MINISTRY OF HIGHER AND SECONDARY SPECIAL EDUCATION OF THE REPUBLIC OF UZBEKISTAN
MINISTRY OF HEALTH OF THE REPUBLIC OF UZBEKISTAN
BUKHARA STATE MEDICAL INSTITUTE NAMED AFTER ABU ALI IBN SINO
DEPARTMENT OF PROPEDYTICS OF INTERNAL DISEASES

"I APPROVE"
Vice-rector for educational work
DSc _____ G.J. Jarilkasinova
«_____» _____2021

FACULTY OF METHODOLOGY FOR 2021-22 ACADEMIC YEAR ON THE SUBJECT OF INTERNAL DISEASES FOR 3-COURSE STUDENTS OF THE FACULTY OF TREATMENT AND MEDICAL PEDAGOGY

Bukhara-2021

3rd year Faculty of Medicine and Medical Pedagogy
The subject "Propaedeutics of Internal Medicine" for students educational and methodical complex

Department of Propaedeutics of Internal Medicine, Bukhara State Medical Institute named after Abu Ali ibn Sino, Ministry of Higher and Secondary Special Education of the Republic of Uzbekistan

Field of knowledge 500000 -Health and social security
Field of education 510000 -Health
Field of study 5510100 -Medical work
5111000 -Professional training
5510200 -Pediatrics
Department of Propaedeutics of Internal Medicine
mudiri, tfd dots. Nurboyev F.E.

Developer: Nurboyev F.E. Nervous system disease department , tf d .
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Reviewers: Axmedova N. Sh. Associate Professor of Internal Medicine, Ph.D.
Nurov U.I. Head of the Department of ENT and Ophthalmology, Associate Professor

Scientific-methodical council of Bukhara Medical Institute
Considered at the 2021 №_2__ meeting

Bukhara - 2021

Mundarija

Annotation

This educational-methodical complex is designed for use by students of 3 courses of treatment, medical pedagogical faculties and is based on a standard program. This educational-methodical complex corresponds to the directions of education 500000 -Health and social security, Education 510000 -Health, Education 5510100 -Medical work. This educational-methodical complex contains the full text of all lectures reflected in the sample program. The complex covers topics such as diseases of the digestive system, urinary system, endocrine system, hematopoiesis and hematopoietic system, bone, muscle and connective tissue diseases, as well as allergies. Assessments on topics (consisting of two tests, concept analysis, case study and practical skills), structured tests on all topics (including complex tests) for the quality of practical training in this complex and the independent preparation and solution of students existing) as well as problematic issues (consisting of a series of questions). In this complex, the texts of all practical training topics for students are covered and questions on the topics are also reflected. The technological map of practical training for practical training is perfectly illuminated. This set of teaching methods provides step-by-step techniques for students to complete their practical skills on all topics so that they can practice and learn. Questions on the topics are provided so that students can prepare for independent work. In the glossary part of the complex I have given an overview of medical terminology and their meaning.

We believe that this set of teaching methods will be convenient for students to use and that they will gain enough knowledge.

Propaedeutics of Internal Medicine »
1 /20 2 2 20 2 science for the academic year

SILLABUSI

A brief description of the science			
university:			
	d k f o Armani line Login		oreign Languages
ıcation:	n 55107 1 00- "Education ion"		
ıcher of the subject (course):	ayevna	**l:**	ru
	with a medical center and franchise education 3,4,5	**ration:**	22
ıdividual schedule:	08.30 14. 50		
	Classroom hours		**ıcation: 65**
ith other disciplines (prerequisites):	with the sciences of human anatomy, normal physiology, general , general physiology, nursing theory, nursing work in therapy, clinical logy.		
	Medicine", "Clinical Pharmacology OXI"		

The content of science	
of science :	**the subject** the subject is to acquaint students with the science of propaedeutics and to them with the methods of medical deontology, iatrogenics, clinical on of patients (interrogation, examination, palpation, percussion, auscultation) ep examination of organs and systems , learning the basic laws of syndromes toms in the system, mastering the skills of laboratory and instinctual on and interpretation of azoles. e : cal techniques or examination methods of syndromes and symptoms ystem step by step training nental control and interpretation of teaching gnostic conclusions based on the results of the examination of nosological forms of internal diseases ciples of treatment of internal diseases
	and teammates; ternal disciplinary rules of the Institute ; during the lesson; formance of assigned homework and independent work assignments; in class; s of classrooms; ohibited; ry, missed classes must be re-learned; ive participation in lessons; ved in the classroom after the teacher; he practical classes should be performed at the required level and reflected in and signed by the teacher. fied with the rating score, 1 day from the date of publication ls Commission.
hip by e-mail	n the faculty and the student can also be done by e-mail, the **issue of**

	nt by phone is not discussed, the assessment is carried out only on the of the institute, in separate rooms and during the lesson. E-mail opening from 15.00 to 20.00

3 . Number of study hours m

	ning load on the classroom (hours)				

The relevance of science

This program is based on the State Educational Standard and the General Practitioner Training Program, designed to teach medical students the subject of propaedeutics of internal medicine.

Nervous system disease symptoms of diseases and syndromes, the diagnosis of diseases of the internal organs q o'llaniladigan diagnosis of the disease and new methods of investigation, q teach the game, and the patient's medical records. Simptomatologiya and diagnostics training q -The private pathology can be divided into teaching, that is, on the one hand, to know the ways and methods of learning, q know a hand lens in far q should not be. Simptomatologiya, diagnostic and pathology basis of an integral Bo orange li q absence of training in albums. The main clinical study of therapeutic diseases, symptoms , etc. Society illustrations. Students diagnostic methods of a particular patient's disease history , etc. See the lil twirling skills. See the clinic in the first days of the student Nervous system disease, one of the functions of the medical deontologiyasi foundations , etc. , to learn am. Propedevtika know later in therapy, but h q a clinical discipline to learn am. Knowledge of the main symptoms of diseases and diagnostic methods, diagnostic methods only q not only therapists, surgeons, gynecologists, neurologist and a clinical specialists h k am able to do.

Bachelor Nervous system disease science, theoretical, methodological, organizational and practical skills or q ali job that is prepared to be a general practitioner, h as well as clinical, laboratory, functional and aids the analysis of the results of inspections q hung diagnosis q Games.

Nervous system disease science students to learn the process of the disease, symptoms, diagnosis, diagnostic, principles, mechanisms for the origin of syndromes to' orange kitty h current time understanding the minds of students, medical ethics and the formation of a h loqi and impregnating deontologiyasi basis.

Nervous system disease science or q ali students are the primary causes of pathological processes in the body, and the mechanisms of their development, the main symptoms and syndromes to' orange kitty, the concept

of physical inspection, inspection, palpation, percussion, hearing, blood pressure, pulse clearly confirm q ECG recording, q on the size of urine, feces, orange am, gastric juice, duodenal ma h dynasty, pleural Condensed q must h h analysis and assess the clinical assessment of clinical death , etc. inadequate resuscitation measures, medical must know the basics of deontology.

Interrelation of science with other subjects in the curriculum and methodological affiliation

The subject " Propaedeutics of internal medicine " is a course included in the block of general professional disciplines and is taught in the 5th, 6th semesters of the 3rd year of medical, medical and pedagogical faculties.

The science of internal medicine propaedeutics is included in the category of clinical sciences, it is taught in all areas of medical undergraduate education. Propaedeutics of internal medicine - is a clinical science. The plan for the implementation of this program talabao'quv planning - vantage of the clinical and scientific training q Fund (normal, normal anatomy, physiology, general science, biology and medical genetics, biochemistry and microbiology science with the vertical integration of pathological anatomy, pathology and physiology, general surgery and farmakalogiya Sciences with horizontal) integration.

The role of science in science, economics and production

Propaedeutics of internal medicine is one of the important disciplines in the formation of the basis of medical knowledge in the general practitioner.

To carry out research activities aimed at studying the prevalence and course of internal diseases of the UAS, early diagnosis, treatment, prevention of the spread of internal diseases among the population and forecasting the course of the disease for the application of professional activities; diagnosis and treatment in treatment and prevention organizations of the health system (therapeutic departments of central district, city, regional, republican hospitals, multidisciplinary central city polyclinics, emergency care centers and stations, maternal and child health care organizations, sanatoriums) It is important to address the main tasks of patients' medical problems with the widespread use of modern methods; including the solution of complex problems in the field of internal medicine at all levels of the health system.

Modern information and pedagogical technologies in science teaching as well as the design of training sessions

The use of advanced and modern teaching methods, the introduction of new information and pedagogical technologies are important for students to master the subject " Propaedeutics of internal medicine ." Textbooks, teaching

aids, lecture notes, handouts, electronic materials, case technologies are used in the study of the subject. Interactive teaching methods (visual, problem-based, author's lectures, two-way analysis, cluster, Venn, Syncway, etc.) are used in lectures and seminars .

On the basis of the principles of pedagogical technology, the teacher of science develops projects of lessons on a subject " Propaedeutics of internal diseases ".

Fan module program (module syllabus)

Full name of the course:	**Propaedeutics of internal diseases**		
Short name of the course:	IKP	Code: IKP	
Chair:	**Propaedeutics of internal diseases**		
Teacher information:	F.I.Sh.	E-mail	
Semester and course duration	5.6 semester 18/18 weeks		
	Fakul - tet	Treatment	tib.ped.
Size of study hours:	**total:**	**250**	**250**
	as well as:		
	report	**32**	**32**
	seminar		
	practical	**130**	**130**
	independent learning	**88**	**88**
Course status	Block of general sciences		
Preliminary preparation:	The course is based on the knowledge of "Anatomy", "Normal Physiology", "Pathological Physiology", "Pathological Anatomy", "Pharmacology", "Histology".		

Subject and content of science:

The purpose of teaching the subject is to acquaint students with the science of propaedeutics and to acquaint students with the methods of medical deontology, iatrogenics, clinical examination of patients (interrogation, examination, palpation, percussion, auscultation), step-by-step examination of organs and systems; ,, is the acquisition of skills in laboratory and instrumental examination and interpretation of members.

The purpose of the science - the main purpose of the program on ICP is to teach future general practitioners (UAS) how to check the condition of internal organs and thus assess the condition of the body, to teach methods of teaching internal medicine in medical institutions.

The task of science is to teach its students:

- syndrome symptoms and learn the basic laws of teeth
- a member of the status of systems and step-step verification learn tooth
- Laboratory and instrumental tests and interpretation of their results
- on the basis of the results of the investigation from the Diag o stick to conclude that you can get ,
- nosologic forms of diseases questions the academic study
- learn the basic principles of treatment of diseases teeth

In the process of mastering the subject **"Propaedeutics of Internal Medicine"** bachelor:

- Anatomy of internal organs;
- Physiology of internal organs;

- responsibilities of internal members;
- have an ***idea of the*** methods of inspection of internal organs ***;***
- Palpation of the chest
- Upka's comparative percussion
- Topographic percussion of Upka
- Upka auscultation
- Palpation of the heart area
- Cardiac percussion (relative boogie boundaries)
- Cardiac percussion (absolute boogie limits)
- Cardiac auscultation
- AD measurement
- Pulse check
- ECG methods
- Normal ECG
- Superficial palpation of the abdomen
- Deep palpation of the abdomen
- Palpation of the liver
- Liver percussion according to Kurlov
- Kidney palpation
- Palpation of lymph nodes
- Palpation and percussion of the spleen
- ***Must have*** analytical ***skills*** ;
- Maintaining a medical history of patients treated in an inpatient setting
- ***Must have*** communication ***skills with*** patients **.**

Thematic structure and content of the course

T/r	**Subject**	**Lecture**	**Practical**	**Mousse is a knock work**
	Faculty of Medicine, Medical Pedagogy			
1.	1. To acquaint students with the task of the subject of ICP. History of development of science , goals and objectives. Types and methods of diagnosis. Basic methods of clinical examination of the patient: subjective and objective Scheme of medical history. Independent work of students with the patient. General examination of the patient: general condition, consciousness, condition, body composition. Examination of the patient by body parts: head, face, neck, skin, subcutaneous fat layers, tumors, muscles, joints, bones, and limbs. Thermometry. Anthropometry. A general understanding of laboratory and instrumental examinations. Importance of modern laboratory and instrumental examination methods.	2/2	6	4
2.	Examination of patients with respiratory diseases. Inquiry. Primary and secondary complaints. Chest examination. Palpation - as a method of objective examination. Palpation of lymph nodes and chest. Percussion as a method of objective examination. Comparative and topographic percussion of the lungs. In norm and pathology.	2	6	3

3.	Auscultation of the lungs. Techniques and rules. Primary (bronchial and vesicular) and secondary (wheezing, crepitation, pleural friction noise) breathing noises. Modern inspection methods . Methods of radiological examination: bronchoscopy, bronchography, tomography. Methods of functional examination of the lungs: spirometry, spirography, pneumotachometry. Examination of sputum.	2	6	4
4.	Impaired bronchial permeability syndrome . Utkirvasurunkalibronchitis. Obstructive and nonobstructive. Flatulence syndrome (bronchial asthma, pulmonary emphysema). Examination of obstructive pulmonary disease . Diagnostics. General concept of etiology and pathogenesis Basic principles of treatment		6	4
5.	Lung tissue hardening (thickening) syndrome (in the example of crouposis and focal pneumonia). Pulmonary cavity syndrome (in the case of pulmonary abscess and bronchiectasis). Diagnostics. General concept of etiology and pathogenesis. Basic principles of treatment.		6	4
6.	Air and fluid accumulation syndrome in the pleural cavity (pleurisy, hydrothorax, pneumothorax). Shortness of breath. Diagnostics. General concept of etiology and pathogenesis Basic principles of treatment. Curation of patients. Write a medical history.		6	4
7.	Methods of examination of patients with cardiovascular diseases. Semiotics. Inquiry: main complaints. Pathogenesis. Examination (general condition, color of skin coatings, swelling, examination of the neck). Diagnostic value. Examination of the heart area and peripheral vascular area. Palpation of the heart area. Diagnostic value.	2	6	4
8.	Cardiac percussion. Determining the relative and absolute limit of heart failure in a healthy person. Heart configuration. Determining the relative and absolute limit of heart failure in pathology. Determination of cardiac configuration in pathology. Diagnostic value. X-ray analysis.		6	4
9.	(To hear the heart auscultation). Auscultation rules. Hear heart tones norm. The order to hear the tones and points. Strengthening the change in tones of cardiovascular pathology (blocked). And the collapse of the split tones. Diagnostic significance. In addition to cardiovascular disease moods .	2	6	4
10.	Cardiac interactions, mechanism of formation, characteristics (in pathology). Pulse check. Features of the pulse in a healthy person and in cardiovascular pathology. Diagnostic value. Blood pressure. Step-by-step measurement of blood pressure. The concept of hypertension and hypotension. Diagnostic value. Modern 3laboratory and instrumental inspection		6	4

	methods.			
11.	Electrocardiography (ECG), recording method and normal ECG analysis. ECG in disorders of cardiac automatism and excitability. ECG with impaired cardiac conduction and contractile function.	2	6	4
12.	The main clinical syndromes. Coronary syndrome. Circulatory insufficiency syndrome. Cardiac asthma, lung swelling. Vascular insufficiency syndrome. Arterial hypertension. The rhythm of the heart failure syndrome. Kardiomegaliya syndrome. A small part of the mining cycle hypertension.		6	4
13	Symptomatology of rheumatic fever and primary rheumatic heart disease. Symptoms and early diagnosis of primary rheumatic heart disease. Mitral regurgitation. Mitral valve insufficiency and stenosis. Diagnostics. Basic principles of treatment.	2	6	4
14	Symptomatology of septic endocarditis. Symptomatology of aortic regurgitation. Aortic valve insufficiency and stenosis. Diagnostics. Basic principles of treatment.	2	6	4
15	Symptomatology of hypertension. The concept of symptomatic hypertension. Diagnostics. Basic principles of treatment. The concept of ischemic heart disease (IHD) . Classification of angina, symptomatology. Symptomatology of myocardial infarction. Diagnostics. Basic principles of treatment. Curation of patients. Record medical history №2.	2	6	4
16	Methods of examination of patients with diseases of the digestive organs. Inquiry. The main complaints. Mechanism of symptoms and diagnostic significance. General examination and examination of the oral cavity, examination of the abdomen. Palpation of the abdominal organs (superficial and deep). Diagnostic value. Modern laboratory and instrumental methods of examination. Basic clinical syndromes.	2	6	4
17	Symptomatology of gastritis (acute and chronic). Symptomatology of gastric and duodenal ulcers. Diagnosis of enteritis and colitis. Basic principles of treatment.		6	4
18	Examination of patients with diseases of the liver and biliary tract. Inquiry. The main complaints, diagnostic value. Examination, liver percussion (according to Kurlov), palpation. Diagnostic value.	2	6	4
19	The main clinical syndromes: jaundice, portal hypertension, liver failure. Modern laboratory and instrumental methods of examination. Diagnosis of chronic hepatitis and cholecystitis. Symptomatology of liver cirrhosis. Basic principles of treatment		6	4
20	Methods of examination of patients with kidney disease. Inquiry, main complaints. Examination,	2	6	4

	palpation of the kidneys, urination (Pasternatsky's symptom). Modern laboratory and instrumental methods of examination. Diagnosis of acute and chronic pyelonephritis and glomerulonephritis. Symptoms of renal failure (acute and chronic). Basic principles of treatment. Crushing medical history № 3.			
21	Methods of examination of patients with diseases of the circulatory system (main complaints, anamnesis, autopsy), palpation of the spleen. Clinical laboratory. General analysis of blood. Basic clinical syndromes. Diagnosis of anemia. Diagnosis of leukemia Basic principles of treatment.	2	6	4
22	Inquiry of patients with diseases of the endocrine glands , anamnesis. Symptomatology of thyroid diseases. Diagnosis of diabetes.	2/2	4	4
Total: 250		**32**	**130**	**88**

Teaching and shooting style:	Lectures, practical classes, independent work (round table, case study, master classes)		
Musta q il works:	Study projects, group presentations, abstracts, cases, reports, crossword puzzles, posters, brochures, essays, etc.		
Time to submit tips and assignments	Days	Time	Aud.
1.			
2.			
3.			

Methods, criteria and procedure for assessing knowledge:

The scores of JN and ON are given in the work program

Evaluation methods	Tests, written assignments, oral questionnaires, presentations, etc.
Monitoring and evaluation of students' knowledge of science	**Forms of control** Types of assessment can be in the form of surveys, oral questions, written assignments, quizzes, or other forms, depending on the nature of the subject. **Criteria for assessing students' knowledge of science**

Ball	**The level of knowledge of the student**
86 - 100 points	- The student is always ready for lessons, very active , well versed in the program materials, able to draw conclusions and decisions, think creatively, apply knowledge in practice ; - The student is able to find new methods and directions that help to find a solution, to understand the essence of the educational material, choosing the scope of application of relevant knowledge in solving creative problems; - The student seeks solutions to the presented

		educational problems, knows and can tell and imagine the program materials.
	71 - 85 ball	- The student, knowing the relevance of the studied phenomena and the ability to describe the object, is able to solve problems by revealing the causal relationship, to connect the studied theoretical knowledge with practice and to make independent observations; - the ability to apply the content of knowledge and skills, to solve problems of the same type, to write and remember, to apply knowledge in practice; - The student is prepared for the lessons, knows the program materials, understands the essence and has imagination.
	55 - 70 ball	- is able to perform tasks on the basis of what the student has heard, the examples given to them, the algorithms and instructions provided, understands the essence; - The student is able to distinguish a certain object on the basis of a number of signs, describe it and explain the learning material and have an idea.
	0 - 54 points	- The student has no imagination; - The student does not know the program materials.
Science lectures, videos: Videos on each topic will be shown (OUM)		
Glossaries: A glossary has been created for each topic.		
Information resource database: **Basic literature** 1. Gadaev A ; Karimov M.Sh ..; " Propaedeutics of internal diseases " T. 2012; 2. Muxin N.A., Moiseev V.I. "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko V.X .. Grebenev A.L “ Propedevtika vnutrennix bolezney ” , M.1989		

MAIN PART
The content of science lectures

for medical and medical pedagogy faculties

1-module. Introduction. History of the development of internal medicine. The science of propaedeutics of internal medicine. Purpose, tasks. Understanding semiotics, symptomatology, and diagnosis.

Nervous system disease symptoms of diseases and syndromes, the diagnosis of diseases of the internal organs q o'llaniladigan diagnosis of the disease and new methods of investigation, q teach the game, and the patient's medical records. Simptomatologiya and diagnostics training q -The private pathology can be divided into teaching, that is, on the

one hand, to know the ways and methods of learning, q know a hand lens in far q should not be.

2-module. Methods of clinical examination of patients. Inquiry. Complaints. Life history. Physical examination method : Examination, pal hats, percussion, Auski unreliable.

Simptomatologiya, diagnostic and pathology basis of an integral Bo orange li q absence of training in albums. The main clinical study of therapeutic diseases, symptoms , etc. Society illustrations. Students diagnostic methods of a particular patient's disease history , etc. See the lil twirling skills. See the clinic in the first days of the student Nervous system disease, one of the functions of the medical deontologiyasi foundations , etc. , to learn am. Propedevtika know later in therapy, but h q a clinical discipline to learn am. Knowledge of the main symptoms of diseases and diagnostic methods, diagnostic methods only q not only therapists, surgeons, gynecologists, neurologist and a clinical specialists h k am able to do.

3-module. Examination of patients with diseases of the respiratory system . Asking - inquiry. Forgiveness in the fall. Methods of objective examination by palpation, percussion and auscultation .

Primary and secondary respiratory interactions.

Respiratory diseases o orange Riga methods of questioning, the main complaints of patients and pathogenesis. Q afasini eye - breast examination, palpation. See the chest o orange settings (the nature, location, duration, intensity, and to reverse the q exchange), h ansirash (physiological, pathological, objective, subjective, and mixed; eksperator, insperator, mixed). Cough (q uru q , damp, duration, appearance and q ti). Bal orange divorce am - nature, mi q , the size of the orange am sick divorce , etc. Part of the Bo orange liqligi. Q on the cutting (the nose, lungs, burun- h q Umdat, q izilo'ngachdan medal), q h on the spit, the duration of a diagnostic role. Disorders of nasal breathing. Yu q to check the high respiratory tract. Q additional complaints: fever, h h olsizlik appetites saw a decrease. Anamnesis of disease and h life. Q afasini examination, chest q sequence of rules. Breast q afasi form of asymmetric and deformed, synchronous h arakatlanishi. Involvement of respiratory muscles in respiration and q . Breathing type. The number of breaths per minute. Impairment of respiratory rhythm on examination and the presence of x congestion. Breast q afasini pathological forms. Types of asthma (norm and pathology). Chest q palpation technique (sound vibration, chest q aphasia resistance). Pulmonary percussion technique. Q Political Percussion Sequence. Percussion sound changes in norm and pathology. Conditional topographic lines of the chest q aphasia. Topographic percussion. See the lungs, but lower high and the lower limit of height, width, q IrrÃ h arakatchanligini identification q care. H H in different physiological and pathological changes in the cases of lung border. Lung auscultation techniques and q rules. (Vesicular and bronchial breathing)

interaction, the mechanism of occurrence, change: increase and weaken the (physiological and pathological) and a diagnostic h role. The mechanism appears to be a wet (q rate, q see the low, and the high tonal sounds, moist and fresh, small, medium, large bubbles), and the associated localization and h q exchange, a diagnostic role. Bronchophonia. Respiratory patients with functional and instrumental methods , etc. belief. Spirometry, pneumotachometry, oxyhemotherapy. Methods of X-ray examination of the lungs, bronchoscopy, bronchography, tomography. Spirometry. Spirography, pneumotachometry, oxyhemometry, pneumotachography. Bal orange am, and the pleural Condensed h q check, make sure you have a diagnostic role. Tarnssudat differs from exudate q i. Interpretation of the results q rights.

4-module. Observed in patients with diseases of the respiratory system basic clinical syndromes.

Bronchial permeability syndrome, pathogenesis. Complaints, review. Palpation. Percussion, auscultation. Symptoms of acute and chronic bronchitis, the role of the profession in its development. Adverse effects and pathogenesis of smoking on the respiratory organs. See the lung to' cut h weather increased the pathogenesis of the syndrome. Complaints, review. Palpation. Percussion, auscultation. Obstructive lung diseases o orange Riga to control patients. Bronchial asthma and pulmonary emphysema. See the lung to' cut compression syndrome pathogenesis. Complaints, examination, palpation, percussion, auscultation. See the lung to' cut Yalla orange depletion of patients with the disease teshkirish. Croupous and will see with symptoms of pneumonia. See the pleural space orange Ida h o BC and liquid up the accumulation of pathogenesis of the syndrome. Complaints, examination palpation, percussion, auscultation. Q rate, q , and exudative bleeding simptomatologiyasi. Hydrothorax, pneumothorax symptomatology. Types of pneumothorax (open q , closed, q op q o q li). Pathogenesis of hollow q syndrome in the lungs . Complaints, examination, palpation, percussion, auscultation. Bronchiectasis, symptomatology of lung abscess.

5-module. Methods of subjective and objective examination of patients with diseases of the cardiovascular system.

Q on patients with cardiovascular disease control methods. Inquiry, review. Heart sa h wear, examine the peripheral blood vessels. Heart sa h wear palpation q rights.

Inquiry. The main complaints, their pathogenesis. Heart sa h o in the field of orange settings q . O orange pi QH to be hung on the nature of the mechanism of localization, intensity, duration, irradiatsiyasi, to reverse the q exchange, the night o orange q is no reduction in the settings . H ansation: mechanism of occurrence, intensity. Cardiac asthma. Heart Rate: continuity, h urujliligi, intensity, duration, etc. See

the ayajonga, rape , etc. Part of the change of the hunt, q art Bo orange li q levels. Heart sa h h really had no hand in various parts of the body, pulsating sense of anticipation. See the cough, spit on the nature of the mechanism appears to be a diagnostic h role. Review. Body structure. Es h ushi h olati. See the color of the skin, Jezer, Q , earn a bruise. Tsianozini heart lung tsianozidan far q i. Oh heart tissue appears to be the mechanism of localization, the diagnostic role of renal tumors are far q i.

Neck vessels examination. Venous pulsation, swelling. Epigastric pulsation. Pulsation of capillaries. Do you come q h causes a diagnostic role. Heart sa h corrosion inspections. Heart fat. Desert q i and moved acoustical heart. Desert qq I moved the definition of the norm: the location, strength, height, to reverse the q exchange. Pathological changes in the motive of choq qq i. Motivation of negative cho qq i. Systolic and diastolic full implementation q care. Symptoms of "cat wheezing". Epigastric factor palpation, heart and liver factor far q care.

Cardiac percussion technique and q . See the heart of relative franchise Calls lock up the border and vessels identified q Confirm input. Ani q lash of the heart . See the heart of relative franchise Calls up the border. Pathology Heart franchise Calls q % change: respiratory and cardiac borders q h on cardiovascular disease, body condition changes.

See the absolute heart of franchising Calls q up to define the boundaries of q Confirm input. See the respiratory and heart on the heart-vascular diseases franchisee Calls q % change of the border. This is a diagnostic changes , etc. role.

6-module. Cardiac auscultation: normal and pathological tones. Heart murmurs.

Cardiac auscultation. Q auscultation rules. Q afasini the front wall of the chest q op q Q s projection and places to hear them. Auskultatsiyasi heart: the body to breath a different phase, different cases, peace h h h, conditions, and physical planners q after work. Auskultatsiyasida systolic heart failure and diastolaning far q i. Heart sounds h a q Ida (1, 2, 3, 4), etc. will be hanging mechanism. The main properties of sounds: timbre, power. Increase and decrease of basic sounds. Basic (1, 2, 3, 4) and q additional mitral q op q o q opening sound, pericardial tone, division of sounds, hesitation, rhythm, changes in their pathology. Horse dupuri rhythm, quail singing rhythm, pendulum rhythm, embryocardia. Tachycardia, bradycardia, arrhythmia. The concept of normal FKG, EXOKG h . See the phonocardiography and diagnosis play a role h h a q specific concepts. The role of Polikardiografik, EXOKG investigation and a h h h a q specific concept, a diagnostic role, Interpretation q rights.

7-module. Electrocardiography. ECG changes in cardiac hypertrophy . ECG changes in acute myocardial infarction . Arrhythmias . About fibrillation of the heart

NormalEKG , training q ishusuli (rhythm , electric learning q i , Yu S , Q spaced teeth). Diagnostic and clinical role of a speed violation in a heart beat .

8-module. Rheumatic fever . Heart defects: mitral valve insufficiency. Symptomatology of left atrioventricular foramen narrowing.

Q Confirmation . Q op q Q s anatomy h a q specific concept . Yurakshov q ins , etc. osilbo'lishmexanizmi . Classification. Functio nal and organic high q ins far q i. See the heart of phase noise due ins. Systolic and diastolic high q nests: protodiastolik, mezodiastolik, presistolik total, timbre, duration. Cobra q ins the best playback. See the pericardium Alan high q Hubie plevroperikardial high q nests. Auscultation of arteries and veins. Traube two sounds, pathological, diastolic Vinagradov- Dyuraze shov q ini. Vascular examination. Q rules and methods. The main characteristic of the pulse. See the arterial q measure the pressure on tours, methods and techniques. Maximum, minimum, medium pressure. “Random” and basic pressure. See the intention of hypo- and hypertension o h a specific concept. Oxyllography, sphygmography, capillary microscopy. Q cashless payment ini speed. Phlebography. See the venous pressure, identifying the method of authentication. Q on Q , a diagnostic for measuring the speed of business , etc. role. Circulating q on mi q drug. See the heart q on me q and earn peripheral resistance. Orthostatic test: a breathing test to stop breathing.

Etiology and pathogenesis of rheumatism, examination of patients. Symptomatology of rheumatism and primary rheumatoid arthritis.

Heart defects. Etiology, pathogenesis of acquired heart disease. Q op q Q mitral insufficiency, gemodinamikasi, simptomatologiyasi (complaint, inspection, palpation, percussion, auscultation). Mitral stenosis. Complaints of hemodynamics. Review. Palpation, percussion, auscultation. ECG, FKG, and EXOK G changes in mitral regurgitation .

9-module. Aortic defects . Symptomatology of tricuspid valve insufficiency.

Aortal q op q o q insufficiency. Hemodynamics. Complaints. Review. Pa lpation, percussion, auscultation. Hemodynamics, symptomatology of aortic stenosis (complaint, examination, palpation, percussion, auscultation ECG, EXOK G , FKG)

The concept of circulatory failure h a q . The mechanism of heart failure. Acute and chronic heart failure syndrome. Left q pixel failure. Clinical manifestations (cardiac asthma, diagnosis of lung tumors, emergency care). Chronic heart failure stages q hearts (ischemia and decompensated). Mechanism of heart failure compensation.

10-module. Hypertension . Ischemic heart disease , heart colic, myocardial infarction, kardioskleroz simptomatologiyasi .

Hypertension h a q specific concepts. Etiology. Pathogenesis. Symptomatology. Complaints, examination, palpation, percussion, auscultation. Laboratory - instrumental diagnostics. Hypertension h a q specific understanding of emergency. Symptomatic hypertension (renal, endocrine, central, hemodynamic).

Ischemic heart disease h a q specific concepts. Etiology. Pathogenesis, symptomatology of angina pectoris classification. ECG diagnostics. Angina pectoris h urujini stop.

Myocardial infarction. Etiology. Pathogenesis. Symptomatology. ECG diagnosis (stage q hearts and localization). Ambulance.

11-module. Methods of subjective and objective examination of patients with diseases of the digestive organs . Acute and chronic gastritis. Ya ra disease. Gastric cancer and methods of early detection. Symptomatology of chronic enteritis and colitis.

H members resolve diseases o orange Riga patients control methods. Inquiry. The main complaints o orange settings q : h hanging mechanism, localization, irradiatsiyasi hunt, q art Bo orange li q , diarrhea linked to the nature of stained orange with q see the intensity levels, throughout the day, when there will continue to be. Oh the orange is no settings q : self-medication or to drink, hot qq reed, say q q and after. Q usish formation mechanism, and h q ti (or agency or hunting q acceptance, and then - in the early evening). Q usu q composition, mi q drug, q an intervention, a h latli q usu q , nausea - mechanism, diagnostic a h significance , occurrence and q ti, number, intensity, treatment. Thyme - h weather, hunting q appear to be at, and q ti, intensity, body , etc. Part of the Bo orange li q levels. Ji orange ildon q nausea, conditions appear to be the duration of the hunt, q art Bo orange li q levels. Ishtar is no A: top, middle, h , o orange taxirlik in a cookie, o see the orange trail off, no h , filling the senses of taste, saliva, Q , ' not knowing m. Swallowing: o orange settings q L q difficult (disfagiya). Diagnostic a h amiyati. Flatulence, q to take place in the orange yldirashi, q medical checkups o orange Weight. Bowel function: number, nature of diarrhea, mi q drug, q an intervention. The cause of diarrhea, diagnostic a h amiyati. Diarrhea q roots mechanism. Oh the orange trail of empty orange Examination of the INI, tonsils, mucous q Picture teeth. Sir John, humidity, color, language, nature, orange hearts, karachlanganligi, the presence of the crack.

Q pixel vertical, horizontal , etc. case examination. Q pixel topographical field h to be raised. See the respiratory Act to take place in the walls of h Movement. Q development to take place in the front surface of the side wall of the venous collaterals. Jellyfish head pigmentation, hernia soup q ozone peristalsis, antiperestalitis. Palpation. Superficial palpation method. Skin and subcutaneous

or PUBLICATIONS Picture h condition. Chur, clearly separate the muscles q care. Shetkin-Blumberg symptom, methodical, sliding, Obraztsov-Strajesko method palpation. The sequence of torque at 4 colon acoustical member of location, mobility, o orange riq, thickness, size, surface, orange yldirash substance no q ligi. See the liver, spleen , kidney acoustical. Q soup ozone Festivals and grass control, Interpretation of the results obtained. Methods of duodenal sensing, 3 portsiyasini the pile, micro, and macroscopic examination, diagnostic h a role.

Methods of instrumeital examination of H azm members. (radiology, esophagogastroduodenofibroscopy, rectoromanoscopy). The soup q h ozone-intestinal tract radiographic examination, a diagnostic role. Q soup ozone tract endoscopic
examination. Gastroscopy. Colonoscopy. Rectoromanoscopy. Advantages of endoscopy over radiological examination.

Gastritis, etiology, pathogenesis and classification. Symptomatology of acute and chronic gastritis. Q soup of ozone and 12 finger qli ulcer disease etiology. Symptomatology of pathogenesis.

Etiology, pathogenesis, symptomatology of enteritis and colitis symptomatology. Coprologic examination. A diagnostic role and Interpretation of the results h and q rights.

12-module. Patients with diseases of the liver and biliary tract of subjective and objective methods. The main clinical syndromes: jaundice, portal hypertension, liver failure. Symptomatology of hepatitis and liver cirrhosis

Inquiry into patients with liver and biliary tract disease. Main complaints: mechanism of pain formation, localization, nature, duration, irradiation, causes, intensification, attenuation, cessation of pain, diagnostic significance. Dyspepsia: changes in appetite, nausea, belching, nausea, vomiting, bloating, nausea. Jaundice: skin discoloration, urine, stool change. Diagnostic value. Itchy skin. Other manifestations of bleeding and hemorrhagic diathesis. Abdominal enlargement. Review. General appearance, changes in subcutaneous fat. Skin changes: jaundice, vascular asterisks, liver plaque, drumsticks, gynecomastia, erythema, diagnostic significance. Abdominal examination: ascites or limited (enlargement of the spleen, liver gallbladder). Navel position. Formation of venous nodules on the anterior wall of the abdomen. Liver palpation, edge, consistency, face, pain. Abdominal percussion, detection of ascites. Determination of liver size according to Kurlov.

Understanding of the main clinical syndromes jaundice portal hypertension, liver failure. Jaundice. Types of jaundice. Mechanism and pathogenesis of jaundice. Scheme of bilirubin exchange. Symptomatology of portal hypertension. Concepts and importance of liver pigment, carbohydrate, fat metabolism (biochemical blood test), micronutrient testing (iron, copper). Enzyme examination, detoxification, excretory activity of the liver,

radioisotope examination of the liver, radiometric examination, methods of liver scanning. Q fire Opinel scanning methods. X-ray examination: cholecystography, cholegraphy, computed tomography.

Mechanism and symptomatology of liver failure. Symptomatology of chronic cholecystitis and hepatitis. Complaints, examination, palpation, tingling. Aetiology and pathogenesis of diseases h a q specific tasa'vur. Symptomatology of liver cirrhosis. Complaints, examination, palpation, tingling. See the aetiology and pathogenesis of diseases h a general idea about .

13-module. Methods of subjective and objective examination of patients with kidney disease . The main clinical syndromes: urinary, nephrotic, hypertensive. Symptomatology of acute glomerulonephritis and pyelonephritis.

Inquiry, examination, palpation, general understanding of the main clinical syndromes, the main complaints and their pathogenesis in patients with kidney disease. Pains, their analysis. Location, distribution of tumors. Dysuric disorders. Methods of laboratory examination of urine. Functional kidney tests (Zimnitsskiy, Nicheporenko, Reberga). Interpretation of the results q rights.

Symptomatology of nephritis. Aetiology and pathogenesis of acute and chronic pyelonephritis, glomerulonephritis h a q a common understanding about.

14-module. Mining system of patients with diseases of the subjective and objective methods . Clinical, laboratory and instrumental examination methods . Symptomatology of anemia and leukemia.

Methods of interrogation and examination of patients with blood diseases (complaints, collection of medical history, hereditary factors). Pain in throat, bones, under right and left ribs. Bleeding: from the nose, gums, gastrointestinal tract, uterus and other organs. Itchy skin, malaria, itching. Changes in the skin and mucous membranes, enlargement of regional lymph nodes. Palpation of lymph nodes. Liver, spleen palpation, consistency, surface and edge, pain. Petechiae. Q verify the moment. Q analyzed on a clinical role and Interpretation of the results h and q rights.

Symptomatology of anemia. Low q decades. The etiology and pathogenesis of TB Calls on the general concepts. Q and use steps. Iron deficiency, postgemorragik, hemolytic q and use simptomatologiyasi. Symptomatology of leukemia. The etiology and pathogenesis to' Calls on the general concepts. Classification of leukemias. Symptomatology of acute and chronic (myelogenous leukemia and lymphocytic leukemia).

15-module. Methods of subjective and objective examination of patients with diseases of the endocrine system . Q Symptomatology of thyroid disease . Symptomatology of diabetes.

Diseases of the glands of internal secretion o Calls Riga questioning (complaints, medical history of patients yi orange , hereditary factors). Complaints: h olsizlik, weight loss, obesity, chan q Q appetites h h a fever condition, if q high q orange skin aluvchanlik, q , heart rate, skin q speck, see the change in color. H penis. Es- h ushi. Change in height and its proportions. Gender and age signs. Q skin speck change. Presence of rashes, stretch marks on the skin. Subcutaneous or orange klechatkasining and even reverse the development of q Exchange. Tumors. H weakness. Changes in the skeletal system. Q identifying symptoms of the appearance of the names of the parts of the body q care. H change in the face, neck, so a change of wear. Q q onsimon diseases. Symptomatology of hypo and hyperthermia. Q overactive thyroid or diabetes, simptomatologiyasi approaches. Diabetic and hypoglycemic coma to' orange on the concept. Basics of ambulance.

15-module. Methods of subjective and objective examination of patients with systemic diseases of the connective tissue . Rheumatoid arthritis, systemic lupus erythematosus, symptomatology of scleroderma.

Practical training

On the organization of practical training instructions and recommendations

The following didactic principles are followed in conducting practical training:

clearly define the purpose of practical training;

to arouse students' interest in opportunities to deepen their knowledge of innovative pedagogical activities of the teacher;

providing the student with the opportunity to achieve the result independently;

theoretical and methodological preparation of students;

practical training is not only a source of completion of knowledge on a specific topic, but also a source of educating students.

The list of practical orange Typo scenario

Tasks of propaedeutics of internal diseases . Patient examination procedure .

- Scheme of medical history .
- The total patient care training setups to forgive .
- Temperature curvature. Anthropometry .
- As a method of palpation examination .

- Percussion is an objective examination method .
- Auscultation .
- Methods of functional and instrumental examination of patients .
- Laboratory classes .
- ECG acquisition and analysis .
- Respiratory system .
- My heart q on vascular system
- Digestive system .
- Urinary system.
- Q on system .
- Endocrine system .
- Connective tissue diseases .
- The concept of allergic diseases .

Instructions for the organization of laboratory work

Science Laboratory works on the training provided for in the plan .

Methodical instructions on the organization of course work

It is planned to check the medical history in the areas of treatment and medical pedagogy.

The form and content of the organization of independent education

It is recommended to use the following forms in the organization of independent study of the subject "IKP", taking into account the characteristics of a particular subject and is assessed as a current control:

1) preparation of abstracts (abstracts, presentations) **on topics .** Such a method, which helps to master the theoretical material, helps to draw more attention to the teaching material. Student syllabus facilitates the preparation for various control tasks, saves time ;

2) learning to work with the unit and automated control systems. Students put their knowledge during the lectures and practical exercises, to prepare for the audit recommended electronic resources , innovative teaching project designs, and self-control test assignments etc. ;

3) work with additional literature on science. In addition to the recommended basic literature, students use additional educational, scientific literature on the topics given for independent study. At the same time Russian and x encouraged the use of foreign languages, literature ;

4) Use of the INTERNET. Mastering science topics, finding INTERNET sources on the topic in the preparation of abstracts , presentations , working with them is encouraged by additional rating points in all types of control ;

5) development and participation in thematic issues, case studies and educational projects ;
6) collection of material on the basis of types of practice, finding solutions to existing problems in practice, preparation of reports;
7) preparation and participation in theses and articles at scientific seminars and conferences ;
Students will be able to complete homework assignments, study new knowledge independently, search for and find the necessary information, collect information and conduct research using the Internet, prepare scientific articles (theses) and reports within the scientific circle or independently using scientific sources. deepens the acquired knowledge, develops their independent thinking and creative ability. Homework is checked and evaluated by the teacher conducting the practical training, and the level of mastery of abstracts and topics is **checked** and evaluated by the teacher conducting the lectures in each lesson.
A set of guidelines and recommendations for the organization of independent work, a case study, a set of situational issues will be developed. It provides students with practical assignments on the main topics of the lecture, case-study methods and tasks for independent work.

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№	TMI topic	hours
1.	Acute respiratory distress syndrome	3
2.	Asthmatic status.	3
3.	O pka emphysema.	3
4.	Diagnosis of obstructive respiratory failure.	3
5.	Diagnosis of bronchiectasis.	3
6.	Upka abscess.	3
7.	Heart disease as a result of lung disease.	3

8.	Infectious ecocarditis.	3
9.	Hypertensive crises.	3
10.	Hypotension.	3
11.	Acute coronary syndrome.	3
12.	Acute mining circulation failure.	3
13.	Chronic mining failure.	3
14.	Human helminthiasis	3
15.	Immunopathology of the gastrointestinal tract	3
16.	Chronic pancreatitis.	3
17.	Reflux esophagitis.	3
18.	Immunopathology of kidney disease.	3
19.	Tubulointerstitial nephropathy.	3
20.	Immunopathology of rheumatoid arthritis.	3
21.	Anemia (iron recognized q is anemia)	3
22.	Diabetes mellitus. Diabetic and hypoglycemic coma	3
23.	Rheumatoid arthritis.	3
24.	Thyrotoxicosis	3
25.	Symptomatology of Addison's disease	3
26.	Symptomatology of Bexteerev's disease	3
27.	Comparative diagnosis of joint syndrome	2
28.	Comparative diagnosis of hyper and hypoglycemic comas	2
29.	Leukemias	2
30.	Symptomatology of erythema	2
31	Diagnostic criteria for systemic lupus erythematosus	2

Total 88 hours

List of used literature

Basic literature

1. Gadaev A ; Karimov M.Sh ..; " Propaedeutics of internal diseases " T. 2012;

2. Muxin N.A., Moiseev V.I. "Propedevtika vnutrennix bolezney", M. 2000.
3. Vasilenko V.X .. Grebenev A.L “ Propedevtika vnutrennix bolezney ” , M.1989

Additional literature

1. Grebenev A.L. "Propedevtika vnutrennix bolezney", M. 2001.
2. Strutynskiy A.V. “Osnovy semiotiki zabolevaniy vn u re n nix organov” M. 2004. MEDpress-inform.
3. Ka rab a eva R.A. Practicum on the propagation of internal diseases, 1992.
4. Therapy per. s angl. \ pod red. Chuchalina M. 1997.
5. Geotar M. Terapevticheskiy spravochnik Vashingtonskogo Universiteta, per. s angl. -1996.
6. Textbook. Harrisons principles of internal medicine. Fauci A. Braunwald E ed. McGraw-Hill, 1998.
7. Textbook of internal medicine. William N. Kelley ed. Lippincott - Ravenpublishers , 1997.
8. Translation from English under the editorship of acad. RAMN V.T.Ivashkina “Internal diseases of Davidson” Geotar M. 2009

Internet site symbols :

1. www.tma.uz
2. www . ziyonet . uz
3. www.medlincs.ru
4. www . medbook . ru .

MINISTRY OF HIGHER AND SECONDARY SPECIAL EDUCATION OF THE REPUBLIC OF UZBEKISTAN
MINISTRY OF HEALTH OF THE REPUBLIC OF UZBEKISTAN
BUKHARA STATE MEDICAL INSTITUTE NAMED AFTER ABU ALI IBN SINO
DEPARTMENT OF PROPEDYTICS OF INTERNAL DISEASES

Training department "I confirm"
Registered by the Vice Rector for Academic Affairs
olindi № _____ tfn , dots _____ G.J. Jarilkasinova
«_____» _____20 21y . “_____” _____ 2021 y

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MODULE FOR PROPEDYTICS OF INTERNAL DISEASES
WORKING CURRICULUM 2021-2022 ACADEMIC YEAR

Course of Study:

Field of knowledge :	**50 0000**	**- Health and social security**
Field of study :	**51 0000**	**- Healthcare**
Field of education :	**5510 7 00**	**- Therapeutic work**
	5111000	**- Vocational education**

Course 3
Hours are 155 hours
Including:
Lecture 20 hours
Practical training 24 hours
Clinical training 46 hours
Independent work 65 hours

Bukhara - 2021

The working curriculum of the science has been developed in accordance with the curriculum, working curriculum and syllabus.

Developer:
N a rziyev Sh. S. - Senior Lecturer of the Department of Propaedeutics of Internal Medicine, Ph.D.

Reviewer:

Axmedova N.Sh. - Associate Professor of Faculty and Hospital Therapy, Hematology and Clinical Laboratory

This working program is based on the curriculum and syllabus 5510700 - medical work, 5111000 - Vocational education and was discussed and approved at the meeting of the department.
Statement № _____ «_____» _____ 2021y.

Chair holder : tfd, prof. _____ Nurboyev FE
(signature)

Head of FUK: Oblokulov A.R. - Infectious diseases, epidemiology, skin and genital diseases
chair of the department, t.fd, associate professor _____

This working program is based on the curriculum and syllabus 5510700 - medical work, 5111000 - Vocational education and was discussed and approved by the Central Methodological Council of Bukhara State Medical Institute.

Protocol № _____ «_____» _____ 202 1 y.

Stylist: Odilova R. H.

INTRODUCTION

Internal medicine propaedeutics teaches the symptoms and syndromes of all diseases, diseases and new diagnostic methods used in the diagnosis of diseases of internal organs, making a diagnosis and writing a medical report of the patient. The teaching of symptomatology and diagnostics is inseparable from the teaching of specific pathology, i.e., there must be no distinction between ways of learning and methods of cognition on the one hand, and objective cognition on the other. The basics of symptomatology, diagnosis, and specific pathology should be studied in an integral relationship. The study of major therapeutic diseases is an illustration of the clinical significance of symptoms. Students become accustomed to diagnostic methods to analyze a patient's medical history. From the first days of work in the clinic, the student should also learn the basics of medical deontology, which is one of the tasks of the propaedeutics of internal medicine. Knowledge of propaedeutics is necessary for the subsequent study of other clinical disciplines as well as therapy. Knowledge of the symptoms of the disease and the basic diagnostic methods, diagnostic methods should be available not only to the therapist, but also to the surgeon, gynecologist, neuropathologist and all other clinical specialists.

The bachelor prepares for independent work through the acquisition of theoretical, methodological, organizational and practical skills in the field of propaedeutics of internal medicine, as well as their diagnosis by analyzing the results of clinical, laboratory, functional and instrumental examinations. learns to put.

In the process of studying the science of propaedeutics of internal medicine, the formation of modern understanding of the principles of disease, diagnosis, diagnostic principles, symptoms, syndromes in students is to inculcate in students the basics of medical ethics and ethics and deontology.

Through the science of propaedeutics of internal medicine, the student conducts a physical examination of the causes of the main pathological processes in the body and the concepts of the main symptoms and syndromes of their development, examination, palpation, palpitations, hearing, blood pressure measurement, pulse detection, ECG general analysis and clinical evaluation of blood, urine, feces, sputum, gastric juice, duodenal products, pleural fluid, resuscitation in case of clinical death, knowledge of the basics of medical deontology.

The teaching of internal medicine propaedeutics is based on vertical integration with normal anatomy, normal physiology, general biology and medical genetics, biochemistry and microbiology, and horizontal integration with pathological anatomy, pathological physiology, general surgery and pharmacology.

Objectives and tasks of the working training module

The purpose of teaching the subject is to acquaint students with the science of propaedeutics and to acquaint them with the methods of medical deontology, iatrogeny, clinical examination of patients (interrogation, examination, palpation, percussion, auscultation) organs and systems step by step. examination, study of the basic laws of syndromes and symptoms in the system, laboratory and instrumental examination of the organs and the acquisition of interpersonal skills.

Vazifasi- science o ' rganuvchilarga:

students to the doctor to check the equipment or methods o ' rgatish

See the syndrome and the symptoms basics o ' rgatish

See the organs and systems adam b a- q step verification o ' rgatish

See the members of laboratory and instrumental investigation and interpretation rights o ' rgatish

Based on the results of the investigation from the Diag o summary of the stick Calls ya know,

to study the questions of nosological forms of internal diseases

to teach the basic principles of treatment of internal diseases

Science students of the imagination, knowledge, skills and qualification requirements for the right to

The main methods of clinical diagnosis are examination, knowledge of palpation, percussion, auscultation and their application in practice. Patients should be interviewed independently and have an idea of the etiology and pathogenesis of pathological processes in the body.

- Fundamentals of medical deontology.
- have an understanding of the main symptoms and syndromes of diseases.

Additional diagnostic lab you want to delete ratoriya analyzes analysis of knowledge and access to them;

- Basic diagnosis of common, typical internal diseases
- have an understanding of first aid measures and have the skills and competencies to keep basic medical records.

The interrelationship and methodological coherence of science with other disciplines in the curriculum

The subject of propaedeutics of internal medicine is a medical-clinical discipline, taught in the 5th and 6th semesters. The program is planned to implement the curriculum mathematical and medical -ilmiy (anayomiyasi and pathological anatomy, normal and pathological physiology, pharmacology) studies necessary knowledge and skills to be able to up the required di.

The role of science in science and production

General practitioners propedevtika science directory in the preparation of a ifican T elicits. Because this subject teaches bachelors how to examine patients (collection of anamnesis, examination, palpation, percussion, auscultation) and in-depth methods of laboratory instrumental examination.

The modern information and science teacher from K Technologies

The use of innovative methods of education, the introduction of new pedagogical, information and Internet technologies play an important role in the study of the propaedeutics of internal medicine. It is recommended to use teaching and methodological aids (textbooks, teaching and methodological manuals, module assignments, lecture notes, handouts, computer programs, electronic materials and ECG, video systems) in mastering the subject. A variety of methods and tools can be used in lectures and practical classes, in particular, brainstorming, clustering, practical work and didactic games, portfolios, case studies, as well as computer programs, Internet systems.

Person-centered education. The essence of this education is the full development of all participants in the educational process. This implies that when designing education, of course, the approach should be based not on the identity of a particular learner, but primarily on learning objectives related to future professional activities.

Systematic approach. Educational technology should embody all the features of the system: the logic of the process, the interconnectedness of all its links, the integrity.

An activity-oriented approach. Represents education aimed at the formation of process qualities of the individual, the activation and intensification of the activities of the learner, the discovery of all his abilities and capabilities, initiative in the learning process.

Dialogic approach. This approach emphasizes the need to build learning relationships. As a result, a person’s creative activities such as self-activation and self-expression are enhanced.

Organizing collaborative learning. It emphasizes the need to focus on the introduction of collaboration in shaping the content of democratic, equitable, educative and recipient activities and in evaluating the results achieved.
Problem-based learning. The problematic way of presenting educational content activates the learner's activity. At the same time, the objective contradiction of scientific knowledge and the creative application of methods of its solution form and develop a dialectical observation, as a result of which the student is provided with independent creative activity.
The use of modern tools and methods of presenting information - the introduction of new computer and information technologies in the educational process.
Teaching methods and techniques. Lecture (introduction, thematic, visualization), problem-based learning, case-study methods, practical work.
Forms of teaching organization: frontal, collective and group based on dialogue, polylogue, communication, collaboration and mutual learning.
Teaching aids. Along with traditional forms of teaching (textbooks, lectures) - computer and information technology.
Methods of communication: direct interactions with the audience based on operational feedback .

Methods and means of feedback: observation, blitz-questionnaire, intermediate, diagnostics of training based on the analysis of the results of current and legal control.
Methods and means of control: planning of lessons in the form of technological maps, which determine the stages of training, the interaction of teacher and student in achieving the goal, not only classroom training, but also independent work outside the classroom. controls.
Monitoring and evaluation: systematic monitoring of learning outcomes in the classroom and throughout the lesson. Assessment of students' knowledge using OSKI at the end of the cycle.

The scope of the subject, the content of the lessons and the rating assessment

semester	Total hours	Classroom hours	report	Practical training - lot	Clinical training	Independent Education	Control type		
							Rating score		
							j / n	o / n	ya / n
5	155	90	20	24	46	65	0.5	0.2	0.3

Interactive methods

1. "Assistment" method
The paper is divided into 4 in the hand of the teacher-trainer.
Describe the symptom of the subject.
Solve the problem.
Test solution.
Completion of practical skills.

2. "Chamomile" method :
Prepare a model of chamomile in advance on a poster, board.

Write a question on the reverse side of the chamomile leaves and write “prize” or “you don’t have to answer the question, relax” on 2-3 of them. The group members take turns taking the chamomile leaves and answer the relevant question.

3. "Find a surprise" game:

Before the lesson begins, a question is written on a piece of paper and pasted in an invisible place.

When participants enter the classroom, some may find them a gift
it is said. The questions on the papers found under the table are answered in turn.

4. The “brainstorming” method.

The main rules of the method:

- Lack of warnings and criticisms that hinder the formation of opinions

-take into account that the more unnatural the idea, the better

-try to get more offers

-combination and development of ideas

- Give a brief description without giving a detailed explanation

-distribute to those who share and rework the ideas of the group

This method helps students to substantiate, defend, and think independently.

5. "Rotation" method

Divide the group into small groups and give them a few problematic questions or situational questions.

Each small group writes their answer for 10 minutes and moves on to the next question. Until the end
a written response to questions. The written answers are discussed and the correct answer is selected.

Uses posters.

6. “Weak ring” method

The students in the group sit in a circle. Students will be asked questions in turn that require quick and short answers on the topic. The student who could not answer the question is out of the game
turns out. At the end, the remaining student will have answered many questions correctly.

7. Question-answer method "Boom" game

Topic hands of the trainer -o'qituvchining materials need to be ready for questions m .

The rules of the game are explained to the participants: they say the numbers 1, 2, 3 aloud, They should say the word “boom” instead of numbers ending in 3, divisible by
3.3. Attention
a pre-prepared question is asked to a student who has not sat down and is lost in the calculation;

Thus, the game between the group participants continues.

8. "Academic controversy" The group "Academic controversy" is divided into 2 teams. They are given a situational question. the student is assigned.

9 . "3-stage interview" "3-stage interview" is given to each group (3 people) of roles. "Doctor", "patient", "VOP expert". "Patients" are diagnosed anonymously. The group will discuss in 10-15 minutes. The "expert" will evaluate the doctor's actions in three parts. what was done right 2. what was done wrong 3. how it was to be done

10. "Group check" - the group is asked 2-3 questions on the topic. Time is given to collect information.

11. "On the gallery" - one task is given to small groups. Each group writes its opinion for 10 minutes and shares the answer sheet with 2 groups. The next group evaluates them and fills in the incomplete answer.

3 . Number of study hours m

Clock size	Distribution of the amount of workload by classroom hours (hours)				Independent work 65
1 55	Jami 90	Lecture 20	Practical training 2 4	Clinical training 4 6	

4 . Lectures

4 .1. Thematic plan of lectures

	Nomi	**Hour**	books
1	Digestive system. Methods of examination of the esophagus, stomach, intestines (interrogation, physical, laboratory-instrumental). Basic clinical syndromes.	2	1. GadaevA; Karimov M.Sh ..; "Propaedeutic internal diseases" T. 2012; 2. Muxin NA, Moiseev VI "Propede vnutrennix bolezney", M. 2000. 3. Vasilenko VX. Grebenev AL “Propede vnutrennix bolezney”, M.1989 4. Narziyev Sh.S. Abdullayeva MA "Propaede of internal diseases". Bukhara.2018;
2	Diseases of the digestive system: esophageal diseases, clinical diagnosis of gastritis .	2	1. GadaevA; Karimov M.Sh ..; "Propaedeutic internal diseases" T. 2012; 2. Muxin NA, Moiseev VI "Propede vnutrennix bolezney", M. 2000. 3. Vasilenko VX. Grebenev AL “Propede vnutrennix bolezney”, M.1989 4. Narziyev Sh.S. Abdullayeva MA "Propaede of internal diseases". Bukhara.2018;
3	Diseases of the digestive system . Y ara, mal`absorbtsiya syndrome, chronic colitis disease diagnostic clinic.	2	1. GadaevA; Karimov M.Sh ..; "Propaedeutic internal diseases" T. 2012; 2. Muxin NA, Moiseev VI "Propede vnutrennix bolezney", M. 2000. 3. Vasilenko VX. Grebenev AL “Propede vnutrennix bolezney”, M.1989 4. Narziyev Sh.S. Abdullayeva MA "Propaede of internal diseases". Bukhara.2018;
4	Diseases of the liver and biliary tract . Methods of examination: interrogation, methods of physical examination (examination, palpation, percussion, laboratory-instrumental methods). Basic clinical syndromes.	2	1. GadaevA; Karimov M.Sh ..; "Propaedeutic internal diseases" T. 2012; 2. Muxin NA, Moiseev VI "Propede vnutrennix bolezney", M. 2000. 3. Vasilenko VX. Grebenev AL “Propede vnutrennix bolezney”, M.1989 4. Narziyev Sh.S. Abdullayeva MA "Propaede of internal diseases". Bukhara.2018;
5	Chronic hepatitis . J Igarassu cirrhosis, chronic cholecystitis clinic diagnosis.	2	1. GadaevA; Karimov M.Sh ..; "Propaedeutic internal diseases" T. 2012; 2. Muxin NA, Moiseev VI "Propede vnutrennix bolezney", M. 2000. 3. Vasilenko VX. Grebenev AL “Propede vnutrennix bolezney”, M.1989 4. Narziyev Sh.S. Abdullayeva MA "Propaede of internal diseases". Bukhara.2018;
6	Renal and urinary system. Methods of examination: interrogation, methods of physical examination	2	1. GadaevA; Karimov M.Sh ..; "Propaedeutic internal diseases" T. 2012; 2. Muxin NA, Moiseev VI "Propede

	(examination, palpation, percussion, laboratory-instrumental methods). Basic clinical syndromes.		vnutrennix bolezney", M. 2000. 3. Vasilenko VX. Grebenev AL "Propede vnutrennix bolezney", M.1989 4. Narziyev Sh.S. Abdullayeva MA "Propaede of internal diseases". Bukhara.2018;
7	Diseases of the kidneys and urinary system. Symptomatology of acute and chronic glomerulonephritis and pyelonephritis. Clinical diagnosis of acute and chronic renal failure .	2	1. GadaevA; Karimov M.Sh ..; "Propaedeutic internal diseases" T. 2012; 2. Muxin NA, Moiseev VI "Propede vnutrennix bolezney", M. 2000. 3. Vasilenko VX. Grebenev AL "Propede vnutrennix bolezney", M.1989 4. Narziyev Sh.S. Abdullayeva MA "Propaede of internal diseases". Bukhara.2018;
8	Endocrine glands and metabolic system. Control methods. Inquiry, physical examination methods. Methods of laboratory and instrumental examination.	2	1. GadaevA; Karimov M.Sh ..; "Propaedeutic internal diseases" T. 2012; 2. Muxin NA, Moiseev VI "Propede vnutrennix bolezney", M. 2000. 3. Vasilenko VX. Grebenev AL "Propede vnutrennix bolezney", M.1989 4. Narziyev Sh.S. Abdullayeva MA "Propaede of internal diseases". Bukhara.2018;
9	Diseases of the endocrine glands and metabolic system , diabetes, neurotoxicosis, hypothyroidism .	2	1. GadaevA; Karimov M.Sh ..; "Propaedeutic internal diseases" T. 2012; 2. Muxin NA, Moiseev VI "Propede vnutrennix bolezney", M. 2000. 3. Vasilenko VX. Grebenev AL "Propede vnutrennix bolezney", M.1989 4. Narziyev Sh.S. Abdullayeva MA "Propaede of internal diseases". Bukhara.2018;
10	Blood system. Control methods. Methods of physical examination. Methods of laboratory and instrumental examination. Clinical diagnosis of diseases of the circulatory system anemia .	2	1. GadaevA; Karimov M.Sh ..; "Propaedeutic internal diseases" T. 2012; 2. Muxin NA, Moiseev VI "Propede vnutrennix bolezney", M. 2000. 3. Vasilenko VX. Grebenev AL "Propede vnutrennix bolezney", M.1989 4. Narziyev Sh.S. Abdullayeva MA "Propaede of internal diseases". Bukhara.2018;
	Jami	20	

4 .2 . Content of lecture materials

Topic 1 . Digestive system. Methods of examination of the esophagus, stomach, intestines (interrogation, physical, laboratory-instrumental). Basic clinical syndromes. Examination of patients with diseases of the gastrointestinal tract: interrogation, examination, palpation. The concept of laboratory-instrumental methods in the gastrointestinal tract. The main syndromes in gastrointestinal diseases. Symptomatology of acute and chronic gastritis. Examination of diseases of the gastrointestinal tract. Inquiry. Review. Abdominal palpation. Complaints: location, distribution of the mechanism of pain formation, time of occurrence during the day, duration, mechanism of vomiting. Whether or not the blood is mixed at lunch or after a meal. Nausea, the mechanism of formation, belching, wheezing, their frequent occurrence, the conditions of their appearance. Appetite, lack of appetite. Disgust with food. Dry mouth, feeling of bitterness, whether swallowing is free or

difficult, the presence of collaterals on the anterior and lateral walls of the abdomen, scarring. Intended surface palpation method. Deep methodical, slippery palpation method on VLObraztsov and NDStrajesko. Smooth palpation of the bowel. Symptomatology of enteritis and colitis The importance of stool examination and diagnosis.

References: 1. (basic literature) -4

2. (additional) - 4, 10, 11, 12, 16, 19, 21

Websites

Topic 2. Diseases of the digestive system: diseases of the esophagus, gastritis . Esophageal diseases, acute and chronic gastritis simptomatologiyasi.

References: 1. (basic literature) -4

2. (additional) - 4, 10, 11, 12, 16, 19, 21

Websites

Topic 3. Diseases of the digestive system: ulcers, malabsorption syndrome, chronic colitis. Etiopathogenesis , symptomatology of gastric and duodenal ulcers . Etiopathogenesis , symptomatology of enteritis and colitis . Coprologic examination. Diagnostic significance. Macroscopic examination: odor, color, shape, consistency. Chemical examination: reaction, detection of occult blood, detection of bile pigments, muscle fibers, fat, plant cells, starch, mucus, epithelium, leukocytes, erythrocytes, macrophages, gels.

References: 1. (basic literature) -4

2. (additional) - 4, 10, 11, 12, 16, 19, 21,

Websites

Topic 4 . Liver and bile ducts. Methods of examination: interrogation, methods of physical examination (examination, palpation, percussion, laboratory-instrumental methods). Basic clinical syndromes. Inquiry , examination, liver palpation, and percussion of patients with liver and biliary tract disease . According to Kurlov. Symptoms, mechanisms of occurrence, methods of diagnosis. Main complaints: mechanism of pain formation, localization, nature, duration, irradiation, causes, intensification, attenuation, cessation of pain, diagnostic significance. Dyspepsia: changes in appetite, nausea, belching, nausea, vomiting, abdominal distention, nausea. Jaundice: skin discoloration, urine, stool change. Diagnostic value. Itchy skin. Bleeding and hemorrhagic manifestations. Abdominal enlargement.

Understanding the main clinical syndromes: jaundice, portal hypertension, liver failure.

References: 1. (basic literature) -4

2. (additional) - 4, 10, 11, 12, 16, 19, 21,

Websites

Topic 5. Diseases of the liver and biliary tract . Chronic hepatitis. J liver cirrhosis, chronic cholecystitis. Symptomatology of chronic hepatitis. Symptomatology of chronic cholecystitis and liver cirrhosis . Mechanism and symptomatology of liver failure. Drug damage to the liver. Understanding the main clinical syndromes: jaundice, portal hypertension, liver failure. Complaints of chronic hepatitis, examination, palpation, tingling. An overview of the etiology and pathogenesis of diseases. Complaints of chronic cholecystitis , examination, palpation, tingling. An overview of the pathogenesis of disease etiology. Cirrhosis of the liver; complaints, examination, palpation, tingling. An overview of the pathogenesis of disease etiology. Mechanism and symptomatology of liver failure.

References: 1. (basic literature) -4

2. (additional) - 4, 10, 11, 12, 16, 19, 21,

Websites

Topic 6 . Renal and urinary system. Methods of examination: interrogation, methods of physical examination (examination, palpation, percussion, laboratory-instrumental methods). Basic clinical syndromes. Inquiry, examination, palpation, basic clinical syndromes of patients with kidney disease. Acute and chronic pyelonephritis,

symptomatology of glomerulonephritis. General understanding of complaints and their pathogenesis. Pain, their analysis. Location and distribution of tumors. Dysuric disorders. Methods of laboratory examination of urine. Functional kidney tests (Zimnisky, Nicheporenko, Reberga). Interpretation of the results.

References: 1. (basic literature) -4

2. (additional) - 4, 10, 11, 12, 16, 19, 21,

Websites

Topic 7. Diseases of the kidneys and urinary system. Symptomatology of acute and chronic glomerulonephritis and pyelonephritis. Acute and chronic renal failure. Understanding the etiopathogenesis of acute and chronic pyelonephritis and glomerulonephritis, acute and chronic renal failure . Inquiry.

References: 1. (basic literature) -4

2. (additional) - 4, 10, 11, 12, 16, 19, 21,

Websites

Topic 8. Endocrine glands and metabolic system. Control methods. Inquiry, physical examination methods. Methods of laboratory and instrumental examination. Inquiry into patients with diseases of the endocrine glands. complaints, anamnesis collection, hereditary factors. fatigue, weight loss, obesity, thirst, loss of appetite, fever, high irritability, itchy skin, palpitations, discoloration of skin coatings. General condition, consciousness, height and its General condition, consciousness, height and changes in its proportions. Gender and age signs. Presence of rashes, stretch marks on the skin. Development of subcutaneous fat and a uniform spread of tumors, weakness.

References: 1. (basic literature) -4

2. (additional) - 4, 10, 11, 12, 16, 19, 21,

Websites

Topic 9. Diseases of the endocrine glands and metabolic system. Diseases of the thyroid gland. Symptomatology of hypo and hyperthermia. Symptomatology of diabetes. The concept of diabetic and hypoglycemic comas. Basics of ambulance. Diseases of the thyroid gland.

References: 1. (basic literature) -4

2. (additional) - 4, 10, 11, 12, 16, 19, 21,

Websites

Topic 10. Blood system. Control methods. Methods of physical examination. Methods of laboratory and instrumental examination. Diseases of the circulatory system. Methods of interrogation and examination of patients with blood diseases. Classification of anemias. Symptomatology of iron deficiency, posthemorrhagic, hemolytic anemia. Symptomatology of leukemia. Blood test. Clinical significance of total blood test and interpretation of the results obtained. Anemia. General concepts of etiology and pathogenesis. General concepts of etiology and pathogenesis of leukemia. Classification of leukemia. Symptomatology of acute and chronic (myelogenous leukemia and lymphalic leukemia). Bleeding: from the nose, gums, gastrointestinal tract, uterus and other organs. Palpation of lymph nodes. Liver, spleen palpation, consistency, surface and edge, pain

References: 1. (basic literature) -4

2. (additional) - 4, 10, 11, 12, 16, 19, 21,

Websites

Thematic plans of practical lessons

№	Lesson topic	Class hour	books
1	Digestive system. Methods of	2	1. GadaevA; Karimov M.Sh

	examination of the esophagus, stomach, intestines (interrogation, physical, laboratory-instrumental).		..; "Propaedeutics of internal diseases" T. 2012; 2. Muxin NA, Moiseev VI "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko VX. Grebenev AL "Propedevtika vnutrennix bolezney", M.1989 4. Narziyev Sh.S. Abdullayeva MA "Propaedeutics of internal diseases". Bukhara.2018;
2	Main clinical syndromes: indigestion syndrome, intestinal absorption syndrome, acute abdomen, acute bleeding from the digestive system.	2	1. GadaevA; Karimov M.Sh ..; "Propaedeutics of internal diseases" T. 2012; 2. Muxin NA, Moiseev VI "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko VX. Grebenev AL "Propedevtika vnutrennix bolezney", M.1989 4. Narziyev Sh.S. Abdullayeva MA "Propaedeutics of internal diseases". Bukhara.2018;
3	Diseases of the digestive system: esophageal diseases - esophagitis , clinical diagnosis of g astritis .	2	1. GadaevA; Karimov M.Sh ..; "Propaedeutics of internal diseases" T. 2012; 2. Muxin NA, Moiseev VI "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko VX. Grebenev AL "Propedevtika vnutrennix bolezney", M.1989 4. Narziyev Sh.S. Abdullayeva MA "Propaedeutics of internal diseases". Bukhara.2018;
4	Y ara, mal`absorbtsiya syndrome, chronic colitis disease diagnostic clinic.	2	1. GadaevA; Karimov M.Sh ..; "Propaedeutics of internal diseases" T. 2012; 2. Muxin NA, Moiseev VI "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko VX. Grebenev AL "Propedevtika vnutrennix bolezney", M.1989 4. Narziyev Sh.S. Abdullayeva MA "Propaedeutics of internal diseases". Bukhara.2018;
5	Liver and bile ducts. Methods	2	1. GadaevA; Karimov M.Sh

	of examination: interrogation, physical examination (examination, palpation, percussion)		..; "Propaedeutics of internal diseases" T. 2012; 2. Muxin NA, Moiseev VI "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko VX. Grebenev AL “Propedevtika vnutrennix bolezney”, M.1989 4. Narziyev Sh.S. Abdullayeva MA "Propaedeutics of internal diseases". Bukhara.2018;
6	Liver and bile ducts. Test methods: laboratory-instrumental test methods). Basic clinical syndromes. Clinical diagnosis of jaundice	2	1. GadaevA; Karimov M.Sh ..; "Propaedeutics of internal diseases" T. 2012; 2. Muxin NA, Moiseev VI "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko VX. Grebenev AL “Propedevtika vnutrennix bolezney”, M.1989 4. Narziyev Sh.S. Abdullayeva MA "Propaedeutics of internal diseases". Bukhara.2018;
7	Liver and bile ducts. Clinical diagnosis of chronic hepatitis	1	1. GadaevA; Karimov M.Sh ..; "Propaedeutics of internal diseases" T. 2012; 2. Muxin NA, Moiseev VI "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko VX. Grebenev AL “Propedevtika vnutrennix bolezney”, M.1989 4. Narziyev Sh.S. Abdullayeva MA "Propaedeutics of internal diseases". Bukhara.2018;
8	Clinical diagnosis of liver cirrhosis, chronic cholecystitis . . Intermediate control № 3	1	1. GadaevA; Karimov M.Sh ..; "Propaedeutics of internal diseases" T. 2012; 2. Muxin NA, Moiseev VI "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko VX. Grebenev AL “Propedevtika vnutrennix bolezney”, M.1989 4. Narziyev Sh.S. Abdullayeva MA "Propaedeutics of internal diseases". Bukhara.2018;
9	Renal and urinary	1	1. GadaevA; Karimov M.Sh

	system. Methods of examination: interrogation, methods of physical examination (examination, palpation, percussion, laboratory-instrumental methods)		..; "Propaedeutics of internal diseases" T. 2012; 2. Muxin NA, Moiseev VI "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko VX. Grebenev AL "Propedevtika vnutrennix bolezney", M.1989 4. Narziyev Sh.S. Abdullayeva MA "Propaedeutics of internal diseases". Bukhara.2018;
10	Diseases of the kidneys and urinary system. Basic clinical syndromes	1	1. GadaevA; Karimov M.Sh ..; "Propaedeutics of internal diseases" T. 2012; 2. Muxin NA, Moiseev VI "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko VX. Grebenev AL "Propedevtika vnutrennix bolezney", M.1989 4. Narziyev Sh.S. Abdullayeva MA "Propaedeutics of internal diseases". Bukhara.2018;
11	Clinical diagnosis of acute and chronic glomerulonephritis . .	1	1. GadaevA; Karimov M.Sh ..; "Propaedeutics of internal diseases" T. 2012; 2. Muxin NA, Moiseev VI "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko VX. Grebenev AL "Propedevtika vnutrennix bolezney", M.1989 4. Narziyev Sh.S. Abdullayeva MA "Propaedeutics of internal diseases". Bukhara.2018;
12	O 'tkir and chronic pielonefritlarni symptom-matologiyasi. Clinical diagnosis of acute and chronic renal failure .	1	1. GadaevA; Karimov M.Sh ..; "Propaedeutics of internal diseases" T. 2012; 2. Muxin NA, Moiseev VI "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko VX. Grebenev AL "Propedevtika vnutrennix bolezney", M.1989 4. Narziyev Sh.S. Abdullayeva MA "Propaedeutics of internal diseases". Bukhara.2018;
13	Musculoskeletal and connective	1	1. GadaevA; Karimov M.Sh

	tissue system. Methods of examination: interrogation, methods of physical examination, methods of laboratory-instrumental examination. Basic clinical syndromes. SQB, Clinical Diagnosis of Systemic Scleroderma .		..; "Propaedeutics of internal diseases" T. 2012; 2. Muxin NA, Moiseev VI "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko VX. Grebenev AL "Propedevtika vnutrennix bolezney", M.1989 4. Narziyev Sh.S. Abdullayeva MA "Propaedeutics of internal diseases". Bukhara.2018;
14	Endocrine glands and metabolic system. Control methods. Inquiry, physical examination methods. Methods of laboratory and instrumental examination.	1	1. GadaevA; Karimov M.Sh ..; "Propaedeutics of internal diseases" T. 2012; 2. Muxin NA, Moiseev VI "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko VX. Grebenev AL "Propedevtika vnutrennix bolezney", M.1989 4. Narziyev Sh.S. Abdullayeva MA "Propaedeutics of internal diseases". Bukhara.2018;
15	Diseases of the endocrine glands and metabolic system. Clinical diagnosis of diabetes, thyrotoxicosis, hypothyroidism .	1	1. GadaevA; Karimov M.Sh ..; "Propaedeutics of internal diseases" T. 2012; 2. Muxin NA, Moiseev VI "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko VX. Grebenev AL "Propedevtika vnutrennix bolezney", M.1989 4. Narziyev Sh.S. Abdullayeva MA "Propaedeutics of internal diseases". Bukhara.2018;
16	Blood system. Control methods. Methods of physical examination. Methods of laboratory and instrumental examination.	1	1. GadaevA; Karimov M.Sh ..; "Propaedeutics of internal diseases" T. 2012; 2. Muxin NA, Moiseev VI "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko VX. Grebenev AL "Propedevtika vnutrennix bolezney", M.1989 4. Narziyev Sh.S. Abdullayeva MA "Propaedeutics of internal diseases". Bukhara.2018;

17	Diseases of the circulatory system. Clinical diagnosis of anemia .	1	1. GadaevA; Karimov M.Sh ..; "Propaedeutics of internal diseases" T. 2012; 2. Muxin NA, Moiseev VI "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko VX. Grebenev AL “Propedevtika vnutrennix bolezney”, M.1989 4. Narziyev Sh.S. Abdullayeva MA "Propaedeutics of internal diseases". Bukhara.2018;
1 8	Medical history. Practical skills.	1	1. GadaevA; Karimov M.Sh ..; "Propaedeutics of internal diseases" T. 2012; 2. Muxin NA, Moiseev VI "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko VX. Grebenev AL “Propedevtika vnutrennix bolezney”, M.1989 4. Narziyev Sh.S. Abdullayeva MA " Propaedeutics of internal diseases". Bukhara.2018;
	Jami	24	

5 .2. The content of the topics of practical and clinical training .

Topic 1. Digestive system. Methods of examination of the esophagus, stomach, intestines (interrogation, physical, laboratory-instrumental). Examination of the abdomen. Palpation of abdominal organs. Palpation of the stomach and intestines. Sequence. Laboratory work: examination of gastric juice and grass. Examination of gastric juice: determination of total, free, bound hydrochloric acid by titration. Duodenal probing, macro, microscopic examination of 3 servings of grass, diagnostic value. Examine the abdomen in a vertical, horizontal position. Dividing the abdomen into a topographic area. Movement of the abdominal wall in the act of breathing. Development of venous collaterals on the anterior surface of the abdomen, on the lateral wall. Medusa head, pigmentation, hernia gastric peristalsis, antiperistalsis. Palpation. Superficial palpation method, condition of the skin and subcutaneous fat layer. Debit-hours, calculate it. Determination of pepsin, method of laboratory examination of gastric juice without a probe (acidotest, determination of uropepsin). Microscopic examination of gastric juice. Basal juice. Stimulating gastric secretion (histamine, cabbage soup).

Interactive method: Academic pol e mica, snow pile, ration method

References: 1. (basic literature) -1,2,3

(additional) - 1,2,3,4,5,6

Websites

Topic 2. Main clinical syndromes: indigestion syndrome, intestinal absorption syndrome, acute abdomen, acute bleeding from the digestive system. Methods of instrumental examination of digestive organs (radiology, esophagogastroduodenofibroscopy, rectoromanoscopy, colonoscopy). Symptomatology of gastritis (acute and chronic). X-ray examination of the gastrointestinal tract. Diagnostic

significance. Endoscopic examination of the gastrointestinal
tract. Gastroscopy. Colonoscopy. Rectoromanoscopy. Advantages of endoscopy over radiological examination. Etiopathogenesis, classification of gastritis. Symptomatology of acute and chronic gastritis.

Interactive method: Beehive, snowdrift, 3-step interview

References: 1. (basic literature) -4

2. (additional) - 4, 10, 11, 12, 16, 19, 21,

Websites

Topic 3. Diseases of the digestive system: diseases of the esophagus, gastritis. Gastritis symptomatology (acute and chronic). Etiopathogenesis, classification of gastritis.

Interactive method: summary method **„** snow pile, 3-step interview

References: 1. (basic literature) -4

2. (additional) - 4, 10, 11, 12, 16, 19, 21,

Websites

Topic 4 . Y ara disease, mal`absorbtsiya syndrome, chronic colitis. Symptomatology of gastric and duodenal ulcers. Symptomatology of enteritis and colitis. Lab: Laboratory examination. Etiopathogenesis , symptomatology of gastric and duodenal
ulcers . Etiopathogenesis , symptomatology of enteritis and colitis . Coprologic examination. Diagnostic significance. Macroscopic examination: odor, color, shape, consistency. Chemical examination: reaction, detection of occult blood, detection of bile pigments, muscle fibers, fat, plant cells, starch, mucus, epithelium, leukocytes, erythrocytes, macrophages, gels.

Interactive method: cluster, snow pile, ruch.stol.ort.

References: 1. (basic literature) -4

2. (additional) - 4, 10, 11, 12, 16, 19, 21,

Websites

Topic 5 . Liver and bile ducts. Methods of examination: interrogation, methods of physical examination (examination, palpation, percussion) . Interrogation of patients with diseases of the liver and biliary tract complainant, anamnesis, examination, palpation and percussion. Understanding the main clinical syndromes: jaundice, portal hypertension, liver failure. Inquiry into patients with liver and biliary tract disease. The main complaints. Localization, nature, duration, irradiation, causes, intensification, attenuation, cessation of pain. Dyspepsia: changes in appetite, nausea in the mouth, nausea, nausea, abdominal cramps. Jaundice: skin discoloration, urine, stool change. Diagnostic significance. Itchy skin. Abdominal enlargement. Review. Abdominal percussion, detection of ascites. Determination of liver size according to Kurlov.

Interactive method: Incident method, ratification method, academic pol e mica

References: 1. (basic literature) -4

2. (additional) - 4, 10, 11, 12, 16, 19, 21,

Websites

Topic 6 . Liver and bile ducts. Test methods: laboratory-instrumental test methods). Basic clinical syndromes. Methods of laboratory and instrumental examination of patients with diseases of the liver and biliary tract (cholecystography, liver scan, examination of the liver and gallbladder with ultrasound). Computed tomography. Examination of liver pigment, carbohydrate, fat metabolism (biochemical examination of blood), examination of trace elements (iron, copper). Examination of enzymes, examination of the detoxifying, excretory activity of the liver. Radioisotope examination of liver structure and function, radiometric examination, liver scan. Scanning the gallbladder. X-ray examination: cholecystography, cholegraphy, computed tomography. The concept of basic clinical syndromes. Mechanism and symptomatology of jaundice, portal hypertension, liver failure. Types, mechanism and pathogenesis of jaundice. Bilirubin metabolism scheme. Symptomatology of portal hypertension.

Interactive method: svod method, weak loop, on gallery

References: 1. (basic literature) -4

2. (additional) - 4, 10, 11, 12, 16, 19, 21,

Websites

Topic 7 . Liver and bile ducts. Chronic hepatitis. Chronic GEP a titlarning simptomatologiyasi. Drug damage to the liver. Complaints, review, palpation, knocking. Etiopathogenesis of the disease.
Interactive method: arrow method, weak loop, on gallery
References: 1. (basic literature) -4
2. (additional) - 4, 10, 11, 12, 16, 19, 21,
Websites

Topic 8 . Cirrhosis of the liver, chronic cholecystitis. Intermediate control № 3. Symptomatology of chronic cholecystitis. Symptomatology of liver cirrhosis. Complaints, review, palpation, knocking. Etiopathogenesis of the disease.
Interactive method: arrow method, weak loop, on gallery
References: 1. (basic literature) -4
2. (additional) - 4, 10, 11, 12, 16, 19, 21,
Websites

Topic 9 . Renal and urinary system. Methods of examination: interrogation, methods of physical examination (examination, palpation, percussion, laboratory-instrumental methods). Inquiry. The main complaints and their pathogenesis. Pains. Their analysis. Location, distribution of tumors. Laboratory tests. General analysis of urine. Examination of urine by the method of Nechiporenko. Leukocytes, erythrocytes, cylinder ratio and diagnostic value. Zimnitsky test. The importance of the relative weight of urine in assessing the functional status of the kidneys. Hyposteniuria. Izosteniruya. Nikturia. Reberg test, determination of serum creatinine, urea
Interactive method: snowdrift, beehive, weak ring
References: 1. (basic literature) -4
2. (additional) - 4, 10, 11, 12, 16, 19, 21,
Websites

Topic 10 . Diseases of the kidneys and urinary system. Basic clinical syndromes. Methods for the detection and diagnostic value of proteinuria, glucosuria, bilirubinuria, urobilinuria, acetonuria. Dysuric disorders.
Interactive method: snowdrift, beehive, weak ring
References: 1. (basic literature) -4
2. (additional) - 4, 10, 11, 12, 16, 19, 21,
Websites

Topic 11. Acute and chronic glomerulonephritis . Understanding the etiopathogenesis of acute and chronic pyelonephritis and glomerulonephritis. Inquiry.
Interactive method: snowdrift, beehive, weak ring
References: 1. (basic literature) -4
2. (additional) - 4, 10, 11, 12, 16, 19, 21,
Websites

Topic 12. Symptomatology of acute and chronic pyelonephritis. Acute and chronic renal failure. Acute and chronic pyelonephritis, acute and chronic renal failure. concept of etiopathogenesis. Inquiry.
Interactive method: snowdrift, beehive, weak ring
References: 1. (basic literature) -4
2. (additional) - 4, 10, 11, 12, 16, 19, 21,
Websites

Topic 13. Musculoskeletal and connective tissue system. Methods of examination: interrogation, methods of physical examination, methods of laboratory-instrumental examination. Basic clinical syndromes. Arthritis. Inquiry. The main complaints and their pathogenesis: pain in the joints and muscles. Fever. Weakness. Joint configuration, swelling, redness, amount of active and passive movements. Palpation, determination of joint temperature, the appearance of nodules, muscle aches. Systematic enlargement of lymph nodes. The concept of rheumatic diseases.

Connective tissue disease. Inquiry. The main complaints and their pathogenesis: pain in the bones, muscles, joints, their dependence on movement. Tumors and their classification.
Interactive method: Round table method, academic pol e mica, weak ring
References: 1. (basic literature) -4
2. (additional) - 4, 10, 11, 12, 16, 19, 21,
Websites

Topic 14. Endocrine glands and metabolic system. Control methods. Inquiry, physical examination methods. Methods of laboratory and instrumental examination. Diseases of the endocrine glands. Complaints. Weakness, weight loss, obesity, thirst, loss of appetite, fever, high irritability, itchy skin, palpitations, discoloration of the skin lining, dryness. General condition. Es-hushi. Change in height and proportions. Gender and age signs. Changes in skin coverings. Presence of rashes, stretch marks on the skin. Development and even distribution of subcutaneous adipose tissue. Tumors. Identify eye symptoms. Changes in the face and neck area.
Interactive method: Cluster, weak ring, snow pile
References: 1. (basic literature) -4
2. (additional) - 4, 10, 11, 12, 16, 19, 21,
Websites

Topic 15. Diseases of the endocrine glands and metabolic system. Diseases of the thyroid gland. Symptomatology of hypo and hyperthermia. Etiopathogenesis, symptomatology of diabetes. The concept of diabetic and hypoglycemic comas. Basics of ambulance.
Interactive method: Ratastic method, group unit, synetics
References: 1. (basic literature) -4
2. (additional) - 4, 10, 11, 12, 16, 19, 21,
Websites

Topic 16. Blood system. Control methods. Methods of physical examination. Methods of laboratory and instrumental examination. Bleeding: from the nose, gums, gastrointestinal tract, uterus and other organs. Itchy skin, malaria, itching. Changes in the skin and mucous membranes, enlargement of regional lymph nodes. Bleeding. Petechiae, palpation of lymph nodes, liver and spleen. Consistency, surface and edge, pain. General clinical analysis of blood. Determination of leukocytes, platelets, erythrocytes (reticulocytes), lymphocytes formula, E.CH.T. Get acquainted with the general methods of determining the state of the rate of blood flow.
Interactive method: Bee hive, weak ring, pen in the middle of the table
References: 1. (basic literature) -4
2. (additional) - 4, 10, 11, 12, 16, 19, 21
Websites

Topic 17. Diseases of the circulatory system. A general understanding of the etiopathogenesis of anemia. Classification. Iron deficiency, posthemorrhagic, hemolytic. A general understanding of the etiopathogenesis of leukemia . Classification.
Interactive method: Cluster, weak ring, snow pile
References: 1. (basic literature) -4
2. (additional) - 4, 10, 11, 12, 16, 19, 21

Topic 18. Medical history. Practical skills. Independent work of students under the supervision of a teacher. Curation of patients. Writing practical skills and medical history.
Interactive method: academic pol e mica, weak ring
References: 1. (basic literature) -4
2. (additional) - 4, 10, 11, 12, 16, 19, 21

K line mashg`ulotlarning thematic plans

№	Lesson topic	Class hour	Books
1	Digestive system. Methods of	2	1. GadaevA; Karimov M.Sh

	examination of the esophagus, stomach, intestines (interrogation, physical, laboratory-instrumental). The concept of coronavirus, its epidemiology and its clinic		..; "Propaedeutics of internal diseases" T. 2012; 2. Muxin NA, Moiseev VI "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko VX. Grebenev AL “Propedevtika vnutrennix bolezney”, M.1989 4. Narziyev Sh.S. Abdullayeva MA "Propaedeutics of internal diseases". Bukhara.2018;
2	Main clinical syndromes: indigestion syndrome, intestinal absorption syndrome, acute abdomen, acute bleeding from the digestive system. Coronavirus diagnostics, differential diagnosis, course	2	1. GadaevA; Karimov M.Sh ..; "Propaedeutics of internal diseases" T. 2012; 2. Muxin NA, Moiseev VI "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko VX. Grebenev AL “Propedevtika vnutrennix bolezney”, M.1989 4. Narziyev Sh.S. Abdullayeva MA "Propaedeutics of internal diseases". Bukhara.2018;
3	Digestive system diseases: diseases of the esophagus -ezofagit g astritlar clinic diagnosis. Treatment regimen for coronavirus	2	1. GadaevA; Karimov M.Sh ..; "Propaedeutics of internal diseases" T. 2012; 2. Muxin NA, Moiseev VI "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko VX. Grebenev AL “Propedevtika vnutrennix bolezney”, M.1989 4. Narziyev Sh.S. Abdullayeva MA "Propaedeutics of internal diseases". Bukhara.2018;
4	Y ara, mal`absorbtsiya syndrome, chronic colitis disease diagnostic clinic.	2	1. GadaevA; Karimov M.Sh ..; "Propaedeutics of internal diseases" T. 2012; 2. Muxin NA, Moiseev VI "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko VX. Grebenev AL “Propedevtika vnutrennix bolezney”, M.1989 4. Narziyev Sh.S. Abdullayeva MA "Propaedeutics of internal diseases". Bukhara.2018;
5	Liver and bile ducts. Methods of examination: interrogation, physical examination (examination, palpation, percussion)	2	1. GadaevA; Karimov M.Sh ..; "Propaedeutics of internal diseases" T. 2012; 2. Muxin NA, Moiseev VI "Propedevtika vnutrennix bolezney", M.

			2000. 3. Vasilenko VX. Grebenev AL “Propedevtika vnutrennix bolezney”, M.1989 4. Narziyev Sh.S. Abdullayeva MA "Propaedeutics of internal diseases". Bukhara.2018;
6	Liver and bile ducts. Test methods: laboratory-instrumental test methods). Basic clinical syndromes. Clinical diagnosis of jaundice	2	1. GadaevA; Karimov M.Sh ..; "Propaedeutics of internal diseases" T. 2012; 2. Muxin NA, Moiseev VI "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko VX. Grebenev AL “Propedevtika vnutrennix bolezney”, M.1989 4. Narziyev Sh.S. Abdullayeva MA "Propaedeutics of internal diseases". Bukhara.2018;
7	Liver and bile ducts. Clinical diagnosis of chronic hepatitis	3	1. GadaevA; Karimov M.Sh ..; "Propaedeutics of internal diseases" T. 2012; 2. Muxin NA, Moiseev VI "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko VX. Grebenev AL “Propedevtika vnutrennix bolezney”, M.1989 4. Narziyev Sh.S. Abdullayeva MA "Propaedeutics of internal diseases". Bukhara.2018;
8	Clinical diagnosis of liver cirrhosis, chronic cholecystitis . . Intermediate control № 3	3	1. GadaevA; Karimov M.Sh ..; "Propaedeutics of internal diseases" T. 2012; 2. Muxin NA, Moiseev VI "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko VX. Grebenev AL “Propedevtika vnutrennix bolezney”, M.1989 4. Narziyev Sh.S. Abdullayeva MA "Propaedeutics of internal diseases". Bukhara.2018;
9	Renal and urinary system. Methods of examination: interrogation, methods of physical examination (examination, palpation, percussion, laboratory-instrumental methods)	3	1. GadaevA; Karimov M.Sh ..; "Propaedeutics of internal diseases" T. 2012; 2. Muxin NA, Moiseev VI "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko VX. Grebenev AL “Propedevtika vnutrennix bolezney”, M.1989

			4. Narziyev Sh.S. Abdullayeva MA "Propaedeutics of internal diseases". Bukhara.2018;
10	Diseases of the kidneys and urinary system. Basic clinical syndromes	3	1. GadaevA; Karimov M.Sh ..; "Propaedeutics of internal diseases" T. 2012; 2. Muxin NA, Moiseev VI "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko VX. Grebenev AL "Propedevtika vnutrennix bolezney", M.1989 4. Narziyev Sh.S. Abdullayeva MA "Propaedeutics of internal diseases". Bukhara.2018;
11	Clinical diagnosis of acute and chronic glomerulonephritis	3	1. GadaevA; Karimov M.Sh ..; "Propaedeutics of internal diseases" T. 2012; 2. Muxin NA, Moiseev VI "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko VX. Grebenev AL "Propedevtika vnutrennix bolezney", M.1989 4. Narziyev Sh.S. Abdullayeva MA "Propaedeutics of internal diseases". Bukhara.2018;
12	O 'tkir and chronic pielonefritlarni symptom-matologiyasi. Clinical diagnosis of acute and chronic renal failure	3	1. GadaevA; Karimov M.Sh ..; "Propaedeutics of internal diseases" T. 2012; 2. Muxin NA, Moiseev VI "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko VX. Grebenev AL "Propedevtika vnutrennix bolezney", M.1989 4. Narziyev Sh.S. Abdullayeva MA "Propaedeutics of internal diseases". Bukhara.2018;
13	Musculoskeletal and connective tissue system. Methods of examination: interrogation, methods of physical examination, methods of laboratory- instrumental examination. Basic clinical syndromes. SQB, Clinical Diagnosis of Systemic Scleroderma	3	1. GadaevA; Karimov M.Sh ..; "Propaedeutics of internal diseases" T. 2012; 2. Muxin NA, Moiseev VI "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko VX. Grebenev AL "Propedevtika vnutrennix bolezney", M.1989 4. Narziyev Sh.S. Abdullayeva MA "Propaedeutics of internal diseases". Bukhara.2018;
14	Endocrine glands and metabolic	3	1. GadaevA; Karimov M.Sh

	system. Control methods. Inquiry, physical examination methods. Methods of laboratory and instrumental examination		..; "Propaedeutics of internal diseases" T. 2012; 2. Muxin NA, Moiseev VI "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko VX. Grebenev AL “Propedevtika vnutrennix bolezney”, M.1989 4. Narziyev Sh.S. Abdullayeva MA "Propaedeutics of internal diseases". Bukhara.2018;
15	Diseases of the endocrine glands and metabolic system. Clinical diagnosis of diabetes, thyrotoxicosis, hypothyroidism	3	1. GadaevA; Karimov M.Sh ..; "Propaedeutics of internal diseases" T. 2012; 2. Muxin NA, Moiseev VI "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko VX. Grebenev AL “Propedevtika vnutrennix bolezney”, M.1989 4. Narziyev Sh.S. Abdullayeva MA "Propaedeutics of internal diseases". Bukhara.2018;
16	Blood system. Control methods. Methods of physical examination. Methods of laboratory and instrumental examination	3	1. GadaevA; Karimov M.Sh ..; "Propaedeutics of internal diseases" T. 2012; 2. Muxin NA, Moiseev VI "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko VX. Grebenev AL “Propedevtika vnutrennix bolezney”, M.1989 4. Narziyev Sh.S. Abdullayeva MA "Propaedeutics of internal diseases". Bukhara.2018;
17	Diseases of the circulatory system. Clinical diagnosis of anemia	2	1. GadaevA; Karimov M.Sh ..; "Propaedeutics of internal diseases" T. 2012; 2. Muxin NA, Moiseev VI "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko VX. Grebenev AL “Propedevtika vnutrennix bolezney”, M.1989 4. Narziyev Sh.S. Abdullayeva MA "Propaedeutics of internal diseases". Bukhara.2018;
18	Medical history. Practical skills	2	1. GadaevA; Karimov M.Sh ..; "Propaedeutics of internal diseases" T. 2012;
	Jami	46	

5 .2. Content of clinical training topics.

Topic 1. Digestive system. Methods of examination of the esophagus, stomach, intestines (interrogation, physical, laboratory-instrumental). Examination of the abdomen. Palpation of abdominal organs. Palpation of the stomach and intestines. Sequence. Laboratory work: examination of gastric juice and grass. Examination of gastric juice: determination of total, free, bound hydrochloric acid by titration. Duodenal probing, macro, microscopic examination of 3 servings of grass, diagnostic value. Examine the abdomen in a vertical, horizontal position. Dividing the abdomen into a topographic area. Movement of the abdominal wall in the act of breathing. Development of venous collaterals on the anterior surface of the abdomen, on the lateral wall. Medusa head, pigmentation, hernia gastric peristalsis, antiperistalsis. Palpation. Superficial palpation method, condition of the skin and subcutaneous fat layer. Debit-hours, calculate it. Determination of pepsin, method of laboratory examination of gastric juice without a probe (acidotest, determination of uropepsin). Microscopic examination of gastric juice. Basal juice. Stimulating gastric secretion (histamine, cabbage soup).

Interactive method: Academic pol e mica, snow pile, ration method

References: 1. (basic literature) -1,2,3

(additional) - 1,2,3,4,5,6

Websites

Topic 2. Main clinical syndromes: indigestion syndrome, intestinal absorption syndrome, acute abdomen, acute bleeding from the digestive system. Methods of instrumental examination of digestive organs (radiology, esophagogastroduodenofibroscopy, rectoromanoscopy, colonoscopy). Symptomatology of gastritis (acute and chronic). X-ray examination of the gastrointestinal tract. Diagnostic significance. Endoscopic examination of the gastrointestinal tract. Gastroscopy. Colonoscopy. Rectoromanoscopy. Advantages of endoscopy over radiological examination. Etiopathogenesis, classification of gastritis. Symptomatology of acute and chronic gastritis.

Interactive method: Beehive, snowdrift, 3-step interview

References: 1. (basic literature) -4

2. (additional) - 4, 10, 11, 12, 16, 19, 21,

Websites

Topic 3. Diseases of the digestive system: diseases of the esophagus, gastritis. Gastritis symptomatology (acute and chronic). Etiopathogenesis, classification of gastritis.

Interactive method: summary method **„** snow pile, 3-step interview

References: 1. (basic literature) -4

2. (additional) - 4, 10, 11, 12, 16, 19, 21,

Websites

Topic 4 . Y ara disease, mal`absorbtsiya syndrome, chronic colitis. Symptomatology of gastric and duodenal ulcers. Symptomatology of enteritis and colitis. Lab: Laboratory examination. Etiopathogenesis , symptomatology of gastric and duodenal ulcers . Etiopathogenesis , symptomatology of enteritis and colitis . Coprologic examination. Diagnostic significance. Macroscopic examination: odor, color, shape, consistency. Chemical examination: reaction, detection of occult blood, detection of bile pigments, muscle fibers, fat, plant cells, starch, mucus, epithelium, leukocytes, erythrocytes, macrophages, gels.

Interactive method: cluster, snow pile, ruch.stol.ort.

References: 1. (basic literature) -4

2. (additional) - 4, 10, 11, 12, 16, 19, 21,

Websites

Topic 5 . Liver and bile ducts. Methods of examination: interrogation, methods of physical examination (examination, palpation, percussion) . Interrogation of patients with

diseases of the liver and biliary tract complainant, anamnesis, examination, palpation and percussion. Understanding the main clinical syndromes: jaundice, portal hypertension, liver failure. Inquiry into patients with liver and biliary tract disease. The main complaints. Localization, nature, duration, irradiation, causes, intensification, attenuation, cessation of pain. Dyspepsia: changes in appetite, nausea in the mouth, nausea, nausea, abdominal cramps. Jaundice: skin discoloration, urine, stool change. Diagnostic significance. Itchy skin. Abdominal enlargement. Review. Abdominal percussion, detection of ascites. Determination of liver size according to Kurlov.

Interactive method: Incident method, ratification method, academic pol e mica

References: 1. (basic literature) -4

2. (additional) - 4, 10, 11, 12, 16, 19, 21,

Websites

Topic 6 . Liver and bile ducts. Test methods: laboratory-instrumental test methods). Basic clinical syndromes. Methods of laboratory and instrumental examination of patients with diseases of the liver and biliary tract (cholecystography, liver scan, examination of the liver and gallbladder with ultrasound). Computed tomography. Examination of liver pigment, carbohydrate, fat metabolism (biochemical examination of blood), examination of trace elements (iron, copper). Examination of enzymes, examination of the detoxifying, excretory activity of the liver. Radioisotope examination of liver structure and function, radiometric examination, liver scan. Scanning the gallbladder. X-ray examination: cholecystography, cholegraphy, computed tomography. The concept of basic clinical syndromes. Mechanism and symptomatology of jaundice, portal hypertension, liver failure. Types, mechanism and pathogenesis of jaundice. Bilirubin metabolism scheme. Symptomatology of portal hypertension.

Interactive method: svod method, weak loop, on gallery

References: 1. (basic literature) -4

2. (additional) - 4, 10, 11, 12, 16, 19, 21,

Websites

Topic 7 . Liver and bile ducts. Chronic hepatitis. Chronic GEP a titlarning simptomatologiyasi. Drug damage to the liver. Complaints, review, palpation, knocking. Etiopathogenesis of the disease.

Interactive method: arrow method, weak loop, on gallery

References: 1. (basic literature) -4

2. (additional) - 4, 10, 11, 12, 16, 19, 21,

Websites

Topic 8 . Cirrhosis of the liver, chronic cholecystitis. Intermediate control № 3. Symptomatology of chronic cholecystitis. Symptomatology of liver cirrhosis. Complaints, review, palpation, knocking. Etiopathogenesis of the disease.

Interactive method: arrow method, weak loop, on gallery

References: 1. (basic literature) -4

2. (additional) - 4, 10, 11, 12, 16, 19, 21,

Websites

Topic 9 . Renal and urinary system. Methods of examination: interrogation, methods of physical examination (examination, palpation, percussion, laboratory-instrumental methods). Inquiry. The main complaints and their pathogenesis. Pains. Their analysis. Location, distribution of tumors. Laboratory tests. General analysis of urine. Examination of urine by the method of Nechiporenko. Leukocytes, erythrocytes, cylinder ratio and diagnostic value. Zimnitsky test. The importance of the relative weight of urine in assessing the functional status of the kidneys. Hyposteniuria. Izosteniruya. Nikturia. Reberg test, determination of serum creatinine, urea

Interactive method: snowdrift, beehive, weak ring

References: 1. (basic literature) -4

2. (additional) - 4, 10, 11, 12, 16, 19, 21,

Websites

Topic 10 . Diseases of the kidneys and urinary system. Basic clinical syndromes. Methods for the detection and diagnostic value of proteinuria, glucosuria, bilirubinuria, urobilinuria, acetonuria. Dysuric disorders.
Interactive method: snowdrift, beehive, weak ring
References: 1. (basic literature) -4
2. (additional) - 4, 10, 11, 12, 16, 19, 21,
Websites

Topic 11. Acute and chronic glomerulonephritis . Understanding the etiopathogenesis of acute and chronic pyelonephritis and glomerulonephritis. Inquiry.
Interactive method: snowdrift, beehive, weak ring
References: 1. (basic literature) -4
2. (additional) - 4, 10, 11, 12, 16, 19, 21,
Websites

Topic 12. Symptomatology of acute and chronic pyelonephritis. Acute and chronic renal failure. Acute and chronic pyelonephritis, acute and chronic renal failure. concept of etiopathogenesis. Inquiry.
Interactive method: snowdrift, beehive, weak ring
References: 1. (basic literature) -4
2. (additional) - 4, 10, 11, 12, 16, 19, 21,
Websites

Topic 13. Musculoskeletal and connective tissue system. Methods of examination: interrogation, methods of physical examination, methods of laboratory-instrumental examination. Basic clinical syndromes. Arthritis. Inquiry. The main complaints and their pathogenesis: pain in the joints and muscles. Fever. Weakness. Joint configuration, swelling, redness, amount of active and passive movements. Palpation, determination of joint temperature, the appearance of nodules, muscle aches. Systematic enlargement of lymph nodes. The concept of rheumatic diseases.
Connective tissue disease. Inquiry. The main complaints and their pathogenesis: pain in the bones, muscles, joints, their dependence on movement. Tumors and their classification.
Interactive method: Round table method, academic pol e mica, weak ring
References: 1. (basic literature) -4
2. (additional) - 4, 10, 11, 12, 16, 19, 21,
Websites

Topic 14. Endocrine glands and metabolic system. Control methods. Inquiry, physical examination methods. Methods of laboratory and instrumental examination. Diseases of the endocrine glands. Complaints. Weakness, weight loss, obesity, thirst, loss of appetite, fever, high irritability, itchy skin, palpitations, discoloration of the skin lining, dryness. General condition. Es-hushi. Change in height and proportions. Gender and age signs. Changes in skin coverings. Presence of rashes, stretch marks on the skin. Development and even distribution of subcutaneous adipose tissue. Tumors. Identify eye symptoms. Changes in the face and neck area.
Interactive method: Cluster, weak ring, snow pile
References: 1. (basic literature) -4
2. (additional) - 4, 10, 11, 12, 16, 19, 21,
Websites

Topic 15. Diseases of the endocrine glands and metabolic system. Diseases of the thyroid gland. Symptomatology of hypo and hyperthermia. Etiopathogenesis, symptomatology of diabetes. The concept of diabetic and hypoglycemic comas. Basics of ambulance.
Interactive method: Ratastic method, group unit, synetics
References: 1. (basic literature) -4
2. (additional) - 4, 10, 11, 12, 16, 19, 21,
Websites

Topic 16. Blood system. Control methods. Methods of physical examination. Methods of laboratory and instrumental examination. Bleeding: from the nose, gums, gastrointestinal

tract, uterus and other organs. Itchy skin, malaria, itching. Changes in the skin and mucous membranes, enlargement of regional lymph nodes. Bleeding. Petechiae, palpation of lymph nodes, liver and spleen. Consistency, surface and edge, pain. General clinical analysis of blood. Determination of leukocytes, platelets, erythrocytes (reticulocytes), lymphocytes formula, E.CH.T. Get acquainted with the general methods of determining the state of the rate of blood flow.
Interactive method: Bee hive, weak ring, pen in the middle of the table
References: 1. (basic literature) -4
2. (additional) - 4, 10, 11, 12, 16, 19, 21
Websites

Topic 17. Diseases of the circulatory system. A general understanding of the etiopathogenesis of anemia. Classification. Iron deficiency, posthemorrhagic, hemolytic. A general understanding of the etiopathogenesis of leukemia . Classification.
Interactive method: Cluster, weak ring, snow pile
References: 1. (basic literature) -4
2. (additional) - 4, 10, 11, 12, 16, 19, 21

Topic 18. Medical history. Practical skills. Independent work of students under the supervision of a teacher. Curation of patients. Writing practical skills and medical history.
Interactive method: academic pol e mica, weak ring
References: 1. (basic literature) -4
2. (additional) - 4, 10, 11, 12, 16, 19, 21

Naimenovaniya lektsionnyx zanyatiy

№	Naimenovaniya lektsionnyx zanyatiy	Chasy	Literature
1	Pishchevaritelnyy tract. Obsledovanie pishchevoda, jeludka, kishechnikov (rasspros, fizikalnaya, labaratorno-instrumalnaya). Basic clinical syndromes. The concept of coronavirus, epidemiology and ego clinic	**2**	1. GadaevA; Karimov M.Sshch ..; "Propaedeutics of internal diseases" T. 2012; 2. Muxin N.A., Moiseev V.I. "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko V.X .. Grebenev A.L "Propedevtika vnutrennix bolezney", M.1989 4. Narziev Sshch.S. Abdullaeva M.A. "Propaedeutics of internal diseases." Buxoro. 2018;
2	Zabolevanie pishchevaritelnogo tract: zabolevanie pishchevoda, gastrity clinic, diagnostics Diagnosis and differential diagnosis and treatment of coronavirus	**2**	1. GadaevA; Karimov M.Sshch ..; "Propaedeutics of internal diseases" T. 2012; 2. Muxin N.A., Moiseev V.I. "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko V.X .. Grebenev A.L "Propedevtika vnutrennix bolezney", M.1989 4. Narziev Sshch.S. Abdullaeva M.A. "Propaedeutics of internal diseases." Buxoro. 2018;
3	Zabolevanii ishchevaritelnogo tract. Yazvennaya bolezn, malabsorbtsii syndrome, chronic colitis clinic, diagnosis. Treatment of coronavirus	**2**	1. GadaevA; Karimov M.Sshch ..; "Propaedeutics of internal diseases" T. 2012; 2. Muxin N.A., Moiseev V.I. "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko V.X .. Grebenev A.L "Propedevtika vnutrennix bolezney", M.1989 4. Narziev Sshch.S. Abdullaeva M.A. "Propaedeutics of internal diseases." Buxoro. 2018;
4	Pechen and jelchnye	**2**	1. GadaevA; Karimov M.Sshch ..; "Propaedeutics of

	puti. Methods of observation: raspross, physical (osmotr, palpation, percussion), labaratorno-instrumental issledovanie. Basic clinical syndromes. Jeltuxi clinic, diagnostics		internal diseases" T. 2012; 2. Muxin N.A., Moiseev V.I. "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko V.X .. Grebenev A.L "Propedevtika vnutrennix bolezney", M.1989 4. Narziev Sshch.S. Abdullaeva M.A. "Propaedeutics of internal diseases." Buxoro. 2018;
5	Zabolevanii p echeni i jelchnyx putey. Chronic hepatitis, cirrhosis of the liver, clinic of chronic cholecystitis, diagnosis.	**2**	1. GadaevA; Karimov M.Sshch ..; "Propaedeutics of internal diseases" T. 2012; 2. Muxin N.A., Moiseev V.I. "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko V.X .. Grebenev A.L "Propedevtika vnutrennix bolezney", M.1989 4. Narziev Sshch.S. Abdullaeva M.A. "Propaedeutics of internal diseases." Buxoro. 2018;
6	Kidneys and urinary system. Methods of observation: raspross, physical (osmotr, palpation, percussion), labaratorno-instrumental issledovanie. . Basic clinical syndromes.	**2**	1. GadaevA; Karimov M.Sshch ..; "Propaedeutics of internal diseases" T. 2012; 2. Muxin N.A., Moiseev V.I. "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko V.X .. Grebenev A.L "Propedevtika vnutrennix bolezney", M.1989 4. Narziev Sshch.S. Abdullaeva M.A. "Propaedeutics of internal diseases." Buxoro. 2018;
7	Zabolevanie kidneys and urinary system. Symptomatology of acute and chronic glomerulonephritis and pyelonephritis. Ostraya and chronic renal lack of availability clinic, diagnostics .	**2**	1. GadaevA; Karimov M.Sshch ..; "Propaedeutics of internal diseases" T. 2012; 2. Muxin N.A., Moiseev V.I. "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko V.X .. Grebenev A.L "Propedevtika vnutrennix bolezney", M.1989 4. Narziev Sshch.S. Abdullaeva M.A. "Propaedeutics of internal diseases." Buxoro. 2018;
8	Endocrine system and exchange of substances. Methods of observation: raspross, physical, laboratory-instrumental research.	**2**	1. GadaevA; Karimov M.Sshch ..; "Propaedeutics of internal diseases" T. 2012; 2. Muxin N.A., Moiseev V.I. "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko V.X .. Grebenev A.L "Propedevtika vnutrennix bolezney", M.1989 4. Narziev Sshch.S. Abdullaeva M.A. "Propaedeutics of internal diseases." Buxoro. 2018;
9	Illness of the endocrine system and the exchange of substances .	**2**	1. GadaevA; Karimov M.Sshch ..; "Propaedeutics of internal diseases" T. 2012; 2. Muxin N.A., Moiseev V.I. "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko V.X .. Grebenev A.L "Propedevtika vnutrennix bolezney", M.1989 4. Narziev Sshch.S. Abdullaeva

			M.A. "Propaedeutics of internal diseases." Buxoro. 2018;
10	Blood system. Methods of observation: raspross, physical, laboratory-instrumental research. Disease of the circulatory system. Anemia clinic, diagnostics.	**2**	1. GadaevA; Karimov M.Sshch ..; "Propaedeutics of internal diseases" T. 2012; 2. Muxin N.A., Moiseev V.I. "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko V.X .. Grebenev A.L "Propedevtika vnutrennix bolezney", M.1989 4. Narziev Sshch.S. Abdullaeva M.A. "Propaedeutics of internal diseases." Buxoro. 2018;
	All	**20**	

C oderjanie lektsionnyx zanyatiy

Lecture 1 Pishchevaritelnyy tract. Obsledovanie pishchevoda, jeludka, kishechnikov (rasspros, fizikalnaya, labaratorno-instrumalnaya). Basic clinical syndromes.
References **:** 1. (osn. Lit.) - 4
2. (dop. Lit.) - 4, 10, 11, 12, 16, 19, 21
internet sites

Lecture 2 Zabolevanie pishchevaritelnogo tract: zabolevanie pishchevoda, gastrity.
References **:** 1. (osn. Lit.) - 4
2. (dop. Lit.) - 4, 10, 11, 12, 16, 19, 21
internet sites

Lecture 3 Zabolevanii pishchevaritelnogo tract: yazvennaya bolezn, malabsorbtsii syndrome, chronic colitis.
References **:** 1. (osn. Lit.) - 4
2. (dop. Lit.) - 4, 10, 11, 12, 16, 19, 21
internet sites

Lecture 4 Pechen and jelchnыe ways. Methods of observation: raspross, physical (osmotr, palpation, percussion), labaratorno-instrumental issledovanie. Basic clinical syndromes.
References **:** 1. (osn. Lit.) - 4
2. (dop. Lit.) - 4, 10, 11, 12, 16, 19, 21
internet sites

Lecture 5 Zabolevanii pecheni i jelchnyx putey. Chronic hepatitis, cirrhosis of the liver, chronic cholecystitis.
References **:** 1. (osn. Lit.) - 4
2. (dop. Lit.) - 4, 10, 11, 12, 16, 19, 21
internet sites

Lecture 6 Kidneys and urinary system. Methods of observation: raspross, physical (osmotr, palpation, percussion), labaratorno-instrumental issledovanie. . Basic clinical syndromes.
References **:** 1. (osn. Lit.) - 4
2. (dop. Lit.) - 4, 10, 11, 12, 16, 19, 21
internet sites

Lecture 7 Illness of the kidneys and urinary system. Symptomatology of acute and chronic glomerulonephritis and pyelonephritis. OPN and XPN.
References **:** 1. (osn. Lit.) - 4
2. (dop. Lit.) - 4, 10, 11, 12, 16, 19, 21
internet sites

Lecture 8 Endocrine system and exchange of substances. Methods of observation: raspross, physical, laboratory-instrumental research.

References **:** 1. (osn. Lit.) - 4
2. (dop. Lit.) - 4, 10, 11, 12, 16, 19, 21
internet sites

Lecture 9 Illness of the endocrine system and the exchange of substances .

References **:** 1. (osn. Lit.) - 4
2. (dop. Lit.) - 4, 10, 11, 12, 16, 19, 21
internet sites

Lecture 10 Blood system. Methods of observation: raspross, physical, laboratory-instrumental research. Disease of the circulatory system.

References **:** 1. (osn. Lit.) - 4
2. (dop. Lit.) - 4, 10, 11, 12, 16, 19, 21
internet sites

Naimenovaniya prakticheskix zanyatiy

№	Naimenovanie zanyatiy	chas y	literature
1	Pishchevaritelnyy tract. Obsledovanie pishchevoda, jeludka, kishechnikov (rasspros, fizikalnaya, labaratorno-instrumalnaya).	2	1. GadaevA; Karimov M.Sshch ..; "Propaedeutics of internal diseases" T. 2012; 2. Muxin N.A., Moiseev V.I. "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko V.X .. Grebenev A.L “Propedevtika vnutrennix bolezney”, M.1989 4. Narziev Sh.S. S. Abdullaeva M.A. "Propaedeutics of internal diseases." Buxoro. 2018;
2	Basic clinical syndromes. Zabolevanie pishchevaritelnogo tract: dyspepsia syndrome, acute abdomen, acute bleeding from the gastrointestinal tract .	2	1. GadaevA; Karimov M.Sshch ..; "Propaedeutics of internal diseases" T. 2012; 2. Muxin N.A., Moiseev V.I. "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko V.X .. Grebenev A.L “Propedevtika vnutrennix bolezney”, M.1989 4. Narziev Sh.S. Abdullaeva M.A. "Propaedeutics of internal diseases". Bukhara.2018;
3	Zabolevanie pishchevaritelnogo tract: zabolevanie pishchevoda-eyasophagitis, gastritis clinic, diagnostics.	2	1. GadaevA; Karimov M.Sshch ..; "Propaedeutics of internal diseases" T. 2012; 2. Muxin N.A., Moiseev V.I. "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko V.X .. Grebenev A.L “Propedevtika vnutrennix bolezney”, M.1989 4. Narziev Sh.S. Abdullaeva M.A. "Propaedeutics of internal diseases". Bukhara.2018;

4	Yazvennaya bolezn, malabsorbtsii syndrome, chronic colitis clinic, diagnosis.	2	1. GadaevA; Karimov M.Sshch ..; "Propaedeutics of internal diseases" T. 2012; 2. Muxin N.A., Moiseev V.I. "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko V.X .. Grebenev A.L “Propedevtika vnutrennix bolezney”, M.1989 4. Narziev Sh.S. Abdullaeva M.A. "Propaedeutics of internal diseases". Bukhara.2018;
5	Pechen and jelchnye puti. Methods of observation: raspross, physical (osmotr, palpation, percussion).	2	1. GadaevA; Karimov M.Sshch ..; "Propaedeutics of internal diseases" T. 2012; 2. Muxin N.A., Moiseev V.I. "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko V.X .. Grebenev A.L “Propedevtika vnutrennix bolezney”, M.1989 4. Narziev Sh.S. Abdullaeva M.A. "Propaedeutics of internal diseases". Bukhara.2018;
6	Pechen i jelchnыe puti: labaratorno-instrumentalnye issledovanie. Basic clinical syndromes. Jeltuxi clinic, diagnostics.	2	1. GadaevA; Karimov M.Sshch ..; "Propaedeutics of internal diseases" T. 2012; 2. Muxin N.A., Moiseev V.I. "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko V.X .. Grebenev A.L “Propedevtika vnutrennix bolezney”, M.1989 4. Narziev Sh.S. Abdullaeva M.A. "Propaedeutics of internal diseases". Bukhara.2018;
7	Pechen and jelchnye puti. Chronic hepatitis clinic, diagnostics.	1	1. GadaevA; Karimov M.Sshch ..; "Propaedeutics of internal diseases" T. 2012; 2. Muxin N.A., Moiseev V.I. "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko V.X .. Grebenev A.L “Propedevtika vnutrennix bolezney”, M.1989 4. Narziev Sh.S. Abdullaeva M.A. "Propaedeutics of internal diseases". Bukhara.2018;
8	Cirrhosis of the liver, clinic of chronic cholecystitis , diagnosis. Promejetochny exam № 3.	1	1. GadaevA; Karimov M.Sshch ..; "Propaedeutics of internal diseases" T. 2012; 2. Muxin N.A., Moiseev

			V.I. "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko V.X .. Grebenev A.L "Propedevtika vnutrennix bolezney", M.1989 4. Narziev Sh.S. Abdullaeva M.A. "Propaedeutics of internal diseases". Bukhara.2018;
9	Kidneys and urinary system. Methods of observation: raspross, physical (osmotr, palpation, percussion), labaratorno-instrumental issledovanie.	1	1. GadaevA; Karimov M.Sshch ..; "Propaedeutics of internal diseases" T. 2012; 2. Muxin N.A., Moiseev V.I. "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko V.X .. Grebenev A.L "Propedevtika vnutrennix bolezney", M.1989 4. Narziev Sh.S. Abdullaeva M.A. "Propaedeutics of internal diseases". Bukhara.2018;
10	Zabolevanie kidneys and urinary system. Basic clinical syndromes.	1	1. GadaevA; Karimov M.Sshch ..; "Propaedeutics of internal diseases" T. 2012; 2. Muxin N.A., Moiseev V.I. "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko V.X .. Grebenev A.L "Propedevtika vnutrennix bolezney", M.1989 4. Narziev Sh.S. Abdullaeva M.A. "Propaedeutics of internal diseases". Bukhara.2018;
11	Acute and chronic glomerulonephritis clinic, diagnosis	1	1. GadaevA; Karimov M.Sshch ..; "Propaedeutics of internal diseases" T. 2012; 2. Muxin N.A., Moiseev V.I. "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko V.X .. Grebenev A.L "Propedevtika vnutrennix bolezney", M.1989 4. Narziev Sh.S. Abdullaeva M.A. "Propaedeutics of internal diseases". Bukhara.2018;
12	Acute and chronic pyelonephritis. Ostraya and chronic renal insufficiency clinic, diagnostics	1	1. GadaevA; Karimov M.Sshch ..; "Propaedeutics of internal diseases" T. 2012; 2. Muxin N.A., Moiseev V.I. "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko V.X .. Grebenev A.L

			“Propedevtika vnutrennix bolezney”, M.1989 4. Narziev Sh.S. Abdullaeva M.A. "Propaedeutics of internal diseases". Bukhara.2018;
13	Bone-мышechnaya and soedenitelnaya tissue. Methods of observation: raspross, physical (osmotr, palpation, percussion), labaratorno-instrumental issledovanie. Basic clinical syndromes. SKV, systemic scleroderma clinic, diagnostics	1	1. GadaevA; Karimov M.Sshch ..; "Propaedeutics of internal diseases" T. 2012; 2. Muxin N.A., Moiseev V.I. "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko V.X .. Grebenev A.L “Propedevtika vnutrennix bolezney”, M.1989 4. Narziev Sh.S. Abdullaeva M.A. "Propaedeutics of internal diseases". Bukhara.2018;
14	Endocrine system and exchange of substances. Methods of observation: raspross, physical, laboratory-instrumental research	1	1. GadaevA; Karimov M.Sshch ..; "Propaedeutics of internal diseases" T. 2012; 2. Muxin N.A., Moiseev V.I. "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko V.X .. Grebenev A.L “Propedevtika vnutrennix bolezney”, M.1989 Narziev Sh.S.
15	Illness of the endocrine system and exchange of substances . Diabetes mellitus, thyrotoxicosis, hypothyroidism clinic, diagnosis	1	1. GadaevA; Karimov M.Sshch ..; "Propaedeutics of internal diseases" T. 2012; 2. Muxin N.A., Moiseev V.I. "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko V.X .. Grebenev A.L “Propedevtika vnutrennix bolezney”, M.1989 4. Narziev Sh.S. Abdullaeva M.A. "Propaedeutics of internal diseases". Bukhara.2018;
16	Blood system. Methods of observation: raspross, physical, laboratory-instrumental research	1	1. GadaevA; Karimov M.Sshch ..; "Propaedeutics of internal diseases" T. 2012; 2. Muxin N.A., Moiseev V.I. "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko V.X .. Grebenev A.L “Propedevtika vnutrennix bolezney”, M.1989 4. Narziev Sh.S. Abdullaeva M.A. "Propaedeutics of internal diseases". Bukhara.2018;

17	Disease of the circulatory system. Anemia clinic, diagnostics	1	1. GadaevA; Karimov M.Sshch ..; "Propaedeutics of internal diseases" T. 2012; 2. Muxin N.A., Moiseev V.I. "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko V.X .. Grebenev A.L "Propedevtika vnutrennix bolezney", M.1989 4. Narziev Sh.S. Abdullaeva M.A. "Propaedeutics of internal diseases". Bukhara.2018;
18	The history of the disease, practical skills	1	1. GadaevA; Karimov M.Sshch ..; "Propaedeutics of internal diseases" T. 2012;
	All	24	

Short content of practical and clinical lessons

Topic 1 Cooking tract. Obsledovanie pishchevoda, jeludka, kishechnikov (rasspros, fizikalnaya, labaratorno-instrumalnaya).

Interactive method: bow, brainstorm, tour of Galeree, snowball method

References **:** 1. (osn. Lit.) - 4
2. (dop. Lit.) - 4, 10, 11, 12, 16, 19, 21
internet sites

Topic **2** Basic clinical syndromes. Zabolevanie pishchevaritelnogo tract: dyspepsia syndrome, acute abdomen, acute bleeding from the gastrointestinal tract .

Interactive method: Slaboe link, round table method, pen on the middle of the table

References **:** 1. (osn. Lit.) - 4
2. (dop. Lit.) - 4, 10, 11, 12, 16, 19, 21
internet sites

Topic **3** Zabolevanie pishchevaritelnogo tract: zabolevanie pishchevoda, gastrity.

Interactive method: 3-x stupen. interview, cerebral palsy, pchelinyy roy

References **:** 1. (osn. Lit.) - 4
2. (dop. Lit.) - 4, 10, 11, 12, 16, 19, 21
internet sites

Topic **4** Yazvennaya bolezn, malabsorbtsii syndrome, chronic colitis.

Interactive method: Akademicheskaya polem., Slab. link method snow **.**

References **:** 1. (osn. Lit.) - 4
2. (dop. Lit.) - 4, 10, 11, 12, 16, 19, 21
internet sites

Topic **5** Biscuits and jelly dishes. Methods of observation: raspross, physical (osmotr, palpation, percussion).

Interactive method: 3-x stupen. interview, round table, snow method

References **:** 1. (osn. Lit.) - 4
2. (dop. Lit.) - 4, 10, 11, 12, 16, 19, 21
internet sites

Topic **6** Pechen and jelchnые ways: laboratory-instrumental research. Basic clinical syndromes.

Interactive method: a ssisment, weak link, pchelinyy roy, ratatsii method

References **:** 1. (osn. Lit.) - 4
2. (dop. Lit.) - 4, 10, 11, 12, 16, 19, 21
internet sites

Theme 7 Pechen and jelchnыe puti. Chronic hepatitis.
Interactive method: Akademicheskaya polem. pen on the middle of the table
References **:** 1. (osn. Lit.) - 4
2. (dop. Lit.) - 4, 10, 11, 12, 16, 19, 21
internet sites
Topic **8** Cirrhosis of the liver, chronic cholecystitis. Promejetochny exam № 3.
Interactive method: Gruperasslede, 3-x stupenchat. interview, snow method.
References **:** 1. (osn. Lit.) - 4
2. (dop. Lit.) - 4, 10, 11, 12, 16, 19, 21
internet sites
Topic **9** Kidneys and urinary system. Methods of observation: raspross, physical (osmotr, palpation, percussion), labaratorno-instrumental issledovanie.
Interactive method: Method of incident, tour of Galeree, academic field.
References **:** 1. (osn. Lit.) - 4
2. (dop. Lit.) - 4, 10, 11, 12, 16, 19, 21
internet sites
Topic **10** Illness of the kidneys and urinary system. Basic clinical syndromes.
Interactive method: arrow, pen on se-red.stola, 3-x stupenchat. interview
References **:** 1. (osn. Lit.) - 4
2. (dop. Lit.) - 4, 10, 11, 12, 16, 19, 21
internet sites
Topic **11** Acute and chronic glomerulonephritis.
Interactive method: ratatsii method, akademicheskaya polemika
References **:** 1. (osn. Lit.) - 4
2. (dop. Lit.) - 4, 10, 11, 12, 16, 19, 21
internet sites
T ema 12 Acute and chronic pyelonephritis. Ostraya and chronic renal insufficiency.
Interactive method: method of problem solving, tour of Galeree
References **:** 1. (osn. Lit.) - 4
2. (dop. Lit.) - 4, 10, 11, 12, 16, 19, 21
internet sites
Topic **13** Kostno-mышechnaya i soedenitelnaya tkan. Methods of observation: raspross, physical (osmotr, palpation, percussion), labaratorno-instrumental issledovanie. Basic clinical syndromes.
Interactive method: assisment, bee sting, cerebral attack
References **:** 1. (osn. Lit.) - 4
2. (dop. Lit.) - 4, 10, 11, 12, 16, 19, 21
internet sites
Topic 14 Endocrine system and exchange of substances. Methods of observation: raspross, physical, laboratory-instrumental research.
Interactive method: Akademicheskaya polemika, met.ratatsii, Cluster
References **:** 1. (osn. Lit.) - 4
2. (dop. Lit.) - 4, 10, 11, 12, 16, 19, 21
internet sites
Topic **15** Illness of the endocrine system and the exchange of substances .
Interactive method: 3-x stupenchat. interview, brainstorming, Gallery
References **:** 1. (osn. Lit.) - 4
2. (dop. Lit.) - 4, 10, 11, 12, 16, 19, 21
internet sites
Topic 16 Blood system. Methods of observation: raspross, physical, laboratory-instrumental research.
Interactive method: snow method, academic controversy

References **:** 1. (osn. Lit.) - 4
2. (dop. Lit.) - 4, 10, 11, 12, 16, 19, 21
internet sites

Topic **17** Disease of the circulatory system.

Interactive method: 3-x stupenchat. interview, incident method

References **:** 1. (osn. Lit.) - 4
2. (dop. Lit.) - 4, 10, 11, 12, 16, 19, 21
internet sites

Topic 18 History of the disease, practical skills

Interactive method: incident method, academician. controversy

References **:** 1. (osn. Lit.) - 4
2. (dop. Lit.) - 4, 10, 11, 12, 16, 19, 21
internet sites

Naimenovaniya klinicheskix zanyatiy

№	Naimenovanie zanyatiy	Ch a s y	literature
1	Pishchevaritelnyy tract. Obsledovanie pishchevoda, jeludka, kishechnikov (rasspros, fizikalnaya, labaratorno-instrumalnaya).	2	1. GadaevA; Karimov M.Sshch ..; "Propaedeutics of internal diseases" T. 2012; 2. Muxin N.A., Moiseev V.I. "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko V.X .. Grebenev A.L “Propedevtika vnutrennix bolezney”, M.1989 4. Narziev Sh.S. Abdullaeva M.A. "Propaedeutics of internal diseases". Bukhara.2018;
2	Basic clinical syndromes. Zabolevanie pishchevaritelnogo tract: dyspepsia syndrome, acute abdomen, acute bleeding from the gastrointestinal tract .	2	1. GadaevA; Karimov M.Sshch ..; "Propaedeutics of internal diseases" T. 2012; 2. Muxin N.A., Moiseev V.I. "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko V.X .. Grebenev A.L “Propedevtika vnutrennix bolezney”, M.1989 4. Narziev Sh.S. Abdullaeva M.A. "Propaedeutics of internal diseases". Bukhara.2018;
3	Zabolevanie pishchevaritelnogo tract: zabolevanie pishchevoda-eyasophagitis, gastritis clinic, diagnostics.	2	1. GadaevA; Karimov M.Sshch ..; "Propaedeutics of internal diseases" T. 2012; 2. Muxin N.A., Moiseev V.I. "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko V.X .. Grebenev A.L “Propedevtika vnutrennix bolezney”, M.1989 4. Narziev Sh.S. Abdullaeva M.A. "Propaedeutics of internal

			diseases". Bukhara.2018;
4	Yazvennaya bolezn, malabsorbtsii syndrome, chronic colitis clinic, diagnosis.	2	1. GadaevA; Karimov M.Sshch ..; "Propaedeutics of internal diseases" T. 2012; 2. Muxin N.A., Moiseev V.I. "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko V.X .. Grebenev A.L "Propedevtika vnutrennix bolezney", M.1989 4. Narziev Sh.S. Abdullaeva M.A. "Propaedeutics of internal diseases". Bukhara.2018;
5	Pechen and jelchnye puti. Methods of observation: raspross, physical (osmotr, palpation, percussion).	2	1. GadaevA; Karimov M.Sshch ..; "Propaedeutics of internal diseases" T. 2012; 2. Muxin N.A., Moiseev V.I. "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko V.X .. Grebenev A.L "Propedevtika vnutrennix bolezney", M.1989 4. Narziev Sh.S. Abdullaeva M.A. "Propaedeutics of internal diseases". Bukhara.2018;
6	Pechen i jelchnыe puti: labaratorno-instrumentalnye issledovanie. Basic clinical syndromes. Jeltuxi clinic, diagnostics.	2	1. GadaevA; Karimov M.Sshch ..; "Propaedeutics of internal diseases" T. 2012; 2. Muxin N.A., Moiseev V.I. "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko V.X .. Grebenev A.L "Propedevtika vnutrennix bolezney", M.1989 4. Narziev Sh.S. Abdullaeva M.A. "Propaedeutics of internal diseases". Bukhara.2018;
7	Pechen and jelchnye puti. Chronic hepatitis clinic, diagnostics.	3	1. GadaevA; Karimov M.Sshch ..; "Propaedeutics of internal diseases" T. 2012; 2. Muxin N.A., Moiseev V.I. "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko V.X .. Grebenev A.L "Propedevtika vnutrennix bolezney", M.1989 4. Narziev Sh.S. Abdullaeva M.A. "Propaedeutics of internal diseases". Bukhara.2018;
8	Cirrhosis of the liver, clinic of chronic cholecystitis , diagnosis. Promejetochny exam № 3.	3	1. GadaevA; Karimov M.Sshch ..; "Propaedeutics of internal diseases" T. 2012;

			2. Muxin N.A., Moiseev V.I. "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko V.X .. Grebenev A.L “Propedevtika vnutrennix bolezney”, M.1989 4. Narziev Sh.S. Abdullaeva M.A. "Propaedeutics of internal diseases". Bukhara.2018;
9	Kidneys and urinary system. Methods of observation: raspross, physical (osmotr, palpation, percussion), labaratorno-instrumental issledovanie.	3	1. GadaevA; Karimov M.Sshch ..; "Propaedeutics of internal diseases" T. 2012; 2. Muxin N.A., Moiseev V.I. "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko V.X .. Grebenev A.L “Propedevtika vnutrennix bolezney”, M.1989 4. Narziev Sh.S. Abdullaeva M.A. "Propaedeutics of internal diseases". Bukhara.2018;
10	Zabolevanie kidneys and urinary system. Basic clinical syndromes.	3	1. GadaevA; Karimov M.Sshch ..; "Propaedeutics of internal diseases" T. 2012; 2. Muxin N.A., Moiseev V.I. "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko V.X .. Grebenev A.L “Propedevtika vnutrennix bolezney”, M.1989 4. Narziev Sh.S. Abdullaeva M.A. "Propaedeutics of internal diseases". Bukhara.2018;
11	Acute and chronic glomerulonephritis clinic, diagnosis	3	1. GadaevA; Karimov M.Sshch ..; "Propaedeutics of internal diseases" T. 2012; 2. Muxin N.A., Moiseev V.I. "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko V.X .. Grebenev A.L “Propedevtika vnutrennix bolezney”, M.1989 4. Narziev Sh.S. Abdullaeva M.A. "Propaedeutics of internal diseases". Bukhara.2018;
12	Acute and chronic pyelonephritis. Ostrau xronicheskaya pochechnaya nedostatochnost clinical diagnostics,	3	1. GadaevA; Karimov M.Sshch ..; "Propaedeutics of internal diseases" T. 2012; 2. Muxin N.A., Moiseev V.I. "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko V.X .. Grebenev A.L

			“Propedevtika vnutrennix bolezney”, M.1989 4. Narziev Sh.S. Abdullaeva M.A. "Propaedeutics of internal diseases". Bukhara.2018;
13	Bone-mыshechnaya and soedenitelnaya tissue. Methods of observation: raspross, physical (osmotr, palpation, percussion), labaratorno-instrumental issledovanie. Basic clinical syndromes. SKV, systemic scleroderma clinic, diagnostics.	3	1. GadaevA; Karimov M.Sshch ..; "Propaedeutics of internal diseases" T. 2012; 2. Muxin N.A., Moiseev V.I. "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko V.X .. Grebenev A.L “Propedevtika vnutrennix bolezney”, M.1989 4. Narziev Sh.S. Abdullaeva M.A. "Propaedeutics of internal diseases". Bukhara.2018;
14	Endocrine system and exchange of substances. Methods of observation: raspross, physical, laboratory-instrumental research	3	1. GadaevA; Karimov M.Sshch ..; "Propaedeutics of internal diseases" T. 2012; 2. Muxin N.A., Moiseev V.I. "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko V.X .. Grebenev A.L “Propedevtika vnutrennix bolezney”, M.1989 4. Narziev Sh.S. Abdullaeva M.A. "Propaedeutics of internal diseases". Bukhara.2018;
15	Illness of the endocrine system and exchange of substances . Diabetes mellitus, thyrotoxicosis, hypothyroidism clinic, diagnosis.	3	1. GadaevA; Karimov M.Sshch ..; "Propaedeutics of internal diseases" T. 2012; 2. Muxin N.A., Moiseev V.I. "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko V.X .. Grebenev A.L “Propedevtika vnutrennix bolezney”, M.1989 4. Narziev Sh.S. Abdullaeva M.A. "Propaedeutics of internal diseases". Bukhara.2018;
16	Blood system. Methods of observation: raspross, physical, laboratory-instrumental research	3	1. GadaevA; Karimov M.Sshch ..; "Propaedeutics of internal diseases" T. 2012; 2. Muxin N.A., Moiseev V.I. "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko V.X .. Grebenev A.L “Propedevtika vnutrennix bolezney”, M.1989 4. Narziev Sh.S. Abdullaeva

			M.A. "Propaedeutics of internal diseases". Bukhara.2018;
17	Disease of the circulatory system. Anemia clinic, diagnostics	2	1. GadaevA; Karimov M.Sshch ..; "Propaedeutics of internal diseases" T. 2012; 2. Muxin N.A., Moiseev V.I. "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko V.X .. Grebenev A.L "Propedevtika vnutrennix bolezney", M.1989 4. Narziev Sh.S. Abdullaeva M.A. "Propaedeutics of internal diseases". Bukhara.2018;
18	The history of the disease, practical skills	2	1. GadaevA; Karimov M.Sshch ..; "Propaedeutics of internal diseases" T. 2012;
	All	46	

Short content of practical and clinical lessons

Topic 1 Cooking tract. Obsledovanie pishchevoda, jeludka, kishechnikov (rasspros, fizikalnaya, labaratorno-instrumalnaya).

Interactive method: bow, brainstorm, tour of Galeree, snowball method

References **:** 1. (osn. Lit.) - 4
2. (dop. Lit.) - 4, 10, 11, 12, 16, 19, 21
internet sites

Topic **2** Basic clinical syndromes. Zabolevanie pishchevaritelnogo tract: dyspepsia syndrome, acute abdomen, acute bleeding from the gastrointestinal tract **.**

Interactive method: Slaboe link, round table method, pen on the middle of the table

References **:** 1. (osn. Lit.) - 4
2. (dop. Lit.) - 4, 10, 11, 12, 16, 19, 21
internet sites

Topic **3** Zabolevanie pishchevaritelnogo tract: zabolevanie pishchevoda, gastrity.

Interactive method: 3-x stupen. interview, cerebral palsy, pchelinyy roy

References **:** 1. (osn. Lit.) - 4
2. (dop. Lit.) - 4, 10, 11, 12, 16, 19, 21
internet sites

Topic **4** Yazvennaya bolezn, malabsorbtsii syndrome, chronic colitis.

Interactive method: Akademicheskaya polem., Slab. link method snow **.**

References **:** 1. (osn. Lit.) - 4
2. (dop. Lit.) - 4, 10, 11, 12, 16, 19, 21
internet sites

Topic **5** Biscuits and jelly dishes. Methods of observation: raspross, physical (osmotr, palpation, percussion).

Interactive method: 3-x stupen. interview, round table, snow method

References **:** 1. (osn. Lit.) - 4
2. (dop. Lit.) - 4, 10, 11, 12, 16, 19, 21
internet sites

Topic **6** Pechen and jelchnые ways: laboratory-instrumental research. Basic clinical syndromes.

Interactive method: a ssisment, weak link, pchelinyy roy, ratatsii method
References **:** 1. (osn. Lit.) - 4
2. (dop. Lit.) - 4, 10, 11, 12, 16, 19, 21
internet sites

Theme 7 Pechen and jelchnыe puti. Chronic hepatitis.
Interactive method: Akademicheskaya polem. pen on the middle of the table
References **:** 1. (osn. Lit.) - 4
2. (dop. Lit.) - 4, 10, 11, 12, 16, 19, 21
internet sites

Topic **8** Cirrhosis of the liver, chronic cholecystitis. Promejetochny exam № 3.
Interactive method: Gruperasslede, 3-x stupenchat. interview, snow method.
References **:** 1. (osn. Lit.) - 4
2. (dop. Lit.) - 4, 10, 11, 12, 16, 19, 21
internet sites

Topic **9** Kidneys and urinary system. Methods of observation: raspross, physical (osmotr, palpation, percussion), labaratorno-instrumental issledovanie.
Interactive method: Method of incident, tour of Galeree, academic field.
References **:** 1. (osn. Lit.) - 4
2. (dop. Lit.) - 4, 10, 11, 12, 16, 19, 21
internet sites

Topic **10** Illness of the kidneys and urinary system. Basic clinical syndromes.
Interactive method: arrow, pen on se-red.stola, 3-x stupenchat. interview
References **:** 1. (osn. Lit.) - 4
2. (dop. Lit.) - 4, 10, 11, 12, 16, 19, 21
internet sites

Topic **11** Acute and chronic glomerulonephritis.
Interactive method: ratatsii method, akademicheskaya polemika
References **:** 1. (osn. Lit.) - 4
2. (dop. Lit.) - 4, 10, 11, 12, 16, 19, 21
internet sites

T ema 12 Acute and chronic pyelonephritis. Ostraya and chronic renal insufficiency.
Interactive method: method of problem solving, tour of Galeree
References **:** 1. (osn. Lit.) - 4
2. (dop. Lit.) - 4, 10, 11, 12, 16, 19, 21
internet sites

Topic **13** Kostno-mышechnaya i soedenitelnaya tkan. Methods of observation: raspross, physical (osmotr, palpation, percussion), labaratorno-instrumental issledovanie. Basic clinical syndromes.
Interactive method: assisment, bee sting, cerebral attack
References **:** 1. (osn. Lit.) - 4
2. (dop. Lit.) - 4, 10, 11, 12, 16, 19, 21
internet sites

Topic 14 Endocrine system and exchange of substances. Methods of observation: raspross, physical, laboratory-instrumental research.
Interactive method: Akademicheskaya polemika, met.ratatsii, Cluster
References **:** 1. (osn. Lit.) - 4
2. (dop. Lit.) - 4, 10, 11, 12, 16, 19, 21
internet sites

Topic **15** Illness of the endocrine system and the exchange of substances .
Interactive method: 3-x stupenchat. interview, brainstorming, Gallery
References **:** 1. (osn. Lit.) - 4
2. (dop. Lit.) - 4, 10, 11, 12, 16, 19, 21

internet sites

Topic 16 Blood system. Methods of observation: raspross, physical, laboratory-instrumental research.

Interactive method: snow method, academic controversy

References **:** 1. (osn. Lit.) - 4

2. (dop. Lit.) - 4, 10, 11, 12, 16, 19, 21

internet sites

Topic **17** Disease of the circulatory system.

Interactive method: 3-x stupenchat. interview, incident method

References **:** 1. (osn. Lit.) - 4

2. (dop. Lit.) - 4, 10, 11, 12, 16, 19, 21

internet sites

Topic 18 History of the disease, practical skills

Interactive method: incident method, academician. controversy

References **:** 1. (osn. Lit.) - 4

2. (dop. Lit.) - 4, 10, 11, 12, 16, 19, 21

internet sites

Names of lecture employment

№	**Names of lecture employment**	**Hours**
1	Digestive tract. Examination of the esophagus, stomach, intestines (interrogation, physical, laboratory and instrumental). The main clinical syndromes.	2
2	Digestive tract disease: disease of the esophagus, gastritis.	2
3	Diseases of the digestive tract. Peptic ulcer, malabsorption syndrome, chronic colitis.	2
4	Liver and bile ducts. Methods of examination: dissemination, physical (examination, palpation, percussion), laboratory and instrumental research. The main clinical syndromes.	2
5	Diseases of the liver and biliary tract. Chronic hepatitis, cirrhosis, chronic cholecystitis.	2
6	Kidneys and urinary system. Methods of examination: dissemination, physical (examination, palpation, percussion), laboratory and instrumental research. . The main clinical syndromes.	2
7	Disease of the kidneys and urinary system. Symptomatology of acute and chronic glomerulonephritis and pyelonephritis. Acute and chronic renal failure.	2
8	Endocrine and metabolic system. Methods of examination: dissemination, physical, laboratory and instrumental research.	2
9	Endocrine and metabolic disease.	2
10	Blood system. Methods of examination: dissemination, physical, laboratory and instrumental research. Disease of the blood system.	2
	Total	2 0

Lecture Content

Lecture 1 Digestive tract. Examination of the esophagus, stomach, intestines (interrogation, physical, laboratory and instrumental). The main clinical syndromes.

Literature: 1. (main lit.) - 4

2. (additional lit.) - 4, 10, 11, 12, 16, 19, 21

internet sites

Lecture 2 Disease of the digestive tract: disease of the esophagus, gastritis.

Literature: 1. (main lit.) - 4

2. (additional lit.) - 4, 10, 11, 12, 16, 19, 21
internet sites

Lecture 3 Digestive tract diseases: peptic ulcer, malabsorption syndrome, chronic colitis.
Literature: 1. (main lit.) - 4
2. (additional lit.) - 4, 10, 11, 12, 16, 19, 21
internet sites

Lecture 4 Liver and biliary tract. Methods of examination: dissemination, physical (examination, palpation, percussion), laboratory and instrumental research. The main clinical syndromes.
Literature: 1. (main lit.) - 4
2. (additional lit.) - 4, 10, 11, 12, 16, 19, 21
internet sites

Lecture 5 Diseases of the liver and biliary tract. Chronic hepatitis, cirrhosis, chronic cholecystitis.
Literature: 1. (main lit.) - 4
2. (additional lit.) - 4, 10, 11, 12, 16, 19, 21
internet sites

Lecture 6 Kidneys and urinary system. Methods of examination: dissemination, physical (examination, palpation, percussion), laboratory and instrumental research. . The main clinical syndromes.
Literature: 1. (main lit.) - 4
2. (additional lit.) - 4, 10, 11, 12, 16, 19, 21
internet sites

Lecture 7 Disease of the kidneys and urinary system. Symptomatology of acute and chronic glomerulonephritis and pyelonephritis. Acute renal failure and chronic renal failure.
Literature: 1. (main lit.) - 4
2. (additional lit.) - 4, 10, 11, 12, 16, 19, 21
internet sites

Lecture 8 Endocrine and metabolic system. Methods of examination: dissemination, physical, laboratory and instrumental research.
Literature: 1. (main lit.) - 4
2. (additional lit.) - 4, 10, 11, 12, 16, 19, 21
internet sites

Lecture 9 Disease of the endocrine and metabolic system.
Literature: 1. (main lit.) - 4
2. (additional lit.) - 4, 10, 11, 12, 16, 19, 21
internet sites

Lecture 10 Blood system. Methods of examination: dissemination, physical, laboratory and instrumental research. Disease of the blood system.
Literature: 1. (main lit.) - 4
2. (additional lit.) - 4, 10, 11, 12, 16, 19, 21
internet sites

Names of a practical training

№	Names of a training	practical training	clinical training
1	Digestive	**2**	1. GadaevA; Karimov M.Sh ..; "Propaedeutics of

	tract. Examination of the esophagus, stomach, intestines (interrogation, physical, laboratory and instrumental).		internal diseases" T. 2012; 2. Muxin NA, Moiseev VI "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko VX. Grebenev AL "Propedevtika vnutrennix bolezney", M.1989 4. Narziyev Sh.S. Abdullayeva MA "Propaedeutics of internal diseases". Bukhara.2018;
2	The main clinical syndromes. Digestive tract disease: dyspepsia syndrome, acute abdomen, acute bleeding from the digestive tract.	**2**	1. GadaevA; Karimov M.Sh ..; "Propaedeutics of internal diseases" T. 2012; 2. Muxin NA, Moiseev VI "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko VX. Grebenev AL "Propedevtika vnutrennix bolezney", M.1989 4. Narziyev Sh.S. Abdullayeva MA "Propaedeutics of internal diseases". Bukhara.2018;
3	Digestive tract disease: disease of the esophagus, gastritis.	**2**	1. GadaevA; Karimov M.Sh ..; "Propaedeutics of internal diseases" T. 2012; 2. Muxin NA, Moiseev VI "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko VX. Grebenev AL "Propedevtika vnutrennix bolezney", M.1989 4. Narziyev Sh.S. Abdullayeva MA "Propaedeutics of internal diseases". Bukhara.2018;
4	Peptic ulcer, malabsorption syndrome, chronic colitis.	**2**	1. GadaevA; Karimov M.Sh ..; " Propaedeutics of internal diseases " T. 2012; 2. Muxin NA, Moiseev VI "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko VX. Grebenev AL "Propedevtika vnutrennix bolezney", M.1989 4. Narziyev Sh.S. Abdullayeva MA "Propaedeutics of internal diseases". Bukhara.2018;
5	Liver and bile ducts. Examination methods: dissemination, physical (examination, palpation, percussion).	**2**	1. GadaevA; Karimov M.Sh ..; "Propaedeutics of internal diseases" T. 2012; 2. Muxin NA, Moiseev VI "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko VX. Grebenev AL "Propedevtika vnutrennix bolezney", M.1989 4. Narziyev Sh.S. Abdullayeva MA "Propaedeutics of internal diseases". Bukhara.2018;
6	Liver and biliary tract: laboratory and instrumental research. The main clinical syndromes.	**2**	1. GadaevA; Karimov M.Sh ..; "Propaedeutics of internal diseases" T. 2012; 2. Muxin NA, Moiseev VI "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko VX. Grebenev AL "Propedevtika vnutrennix bolezney", M.1989 4. Narziyev Sh.S. Abdullayeva MA "Propaedeutics of internal diseases".

			Bukhara.2018;
7	Liver and bile ducts. Chronic hepatitis.	**1**	1. GadaevA; Karimov M.Sh ..; "Propaedeutics of internal diseases" T. 2012; 2. Muxin NA, Moiseev VI "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko VX. Grebenev AL “Propedevtika vnutrennix bolezney”, M.1989 4. Narziyev Sh.S. Abdullayeva MA "Propaedeutics of internal diseases". Bukhara.2018;
8	Cirrhosis of the liver, chronic cholecystitis. Interim exam number 3.	**1**	1. Gadaev A; Karimov M.Sh ..; "Propaedeutics of internal diseases" T. 2012; 2. Muxin NA, Moiseev VI "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko VX. Grebenev AL “Propedevtika vnutrennix bolezney”, M.1989 4. Narziyev Sh.S. Abdullayeva MA "Propaedeutics of internal diseases". Bukhara.2018;
9	Kidneys and urinary system. Methods of examination: dissemination, physical (examination, palpation, percussion), laboratory and instrumental research.	**1**	1. GadaevA; Karimov M.Sh ..; "Propaedeutics of internal diseases" T. 2012; 2. Muxin NA, Moiseev VI "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko VX. Grebenev AL “Propedevtika vnutrennix bolezney”, M.1989 4. Narziyev Sh.S. Abdullayeva MA "Propaedeutics of internal diseases". Bukhara.2018;
10	Kidney and urinary system disease. The main clinical syndromes.	**1**	1. GadaevA; Karimov M.Sh ..; "Propaedeutics of internal diseases" T. 2012; 2. Muxin NA, Moiseev VI "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko VX. Grebenev AL “Propedevtika vnutrennix bolezney”, M.1989 4. Narziyev Sh.S. Abdullayeva MA "Propaedeutics of internal diseases". Bukhara.2018;
11	Acute and chronic glomerulonephritis.	**1**	1. GadaevA; Karimov M.Sh ..; "Propaedeutics of internal diseases" T. 2012; 2. Muxin NA, Moiseev VI "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko VX. Grebenev AL “Propedevtika vnutrennix bolezney”, M.1989 4. Narziyev Sh.S. Abdullayeva MA "Propaedeutics of internal diseases". Bukhara.2018;
12	Acute and chronic pyelonephritis. Acute and chronic renal failure	**1**	1. GadaevA; Karimov M.Sh ..; "Propaedeutics of internal diseases" T. 2012; 2. Muxin NA, Moiseev VI "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko VX. Grebenev AL “Propedevtika vnutrennix bolezney”, M.1989

			4. Narziyev Sh.S. Abdullayeva MA "Propaedeutics of internal diseases". Bukhara.2018;
13	Musculoskeletal and connective tissue. Methods of examination: dissemination, physical (examination, palpation, percussion), laboratory and instrumental research. The main clinical syndromes.	**1**	1. GadaevA; Karimov M.Sh ..; "Propaedeutics of internal diseases" T. 2012; 2. Muxin NA, Moiseev VI "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko VX. Grebenev AL "Propedevtika vnutrennix bolezney", M.1989 4. Narziyev Sh.S. Abdullayeva MA "Propaedeutics of internal diseases". Bukhara.2018;
14	Endocrine and metabolic system. Methods of examination: dissemination, physical, laboratory and instrumental research.	**1**	1. GadaevA; Karimov M.Sh ..; "Propaedeutics of internal diseases" T. 2012; 2. Muxin NA, Moiseev VI "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko VX. Grebenev AL "Propedevtika vnutrennix bolezney", M.1989 4. Narziyev Sh.S. Abdullayeva MA "Propaedeutics of internal diseases". Bukhara.2018;
15	Endocrine and metabolic disease.	**1**	1. GadaevA; Karimov M.Sh ..; "Propaedeutics of internal diseases" T. 2012; 2. Muxin NA, Moiseev VI "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko VX. Grebenev AL "Propedevtika vnutrennix bolezney", M.1989 4. Narziyev Sh.S. Abdullayeva MA "Propaedeutics of internal diseases". Bukhara.2018;
16	Blood system. Methods of examination: dissemination, physical, laboratory and instrumental research.	**1**	1. GadaevA; Karimov M.Sh ..; "Propaedeutics of internal diseases" T. 2012; 2. Muxin NA, Moiseev VI "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko VX. Grebenev AL "Propedevtika vnutrennix bolezney", M.1989 4. Narziyev Sh.S. Abdullayeva MA "Propaedeutics of internal diseases". Bukhara.2018;
17	Disease of the blood system.	**1**	1. GadaevA; Karimov M.Sh ..; "Propaedeutics of internal diseases" T. 2012; 2. Muxin NA, Moiseev VI "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko VX. Grebenev AL "Propedevtika vnutrennix bolezney", M.1989 4. Narziyev Sh.S. Abdullayeva MA "Propaedeutics of internal diseases". Bukhara.2018;
18	Medical history, practical skills	**1**	1. GadaevA; Karimov M.Sh ..; "Propaedeutics of internal diseases" T. 2012;

			2. Muxin NA, Moiseev VI "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko VX. Grebenev AL “Propedevtika vnutrennix bolezney”, M.1989 4. Narziyev Sh.S. Abdullayeva MA "Propaedeutics of internal diseases". Bukhara.2018;
	Total.	**24**	1. GadaevA; Karimov M.Sh ..; "Propaedeutics of internal diseases" T. 2012;

Names of a clinical training

№	**Names of a training**	**clinical training**	
1	Digestive tract. Examination of the esophagus, stomach, intestines (interrogation, physical, laboratory and instrumental).	**2**	1. GadaevA; Karimov M.Sh ..; "Propaedeutics of internal diseases" T. 2012; 2. Muxin NA, Moiseev VI "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko VX. Grebenev AL “Propedevtika vnutrennix bolezney”, M.1989 4. Narziyev Sh.S. Abdullayeva MA "Propaedeutics of internal diseases". Bukhara.2018;
2	The main clinical syndromes. Digestive tract disease: dyspepsia syndrome, acute abdomen, acute bleeding from the digestive tract.	**2**	1. GadaevA; Karimov M.Sh ..; "Propaedeutics of internal diseases" T. 2012; 2. Muxin NA, Moiseev VI "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko VX. Grebenev AL “Propedevtika vnutrennix bolezney”, M.1989 4. Narziyev Sh.S. Abdullayeva MA "Propaedeutics of internal diseases". Bukhara.2018;
3	Digestive tract disease: disease of the esophagus, gastritis.	**2**	1. GadaevA; Karimov M.Sh ..; "Propaedeutics of internal diseases" T. 2012; 2. Muxin NA, Moiseev VI "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko VX. Grebenev AL “Propedevtika vnutrennix bolezney”, M.1989 4. Narziyev Sh.S. Abdullayeva MA "Propaedeutics of internal diseases". Bukhara.2018;
4	Peptic ulcer, malabsorption syndrome, chronic colitis.	**2**	1. GadaevA; Karimov M.Sh ..; "Propaedeutics of internal diseases" T. 2012; 2. Muxin NA, Moiseev VI "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko VX. Grebenev AL “Propedevtika vnutrennix bolezney”, M.1989 4. Narziyev Sh.S. Abdullayeva MA "Propaedeutics of internal diseases". Bukhara.2018;
5	Liver and bile	**2**	1. GadaevA; Karimov M.Sh ..; "Propaedeutics of

	ducts. Examination methods: dissemination, physical (examination, palpation, percussion).		internal diseases" T. 2012; 2. Muxin NA, Moiseev VI "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko VX. Grebenev AL "Propedevtika vnutrennix bolezney", M.1989 4. Narziyev Sh.S. Abdullayeva MA "Propaedeutics of internal diseases". Bukhara.2018;
6	Liver and biliary tract: laboratory and instrumental research. The main clinical syndromes.	**2**	1. GadaevA; Karimov M.Sh ..; "Propaedeutics of internal diseases" T. 2012; 2. Muxin NA, Moiseev VI "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko VX. Grebenev AL "Propedevtika vnutrennix bolezney", M.1989 4. Narziyev Sh.S. Abdullayeva MA "Propaedeutics of internal diseases". Bukhara.2018;
7	Liver and bile ducts. Chronic hepatitis.	**3**	1. GadaevA; Karimov M.Sh ..; "Propaedeutics of internal diseases" T. 2012; 2. Muxin NA, Moiseev VI "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko VX. Grebenev AL "Propedevtika vnutrennix bolezney", M.1989 4. Narziyev Sh.S. Abdullayeva MA "Propaedeutics of internal diseases". Bukhara.2018;
8	Cirrhosis of the liver, chronic cholecystitis. Interim exam number 3.	**3**	1. GadaevA; Karimov M.Sh ..; "Propaedeutics of internal diseases" T. 2012; 2. Muxin NA, Moiseev VI "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko VX. Grebenev AL "Propedevtika vnutrennix bolezney", M.1989 4. Narziyev Sh.S. Abdullayeva MA "Propaedeutics of internal diseases". Bukhara.2018;
9	Kidneys and urinary system. Methods of examination: dissemination, physical (examination, palpation, percussion), laboratory and instrumental research.	**3**	1. GadaevA; Karimov M.Sh ..; "Propaedeutics of internal diseases" T. 2012; 2. Muxin NA, Moiseev VI "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko VX. Grebenev AL "Propedevtika vnutrennix bolezney", M.1989 4. Narziyev Sh.S. Abdullayeva MA "Propaedeutics of internal diseases". Bukhara.2018;
10	Kidney and urinary system disease. The main clinical syndromes.	**3**	1. GadaevA; Karimov M.Sh ..; "Propaedeutics of internal diseases" T. 2012; 2. Muxin NA, Moiseev VI "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko VX. Grebenev AL "Propedevtika vnutrennix bolezney", M.1989 4. Narziyev Sh.S. Abdullayeva MA "Propaedeutics of internal diseases".

			Bukhara.2018;
11	Acute and chronic glomerulonephritis.	**3**	1. GadaevA; Karimov M.Sh ..; "Propaedeutics of internal diseases" T. 2012; 2. Muxin NA, Moiseev VI "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko VX. Grebenev AL “Propedevtika vnutrennix bolezney”, M.1989 4. Narziyev Sh.S. Abdullayeva MA "Propaedeutics of internal diseases". Bukhara.2018;
12	Acute and chronic pyelonephritis. Acute and chronic renal failure	**3**	1. GadaevA; Karimov M.Sh ..; "Propaedeutics of internal diseases" T. 2012; 2. Muxin NA, Moiseev VI "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko VX. Grebenev AL “Propedevtika vnutrennix bolezney”, M.1989 4. Narziyev Sh.S. Abdullayeva MA " Propaedeutics of internal diseases". Bukhara.2018
13	Musculoskeletal and connective tissue. Methods of examination: dissemination, physical (examination, palpation, percussion), laboratory and instrumental research. The main clinical syndromes.	**3**	1. GadaevA; Karimov M.Sh ..; "Propaedeutics of internal diseases" T. 2012; 2. Muxin NA, Moiseev VI "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko VX. Grebenev AL “Propedevtika vnutrennix bolezney”, M.1989 4. Narziyev Sh.S. Abdullayeva MA "Propaedeutics of internal diseases". Bukhara.2018;
14	Endocrine and metabolic system. Methods of examination: dissemination, physical, laboratory and instrumental research.	**3**	1. GadaevA; Karimov M.Sh ..; "Propaedeutics of internal diseases" T. 2012; 2. Muxin NA, Moiseev VI "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko VX. Grebenev AL “Propedevtika vnutrennix bolezney”, M.1989 4. Narziyev Sh.S. Abdullayeva MA "Propaedeutics of internal diseases". Bukhara.2018;
15	Endocrine and metabolic disease.	**3**	1. GadaevA; Karimov M.Sh ..; "Propaedeutics of internal diseases" T. 2012; 2. Muxin NA, Moiseev VI "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko VX. Grebenev AL “Propedevtika vnutrennix bolezney”, M.1989 4. Narziyev Sh.S. Abdullayeva MA "Propaedeutics of internal diseases". Bukhara.2018;
16	Blood system. Methods of examination: dissemination, physical, laboratory	**3**	1. GadaevA; Karimov M.Sh ..; "Propaedeutics of internal diseases" T. 2012; 2. Muxin NA, Moiseev VI "Propedevtika vnutrennix bolezney", M. 2000.

	and instrumental research.		3. Vasilenko VX. Grebenev AL “Propedevtika vnutrennix bolezney”, M.1989 4. Narziyev Sh.S. Abdullayeva MA "Propaedeutics of internal diseases". Bukhara.2018;
17	Disease of the blood system.	**2**	1. GadaevA; Karimov M.Sh ..; "Propaedeutics of internal diseases" T. 2012; 2. Muxin NA, Moiseev VI "Propedevtika vnutrennix bolezney", M. 2000. 3. Vasilenko VX. Grebenev AL “Propedevtika vnutrennix bolezney”, M.1989 4. Narziyev Sh.S. Abdullayeva MA "Propaedeutics of internal diseases". Bukhara.2018;
18	Medical history, practical skills	**2**	1. GadaevA; Karimov M.Sh ..; "Propaedeutics of internal diseases" T. 2012;
	Total.	**46**	

Meanings of a practical and clinical training

Theme 1 Digestive tract. Examination of the esophagus, stomach, intestines (interrogation, physical, laboratory and instrumental).

Interactive method: arch, brainstorming, Gallery tour, snowball method

Literature: 1. (main lit.) - 4

2. (additional lit.) - 4, 10, 11, 12, 16, 19, 21

internet sites

Theme 2 The main clinical syndromes. Digestive tract disease: dyspepsia syndrome, acute abdomen, acute bleeding from the digestive tract.

Interactive Method: Weak link, round table method, handle in the middle of the table

Literature: 1. (main lit.) - 4

2. (additional lit.) - 4, 10, 11, 12, 16, 19, 21

internet sites

Theme 3 Digestive tract disease: disease of the esophagus, gastritis.

Interactive method: 3 steps. interview, brainstorming, bee swarm

Literature: 1. (main lit.) - 4

2. (additional lit.) - 4, 10, 11, 12, 16, 19, 21

internet sites

Theme 4: Peptic ulcer, malabsorption syndrome, chronic colitis.

Interactive method: Academic field., Weak. link method snow.

Literature: 1. (main lit.) - 4

2. (additional lit.) - 4, 10, 11, 12, 16, 19, 21

internet sites

Theme 5 Liver and bile ducts. Examination methods: dissemination, physical (examination, palpation, percussion).

Interactive method: 3 steps. interview, round table, snowball method

Literature: 1. (main lit.) - 4

2. (additional lit.) - 4, 10, 11, 12, 16, 19, 21

internet sites

Theme 6 Liver and biliary tract: laboratory and instrumental research. The main clinical syndromes.

Interactive method: Assistance, weak link, bee swarm, ratie method

Literature: 1. (main lit.) - 4
2. (additional lit.) - 4, 10, 11, 12, 16, 19, 21
internet sites

Theme 7 Liver and biliary tract. Chronic hepatitis.
Interactive method: Academic Field. a pen in the middle of the table
Literature: 1. (main lit.) - 4
2. (additional lit.) - 4, 10, 11, 12, 16, 19, 21
internet sites

Theme 8 Cirrhosis of the liver, chronic cholecystitis. Interim exam number 3.
Interactive method: Groupsexled, 3 steps. interview, snow method.
Literature: 1. (main lit.) - 4
2. (additional lit.) - 4, 10, 11, 12, 16, 19, 21
internet sites

Theme 9 Kidneys and urinary system. Methods of examination: dissemination, physical (examination, palpation, percussion), laboratory and instrumental research.
Interactive method: Incident method, Gallery tour, academic field.
Literature: 1. (main lit.) - 4
2. (additional lit.) - 4, 10, 11, 12, 16, 19, 21
internet sites

Theme 10: Disease of the kidneys and urinary system. The main clinical syndromes.
Interactive method: arch, handle on the central table, 3 steps. interview
Literature: 1. (main lit.) - 4
2. (additional lit.) - 4, 10, 11, 12, 16, 19, 21
internet sites

Theme 11 Acute and chronic glomerulonephritis.
Interactive method: ratation method, academic polemic
Literature: 1. (main lit.) - 4
2. (additional lit.) - 4, 10, 11, 12, 16, 19, 21
internet sites

Theme 12 Acute and chronic pyelonephritis. Acute and chronic renal failure.
Interactive Method: Problem Solving Method, Gallery Tour
Literature: 1. (main lit.) - 4
2. (additional lit.) - 4, 10, 11, 12, 16, 19, 21
internet sites

Theme 13 Musculoskeletal and connective tissue. Methods of examination: dissemination, physical (examination, palpation, percussion), laboratory and instrumental research. The main clinical syndromes.
Interactive method: Assistance, Bee Swarm, Brainstorming
Literature: 1. (main lit.) - 4
2. (additional lit.) - 4, 10, 11, 12, 16, 19, 21
internet sites

Theme 14 Endocrine and metabolic system. Methods of examination: dissemination, physical, laboratory and instrumental research.
Interactive method: Academic polemic, met.ratation, Cluster
Literature: 1. (main lit.) - 4
2. (additional lit.) - 4, 10, 11, 12, 16, 19, 21
internet sites

Theme 15 Endocrine and metabolic diseases.
Interactive method: 3 steps. interview, brainstorming, gallery
Literature: 1. (main lit.) - 4
2. (additional lit.) - 4, 10, 11, 12, 16, 19, 21
internet sites

Theme 16 Blood system. Methods of examination: dissemination, physical, laboratory and instrumental research.
Interactive method: snow method., Academic debate
Literature: 1. (main lit.) - 4
2. (additional lit.) - 4, 10, 11, 12, 16, 19, 21
internet sites
Theme 17 Disease of the blood system.
Interactive method: 3 steps. interview, incident method
Literature: 1. (main lit.) - 4
2. (additional lit.) - 4, 10, 11, 12, 16, 19, 21
internet sites
Theme 18 Medical history, practical skills
Interactive Method: Incident Method, Academic. controversy
Literature: 1. (main lit.) - 4
2. (additional lit.) - 4, 10, 11, 12, 16, 19, 21
internet sites

7 . The form and content of the organization of independent education

The main purpose of the student's independent work is the formation and development of knowledge and skills to perform certain educational tasks independently under the guidance and supervision of the teacher.

Requirements for independent work:

- to independently acquire new knowledge and acquire skills.
- Identify convenient ways and means to find the necessary information.
- effective use of information sources and addresses.
- Work with traditional educational and scientific literature, normative documents
- Work with electronic textbooks and databases.
- Targeted use of the Internet.
- to determine the rational solution of the given task.
- Database analysis.

In the organization of independent work of the student follows the subject of propaedeutics of internal medicine

Forms are used:

independent study of some theoretical topics with the help of textbooks;
preparation of information (abstract) on the given topics;
application of theoretical knowledge in practice;
work with automated training and control systems;
Scientific article, preparation of a report for the conference, etc.
Crossword, chainword, weak ring, problem solving, etc. training.

Independent work

№	TOPIC.	Hour
1.	Causes of development of dysphagia, risk factors etiology, pathogenesis, mechanism of pain formation.	4
2.	Clinical manifestations of wound disease, clinical-laboratory criteria, complications and methods of treatment.	4
3.	Etiology, pathogenesis, and classification of pancreatitis. Diagnostic tests for pancreatic tumors .	4
4.	Complications of nonspecific ulcerative colitis and diagnostic examination of the intestine.	4
5.	Etiopathogenesis of diseases associated with hepatomegaly, clinical manifestations, syndromic diagnosis of the disease.	4

6.	Etiopathogenesis, clinical manifestations, and diagnosis of liver cirrhosis.	4
7.	Etiopathogenesis of jaundice, clinical manifestations, syndromic diagnosis of the disease.	4
8.	Etiology, pathogenesis, clinical manifestations, diagnosis and activity criteria of inflammatory and immune inflammatory diseases of the kidneys.	4
9.	Etiology, pathogenesis and methods of diagnosis of nephropathy.	4
10.	Drugs used in the etiology, pathogenesis, classification, clinical syndromes, diagnosis and treatment of chronic renal failure	4
11.	Etiology, pathogenesis, clinical manifestations and diagnosis of tumor syndrome.	4
12.	Diagnostic criteria for rheumatism and rheumatoid arthritis.	4
13.	Diagnostic criteria of seronegative spondyloarthritis (reactive arthritis, Bexterev's disease, psoriatic arthritis).	4
14.	Diagnostic criteria, activity levels and treatment methods of diffuse connective tissue diseases.	4
15.	Diagnostic criteria, activity levels, treatment methods and prevention of dermatomyositis and systemic vasculitis.	4
16.	Diagnostic criteria, activity levels, treatment methods and prevention of nodular periarthritis and nonspecific aortoarteritis.	5
	Total:	65

Themes self-employment

№	**N a z v a n i e t e m y**	**Chasy**
1.	Prichiny developmental dysphagia, risk factors, etiology, pathogenesis, mechanism of developmental disease.	4
2.	Clinical variants of ulcerative colitis, clinical and laboratory criteria, complications and treatment.	4
3.	Etiology, pathogenesis, classification of pancreatitis. Diagnosticheskie issledovaniya provodimye pri rake podjeludochnoy jelezы.	4
4.	Diagnostic criteria and complication of nonspecific ulcerative colitis.	4
5.	Etiopathogenesis, clinical manifestations, syndromic diagnosis of diseases with hepatomegaly.	4
6.	Etiopathogenesis, clinic and diagnosis of cirrhosis of the liver.	4
7.	Etiopathogenesis, clinical manifestations, syndromic diagnosis of disease with gallstones	4
8.	Etiology, pathogenesis, clinical picture, diagnostics and criteria of the activity of the disease with immune-inflammatory and inflammatory processes in the kidney.	4
9.	Etiology, pathogenesis and methods of determining the treatment of nephropathy.	4

10.	Etiology, pathogenesis, classification, clinical syndromes, diagnosis and treatment of chronic renal failure.	4
11.	Etiology, pathogenesis, clinical manifestations and diagnosis of edema syndrome.	4
12.	Diagnosticheskie criteria rheumatism and rheumatoid arthritis.	4
13.	Diagnosticheskie kriterii seronegativnyx spondyloarthritis (reactive arthritis, Bextereva's disease, psoriatic arthritis).	4
14.	Diagnosticheskie kriterii, stepen aktivnosti i lechenie sistemnyx zabolevaniy soedinitelnyx tkaney.	4
15.	Diagnostic criteria, degree of activity, treatment, prevention of dermatomyositis and systemic vasculitis.	4
16.	Diagnostic criteria, degree of activity, treatment, prevention of nodular periarteritis and nonspecific aortoarteritis.	5
	All	65

Names of independent work

№	**N a Theme**	**Hours**
1.	Causes of dysphagia, risk factors, etiology, pathogenesis, mechanism of pain development.	4
2.	Clinical options for peptic ulcer disease, clinical and laboratory criteria, complication and treatment.	4
3.	Etiology, pathogenesis, classification of pancreatitis. Diagnostic tests for pancreatic cancer.	4
4.	Diagnostic criteria and complication of ulcerative colitis.	4
5.	Etiopathogenesis, clinical types, syndromic diagnosis of diseases with hepatomegaly.	4
6.	Etiopathogenesis, clinic and diagnosis of cirrhosis.	4
7.	Etiopathogenesis, clinical types, syndromic diagnosis of diseases with jaundice	4
8.	Etiology, pathogenesis, clinical picture, diagnosis and criteria for the activity of diseases with immune-inflammatory and inflammatory processes of the kidneys.	4
9.	Etiology, pathogenesis and methods for determining the disease of nephropathy.	4
10	Etiology, pathogenesis, classification, clinical syndromes, diagnosis and treatment of chronic renal failure.	4
11.	Etiology, pathogenesis, clinical presentation and diagnosis of edematous syndrome.	4
12.	Diagnostic criteria for rheumatism and rheumatoid arthritis.	4
13.	Diagnostic criteria for seronegative spondylitis (reactive arthritis, ankylosing spondylitis, psoriatic arthritis).	4
14.	Diagnostic criteria, degree of activity and treatment of systemic diseases of connective tissues.	4
15.	Diagnostic criteria, degree of activity, treatment, prevention of dermatomyositis and systemic vasculitis.	4

16.	Diagnostic criteria, degree of activity, treatment, prevention of periarteritis nodosa and nonspecific aortoarteritis.	5
	Total	65

During the transition to clinical practice in the field of propaedeutics of internal medicine, students practice
they are expected to master the skills.

List of practical skills:
1. Palpation of the chest
2. Specific percussion of the lungs
3. Topographic percussion of the lungs
4. Pulmonary auscultation
5. Palpation of the heart
6. Cardiac percussion (determination of the relative limit)
7. Cardiac percussion (determination of the absolute limit)
8. Cardiac auscultation
9. Measuring blood pressure
10. Checking money
11. ECG recording technique
12. Normal ECG analysis
13. Superficial palpation of the abdomen
14. Deep palpation of the abdomen
15. Palpation of the liver
16. Liver percussion according to Kurlov
17. Palpation of the kidneys
18. Palpation of lymph nodes
19. Palpation and percussion of the spleen
20. Analysis of analyzes
21. Measure the neck
22. Measurement of body weight

List of practical skills:
1. Palpation of the breast
2.Sravnitelnaya percussion legkix
3.Topograficheskaya percussion legkix
4.Auscultation legkix
5. Palpation is cold
6.Percussion of the heart (determine the relative granite)
7. Percussion heart (determine the absolute granite) 8. Cardiac auscultation
9. Measure arterial pressure
10. determine the pulse
11. The technique of performing ECG
12. Decoding of normal ECG
13. Poverxnostnaya palpation of the abdomen
14. Deep palpation of the abdomen
15. Palpation biscuits
16. Percussion cookies by Kurlov
17. Palpation of the kidneys
18. Palpation lymphatic node
19. Palpation and percussion selezenki
20. Interpretation analysis
21. Izmerenie rosta

22. Izmerenie vesa

List of practical skills:

1. Palpation of the chest
2. Comparative lung percussion
3. Topographic lung percussion
4. Long auscultation
5. Palpation of the heart
6. Percussion of the heart (determine the relative border)
7. Heart percussion (determine the absolute boundary). 8. Cardiac auscultation
9. Measure blood pressure
10. determine the pulse
11. ECG technique
12. Decoding of a normal ECG
13. Superficial palpation of the abdomen
14. Deep palpation of the abdomen
15. Palpation of the liver
16. Kurlov liver percussion
17. Palpation of the kidney
18. Palpation of the lymph node
19. Palpation and percussion of the spleen
20. Interpretation of analyzes
21. Measurement of growth
22. Weight Measurement

Guidelines and recommendations for the organization of educational clinical practice

Clinical practice of students on the subject "Internal Medicine" (practical part of the course) is 50% of the daily educational process, and practical training is provided in internal medicine, cardiology, pulmonology, gastroenterology, hepatobiliary departments and counseling clinic. , held in family clinics.

In the practical training, the process of teaching practical skills is planned in detail and includes several stages:

1. The first stage - based on the goals and objectives of the training, the motivational basis for the study of practical skills is determined, its theoretical aspects are discussed. If it is required to use the necessary equipment to implement practical skills (peak flow meter, electrocardiograph), the student is acquainted with the mechanism of their operation, the rules of use.

To carry out the first stage, the department has all the tools and must be in working condition.

2. The second stage is to demonstrate practical skills and practice many times. The step-by-step algorithm of practical skills for the implementation of this stage is demonstrated by the teacher and through videos, with special emphasis on the correct implementation of the step-by-step algorithm. The student learns the practical skill independently, but under the supervision of the educator, the students practice each other many times. Initially, it is allowed to apply to the patient after complete and correct completion of all stages separately, then generalized (simulation training). In order to implement the second stage, the practical skills developed by the department should be developed step-by-step algorithm and video, teaching aids, implementation scheme or technique, etc., evaluation criteria.

To conduct the practical skill, the equipment must be equipped and the necessary conditions (modeled as close as possible to the working conditions) must be created. At this stage, the educator monitors and corrects errors in student work as needed. In this process, student actions can be videotaped, demonstrated, and critically discussed. The student explains to the teacher and other students what his or her mistake is and then

repeats the procedure. Interactivity is manifested in the fact that other students participate as experts and in assessing the correct mastery of practical skills by the student being taught. It is desirable to bring practical skills to the level of automatism.
3. The third stage is the application of the learned knowledge and practical skills to the patient. At this stage, the student is taught under pedagogical supervision to apply the acquired knowledge and practical skills in various clinical situations (including emergencies), to analyze the results obtained and to determine tactics of action on the basis of this information.
To implement the third stage, a set of teaching, methodological manuals, photographs, situational tables and tests developed by the department, cases, diagnostic and treatment standards, medical histories and outpatient cards, etc. should be used. Interactivity is manifested in the fact that other students act only as experts
4. The fourth stage is conclusion. At this stage, the educator must make sure that the knowledge and skills acquired by the student can be applied correctly and fully in patients, in different situations, in the process of activity, and then the practical skill is considered mastered.
For the implementation of the fourth stage, the student's independent work with the patient is supervised by the educator, evaluated when writing and defending medical documents, clinical audit.
At the end of the lesson, the teacher confirms that each student has mastered the practical skill. In cases where the student is unable to master the practical skill, it is recommended to master it independently outside of class time and re-assign it to the teacher. The student is considered to have mastered the science while having mastered all the practical skills.

Basic and additional educational literature and information sources

Basic literature

1. GadaevA; Karimov M.Sh ..; "Propaedeutics of internal diseases" T. 2012;
2. Muxin NA, Moiseev VI "Propedevtika vnutrennix bolezney", M. 2000.
3. Vasilenko VX. Grebenev AL “Propedevtika vnutrennix bolezney”, M.1989

Additional literature

1. Grebenev AL "Propedevtika vnutrennix bolezney", M. 2001.
2. Struto`nskiy AV “Fundamentals of semiotics of diseased internal organs” M. 2004. MEDpress-inform.
3. Karabaeva RA Practicum on propaedeutics of internal diseases, 1992.
4. T e rapiya p e r. s angl. g` pod r e d. Chuchalina M. 1997.
5. G e t-lethal M. e rap e vtich e Eugeny spravochnik Vashingtonskogo University e rsit e p e r. s angl. -1996.
6. Textbook. Harrisons principles of internal medicine. Fauci A. Braunwald E ed. McGraw-Hill, 1998.
7. Textbook of internal medicine. William N. Kelley ed. Lippincott - Ravenpublishers, 1997.
8. P e r e vod s angliyskogo pod r e daktsi e y akad. VTIvashkina fpr "Payment e TA Banks e bol email Forgot PO Davidson's" G e -lethal M., 2009
9. Narziyev Sh.S. Abdullayeva MA "Propaedeutics of internal diseases". Bukhara.2018;

Internet site symbols :

1. www.tma.uz
2. www . ziyonet . uz
3. www.medlincs.ru
4. www.medbook.ru .

Didactic tools

Equipment, tools, models, hardware: computer, projector.
Structure of centralized and decentralized hospital kitchen,
"Symptomatology of depression",
"Symptomatology of pleurisy",
"Complications of diabetes"
"Diseases of the endocrine system",
See the "soup of ozone and 12 finger q li ulcer"
"Arrhythmias",
"Bronchial asthma",
"Congenital heart disease",
"Rheumatoid arthritis",
"Myocardial infarction",
“Symptomatology of diabetes.
The concept of diabetic and hypoglycemic coma.
"Symptomatology of leukemia",
"Anemia",
"Cirrhosis of the liver"
“Symptomatology of rheumatism and primary rheumatic heart disease
“Examination of blood vessels.
Hypertension, hypotension. Izmerenie AG`D,
We have prepared on the following topics: "Laboratory and instrumental methods of research in the field of pathology."
Changes to the working program :
Created for the 2018-2019 academic year.
(next year there will be a similar application form but the change is here
will be
added. _____
_____
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The work program was considered at a meeting of the department
«__» _____ 202 1 year

Chair holder , tf d ., Prof. _____ Nurboyev FE

Presentations of lectures

Lectures № 1 and 2

Examination of patients with diseases of the gastrointestinal tract: interrogation, examination, palpation. The concept of laboratory-instrumental methods in the gastrointestinal tract. The main syndromes in gastrointestinal diseases.

Symptomatology of acute and chronic gastritis .

1. Training module of lecture technology .

Training time - 2 hours	Number of students : 2 0 to 80
Form of training	Lecture information lesson
Lecture plan	1. Examination of patients with diseases of the gastrointestinal tract: 2. inquiry, examination, palpation. 3. The concept of laboratory-instrumental methods in the gastrointestinal tract. 4. The main syndromes in gastrointestinal diseases. 5. Symptomatology of acute and chronic gastritis .
The aim of the exercises Ma Paper will contain :	To teach students to examine patients with diseases of the gastrointestinal tract .
Teaching style	Lecture interview
Form of teaching	Large, grouped.
Training equipment	Textbook, lecture content, projector, computer.
Training mode	Methodically equipped auditorium.
Monitoring and evaluation	Oral control: question and answer.

1.2 Technological map of lectures

See the stages of hearts and q ti.	Educator	Learners
In the preparation stage of q	1. Preparation of educational content on the topic. 2. Preparation of presentation slides for the introductory speech 3. Develop a list of references used in the study of science	
1. Introduction to the topic (15 da q i q a)	1. Subject ma q , with the figure and function 2. Subject b o ' on the questions.	They listen Students answer the questions posed
2 - the main stages of diarrhea q 65da q i q a)	1. Explains the topic, showing slides 2. K o ' demonstration placards	They listen They listen
1 final press q ich (10 da q i q a)	1. Concludes 2. Provides independent work 3. Gives homework	He listens Takes notes Takes notes

Brief description

Examination of diseases of the gastrointestinal tract. Inquiry. Review. Abdominal palpation. Complaints: location, distribution of the mechanism of pain formation, time of occurrence during the day, duration, mechanism of vomiting. Whether or not the blood is mixed at lunch or after a meal. Nausea, the mechanism of formation, belching, wheezing, their frequent occurrence, the conditions of their appearance. Appetite, lack of appetite. Disgust with food. Dry mouth, feeling of bitterness, whether swallowing is free or difficult, the presence of collaterals on the anterior and lateral walls of the abdomen, scarring. Intended surface palpation method. Deep methodical, slippery palpation method on VLObraztsov and NDStrajesko. Smooth palpation of the bowel.

Multimedia of lecture texts

Ma ' ruza №3

GERB symptomatology .

Oh shqozon and 12 duodenal ulcer disease etiology . symptomatology of pathogenesis.

1. Training module of lecture technology .

Training time - 2 hours	Number of students : 2 0 to 80
Form of training	Lecture information lesson
Lecture plan	1. GERB symptomatology . 2. O Etiology of gastric and duodenal ulcers. 3. symptomatology of pathogenesis.
The aim of the exercises Ma Paper will contain :	To teach students the symptomatology of GERB .
Teaching style	Lecture interview
Form of teaching	Large, grouped.
Training equipment	Textbook, lecture content, projector, computer.
Training mode	Methodically equipped auditorium.
Monitoring and evaluation	Oral control: question and answer.

1.2 Technological map of lectures .

See the stages of hearts and q ti.	Educator	Learners
In the preparation stage of q	1. Preparation of educational content on the topic. 2. Preparation of presentation slides for the introductory speech 3. Develop a list of references used in the study of	

	science	
1. Introduction to the topic (15 da q i q a)	1. Subject ma q , with the figure and function 2. Subject b o ' on the questions.	They listen Students answer the questions posed
2 - the main stages of diarrhea q 65da q i q a)	1. Explains the topic, showing slides 2. K o ' demonstration placards	They listen They listen
1 final press q ich (10 da q i q a)	1. Concludes 2. Provides independent work 3. Gives homework	He listens Takes notes Takes notes

Brief description

Etiology, pathogenesis, clinic, prevention of GERB . Etiology, pathogenesis, symptomatology of gastric and duodenal ulcers . Coprologic examination. Interpretation of diagnostic value and results obtained.

Multimedia of lecture texts

Lecture №4

Symptomatology of enteritis and colitis The importance of stool examination and diagnosis. Inquiry, examination, liver palpation, and percussion of patients with liver and biliary tract disease. According to Kurlov. Symptoms, mechanisms of occurrence, methods of detection. .

1. Training module of lecture technology .

Training time - 2 hours	Number of students : 2 0 to 80
Form of training	Lecture information lesson
Lecture plan	1. Symptomatology of enteritis and colitis 2. Garbage inspection and diagnostic value. 3. Inquiry, examination, liver palpation, and percussion of patients with liver and biliary tract disease. 4. According to Kurlov. Symptoms of the disease, mechanisms of occurrence, methods of detection.
The aim of the exercises Ma Paper will contain :	To teach students the symptoms of enteritis and colitis .
Teaching style	Lecture interview
Form of teaching	Large, grouped.
Training equipment	Textbook, lecture content, projector, computer.
Training mode	Methodically equipped auditorium.
Monitoring and evaluation	Oral control: question and answer.

1.2 Technological map of lectures

See the stages of hearts and q ti.	Educator	Learners
In the preparation	1. Preparation of educational content on the topic. 2. Preparation of presentation slides for the introductory	

stage of q	speech 3. Develop a list of references used in the study of science	
1. Introduction to the topic (15 da q i q a)	1. Subject ma q , with the figure and function 2. Subject b o ' on the questions.	They listen Students answer the questions posed
2 - the main stages of diarrhea q 65da q i q a)	1. Explains the topic, showing slides 2. K o ' demonstration placards	They listen They listen
1-final stage of q diarrhea (10 da q i q a)	1. Concludes 2. Provides independent work 3. Gives homework	He listens Takes notes Takes notes

Brief description

Main complaints: mechanism of pain formation, localization, nature, duration, irradiation, causes, intensification, attenuation, cessation of pain, diagnostic significance.

Dyspepsia: changes in appetite, nausea, belching, nausea, vomiting, abdominal distention, nausea. Jaundice: skin discoloration, urine, stool change. Diagnostic value. Itchy skin. Bleeding and hemorrhagic display format . Abdominal enlargement .

Multimedia of lecture texts

Lecture №5

Understanding the main clinical syndromes are jaundice, portal hypertension, liver failure. Symptomatology of chronic hepatitis .

1. Training module of lecture technology .

Training time - 2 hours	Number of students : 2 0 to 80
Form of training	Lecture information lesson
Lecture plan	1. The basic concept of the clinical syndromes 2. jaundice , portal hypertension , 3. liver failure . 4. Symptomatology of chronic hepatitis .
The aim of the exercises Ma Paper will contain :	To teach students the symptoms of liver failure, chronic hepatitis .
Teaching style	Lecture interview
Form of teaching	Large, grouped.

Training equipment	Textbook, lecture content, projector, computer.
Training mode	Methodically equipped auditorium.
Monitoring and evaluation	Oral control: question and answer.

1.2 Technological map of lectures

See the stages of hearts and q ti.	Educator	Learners
In the preparation stage of q	1. Preparation of educational content on the topic. 2. Preparation of presentation slides for the introductory speech 3. Develop a list of references used in the study of science	
1. Introduction to the topic (15 da q i q a)	1. Subject ma q , with the figure and function 2. Subject b o ' on the questions.	They listen Students answer the questions posed
2 - the main stages of diarrhea q 65da q i q a)	1. Explains the topic, showing slides 2. K o ' demonstration placards	They listen They listen
1 final press q ich (10 da q i q a)	1. Concludes 2. Provides independent work 3. Gives homework	He listens Takes notes Takes notes

Brief description

Complaints of chronic hepatitis, examination, palpation, tingling. An overview of the etiology and pathogenesis of diseases

Multimedia of lecture texts

Ma ' ruza №6

For patients with kidney disease sa ' Lord , is forgiveness of your question examined , the main clinical syndromes . Acute and chronic pyelonephritis, symptomatology of glomerulonephritis.

1. Training module of lecture technology .

Training time - 2 hours	Number of students : 2 0 to 80
Form of training	Lecture information lesson
Lecture plan	1. Inquiry, examination, palpation of patients with kidney disease, 2. basic clinical syndromes . 3. O tkir and chronic pyelonephritis , 4. symptomatology of glomerulonephritis .
The aim of the exercises Ma Paper will contain :	To teach students to examine patients with kidney disease, to examine, to teach palpation .
Teaching style	Lecture interview
Form of teaching	Large, grouped.
Training equipment	Textbook, lecture content, projector, computer.
Training mode	Methodically equipped auditorium.
Monitoring and evaluation	Oral control: question and answer.

1.2 Technological map of lectures

See the stages of hearts and q ti.	Educator	Learners
In the preparation stage of q	1. Preparation of educational content on the topic. 2. Preparation of presentation slides for the introductory speech 3. Develop a list of references used in the study of science	
1. Introduction to the topic (15 da q i q a)	1. Subject ma q , with the figure and function 2. Subject b o ' on the questions.	They listen Students answer the questions posed
2 - the main stages of diarrhea q 65da q i q a)	1. Explains the topic, showing slides 2. K o ' demonstration placards	They listen They listen
1 final press q ich (10 da q i q a)	1. Concludes 2. Provides independent work 3. Gives homework	He listens Takes notes Takes notes

Brief description

General concept of complaints and their pathogenesis. Pain, their analysis. Location, distribution of tumors. Dysuric disorders . Methods of laboratory examination of urine. Functional kidney tests (Zimnisky, Nicheporenko, Reberga). Interpretation of the results.

Multimedia of lecture texts

Lecture №7

Methods of interrogation and examination of patients with blood diseases. Classification of anemias. Symptomatology of iron deficiency, posthemorrhagic, hemolytic anemia. Symptomatology of leukemia.

1. Training module of lecture technology .

Training time - 2 hours	Number of students : 2 0 to 80
Form of training	Lecture information lesson
Lecture plan	1. Methods of interrogation and examination of patients with blood diseases. 2. Classification of anemias . 3. Symptomatology of iron deficiency, posthemorrhagic, hemolytic anemia. 4. Symptomatology of leukemia .
The aim of the exercises Ma Paper will contain :	To teach students the methods of interrogation and examination of patients with blood diseases .
Teaching style	Lecture interview
Form of teaching	Large, grouped.
Training equipment	Textbook, lecture content, projector, computer.
Training mode	Methodically equipped auditorium.
Monitoring and evaluation	Oral control: question and answer.

1.2 Technological map of lectures

See the	Educator	Learners

stages of hearts and q ti.		
In the preparation stage of q	1. Preparation of educational content on the topic. 2. Preparation of presentation slides for the introductory speech 3. Develop a list of references used in the study of science	
1. Introduction to the topic (15 da q i q a)	1. Subject ma q , with the figure and function 2. Subject b o ' on the questions.	They listen Students answer the questions posed
2 - the main stages of diarrhea q 65da q i q a)	1. Explains the topic, showing slides 2. K o ' demonstration placards	They listen They listen
1 final press q ich (10 da q i q a)	1. Concludes 2. Provides independent work 3. Gives homework	He listens Takes notes Takes notes

Brief description

Blood test. Clinical significance of general blood analysis and interpretation of the results. Anemia. General concepts of etiology and pathogenesis. General concepts of etiology and pathogenesis of leukemia. Classification of leukemia. Acute and chronic (myelogenous and lymphalic leukemia) symptomatology of leukemias. Bleeding: from the nose, gums, gastrointestinal tract, uterus and other organs. Palpation of lymph nodes. Palpation of
the liver , spleen , konsistensiyasi , surface and edge , OG riqlar

Multimedia of lecture texts

Lecture №9

Riga, LV patients with diseases of the glands of internal secretion sa question the Lord . Diseases of the thyroid gland . Symptomatology
of hypo and hyperthermia . Epiopatogenezi diabetes , simptomatologiyasi . Di abetic coma and gipogligemik to ' D ' on the concept . Emergency basis .

1. Training module of lecture technology .

Training time - 2 hours	Number of students : 2 0 to 80
Form of training	Lecture information lesson

Lecture plan	1. Riga, LV patients with diseases of the glands of internal secretion sa question the Lord . 2. Diseases of the thyroid gland. 3. Symptomatology of hypo and hyperthermia. 4. Epiopathogenesis, symptomatology of diabetes. 5. The concept of diabetic and hypoglycemic comas. 6. Basics of ambulance.
The aim of the exercises Ma Paper will contain :	To teach students to interrogate patients with diseases of the endocrine glands .
Teaching style	Lecture interview
Form of teaching	Large, grouped.
Training equipment	Textbook, lecture content, projector, computer.
Training mode	Methodically equipped auditorium.
Monitoring and evaluation	Oral control: question and answer.

1.2 Technological map of lectures

See the stages of hearts and q ti.	Educator	Learners
In the preparation stage of q	1. Preparation of educational content on the topic. 2. Preparation of presentation slides for the introductory speech 3. Develop a list of references used in the study of science	
1. Introduction to the topic (15 da q i q a)	1. Subject ma q , with the figure and function 2. Subject b o ' on the questions.	They listen Students answer the questions posed
2 - the main stages of diarrhea q 65da q i q a)	1. Explains the topic, showing slides 2. K o ' demonstration placards	They listen They listen

1 final press q ich (10 da q i q a)	1. Concludes 2. Provides independent work 3. Gives homework	He listens Takes notes Takes notes

Brief description

Diseases of the thyroid gland; complaints, anamnesis collection, hereditary factors. weakness, weight loss, obesity, thirst, appetite, fever, high irritability, itchy skin, palpitations, discoloration of the skin. General condition, consciousness, height and its proportions o Changes. Sex and age. Skin rashes, stretch marks. Development and even distribution of subcutaneous fat. Tumors, weakness. Changes in the skeletal system. Change in the field of 'yin.

Multimedia of lecture texts

Lecture № 1 0

Anemia . Iron, vitamin B12 deficiency and hemolytic anemia.
Allergic diseases, etiopathogenesis. Major allergic reactions: ischemia, Quincke's tumor, symptomatology of anaphylactic shock, and first aid.

1. Training module of lecture technology .

Training time - 2 hours	Number of students : 2 0 to 6 0
Form of training	Lecture information lesson
Lecture plan	1. Examination of patients with iron, vitamin B12 deficiency and hemolytic anemia . 2. Inquiry. Symptomatology of iron, vitamin B12 deficiency and hemolytic anemia 3. Methods of screening patients with iron, vitamin B12 deficiency and hemolytic anemia . 4. Methods of instrumental examination of patients with iron, vitamin B12 deficiency and hemolytic anemia .

The aim of the exercises Ma Paper will contain :	To teach students the examination, questioning, allergic diseases, etiopathogenesis of patients with iron, vitamin B12 deficiency and hemolytic anemia .
Teaching style	Lecture interview
Form of teaching	Large, grouped.
Training equipment	Textbook, lecture content, projector, computer.
Training mode	Methodically equipped auditorium.
Monitoring and evaluation	Oral control: question and answer.

1.2 Technological map of lectures

See the stages of hearts and q ti.	Educator	Learners
In the preparation stage of q	1. Preparation of educational content on the topic. 2. Preparation of presentation slides for the introductory speech 3. Develop a list of references used in the study of science	
1. Introduction to the topic (15 da q i q a)	1. Subject ma q , with the figure and function 2. Subject b o ' on the questions.	They listen Students answer the questions posed
2 - the main stages of diarrhea q 65da q i q a)	1. Explains the topic, showing slides 2. K o ' demonstration placards	They listen They listen
1-final stage of q diarrhea (10 da q i q a)	1. Concludes 2. Provides independent work 3. Gives homework	He listens Takes notes Takes notes

Multimedia of lecture texts

Handouts. Tests.

Digestive system related tests

1. What is the cause of vomiting in esophageal disease ? 1). Narrowing of the esophagus 2). The vomit mass consisted of chewed oats; 3). Nausea, narrowing of the esophagus; 4). Pain, nausea, narrowing of the esophagus; 5). Antiperistaltic contraction of esophageal muscles

1) .- 1 -4; 2) .- 1-5; 3) .- 2-3; 4) .- 2-4;

2. Mass of vomit from the esophagus? 1). C contains boiled food and hydrochloric acid; 2). Consists of undigested chewed food; 3). It comes without nausea and does not contain pepsin; 4). Undigested, chewed food and contains pepsin; 5). It consists of pepsin, hydrochloric acid, and digestible food.

1) .- 1-3; 2) .- 1-2; 3) .- 2-3; 4) .- 2-4;

3. What is the cause of bleeding from the esophagus? 1). Esophageal tumors and esophageal burns; 2) .After the rupture of the esophageal tumor; 3). Narrowing of the esophagus and esophageal ulcer; 4). From esophageal ulcer to rupture of dilated esophageal veins; 5) .In dilation of esophageal veins and esophageal burns

1) .- 1-2; 2) .- 1-3; 3) .- 2-3; 4) .- 2-4;

4. What is the cause of jaundice? 1). Me ' da juice is thrown into the lower part of the esophagus; 2). Reflux esophagitis; 3). In vomiting, in recording; 4). When bleeding from the esophagus, reflux esophagitis; 5). Reflux in esophagitis, vomiting, when noted.

1) .- 1-2; 2) .- 1-3; 3) .- 2-3; 4) .- 2-4;

5. Sputum mass characteristic for Privratnik stenosis; 1). Mass of odorous vomit; 2). The vomit mass consisted only of gastric juice; 3). Smells 10-15 minutes after eating; 4). The vomit mass contains fresh food and pepsin; 5). After vomiting, the patient feels relieved.

1). -2-3; 2) .- 1-3; 3). -1-4; 4) .- 2-4;

6. Dysphagia is… 1). Oral secretion of gastric juice; 2). Occurs after esophageal burns; 3). Disorders of the passage of food through the esophagus; 4). Return of food eaten; 5). Pain during feeding in the chest area;

1) .- 1-2; 2) .- 1-3; 3) .- 2-3; 4) .- 2-4;

7. Determine the cause of bad breath? 1). Esophageal cancer; 2). Difficulty passing food as a result of breast cancer; 3). Difficulty passing food from esophageal burns; 4). Food stagnation in cardiac achalasia; 5). Difficulty passing food as a result of narrowing of the esophagus;

1) .- 1-5; 2) .- 1-3; 3) .- 2-3; 4) .- 1-4;

8. When examining a patient with cirrhosis? 1). The liver is enlarged, the skin is yellow. 2). Hood jellyfish, vascular asterisks. 3). Drum stick finger. 4). Enlargement of the abdomen, (ascites). 5). Liver pulsation, vascular pulsation.

1). -1-3; 2). -2-3-4; 3) .- 2-3; 4) .- 2-4;

9. What are the specific laboratory changes in hemolytic jaundice? 1). An increase in the amount of incorrect bilirubin in the blood. 2). Bilirubin is not present in urine. 3). Binding bilirubin in the blood increases. 4). Bilirubin appears in the urine. 5). Garbage turns white.

1) .- 3-4; 2) .- 1-2; 3) .- 4-5; 4) .- 2-3;

10. Hemolytic jaundice occurs? 1). As a result of the effects of viruses on the liver. 2). From an increase in bile acids in the blood. 3). As a result of the breakdown of erythrocytes in the spleen, liver and bone marrow. 4). Increased bilirubin glucuronide in the blood. 5). Improper (unbound) bilirubin increase in the blood.

1) .- 3-5; 2) .- 1-2; 3) .- 2-3; 4) .- 4-5;

Tests of the urinary system

1. Addis-Kokowski is determined by test?

a) Relative density of urine c) Proteins

b) Shape elements d) Salts

2.Addison disease b e h disease?

a) pancreas disease c) adrenal b e zi disease

b) thyroid b e h d) The pituitary disease b e zi disease

3. Symptoms of Addison's disease

a) Changes in pigmentation c) Intoxication

b) Hemorrhage d) Oliguria

4. Addisson's disease is caused by chronic organ failure?

a) Divorce c) Liver

b) Heart d) Kidney

5. Angie e nzin 2 - in which the collapse of the f e rm e nt b occur under the influence of e worldwide.

a) Angiot e nzinaza c) Ad e niltsiklaza

b) Amminop e ptidase d) Kallikr e inaza

6. One day m e contains the amount of urine

a) 1000-2000ml c) 200-500ml

b) 500-800ml d) 500-1000ml

7. Kidney GIP e rt e nziyasida more ... pressure:

a) Pulse pressure c) Diastolic

b) Systolic and diastolic d) Systolic only

8. Patient color in kidney disease

a) Pale c) Brown

b) Hyperemic d) Cyanosis bruising

9. Where do tumors most often start in kidney disease?

a) In the heart c) In the spleen

b) On the face d) On the feet

10. Kidney disease diagnosis equipment with T E does not bind kshim

a) UZI c) Kidney scan

b) Kidney biopsy d) Laparoscopy

11. Kidney disease b e morlarning main complaints included:

a) Disorders of urinary excretion c) The onset of swelling in the foot

b) Pain in B e l area d) Swelling in faces

12. kidney tumors to try to e rli

a) His face is swollen, pale and emaciated

b) T e z-developing and spreading to all parts of the body

c) Blue tumors

d) Top-down spread of tumors

13. The symptoms of chronic kidney disease, which try to e rli?

a) Pain in the B e l area

b) Art e rial gip e rt e nziya

c) Art e rial gipot e nziya

d) Art e rial gip e rt e nziya, pain in the b e l area, tumors

14. Grom e rolls e frit for action, e rli.

a) Proteinuria c) Hypertension

b) Pyuria d) Hyperstenuria

15. Glom e roll e frit cannot cause development

a) High insolation c) Viruses

b) cold d) Inf e ktsiyalar

16. Glucosuria bu-

a) Separation of k e ton bodies with urine
b) Protein excretion in urine
c) Separation of bilirubin in urine
d) Separation of sugar from urine

17. What is polyuria?

a) Excessive excretion of urine c) Presence of protein in urine
b) Decreased urination d) Blood in the urine

18. Polyuria cannot be the cause

a) How Diab e t c), hypo- e nziv pr e Paraty
b) How Diab e t d), 2 and 3

19. Pollakiuria is:

a) K e chqurungi urine e zning at day rule
b) T e z-t e z separation of urine
c) Urinary incontinence
d) Increased daily urine output

20. Pollackuria is specific to which disease.

a) Kandli diab e t c) Gipotir e oz
b) Tyr e otoxicosis d) Addison's disease

Endocrine system test questions

1. The amount of sugar in the blood of a healthy person.

a) 14-16 mmol / l **c)** 3, 5 - 6 , 5 mmol / l
b) 9-12 mmol / l **d)** 12-14 mmol / l

2. Characteristic of Shtelvag's symptom (rare blinking of the eye):

a) Akram e Galina for **c)** The prime e rkulyoz
b) For baz e dov disease **d) For** cancer

3. Facial expression as a result of hypothyroidism?

a) Lion face **c)** Mexidematous face
b) Parkinson's face **d)** Moon face

4. What hormone does the thyroid gland produce?

a) Thyroxine, triiodothyronine **c)** Estradiol
b) Progesterone **d)** Testosterone

5. What is used as first aid in diabetic coma.

a) 40% - 20.0 glucose v e na into **c**) Insulinot e rapiya
b) antiarrhythmic pr e Paraty **d)** cardiac glycosides

6. How Diab e tried concurrently for the first stages of e rli:

a) Polyuria **c)** Isosthenuria
b) Hypoizost e nuriya **d)** Anuria

7. How Diab e t s e cr e police b e zi disease.

a) Adrenal **c** e zi **c)** Thyroid b e zi
The pituitary b e **b)** some **d)** stomach b e zi

8. How Diab e sionmaking basic laboratory character counts

a) Glucosuria **c)** Anor e xia
b) Polyuria **d)** Polydipsia

9. Polydipsia is a specific symptom for which disease.

a) hypothyroidism **c)** diabetes Diab e t
b) Hyperthyroidism **d)** Its e nko-Kushing syndrome

10. What does the face look like in a mix e d e mada?

a) The eyes are sunken, the nose is sharp, t e ri is blue

b) the face, lips, nose, eyebrows arch over a large range of dental care e ngayib, the lower jaw forward

c) face k e ng, round, pale, t e Rising smooth, thickened, and his eyes, pale and smiling,

d) A large open, flushed, shiny, eye, face frozen in fear.

11. Ksantelazma evidence of a breach of the following b e worldwide:

a) Water-salt metabolism **c)** Protein metabolism

b) Hol e st e rin metabolism **d)** Enzyme metabolism

12. Xant e places are located:

a) On the lids **c) On the** hand

b) On the body **d) On the** neck

13. GIP e rtir e bit to try to e rli

a) Increased metabolism

b) GIP e rxolest e rin e brain and bradycardia

c) Tachycardia, cholesterol m e will not help '

d) Gipoxolest e rinemiya and tachycardia

14. thyroid b e h GIP e rfunktsiyasi for action, e rli.

a) Exophthalmos, bradycardia **c)** Exophthalmos

b) Exophthalmos, t e z-t e z eye closure **d)** Exophthalmos, tachycardia

15. Gip e rglik e miya-bu.

a) Increased blood sugar

c) Increased bilirubin in the blood

b) Decreased blood sugar

d) Decreased lipids in the blood

16. Angulyar stomatitis, gingivitis, tooth elderly e have paradontoz.

a) diabetes Diab e sionmaking **c)** Gipotir e paper

b) Addison's disease **d)** Akram e Galiyev

17. He looks ats e ton which spread the smell of coma for action, e rli

a) Apopl e xic coma **c)** Ur e mik coma

b) Diab e coma **d)** Gipoglik e Mick coma

T est questions about the blood system

1. Arrange the following in order; In hereditary hemolytic anemia, indicate the causes of 1. membranopathy and 2. hemoglobinopathy; A) accumulation of sodium in the cell; **B)** a defect in the amino acid chain a ; **C)** intracellular decrease in potassium; **D) a** defect in the b amino acid chain; **E)** accumulation of water inside the cell.

A) 1-ACE and 2-BD

B) 1-A- E -D and 2-BC-

C) 1-ABD and 2-CE

D) 1-ABE and 2-CD

2. What disease is characterized by enlarged, painless, mobile and incoherent lymph nodes?

A) For asthma

B) For leukemia

C) For lymphagranulomatous disease

D) For hematosarcomas

3. Identify the causes of Minkowski-Shoffar spherocytic anemia; A) intracellular accumulation of sodium; **B)** extracellular release of sodium; **C)** intracellular decrease in potassium; **D)** accumulation of potassium in the cell; **E)** accumulation of water inside the cell. **J)** depletion of intracellular water;

A) ACE

B) BCE

C) ACD

D) DE- J

4. Separate the lymph nodes specific to leukemia?

A) Enlarged, painful, red, and incoherent
B) Enlarged, painless, mobile, incoherent
C) Enlarged, painless, mobile, and interconnected
D) Red, painful, purulent, enlarged lymph node

5. Show the typical symptoms of vitamin B12 deficiency and folic acid deficiency anemia. A) Decreased pay reflexes; **B)** Bone marrow hyperplasia; **C)** Color index hyperchromia; **D)** Rossolimo pathological reflexes appear; **E)** Terisi rangpar; **J)** Atrophic Gunter glossitis; **Z)** Raspberry colored tongue; **I)** Atrophy of the subcutaneous fat;

A) ACDJZ
B) BCDIZ
C) ACJZ -B
D) EDJZB

6. Identify the causes of hereditary hemolytic anemia type of enzymopathy; A) G glucose-6-phosphate dehydrogenase deficiency; **B)** Decreased oxidation in erythrocytes; **C)** P iruvatkinase enzyme deficiency; **D)** G lutation peroxidase enzyme y deficiency ; **E)** Increased returned glutathione; **J)** Decreased oxidized glutathione; **Z)** slowing down the return of NADF to NADFH ; **I)** O ksidlangan glyutationdan returned (restored) glutationning sure to be broken.

A) ACDZI
B) ABCEJ
C) ABDZI
D) ECDJI

7. Indicate the radiological changes that lead to thalassemia in hereditary hemolytic anemia; A) The fragility of the bones; **B)** S uyaklarla operates osteoporosis ; **C)** Short tubular bones; **D)** Thinning of N-shaped bones ; **E)** Deformation of flat bones; **J)** B soup bones cho`tkasimon (hedgehog ignachalari) changes ;

A) ACD
B) ACE
C) ABJ
D) BDJ

8. Identify the etiological factors of hypoplastic and aplastic anemia; A) Blood loss; **B)** Decomposition of blood; **C)** Radiation radiation; **D)** antibodies against bone marrow ; **E)** Iron and vit B12 deficiency; **J)** Influence of benzene, trinitrotoluene, tetraethyl lead on bone marrow ;

A) ACD
B) CDJ
C) ABJ
D) ACE

9. Arrange the following in order; In iron deficiency anemia 1. sideropenic syndromes and 2. symptoms characteristic of cologne. A) Dysphagia; **B)** Hair breakage ; **C)** thinned nail; **D)** Dry skin; **E) The** tongue is smooth, shiny; **J)** Nails are brittle; **I)** Spoon nails; **Z)** Flattened nails;

A) 1-A- E -JZ and 2-BCDI
B) 1-ACDE and 2-BJIZ
C) 1- AB- D- E and 2- CJIZ
D) 1- ACJ- I and 2- AEDJ

10 . Separate the lymph nodes specific to lymphagranulomatosis?

A) Enlarged, painless, mobile, and unconnected
B) Enlarged, painful, red, and incoherent
C) Enlarged, painless, mobile, and interconnected
D) Red, painful, purulent, enlarged lymph node

11, place the following in order; 1. Distinguish between symptoms characteristic of chronic myelogenous leukemia and 2. characteristic of chronic lymphocytic leukemia. A) The origin of myelopoiesis cells; **B)** Cardiac examination reveals Philadelphia chromosomes; **C)** Origin of lymphopoiesis cells; **D)** Botkin-Gumprecht bodies are found **; E)** Mature lymphocytes multiply in the blood; **J)** Promyelocyte, metamyelocyte in the blood **;**

A) 1-ACD and 2-BJE
B) 1-A-EJ and 2-BCD
C) 1-CJE and 2-ABD
D) 1-ABJ and 2-CDE

12. Arrange the following in order; 1. Characteristic for lymphadenitis and lymphagranulomatosis; A) Enlarged painless lymph node; **B)** Enlarged painful lymph node **C)** Enlarged painful lymph node, redness around; **D)** Enlarged painful lymph node, redness around and moves with surrounding tissue; **E)** Enlarged painless lymph node and not attached to the surrounding tissue; **J) The** lymph node is enlarged, painless in motion, connected to each other;

A) 1-ADE and 2-ACE
B) 1- AEB and 2-DEJ
C) 1- BCD and 2- AEJ
D) 1-CDE and 2-DEJ

13. What are the symptoms of Pico chloratica in iron deficiency anemia? A) Loss of complete cognition; **B)** hair loss; **C)** Consumption of cuttings, chalk; **D)** Dry skin; **E)** Smell of kerosene and gasoline; **J)** Thinning and breaking of nails;

A) ACD
B) CDJ
C) ACE
D) ABJ

Digestive system issues

1 - masala

B e mor, 59 years old, complains of weight loss, difficulty swallowing (dysphagia), chest pain, nausea, bad breath, vomiting. The vomit mass consists of chewed food and smells. B e is sensible not to eat meat appetite ist e can not afford k e chirp kax e ktsiya t e settings identified a lack of subcutaneous fat layer developed, enlarged lymph nodes in the neck.

What pathological process can occur in B e mor ?

How instrum e ntal t e kshiruvlar probably be conducted ?

R e NTG email technology and endoscopic t e kshiruvda to see any changes ?

2 issues

B e defeated the 35-year-old, male, n e cha dispans since e is continuing under the supervision k e sick and want to d e b calculation. B e snout permanent kabziyat, sometimes - sometimes there is pain in the lower part of the abdomen b e zovta. B e defeated the faeces of k e camel feces obstructed light - round and round like the surface of the mucosa have been identified. B e morning blood t e kshirilganida a n e ytrofil l e ykotsitoz drowning in red blood cells and T e zligi t e zlashgan (14 mm / s).

How do you diagnose B e m o r?

What are the palpator changes in the b e mor?

How instrum e ntal t e probably took kshiruvlar d e b can think of, and any changes?

3 - masala

B e defeated the 31-year-old, he has been ill d e b know and disease, mainly in spring and autumn b e zovta. Pass the complaint sometimes inside, sometimes with constipation, fatigue, pain in the side of the lower part of the abdomen, hips b e zovta. The

pains intensify after eating and before d e f e cation. Blood t e kshirilganida a n e ytrofil l e ykotsitoz and Echt t e zlashgan. B e defeated in Najaf t e kshim seen covered with feces hard and the surface of the mucosa.

How do you diagnose this b e morda?

Tell palpator changes?

How instrum e ntal t e kshiruvlar into the route and any changes?

B e Morley said changes in fecal b e ring?

4 masala

B e defeated the 22-year-old male driver due to the use of nutrition r e jimi always be broken, and sometimes epigastric pain doctor noted. B e mor said the disease mainly attacks in spring and autumn. Weather ' e ktiv acoustical presence of epigastric pain and p e rkussiyada epigastrida m e pro e ktsiyasida M e nd e l symptom positive. Gastrofibroskopiyada m e exceed 0.5 X 0.6 smli curve d e struktiv changes and redness around it.

Put an approximate diagnosis of this b e morga?

B e morning listed the main complaints b e ring?

Palpator b e ring to tell changes ?

How instrum e ntal t e kshiruvlar need to be conducted and the change in you?

5 masala

The patient, 33 years old, said when he came to the doctor's office that he had been ill for many years and that there was pain in the left side of the abdomen, which was transmitted to the left chest and lumbar spine. Appearing after 30 minutes, he said it was more annoying in the spring, along with wheezing, wheezing, nausea, sometimes vomiting, and he felt relief after a little vomiting. Percussion revealed a positive Mendel symptom in the epigastric area. X-ray examination revealed a shelf (niche) symptom.

How do you diagnose a patient ?

List the etiological factors for this disease ?

It is the disease complications o Framework counted ?

Diet e z desktop and treatment r e war ?.

Issues related to the urinary system

1 - masala

B e defeated the 19-year-old, female gender, rural medical point of visiting a doctor k e a decrease in the ability to configure headache, dyspnea, and heart pain to complain about the presence of the field k e i and Anamur e z gathered himself He has been ill for 2 weeks and has been diagnosed with redness of the p e shobe, oliguria, and angina. B e Morning k e lamp t e settings covering catching color, the presence of edema of the eyelids visible.

Make a tentative diagnosis of B e morgue.

Palpator, p e rkutor, say auscultatory changes.

What changes can be expected in the B e mor p e branch.

How instrum e ntal t e kshiruvlar on the route.

2 - masala

B e mor is 22 years old, a female. Section k e to configure inquiry and complaints found 15 days ago skarlatina diseases, deterioration of eyesight, heart and b e l in the presence of pain b e zovta. B e defeated k e chirp, the presence of edema of the eyelids, laboratory detected the presence of red blood cells in the urine.

B e morning tell the approximate diagnosis.

B e Morley k e lamp.

What changes can occur in B e mor p e branch.

3 - masala.

Rural health units k e b want to e defeated, b e l in the two nagging pain, p e Shobha t e h t e h k e to be made, and a small amount of N e to go, headache and the ability to

complain about the decline in k e i. He considers himself b e mor d e b for 3 weeks and said that k e yin started from otitis . Laboratory t e kshiruvlar writ of p e Shobha the presence of blood cells and proteins.

Make a tentative diagnosis of B e morgue.

State the p e rcutor changes in B e mor .

B e Morley r e NTG e technology t e kshiruv t e machinery b e ring.

B e defeated p e Shobha macroscopic and microscopic changes to b e ring.

4 –masala.

B e mor 20 years old, female, admitted to the hospital, patient for 3 weeks, after all t e examinations by the reception physician , p e shob was found to be low in protein, erythrocytes, hyaline cylinders, and urine volume (oliguria). The macroscopic appearance of the urine is similar to that of meat washed water.

Determine the approximate diagnosis of B e mor.

K e see the light of any changes.

Describe the changes in the ECG.

How instrum e ntal t e kshiruvlar executed and t e machinery.

5 - masala

B e defeated, female kasalxanaga k e configure the medical examination of a n e cha are asking b e sick rin d e b of the century and following the complaint, b e l in the presence of pain, and a reduction in the amount of urine said he was red in color and had swelling on his face and legs. B e purple Anamur e accrue z k e gaining increased frequency of urination, and increased the amount of urine.

Make a tentative diagnosis of B e morgue.

B e defeated k e muted any changes.

State the p e rcutor changes in B e mor .

B e purple laboratory and instrum e ntal t e kshiruvlar, and a t e machinery.

Addis Kakovskiy method of collecting urine test t e machinery and how changes to a b e ring.

6 masala

B e mor is 16 years old, has known himself as a patient d e b for 15-20 days and associates his illness with angina. B e purple Anamur e z collection of k e mold and the eyelids and leg tumor, laboratory p e Shobha l e ykotsitlar. And instrum e ntal t e kshiruvda kidney par e nximasida inflammatory b e is defined.

B e morning tell the approximate diagnosis.

What changes will occur in the analysis of B e mor p e shobi.

How instrum e ntal t e kshiruvlar carried out, and any changes will be viable.

Problems with the endocrine system

1 masala

B e defeated Sarah investigated Gesture g'aluvchan, stinginess, crying for no reason, heart, t e z palpitations, poor sleep, t e rlash t e z rising fatigue, fever, shaking hands, and to lose jobs a decrease in ability was found. B e defeated k e chirp k e dance movements, words completed another sentence caps, t e GIP e rpigm on the e ntatsiya (symptom e llin e k). B e morning pulse t e increased 110 times min. She has lost 5kg in recent days, a major metabolism increase of 30%.

Make a tentative diagnosis of B e morgue.

B e morning the face image of the k e can see the light of any changes?

What changes do we see in the cardiovascular system?

What changes do we hear when the heart is auscultated?

2 issues

Visiting a doctor k e b want to email the defeat of the k e chirp thyroid b e some large, t e Rising shining, t e settings layer of subcutaneous fat atrophy, symptoms of eye positive, that is, to display the shining eyes, Self slot k e ngaygan, posing for the costs

(ICI e lvag symptom), qonv e rg e ntsiyasi declines (M e bius symptom), the eye moves down to the top of the pumpkin behind SKL e Here, the white line (Gr e f symptom), a similar white line appears when the eyeball moves upwards (Cox e r symptom).

Make a tentative diagnosis of B e morgue.

Name the changes that occur in B e mor blood.

Changes in the cardiovascular system, b e ring.

EKGdagi b changes in the E ring.

3 masala

B e defeated visiting a doctor k e configure intensification of the stomach, vomiting, stomach pain xurujsimon and liquid to pass through the inside of the complaint. Cardiovascular system t e kshirilganida pulse t e zlashgan, tachycardia, systolic art e rial pressure is increased and decreased diastolic pressure, pulse pressure is enlarged. Cardiac arrhythmias, such as extrasystoles and palpitations, were found. B e morning thyroid b e some large, shiny eyes, eye lid k e ngaygan, posing for the costs and b e took the defeat in the last days of the 10 kg weight century. The main exchange was found to have increased by 55%.

How do you make a tentative diagnosis of B e morga?

B e Morning k e can see the light of any changes?

What changes are heard when the heart is auscultated?

B e defeated the blood changes to b e ring.

How instrum e ntal t e k sweet conducted and how the results ?.

4 masala

B e defeated the 38-year-old woman, sex, medical appointments k e configure crying for no reason, heart, t e z palpitations, poor sleep, t e rlash t e z fatigue, fever, hand tremors, weight loss and removal of business noted that the decrease in ability b e zovta. B e Morning height of 1.67 m and weighing 49 kg, K e tl e ind e 17 ksi b e defeated very thin k e , passing the pulse t e zligi 142 times, and 68% increase in the exchange.

Make a tentative diagnosis of B e morgue.

B e Morning k e lamp.

Mention eye symptoms.

5 masala

B e defeated the 49-year-old woman, sex, medical appointments k e configure fatigue, sleep, memory Diner, impairment loss, dryness and brittle hair, eyebrows ch e ti and complain about loss of eyelashes, nails, broken and dry does. In the oral cavity, periodontitis in the teeth, damaged teeth, constipation and m e t e orism were detected. In the nervous system, par e st e zia, seizures, and inability to walk t e kis. Ignoring the environment, int e ll e kt decreased. The doctor b e purple Anamur e z collector before becoming sick with radioactive iodine n e cha treatments identified. B e morning face was masked and the eyelids narrowed.

How do you make a tentative diagnosis of B e morga?

B e Morning k e see the light of any changes ?.

What changes are observed in the cardiovascular system?

ECG changes to b e ring.

6 masala

The doctor, heir k e b want to email the defeat of the k e muted red jowls (Rumy e ts), p e glory, cheek, jaw and underground areas kapilyarlari k e ngaygan rubles (e), muscle atrophy, and less support 'feet and hands on the yellow t l e tracks are usually qashalgan, xanthomas presence, the presence of foot ulcers and b e morning flow cavity of the k e chirp gingivitis, stomatitis and teeth paradontozga broke down in tears discarded. B e purple Anamur e accrue z k e gaining a lot of p e Shobha divorce and t e kshirilganda the presence of glucose in the urine.

How do you make a tentative diagnosis of B e morga?

Changes in the cardiovascular system, b e ring.

Changes in the nervous system that b e ring.

7 masala

B e defeated the 55-year-old hospital k e configure thumb on his right leg ulcers appear, itch, and p e Shobha large amounts of divorce complaint. B e morning pulse 94 times min. 170/100 mm when measuring blood pressure. sim. ust. T e teaching at the same time the blood GIP e rglik e brain and p e Shobha t e kshirilganida the presence of protein and glucose.

Make an approximate diagnosis of B e morga.

B e morning urine separation system changes will b e ring.

Changes in the bottom of the eye b e ring.

8 masala

B e defeated visiting a doctor k e configure itching, hunger, fatigue, p e Shobha k e chqurunlari Only complaint. B e Morning k e the presence of the lamp body of the smoker monitors, blood t e kshirilganida Failures of the amount of glucose in the blood of 18 mmol / l, p e Shobha 3% of the amount of glucose detected.

Make a tentative diagnosis of B e morgue.

What changes occur in protein metabolism?

What changes are observed in the digestive system.

What changes occur in the urinary system?

Issues related to diseases of the circulatory system

1 masala

School children held periodic medical examination, the student teacher always chalk ist e 'stance, sometimes in conjunction with k e Sak ist e said, and the girl child At the age of 14, the doctor said that when the anamn e z was collected, the menstrual cycle (m e nstruation) began at the age of 10 and lasted for 7-8 days. B e mor hair is brittle, dry, the corner of the mouth is cracked, the hair falls out according to the girl. Blood t e kshirilganida color indicator of 0, 65 (gipoxrom) levels and the number of red blood cells (less than 3.2 million) levels were determined.

How do you diagnose B e morga?

Classification of the disease b e ring indicator (color and morphology, etc.).

List b e mor complaints.

B e defeated the blood changes to b e ring.

2 issues

Female 21 years, 30 weeks of pregnancy, women maslahatgohiga k e configure it claims heard by a doctor, sub e ktiv t e kshiruvda eyes, dizziness and overcast weather ' e ktiv t e settings pale nails into the sinking of was found to be thinner. B e defeated the red blood cells in the blood of 2.8 million and g e moglobin amount of less than 64 g / l (spotted) and the cardiovascular system, heart t e zligi 104 pulses / min and a soft systolic heart cashmere V noise heard in the ribs .

B e morning tell the approximate diagnosis.

List b e mor complaints.

B e Morning Weather ' e ktiv t e kshirishda see any change.

Changes in the cardiovascular system, b e ring

B e morning blood changes that can b e ring.

3 masala

The patient, 24 years old, was examined by a physician at the time of admission to the hospital and found to have the following changes. The skin and mucous membranes are white-yellow, the face is swollen, the tongue is dark red and shiny, the surface is smooth due to atrophy of the nipples (atrophic Gunterov glossitis), the oral mucosa and the back of the palate are atrophied, around the tongue ulcers appeared in the oral mucosa

and caries was found in the teeth. Blood color index (1.1) revealed the presence of hyperchromia and Jolly bodies, Kebot rings.

What approximate diagnosis do you make to the patient ?

List the etiological factors of the disease.

Describe the pathogenesis of the disease.

Describe the changes in the blood.

4 masala

The lobby and k e b want to e Morning ask the doctor questions, headache, dizziness b e zovta and weather e ktiv- t e settings and yellowish mucous rangdaligi and disk space ' i k e chirp smooth, dark red in color, with glossitis, there are instrum e ntal (CT) of the liver and spleen size and the color of blood and laboratory index of 1.2, Jolly found the cells.

Make a tentative diagnosis in B e morda.

B e defeated the complaints b e ring.

The etiology of the disease and Pato e n e self-b e ring.

B e Morning k e can see the light of any changes?

B e can see any changes in the blood of the morning?

5 masala

The patient, 48 years old, has been under dispensary supervision with rheumatoid arthritis for several years, suffering from fatigue, rapid fatigue, palpitations during physical exertion after being infected with the flu. Examination of the patient showed yellowing of the skin, eyes and other mucous membranes, darkening of the color of urine. The number of erythrocytes in the blood was found to be 3.2 million less and reticulocytes slightly higher than normal. The color of the blood did not change.

What type of anemia does this anemia belong to?

Describe the mechanism of disease development.

Describe palpator and percussion changes.

Describe the changes in the patient's blood, feces, urine.

6 masala

The patient, who came to the doctor's office, was examined and found to have cachexia, swollen abdomen, moist skin, pale skin, yellowish tinge, swelling in the legs, skin, subcutaneous hemorrhage, rash, in the oral cavity, tonsils we can see the presence of necrotic lesions, the presence of gingivitis and stomatitis, enlarged lymph nodes. There were blood clots in the skin, and when the blood was examined, it was found that the number of leukocytes increased mainly due to myelocytes, promyelocytes.

What approximate diagnosis do you make to the patient ?

List patient complaints.

Name the palpator and percussion changes.

State the changes in Q.

Name the cariological changes.

7 masala

On examination by an outpatient, the patient presented with cachexia, swollen abdomen, dry skin, pale skin, swelling in the legs, hemorrhage in the skin, subcutaneous hemorrhage, rash, necrotic lesions in the oral cavity, tonsils, we can see the presence of gingivitis and stomatitis, enlarged lymph nodes. There were blood clots in the skin, and a blood test revealed an increase in the number of leukocytes , mainly due to lymphocytes. In addition, the presence of the Botkin-Gumprecht shadow was found.

What approximate diagnosis do you make to the patient?

List patient complaints.

Name the palpator and percussion changes.

Name the changes in the blood.

8 masala

During the medical examination of the patient who came to the doctor's office, the patient reported a prolonged fever and increasing weakness, rapid fatigue and sweating, as well as pain in the bones and sometimes the left rib. He complained of discomfort and pain underneath, and enlarged lymph nodes. The doctor carefully examined the patient and recommended a blood test, and it was determined that the number of lymphocytes was excessive and 90-95% were mature cells, and that there was a Botkin-Gumprext shadow.

Make an approximate diagnosis of the patient.

What changes can we see when examining a patient objectively?

What are the changes in palpation and percussion ?

What changes do we see in the blood test?

9 masala

The patient, a 36-year-old female, came to the doctor for advice because of the constant yellowing of the skin and the color of the visible mucous membranes. Subjective weakness, dizziness and objective yellowing of the skin, laboratory tests showed a decrease in the number of erythrocytes to 3.0 million, followed by a lack of glucose-6 - phosphate dehydrogenase in the blood, darkening of the urine and hemosiderin was found to be present, slightly enlarged liver and more spleen on palpation.

Make an approximate diagnosis of the patient.

Explain what type of anemia it belongs to.

Explain the mechanism of disease development.

Classification of diseases of the blood b e ring.

Assemblies on topics
Digestive system assistants
Assessment 1

Te s t **1. Identify the radioscopic signs of wound disease? 1**). The gastric mucosa is hypertrophied.2). The gastric mucosa is atrophied.3). Erosion and bleeding in the gastric mucosa.4). Niche (shelf) symptom appears.5). The index fingerprint is displayed. 1) .- 4-5; 2) .- 1-2; 3) .- 2-3; 4) .- 3-4; **57. What is "Melena"?** a) Black garbage b) Colorless (gray) garbage c) Garbage with undigested food debris g) Oily, shiny, poorly washed garbage	**Situational issue** 1 masala The patient is 31 years old, male, and has been ill for 10 years. The patient complains of pain in the epigastric area 1.5-2 hours after eating, nausea and vomiting, and sometimes nausea and vomiting. The pain subsides after eating, often bothering in the spring and fall. What is your approximate diagnosis. What changes do we see on X-ray examination. Tell the palpator changes.
Analysis of t : Chronic enteritis is	**A financial ability:** Sigmoid colon palpation technique

Assessment 2

Test **22 . Symptoms of ulcer disease?** 1). Occurrence of pain after eating.2). The relationship of pain to food and its onset in spring and autumn.3). Occurrence of evening pain in 12-finger ulcer.4). Occurrence of pain in duodenal ulcer 20-30 minutes after eating.5). In 12-finger ulcers, the pain is exacerbated after a meal. 1). -3-4; 2) .- 1-3; 3). -2-3; 4) .- 4-5; **58. The smell of "eggs in the palate" is observed:** a) in compensated stenosis b) in gastric esophageal reflux v) in duodeno-gastro-esophageal reflux g) in decompensated stenosis	**Situational issue** 2 issues The patient is 23 years old, male, student. When he came to the diagnostic center, he said he had been ill for many years and said that every evening between 2 and 3 p.m., the pain in the epigastric area was bothersome, and that nausea and weakness were bothersome. The patient gets up at this time, eats a little, and lies down again when the pain subsides. When the doctor palpated the patient's abdominal area, he found that there was pain on the navel to the right of the midline of the abdomen. What instrumental examinations are preferred to make a definitive diagnosis in the patient. What are the radiological signs of this disease. State the signs of gastrofibroscopic.
Analysis of t : Chronic colitis is	**Practical skills:** Lateral and cecal palpation techniques

Assessment 3

Te s t **21. What are the complications of wound disease? 1**). Pain, bleeding,	**Situational issue** 3 masala When the patient came to the doctor, he

perforation.2). Weight loss, penetration, malignancy.3). C tenosis, perforation, malignancy.4). Q ongoing departure, penetration.5). E pain in the pigastral area , vomiting, gastric perforation, stenosis. 1) .- 3-4; 3) .- 2-3; 2) .- 1-2; 4) .- 4-5; **59. Food after 7-8 hours after eating " flush (Plesk) indicates noisy" epigastric fossa:** a) M undergoing changes in function b) HCl -hyperproduction c) Ev aquatic feature can be blocked g) Sh trapped (sliz) giperproduktsiyasidan	said he had been ill for 16 years, and in the evenings there was pain in the epigastric area, he woke up and lay down again to drink some milk, his tongue covered with a white look when he examined the patient. On palpation there is pain above the navel to the right of the midline of the abdomen. Describe the specific radioscopic signs for the disease. What complications specific to the disease may remain in this patient. Establish a diet regimen for this patient.
Analysis of t : Chronic sigmaiditis is… ..	**Practical skills:** Outgoing small bowel palpation technique

Assessment 4

Te s t **25. Is it detected on gastrofibroscopic examination? 1**). Gastric or peptic ulcer: 12). Gastric or peptic ulcer: i .3). 12 duodenal and sigmoid ulcers i .4). Inflammatory tumors of the stomach and duodenum, polyps.5). Ulcers and tumors of the colon and small intestine 1). 2-3; 2) .- 1-4; 3) .- 3-4; 4) .- 4-5; **62. “Fatty feces (garbage)” is specific to which of the following diseases;** a) Liver cirrhosis b) Chronic pancreatitis c) Chronic gastritis g) Chronic hepatitis	**Situational issue** 7 masala The patient is 27 years old, female, has a constant rest on the abdomen, laughter, diarrhea is sometimes fluid, subjective - the above symptoms are more annoying in the spring and autumn. The patient has a disturbed diet, consumes alcohol. Objective - palpation revealed the presence of pain above the umbilicus and on the left side (symptom Porges) and along the small bowel (symptom Sternberg), laughter in the abdominal area. Your approximate diagnosis? Create an examination plan in the patient and state the changes in it? What changes may occur in the patient's stool ? Create a par h ez table and meal regimen for this patient ?
Analysis of t : The concept of 12-finger bowel probing	**Practical skills:** Transverse ileal palpation technique

Assessment 5

Te s t **15.O'tkir gastritis EGD changes?** 1). Erosions, hemorrhages. 2). Hyperemia of the mucous membrane. 3). Atrophy of the stomach folds. 4). Stomach and 12 b / i ulcers and bleeding. 5). Hypertrophy of the gastric mucosa. 1) .- 1-2; 2) .- 1-3; 3) .- 2-3; 4) .- 3-5; **60. The main reason for the appearance of "vascular asterisks" in the body is:** a) O rganizmda esterogenlar meeting and the	**Situational issue** 17 - masala The patient, a 23-year-old female, was taken to the Emergency Medical Center, and an interrogation revealed that she had been ill for many years and that the disease had bothered her in the spring and fall, and that she had a history of gastric ulcer. The patient's complaint is weakness, vomiting, and the vomit mass is coffee-like. On examination of the patient, the color is pale, the stool is dark (decteobraz), he is weak, the pulse is accelerated, blood

reduction of the liver to inactivate them b) It is a disorder of carbohydrate metabolism c) T Amir increase in permeability g) J Igarassu hardware reticulo-endothelial dysfunction	pressure is 80/50 mm. sim. ust. teng. Make an approximate diagnosis of the patient? What tests should be performed to confirm the diagnosis? What are the changes in FGDS?
Analysis of t : Radiological signs of wound disease…	**Practical skills:** Stomach palpation technique

Urinary tract ascents

Assessment 1

Te s t	**Situational issue**
1. Changes in the oral cavity in kidney disease : **A) .** Tongue pale, dry; **B)** . Tongue raspberry, angular stomatitis; **C)** . Tongue and lips bluish-red, atrophy of tongue suckers; **D).** Whitening of the tongue, stomatitis, ulcerative-necrotic angina; **E)** . The smell of ammonia comes from the mouth A) AB C) CE B) BD D) AE **2 . Normal urea content in blood serum -…** A) 3.23-8.32 mmol / l B) 9-12 mmol / l C) 14-18 mmol / l D) 5.2-9.30 mmol / l	10 issues The patient is 33 years old, female. When the doctor came to the reception, he noted that he had been ill for a long time, the patient complained of severe stabbing pain in the left lumbar region and the pain was transmitted to the lower abdomen. When collecting a medical history from the patient, it was found that there was pain a on the left side in the lumbar region while walking in the vehicle, the pain is transmitted to the bladder and between the legs. When the patient was questioned, it was found that putting a heater (hot water heater) in the lumbar region and taking hot anna slowed down the pain. How can this patient be diagnosed ? What are the percussion changes in the patient? Describe the changes in the patient's urine? What instrumental examinations will be acceptable to the patient and what changes will be made What are your treatment tactics for this patient ?
Analysis of t : **Explain chronic pyelonephritis**	**Practical skills** **Renal palpation technique**

Assessment 2

Te s t	**Situatio issue**
3. Instruments for the diagnosis of chronic kidney disease in the brain are not included: **A)** . Exography; **B)** . Laparoscopy; **C)** . Phonography; **D)** .Scanning; **E)** .Puncture biopsy; **J)** . Retrograde pielography; A) AB C) DJ B) AC D) EJ **4 . Macrohematuria is -…** a) Separation of epithelium in the fluid b) Increased red blood cell count in tissues c) Leukocyte secretion in urine d) Direct presence of blood in the urine	15 masa A patien who cam the villag doctor's office complai as follows. noted tha left side the lumb region w

	sore, wit frequent urination and that urine wa clear. W question the patie was foun have occasion fever, headache and hear palpitatio and state that he h had the disease f several y and was being treated. U analysis revealed presence large numbers leukocyt Make an approxin diagnosi the patie Describe changes the patie blood an urine . What instrume inspectio are performe and wha changes be seen ?
Analysis of t : **Explain chronic glomerulonephritis**	**Practica skills** **Urine collectio techniqu**

	Zimnitsk method

Assessment 3

Te s t **5 . Indicate changes in the blood in renal failure syndrome: A**) . Increased creatinine levels; **B).** Increased urea content; **C)** . Increase in the amount of aldolase; **D)** . Increased bilirubin levels; **E)** . Proteinuria; **J)** . glucosuria A) AB -E C) DJ -A B) EC -B D) BD -E **6. In the Zimnitsky test… is determined?** a) Salts b) O qsillar c) Shape elements d) Relative density of urine	**Situational issue** 20 issues B e defeated the 26-year-old woman, sex, accommodation polyclinic of the zone T e rap e vtida chronic Grom e rolls e frit diagnosed with dispans e control of the game, in the last days k e gaining increased frequency of urination, and e defeated for the treatment of n e applied to the frology department and was hospitalized for treatment. This is what is called a urinary incontinence disorder. K e chqurungi urinary diseases in which the frequency of divorce Cup. This type of separation of urine which is determined by the test and assembly t e machinery.
Analysis of t : **Explain chronic cystitis**	**Practical skills** **Urine collection technique by Addis-Kakovsky method**

Assessment 4

Te s t **11 . Changes in general urinalysis in chronic pyelonephritis (3):** **A)** . Leukocyturia; **B**). Bacteriuria; **C)** . Erythrocyturia; D). Massive cylindruria; **E)** . Dysuria A) ABE C) CDE B) ACD D) BDC **12. Cannot cause true anuria.** a) Acute glomerulonephritis b) When transfusing blood from another group c) When the spinal cord is damaged d) Anaphylactic shock	**Situational issue** 26 issues The patient was a 67-year-old male male, under the supervision of a dispensary with a prostate adenoma in a rural therapist at a rural medical center, sometimes disturbed by simmering pains in the lower abdomen and lumbar region, and suddenly stopped urinating. The patient was referred to the regional diagnostic center for treatment and hospitalized in the nephrology

	department. This is what is called a urinary incontinence disorder. In what diseases this type of urinary incontinence occurs. Urine collection and its technique in Ambyurje method i . Provide emergency assistance to B e Morga.
Conceptual analysis: **Explain pollakiuria**	**Practical skills** **Technique of urine collection by Hechiporenko method**

Assessment 5

Test

13. Chronic e Grom rolls of great importance to the e Fritz etiology (3); A) . Systemic lupus erythematosus ; **B)** . Str e ptakokkli inf e ksiya ; violation of the coal transport ; **E)** . Radiation effects ; **J)** . Excessive fat foods li ist e pro

A) ABE C) CDJ

B) ACD D) ABC

14 . Not characteristic of urinary syndrome of acute and chronic glomerulonephritis :

a) The predominance of leukocytes over erythrocytes

b) Proteinuria

c) G ematuria

d) T cylindruria

Conceptual analysis: **Nocturia Explain**

Assessment 6

Te s t **15. I plays a crucial role in the development of pyelonephritis (3); A)** . Urinary stone disease; **B)** . Nervous tension **C)** . Heredity; **D)** . Urodynamic disorders; **E)** . Hunting q atlanishning disorder; **J)** . J physical boredom A) ABE C) CDE B) ACD D) DCJ **16 . Symptoms characteristic of urinary syndrome in acute nephritis, except one** a) Proteinuria b) Cylindruria c) Macrohematuria d) M microhematuria	**Situational issue** 24 masala B e defeated 30-year-old woman, sex, clinics zone t e rap e vtida kidney disease dispans e r-controlled, in recent days has increased the daily amount of urination, and e defeated accommodation and appealed to the clinic for treatment for the treatment of B e Moguls n e was hospitalized in the frology department, the relative density of urine decreased when t e x-ray (norm 1,015–1,028). This is what is called a urinary incontinence disorder. In what diseases this type of urinary incontinence occurs. Zimnitskiy method of urine collection and the T e machinery, m e m todikasi email contains content
Conceptual analysis: **Explain polyuria**	**Practical skills** **Techniques of retrograde pyelography**

Hematopoietic system assimilations

A session 1

Te st **28. Foods that affect the absorption of the trace element iron; A)** Liver; **B)** Cocoa; **C)** Veal; **D)** Black coffee; **E)** Meat; **J)** Karachay and milk; **Z)** Kisel; **I)** Black beef; **A)** BDJZ **B)** BCDI **C)** ACEZ **D)** CDEI **16. What are the typical symptoms of pica chlorotica:**	**Situational issue** 1 masala The patient was 25 years old, was examined by a physician at the time of admission to the hospital, and the following changes were noted. The skin is dry and mobile, pale, mucous membranes are flowing, nails are brittle, coylonychia is observed, the corners of the mouth are cracked, the nipples are atrophied, there are black spots on the teeth, destroyed, alveolar piorea is observed, the hair is thin. brittle,

A) T e ri dryness **B)** D e formation of nails **C)** Dysphagia **D)** Likes unpleasant odors	dry and brittle. The color index in the blood is 0.63 (hypochromia). How do you diagnose a patient? List the etiological factors of the disease. List patient complaints. Describe the changes in the cardiovascular system. Describe the macroscopic and microscopic changes in the blood.
Conceptual analysis: **Mechanism of development of vitamin B12 and folate deficiency anemia**	**Practical skills** **Techniques for determining the amount of hemoglobin.**

A session 2

Te st	**Situational issue**
19. Which food products, t e mir well tolerated in the body: **A)** Fish; **B)** Liver; **C)** Chicken; **D)** Veal; E) Turkey meat; J) camel meat; **A)** AB **B)** BE **C)** BD **D)** DC **15. T e mir is absorbed in the digestive tract :** **A) The** colon **B) In the** stomach **C) In the** upper part of the duodenum and small intestine **D)** Stomach and 12-fingered intestine	3 masala B e mor is 23 years old, female, 28 weeks pregnant. The patient's problems, fatigue, dizziness, and his eyes grew dim, impairment, t e z fatigue, dyspnea, ear noise, body drenched with feeling of the march, t e ri s e zuvchanligini blocked, or the ability to know the full ' remain, nausea, disfagiya b e zovta said, and ask investigated k e Sak and chalk ist e wants k e b contain e RDI. B e morning blood t e kshirilganida blood color display 0.71 (g), which focused e moglobin 85 g / l May and 3.6 million red blood cells (DA), different shapes and different look at e rli (anizotsitoz paykilotsitoz) the presence of red blood cells, micro cytosine that is, performing an average of magnifying e reimbursing the picture settings. What is your approximate diagnosis? B e Morning k e can see the light of any changes? What changes can we see in the cardiovascular system?
Conceptual analysis: **Explain lymphagranulomatosis**	**Practical skills** **Determination of erythrocyte count.**

A session 3

Te st	**Situational issue**
21. Does it enter the blood-forming organ? A) Bone marrow, lymph nodes, lungs; **B)** Spleen, liver, bone marrow; **C)** liver, bone marrow, heart; **D)** Spleen, liver, lymph nodes; **E)** lymph node, heart, liver; **A)** BD **B)** AB **C)** BE **D)** BC **14. The composition of the food t e Mirna maximum amount of absorption:**	4 masala The woman was 21 years old, 30 weeks pregnant, and when she came to the women's clinic, a subjective examination by a doctor revealed that her eyes were dark, dizzy, and the objective skin was pale, her nails were sunken and thin. In the patient's blood there are 2.8 million erythrocytes and 64 g / l (low) hemoglobin, a heart rate of 104 beats per minute in the cardiovascular system and a soft systolic murmur between the V ribs at the apex of the heart.

A) 4.5 mg / day **B)** 1.5 mg / day **C)** 2.5 mg / day **D)** 3.5 mg / `per day	Tell the approximate diagnosis of the patient. What a change we can see in an objective examination of the patient. Describe the changes in the cardiovascular system Describe the possible changes in the patient's blood.
Conceptual analysis: **The concept of chronic myelogenous leukemia**	**Practical skills** **Determination of leukocyte count.**

A session 4

Te st	**Situational issue**
35. Arrange the following in order; 1) What are iron-rich products and 2) What products reduce iron absorption? **A)** Liver; **B)** Cocoa; **C)** Veal; **D)** Black coffee; **E)** Meat; **J)** Karachay and milk; **Z)** Kisel; **I)** Black beef; **A)** 1-ACEI and 2-BDJZ **B)** 1-ABCE and 2-IDJZ **C)** 1-ACJZ and 2-EDJZ **D)** 1-ACDI and 2-EBZJ **17. Lymph nodes that are not red, enlarged, painless, do not stick to the skin and do not stick together are typical for which disease?** **A) For** erythremia **B) For** lymphosarcomas **C) For** lymphagranulomatous disease **D) For** leukemia	26 issues The patient, a 47-year-old female, complained of weakness, sweating, decreased ability to work, itching of the body, swelling in the neck area and under the lumbar region, and fever when she came to the doctor's office. A biopsy of the lymph node revealed multinucleated Berezovsky-Sternberg cells. The patient's blood count was 0.67 hypochromia, erythrocytes 2.5 million, ie anemia, neutrophil leukocytosis, eosinophilia, thrombocytopenia and increased ECG (50-70 mm / s). Make an approximate diagnosis of the patient. What changes do we see when the patient is examined? Describe palpator and percussion changes. What instrumental tests are performed and what results can be expected ?
Conceptual analysis: **The concept of chronic lymphocytic leukemia.**	**Practical skills** **Determining the time of blood clotting.**

A session 5

Te st	**Situational issue**
7. Arrange the following in order; 1) Iron deficiency is characteristic of anemia and 2) Vit. B12 and folate deficiency anemia-specific changes; A) Sid e ropenik syndrome **B)** The yellow shade pale t e settings to try to e rli: **C)** kattalashuvi liver; **D)** Kebot rings and Jolly bodies; **E)** Cologne; **J)** Pica chlorate: **Z)** Rangpar skin; **I)** Hyperchromia; **A)** 1-A- E -JZ and 2-BCDI **B)** 1-ACEI and 2-BDJZ **C)** 1-ACJZ and 2-EDJZ **D)** 1-ACDI and 2-EBZJ **11. Microcytes appear in the following cases**	10 issues. B e defeated visiting a doctor k e configure weakness, heart palpitations, nausea, dyspnea, language impairment, poor appetite, irritation (see below) b e zovta to stay human. B e morning flow cavity of the k e muted red and orange shining, smooth surface of the papilla due to atrophy (atrophic Gunter e glossiti survey), mucous membrane of the mouth and the back sides of the palate atrophy language, those around the teeth appear in the mucous membrane of the mouth ulcers, as well as the Caribbean, e s

A) G e molitic an e brain **B)** Vit B12 d e fitsitli an e miya **C)** T e mir d e fitsitli an e miya **D)** Hypoplastic an e brain	existence. B e defeated the blood t e kshirilganda m e m galotsit and e galoblast cells have been identified. What is your approximate diagnosis of B e morga ?. The development of the disease m e mechanisms to explain b e ring. N e rv sectors e Christ and the effects of m e mechanisms to b e ring.
Conceptual analysis: **The concept of hereditary hemolytic anemia.**	**Practical skills** **Determination of erythrocyte sedimentation rate.**

A session 6

Te st **10. Arrange the following in order; 1) Hereditary hemolytic anemia and 2 - Acquired hemolytic anemia; A)** Fermentopathy; **B)** Membranopethy **; C)** Systemic diseases; **D)** Infection; **E)** Hemoglobinopathy; **J)** Enzymopathy; **Z)** Toxic substances; **I)** Rhesus asymmetry; **A)** 1-ACEI and 2-BDJZ **B)** 1-A- E -JZ and 2-BCDI **C)** 1-ACJZ and 2-EDJZ **D)** 1-ABEJ and 2-CDZI **26. Sid e ropenik syndrome b e lgilariga, with one addition:** **A)** Dryness of T e ri coatings **B)** Difficulty swallowing **C)** Painful swallowing **D)** Stuck food in the esophagus	**Situational issue** 8 masala The patient was elderly, in an outpatient setting, when he was seen by a neurologist. On examination of the patient's oral cavity, the tongue is dark red and shiny, the surface is smooth (atrophic Gunterov glossitis) due to atrophy of the nipples, the mucous membranes of the mouth and the back of the palate are atrophied, ulcers appeared in the mucous layer of the trace and the presence of caries on the teeth. Jolly bodies and Kebot rings were detected in the blood. What is your approximate diagnosis ? List patient complaints. What instrumental

	inspections are performed and what changes are observed ? Changes in the laboratory b e ring
Conceptual analysis: **The concept of acquired hemolytic anemia**	**Practical skills** **Color index detection technique**

A session 7

Te st **1. Changes in hair observed in iron deficiency anemia?** **A)** Oily hair is observed; Hair split ends and brittle **b) C)** C feeding the tunic, ingichkalashishi ; **D)** M becomes brittle and spills ; **E)** No change in hair; **J)** Q seed and brittleness are observed ; **A)** ADEJ **B)** BCDJ **C)** ACED **D)** CDEJ **13. Microscopy of the blood revealed Jolly bodies and Kebot rings. Determine which disease you need:** **A)** Iron deficiency anemia **B)** Posgemorrhagic anemia **C) In** aplastic and hypoplastic anemia **D)** Vitamin B12 and folate deficiency anemia	**Situational issue** **27 masala** Examination of the patient at the doctor's office revealed that the peripheral lymph nodes were enlarged and connected to each other, there were scars on the body and some parts of the body were bruised. Multi-nuclear Berezovsky-Sternberg cells were found in smears taken from the patient's lymph node, bone marrow. Determine the approximate diagnosis of the patient. List the patient's complaints. Palpator, tell the percussion changes Describe the changes that occur in the patient's blood.
Conceptual analysis: **Mechanism of development of vitamin B12 and folate deficiency anemia**	**Practical skills** **Technique for determining the number of erythrocytes.**

A session 8

Te st

3. the

following order ; 1. chronic 2. mieloleykozga and chronic limfoleykozga kind o ' changes chromosomes ; **I)** Botkin - Gumprext bodies ; **Z)** Caesarean section syndrome ;

A) 1-ACEI and 2-BDJZ

B) 1-ABCE and 2-IDJZ

C) 1-ACJZ and 2-EDJZ

D) 1-ACDJ and 2-BE-IZ

9. Whitening, thinning of hunger ; m secondary Suffer wipping and i , q thorns and fragility

A) Hereditary hemolytic anemia

B) Acquired hemolytic anemia

C) In iron deficiency anemia

D) Vitamin B12 and folate deficiency in anemia

Conceptual analysis:
Explain cologne and what disease it occurs

A session 9

Te st	Situational issue
18. Changes in hair observed in anemia? **A)** C feeding the tunic, ingichkalashishi ; **B)** Oily hair is observed; **C)** M becomes brittle and spills ; **D)** No change in hair is observed; **E)** Q seed and brittleness are observed ; **A)** A-D_E **B)** BCD **C)** ACE **D)** CDE **16. What are the symptoms of pica chlorotica:** **A)** Rest in the abdomen **B)** Increased appetite **C)** Dysphagia **D)** Disorders of taste	21 masala The patient was 24 years old, female, 21 weeks pregnant, was examined at the obstetrician-gynecologist's appointment and the skin was pale, yellowish, with bleeding and necrotic areas on the skin, oral cavity and the appearance of necrotic lesions in the mucous membranes, ulcerative necrotic gingivitis, stomatitis, angina, enlarged lymph nodes were detected and blood was drawn for RW. Seeing the positiveity of the tow symptom, the patient's blood was examined and the presence of lymphoblast cells was detected. Make an approximate diagnosis of the patient. List patient complaints. Describe palpation, percussion, auscultatory changes.
Conceptual analysis: **The mechanism of origin of enzymopathy.**	**Practical skills** **Technique for determining the rate of erythrocyte sedimentation.**

Thematic photos

Digestive system

Picture

Helicobacter pylori is not so terrible?

According to the WHO, about 60% of the world’s population is a carrier of Helicobacter pylori bacteria. It can cause many diseases from banal gastritis to stomach cancer. However, 60 percent of the population is absolutely peaceful to Helicobacter pylori and does not suffer from any disease. Why is that? It is known to rarely cause disease, but along with a weakened immune system, unhealthy lifestyle and poor nutrition, it can cause a number of diseases. So I advise you to give up bad habits, normalize your immunity and diet, then you will not be intimidated by any case with Helicobacter pylori.

Endoscopic examination. Esophageal ulcer

Endoscopic examination. Esophageal polyp

Red arrow is a malignant tumor of the stomach

The yellow arrow is in the median fold

The endoscope is depicted with a scalpel

A biopsy is taken from the med

Gastric lavage process. Funnel during gastric lavage insertion sequence.

Fractional examination of the composition of gastric juice.

Duodenal probing technique and patient status

Procedure for installing the exhaust pipe

Huqna transfer procedure

The process of siphoning: a - water is poured into the intestine through the funnel; b - After the funnel level is lowered, intestinal fluid exits through the funnel.

X-ray image display clarify finger symptom of what rentgenoskopiyu izobrajeno symptoms ukazatelnogo coat

Gastroskan 5 5 **M** with a single person or a medal in the field of photodynamic 5 medals pH metriyasi o ' the possibility of class and , a t the same time asked to assess the functional condition of the gastrointestinal tract upper parts of diagnostic tests o ' dogs .

The test can be performed on an empty stomach and after meals.
Gastroskan GEM » Computer equipment for peripheral electogastroenterography. Gastroskan GEM immediately to determine the acidity of the digestive tract for a
long period of electrical activity and mechanical spaces .

Diseases of the urinary system

Polycystic kidney disease

2. intravenous e kskr e narrow urografiyasi photo (116 a) and (b) . This method of investigation by the kidney ekskr e police to be contrast b e the root of the morning have been sent to radiology, renal failure, urinary tubes and siuda box shadows. Not only does it assess its anatomical condition but also its functional state, and it is also possible to see how quickly and well the contrast material separates. An X-ray is taken every 1-10-15-20-30-60 minutes for a patient sent for contrast. This screening method is an effective way to diagnose chronic pyelonephritis. In particular, the condition of the calyx and cups of the kidney is assessed.

116 Figures a and b

a) X-ray image shows hesitation of the kidneys and urinary tract.

b) pyelouretral stenosis of the right kidney and enlargement of the vessels over the stenosis.

A B

C 117 a, b, c photo

Kidney MRI. A kidney tumor is indicated by an arrow on the left kidney

Method of magnetic resonance imaging of the kidney; Magnitno - rezonansnaya Computers CT -mail

computed tomography examination method;

Renal artery stenosis on renal vascular angiogram

described.

Cystouretrogram Bilateral bladder and urethral reflux. Hydronephrosis. (puzырnomochetochnikovыy).

Excretory urography. Urinary hypotension.

Ultrasound examination of the kidneys

Healthy kidney exogram

Nephrolithiasis

Ultrasound examination of the kidneys and abdominal organs

Pictures of the endocrine system

Topic: Endocrine glands and metabolic system. Control methods. Inquiry, physical examination methods. Methods of laboratory and instrumental examination.

A ddison k honey

Double-sided knotted stalk

glycomer

hypothyroidism

X-ray images related to the subjects.

Methods of X-ray examination of diseases of the digestive system
A two-day irigoscopic (fluoroscopic) contrast clot, with biopsy-confirmed Girshprung's disease, shows a small-caliber bowel that easily separates the proximal parts of the colon. Notice the "bear" appearance of the aganglionic segment (arrowheads), which reflects incorrect contractions.

Ileal atresia results from mesenteric vessels in the uterus. (a) Radiography of the newborn with ileal atresia shows that several intestinal loops are separated in the abdominal cavity, which is a low obstruction. (b) In the same child, a fluoroscopic contrast clot indicates that the typical 'microcolon' (unused colon) and unexpanded distal flank are filled to the level of atresia (arrow). The proximal loops of the greater intestine are enlarged and filled with air

Esophageal atresia and tracheoisophageal effusion represent a spectrum of abnormalities resulting from abnormal formation and embryological anterior separation. These anomalies range from isolated EA to isolated TEF. the classification of congenital tracheysophageal anomalies depends on the presence and location of the flow connection between the two structures. (a) X-ray of esophageal atresia (no fistula) of the newborn indicates the absence of distal gas from the bowel. Patients with isolated EA may have polyhydramnios in the uterus, and an oogastric tube that is above the level of atresia at birth may not pass. (b) An X-ray of the newborn shows gas in the gastrointestinal tract despite esophageal atresia and a leak between the airway and the distal atrial segment, even though the feeding tube has not passed. (c) Fluoroscopic imaging of an infant with an isolated tracheosophageal effusion (arrow) after stimulation of a water-soluble contrast agent in the esophagus.

An extensive radiograph of the abdomen shows intestinal obstruction and enlarged intestinal loops. Shows white bile or possibly apricot peel.

The radioscopic image shows the passage of barium sulfate contrast agent through the esophagus

Radioscopic imaging of gastroesophageal reflux disease

An X-ray image shows intestinal obstruction

X-ray examination of a patient with constipation

An X-ray image shows Crohn's disease

An X-ray image shows Crohn's disease

Methods of instrumental examination of the urinary system Instrumentalnye techniques issledovaniya mochevydelitelnoy sistem y

Method of magnetic resonance imaging of the kidney;

The renal artery angiogram describes stenosis of the right renal artery.

Cystouretrogram Bilateral bladder and urethral reflux. Hydronephrosis. (puzырnomochetochnikovыy).

Excretory urography. Urinary hypotension.

Ultrasound examination of the kidneys

Healthy kidney exogram

Nephrolithiasis

Practical training materials

Practical d ' Typo № 1

Digestive system. Methods of examination of the esophagus, stomach, intestines (interrogation, physical, laboratory-instrumental).

1. Practical training module .

Training time - 4 hours	The number of students 1 : 0 to 12
Form of training	Practical training
Practical training plan	A method of examining patients with diseases of the digestive organs. Inquiry, anamnesis collection, general and oral examination.
The purpose of practical training :	Methods of examination of patients with diseases of the digestive organs: interrogation, collection of anamnesis, general and oral examination.
Teaching style	Inquiry. Demonstration of patients, interactive teaching methods, practical skills.
Form of teaching	In small subgroups.
O ' unit equipment	Calls to O Training Guidebook , practical Typo content , projectors , computer .
Training mode	Methodically equipped auditorium.
Monitoring and evaluation	Oral control: questions and answers, tests, problem solving.

1.2 . ***Technological card of practical training***

See the stages of hearts and q ti.	Educator	Learners
In the preparatory stage of q	1. See the audience to control the purity karîm 2. Checks students ' readiness for training 3. And control q karîm	
1. Drink and training g contain levels of intra q (10 da q i q a)	1. Preparation of educational content on the topic. 2. Preparation of presentation slides for the introductory speech 3. Develop a list of references used in the study of science	
See the 2 basic levels of diarrhea (160 da q i q a)	1. Divide students into small groups and ask questions on the topic. 2. Uses display posters 3. Uses slides, multimedia 4. Conducts treatment 5. Summarizes and summarizes the information provided on the basis of topics, encourages and actively evaluates the active participant students	They are divided into small groups They watch They participate They listen and answer questions
1- final press q ich (10 da q i q a)	1. Concludes 2. Provides independent work 3. Gives homework	He listens Takes notes Takes notes

3. Assessment of students' theoretical knowledge:

A) Frontal method:

1. List the main complaints of patients with esophageal pain
2. List the main complaints of patients with gastric diseases
3. List the main complaints of patients with intestinal diseases
4. A general review of patients with gastrointestinal disorders
5. A general review of patients with intestinal diseases
6. Language changes in diseases of the digestive organs
7. The diagnostic value of a general examination of the oral cavity

Brainstorming method

Basic rules :

- Not to mention the shortcomings that hinder the emergence of the idea
- The height of ideas and thought, because the more unusual an idea, the better it is
- Accept many offers
- Combination of ideas and their development
- Present the idea succinctly without argumentation
- Divide the group into two: thought generators and thought analysts

This method allows you to argue ideas and opinions, your own personal opinion, to find the optimal solution in any situation.

Interrogatio b e mor in diseases of the digestive system (interrogatio)

Anatomy and endoscopic examination of organs of the digestive system

It's time

to digest

The scheme of examination of patients with diseases of the gastrointestinal tract:

1. Assessment of the patient's condition.
2. Asking the patient.
3. Review.

4. Palpation.
5. Percussion.
6. Auscultation method is less informative.
7. Consists of instrumental inspection methods.
8. Methods of laboratory examination, examination by gastroscan.
9. Methods of functional testing.

I- When assessing the condition of patients with diseases of the gastrointestinal tract, attention is paid to the following;

1. To his consciousness (in himself, not in himself, stupor, sopor, in a coma, answers questions, answers correctly, answers late, answers incorrectly, etc.
2. Attention is paid to his condition (active, passive, obligatory state).

II-Patient Inquiry The following should be noted.

1. The main complaints
2. Additional complaints
3. Medical history (anamnesis morbidity)
4. Life history (anamnesis vitae).

III- The following should be considered during the review.

1. To the Constitution (asthenic, normastenic, hypertensive).
2. Absence of pathological changes in the abdominal area (abdominal volume, abdominal wall vessels, umbilical position, abdominal skin rash, skin color are taken into account);
3. The condition of the skin is assessed (skin elasticity, its moisture);
4. Tumor (Acit);
5. To the condition of the fingers of patients (drumstick finger) in purulent diseases.
6. The condition of the nails (the presence of clock-like nails (in purulent diseases)).
7. Body temperature (acute infectious gastrointestinal diseases).
8. Muscle condition (atrophied, absent) is assessed.

The following should be considered when palpating a patient with diseases of the IV-gastrointestinal system.

1. On superficial palpation (presence of painful spots, the condition of the skin of the abdomen is assessed);
2. Deep palpation (palpation of the stomach, intestines, liver, spleen and kidneys, which reveals the presence of pain and other pathological processes);
3. Detection of symptoms of tremor on palpation, in stenosis of the pyloric part of the medulla.

V- The following are important in the percussion of diseases of the gastrointestinal tract:

1. Type of percussion sound: tympanic, blunt;
2. Painful points are identified (for example, when percussive percussion in the epigastric area - Mendel's symptom is detected);
3. The size of the liver and spleen is determined.

VI-Auscultation reveals the following.

1. Laughter in the abdomen;
2. The lower part of the stomach is determined (auscultatory-atrial method)

VII- Instrumental inspection methods.

The following instrumental examination methods are performed in diseases of the gastrointestinal tract.

1. X-ray examination method (barium sulfate by sending a contrast agent);

a) X-ray of the esophagus, stomach, duodenum and other intestines;

b) Irigography or irigoscopy;

2. Method of endoscopic examination of the gastrointestinal tract;
    a) fibroesophagogastroduodenoscopy
    b) Colonoscopy
    c) Rectoromonoscopy
    g) capsule endoscopy (allegedly Gavriel (1981)) ;
    d) Double balloon enteroscopy (X ironori Yamamato 2004 .) ;

3. Ultrasound examination methods
4. Method of examination with gastroscan
5. Computed tomography, MSKT;
6. The method of scanning parenchymal organs

VIII-Laboratory test methods.

The following laboratory tests are performed in diseases of the gastrointestinal tract.

1. General analysis of blood.
2. Biochemical analysis of blood
3. Acetic fluid is examined clinically, cytologically, chemically.
4. The stool is coprologically examined.
5. Urine is checked.
6. Methods of morphological examination.
7. Methods of microscopic examination.

Laboratory tests (cholesterol-low and high-density lipoproteins, triglycerides, blood sugar, AlT, AsT, hemoglobin). Precise adherence to these methods is essential in making a definitive diagnosis in patients.

IX-Functional inspection methods

Methods of functional examination in diseases of the gastrointestinal tract.

1. Probing the stomach **.**
2. 12-finger bowel probing.
3. Check with gastroscan

Gastroskan belongs to the family of computerized instruments and is designed to check the function of the gastrointestinal tract. Intra-medial pH meter, daily pH meter, gastrocardiography, manometry, electrogastroenterography are performed using this test method. There are Gastroskan 5 M, Gastroskan 24, Gastroskan D, Gastroskan GEM, Gastroskan ECG and Gastroskan used in drug approbation.

With Gastroskan 5 M (Fig . 77 a) it is possible to perform intra-medial pH metrics at a time in 5 people or in 5 areas of the gastrointestinal tract, and at the same time diagnostic tests are performed to assess the functional status of the upper gastrointestinal tract. When measuring the acidity of meda juice, the pH is adjusted to a pH accuracy level of 0.2 to 1.1 to 9.2 units. The test can be performed for 2-3 hours and can be performed by 1 to 5 people at a time.

The test can be performed on an empty stomach and after meals. This device helps to determine the course of diseases of the stomach and duodenum and a number of other diseases. The gastroscan is programmed to display each patient's data on the monitor screen at the time of examination, it is possible to print them and the data can be stored in memory.

Gastroskan-24 (77 b picture) won the pH of the juice daily metriyasi is designed to hold the computer equipment. The patient keeps a computer-recording blog on a belt around his waist until the examination is complete. The patient is transplanted nasally to ensure that the pH probe does not interfere with feeding and lying down, and the pH meter is monitored by placing it at three points in the digestive tract selected by the health worker. Determining the presence of gastroscan-24 reflux, its degree of development (physiological or pathological), its aggravating factors (diet, body condition, medication

intake, etc.), the acidity of gastric juice at any time of day and determining the appropriate course of treatment will give. In the selection of proton pump inhibitors, drugs that act for 36 or 48 hours are selected.

In the detection of reflux eruptions and atypical forms of reflux disease, reflux induced asthma, pharyngitis, laryngitis, caries, etc., measuring electrodes are placed on the upper part of the throat or esophagus.

Gastroskan -**D** is a computer device for perfusion monometry. There are diseases of the gastrointestinal tract, which are accompanied by a violation of the motility of this or that part of the digestive tract and the functioning of their sphincters. In determining the motility of the esophagus and meda, the lower and upper sphincter of the esophagus, the peristalsis of the sphincter of Oddi, the method of their monometric examination is used. This method is through the digestive tract or that calm and normal pressure in the cavity or the (stress tests) to be held in time change **78 photo** based on. Gastroskan D is measured in the digestive tract through an 8-channel aqueous perfusion catheter. 0.5 ml of fluid per minute is delivered through the side wall or hole of each catheter canal. The liquid level pressure is measured by applying it to the sensor and displaying it on the monitor screen. Pressure measurement from 0 to 220 mm.sim above. measured in different parts of the digestive tract at the same time and 3 mm.sim. the upper is determined to the level of accuracy. This is determined by the outlet ports of the catheter ducts. The test takes 30 minutes. As a result, a wavy contraction of the esophagus is recorded on the monitor screen.

Gastroskan GEM (Fig. 78) is a computer device for peripheral electogastroenterography. Gastroskan GEM immediately to determine the acidity of the digestive tract for a long period of electrical activity and mechanical spaces . In this way, 40 -minute elektogastroenterografiya o ' computers . This way the digestive tract motility nahorda and to disarm Calls riqish llaganda or prokinetiklardan health certificate is also used in searches . The test method is noninvasive and is based on signals from three electrodes attached to the limbs. Signals received from this or that part of the digestive tract are processed on monitor screens. According to the results of the examination, the medical staff determines the activity and function of this or that part of the digestive tract. Priborda also padbor treats with the drug substance.

Gastroskan ECG (Fig. 79) is a computer device designed to monitor the movement of the acidity of the juice of the digestive tract and ECG for a long time. Because the pains of esophageal diseases are transmitted to the chest and these pains are also transmitted to the heart. Therefore, measuring the pH of the esophagus and gastric juice at once and taking an ECG will help in the differential diagnosis. Digestive tract acidity is conducted at three points and the same ECG is performed on three chest conductors CS-1, CS-2, SM -5. The device also provides correlation processing of the results obtained in pH meter and ECG. Detects heart rhythm disturbances, magnitude of ST segment displacement. This method not only assesses the pH of the digestive tract and the activity of the cardiovascular system, but also optimizes treatment by detecting gastroesophageal reflux disease. Correlation processing of the results of daily pH metrics and ECG monitoring plays an important role in the detection of cardiac arrhythmias and ST segment changes in the cardiac form of gastroesophageal reflux disease.

The instruments in the Gastroskan family are designed to handle most scientific research. These are:

-In the approbation and development of a new method of diagnosis and treatment;

-Choice of drugs and their dosage in the medical treatment of patients;

-It is a computer device designed to determine the effectiveness of the action of drugs in the treatment of specific diseases.

79 photos

Gastroskan used in the approbation of drugs.

Capsular endoscopy

The capsule endoscopic examination method consists of a capsule capable of viewing the stomach, 12-fingered intestine, small and lateral head, and colon using a miniature video camera. Patients should not eat for 8 hours before the examination. The capsule is easily swallowed, moving naturally in all members of the digestive tract, allowing 70,000 to 80,000 images to be taken. The resulting images are transmitted to a recording device attached to the patient's waist. The examination is painless and lasts 4-9 hours. It does not affect the patient's normal lifestyle. It is absolutely harmless to the patient, is used 1 time and is excreted naturally. With this method, information can also be obtained from areas not covered in the FGDS review. Pill Cam SB3 is used to examine the small intestine, Pill Cam COLON 2 is used to examine the colon, and Pill Cam patency is used to check the digestive tract . The method is absolutely painless, safe, comfortable and highly informative. The disadvantage of this capsular endoscopy is that the camera captures that side of the intestinal tract in whichever direction it is on and cannot photograph the other walls.

Double balloon enteroscopy (80 photos)

This enteroscope was created in 2001 by Hironi Yamamoto. The enteroscope consists of a flexible tube with a small diameter. Its entry into the intestine is accomplished by filling and expelling air from the balloons mounted on the tube. To check with this instrument, the enteroscope uses a telescope as well as an external tube balloon and an air pump system. This procedure is performed under general anesthesia.

Enteroscopy esophagus, held in the mouth me the opportunity to see Daniel, 12, duodenum and small bowel. Oral enteroscopy consists of 4 stages .

1. Enteroscopy, esophagus me privratnikdan 12 at the top of the vertical duodenum;
2. Crossing the Traits link;
3. Small bowel **assembly i ;**
4. **Tu** bowel **language;**

With a high level of reference, it is also possible to see parts of the bowel that cannot be seen with a colonoscope. The transanal enteroscopy method provides the results of a complete colonoscopic examination. In this method, the rectum, sigmoid colon, and small intestine can be seen and all changes in them can be detected.

Advantages of inspection. With double balloon enteroscopy, the entire portion of the small and large intestine can be seen.

The downside. **With a thin enteroscope tube, the process of turning or rotating within an organ is difficult .**

Preparing patients for examination. Before the test, patients are psychologically Thai or care for the sick must determine at this important and completely painless and easy method to be explained. Check before you go on a special Thai orgarlik, a series of laboratory analysis papers, an electrocardiogram, a therapist counseling and intestines huqna or laxative drugs through treatment.

This screening method is an effective method in the diagnosis of small bowel disease, Crohn's disease, intestinal bleeding, cessation of bleeding, tumor detection, biopsy, removal of polyps and foreign bodies, as well as treatment of small bowel stenosis . In addition, double balloon enteroscopy - ingicka inflammation of the intestine, small intestine, structure of the balloon dilyatatsiyasini me ' and postoperative bowel disease, chronic pain and diarrhea because of the determination. Today, this method is a new modern method in the diagnosis of small bowel diseases.

Complications of enteroscopy - pain syndrome, sore throat, hyperthermia, vomiting, bleeding 1.2%, perforation of the mucous membrane 1.7%, acute pancreatitis 0.6%, aspiration pneumonia and paralysis intestinal obstruction may be observed.

Diseases of the esophagus

In practice, patients with esophageal diseases are more common. Such diseases include developmental anomalies, functional diseases (esophageal dyskinesia), esophageal hernia of the diaphragm, inflammation of the esophageal mucosa (esophagitis), esophageal peptic ulcer, cardiospasm, diverticula, esophageal varices and a malignant tumor.

Inquiry of patients with esophageal disease. Patients are asked about their main and additional complaints, medical history and life history.

The main complaints: 1- Relatively neuropsychiatric symptoms: functional dysphagia, sore mouth, constipation.
2 - relatively motor-evacuatory in nature: organic dysphagia, odenophagia, ruminatio.
3 - relatively dyspeptic in nature: pyrosis (jaundice), eructatio (stuttering), regurgitatio (recording), impaired cognition (metallic taste, sour taste) (dysgevzia), agevziya complete loss of cognition.
Additional complaints include: Weakness, decreased ability to work, weight loss.

In the survey, patients with esophageal diseases had difficulty swallowing esophagus (dysphagia), sore throat esophagus (odenophagy), vomiting, hoarseness, wheezing , hypersalivation - complain of excessive salivation or impaired salivation (Shegren's syndrome).

Dysphagia (Greek dys-difficulty, dysfunction, phagein-eating) is a disorder of the passage of food into the esophagus and is a major symptom in esophageal diseases. At the same time, patients feel that the bite is stuck in the esophagus, there is pain and discomfort in the esophagus (behind the chest) while eating. In esophageal diseases, dysphagia can be permanent or temporary. Persistent dysphagia occurs in organic lesions of the esophagus (new tumors, scarring structures of the esophagus). At the onset of the disease, it is difficult to pass only solid foods. Later, due to increased esophageal constriction, even soft or liquid food becomes difficult to pass.

Paroxysmal dysphagia occurs in functional lesions of the esophagus (esophagospasm) and this often leads to malnutrition and agitation. Soft and liquid food is more difficult to pass through the esophagus, while solid food is relatively easier to pass (paradoxical dysphagia).

Difficulty passing food from the esophagus in diseases of the larynx and thyroid gland; in malignant tumors of the chest, enlargement of the mediastinal lymph nodes, aortic aneurysm; cardiac achalasia (diseases associated with reflex opening of the heart when swallowed), peptic strictures, and esophageal tumors. Hypertrophy of the left ventricle can also be observed due to displacement of the esophagus.

Pain. In diseases of the esophagus, the pain is constant, aggressive in nature, localized in the back of the chest, irradiated to the shoulder, neck, left side of the chest, resembling an angina attack. In functional disorders (dyskinesia), pain is often associated with spastic contraction of the esophageal wall. Pain in esophagitis is associated with inflammation of the mucous membrane of the esophagus. The occurrence of pain in such cases leads to the release of substances from the stomach into the esophagus (gastro-esophageal reflux disease) when eating a lot of food, the body is bent forward, in a horizontal position. In new tumors of poor quality, patients complain of persistent excruciating pain associated with tumor growth in the esophageal serous layer.

Vomiting. Vomiting from the esophagus occurs in narrowing of the esophagus (e.g., narrowing of the esophagus with scarring strictures or tumors). Unlike vomiting in diseases of the stomach and duodenum, vomiting from the esophagus is trapped in the

esophagus, there is no nausea before vomiting, and occurs without the involvement of the muscles of the anterior abdominal wall. In this case, the sputum mass consists of a low-altered, chewed mass of food mixed with saliva that does not contain HCl acid, pepsin. In esophageal cancer, the sputum mass has a purulent odor and is mixed with blood, retaining a certain amount of mucus from food debris that has already been consumed.

Regurgitation. It is observed when a certain part of the ingested food returns to the oral cavity and also obstructs the passage of food from the esophagus. Evening regurgitation of substances accumulated in the esophagus is often observed in cardiac achalasia.

Bruising (eructatio) - Blowing with air can be observed. At the base there is snoring with odorous air, which occurs as a result of prolonged stay of food in the digestive tract.

Heartburn (pyrosis) is a burning sensation in the area of the sternum of the sternum, a symptom common in esophageal diseases (reflux esophagitis, esophageal hernia of the diaphragm, cardiac insufficiency). The mechanism of occurrence of urticaria is due to the effect of gastric and duodenal secretions from the gastric mucosa of the distal part of the esophagus. In addition to the listed complaints, patients with esophageal disease complain of excessive salivation (hypersalivation), wheezing with air or stomach substances, bad breath, and hiccups.

Bleeding . An important symptom in the diagnosis is bleeding. Its causes are esophageal peptic ulcer and malignant tumor, damage to the esophageal wall with foreign substances, rupture of dilated varicose veins of the esophagus (in patients with cirrhosis of the liver), linearization of the mucous membrane of the esophageal cardiac area caused by severe vomiting in incisions, such as in patients consuming alcohol (Mellori-Weiss syndrome). Unlike bleeding in gastrointestinal diseases, arterial bleeding of the esophagus (e.g., from a peptic ulcer, from an affected tumor) is characterized by unchanged blood mixing in the sputum mass. When bleeding from dilated varicose veins of the esophagus, the color of the blood in the vomit mass becomes dark red.

Anamnesis of morbi. When collecting a medical history, we should pay attention to the acute onset of symptoms. For example, adenophagy painful swallowing often begins acutely with burning of the mucous membrane of the esophagus. Dysphagia - a disorder of swallowing, which begins acutely in esophageal burns, and gradually develops in esophagitis. Attention is also paid to pain irradiation. In most cases of esophageal disease, the pain is constant, simultaneous or aggressive, and is given under the stone, shoulder, neck, and left chest. In esophageal achalasia, dysphagia begins suddenly and patients can also tell when it started and often develops after psychiatric factors. Outbreaks appear to be exacerbated during remission.

Anamnesis vitae. When collecting a life history, it is advisable to ask about the factors that lead to esophageal disease. This includes hot food or other thermal or chemical substances that burn the esophagus (whether acetic acid, acids and alkalis were consumed as suicide), whether or not there were esophageal traumas, whether the patient had harmful habits such as smoking, alcohol , are asked to constantly consume excessively spicy foods, which can lead to reflux esophagitis. Patients are reported to have other internal diseases that damage the esophagus. These diseases are inquired into by diseases such as systemic diseases (systemic scleroderma), iron deficiency anemia.

• **Methods of physical examination.** In esophageal diseases, the possibility of physical examination methods is severely limited, only examination is important. Examination of patients reveals a number of remarkable symptoms - weight loss, and even cachexia (esophageal cancer and achalasia (violation of esophageal sphincter emptying)), dry skin, whitening (esophageal cancer). Sometimes dehydration , vitamin deficiency is observed. Traube area loss is observed when percussion is performed in cardiac achalasia and tumors covering the esophageal surface, but percussion is less

informative. Palpation in esophageal diseases, and auscultatory examination methods are almost not used for low informativeness.

Instrumental and laboratory testing methods . X-ray, endoscopic, morphological, esophagotanochemical examination methods are used in the diagnosis of esophageal diseases.

1. X-ray barium sulfate into the esophagus patients to take and you can see some esophageal diseases or that side (for example, you can see the hypertrophied left ventricle (mitral stenosis), thorax authorities o ' smalarda). X-ray examination allows to assess the condition, shape, length, contours of the esophagus, to determine the relief of its mucous layer, to know the various disorders of motor skills. Various radiological methods are used today to make a definite diagnosis (simple radioscopy and radiography with barium sulfate, double contrast, computed tomography, X-ray cinematography and X-ray television, nuclear magnetic resonance imaging, pneumomediastenography, etc.) allows to identify changes in the thoracic organs that lead to esophageal damage (lymph nodes, tumor enlargement, aortic aneurysm, etc.). For more accurate information when examining patients, X-rays of different projections can be taken on them in different situations.

• 2. Endoscopic examination method is a clear method that can assess the condition of esophageal mucosa inflammation, redness, mucosal swelling, mucosal ulcers, polyps, muscle contraction, dilation, esophageal sphincter condition. **Esophagoscopy.** For endoscopic examination of the esophagus, a fibroesophagoscope or a capsular endoscope (Fig . 81) is used , which consists of a special lateral illuminating optics . Esophagoscopy helps in the diagnosis of esophageal diseases, allows biopsy of the affected area, if necessary, histological examination of the material, various therapeutic manipulations: esophageal dilatation, bleeding vascular electrocoagulation (eg in peptic ulcers), esophageal varicose veins creates conditions for sclerotherapy.

Capsule edoscopy examination method;

a b

v g d

81 photos. a) Radiocapsule structure; b) Receiving a radio capsule; c) Radiocapsule operation process; g) Installation of the radio capsule; d) Radiocapsule receiver;

4. esophagus Manor e trick checking the esophagus Sphinx e tone settings condition score. 4 - daily pH - monitoring; 5 - Impedanceometry examination determines the motor activity of the esophageal muscles. 6. Determination of daily pH. Determines the pH value from 4 to more than 7 and the duration. It also determines its association with subjective symptoms. (Food intake, body condition, medication intake, smoking) provides control over the effectiveness of the use of drugs and the possibility of individual therapy. 7. Determination of pH for 48 hours (without probe) using Bravo radio capsule. The environment in the esophagus allows to determine the pH of the esophagus for 48 hours without a probe without causing excessive discomfort in the patient (see photo V.GD above).

5. The Bravo pH radio capsule, manufactured by the US company Medtronic since 2003, does not have the ability to move freely. Using a special device it esophagus EPITO e liysiga set, usually in the lower esophagus Sfinks e lied about 5 cm above and n e cha days (usually 48 hours), the lining of the esophagus layer pH Coaster 'on the b e stature morning or marks on a continuous integrated priyomnikka b e rib. Then the information from the computer e RGA removed and then analyzed. The capsule carrying a n e cha night k e yin EPITO e liydan cut, and along with the feces out through the anus.

Other inspection methods. An additional method in the diagnosis of malignant tumors of the esophagus is cytological examination, in which the esophagus is washed

with water or taken as a separation from the mucous membrane, or using bioptat material. The separation is obtained using a special probe consisting of a wide-surface illuminating balloon.

Esophagotonochemography is used to record the activity of various parts of the esophagus, as well as to determine the tone of the lower esophageal sphincter and in the diagnosis of esophageal dyskinesia, cardiac achalasia, diaphragmatic esophageal sphincter hernia. Intra- esophageal pH meter is used to diagnose gastroesophageal reflux disease . In this case, when the pH of the esophagus is below 4.0, it means that the sour substances in the stomach are transferred to the esophagus.

Endosonography. In recent years, endosonography is one of the most common methods in the diagnosis of esophageal diseases , which is an ultrasound examination that involves inserting an ultrasound sensor into the esophagus using an esophagoscope. This method helps to identify esophageal tumors (especially if they are under the mucosa) and is used to assess the condition of regional lymph nodes, which answers important questions in surgical treatment.

Various pharmacological agents are currently used in the differential diagnosis of organic and functional constrictions of the esophagus . Nitroglycerin reduces the tone of the lower esophageal sphincter, facilitates the passage of food from the esophagus in patients with cardiac achalasia, and does not have such an effect in organic lesions of the esophagus.

Diseases of the digestive system diseases fail to b e Morley's claim pathological process and will vary depending on which part of the digestive Road damage. Bo'shli'g'ida local inflammation of the mouth or teeth, the Caribbean, e si k e lib out of the field of dentistry, surgery, pain elsewhere in the nation. Stomatitis (inflammation of the mucous membrane of the oral cavity) - local or general causes of diseases as the appearance of a k e may contain. Stomatitis is accompanied by pain in the oral cavity. The pains are mainly on the tongue and gums, and these pains are exacerbated when consuming sharp and salty foods. Tongue soreness can be one of the symptoms of poor quality anemia (Addison-Birmer disease). Bitterness and unpleasant taste in the mouth are observed in gastritis and especially in diseases of the liver and biliary tract.

Excessive dry mouth is observed in diabetes. Patients with esophageal disease complain of: dysphagia, pain throughout the esophagus, vomiting, heartburn, and bleeding. Dysphagia is a condition in which it is difficult to swallow or not swallow the food taken at all. Dysphagia can occur in esophageal tumors, ingestion of a foreign body, scarring after burning of the esophagus under the influence of alkalis or acids, as well as compression with an aortic aneurysm or posterior thoracic tumor and diverticulum disease . Bleeding from the esophagus occurs as a result of rupture of dilated varicose veins in liver cirrhosis. The main complaints in diseases of the stomach are pain in the epigastric area, nausea, vomiting, loss of appetite, belching, heartburn. When there is pain in the stomach, it is necessary to determine not only its localization, but also (irradiation, nature and under what conditions it occurs (meal time, nature of eating, etc.). Vomiting is one of the most common complaints and can be observed in various diseases of the stomach (acute and chronic gastritis, ulcers, piloroduodenal stenosis, gastric cancer, etc.).

When questioning the patient, it should be borne in mind that the symptom of vomiting can be observed in other diseases, regardless of the stomach. For example, it can be found in brain tumors, meningitis, uremia, cholecystitis, peritonitis and other diseases. However, vomiting can be observed when taking certain drugs (digitalis) and as a result of exposure to certain chemicals in the gastric mucosa (sulema, arsenic).

In acute gastritis, vomiting begins after eating. In gastric atony and hypersecretion, vomiting begins 3-4 hours after a meal.

Stenosis of the gastrointestinal tract is characterized by vomiting in the stomach, in the mass of vomit is stored food debris in the amount of 3-4 liters, and has an odor similar

to the smell of "eggs in the stomach." Sometimes during severe vomiting, small blood vessels in the gastric mucosa are damaged, resulting in the appearance of blood droplets in the vomit masses.

Excessive bleeding from the stomach is a serious symptom and occurs as a result of large vascular damage in gastric tumors and ulcers. Sometimes bleeding can also be caused by burning of the gastric mucosa, hemorrhagic diathesis, varicose veins of the stomach in portal hypertension. In such cases, the blood in the vomit mass is light red. If vomiting occurs some time after the bleeding, during which time the blood has time to join with the gastric mucosa, and as a result the vomit mass becomes the color of a coffee grounds. This is most often seen in stomach cancer.

Before vomiting, the patient develops nausea. This symptom may or may not be related to gastric disease. This symptom is also found in hypertensive crises, kidney disease and other diseases.

As a result of the intensification of the process of digestion in the stomach, the patient experiences bloating with air or food mixture. In patients with neurosis, long-distance audible, in children, hoarseness with vocal air is observed. This symptom is called “aerophagia”. There is also a bitterness similar to the smell of bitter oil. Such fermentation occurs when organic acids (fat, lactic, acid, etc.) are formed during the fermentation process. Sometimes patients complain of heartburn. This often occurs as a result of an increase in acidity that is not stored in the stomach, but heartburn can be observed when the acidity is normal and even low. The cause of heartburn is the return of gastric contents to the esophagus as a result of cardiac sphincter insufficiency.

The patient's appetite should also be taken into account when questioning. When the acidic environment in the stomach is low, the appetite is reduced, whereas in gastric diseases accompanied by a high acidic environment, the appetite is high. However, it should be borne in mind that in patients with peptic ulcer disease, accompanied by an increase in the acidic environment, they refrain from eating for fear of an attack of pain, despite the fact that the patient's appetite is high. Some patients have an extremely high appetite, i.e. bulimia (wolf appetite).

Complete loss of appetite (anorexia) and inability to eat especially meaty foods are characteristic of stomach cancer. Weight loss will be followed by loss of appetite. In intestinal diseases, the main complaint of the patient is a feeling of pain throughout the intestines, flatulence, diarrhea, constipation and sometimes bleeding from the intestines. Intestinal pain occurs as a result of spasm of the intestinal muscles and is often observed in inflammatory processes of the small intestine (enteritis) and colon (colitis). Spastic pains can also occur when poisoned with kim-wild substances such as arsenic, miss. The pain becomes numb, especially in chronic poisoning with miss.

Pain can also occur as a result of a sudden enlargement of the bowels or accumulation of large amounts of gas due to diarrhea.

When pain in the intestine is observed, it is important to determine its localization. If the pain is in the left flank area, it indicates sigmoid bowel disease, and if there is pain in the right flank area, it indicates appendicitis. Pain in the middle part of the abdomen is observed in diseases of the small intestine. Pain that occurs during the act of defecation is observed in diseases of the rectum (hemorrhoids, anal fissures, sigmoid and rectal tumors), and the mixing of blood with feces is detected.

If the bleeding is from the upper part of the intestine, it is characteristic of duodenal ulcer disease, in which the stool is black.

Bleeding from the intestine can also occur in other diseases. For example, it is observed in capillary toxicosis, Verlgoff's disease, mesenteric vascular thrombosis. With a certain amount of blood loss, the patient develops pale skin, general weakness, dizziness, cold sweats. As a result of the intensification of the processes of digestion and putrefaction in the intestines, the abdomen relaxes (flatulence) and pain occurs.

See the table q ozone and 12 finger with intestinal diseases o g of Riga patients with clinical methods: (S ub y and timely weather ' timely)

Sub yektiv research: asking the question (interrogatio): the main complaints of patients - and more.

Anamnesis morbi

Anamnesis vitae

The syndromes observed in diseases of the gastrointestinal tract are as follows.

1 - Pain Syndrome: try to email settings, int e nsivligi, localization, irradiatsiyasi length, k e purified factors (usually dinner reception k e Games after 30-50 min) ;

2 - Dyspeptic syndrome: nausea, vomiting, haemotemesis, vomiting, eructatio, regurgitation, melena, diarrhea;

3 - Asthenovegetative syndrome: anorexia (loss of appetite), sore mouth, hypersalivation or dry mouth

4 - Gastrointestinal bleeding (hemorrhagic syndrome);

5 Decreased body mass;

Pains. Pain in the abdomen of the body is more disturbing in pathology of the stomach and duodenum. It usually spreads to the epigastric area and has a persistent or aggressive character. Most often, aggressive pains are associated with food intake and are seasonal in nature (spring and autumn). Depending on the time of onset of pain, the localization of the wound can be determined. Early and evening pains are different.

- **Early pain is** usually associated with gastric pathology. O vqat affect where the mass consumer after the injury and pain. They appear quickly after eating (usually after 30-60 minutes), lasting 1-1.5 hours. The mass in the stomach decreases after evacuation.

-Evening pains are associated with diseases of the 12-fingered intestine, and these pains are caused by the effect of hydrochloric acid contained in the gastric juice on the wound site. They appear after a meal (1.5–3 hours). There is also a distinction between hunger pains and night pains. The pain weakens or slows down after eating, which is due to the neutralization of the hydrochloric acid contained in the gastric juice with food. Patients complain of the following; complaints of pressure or pain in the epigastric area , indigestion. There is also flatulence in the abdomen as a result of increased gas production in the intestines.

Dyspeptic syndrome . Symptoms associated with dyspepsia-, digestive disorders (nausea, vomiting, heartburn, rest in the stomach, laughter, pain in the epigastric area and a feeling of heaviness in the intestines) are disturbed.

Causes of dyspeptic syndrome s : Be organic and functional .

Causes of organic dyspepsia: reflux esophagitis; wound diseases; gastritis; chronic pancreatitis; gallstone diseases; stomach cancer.

Functional dyspepsia takes the form of dyskinetic, nonspecific:

-Dyskinetic dyspepsia is characterized by the following symptoms: a feeling of rapid satiety of the stomach, discomfort in the epigastric area, pain may not be observed, nausea, poisoning from certain products, sometimes damage to the small intestine.

-Repetitive belching, abdominal rest, nausea, stress, characteristic for nonspecific dyspepsia.

Causes of **gastrointestinal bleeding** :

Wound disease

Gastritis and gastric erosion

Esophagitis or esophageal ulcer

Varicose veins of the esophagus (cirrhosis of the liver).

Mellori-Weiss syndrome is bleeding from the stomach as a result of rupture of the capillaries of the mucous membrane of the cardiac part of the stomach due to repeated vomiting and seizures. Gastrointestinal bleeding causes mixed vomiting of blood and melena. The color of the vomit mass depends on the amount of blood going out and how long it has been in the stomach.

Additional complaints:

Nausea, a feeling of discomfort in the epigastric area, is associated with damage to the stray nerve. In gastric diseases, nausea usually occurs with pain as well as frequent vomiting. Nausea occurs in many other cases as well. In pathology of the gastrointestinal tract is usually observed when eating.

Vomiting is a reflex act in which the products of the stomach and 12-fingered intestine esophagus, the oral cavity sometimes returning to the nose. This is due to the contraction of the abdominal muscles, the movement of the respiratory muscles. The pyloric sphincter is in a closed position. The vomiting reflex is associated with food intake, food stagnation in the stomach, pain sensation. The center of vomiting is located in the elongated brain.

-Morning vomiting. A large amount of mucus is characteristic of chronic gastritis (in alcoholism). Morning vomiting is characteristic of nocturnal hypersecretion if bile acid is retained. Vomiting after a meal (10-15min) is characteristic of cancer of the ulcer or cardinal part of the stomach, as well as chronic gastritis. -Vomiting in ulcers or stomach cancer occurs 2-3 hours after a meal.

In pyloric gastric ulcer or duodenal ulcer, vomiting occurs 4-6 hours after ingestion. Vomiting of food collected 1-2 days ago is characterized by accumulation of food in the stomach in this sphincter stenosis, and there is the smell of eggs in the sputum.

Vomiting mass character i :

-Qusiq mass have to be mixed in the blood . This means bleeding from the esophagus, gastrointestinal tract. This vomit mass is in the form of "dark coffee".

- If there is a putrid odor from the vomit mass, the food is stored in the stomach for a long time, which is typical for stenosis and atony of the pyloric part of the stomach.

Usually vomiting is observed in diseases of the stomach and esophagus, sometimes in addition to pathology of the gastrointestinal tract, in hypertension, poisoning, infectious diseases, increased pressure in the brain, encephalitis, vomiting center excitation. It is characterized by sudden onset of vomiting , absence of nausea and other dyspeptic symptoms, lack of relief and continuity after vomiting.

Medical history. When the symptoms of the disease begin, whether it is related to diet, whether it is seasonal (spring and autumn), the duration of pain, exacerbation, slowing (increases after eating in the stomach ulcer, in 12-finger ulcers postprandial dehydration and evening, nocturnal pain characteristic), the localization of pain is also of great importance in the diagnosis of diffuse or local character. In order to determine whether the patient has taken nonsteroidal anti-inflammatory drugs, past diseases (liver cirrhosis) bleeding from the gastrointestinal tract, past diseases and diseases recorded in the offspring are inquired. It is important to assess the nature of the vomit mass. The passage of the bowel with constipation or a mixture of liquid, dark (dextrose), blood, mucus and foam is an indication of intestinal diseases.

Life history. In collecting the anamnesis, it is necessary to assess the patient's nutrition (it is important to ask if the diet was disturbed, consumed more spicy, salty and dry foods). The main factor in diseases of the digestive organs is to determine whether the eating sequence is disturbed. It is necessary to determine what kind of food the patient consumed (fatty, fried, spicy, salty, spicy, spicy drinks (acetic acid)), how much. It is of

great importance in the diagnosis of diseases such as systemic diseases, diabetes, renal failure, diffuse toxic goiter.

Methods of objective examination: On examination - (inspectsio): On examination of the abdominal organs, the abdominal wall is studied in three layers, the epigastrium, mesogastric and hypogastric, and each layer is divided into three parts and a total of 9 divided into squares. This square, palpation and percussion, the members of such diseases is taken about **82 pictures** .

Attention should be paid to his condition when examining patients . In this case, the patient's condition can be active, passive and compulsive. The obligatory condition is observed when the pain in patients with ulcerative colitis is severe or in peritonitis (see Fig . 83 **a** and **b**) .

a 83 photo b

In patients - weight loss, even cachexia (in cancer and esophageal achalasia), dry skin, paleness (in esophageal cancer). The shape of the abdomen focuses on peristalsis. Scars on the skin, striae, pink stripes - in Cushing's syndrome, dilatation of subcutaneous veins (in cirrhosis or v. Cava inferior obstruction), swelling of the umbilicus (ascites, tumors), pink staining (in pancreatitis, ecchymoses (bleeding into soft tissues), around the navel - Kullen's symptom (photo 84), on the side walls of the abdomen - Gray Turner's symptom (inside or behind the peritoneum) in subcutaneous hemorrhage), asymmetry is observed in abdominal tumors. It is advisable to pay special attention to the participation of the anterior abdominal wall in the respiratory excursion when observing the abdominal level; subcutaneous venous anastomoses and postoperative scarring.

84 photos

Palpation - one-handed (mono manual) and two-handed (bimanual) palpation. On surface palpation, attention is paid to the tension (resistance) of the abdominal wall muscles, muscle tone, the absence of painful spots, pain in the epigastric area associated with peptic ulcer disease and gastritis, abdominal muscle tension. Deep palpation consists of four moments; 1 to place the hand on the abdominal wall, 2 to pull the skin to form a fold, 3 to enter, 4 to palpate. Palpation sequence-sigmoid colon, appendix, transverse ileum, ascending (descending) ileum, descending ileum, and stomach, are percussed in the same order. On deep palpation we identify the large and small curvature and pyloric portion of the stomach. Sometimes enlarged tumors are palpated. The condition of the bowel is assessed and pain is identified. **Percussion** (information is low and therefore rarely used) nevertheless in pylorostenosis positive symptom of tremor, liver, spleen volume, and tympanic sound in the gastrointestinal tract are detected. **Auscultation** kuldirashlarni, abdominal aortic aneurysms heard over the noise. Currently, physical examination methods are performed in a combined state. Such methods are of great importance in determining the diagnosis.

"Combination" methods:

1 - auscultoaffriction:

A phonendoscope is placed in the epigastric area and lin along the anterior wall of the abdomen using the fingers of the hand. in the median anterior projection, a sliding motion is made from the dagger-shaped tumor to the umbilicus. The lower border of the stomach is usually located at the point where the slippery sound subsides.

2 - auscultatory percussion

When this combination method is performed, the inspector’s fingers are not slid across the skin, but are lightly tapped.

Examination of gastric juice

Examination of gastric juice (secretory function of the stomach) is one of the main methods that allows to study the morphological and functional state of the gastric

mucosa, as well as to assess its evacuation function remotely. Probe and non-probe methods of examining gastric juice are common. Probing is a method of checking the secretory (amount of gastric juice) acid and enzyme production function (secretory function) of the stomach. To get an idea of the secretory function of the pancreas, it is necessary to probe with a special (thin) probe and suck the gastric fluid constantly or after 1 different time interval. In all cases, in the initial stage, intensive gastric juice is taken for 1 hour (every 15min) between its calm and digestive intervals, and it is called basal secretion. In the laboratory, in order to check the secretory activity of the gastric mucosa during digestion through a probe, patients are given subcutaneous drugs (histamine, pentagastrin,) that stimulate the production of syrup, which is formed in response to food. Chemicals that stimulate gastric secretion help to obtain pure gastric juice that is suitable for lab o rator examination. After the injection of histamine, the person being examined experiences redness that passes after a few minutes, a feeling of pressure and heat in the head. To prevent the above side effects, the nurse should administer diphenhydramine, suprastin, and other anti-allergy medications 20-30 minutes before intramuscular injection. At present, it is forbidden to take foods that stimulate gastric juice (caffeine, alcohol, cabbage broth, meat broth). Because they have a weak stimulating property, they also do not allow an objective assessment of the acid and enzyme production function of the stomach.

Method of probe examination of meda juice

Due to the fact that some patients are contraindicated in the probe method, the probe method is used.

This method includes desmoid test on Sali, ion exchange resin method, acidotest, gastrotest. These methods provide less data than probe methods.

Desmoid test on Sali. To do this, Methylene blue is placed in a small thin elastic rubber bag and this bag is tied with a catgut thread. The examinee swallows the bag at lunch and then makes breakfast. Once the sac enters the stomach, the catgut is digested under the action of hydrochloric acid and pepsin, and the sac opens. The methylene blue in the sac is absorbed into the blood and excreted in the urine. During the examination, urine is collected after 3-5 and 20 hours, and the time of urine staining (staining) intensity is determined. If the secretory activity in the medulla is normal, **1** portion is colorless, **2** portions are white-green and **3** portions are blue-green.

Ion exchange resin method. This method is based on the ingestion of ion exchange resin bound to a small molecular compound (quinine, papaaminosalicylic acid). The hydrogen of the hydrochloric acid in the meda juice is replaced by an indicator of ions, and the indicators are separated from the resin and absorbed in the intestine and excreted in the urine.

Probe-free examination of gastric juice (detection of uropepsin in urine, desmoid probe, ion exchange test-acidotest) is of only guiding importance. This method provides only approximate information on whether gastric secretory function is preserved or not. Quantitative indicators of gastric secretory activity cannot be determined without a probe. The probe-free screening method only allows a limited number of people to be screened en masse. It is not advisable to examine patients with diseases of the stomach, duodenum and other digestive tract in a polyclinic, inpatient setting without a probe. **Preparation of patients t :** a light lunch should be made at 8 pm the day before the examination. Breakfast, fluids and medications will not be taken on the day of the test.

Determination of acidity of meda juice by means of acid test without a probe

The acidotest test method is used as a diagnostic tool to determine the acidic environment in the gastric juice. To do this, the patient is given a portion of urine in the morning. To perform an acid test, the patient empties the bladder and this portion is not taken for analysis. After emptying the bladder, the patient takes Hungarian-made 2,4-

diamino-4-ethoxy-azobenzene 3 dragees at a dose of 0.05 and 2 tablets at a dose of 0.2 caffeine benzoate sodium. Along with the tablets, the package contains a colored scale **(A-free hydrochloric acid is present and B-free hydrochloric acid is not present)** to evaluate the acidotest results, and is evaluated accordingly.

The patient takes 2 tablets of caffeine benzoate sodium after emptying the bladder, and after 1 hour the bladder is emptied and analyzed in the container and the container is labeled as the control group. After emptying the bladder a second time, the patient receives 3 dragees of 2,4-diamino-4-ethoxy-azobenzene , and 1.5 hours later, the patient is re-analyzed and collected in a container, and 1, It is set to 5 hours. The urine obtained in the control group and after one and a half hours is diluted with up to 200 ml of water, and an acidic medium is formed with hydrochloric acid. In addition, 5 ml of the solution is taken and the color of the urine in each solution is assessed.

In this case, the urine in the control group does not change color as usual.

An hour and a half urine is stained red (aliy). The staining intensity is of diagnostic importance and is determined by comparing it with a color scale to determine the acidity of the gastric juice.

Caffeine benzoate is used to stimulate the secretion of sodium meda juice.

Ion exchange resin dragee, saturated with 2,4-diamino-4-ethoxy-azobenzene. 2,4-diamino-4-ethoxy-azobenzene is a dye that forms a bond with ion exchange resin. Meda juice is squeezed out in an acidic environment under the influence of hydrogen ions, does not form a bond with the ion-exchange resin. The dye is absorbed into the fatty intestine and excreted in the urine. Depending on the intensity of the color of the urine, the acidity of the gastric juice is determined.

* In the anacid state, there are almost no hydrogen ions and the dye is not squeezed out of the ion exchange resin. The amount of dye absorbed and excreted in the urine is low. Therefore, the color of the urine corresponds to section " V " on a color scale .

* In the hyperacid state, due to the abundance of hydrogen ions, it completely displaces the dye from the dragee. Excessive absorption and excretion of the dye in the urine increases the intensity of urine color falls into the "A" category.

* In the case of normatsid, the color of urine is between hyperacid and hypoacid.

Examination of serum gastrin. Radioimmune testing of gastrin for serum gastritis in diseases of the stomach and duodenum is of diagnostic value. Physiologically, the amount of gastrin in the serum is 100-200 ng / l. Its increase above 600 ng / l (hypergastrinemia) is observed in Zollinger-Ellison syndrome (gastric and duodenal ulcers accompanied by pancreatic adenocarcinoma) and pernicious anemia.

Detection of Helicobacter pylori. Helicobacter pylori (Fig. **85**) is detected by examination of material obtained by biopsy of the stomach (bioptat is obtained using FEGDS). The Ureaz test is handy for testing. In this case , the bioptat is applied to a gel containing machevina. If you have Helicobacter pylori, the color will change after a few minutes. Helicobacter pylori is difficult to study in culture and cannot be tested serologically.

Biopsy of the gastric mucosa and histological examination .

This screening method is used to determine if there is a tumor. Tissue is taken from several places for inspection. In 80-90% of cases the diagnosis is correct. Recently, this method has been used to detect Helicobacter pylori. Bioptat examination not only helps to identify the microorganism in a timely manner, but also to identify morphological changes (eg, inflammation, atrophy, metaplasia.)

X-ray examination

X-ray and radioscopic methods are used to examine the stomach. An approximate assessment of gastric motility can be made by radioscopic examination. The patient is given a light meal in the evening to prepare for the examination, and a cleansing enema is given that morning. The examination is performed on an empty stomach and in a vertical

position. Barium sulfate is used as a contrast agent. The examination begins with the study of the relief of the gastric mucosa. Depending on the stage of digestion, the folds of the stomach vary widely (folded or flattened). The area where the direction of the folds is disturbed is a pathological lesion. An important component of the examination is the study of the contours of the stomach. The shadow of a permanent depression in a known place is referred to by the term “shelf” (a typical sign of a stomach ulcer), the symptom of the index finger can be identified See Fig . 86 .

A contrast with the stomach area of the stomach called the deffekti (tumor). **86 photos Fibroesophagogastroduodenoscopy.** Gastroduodenoscopy has developed intensively since the use of fiber optics, and this method is a method that provides quick and accurate information on diseases of the stomach and duodenum. Using this method, a biopsy can be obtained for morphological examination. One of the advantages of this method is that local treatment of this prolonged bleeding can also detect changes in the mucous membrane that cannot be detected on X-ray examination.

When a radiographic examination reveals a gastric ulcer, an endoscopic examination should be performed to histologically confirm that the tumor has not become ulcerated. Endoscopic examination is the main indication when gastric tumor is suspected (as well as loss of body mass, anemia). However, endoscopic examination, unlike X-ray examination, in some cases does not allow the detection of gastric tumors. For example, an infiltrative growing gastric tumor cannot be detected because it almost does not change the integrity of the mucous membrane. It is convenient to take a biopsy on endoscopic examination and burn bleeding wounds.

Practical training 2

Main clinical syndromes: indigestion syndrome, intestinal absorption syndrome, acute abdomen, acute bleeding from the digestive system. Coronavirus diagnostics, differential diagnosis, course

1. Practical training module .

Training time - 4 hours	The number of students 1 : 0 to 12
Form of training	Practical training
Practical training plan	A digest ' Zola instrumental methods (radiological , rector of the ezofagogastroduodenofibroskopiya - manoskopiya , colonoscopy). Symptomatology of gastritis (acute and chronic).
The purpose of practical training :	Methods of instrumental examination of digestive organs (radiology, esophagogastroduodenofibroscopy, recto-manoscopy, colonoscopy). Study of the symptomatology of gastritis (acute and chronic).
Teaching style	Inquiry. Demonstration of patients, interactive teaching methods, practical skills.
Form of teaching	In small subgroups.
O ' unit equipment	Calls to O Training Guidebook , practical Typo content , projectors , computer .
Training mode	Methodically equipped auditorium.
Monitoring and evaluation	Oral control: questions and answers, tests, problem solving.

1.2 . ***Technological card of practical training***

See the stages of hearts and q ti.	Educator	Learners
In the preparatory stage of q	4. See the audience to control the purity karîm 5. Checks students ' readiness for training 6. And control q karîm	
1. bullet and training g contain levels of intra q (At 10 p.m.)	1. Preparation of educational content on the topic. 2. Preparation of presentation slides for the introductory speech 3. Develop a list of references used in the study of science	
See the 2 main stages diarrhea (160 da q i q a)	1. Divide students into small groups and ask questions on the topic. 2. Uses display posters 3. Uses slides, multimedia 4. Conducts treatment 5. Summarizes and summarizes the information provided on the basis of topics, encourages and actively evaluates the active participant students	They are divided into small groups They watch q participate They listen and answer questions
2- final press q ich (10 da q i q a)	1. Concludes 2. Provides independent work 3. Gives homework	He listens Takes notes Takes notes

3. Assessment of students' theoretical knowledge:

A) Frontal method:

1. 1 Understanding the methods of instrumental examination of digestive organs
2. What is X-ray and its importance
3. What is radiography and its diagnostic value
4. Preparing patients for radiography
5. Contraindications to X-ray and X-ray
6. Guidelines for X-ray and X-ray
7. What is EGDFS
8. Diagnostic significance of EGDFS
9. EGDFS technique
10. EGDFS guidance and contraindications
11. What is the diagnostic value of rectoromonoscopy
12. Preparation and techniques of patients for rectoromonoscopy
13. Indications and contraindications to rectoromonoscopy
14. What is the diagnostic value of colocoscopy
15. Indications and contraindications to colocoscopy
16. Describe gastritis
17. Etiopathogenesis of acute and chronic gastritis
18. Classification of chronic gastritis

19. Diagnostic methods of chronic gastritis
20. Clinic, course, prevention of chronic gastritis

Beehive ”method

In this method, the problem is analyzed with the whole group or with two small groups. The assigned task can be assigned to one or two different groups for the whole group. Within 10-15 minutes, group participants analyze the task solution and report to each other. The best option will be selected from them.

Practical training 3

Diseases of the digestive system: esophageal diseases esophagitis, gastritis clinic, diagnosis .

1. Practical training module .

Training time - 4 hours	The number of students 1 : 0 to 12
Form of training	Practical training
Practical training plan	GERB symptomatology . Modern diagnostic methods Diseases of the liver and the ' T ' or ways to query the MV ' sa ' Lord of the patients in Riga - (shikoyatkari history), making at least - to view the display , acoustical and perkussiyasi . The basic concept of the clinical syndromes : jaundice , portal hypertension , liver failure .
The purpose of practical training :	Introduction to GERB. Interrogation of patients with liver and biliary tract diseases (complainant, anamnesis), examination, palpation and percussion. Understanding the main clinical syndromes: jaundice, portal hypertension, liver failure.
Teaching style	Inquiry. Demonstration of patients, interactive teaching methods, practical skills.
Form of teaching	In small subgroups.
O ' unit equipment	Calls to O Training Guidebook , practical Typo content , projectors , computer .
Training mode	Methodically equipped auditorium.
Monitoring and evaluation	Oral control: questions and answers, tests, problem solving.

1.2 . ***Technological card of practical training***

See the stages of hearts and q ti.	Educator	Learners
In the preparatory stage of q	1. See the audience to control the purity karîm 2. Checks students ' readiness for training 3. And control q karîm	
1. bullet and training g contain	1. Preparation of educational content on the topic.	

levels of intra q (10 da q i q a)	2. Preparation of presentation slides for the introductory speech 3. Develop a list of references used in the study of science	
2 - the main stages of diarrhea q (160 da qiq a)	1. Divide students into small groups and ask questions on the topic. 2. Uses display posters 3. Uses slides, multimedia 4. Conducts treatment 5. Summarizes and summarizes the information provided on the basis of topics, encourages and actively evaluates the active participant students	They are divided into small groups They watch participate They listen and answer questions
2- final press q ich (10 da q i q a)	1. Concludes 2. Provides independent work 3. Gives homework	He listens Takes notes Takes notes

3. Assessment of students' theoretical knowledge:

A) Frontal method:

"Snowflake" method

Two groups of students discuss a problem or situational problem in order to find a more accurate answer. For example, the differential diagnosis of diseases associated with symptoms of acute cholecystitis, or abdominal pain syndrome. Each correct answer is evaluated by one point and one piece of snow is given to this group. The team members who collect the most snowflakes are rated with excellent grades.

Esophagitis

Esophagitis is an inflammation of the mucous membrane of the esophagus that is accompanied by dysphagia, odenophagy, and a number of other symptoms. Usually primary and secondary esophagitis are distinguished. If the primary is direct esophageal injury, the secondary is the esophagus affected as a result of these other internal organ diseases, these diseases include systemic diseases (systemic scleroderma, systemic lupus erythematosus, dermatomyositis), anemia (iron deficiency anemia), Shagas disease (esophageal dilatation (American trypanosomiasis)), eosinophilic esophagitis (chronic autoimmune disease with esophageal dysfunction and inflammation of the esophageal walls) disease with eosinophilic infiltration (which develops after food allergy), gastroesophageal reflux disease, which develops if there is persistent vomiting in alcoholics. Primary esophagitis may be very rare.

Etiology - Constant consumption of raw food, excessive consumption of hot or cold foods, consumption of acetic acid or others as suicide.

Acute and chronic esophagitis are distinguished.

Depending on the nature of inflammation, the following types of acute esophagitis are distinguished:

Catarrhal;
Fibrinosis;
Fl e gmonoz;
Wounded;
Gang romantic e

Necrotic

Erosive

There are 3 levels of acute esophagitis. Grade I - the surface of the esophagus is damaged, there are no wounds and erosions.

Grade II - crossing of the mucous membrane of the esophagus with necrosis and wound defects.

Grade III - subcutaneous esophageal mucosa is damaged, with deep defects and bleeding.

Clinic of esophagitis - heartburn, sour throat, odenophagia (painful swallowing), difficulty swallowing, irritability behind the chest, hiccups (nausea), nausea, vomiting, feeling of foreign body behind the chest symptoms such as the appearance of irritating.

The diagnostic value of palpation, percussion, and auscultatory examinations is less informative.

Diagnostics. Basically, radiological and endoscopic examination methods provide sufficient information in the diagnosis. On X-ray examination, the patient consumes liquid snacks in the evening before the examination and arrives at breakfast in the morning. Before examining the patient, 200 ml of barium sulphate is ingested and monitored while drinking X-rays. Changes in the mucous membrane of the esophagus, narrowing or enlargement of the esophagus, shifting it to one side or another (hypertrophy of the left ventricle, tumors of the thoracic cavity, excessive enlargement of lymph nodes around the trachea) We can see. At the endoscopic examination, patients are mentally and physically prepared. In spiritual preparation, patients are explained that endoscopic examination is absolutely harmless to health and painless, leaves no complications for the patient, and is very important in diagnosis. In physical preparation, the patient consumes a liquid snack in the evening before the examination and arrives at breakfast in the morning, gastric lavage by siphoning before the examination. Dikain or lidocaine is injected into the throat, larynx, and throat to reduce vomiting reflexes 10 minutes before examination. The endoscope is then inserted into the esophagus. The mucous layer of the esophagus becomes red and swollen in esophagitis, burns, in catarrhal inflammation can also be seen white coating on the mucous membrane. Occasionally, on endoscopic examination of the esophagus, we may also see polyps, esophageal cancer, narrowing or enlargement. Examination methods such as esophageal monometry (determination of esophageal sphincter tone), impedanceometry examination method (determination of esophageal muscle activity) and daily pH monitoring (see above) are performed. Determines the pH value from 4 to more than 7 and the duration. It also determines its association with subjective symptoms. (Food intake, body condition, medication intake, smoking) provides control over the effectiveness of the use of drugs and the possibility of individual therapy.

Chronic esophagitis

Chronic esophagitis esophagus by a variety of factors, chronic irritation
develops as a result of (alcohol, hot food or Dagali consumer), circulatory disorders, and so on a separate form (r e gurgitatsiya), r e flyuks esophagitis, etc.

According to Savari-Miller's classification, 4 levels of chronic esophagitis are distinguished. Grade I - there is redness in the distal areas of the mucous membrane of the esophagus.
Level II - small erosions are added, but not all parts of the mucous layer are damaged.
Grade III - the lower third of the esophagus is accompanied by a wound injury.
Grade IV - chronic ulcers appear and the esophagus narrows.
See clinic, diagnosis acute esophagitis.

Esophageal cancer

It mainly develops in the middle and lower part of the esophagus. Although the causes of esophageal cancer are unclear, it is often the result of excessive consumption of hot foods and beverages, constant exposure to the mucous membranes, constant consumption of alcohol, smoking, radiation, Barrett's esophagus, previous diseases esophageal cancer will bring. The disease is accompanied by injury to the mucous membrane, which spreads around the esophagus and narrows it, making it difficult for food to pass from the esophagus to the stomach, disrupting the evacuation function. The disease rapidly metastasizes to surrounding regional lymph nodes.

Clinic. Dysphagia, which is exacerbated in patients due to narrowing of the esophagus, is also difficult to swallow, first solid and then liquid food. Patients experience pain when swallowing, which occurs under the chest. These pains are given to the neck, back and spine area. This is because the tumor occupies the entire circumference of the esophagus. In the esophagus, bleeding is also observed from areas where the tumor has grown. When we examine patients, we can see that they are losing weight. Skin tension decreases. X-ray examination and endoscopic examination methods are used for diagnosis. At the time of endoscopic examination, a biopsy is taken from the tumor area to obtain material for morphological and histological examination, and atypical cells are identified.

Pathological anatomy. Macroscopic forms:. An annular solid konsist e ntsiyali cancer; Sucker cancer; We can see wounded cancer. Microscopic forms. Esophageal cancer often has a flat-cell frozen or non- frozen structure. If the esophagus develops from b e s - ad e nokartsinoma structure. M e tastazylasy - lymphog e n way.

Complications. Associated with the growth of adjacent organs - trax e ya, thoracic cavity, pl e vra. The Trax e fistulae, aspiratsion pn e vmoniya, lung Abst e SSI and the Gang email , utensils, PL e vra empi e Christ, purulent m e Dias e nit develop. Esophageal cancer is very early kax e ksiya k e is located.

The cure. In the initial stages, surgical treatment is effective, in the evening stages, light and chemotherapy methods (fluorouracil, cisplatin) are used.

Acute gastritis

Gastritis is an acute inflammation of the gastric mucosa . Morphological and funk t Zion with atrophy of the building and developing a chronic form can be .

Etiology. This includes rough and incompletely crushed low-quality food, contamination with various microorganisms (Helicobacter pylori, staphylococcus, salmonella), the use of drugs primarily aspirin and other nonsteroidal anti-inflammatory drugs (NYQD), ethanol and exogenous factors such as exoallergens, excessive hot food intake, and ingestion of toxic substances play an important role. Endogenous factors are also important in the origin of gastritis. These occur after respiratory, renal, hepatic and heart failure, as well as systemic diseases. NYQD is associated with inhibition of prostaglandins, which often results in acute erosive gastritis. Acute drinking can also be caused by stress, as a result of allergies to certain foods, burns, trauma, and surgery.

The origin of erosive gastritis is accompanied by a violation of blood circulation in the gastric mucosa, the above causes play an important role.

Inquiry. Acute catarrhal gastritis is characterized by pain in the epigastric area, dyspeptic syndrome (nausea, vomiting mixed with food, mucus and bile), and sometimes subfebrile fever. Symptoms appear after 6-8 hours under the influence of pathogenic factors in the gastric mucosa. Acute erosive gastritis can be complicated by bleeding of various appearances and duration (hence this is a dangerous form of acute gastritis). Mild pain or discomfort on palpation of the epigastric area of the patient is accompanied by a tympanic sound when percussion is performed. X-ray examination provides little information. The diagnosis is confirmed by FEGDS, in which a large number of erosions and hyperemia are detected. Diffuse hyperemia in the gastric mucosa is usually observed when FEGDS is used. We can see atrophy or hypertrophy of the mucous membrane in the later stages. The folds of the gastric mucosa may thicken or flatten.

Control methods; radiologist, endoscopy, laboratory examination methods.

Chronic gastritis

Chronic gastritis is a chronic inflammation of the gastric mucosa, which is a disease characterized by structural reconstruction and developing atrophy, impaired motor, secretory, evacuatory and other functions.

Etiology. Mainly exogenous and endogenous factors play a role in the origin of chronic gastritis. **Inadequately** chewed foods that are susceptible to **exogenous factors** ; ethanol and a number of other substances; nonspecific anti-inflammatory drugs; Helicobacter pylori; acid and alkali vapors; radiation.

Endogenous factors; Grass reflux; Increased urea in the blood; Hypoxia of the gastric mucosa as a result of cardiovascular insufficiency; Disorders of the microcirculation of the meda mucosa;

In diagnosis, the endoscopic method provides accurate information and biopsy material is obtained for histological examination. In the early stages of the disease, superficial disorders of the mucous membrane are accompanied by lymphocytic and plasma infiltration, and in the late stages, the mucous glands are damaged, and eventually gastritis develops, the mucous membrane atrophies, wrinkles are reduced. Among the etiological factors are primarily Helicobacter pylori, as well as non-microbial factors (alcohol, NYQD, exposure to chemical agents, bile reflux, drugs) and autoantibody production.

Depending on the secretory function of chronic gastritis ;

Chronic hypoacid and anacid gastritis;

Chronic hyperacid gastritis;

Depending on the morphological change;

Chronic atrophic gastritis;

Chronic hypertrophic gastritis (Menetre's disease 1888);

Erosive gastritis;

Chronic antral (rigid) gastritis

Inquiry; Less symptomatic disease : 1 - pain syndrome: nature, intensity, localization, irradiation, duration, causative factors (usually 30-50 min after a meal); clearly not localized, scattered.

2 -Dyspeptic syndromes: rest of the epigastric area, which is associated with food consumption; belching, nausea, vomiting, loss of appetite, bloating, bloating, flatulence, irregular diarrhea (nausea, vomiting, haemotemesis, eructatio, regurgitatio, melena) ; .

3 AST e Nov e g e syndrome: the taste of pomegranate e ksiya, irritation of the mouth, GIP e rsalivatsiya or mouth;

The clinical manifestation of high or normal secretory function in gastritis is characterized by insufficient production of acid and pepsin, which are formed in gastritis. - In gastritis with **normal** or high **secretory function** , heartburn, sour gut, aggravation of the epigastric region after eating and the presence of dull stabbing pain (sometimes "starvation" or "evening ”Pains), prone to constipation. - In gastritis

with **insufficient** secretion are characterized by: blunt pain and swelling in the epigastric area, nausea, loss of appetite, unpleasant taste in the mouth, smelly gut, laughing, prone to diarrhea. In addition, there are signs of hypovitaminosis (dry skin, changes in nails) and sometimes dumping syndrome (weakness after eating, excessive sweating, dizziness, rapid heartbeat). **Changes in the oral cavity .** Examination of the oral cavity of patients reveals bleeding from the gums, thickening of the tongue, redness, flattening of the nipples, the presence of traces of teeth around it, the development of chelos and angular stomatitis, as well as dry skin.

Instrumental verification methods

The final diagnosis is often a histological examination of the FEGDS or also biopsy material. The most important is the detection of Helicobacter pylori (can be treated with eradication therapy). Gastric secretory function is determined by fractional probing of the stomach with the use of stimulants of gastric secretion (histamine, pentahistamine). Decreased serum gastrin levels are mainly detected when the antral area of the stomach is injured.

Treatment. Mild chronic gastritis usually does not require a special course of treatment. Adherence to the diet is prescribed, it consists of light digestible food and should not take alcohol and NYQD. -B12 as needed, as well as hydrochloric acid with gastric juice or food. -If Helicobacter pylori is detected, eradication therapy (trixapol, metronidazole, etc.) is performed. No-spa, platiffilin for pain relief; Aloe 1.0 to improve reparative processes; festal, mezim, creon, etc. to improve digestive processes; vitamin therapy is carried out. **Gastropathy of nonsteroidal anti-inflammatory drugs**

In 1986, the term NSAID-gastropathy (nonsteroidal anti-inflammatory drug (NYQD) -gastropathy) was proposed by SN Roth. This is an erosive wound injury of the gastroduodenal zone, associated with the consumption of these drugs and is characterized by a clinical endoscopic appearance.

Pathogenesis. Modern understanding of the pathogenesis of NYQD -gastropathies is based on the concept of cyclooxygenase (TSOK, prostaglandin synthetase). However, the main role of these agents in the early days of NYQD therapy depends on local traumatic effects. Most NYQDs are weak organic acid derivatives that do not ionize in the acidic environment of the stomach and pass through the hydrophobic membrane of epitheliocytases. At the same time there is a local lesion of the mucous membrane - the phenomenon of adaptation is prescribed for short-term semi-excretory drugs (voltaren, diclofenac, ibuprofen): they are not expressed by prostaglandins, but by growth factors, and adaptation to early NYQD -gastropathy is spontaneous tends to run out. When the constitutional isoform TSOK-1 is inhibited, NYQD causes severe gastroduodenal injury, resulting in endoscopic and clinical lesions. In prostaglandin 12 deficiency, microcirculation in the gastric wall deteriorates, which is an important pathogenetic link in NYQD -gastropathy. A decrease in prostaglandin E2 synthesis reduces bicarbonate and mucus secretion, an increase in acid production increases the imbalance of protective and aggression factors. It has ultrogenogenesis and is explained by the strong prophylactic effect of antisecretory and gastroprotective substances. Localization of NYQD-gastropathy is most often in the antral part of the stomach. Finally, secondary adaptation of NYQD occurs in the gastroduodenal zone (the phenomenon of prostaglandin gastroprotection). After 3-4 months, the risk of receiving NYQD-gastropathy decreases.

Practical training 4

Y ara, mal`absorbtsiya syndrome, chronic colitis disease diagnosis .

1. Practical training module .

Training time -	The number of students 1 : 0 to 12

4 hours	
Form of training	Practical training
Practical training plan	Stomach and duodenal ulcer disease is symptomatic - 12 logiyasi . Symptomatology of enteritis and colitis. Lab: Laboratory examination.
The purpose of practical training :	The concept of gastric and duodenal ulcers, enteritis and colitis.
Teaching style	Inquiry. Demonstration of patients, interactive teaching methods, practical skills.
Form of teaching	In small subgroups.
O ' unit equipment	Calls to O Training Guidebook , practical Typo content , projectors , computer .
Training mode	Methodically equipped auditorium.
Monitoring and evaluation	Oral control: questions and answers, tests, problem solving.

1.2 . ***Technological card of practical training***

See the stages of hearts and q ti.	Educator	Learners
In the preparatory stage of q	7. See the audience to control the purity karîm 8. Checks students ' readiness for training 9. And control q karîm	
1. bullet and training g contain levels of intra q (10 da q i q a)	1. Preparation of educational content on the topic. 2. Preparation of presentation slides for the introductory speech 3. Develop a list of references used in the study of science	
See the 2 basic levels of diarrhea (160 da q i q a)	1. Divide students into small groups and ask questions on the topic. 2. Uses display posters 3. Uses slides, multimedia 4. Conducts treatment 5. Summarizes and summarizes the information provided on the basis of topics, encourages and actively evaluates the active participant students	They are divided into small groups They watch participate They listen and answer questions
2- final press q ich (10 da q i q a)	1. Concludes 2. Provides independent work 3. Gives homework	He listens Takes notes Takes notes

3. Assessment of students' theoretical knowledge:

A) Frontal method:

1. Factors to be analyzed in the etiology of wound disease
2. Pathogenesis of gastric and duodenal ulcers
3. Modern Theories in the Occurrence of Wound Disease
4. Classification of wound disease
5. The main causes of wound disease
6. Results of palpation examination in gastric and duodenal ulcers
7. Functional examinations in the diagnosis of wound diseases
8. Importance of laboratory examination methods in wound diseases
9. Wound Disease Clinic
10. Clinical features of wound diseases
11. Changes in gastric function depending on wound localization
12. The importance of radiological examination in wound disease
13. The role of EGDFS in the diagnosis of ulcer disease
14. Complications and prevention of wound disease
15. Modern methods of treatment of wound disease
16. Etiopathogenesis of chronic enteritis
17. Classification of chronic enteritis, clinical course, diagnosis
18. Etiopathogenesis of chronic colitis
19. Clinical manifestations, diagnosis of colitis
20. Coprological examination: macroscopic, microscopic and chemical

Brainstorming method

Basic rules :

- Not to mention the shortcomings that hinder the emergence of the idea
- The height of ideas and thought, because the more unusual an idea, the better it is
- Accept many offers
- Combination of ideas and their development
- Present the idea succinctly without argumentation
- Divide the group into two: thought generators and thought analysts

This method allows you to argue ideas and opinions, your own personal opinion, to find the optimal solution in any situation.

Gastric or peptic ulcer:

12 gastric and duodenal ulcer disease is a chronic body and t e h t e z r e tsidivlovchi a common disease, the mucous membrane (87 photos) linked to the d e f e ktlar appear to be feasible.

The appearance of the ulcer depends on the acid content of the gastric juice. **The** integrity of the mucous membrane is compromised by the storage of hydrochloric acid and pepsin in the gastric juice. Ulceration of the mucous membrane is a major cause of dyspeptic syndrome and prolonged pain in the abdomen. The disease is smooth and seasonal (spring and autumn).

12-finger ulcers are more common among ulcers. **Symptomatic (secondary) ulcers of the gastrointestinal tract also occur.** These include nonsteroidal anti-inflammatory drugs in thyrotoxicosis, Zollinger-Ellison syndrome (pancreatic adenoma, ulceration with gastrinoma) caused by Cushing's disease and post-stress, some cardiovascular, respiratory, and kidney diseases. , Can include wounds caused by curling (burns). Symptomatic wounds are different from true wounds. Symptomatic ulcers are not seasonal in nature and are numerous, linear in shape, up to 50% of these wounds are at risk of bleeding, mainly in the 12-fingered intestine, and the underlying disease must be treated.

On the location of wound diseases; Medicinal **ulcers -** cardiac, subcardial, body part, antral part, pyloric part. **12-finger intestinal ulcer;** Bulbar, postbulbar wounds occur. There may be one or more wounds depending on the number of wounds. Wounds; sensitivity period; scarring period; remission period varies;

Etiology. Wound development is characterized by a disturbance of the balance between protective and aggression factors that ensure the integrity of the mucous membrane. Genetic predisposition is also important in the etiology of ulcer disease. There is no definite information about the transmission from generation to generation. Causes of ulcer formation may include the following.

Infectious theory was discovered in 1983 by Australian scientists Helicobacter pylori (B. Marshall, A. Warren) (Fig. 63), neuropsychic, alimentary, harmful factors, drug factors also play a role

Reproduction of pepsinogen Peptic - 1856-1878 (C. Bemard, H. Quincke) ;
After inflammation (gastritis) -1817-1923 (F. Uden and GE Konjetzny),
Vascular theory (Vascular - 1852 (R. Virchov)),
Stress -1953 y (Seiye),
Acidic peptic factor - 1956 (LR Dragstedt), G cell hyperfunction;
Cortico-visceral - 1949 y (kM Bikov, IT Kurtsin),
Traumatic - 1912 (L. Aschoff),
I (0) blood group;
Alpha antitrypsin deficiency;
Alpha macroglobulin deficiency;
Mucoprotein deficiency;
Wounds are more common in people who have the B5, B15, B35 HLA antigen.

87 photos

Pathogenesis. The development of the disease lies in the imbalance between aggressive and protective factors. As the strength of the aggressive factors increases, the protective factors also increase or weaken the compensator. Aggressive factors and gastroduodenal hypermotor dyskinesia rapidly evacuate food and gastric juice from the stomach to the 12-fingered intestine, and the hydrochloric acid contained in the gastric juice affects the mucous membrane.

The following table lists the aggressive and protective factors

	Aggressive factors	Protective factors
1	Hydrochloric acid in meda juice	Mucus formation in the stomach
2	Pepsin in meda juice	Production of bicarbonates
3	Gastrin in meda juice	Microcirculation of the mucosa be enough
4	Nosteroid anti-ulcer drugs	Prostoglandins in the mucous membrane enough
5	Helicobacter pylori	Regenerative condition of the mucous membrane
6	Bile acids	Protects the immune system
7	Impaired motor evacuation function of the meda and 12-fingered intestine	Resistance of the mucous membrane to aggressive factors;

Increases the production of gastrin, pepsin and hydrochloric acid, which **are** synthesized in **G cells** , leading to the formation of an aggressive factor. Gastrin

synthesis is enhanced by vagus cholinergic (stray nerve) enhancement (stimulation), medullary antral elongation, partial digestion of proteins, bile fluid, catecholamines, alcohol, and medial antral alkaline environment. Conversely, its synthesis is reduced by a decrease in pH in the antral part, cholinergic blockade, secretin, somatostatin, prostaglandins, and vasoactive interstitial peptides. Stray nerve parietal cells act on M cholinergic receptors to enhance hydrochloric acid synthesis, also by hypersecretion of gastrin. **Histamine** antral part of the medal is released from fat cells (lamina propria) and impact.

Histamine stimulates gastrin or cholinergic mediators, acid production of parietal cells. This confirms the therapeutic effect of H_2-histamine receptor antagonists (cimetidine, ranitidine, and bq). AKTG increases gastric secretion and reduces mucus in the gastric mucosa, thereby increasing the effect of aggressive factors.

Factors that reduce meda secretion.

Increases the production of mucus secretion in the C cells of the duodenum and in the proximal part of the small intestine, reduces the production of gastrin and hydrochloric acid, reduces smooth muscle. Somatostatin, produced in D cells of the antral part of the stomach and pancreas, inhibits gastrin production. Vasoactive interstitial peptides produced in small intestinal cells as well as large amounts of fats and carbohydrates slow down gastric production.

Protective factors include the following (see table above).

Factors that reduce the protective function of the medulla mucosa .

Hydrochloric acid, pepsin, bile acid.

In the pathogenesis of the disease lies a decrease in the resistance of the mucous membrane, in addition, the release of bile acids into the stomach as a result of weakening of the pyloric sphincter is also important. Acid-peptic factor plays a key role in duodenal ulcer disease. Due to gastroduodenal reflux, bile acids entering the stomach increase the permeability of the gastric mucosa to hydrogen ions, which increases the formation of hydrochloric acid in the stomach, ie increases the acidic environment in the stomach.

Helicobacter pylori is located in the mucous membrane, between the villi, in the intercellular space, produces many enzymes urease, protease, NO synthetase, has a direct cytotoxic effect, activates stem cells in the stomach, enhances the production of hydrochloric acid and pepsinogen-1, causes local inflammation, reduces the growth and regeneration of cells in the mucous membrane, slows the movement of hydrogen ions in the stomach, and reduces the protective barrier of the mucous membrane.

Spasm of arterioles and capillaries in the mucous membrane, dilation of venules leads to the development of hypoxia in the mucous membrane. It slows down metabolic processes, slows down the regeneration process, increases cell permeability. Adrenaline and noradrenaline activate adenylate cyclase, disrupting the activity of lysosomes of the gastric mucosa by increasing the amount of tsAMF in the cells of the stomach and duodenum. IgA deficiency and microcirculation leading to ischemia also lead to decreased mucosal resistance

Factors that increase the protective function of the medulla mucosa .

Prostaglandin E enhances the formation of bicarbonates in the mucous membrane, has a cytoprotective effect, improves microcirculation in the gastroduodenal mucosa. They play a central role in protecting the mucous membrane epithelium from aggressive factors. The key enzyme in the synthesis of prostaglandins is cyclooxygenase (TsOG), which occurs in 2 forms in the body. TsOG-1 and TsOG-2

TsOG-1 - it is found in the stomach, kidneys, platelets, endothelium.

Induction of TsOG-2 occurs as a result of inflammation, the production of this enzyme is carried out by inflamed cells.

The clinical effect of NYQD is associated with a decrease in TsOG-2 and also a decrease in TsOG-1 as a side effect. This leads to the development of gastropathy, which is

close to ulcerative colitis, accompanied by a violation of the protective layer of the gastric mucosa. There is great hope for selective inhibitors of TsOG-2, which are currently effective and safe in inflammatory disease. Nosteroid anti-inflammatory drugs reduce the synthesis of prostaglandins.

Endogenous opiates reduce the amount of gastrin and somatostatins and have antispasmodic effects. Unpleasant factors in the development of ulcers, of course, also serve external factors: stress, dietary disorders, smoking.

Clinical course. Clinical symptoms of ulcer disease are associated with the localization of the wound, its depth and the presence of additional gastritis, gastroduodenitis. The disease is characterized by sensitivity and remission, mainly in autumn and spring.

Characteristic symptoms are abdominal pain and dyspeptic symptoms associated with eating.

- The pain is localized in the epigastric area, if it appears soon after eating, it is characteristic of wounds of the cardiac part of the stomach, and patients are afraid to eat. The pain radiates to the waist, spine (lumbar region), which is more typical when the wound is on the back wall of the stomach. Pain that occurs when eating - early (30-40 min after a meal, which is typical of peptic ulcer disease). At the onset of pain, vomiting with an acidic stomach mass is observed, after which the pain subsides rapidly, the patient feels relief. Appears in the evening (3-4 hours after a meal and at night pains, typical of 12-finger intestinal ulcers). Pain syndrome reduces the consumption of a substance that reduces acidity (e.g. milk) after a meal, which has a seasonal (spring, autumn) character.

- Exacerbation of ulcer disease (especially duodenal ulcer) is often accompanied by constipation associated with impaired motility of the colon (in 5% of patients this may be the main symptom).

- Boiling, wheezing may be observed.

When viewed, the tongue is covered with a look. On palpation of the abdomen, the muscles may be slightly tense, and pain may be detected in the epigastric area or in the area of the 12-fingered intestinal root. The pain is mostly at one point. Some patients experience pain in the epigastric area during percussive percussion over the stomach- **Mendel's symptom.** At the base there are painful spots in the body and tumor area of the thoracic and lumbar spine (symptom Boasa, Openxovskogo, Pevznera). In the same patients, Zacharin-Ged hyperesthesia zones are detected on the anterior and posterior surfaces of the chest, mainly in the D5-D9 segments.

On radioscopic examination of patients, barium sulfate is given and the shelf (niche) symptom and index finger symptom are positive, wound deformity.

On examination of **gastrofibroduodenoscopy** we can see the wound, post-wound scar, gastritis, duodenitis. On endoscopic examination, there is redness around the flattened folds of the mucous membrane, we can see it in the bleeding phase, polyps are visible (see Fig . 88). A biopsy is available (see photo below). **Laboratory testing** . Blood is checked — during the bleeding period of the wound, the amount of erythrocytes and hemoglobin is reduced and the color index is low. We can see reticulocytes in the blood due to increased hematopoiesis . In ulcerative colitis, hydrochloric acid is examined in the gastric juice. In duodenal ulcers, basal and stimulated hydrochloric acid are examined and hypersecretion is observed, while in subcardiac and medial body wounds, hydrochloric acid is normal or decreased. If achlorhydria is observed after administration of histamine, it indicates the absence of a 12-finger ulcer. Stools are examined by the Gregerson method to detect occult bleeding.

88 photos

Complications

-Perforation (6-20%);

- Bleeding (10-15%);

- Pyloric stenosis (6-15%);
- Penetration into the neighboring organ (15%);
- Malignancy is now very rare;
-Jaundice -12-finger ulcer is observed when the sphincter of Oddi is closed with a scar and hanging;

Perforation is characterized by "dagger" pain, which then leads to the development of acute peritonitis.

Bleeding can be observed in wound disease and can be detected by examining the stool for occult bleeding. **Garbage is checked by the Gregerson method.** The waste is taken in a plastic **SARSTEDT** container the **size of a** walnut and sent to the laboratory for testing. If you need to check for worms in the litter, then you will need 2 containers. In the Gregerson method, it is necessary to keep a diet for 3 days before checking for occult bleeding . To do this, the diet should not contain fish, meat, green melons, tomatoes and foods that contain iron . The waste obtained for analysis is checked no later than 12 hours and should be stored in the refrigerator at a temperature not higher than +2 +8 ° C. Because the giardiasis is somewhat resistant, they can even be sent for analysis within 24 hours to find a cyst.

Pyloric stenosis is most often seen in duodenal ulcers, rarely in the stomach. As this process progresses, patients will notice that eating increases rather than decreases pain. The most common symptom is excessive vomiting, in which the patient loses large amounts of gastric juice, which can lead to hypochloremia and renal failure. On superficial sliding palpation, a "swaying" noise can be felt in the epigastric area. The cause of stenosis is determined by FEGDS and a biopsy obtained for histological examination (this is to rule out gastric cancer and pyloric hypertrophy).

Penetration is defined by this term as the transition of the infiltrating-destructive process to the neighboring organs of the stomach and duodenum - the liver, pancreas, intestines. At the same time there is a subfebrile increase in body temperature, an increase in ECG.

Diet. It is necessary to exclude from the diet products that cause dyspepsia and increase gastric secretion (spicy spices, canned food, hot tea, coffee, alcohol, fried meat). Sliced meals (every 3-4 hours), i.e. a one-time meal reduction, are important in reducing gastric secretion. Because the amount of food stimulates secretion, drinking at the same time with the diet, smoking should also be limited.

Treatment- Treatment is often complicated. For wound healing it is necessary to neutralize the acidity of gastric juice and normalize the processes of excitation and inhibition in the CNS. Pain-no-spa, platyphyllin; proton pump inhibitors (omeprazole, omez, kvamatel, etc.), solcoseryl, ranitidine, etc .; Aloe 1.0 to improve reparative processes; de-zero; vitamin therapy.

Eradication therapy. Once Helicobacter pylori is detected, treatment is focused on eradicating it. Treatment is carried out on the basis of a strictly standardized scheme, using 3 or 4 component bactericidal substances (tetracycline, clarithromycin, metronidazole, trixapol) and bismuth and proton pump inhibitors (omeprazole). Eradication was effective in 85–90% of patients, with no recurrence of Helicobacter pylori.

Surgical treatment. Surgical treatment is the main method of treatment when conservative treatment is ineffective and in urgent cases (wound perforation, profuse bleeding wound malignancy, scar-wound pyloric stenosis).

Intestinal Syndrome

Affected bowel syndrome is a set of persistent functional disorders, in which there is pain or discomfort in the abdomen, their decrease after defecation is accompanied by changes in the frequency and consistency of diarrhea, and not less than 2 times. flour (not less than 3 months per year) includes symptoms of intestinal dysfunction.

-Variation in the frequency of diarrhea

-Changes in the act of defecation
-Changes in stool consistency
-Muscular separation with feces
-Meteorism

These pathological changes are the result of impaired colon function. In this case, the morphological changes in the colon are weak

Clinic of the disease. Some patients have spastic constipation and abdominal pain, while others have diarrhea. In some patients, painful constipation is replaced by diarrhea, which is often observed in the morning, after breakfast. After 3-4 morning diarrhea with water and mucus in the morning, the patient may feel better during the day, with diarrhea not bothering him at all. Such events can last periodically for weeks or even months and stop indefinitely. With the development of dysbacteriosis, flatulence is observed due to intestinal rot and putrefaction.

Physical examination. The abdomen is swollen during pain, and there are no signs of increased peristalsis. Palpation reveals a soft abdomen, pain in all parts of the small intestine. The sigmoid colon is spasmodic, hardened, the appendix is often swollen, and palpation reveals laughter, sometimes pain.

Diagnostics. - There are no changes in blood analysis, biochemical examination;

Examination of feces can help identify signs of dysbacteriosis.

On radiological examinations (irrigography, irrigoscopy) are characteristic signs of dyskinesia: uneven filling and loosening, spastic contraction and dilation of large parts of the intestine, the exchange of secretions into the intestinal cavity in large quantities; Colonoscopy is definitely performed with a biopsy because biopsies help differentiate the affected bowel syndrome in inflammatory bowel disease.

In addition, for the diagnosis of affected bowel syndrome, the entire gastrointestinal tract should undergo a special examination to rule out inflammation and tumor.

Practical training 5

Liver and bile ducts. Methods of examination: interrogation, physical examination (examination, palpation, percussion)

1. Practical training module .

Training time - 4 hours	The number of students 1 : 0 to 12
Form of training	Practical training
Practical training plan	Inquiry (complainant, anamnesis), examination, palpation, and percussion of patients with liver and biliary tract disease. Understanding the main clinical syndromes: jaundice, portal hypertension, liver failure.
The purpose of practical training :	Understanding the main clinical syndromes: jaundice, portal hypertension, liver failure .
Teaching style	Inquiry. Demonstration of patients, interactive teaching methods, practical skills.
Form of teaching	In small subgroups.
O ' unit equipment	Calls to O Training Guidebook , practical Typo content , projectors , computer .
Training mode	Methodically equipped auditorium.
Monitoring and evaluation	Oral control: questions and answers, tests, problem solving.

1.2 . ***Technological card of practical training***

See the stages of hearts and q ti.	Educator	Learners
In the preparatory stage of q	10. See the audience to control the purity karîm 11. Checks students ' readiness for training 12. And control q karîm	
1. bullet and training g contain levels of intra q (10 da q i q a)	1. Preparation of educational content on the topic. 2. Preparation of presentation slides for the introductory speech 3. Develop a list of references used in the study of science	
2 - the main stages of diarrhea q (160 da q i q a)	1. Divide students into small groups and ask questions on the topic. 2. Uses display posters 3. Uses slides, multimedia 4. Conducts treatment 5. Summarizes and summarizes the information provided on the basis of topics, encourages and actively evaluates the active participant students	They are divided into small groups They watch They participate They listen and answer questions
2- final press q ich (10 da q i qa)	1. Concludes 2. Provides independent work 3. Gives homework	He listens Takes notes Takes notes

3. Assessment of students' theoretical knowledge:

A) Frontal method:

1. 1 Primary and secondary complaints of patients with liver and biliary tract diseases
2. A general review of patients with liver and biliary tract disease
3. What changes will be identified in the review
4. The importance of palpation in diseases of the liver and biliary tract
5. Liver and bile duct percussion techniques
6. Liver percussion on Obraztsov VP and Strajesko ID
7. Liver percussion according to Kurlov
8. Methods of palpation of the liver
9. Give an idea about jaundice
10. Causes of jaundice
11. What is mechanical jaundice and diagnostic
12. What is parenchymal jaundice and diagnostic
13. What is hemolytic jaundice and diagnostic
14. The concept of liver failure
15. Factors leading to liver failure
16. Which process underlies the development of liver failure
17. Stages of liver failure
18. Clinical course of liver failure

19. The concept of portal hypertension
20. Characteristic symptoms of portal hypertension

Incident method

In clinical departments, the incident method is used to teach students to act in extreme situations. Students are given information about any situational issue. This issue needs to be resolved quickly (0.5-1.5 minutes).

This method speeds up students 'thinking and activates their activities in extreme situations. Student movement error should be 1.0.

I. Methods of subjective examination of patients with diseases of the hepatobiliary system

1.1 The main and additional complaints of patients (pain, jaundice, itchy skin, edema, bleeding, skin rash, subcutaneous hemorrhage, dyspepsia, fever, impaired olfactory function and additional complaints of headache pain, fatigue, decreased ability to work, pain in muscles, joints, sweating, etc.).

1.2 Medical history (how the symptoms first appeared, the dynamics of the course of the disease symptoms, previous treatment measures and its effectiveness, the reason for the current referral to a medical professional).

1.3 Life history (occupational, family, allergic, epidemiological, gynecological history, previous illnesses, transfusion, nutrition (rich in protein and vitamins), consumption of alcohol and toxic fungi, taking toxic drugs anamnesis), Botkin's disease, typhoid fever, malaria, liyamliosis, syphilis, work with toxic substances such as carbon tetrachloride, copper, lead, phosphorus, arsenic, aminazine in the course of work, systemic diseases, diabetes, biliary tract and duodenal ulcers and tumors cause liver disease.

II. Methods of objective examination

1. Review;
2. Palpation;
3. Percussion.

III. Instrumental inspections.

- Radioscopy and radiography of the biliary tract (including contrast cholangiography)
- UTT crossing of grass roads
- Computed tomography of the bile ducts

TJXG (skin, liver cholangiography)

Endoscopic retrograde cholangiopancreatography (ERXPG)

EFGDS (Esophagogastroduodenoscopy)

Oral cholecystography (iodine-containing contrast agent - bilitrast (3-3.5 g), yapanoevic acid (3-6 g) is given after dinner)

Intravenous cholegraphy (30-40 ml of 20% bilignost is sent and after 5-10 minutes X-ray and examination of the bile ducts)

X-ray and radiography of the liver and gallbladder

Dynamic hepatobiliocintigraphy

Elastography examination method with fibroscan

Ultrasound of the liver

Computed tomography

Positron emission tomograph

Laparoscopy (for liver biopsy)

IV. Radioisotope inspection method

Radioisotope hepatography (administered intravenously with J131 sodium chloride)

Liver scan

V. Laboratory tests

a) Total blood test (erythrocytes and hemoglobin);

b) Determination of osmotic resistance of erythrocytes (decreases)

c) Determination of erythrocyte survival (SR-51 test)

d) Determination of enzymes in the blood (G-6-FD (glucose-6-phosphate dehydrogenase), (GR) Glutathione reductase, glutathione peroxidase, AlT, AsT, IF (alkaline phosphatase) , etc.).

e) Blood smear (detection of Heinz-Ehrlich bodies in erythrocytes)

J) Blood AT (antibody to erythrocytes)

h) Determination of the number of reticulocytes in the blood

I) Antinuclear AT in the blood (antibody against erythrocyte nucleus);

Biochemical analysis;

Total bilirubin (bound, unbound)

The amount of copper in whey;

Ceruloplasmin in serum;

The amount of copper in the urine;

Determination of pigments in urine and feces;

Prothrombin time;

Platelet count;

Increased levels of albumin in the blood (hypoalbuminemia), decreased protein synthesis (hypoproteinemia) and increased levels of g -globulins;

Determination of immunoglobulins (Ig A);

Virological examination (hepatitis A, B, C, D, yellow fever virus, etc. **)**

Morphological examination (examination of bile duct biopsies)

Fibroscan test method

Ultrasound elastography is designed to determine the bioelasticity of tissues. The operation of the apparatus is based on the results of histological examinations. The harder the liver, the higher the rate of fibrosis development. To determine the degree of fibrosis, the apparatus sends a pulse wave. Its velocity is then measured from the chest wall using an ultrasound wave package . If the hard tissue of the ultrasound wave in violation of development of the greater speed and fibrosis rate is too high. Fibroscan is a modern non-invasive device designed to determine the stages of fibrosis and the degree of liver damage.

Advantages of fibroscan.

1. A simple, painless and quick non-invasive method of determining the degree of fibrosis in the liver.

2. T Medicine at the employee the right to delete founding able to get the results immediately gives you the option .

3. Can be checked many times, is harmless.

4. Fibroskandan needle biopsy difference is that the liver invasive method , against restricting or not.

5. The non-invasive method ma ' information, and it is recommended to determine the degree of fibrosis and morphological control method remains .

6. Elastometriyada liver tissue in the size of the assessment .

Elastometry should be performed on the fibroscan apparatus in the following cases :

1. For prophylaxis, it is advisable to perform elastometry in people older than 40 years.

2. Fibroscan examination is mandatory to determine the degree of damage to the liver in all diseases ;

-Viral hepatitis B, C, D.

- Fatty hepatosis, alcoholic steatohepatitis;

- Autoimmune diseases of the liver (**autoimmune** hepatitis, primary liver cirrhosis);

-Gilber syndrome and other hereditary diseases of the liver;

3. Elastography is mandatory in patients at high risk of liver injury ;

-Diabetes mellitus;

-High cholesterol or triglycerides in the blood;

-Tsitoliz above (ALT, as a T, GGT);

-The clinical analysis o ' z , Gary (decrease in the number of white blood cells and platelets);

-In obesity;

- Frequent increase in bilirubin in the blood;

- When cirrhosis of the liver is suspected and liver cirrhosis is diagnosed;

-Disposal elastometriya alkagol and toxic to the liver of those ' secret agents in patients receiving the opportunity to find diseases early.

Endoscopic r e trograd punk e atoxolangiografiya

Endoscopic r e trograd pancreatic e atocholangiography (ERPXG) is a method of examining bile duct and pancreatic protocols. It is a method of observation of the bile ducts by filling them with an X-ray contrast agent with an endoscope using an X-ray device 95 a and b .

Guide to ERPXG;

Gallstone diseases;

Narrowing of the bile ducts and their tumors;

Pancreatic cancer and other diseases and suspicion of them; **95 Figures a and b**

a

ERPXG radiograph;
Gallbladder cancer

If stones are found in the gallbladder and ducts during ERPXG, the stones can be removed without removing the endoscope. In 2-4% of cases, ERPXG is performed in an inpatient setting when frequent treatment procedures are required .

Positron emission tomograph

Positron emission tomography or dual photon emission tomography (PET) is a method of radionuclide tomography examination of human internal organs .

This method is based on the registration of γ-quantum vapors formed as a result of the interaction of positrons with electrons.

Positrons are formed from the positron b decay of a radionuclide that is part of a radiopharmaceutical. This drug is administered to the body before the examination.

The positron annihilation left in the tissue produces two γ-quanta of the same energy that are directed in one direction from the electrons in this medium.

A large set of detectors around the inspected object and computer processing of signals from them ensures that the distribution of radionuclide to the scanned object is performed on the basis of three-dimensional reconstruction.

PET is an actively developing diagnostic test method in nuclear medicine. At the heart of this method is the ability to control the spread of biologically active compounds in the body by radioisotopes emitting positron radiation, using a special detector (PET-scanner).

Ftordezoksiglyukoza. (radioactive indicator-fluorine-18, ^{18}F) PET-scanning is widely used in clinical oncology. Fluorodeoxyglucose (FDG) is an analogue of glucose and is well absorbed into tissues, including tumor cells.

In addition, most tissues other than the liver and kidneys are unable to excrete phosphate added to hexokinase. It follows that FDG is retained in all cells until it is broken down. Phosphorylated sugar cannot get them out of the cells for an ionized charge. This leads to intensive celebration of azo, such as brain, liver, and many cancer cell tissues, with excessive absorption of glucose. Therefore, FDG-PET can be used in the diagnosis and monitoring and treatment of oncological diseases. Lymphoma Hodgkin is a convenient and effective screening method for the diagnosis of non-Hodgkin's lymphoma and lung cancer.

History. David E. Gray, Luke Chapman and Roy Edwards developed the PET concept in the late 1950s. Their work later led to the design and creation of several tomographic instruments at the University of Pennsylvania. Later, Michael Tep-Pogrosyan and his co-workers Dj. Eugen-Robinson and K. Sharp Cook completed tomographic screening methods in 1975.

Radiopharmaceuticals used in PET. If the right radiopharmaceutical is selected, PET will be used to study various processes - metabolism, substance transport, gene expression, ligand receptor interactions, and so on.

Nowadays, the second cycle elements of mainly positron irradiating isotopes are used in PET.

Carbon-11 ($T_{½}$ = 20.4 min .);

Nitrogen-13 ($T_{½}$ = 9.96 min .);

Oxygen- ($T_{½}$ = 2.03 min .);

Ftor-18 ($T_{½}$ = 109.8 min .).

Ftor-18 is optimal for use in PET, has a half-life and low energy irradiance. Because fluoride -18 is administered to the patient in small doses due to the presence of a small half-life, a high-contrast PET image can be obtained.

Inquiry of patients with diseases of the hepatobiliary system

Inquiry into patients allows for early detection and diagnosis of liver disease. Patients with liver and biliary tract diseases may experience loss of appetite, nausea, vomiting, abdominal discomfort, fatigue, irritability, headache, itchy skin, decreased potency, complains of menstrual cycle disorders. Jaundice is often undetectable on examination and can be detected using a medical history. In addition, the survey will help determine the nature of jaundice. There may also be a mass of vomit or blood in the stool. An increase in the size of the abdomen and associated body mass increases (due to accumulated fluid).

In pathology of the biliary tract, there are other complaints associated with the ingestion of bile into the stomach and esophagus.

Unpleasant, usually bitter taste in the oral cavity, belching, nausea and vomiting after consumption of fatty and fried foods. Constantly non-uniform stools (constipation or diarrhea), abdominal relaxation are observed. There is sometimes pain in the area of the right subcostal arch. In addition to the liver, skin itching associated with bile duct obstruction occurs.

Complaints of a general nature

The onset of fatigue, irritability, and headache is associated with central nervous system dysfunction (liver encephalopathy) intoxication. Impairment of the detoxification functions of the liver leads to intoxication of the organism. Sleep disturbances (sleep inversion — nocturnal wakefulness and daytime drowsiness) and subsequent loss of consciousness (liver coma) are also characteristic.

Bleeding. The presence of blood in the sputum mass usually indicates bleeding from dilated varicose veins of the esophagus or with the presence of blood stasis (portal hypertension) or erosive gastritis in the portal vein, which is explained by liver disease. In this case the defendants appear red blood in the stools (gemmoraidal bleeding) and feces (melena). Bleeding is caused by erosive gastritis, alcoholic gastritis, Mellori -Weiss syndrome. Frequent bleeding from the gums and nose, subcutaneous hemorrhage is observed in normal exposures. Examples include uterine bleeding in women or prolongation of the menstrual cycle.

Pain. There may be heaviness, pressure, pain under the right rib arch. Pain may be associated with peritoneal exposure surrounding the liver, such as perigepatitis (liver tumor and abscess) and pericholecystitis. They are usually intense in nature and radiate upwards (to the right shoulder area), intensifying when palpated under the right rib. Elongation of the glisson capsule in hepatic enlargement causes a symptom of pain, which is often observed in the occurrence of blood stasis in the liver as a result of heart failure. In gallbladder (liver) colic, it leads to spastic contraction of the smooth muscles of the gallbladder and bile ducts. The latter occurs when the stone slides along the grass paths. These pains occur suddenly and become intense, often irradiated upwards, there is nausea, vomiting, which does not relieve the patient.

Dyspepsia. Symptoms such as decreased appetite, unpleasant or sour fullness in the mouth, bloating, nausea, vomiting, rest in the abdomen, laughter, constipation, or diarrhea are disturbing. The onset of these symptoms is caused by a violation of the secretion of bile.

Fever. It often occurs in diseases such as acute inflammation of the liver and bile ducts (abscess, liver cancer, hepatitis, active period of cirrhosis).

Asthenoneurotic syndromes (decreased ability to work, fatigue, rapid fatigue, insomnia, headache, etc.).

Itchy skin is characteristic of liver disease and is accompanied by cholestasis. The degree of exacerbation of itchy skin (painful, intensifies at night) is accompanied by jaundice (in cholestasis syndrome), intrahepatic and extrahepatic bile duct obstruction.

Jaundice. The main symptom encountered in liver disease.

Reasons. Inflammation of the liver cells (viral and other), ischemia, they are tumors, necrosis. Toxic liver damage (hepatotropic poisons, alcohol, drugs). Autoimmune hepatitis. Intrahepatic cholestasis. Cholangitis with primary sclerosis. Scarring of the bile ducts after surgery on the bile ducts. Drug-induced cholestasis (contraceptives and testosterone and bq).

Pregnancy cholestatic jaundice. Hemolytic jaundice of the baby. Sepsis. As a result of gallstones getting stuck in the bile ducts. Compression of the bile ducts by tumors. Tumors in the duodenum grow into the common bile duct and close it. Closure of the bile ducts with vomiting. Badda-Kiari syndrome. Cirrhosis caused by thrombosis of the hepatic veins (see below).

Changes in the sense of **smell and smell** (obonyaniya) are often observed in patients with hepatitis A (decreased sensitivity of the olfactory analyzer-hypoosmia, poor odor perception-dysosmia, decreased sense of full-sense-hypogevsia, nausea (dyskinesia) -dysgevia) .

The history of the disease turbaned, hepatitis B and C and other waste transfer otkir other diseases of the liver and biliary tract, cholangitis and liver colic as well as enlarged liver, spleen, liver diseases and the emergence of capacity. When collecting the history of the disease, it is necessary to pay attention to the nature of the pain, pain in the liver and biliary tract - pain in the liver (due to elongation of the Glisson capsule) or stabbing pain in gallstones and liver observed in colic, cholangitis.

During the collection of **life history,** patients' nutrition, whether they ate vitamins and proteins, alcohol, contact with toxic substances, blood and blood products, surgical, dental, gynecological treatments, some toxic effects It is important to assess whether the recipient has taken drugs, conducted hepatitis B and C, cholestasis, and the presence of wounds and tumors in the common bile duct in the duodenum .

Methods of objective examination of patients with diseases of the hepatobiliary system

Overview ; Check skin color, integrity, hair coverage. Determining the color of the sclera. Check the condition of the mammary glands and testicles. - It is advisable to examine the shape of the abdomen and the anterior wall.

On general examination, attention should be paid to the degree of weight loss, which is observed in decompensated cirrhosis or liver cancer; At the same time, a decrease in body mass is observed with an enlarged abdomen due to ascites. To understand the cause of isolated ascites, the dilated veins of the abdominal wall are important (“Jellyfish Head” Fig. 96-3), which is the reason for the onset of portal hypertension. In long-lasting liver cirrhosis of various etiologies, changes in the phalanges of the fingers can be felt, just like “drumsticks”.

Some diseases of the developing liver (chronic active hepatitis, cirrhosis of the liver), accompanied by general nonspecific syndromes-non-infectious fever, (in contrast to these diseases in acute cholecystitis, cholangitis, liver abscess, infectious hepatitis, often with hectic, shivering and sweating observed temperature), arthritis, vascular changes with Reynaud's syndrome ("dead fingers" syndrome), dry syndrome Shegren's syndrome, lack of saliva (xerostomy), tears (dry keratoconjunctivitis)), diffuse caries of the

teeth. Sometimes liver disease occurs as scleroderma, systemic lupus erythematosus, rheumatoid arthritis, the results of the study and examination of the anamnesis, as well as biopsy, help to diagnose primary liver disease from non-hepatic diseases. Liver pathology typical characters under the special terminology, "" Signs of liver, small and large groups of pictures (78). **Eye examination** to display the yellow and green colors on the floor of the mucous Kaiser Fleischer ring You can view 109 photos.

Small signs of liver. Telangiectasia ("vascular asterisks" 96 photo - 1); Gynecomastia (96 photos-2); Red lacquered lip, tongue (96 photos -4), oral mucosa; Palmar erythema (liver plaque); Xanthomatous plaques (Fig . 96 -5) (plaque), xanthelasmas; drumstick rod-shaped fingers.

The oral and nasal mucosa, skin hemorrhage ekximoz and petexiya be observed gemmoragik syndrome and liver alkagolli disease (like a giant ear Paraty gland growth, Dyupyuitren contracture of 97 straight Cafu obsessive-T fibrous-scarring and dryness of the flexor muscles of the fingers, injectation of scleral vessels).

Major signs of liver

These symptoms include jaundice and hepatomegaly, accompanied by the main syndromes of the liver - portal hypertension, hepatocellular insufficiency, hepatic encephalopathy, hepatolienal syndrome.

When examining the abdomen, attention should be paid to changes in the size, shape, and skin of the abdominal wall. We can see an increase in the size of the abdomen due to ascites fluid and the shape of the abdomen resembling a frog's abdomen, dilation of the veins of the anterior wall of the abdomen, bulging of the umbilicus, symptom of jellyfish head, vascular asterisks. Yellowing of the skin of the abdomen, hemorrhagic rashes on the skin of the abdomen can also be seen. When examining the liver area, it can be seen that it is enlarged to a significant size and at the expense of tumors (nodules, tumors, echinococcosis cysts, large abscesses) with a slight enlargement of the abdominal wall (hepatomegaly). In all of these cases, attention should be paid to the asymmetry of the abdomen caused by liver tumors, as well as the retardation of the right subclavian and epigastric area during movement. It is also possible to see an enlarged liver pulsation due to a wave of blood regurgitation in tricuspid valve insufficiency. An enlarged gallbladder forms a visible bulge.

96 photos

97 photos

Liver palpation. Palpation of the liver is the main method of examination of this organ, which is performed in 4 stages (Fig. 98). palpation Palpation determines the lower border-shape, integrity, irregularities, and sensitivity of the liver. Normally, when the liver is

palpated, the surface is smooth , smooth, painless. Palpation of the liver is performed according to the rules of deep sliding palpation on Obraztsov. In this case, the patient should stand on his right side, with the patient's arms outstretched and his legs bent at the knees. The patient is palpated during the exhalation phase when the abdominal wall muscles are in a state of maximum relaxation. Normally the liver is soft and the surface is smooth. Hepatitis, in cirrhosis of the liver, is organized, hardened, and the surface is notxious.

98 photos

In order to enhance liver excursion, the pressure of the left palm is applied to the lower sections of the right anterior chest wall. The palpated right arm is placed on the anterior abdominal wall below the border of the liver. In this case, the fingers enter the abdomen during exhalation . If there is fluid in the abdomen (in ascites), liver palpation and percussion become more difficult, depending on the amount of fluid. To do this, place the right finger below the navel in the right mesogastric area, lift it upwards until the organ is felt under the fingers with a provocative movement of the fingers, and information about the boundaries and surfaces of the liver is obtained.

Perkussiyasi of the liver (99 and 101) . Percussion can be used to detect changes in liver size, lower border, upper border in rare cases (abscess, large cyst, large tumor nodule). Usually the upper border of the liver corresponds to the lower border of the right lung. Determining the location of the lower border is determined using liver percussion or palpation.

99 photos

The lower limit of the liver can be determined using quiet percussion. Percussion is percussed in the area of the tympanic sound from the navel or below until the plexiglass-finger is upwards until a muffled sound is formed. This will be the lower limit of the liver. Normally, the liver does not protrude beyond the rib cage, while in asthenics it is below the rib cage. When breathing deeply and in a vertical position, the lower limit of the liver drops to 1-1.5 cm.

100 photos

To determine the size of the liver, the upper border (100) was percussed along the right midline to the bottom of the rib cage until a distinct lung sound was produced. picture) and along this line the lower border of the liver is percussed from the level of the umbilicus or below and upwards until the tympanic sound is percussed until the sound of hepatic suffocation is produced .. This will be the lower border of the liver. By combining the two points, the **first measurement of the liver is measured according to Kurlov. Usually it is 9 cm (9-11).** It is used to determine two other dimensions from the upper limit of liver obstruction.

Along the midline of the abdomen, the liver is percussed upwards until a lumen is formed, and a secondary dimension is determined. Determining the upper limit along the midline is difficult because the presence of a breast under the skin gives a fading percussion sound, so as the high point of this measurement is conditionally the first hepatic obstruction measurement with the upper limit a different point is obtained. **By combining these points, a second dimension is obtained according to Kurlov, usually 8 cm (7-9).**

The third dimension on the curvature begins percussion inward from the anterior axillary line parallel to it near the left rib arch. Usually it is 7 cm (6-8). If the liver is enlarged, the first major dimension is represented by a fraction, in the figure - the total dimension along the right midline, and in the maxilla - the dimension below the rib arch.

To determine the size of the right part of the liver, percussion is performed in a quiet percussion method from the top of the lungs until a hoarse voice is heard, and the top of the finger is marked on the skin and identified from above and below along three topographic lines . The symbol is placed under the finger 99 See photo.

The table of upper, lower boundaries and dimensions of the liver in the norm

Topographic lines	Upper border of the liver	Lower border of the liver	Dimensions
L. parasternalis	VI upper edge of rib	2 cm below the arch of the right rib	8-11 cm
L. medioclavicularis	VI rib	Lower right rib arch	9-11 cm
L. axillary anterior	VII rib	X ribs	10-12 cm

Normally, the condition of the liver depends on the constitution, being higher in hypertensives and below the threshold indicated in asthenics. When percussion is performed in a vertical position, the liver moves normally 1-15 cm down.

To determine the dimensions of the left side of the liver, percussion is performed along the left rib arch, from the VIII-IX rib arch or from the left axillary anterior line to the right until the tympanic (Traube area) is free of sound. until the squeak goes into the sound and is set.

Percussion is of diagnostic importance.

Depending on the enlargement is observed in other liver diseases. **101 picture**

Practical training 6

Liver and bile ducts. Test methods: laboratory-instrumental test methods). Basic clinical syndromes. Jaundice clinic, diagnosis.

1. Practical training module .

Training time - 4 hours	The number of students 1 : 0 to 12
Form of training	Practical training
Practical training plan	Methods of laboratory and instrumental examination of patients with diseases of the liver and biliary tract (cholecystography, liver scan, examination of the liver and gallbladder with ultrasound). Computed tomography. Symptomatology of chronic cholecystitis and hepatitis.
The purpose of practical training :	Familiarity with the methods of laboratory and instrumental examination of patients with diseases of the liver and biliary tract . Symptomatology of chronic cholecystitis and hepatitis.
Teaching style	Inquiry. Demonstration of patients, interactive teaching methods, practical skills.
Form of teaching	In small subgroups.
O ' unit equipment	Calls to O Training Guidebook , practical Typo content , projectors , computer .
Training mode	Methodically equipped auditorium.
Monitoring and evaluation	Oral control: questions and answers, tests, problem solving.

1.2 . ***Technological card of practical training***

See the stages of hearts and q ti.	Educator	Learners
In the preparatory stage of q	13. See the audience to control the purity karîm 14. Checks students ' readiness for training 15. And control q karîm	
1. bullet and training g contain levels of intra q (10 da q i q a)	1. Preparation of educational content on the topic. 2. Preparation of presentation slides for the introductory speech 3. Develop a list of references used in the study of science	
2 - the main stages of diarrhea q (160 da q i q a)	1. Divide students into small groups and ask questions on the topic. 2. Uses display posters 3. Uses slides, multimedia 4. Conducts treatment 5. Summarizes and summarizes the information provided on the basis of topics, encourages and actively evaluates the active participant students	They are divided into small groups They watch They participate They listen and answer questions
2- final press q ich	1. Concludes	He listens

(10 da q i q a)	2. Provides independent work 3. Gives homework	Takes notes Takes notes

3. Assessment of students' theoretical knowledge:

A) Frontal method:

1. 1. Describe normal bilirubin metabolism
2. Explain the determination of bilirubin
3. Name the galactose and adrenaline test
4. Explain the normal protenogram
5. Describe protein precipitation tests
6. What is Thymol and Formal Test
7. How the liver is involved in copper and iron metabolism
8. Examination of the bile ducts and gallbladder for radiography and rengenoscopy
9. Describe the method of splenoportography
10. What is holegraphy
11. What is cholecystography
12. Describe the radiostop inspection method
13. What is exography
14. Describe a liver biopsy
15. What is loparoscopy
16. What is scanning
17. The role of computed tomography in the diagnosis of liver and biliary tract diseases
18. Etiopathogenesis of chronic cholecystitis
19. Clinic of chronic cholecystitis
20. Clinical and diagnostic diagnosis of chronic cholecystitis

Brainstorming method

Basic rules :

- Not to mention the shortcomings that hinder the emergence of the idea
- The height of ideas and thought, because the more unusual an idea, the better it is
- Accept many offers
- Combination of ideas and their development
- Present the idea succinctly without argumentation
- Divide the group into two: thought generators and thought analysts

This method allows you to argue ideas and opinions, your own personal opinion, to find the optimal solution in any situation.

Check bile fluid . Duodenal probing

Composition of duodenal juice 1) Gastric juice; 2) Bile fluid; 3) pancreatic secretion; 4) Consists of intestinal juice secreted from the intestinal mucosa. Duodenal examinations provide information about the bile ducts and gallbladder. This method Lyutkens and the sphincter of Oddi and funk t know the concentration of Zion, and a reduction in the state. Inflammation of the bile ducts, pathogenic and excretory microbes can also be detected.

Duodenal examinations are performed using special "duodenal probes" - thin rubber tubes 1.5 meters long and marked on the surface indicating the depth of its penetration. The rubber is mounted on a metal container that collects the analysis at one end of the tube. In this case, the juice is taken from

the duodenum at lunch. In this case, the patient swallows the duodenal tube until the first sign and lies on the tie with the right side. A pillow or roller is placed in the pelvic area for easy passage from the stomach to the duodenum. The patient slowly swallows the probe to the marked mark. In this case, the probe enters the bile duct and pancreatic duct from the duodenum below. Usually this process takes 1-1.5 hours and in some cases 15-20 minutes. At the same time a yellowish liquid- **A** portion begins to separate. It consists of bile, pancreas, intestines and gastric juice. Withdrawal of portion **B** (grass in the gallbladder) is performed only 20-30 minutes after portion **A** begins to separate. To do this, a stimulus of gradual contractions is introduced into the gallbladder through a probe (30-50 ml, 33% magnesium sulfate, 40 ml of olive oil, 30-40 ml of 10% salt, 30-50 ml of 10% glucose solution, etc.). Usually, the contraction of the gallbladder is observed under the influence of food, which regulates and stimulates its activity, and other mechanisms . Weakening of the Oddi and Lutkens sphincters is also observed along with gallbladder muscle contractions. Duodenal probing uses special compounds that allow the removal of **B** portion of black-red or bluish or dark-colored bubbles after 5-10 minutes. Once the gallbladder is emptied, a golden-reddish transparent bile **C** portion begins to separate from the bladder neck and intrahepatic bile ducts.

In addition to the traditional duodenal sounding methods described above, 5-fractional duodenal sounding methods have also been used in recent years. It allows the time of separation of its individual fractions and quantitative analysis of each fraction, taking into account the area of grass coming into the gallbladder. The chromatic fractional method of duodenal probing is used to accurately determine the characteristics of bile secretion from the gallbladder. In this case, the patient receives methylene gelatin capsule dye 10-15 hours before the duodenal probe. This drug allows you to clearly separate the grass from other organs.

Concentrated suction fractions in the mucous membrane of the gallbladder and their pathology can be established using the above methods. The chemical composition of bile also allows to know the metabolism that occurs in the gallbladder, the condition of the liver and the metabolism in the body (including pathological processes).

Bile fluid microscopy . Leukocytes in the stomach may belong to the mouth, stomach, or intestines, so it is advisable to use a two-channel probe in duodenal probing, which allows the residue in the stomach to be absorbed.

The presence of giardiasis leads to various pathological (mainly inflammatory and dyskinetic) processes in the gastrointestinal tract. There are no giardiasis in the gallbladder of healthy people. In patients with cholecystitis, the giardiasis is located in the mucous membrane of the gallbladder, (along with microbes) exacerbates dyskinesia and inflammatory processes.

In the diagnosis of cholecystitis, increased concentrations of G immunoglobulin, A, R-proteins, C-reactive protein, alkaline phosphatases, S-nucleotidases, malon dialdehydes are noted. However, a decrease in the amount of lysozyme, bilirubin is observed.

Knowing the ratio of bile acids, cholesterol and lecithins, the lithogenicity index (or coefficient) can be determined and it is calculated as follows:

$$\text{Lithogenicity index} = \text{The } \frac{\text{amount of cholesterol in the bile}}{\text{Bile acids, cholesterol and lecithins which can be melted in the given proportions amount of cholesterol}}$$

If the lithogenicity is less than one, it is called unsaturated, and if it is greater than one, it is called saturated (sometimes called the "saturation index").). Cholesterol-saturated grass is characterized by crystallization of cholesterol, which is why they are called lithogenic.

Biochemical analysis of blood. Examination of biochemical parameters is important in the diagnosis of diseases of the liver and biliary tract. First of all, it is important to determine bilirubin, which is a key indicator of the liver. One of the main biochemical indicators manifested in liver injury is a change in the activity of enzymes produced by liver tissue.

This analysis is used to assess liver function and differential diagnosis of liver diseases (inflammatory, infiltrative, metabolic, cardiovascular, hepatobiliary diseases.)

Bilirubin. The amount of bilirubin is an indicator that characterizes the absorption, metabolism and excretory functions of the liver. Bound bilirubin is differentiated according to test results with unbound bilirubin.

Transaminase. Glutamate oxaloacetate transaminase and alanine aminotransferase are sensitive enzymes that indicate the state of liver cells. The maximum increase in their number is observed in necrotic changes in the liver (viral hepatitis, liver intoxication, acute circulatory failure, impaired hepatic venous permeability) and sometimes in impaired bile duct permeability (concretion). Transaminase activity indicators do not indicate the severity of the disease.

Less well-known changes are observed in cholestasis and infiltration. Alanine aminotransferase indicators indicate the degree of liver damage. In this case, aspartate aminotransferase is found in the transverse skeletal muscle. Alcoholic hepatitis causes a significant increase in aspartate aminotransferase levels relative to alanine aminotransferase.

Lactate dehydrogenase - an enzyme test is not of great importance in the diagnosis of liver disease.

Alkaline phosphatase - this cholestatic syndrome is a sensitive indicator of biliary tract permeability disorder (increases rapidly in the blood bilirubin) in infiltrative diseases of the liver. Its specificity is low because this enzyme is also found in other tissues. Alkaline phosphatase activity is high in children, pregnant women, and bone patients, characterized by thermostability of

specific tissue isoenzymes (liver enzymes are thermostable at low bone enzyme activity).

5-nucleotidase - An increase in enzyme activity in diseases of the hepatobiliary system is the same as changes in alkaline phosphatase, however, it more shows the state of liver cells. It is examined to differentiate liver-related changes in alkaline phosphatase, especially in childhood, pregnancy, and bone disease.

Gamma-glutamyl transferase is the same as the amount of alkaline phosphatase. When the hepatobiliary system is damaged, its activity increases rapidly. It is also susceptible to diseases of the pancreas, kidneys, heart, lungs.

Prothrombin time is an indicator of the activity of hemocoagulation factors. Its increase indicates a lack of activity of this factor. All factors of hemocoagulation are produced in the liver except factor 8. Extensive focal damage to liver tissue results in its deficiency (hepatitis, intoxication, cirrhosis). Factors 2, 7, 9, 10 are active only in the presence of vitamin **K.**

Albumin - A decrease in the concentration of albumin in the blood indicates its low production in the liver (chronic liver pathology or prolonged starvation) or intensive excretion in urine or feces. Hepatic dysfunction is a less sensitive indicator of t politics because its half-life in the blood is 15-20 days. It is characterized by the manifestation of albumin-deficient dysfunction in patients with chronic liver pathology. Decreased globulin is mainly observed in chronic liver disease. Decreased globulin is observed in the autoimmune form of chronic hepatitis.

An increase in **ammonia in the** blood serum indicates impaired detoxification function of the liver. This fulminant hepatitis has been observed in liver cirrhosis when exposed to hepatotoxic agents and in liver cirrhosis. Many of ammonia in blood serum in patients with liver en t sefalopatiyasidan. It is more common in encephalopathy, but does not allow to detect disorders of metabolic processes (liver injury, uremia or an increase in carbon dioxide in the body).

Instrumental inspection methods.

Contrast radiology, (CT) UTT (ultrasound) endoscopy, duodenal probing, laparoscopy, biopsy, for the diagnosis of liver and biliary tract diseases,

radionuclide inspection methods are used.

X-ray, radionuclide, ultrasound methods.

X-ray examination using a contrast agent is the most convenient method for detecting varicose veins of the esophageal veins - an increase in pressure in the portal venous system. Choleography is also used to examine the bile ducts. In this case, the contrast agent is administered parenterally. X-ray examination of the bile ducts is also performed - cholangiography through the skin of the transverse liver. In this case, the contrast agent is injected into the common bile duct or gallbladder through the puncture route to the abdominal

wall or liver tissue. In addition to X-ray contrast cholegraphy, a contrast agent is delivered retrograde to the common bile duct (ERXPG) through endoscopy. This method helps to detect biliary tract hypertension caused by narrowing of the bile duct. This is the gold standard in the detection of primary sclerotic cholangitis. UTT (UZI), CT, radionuclide examination are used to detect focal and diffuse changes in the liver parenchyma. If necessary, angiography is performed.

Among these techniques, FEGDS is important. This method detects changes in the esophageal veins, gastric mucosa (characteristic of portal hypertension syndrome), as well as erosive esophagitis (often observed in alcohol-induced liver damage) and duodenal ulcers.

Laparoscopy and biopsy. Laparoscopy and biopsy methods are important for the diagnosis of liver and biliary tract diseases. Instructions for biopsy.

Hepatomegaly of unknown etiology. ALT, AST, **g-** steady increase in GTP activity. Detection of viral hepatitis B, C, D in liver tissue. Liver damage under the influence of drugs. Liver damage under the influence of alcohol.

Infectious and systemic diseases. Examination of the patient's relatives to determine hereditary diseases of the liver.

Furnace changes.

Laparoscopy is used to determine the causes of chronic liver disease (hepatitis, cirrhosis, small, large nodules, mixed), portal hypertension, focal liver damage.

Basic clinical syndromes . Jaundice

Normally, after erythrocytes break down, bilirubin is released from hemoglobin and goes to the liver through the blood and binds to glucuronic acid. Link bilirubin through the bowel and biliary tract, urinary tract and goes out **Jaundice (ictherus)** - bilirubin, liver cells by withdrawing glyukuronid acid, associated with the violation of its transport (102 images) at the expense of the blood bag is a syndrome characterized by an increase in the amount of binded or unbound bilirubin. **102 photos**

As a result of an increase in the amount of bilirubin in the blood is observed yellowing of the skin , palms of the hands and feet, mucous membranes and sclera, itchy skin . As a result of an increase in the amount of bound or unbound bilirubin in the blood, there is a change in the color of urine (beer color) and a change in the color of feces - whitening (liver and subcutaneous jaundice) and even darkening (liver)

upper jaundice) (Fig . **103)** . An increase in the amount of bilirubin in the blood can be caused by excessive breakdown of blood, liver cell deficiency or a pathological process in the bile ducts. Jaundice and know it exceeds the amount of compound in the blood accumulate in the skin, mucosa and sclera. Yellowing of the skin (increased carotene and acrycin) can be caused by other factors **103 photos**

(in fake yellow); Consumption of products containing large amounts of carotene, acrycin (carrots, tomatoes) also causes jaundice - in contrast to

jaundice, the sclera of the eye does not turn yellow. Clinically, jaundice in various areas of the body depends on the degree of hyperbilirubinemia. The sequence of development of jaundice in the body is as follows; at the beginning the sclera then the lower part of the tongue becomes the mucous layer and palate, then the skin of the face, and at the end of the palms the whole body turns yellow.

Jaundice is less noticeable in anemia, hypoproteinemia, obesity tumors.

In thin and muscular patients, the skin becomes brighter yellow. When blood clots in the liver as a result of heart failure, the upper part of the body turns yellow. In persistent hyperbilirubinemia, the body color becomes green or even bronze-black (melanosaricity).

Jaundice syndrome requiring inspections -differentsial diagnostic syndrome . Therefore, it is important to know the physiology of bilirubin formation and the mechanisms of its distribution in the body.

Bilirubin is the end product of gem breakdown, of which 80-85% is formed from hemoglobin and the remaining 20-25% from heme-storing proteins (cytochrome P-450). The formation of bilirubin takes place in the cells of the reticuloendothelial system, and around 300 mg of bilirubin is produced daily. The bilirubin formed as a result of gem breakdown is free or unbound, and it is then bound to plasma proteins (albumin). Protein-bound bilirubin comes to the liver through the rock. It is then converted to bound bilirubin (conjugated) in the liver under the action of uridindiphosphate glucuronyltransferase (UDFGT).

Conjugated bilirubin enters the duodenum through the bile ducts. Under the influence of bacteria in the intestine is broken down into 2 types of pigment: stercobilin (gives color to feces) and urobilinogen. Urobilinogen is well absorbed in the small intestine, and a certain part is normally excreted again through the liver and kidneys (enterohepatic circulation). Bilirubin in the blood is not evenly distributed in the tissues. It is retained in large amounts in elastic tissues (skin, sclera, and vascular walls), so in jaundice these tissues become more yellow.

Jaundice syndrome also develops in most severe diseases of the internal organs. In such cases, of course, it is advisable to make a differential diagnosis. For example, jaundice syndrome is observed in all liver diseases . Jaundice syndrome is observed in hemolytic anemia, vitamin **B 12** and folic acid deficiency anemia. Jaundice syndrome of the emphysematous type of chronic obstructive pulmonary disease is observed in 5% of patients with pneumococcal pneumonia. Therefore, it is advisable to thoroughly examine the lungs of those who have developed jaundice.

In severe heart failure, there is often blood stasis in the liver, which in turn not only develops jaundice, but also leads to portosystemic encephalopathy. Severe renal failure also leads to jaundice syndrome.

Types of jaundice

An increase in bound or unbound bilirubin in the blood occurs in hereditary, benign, and acquired types of jaundice. Jaundice is a symptom of

some disease. Acquired disorders of bilirubin metabolism are divided into: hepatic, parenchymal (liver-cell), mechanical jaundice. The most common type is hepatic jaundice-hemolytic, hepatocellular jaundice-hepatocyte necrosis or acute and chronic liver disease, and subhepatic jaundice is the result of intrahepatic or common bile duct obstruction. .

Hemolytic jaundice

It is characterized by jaundice konyugirlangan the amount of bilirubin in the blood Photo 103-104 . **Occurs in the excretion of** unbound bilirubin in the case associated with multiple hemolysis of erythrocytes: **Fig . 104**

When examining patients with hemolytic jaundice, the skin color turns lemon-yellow , see photo 105 below , there is no itching of the skin, on palpation the spleen is enlarged, anemia develops. **UTT examination** can detect enlarged spleen.

The amount of **free** (unbound) bilirubin in the blood increases because the liver does not have time to bind the bilirubin that is produced in large quantities. **The color of the** stool is intense, which is due to the formation of large amounts of bilirubin and its excretion in the intestine in the form of stercobilinogen. Urine contains **large** amounts of urobilin and stercobilinogen, but no bilirubin.

Liver-cell (parenchymatosis) jaundice.

Liver cell damage due to their **10 5 photos**
due to a decrease in detoxification function, the binding activity of bilirubin to glucuronic acid, which is formed as a result of the breakdown of hemoglobin in the blood, i.e., a decrease in bilirubin glucuronide formation and low excretion into the bile ducts , see Fig . 106 . This type of jaundice is caused by damage to the hepatocytes and is bound to the blood, but not bound
an increase in bilirubin is observed.

The color of the skin coatings on examination is **106 photos**
the changed color is reddish-yellow and redder, the color of the urine is darker and the color of the stool is whiter than usual. Palpation and percussion may reveal enlarged spleen. The amount of total bilirubin in the blood increases due to bound and unbound bilirubin. As with hemolytic jaundice, the amount of conjugated bilirubin in the urine increased. **The following 107 pictures a and b.**

a b

107 Figures a and b

Mechanical jaundice

This type of jaundice occurs as a result of obstruction of the bile ducts under the influence of endogenous or exogenous causes Figures 108 and 109 . Endogenous occlusion occurs due to occlusion of gallstones into the bile ducts and sclerotic changes of the bile ducts in sclerosing cholangitis. Exogenous closure externally narrows the bile ducts (tumors, liver metastases in tumor metastases, pancreatic head tumors, tumors and tumors of the 12-fingered intestinal mucosa, penetration of the same intestinal ulcers, etc.). These changes pressure upwards from the place where the bile is stagnant **108 Fig**

Ensures that bilirubingly glucuronide is reabsorbed into the blood due to the increase. Due to regurgitation in jaundice, the amount of conjugated bilirubin in the blood increases.

When patients are examined, the skin color is maximally altered, with persistent jaundice the skin becoming darker in color. The amount of bound bilirubin in the blood increases, and if this continues for a long time, unbound bilirubin also appears due to impaired liver function.

Binding bilirubin and bile acids appear in the urine. The color of the urine is brown. The color of the stool becomes discolored because stercobilin does not enter the intestine. Patients almost always have itchy skin due to an increase in bile acids in the blood (cholemia), on examination can be seen scars on the skin, the skin is tight. Due to bile acids tickling the stray nerve, patients' pulse decreases (bradycardia), blood pressure drops. The central nervous system is poisoned by cholemia, causing astheno-neurotic syndromes (headache, dizziness, rapid fatigue, weakness, decreased ability to work, irritability, insomnia, etc.). Based on the above signs of mechanical jaundice, its wavy character is felt; this is explained by the obstruction of the bile duct by the tumor.

109 photos

There are 8 clinical forms of hereditary pigmented hepatosis.

A. Conjugated hyperbilirubinemia

Gilbert's syndrome,
Meylengracht's disease,
Kriegler Nayar syndrome,
Lucy-Driscoll syndrome,

B. Unconjugated hyperbilirubinemia

Dabin Johnson syndrome
Rotop syndrome
Bayler's disease (congenital intrahepatic cholestasis)
Aagenesa-Summerskill syndrome (congenital intrahepatic cholestasis)
Alajilya syndrome (accompanied by cholestasis)

Wilson-Konovalov disease

Wilson-Konovalov disease - In 1912, the British neurologist Wilson recorded 12 familial cases, all of which were accompanied by progressive degenerative changes in the liver with cirrhosis of the liver, and its pathogenesis remained unknown until 1948. In the same year, the pathogenesis of Wilson's disease was determined after the release of an increase in the amount (concentration) of copper in the liver and brain. There is currently a lot of data on the hepatotoxic properties of copper, and a genetic defect in Wilson's disease has also been identified.

Wilson's disease is a rare inherited disease in which the autosome is inherited through a recessive pathway and the gene (a mutation of ATP7B causes the disease) is located on chromosome 13 (part 13q14-q21) and impairs protein and copper metabolism. The Wilson disease gene was isolated in 1993 and contains more than 40 mutated genes. There are 30 patients for every 1,000,000 population. Marriages between close relatives account for 1 patient per 90 people. It is mainly manifested from a young age and is accompanied by the accumulation of large amounts of copper in the blood in the body. In the early stages, copper accumulates in the liver, leading to hepatitis, cirrhosis, liver failure. Copper then falls on other organs and systems, damaging them. Injury to the brain leads to mental nerve changes.

M is metabolism. Once the copper in the food (2.5-5 mg) is absorbed in the small intestine, the proteins combine with the amino acids to enter the liver through the portal vein, and a large amount (90%) remains there. The rest is excreted in the intestine with copper feces and the part that does not enter the portal vein is excreted by the kidneys. In hepatocytes, copper is present in metalloenzymes. These are superoxide dismutase, cytochromoxidase, monoamine oxidases. The export of copper is carried out by the transport protein ceruloplasmin, which belongs to the a_2 globulin fraction. The introduction of copper into the ceruloplasm is carried out in the Goldji apparatus. This is done in conjunction with the P-type protein-ATPase, which transports copper through the Wilson disease gene. They are actively involved in the lysosomal excretion of copper, keeping the amount of copper in the norm. If this condition persists, of course, copper will accumulate in the liver. Preservation of large amounts of copper in the liver (detoxifying property), copper is associated with glutathione, metalothonane and excreted in the biliary system in the presence of lysosomes. 80% of copper is excreted by the bile into the liver. Copper in bile is bound to high-molecular-weight proteins that are not reabsorbed into the small intestine. Copper-bound high-molecular-weight proteins prevent them from being absorbed into the small intestine. **Pathogenesis.** The main role in the pathogenesis of disease development depends on the balance of its excretion with the amount of copper taken. In Wilson's disease, the accumulation of copper is due to a decrease in its excretion by bile, mainly due to the lysosomal fraction, and leads to the accumulation of copper in the liver. Decreased copper excretion is associated with Wilson disease gene deficiency. This gene ensures that copper is transported to the Goldji apparatus and broken down into bile. Due to this,

the binding of copper to apoceruloplasmin is disrupted, which in turn leads to a decrease in ceruloplasmin in the blood serum. Ceruloplasmin contains copper, which replenishes it in the blood due to copper, and this substance is involved in erythropoiesis. Hereditary deficiency of this substance leads to an increase in the trace element copper in the blood serum, and mainly develops jaundice in the eye and the formation of a green ring. This results in liver damage, cirrhosis, and secondary brain damage (dystrophic changes in the nucleus accumbens). Extrapyramidal rigidity, hyperkinesia are observed. The bulbar sphere and brain activity are disrupted, mood and speech are altered, which is accompanied by signs of liver damage.

Mechanism of toxic effect of copper. Copper catalyzes free radicals as a prooxidant and enhances peroxidation of lipids. It disrupts the function of the plasma membrane and mitochondrial membrane, lysosomal enzymes are released into the cell, the function of DNA and proteins is disrupted, the antioxidants lead to a decrease in glutathione and Vitamin E levels. Malonovoy dialdehyde, formed as a result of peroxidation of lipids, enhances collagen synthesis and leads to fibrogenesis. The accumulation of copper in the horny (rogovitsa) membrane destsemet membrane is manifested by the formation of a Kaiser-Fleischer ring (Fig . 110).

110 photos

Specific to Wilson-Konovalov disease.

Clinical status: Patient S. complained of general weakness, memory loss, daytime sleepiness, slurred speech during speech, impaired spelling due to trembling hands, intermittent nosebleeds, swelling in the legs, and dry cough upon admission at the age of 25 years . He considers himself a patient since 2011 and developed jaundice on the skin after performing an ARVI. The patient was admitted to the gastroenterology department of the hospital, diagnosed with cirrhosis of the liver, portal hypertension syndrome, ascites. The patient was prescribed 300 mg prednisolone, infusion therapy in combination with hepatoprotectants, and the patient's condition improved and he was discharged from the hospital. He was later hospitalized several times and given diuretics along with hepatoprotectants, his condition improved slightly. From 2011 to 2012, he regularly took prednisolone in a 30 mg tablet, and the dose was gradually reduced and completely eliminated from the hormone. In 2011, the activity of the enzyme AlAT increased to 122 one / l, AsAT to 118 one / l, IF-549.4 one / l, GGTP-133 one / l, total bilirubin to 47 mmol / l, ceruloplasmin to 0.072, platelets to 40,000, leukocytes 1 , Decreased to 8,000. Antibodies against neutrophil cytoplasm, cardiolipin, mitochondria gave a negative result. In October 2013, an endoprosthesis was placed in the left pelvic joint due to aseptic necrosis of the femoral head. Due to blood loss during surgery, 600 ml of erythrocyte mass and 600 ml of plasma were transfused. At the endocrinologist, hypothyroidism was accounted for with grade II. The menstrual cycle is disrupted. Over the years, his condition worsened, and his ability to speak and memory declined. When the history of the disease was studied, the spleen was enlarged from childhood.

On examination, the condition is moderate, the skin is pale, there are vascular asterisks, the tongue is moist, clean. Vesicular breathing in the lungs, shortness of breath in the lower parts. Heart tones are muffled, rhythmic, heart rate 90 beats, AQB 110/70 mm.sim.ust.teng. abdomen soft, slightly painful in the projection of the gallbladder, liver under the rib arch, spleen +3 cm. Stool 1 time per day unchanged. There is swelling in the legs up to the upper third of the knee. Urinary incontinence is the same as urinary incontinence.

Inspections conducted; Blood analysis - erythrocytes 4 thousand, leukocytes 1.8 thousand, hemoglobin 122 g / l, erythrocytes 4.2 million. Biochemical analysis of blood; total protein 52.7 g / l, bilirubin 47.2 mmol / l (right-27.9 and wrong 19.3), AlAT-70, AsAT-118. GGTP-109, IF-159 increased to one / l and ceruloplasmin decreased to 0.072 g / l. On the coagulogram, the prothrombin index was 45% (1.79), the thromboplastin time was partially activated, and the fibrinogen was 1.82 g / l.

UTT-hepatosplenomegaly, diffuse changes in the liver (symptoms of cirrhosis). Gallstone disease, chronic pancreatitis, small pelvic fluid.

Oculist's conclusion - Kaiser-Fleischer ring, EFGDS grade 3 esophageal varices, superficial gastritis, bulbitis.

Color dopplerographic examination of the portal system-portal vein postrombophlebitis, partial recanalization, portal hypertension.

MRI of the brain reveals hepatocerebral dystrophy, less developed external hydrocephalus.

The diagnosis was made on the basis of the above clinical, laboratory, instrumental examinations.

Mixed form of Wilson-Konovalov disease. Cirrhosis of the liver, Class B on Child-Pew. Portal hypertension syndrome. Dilation of the esophageal veins is 3 degrees. Splenomegaly with hypersplenism. Severe ataxic damage to the brain. Gallstone disease. Period of remission of chronic pancreatitis. Period of remission of chronic superficial gastritis. Duodenitis remission period.

Treatment plan: D-penicillamine (cuprenyl) 250 mg X 1 m / k, then the dose was increased from 250 mg to 1 g. Vitamin B_1 and B_6 m / o. The patient’s condition worsened for 2 weeks despite pathogenetic therapy.

The patient developed general weakness, pain under the right rib. The patient excreted 492 mcg of copper (N-2-80mkg) per day in the urine. After 2 weeks, the patient's condition improved. Weakness decreased, speech improved slightly, and copper excretion in the urine increased from 38 to 40 mcg (N-2-80 mcg).

Repeated blood tests revealed a platelet count of up to 52,000 g / l, leukocytes up to 2,000, and erythrocytes up to 4.3 million. Biochemical analyzes showed total protein-52.3 g / l, total bilirubin 23 μmol / l, AlAT 40 a / l, AsAT-50 a / l.

Conclusion: Wilson-Konovalov disease is a rare disease with copper accumulation. Patients with chronic hepatitis and cirrhosis should be screened for patients with an increase in the amount of aminotransferases in the blood,

the development of psychoneurological symptoms and tremors, changes in behavior, liver failure of unknown etiology.

Gilbert's syndrome

Gilbert's syndrome is a good quality congenital functional hyperbilirubinemia, an inherited disease. It is accompanied by an increase in the amount of unbound bilirubin. This condition is caused by a mutation in a gene that encodes an enzyme involved in bilirubin metabolism. Jaundice develops without signs of liver damage. This closes the bile ducts and causes hemolysis. It is an inherited disease in which bilirubin metabolism is disrupted due to a decrease in the activity of the enzyme glucuronyltransferase (30% below normal) or a deficiency. Autosom is passed from generation to generation through a dominant pathway, with no functional or morphological changes in the liver. It is more common in men during adolescence. According to some authors (Strassburg CP), Gilbert syndrome occurs in 3-9% of the population in European countries.

The causative agents of the disease are a decrease in the activity of the enzyme glucuronyltransferase, which occurs as a result of mutations in the gene encoding the enzyme involved in the metabolism of bilirubin from endogenous factors. Exogenous factors include starvation, physical exertion, exposure to alcohol and toxic substances, long-term drug intake, and concomitant diseases.

Symptoms of the disease; rapid fatigue, weakness, dizziness, insomnia, sleep disturbances, discomfort under the right ribs, varying degrees of yellowing of the sclera (physical exertion, alcohol consumption, no itching of the skin after infectious diseases) , symptoms such as the formation of stones in the gallbladder and tract. In rare cases, dyspepsia, loss of appetite, sour fullness in the mouth, belching, irritability, nausea, in rare cases, vomiting, constipation or diarrhea, frequent diarrhea, abdominal discomfort, o Symptoms such as numbness, tingling pain under the ribs are annoying. Pain mainly occurs when the diet is disrupted, consuming fatty, spicy foods. Sometimes an enlarged liver can be detected. Sensitivity is mainly when the diet is disturbed, fatty, spicy foods, alcohol, anabolic steroids, sex hormones, glucocorticoids, antibiotics, consumption of nonsteroidal anti-inflammatory drugs, stress, physical exertion, trauma, operations, viral infections , hepatitis leads to disease exacerbation.

Diagnosis Total bilirubin may increase to 21-51 mmol / l. Normal total bilirubin is 5.1-17.1 (8.6-20.5) mmol / l, Bound 1.7-5.1 mmol / l, Unbound 3.4-12 mmol / l

In Gilbert's syndrome, special diagnostic tests are performed — starvation test; If the patient follows a low-calorie diet (400 kcal per day) for 48 hours, the amount of bilirubin increases by 50-100%. Bilirubin is detected at breakfast and after 2 days. Phenobarbital test - after consumption of this substance, the activity of the enzyme glucuronyltransferase increases and the amount of bilirubin decreases.

Basic diagnostic criteria : .

-Strong, intermittent jaundice, physical exertion, nervousness, dietary disorders, fatty, spicy foods, alcohol, anabolic steroids, sex hormones, glucocorticoids, antibiotics, nonsteroidal anti-inflammatory drugs consumption, stress, physical exertion, trauma, surgeries, viral infections, jaundice in hepatitis.

-Hyperbilrubinemia is observed due to the unconjugated fraction.

-Hyperbilrubinemia due to this fraction is also observed in patients' relatives.

- Bilirubin returns to normal when treated with phenobarbital.

Comparative diagnosis of Gilbert syndrome and congenital hemolytic anemia.

Gilbert's syndrome -Time of onset of primary symptoms -11-30 years, Splenomegaly-in rare cases, the effectiveness of fer phenobarbital accumulated in hepatocytes ment-lipofuzcin, the effectiveness of phenobarbital-effective.

Decreased activity of the enzyme glucuronyltransferase is characteristic.

Congenital hemolytic anemia - The time of onset of primary symptoms is up to 10 years, splenomegaly-permanent. Anemia-specific, microspherocytosis-specific, erythrocyte survival-reduced, erythrocyte osmotic resistance, decreased, biliary stones-common, enzyme accumulated in hepatocytes-hemosiderin, phenobarbital-ineffective.

Dabin - Johnson syndrome

Dabin-Johnson syndrome is an enzymopathic (fermentopathic) jaundice that belongs to the group of rare hepatoses and is a pathological process characterized by impaired secretion of bound bilirubin from hepatocytes to bile capillaries. This in turn leads to regurgitation of bilirubin, i.e. reabsorption. This is due to a hereditary defect of the ATF-responsive transport system in the tubules of hepatocytes (postmicrosomal hepatocellular jaundice). At the same time , the excretion of dyes (sulfbromphthalein sodium) is reduced, hepatocyte pigments that do not store iron, there is no pathology in the gallbladder. The reason that bilirubin is retained in hepatocytes is due to adrenaline metabolism, where bilirubin and melanin accumulate. Later melanotic hepatosis develops.

According to the literature, the **Dabin-Johnson** s **indrome is** more common among Iranian Jews, accounting for 1: 1300 people. Jaundice, which is common and recurrent in families. 60% of patients have a decrease in prothrombin levels, which is due to low factor VII. 70% of patients are young and the remaining percentage is after 50 years.

Dabin-Johnson s **indroma is an** inherited disease that is inherited through an autosomal recessive pathway. A gene defect is caused by a mutation in the ion channel gene (cMOAT) that transports organic anions. As a result, the hepatobiliary transport of bilirubin and organic anions is disrupted. The amount of bound bilirubin fraction increases in the blood and bilirubinuria is observed in the urine because the bound bilirubin is well soluble in water. If the amount of bound bilirubin exceeds 0.3 mg / l

(5.2 μmo l / l) and total serum bilirubin exceeds 2.0 mg / l (34.2 mmol / l), we say conjugative jaundice (or the correct bilirubin fraction) More than 15% of total serum bilirubin, which increases by 2.0 mg / l).

In the Dabin-Johnson s **indrome, the** color of the liver is greenish-brown or dark brown, and black spots can be seen on the surface. It is associated with impaired secretion of tyrosine, tryptophan and phenylalanine. Pigment also accumulates in the spleen. Hepatocytes and Kupfer cells are filled with black pigment, which looks good when cramped with lipofucin. Pigments are found in solid bodies bound to lysosomes. Lysasomes are uneven, enlarged, oily, membrane-bound. In patients, jaundice syndrome is persistent and is often manifested by itchy skin. The disease can go asymptomatic for years. Most often, young men get sick. The diseased liver does not excrete bilirubin, bromsulfalein, contrast agents for cholecystography. For this reason, the amount of bilirubin exceeds the norm, the amount of bromsulfalein probe, alkaline phosphatase changes. In patients with gallbladder shadow is not visible during the examination due to a violation of the excretion of the contrast agent. On separate examination, the amount of bound bilirubin in the blood is increased, in connection with which bilirubinuria is observed.

Basic diagnostic criteria:

- It is most common in men
- The first clinical signs appear from birth to 25 years of age.
- The main symptom is chronic non-severe jaundice.
- Jaundice is manifested by rapid fatigue, nausea, and sometimes abdominal pain. Patients have common symptoms-fatigue, weakness, feeling unwell, dyspepsia-poor appetite, diarrhea, pain under the right rib, colic, fever. Patients' throats become red.
- The liver can grow 1-2 cm
- Urine is dark in color, bilirubinuria is observed.
- In many patients, the enlarged, mostly conjugated fraction in the blood is increased.
- There will be no change in the blood test.

Of course, laboratory tests.

General blood test

General urine analysis

Bilirubin in the blood is detected-bound increases

Bilirubin in the urine

Bilirubin is reduced when taking proba-phenobarbital with phenobarbital.

Enzymes are detected in the blood (AcHT, AlAT, GGTP, SHF) are slightly increased

The amount of bromsulfalein in the probe serum increases when the curve is drawn for 45 min. compared to 90 min.ga.

The amount of coproporphyrin in the daily urine does not change

The type I isomer of coproporphyrin is increased in daily urine.

Laboratory tests performed according to the instructions.

Virological examination Hepatitis B, C, D, etc.

Instrumental examinations (liver, spleen, gallbladder, bile duct UTT)

Cholecystography with oral and intravenous contrast (in the disease there is no contrast in the gallbladder)

According to the instructions;

Puncture biopsy (to find pigments characteristic of liver cells)

Diagnostic laparoscopy (black staining of the liver)

Differential diagnosis is made with syndromes Krigler-Nayar, Gilbert, Rotor, viral hepatitis, chronic hepatitis, cholestatic syndrome, mechanical jaundice, liver cirrhosis, etc.). Diagnosis is assisted by bromsulfalein test and other functional tests, family nature of the disease, the onset of the disease (from childhood, adolescence), laparoscopy, puncture biopsy, etc.

Rotor syndrome

Rotor syndrome is a hereditary pigmented hepatosis, the development of the disease is similar to Dabin-Johnson syndrome, it is milder, but the bilirubin-excretory function of hepatocytes is milder. The difference is that bromosulfalein gives a negative result 2 times in the test, ie the concentration of dyes does not increase. Cholecystography shows a gallbladder, black spots do not form on the liver cells. Morphological examination may show signs of fatty dystrophy in the liver. **In rotor syndrome** - a slight increase in the amount of bound bilirubin in the blood and the excretion of coproporphyrin in the urine, the activity of liver enzymes is not impaired. There are no other changes in the liver. **Rotor syndrome is an** inherited disease that is inherited through an autosomal recessive pathway and the disease develops from childhood. The clinical course of the disease is often asymptomatic, manifested by jaundice syndrome. In Dabin-Johnson syndrome, the excretion of bromsulfalein test is impaired, while in Rotor syndrome, its absorption into liver cells is impaired. In Rotop syndrome, the liver, gallbladder, and bile ducts appear when examined with lidofenin.

Basic diagnostic criteria:

- Chronic jaundice, not severe.
- Subjective complaints will not be clear.
- The liver is normal in size
- Hyperbilirubinemia due to conjugated fraction.
- Periodically, bilirubinuria is observed in the urine.
- There will be no change in blood tests and liver tests.
- Liver biopsy shows a normal histological appearance and no pigment accumulation is detected.

Portal hypertension

Portal hypertension-liver syndrome is caused by a violation of the flow of blood from the portal vein to the liver, ie from the outside and inside

c. characterized by closure or compression of the port and an increase in pressure in it.

Etiology and pathogenesis. Circulatory disorders in the portal veins are divided into 3 groups: intrahepatic (sinusoidal), subhepatic (postsinusoidal), suprahepatic (presinusoidal). Complications of circulatory disorders in the portal vein are observed in viral or alcoholic liver cirrhosis, as well as in cirrhosis of the liver observed in Wilson-Kanavalov disease, when the connective tissue on the interstitial surface grows. In the subhepatic group, portal vein occlusion is observed: inflammation of the wall, thrombosis, obstruction of the tumor, enlarged lymph nodes (lymphagranulomatosis and leukemia), and in rare cases, schistosomiasis, sarcoidosis. The subhepatic group includes hepatic veins occlusion, Badda-Kiari syndrome.

Clinical manifestations

In the portal hypertension clinic-clinical triad is observed:

1. Occurrence of collateral venous circulation;
2. Ascites
3. Enlargement of the spleen.

Symptoms in this triad are due to the development of anastomoses as a result of increased pressure in the portal vein. 1. In the area of hemorrhoidal venous congestion (anastomosis between the inferior vena cava vein and hemorrhoidal veins makes it difficult for blood to enter the inferior vena cava) due to which the hemorrhoidal veins dilate.

2. Esophageal-gastric vein tangled area (from the left gastric vein to the esophageal tangle and from the semicircular vein to the superior vena cava. Due to this, varicose expansion of the inferior esophageal veins is observed. 3. With abdominal veins anastomotic umbilical veins and abdominal wall, diaphragm vein, which should carry blood to the superior and inferior vena cava. The cause of ascites can be explained as follows: 1. Blood serum is released into the abdomen due to increased pressure in the veins of the abdomen. 2. Oncotic pressure of the blood decreases because the synthesis of albumins in the liver decreases. and the production of antidiuretic hormone by the liver due to a decrease in the inactivation process of water and sodium to keep a large amount of ascites development role. **bemos On examination, the** patient was thin, the skin and subcutaneous fat layer was poorly developed, the abdomen was enlarged, the abdominal wall veins were dilated, vascular asterisks, jellyfish head symptom, umbilical bulge, bleeding marks from the nose, gums, etc. We can see the changes. When patients lie down, their abdomen expands to both sides. On palpation, the symptom of fluctuation in the abdomen is positive, it is possible to detect an enlarged spleen. Information on liver and spleen dimensions can be obtained on percussion.

Liver cell deficiency

In the liver, there is a deficiency of liver cells due to a decrease in the number of hepatocytes that fully perform their function . Liver failure is divided into acute and chronic types. Three stages are

distinguished; compensated; decompensated and terminal or dystrophic. The third stage ends with a coma. Acute and chronic liver failure are different.

Etiological factors ; acute and chronic liver failure is caused by poisoning with toxic substances, viruses, tumors, protein and vitamin deficiencies, cirrhosis of the liver and bqs. 30-60% of patients with cirrhosis die from hepatic coma, which causes hepatic encephalopathy. Factors leading to hepatic encephalopathy:

-alcohol;

- bleeding from the digestive tract;

-collapse, shock;

- The effect of drugs;

-consumption of large amounts of protein;

-infection.

In hepatic coma, water-electrolyte balance changes due to increased diuresis and vomiting. Clinical manifestations include hepatic encephalopathy, developing ascites and hemorrhagic syndromes, progressive jaundice. Hepatic encephalopathy is a syndrome characterized by acute, chronic liver disease or neurological and psychoemotional disorders as a result of the passage of toxic substances from the intestines into the blood when a shunt is formed between the vessels of the portal veins and the general circulatory system.

Underlying liver failure is deep hepatic dystrophy and necrobiosis of liver cells. Such changes in the liver lead to a violation of its detoxification function, toxic substances formed in the intestine (indole skatol) and as a result of metabolism fall into the bloodstream from the collaterals and poison the body.

Pathogenesis. In hepatocellular insufficiency, all functions of the liver are impaired, leading to increased toxicity, hemorrhagic syndrome, which occurs when the synthesis of coagulation factors is impaired, the development of ascites and edema due to decreased albumin synthesis. The development of hepatic encephalopathy is associated with the retention of nitrogen compounds in the body, which leads to edema and functional disorders.

Clinical manifestations. The clinical symptoms of this disease are primarily encephalopathy, which leads to cirrhosis, death, and deep liver coma. There are several stages of hepatic encephalopathy:

Stage 1 (prodromal period) is a disorder of the emotional environment and sleep, inability to sleep at night is accompanied by drowsiness during the day.

Stage 2 - deep neuropsychic disorders. This is characterized by "tremor" of the fingers.

Stage 3 - pre-coma: loss of consciousness - sopor, dysarthria, the appearance of pathological reflexes, increased "clapping" tremors, the appearance of Cheyn-Stokes or Kussmaul breathing type. There is a sweet-smelling odor in the mouth, a decrease in liver size, jaundice that develops without itchy skin, hemorrhagic syndrome, hypoalbuminemia, fever.

Stage 4 is the period of hepatic coma, areflexia is typical for this period.

In addition, in hepatocellular insufficiency there is hypoalbuminemia, hyperbilluribinemia, as well as a decrease in the amount of prothrombin and cholesterol in the blood.

Patients are mainly bothered by asthenoneurotic, dyspeptic, jaundice, edema, hemorrhagic syndromes, and fever. Symptoms of endocrine disorders can be seen in patients and mainly in men (testicular atrophy, gynecomastia, hair loss in the scalp, underarms and other areas), which is associated with inactivation of estrogens. In women, there are changes such as atrophy of the uterus and mammary glands, menstrual disorders.

Practical training 7

Liver and bile ducts. Clinic, diagnosis of chronic hepatitis .

1. Practical training module .

Training time - 4 hours	The number of students 1 : 0 to 12
Form of training	Practical training
Practical training plan	Methods of laboratory and instrumental examination of patients with diseases of the liver and biliary tract (cholecystography, liver scan, examination of the liver and gallbladder with ultrasound). Computed tomography. Symptomatology of chronic cholecystitis and hepatitis.
The purpose of practical training :	Familiarity with the methods of laboratory and instrumental examination of patients with diseases of the liver and biliary tract . Symptomatology of chronic cholecystitis and hepatitis.
Teaching style	Inquiry. Demonstration of patients, interactive teaching methods, practical skills.
Form of teaching	In small subgroups.
O ' unit equipment	Calls to O Training Guidebook , practical Typo content , projectors , computer .
Training mode	Methodically equipped auditorium.
Monitoring and evaluation	Oral control: questions and answers, tests, problem solving.

1.2 . ***Technological card of practical training***

See the stages of hearts and q ti.	Educator	Learners
In the preparatory stage of q	16. See the audience to control the purity karîm 17. Checks students ' readiness for training 18. And control q karîm	
1. bullet and training g contain	1. Preparation of educational content on the topic. 2. Preparation of presentation slides for the introductory	

levels of intra q (10 da q i q a)	speech 3. Develop a list of references used in the study of science	
2 - the main stages of diarrhea q (160 da q i q a)	1. Divide students into small groups and ask questions on the topic. 2. Uses display posters 3. Uses slides, multimedia 4. Conducts treatment 5. Summarizes and summarizes the information provided on the basis of topics, encourages and actively evaluates the active participant students	They are divided into small groups They watch They participate They listen and answer questions
2- final press q ich (10 da q i q a)	1. Concludes 2. Provides independent work 3. Gives homework	He listens Takes notes Takes notes

3. Assessment of students' theoretical knowledge:

A) Frontal method:

21. 1. Describe normal bilirubin metabolism
22. Explain the determination of bilirubin
23. Name the galactose and adrenaline test
24. Explain the normal protenogram
25. Describe protein precipitation tests
26. What is Thymol and Formal Test
27. How the liver is involved in copper and iron metabolism
28. Examination of the bile ducts and gallbladder for radiography and rengenoscopy
29. Describe the method of splenoportography
30. What is holegraphy
31. What is cholecystography
32. Describe the radiostop inspection method
33. What is exography
34. Describe a liver biopsy
35. What is loparoscopy
36. What is scanning
37. The role of computed tomography in the diagnosis of liver and biliary tract diseases
38. Etiopathogenesis of chronic cholecystitis
39. Clinic of chronic cholecystitis
40. Clinical and diagnostic diagnosis of chronic cholecystitis

Brainstorming method

Basic rules :

- Not to mention the shortcomings that hinder the emergence of the idea
- The height of ideas and thought, because the more unusual an idea, the better it is
- Accept many offers
- Combination of ideas and their development
- Present the idea succinctly without argumentation

- Divide the group into two: thought generators and thought analysts

This method allows you to argue ideas and opinions, your own personal opinion, to find the optimal solution in any situation.

Autoimmune hepatitis

Autoimmune hepatitis (AIG) is a chronic disease of unknown etiology of the liver, characterized by periportal or extensive inflammation and manifested by pronounced hypergammaglobulinemia and the appearance of broad-spectrum autoantibodies in serum.

The first symptoms of the disease are a ratio of 8: 1 between women and men

It begins to appear between the ages of 10 and 20-30, and the second peak period occurs after menopause.

Autoantibodies are differentiated depending on the spectrum

Type 1 AIG (85%)

Type 2 AIG (15%)

Epidemiology. The prevalence of AIG ranges from 0.1 to 1.9 per 100,000 population in Europe and North America. According to European and North American statisticians, autoimmune hepatitis accounts for 20% of patients with chronic hepatitis. This figure is less than% in Asia and Africa, probably due to a lack of in-depth research. In Japan, AIG accounts for 5% of chronic hepatitis.

Etiology . AIG is characterized by the association of cell imbalances with a number of antigens that control immune processes (MNS; NLA in humans). The predisposition to AIG is primarily in the HLA B8 and DR3 gene alleles. The incidence in homozygous DR3 * 0101 ranges from 4.2 to 14.7% in **type AIG I.**

After corticosteroid discontinuation, HLA-DR3-positive individuals are characterized by early onset, severe course, and frequent recurrence. A1, B8 and DR3 allelic genes are rapidly inherited from generation to generation. PL attention Bittencourt and colleagues (2001), Geng associated with genetic HLA Class III a llellari TNF A * 2 variant alleles linked to the DRB 1 * 0301 noted.

The development of **AIG type II is** poorly understood due to its low prevalence. AIG type II HLA DR3, DQ2, V 14, C4AQ0 Studies in the UK and America have shown that DRB50101 - DRB11501 plays a role. Along with antigens, the transcription factor also plays a role. In this case, the type I autoimmune regulator (AIRE-1), AIRE-1 is expressed in the pancreas, lymph nodes, paracortical and cerebral substance, spleen, fetal liver. AIRE-1 is involved in the formation and maintenance of immunological tolerance.

AIRE-1 defect has been shown to lead to the development of autoimmune polyglandular syndrome, and its component is AIG in 10-20%

of cases. Such a transcription factor mutation leads to a loss of tolerance to liver autoantigens

Although these factors are not considered to be a direct etiological factor, they are a trigger for the development of AIG. Most observations showed that AIG developed after infection with viral hepatitis A, B, C, herpes (HHV-6 and HSV-1), and Epstein-Barr viruses. The role of viral infection in the development of AIG can be seen as twofold. On the one hand, after the virus enters the body, the production of cytokines and IFN α increases, which increases the expression of HLA I, HLA class II in hepatocytes. Similarly, the HLA II antigen plays a cellular role. In the late stages of infection, specific cellular and humoral immune mechanisms are activated. This is because most viruses produce a protein with antigenic properties similar to human protein, and in response, antibodies and T-lymphocytes can easily react with these proteins. Under certain conditions, the similarity of viral epitopes and autoepitopes leads to the activation of autoreactive immunocompetent cells under certain conditions. In the tolerant state, normally autoantigens support autoreactive T helpers in small concentrations. The infectious agent causes the activation of autoreactive T helpers by inducing the expression of CD2, CD28, LFA-1. Activated autoreactive lymphocytes have the property of interacting with autoepitopes (hepatocytes, biliary epithelial cells). B-cell epitopes can cause tolerance disruption, and the mechanism is different. Most autoreactive B lymphocytes may not be active, but may be in a state of tolerance to low concentrations of antigen, either by interacting with T helpers, or by recognizing latent epitopes. As a result of external influences, the stimulator function is taken out of the tolerance state by autoepitopes and T-helpers and activates B lymphocytes. The peptide cytochrome P 450 2D6 (CYP2D6), which contains 33 amino acid residues, is included. This peptide is considered the primary autoepitope and to which T-lymphocytes are sensitized. These have the property of damaging hepatocytes in the AIG-2 type. The structure of this peptide is similar to that of type 1 (HSV-1) herpes simplex virus IEP-175.

In addition to viruses, environmental factors also play a role in the transmission of immunopathological processes, including reactive metabolites of drugs. Phase I and II drugs enhanced by drugs and viruses are targets of the autoimmune reaction of metabolic enzymes CYP and UDF-glucuronyltransferase 1. This means that different agents can cause autoimmune processes and are directed against molecular targets.

AIG type 1 circulates in 70-80% of antinuclear autoantibodies, 50-70% of smooth muscle antibodies, and in rare cases atypical antineutrophil cytoplasmic antibody p-type (pANCA) in 60% of patients.

AIG type **2** autoantibodies against liver and kidney microsomes (antiLKM-1) are found in 100% of patients. At the base, anti-LKM-3 and liver cytosol protein are combined with anti-LC-1.

AIG has antibodies against **type 3** soluble liver (anti SLA) and liver pancreatic (antiLP) antigens. This type is less segregated and is considered AIG 1 pod type because the clinical course with AIG-1 type and the frequency of encountering ANA i SMA serologic markers occur in 74% of cases.

The ANA in AIG is heterogeneous in nature and is associated with single- and double-stranded DNA, ribonucleoproteins, transport RNA, histones, cyclin A, and other nuclear components. In general, ANA and SMA disappear when immunosuppressive therapy is given . Sensitization of T lymphocytes to ASYP-R day CYP2D6 autoepitopes was found in AIG. Similar sensitized lymphocytes were found in inflammatory infiltrates of the liver of patients with AIG-2. 85% of these lymphocytes carry the phenotype CD4 + CD8 and produce g-interferon, interleukin-4. This is evidenced by the type 1 subpopulation of T-helper.

Thus, the underlying pathogenesis of AIG lies in the following mechanisms;

Genetic predisposition to the emergence of autoimmune processes (decreased immunological control over autoreactive clones of lymphocytes)

The decisive influence of the anonymous factor.

High expression of liver autoantigens and class II HLA

Activation of autoreactive clones of T- and B-lymphocytes

Inflammatory mediators and liver tissue injury, development of systemic inflammation.

AIG clinical picture

Clinical;

- 1 - helplessness
- 2 - arthralgia
- 3 - jaundice
- 4 - anorexia
- 5 - nausea
- 6 - Unpleasant condition under the right rib
- 7 - myalgia (polymyositis)
- 8 - skin rash
- 9 - amenorrhea
- 10 - fever

Patients inspection cardiovascular star, groin bright purple planing in the field of the white line (string) , due to a decrease in skin tromdotsitlar hemorrhagic and spoil the beauty of rashes, body fat kushingsimon to see. On palpation, hepatomegaly, splenomegaly are observed. 10-40% of patients experience pain in the liver area under the right rib. Symptoms of portal hypertension and hepatic encephalopathy occur when complicated by liver cirrhosis. Percussion can detect enlargement of the liver and spleen. Swelling, redness may be seen when the joints are examined.

X-ray signs of arthritis that is, joints can be narrowed and the erosion of the articular surface of the slot.

Laboratory;

- 1 - Hemolytic anemia
- 2 - ECG increase
- 3 - leukopenia
- 4 - thrombocytopenia
- 5 - anemia
- 6 - hyperbilirubinemia due to direct bilirubin (2 - 10 times, in 83% of patients)
- 7 - transaminaseemia (5-10)
- 8 - hypoalbuminemia
- 9 - hypergammaglobulinemia (up to 2 times)
- 10 - Rheumatoid factors increase
- 11 - Slight increase in alkaline phosphatase
- 12 - Increased levels of immunoglobulin G (IgG)
- 13 - an increase in the amount of a fetoprotein

Complications of AIG ; Lymphadenopathy, pneumonitis, fibrous alveoli, pleurisy, myocarditis, thyroiditis hosimoto (10-13% of patients), glomerulonephritis, tubulointerstitial nephritis, shegren's syndrome, ulcerative colitis, diabetes mellitus, hemolytic anemia, idiopathic thrombocytopenia.

Complications of the skin are red flatulence, allergic capillaries, vascular wounds on the hands and feet, hemorrhagic vasculitis, psoriasis, nodular erythema. Skin candidiasis is observed in the mucous membranes, ectodermal tissue damage in 80% of patients, intestinal damage in 30% of patients.

The basis of the case. Immunosuppressive therapy Glucocorticoid therapy - prednisolone 5mg, the mechanism of action in order to reduce the production of cytokines. Triamcinalone and dexamethasone are used as fluorinated glucocorticosteroids. A new generation of glucocorticosteroids, budesonide, can also be given.

Immunosuppressants 6 mercaptopurine series drugs - azathiopyrine 50mg - have antiproliferative effects. Cyclosporine and macrolimus are given because T is a selective blocker of the cellular immune response. Cyclophosphamide is given 1-1.5 mg / kg. Group of **hepatoprotectors** essential 5.0; gepamerts-10.0; **Drive up liquid** medicine bositalari xolenzim, allaxol, Liv-52 wards; **Total support** group B vitamins; **Desintoxication** therapy poliglyukin, reopoliglyukin, infezol, amminosol and prescribed bq.

AIG treatment regimen

1	Prednisolone	Prednisolone and azathiopyrine

2	60 mg / day - 1 week	Prednisolone
3	40 mg / day - 2 - weeks	30 mg / day - 1 week
4	30 mg / day - 3-4 - weeks	20 mg / day - 2 weeks
5	20 mg / day - mg / day - a constant maintenance dose	15 mg / day - 3-4 weeks
6		10 mg / day - a constant maintenance dose
7		Azathiopirin *:*
8	Relative contraindication	100 mg / day - 1 week
8	Postmenopausal	50 mg / day - continuous after 1 week
		Contraindications
10	About steoporosis	pregnancy
11	Diabetes	cytopenia
12	Arterial hypertension	Poor quality tumors
13	Cushingoid syndrome	Indications and side effects against azathioprine

If conservative treatment does not work, a liver transplant is performed. 90% of patients live more than 5 years. Mycophenolate mofetil is used at this time. This drug is a potent immunosuppressant in transplantology. **Chronic viral hepatitis**

Chronic viral hepatitis is a chronic diffuse inflammation of the liver of viral etiology. Hepatitis B is a disease caused by viruses that are widespread and severe in the population. occurs with liver damage and other manifestations.

Epidemiology. According to the World Health Organization, more than 350 million people worldwide suffer from hepatitis B, mostly in developing countries. Hepatitis B is associated with hepatitis B in 7% of patients. The disease damages the liver and kidneys and causes oncological disease in 10% of cases . Each year, more than 700,000 people die from the disease . Patients take antiretroviral drugs until a liver transplant is performed. The disease is difficult to treat and after a few years, 60-80% of patients develop liver cirrhosis and cancer. Hepatitis B virus is resistant to the external environment and lives for a week on dried blood droplets, blades, razors, syringe needles.

Etiology. In recent years, viral etiology has received increasing attention. Hepatitis B and hepatitis C viruses are mainly transmitted through blood transfusions, damaged surgical instruments, needles, patients using toothpaste, hairdressers' equipment and bqs, and its asymptomatic transmission is common for many years. In particular, 3 types of viruses are most common - B, C and D. At the peak of the disease in 1 ml of blood are found billions of lab viruses .

111 photos

Infectious routes - vertical route, blood transfusion, surgical, gynecological, dental procedures, barber razor (razor), genital tract (sperm, pubic lip (vagina) mucus), syringe needles, transplant (liver) genitals, saliva, toothbrushes, tools used in cosmetology (manicure, pedicure), piercings and tattoos in salons. Therefore, care should be taken to use the services of hairdressers and cosmetologists. The disease is transmitted from damaged skin and mucous membranes. The incubation period is 2–6 months.

Risk factors for the transmission of viral hepatitis B and C.

1. History of blood transfusion and its drugs.
2. Taking drugs.
3. Sexual intercourse; (anal sex, sexually transmitted diseases, history of five or more sexual partners, sex with carriers of viral hepatitis B and C).
4. Needle treatment.
5. Work in the hemodialysis department and blood transfusions.
6. Work in surgical departments, intravenous injectors, huqna conductors, endoscopic examiners, laboratory assistants and others.
7. Sharing common hygiene items, especially for patients (razors, toothbrushes, massagers, shower and bath utensils, etc.).
8. Piercing of the skin (piercing of the ear and nose for the purpose of sharpening), tattooing.
9. Medical staff and students of the medical institute .
10. G ePati B with infected family members .
11. Q amoqxona prisoners.

According to Israeli scientists (Rifat Safadi and Daniel Shuval, professors at Hadassah Medical Center), there are the following forms of the disease;

Acute (period of jaundice);

Acute heat (yellow-free period);

Subclinical;

Chronic;

Clinic. If the disease is acute, it occurs in subclinical or jaundice, cholestatic and chronic form. Inquiry into patients is important in the diagnosis of hepatitis B and C. Fatigue, nausea, pain in the epigastric area, diarrhea, skin rash, pain in the muscles and joints, jaundice are disturbed. In patients, an increase in body temperature, headaches are disturbed, and after a few days the appetite is suppressed and jaundice appears on the skin. In patients with chronic viral hepatitis, discomfort and pain appear under the

arch of the right rib, the color of the urine becomes dull, the stool becomes discolored. Chronic hepatitis C is characterized by a latent course, which manifests itself several years later and can lead to liver cirrhosis and liver-cell carcinoma. When hepatitis B + D comes together, the disease becomes severe.

In addition to the development of cirrhosis of the liver, there is a syndrome of liver failure - bleeding from the esophagus, intestines, gums, uterus, jaundice, ascites, encephalopathy. This syndrome is observed during the decompensation period of cirrhosis or in the severe course of acute hepatitis. **When patients are examined, we see that the** subcutaneous fat layer is poorly developed and the color of the skin and visible mucous membranes is yellow. **Palpation** reveals that the liver is enlarged, the surface is slightly hardened, and there is pain in it. **When the liver is percussed** , it is found to be enlarged in size.

Instrumental inspection. Fibroscan. In viral hepatitis, hepatocytes enlarge and the liver hardens. If the hard tissue of the ultrasound wave in violation of development of the greater speed and fibrosis rate is too high. The operation of the apparatus is based on the results of histological examinations. The harder the liver, the higher the rate of fibrosis development. To determine the degree of fibrosis, the apparatus sends a pulse wave. Its velocity is then measured from the chest wall using an ultrasound wave package . Abdominal UTT provides sufficient information and can be used to detect enlarged and hardened liver.

Morphological signs of chronic hepatitis B. Hydropic dystrophy of hepatocytes; Acidophilic body detection (Kaunsilmen body); -Focal foci in hepatocytes (necrosis is replaced by apoptosis); Lymphohistiocytic infiltration; We see changes such as portal tract fibrosis;

Morphological signs of chronic hepatitis C. Large droplet fatty dystrophy is characterized by the formation of lymphoid nodules as a result of lymphoid aggregation in the portal tract, purulent cholangitis in the bile ducts is accompanied by lymphoid and plasma cell infiltration. Cellular infiltration is also common in periportal zones. The development of liver cirrhosis is possible due to the formation of fibrous tissue.

Laboratory examination. HB_sAg is a surface antigen of viral hepatitis B. Its presence in the blood is typical of virus carriers and patients with chronic hepatitis. HB_cAg is the core antigen of viral hepatitis B: it is expressed by liver cells. It is found in the blood only when parts of the virus are broken down. HB_eAg - Ag e: its presence in the blood indicates active replication of the virus and its ability to cause maximum blood damage. In addition to the detection of antigens in the blood, the detection of antibodies to the 3 antigens shown is also of great importance. There is an increase in ALT, AST, bilirubin in the blood. Sandy nuclei Hb core Ag marker.

Decreased blood clotting time in the blood of patients, hepatitis B markers (HBsAg, a nti-HBc IgM, a nti-HBc total, HBeAg, a nti-Hbe, HBV-DNA) are detected.

The following diets and medications are used for treatment. A special diet is prescribed to treat patients. Foods are limited to fatty, salty, spicy, fried, canned products and whole alcohol.

Patients are recommended detoxification therapy and antiviral drugs to remove liver toxins, vitamins and general stimulants to restore liver cells. Antiviral alpha-interferon (Alviron, Alfar e kin, Vif e ron, Galavit, Groprinozin, Z e ffix, Laf e robion, Laf e ron, N e ovir, Novirin, Prot e flazid, Flavozid) and nucleoside analogues lamivudine, adenovir ordered. These drugs slow down the multiplication of viruses and their accumulation in liver cells. Hepatoprotectors are also used (zadaksin. Essential, gepamerts, etc.). **The main goal of treatment is to** stop the replication of the HBV virus and to use antiviral drugs to bring the disease into remission.

Normalization of AlAT levels; Loss of HBV DNA and HBe Ag (with or without anti-HBe formation); Interferon and lamivudine (zeffix) are currently used for treatment. a -interferon (realderon, roferon-A) in the event of ALT and HBeAg and HBV DNA in the blood of 25-40% of patients normally be determined. In severe cases, corticosteroids are used.

After studying HBV-DNA, HCV-RNA, HDV-RNA, replication of hepatitis B, C, D was detected . It is the use of antiviral drugs to stop HCV virus replication and transfer the disease into remission. Ribavirin is prescribed at 1000-1200 mg with the use of antiviral drugs. Side effects can cause hemolytic anemia. Pegylated interferon is more effective in the treatment of hepatitis C. Peg interferon **a** $_{2a}$ from 180 mcg (Peg-IFN **a** $_{2a}$, Peg-IFN **a** $_{2b}$ -1.5 mg once a week per kg of body weight) is administered subcutaneously once a week. Peg-IFN can be given in combination with **a** $_{2a}$ / ribavirin (pegasis / ribavirin).

contraindications to treatment with **a** -interferon

Absolute contraindication; -Severe heart disease; Uncontrolled drug-induced epilepsy; Neutropenia (neutrophil count less than $1.5x10^{9}$/ l); Thrombocytopenia (platelet count less than $90.0x10^{9}$/ l); Organ transplantation (except liver); Decompensated liver cirrhosis;

Relative contraindication - Uncontrolled drug-induced diabetes mellitus; Autoimmune diseases;

Adefovir is a dipivoxyl (10–30 mg / day) -adenosine monophosphate analogue, is taken orally, and is used when treatment with lamivudine (100–150 mg orally for 12 months) is ineffective. It is used against viruses, after liver transplantation, if there is a process of decompensation in the liver, and against HIV coinfection. Side effects - nephrotoxic effects.

Entecavir (1 mg / day) is an analogue of guanosine. HBV reduces polymerase function, preventing the formation of DNA strands. Thymosin a $_{1}$ is an acetylated polypeptide consisting of 28 amino acids, zadaxine ™ (1.6 mg / day. T / o 2 times 6 months) . Protects the body by participating in the development and differentiation of T-lymphocytes. Although patients are resistant to α-interferon, the following scheme is used. Thymosin a $_{1+}$ a-interferon; Thymosin **a** $_{1+}$ lamivudine $_{+}$ a-

interferon; Thymosin **a** ₁₊ adefovir ₊ a-interferon; Thymosin **a** ₁₊ lamivudine if contraindicated in α-interferon ; It is advisable to use thymosin **a** ₁₊ adefovir. Prevention consists of vaccination against the virus and prevention of routes of transmission.

The course of treatment is 6 months or more.

Complications are divided into two groups; Specific - liver injury is accompanied by hepatic encephalopathy, liver failure and subsequent transition to cirrhosis and the development of oncological disease, as well as the development of coma.

Nospetsifik- of the gall bladder and accompanied by the development of proton damage cholecystitis and cholangitis.

Acute viral hepatitis can progress to chronic. Chronic transmission of the disease is observed more often from 1 to 5 years of age (25 - 50%), if the virus is transmitted at birth, this figure is 90%. Therefore, it is necessary to vaccinate children at a young age.

Prophylaxis. Specific - vaccination of children and people at risk for viral hepatitis B (0-1-6 months). **Nonspecific** - elimination of infectious routes.

Practical training 8

Clinic of liver t cirrhosis, chronic cholecystitis , diagnosis .

1. Practical training module .

Training time - 4 hours	The number of students 1 : 0 to 12
Form of training	Practical training
Practical training plan	Methods of laboratory and instrumental examination of patients with diseases of the liver and biliary tract (cholecystography, liver scan, examination of the liver and gallbladder with ultrasound). Computed tomography. Symptomatology of chronic cholecystitis and hepatitis.
The purpose of practical training :	Familiarity with the methods of laboratory and instrumental examination of patients with diseases of the liver and biliary tract . Symptomatology of chronic cholecystitis and hepatitis.
Teaching style	Inquiry. Demonstration of patients, interactive teaching methods, practical skills.
Form of teaching	In small subgroups.
O ' unit equipment	Calls to O Training Guidebook , practical Typo content , projectors , computer .
Training mode	Methodically equipped auditorium.
Monitoring and evaluation	Oral control: questions and answers, tests, problem solving.

1.2 . ***Technological card of practical training***

See the stages of hearts and q ti.	Educator	Learners
In the preparatory stage of q	19. See the audience to control the purity karîm 20. Checks students ' readiness for training 21. And control q karîm	
1. bullet and training g contain levels of intra q (10 da q i q a)	1. Preparation of educational content on the topic. 2. Preparation of presentation slides for the introductory speech 3. Develop a list of references used in the study of science	
2 - the main stages of diarrhea q (160 da q i q a)	1. Divide students into small groups and ask questions on the topic. 2. Uses display posters 3. Uses slides, multimedia 4. Conducts treatment 5. Summarizes and summarizes the information provided on the basis of topics, encourages and actively evaluates the active participant students	They are divided into small groups They watch They participate They listen and answer questions
2- final press q ich (10 da q i q a)	1. Concludes 2. Provides independent work 3. Gives homework	He listens Takes notes Takes notes

3. Assessment of students' theoretical knowledge:

A) Frontal method:

41. 1. Describe normal bilirubin metabolism
42. Explain the determination of bilirubin
43. Name the galactose and adrenaline test
44. Explain the normal protenogram
45. Describe protein precipitation tests
46. What is Thymol and Formal Test
47. How the liver is involved in copper and iron metabolism
48. Examination of the bile ducts and gallbladder for radiography and rengenoscopy
49. Describe the method of splenoportography
50. What is holegraphy
51. What is cholecystography
52. Describe the radiostop inspection method
53. What is exography
54. Describe a liver biopsy
55. What is loparoscopy
56. What is scanning
57. The role of computed tomography in the diagnosis of liver and biliary tract diseases
58. Etiopathogenesis of chronic cholecystitis
59. Clinic of chronic cholecystitis

60. Clinical and diagnostic diagnosis of chronic cholecystitis

Brainstorming method
Basic rules :

- Not to mention the shortcomings that hinder the emergence of the idea
- The height of ideas and thought, because the more unusual an idea, the better it is
- Accept many offers
- Combination of ideas and their development
- Present the idea succinctly without argumentation
- Divide the group into two: thought generators and thought analysts

This method allows you to argue ideas and opinions, your own personal opinion, to find the optimal solution in any situation.

Cirrhosis of the liver

Cirrhosis of the liver is characterized by chronic, progressive and dystrophic and necrotic changes in the liver parenchyma, diffuse proliferation of connective tissue in it due to nodular regeneration and deep reconstruction of organ architecture.

Depending on the size of the cirrhotic part, it is divided into three morphological types: Micronodular (small nodules), in which the size of the cirrhotic nodules is less than 3 mm. Macronodular (large nodules), in which the size of the cirrhotic nodules is greater than 3 mm, and the nodules reach 2-3 cm; In this case, the size of the cirrhotic nodules is small and large.

Depending on the etiological types of cirrhosis (biliary cirrhosis, hemochromatosis), the formation of nodules is slow, which is associated with a slowing of regeneration processes in hepatocytes. Because of the small size of micronodular cirrhosis , diagnosis by puncture biopsy is easy. In most subjects, in liver disease and in the absence of cirrhosis, the liver capsule becomes thickened due to an increase in fibrous tissue volume. Dermal puncture biopsy poses a challenge for the morphological diagnosis of macronodular cirrhosis, as the regenerative node may not fall into a complete puncture needle.

Morphological examination in cirrhosis allows to determine its morphological type (large, small, mixed nodules), its level of activity and, if possible, its etiological factors. If one and a half regenerate nodes are found, it indicates the development of cirrhosis, and in advanced cirrhosis, the histoarchitectonics of the structure of the liver fragments is completely lost. The etiology of cirrhosis can also be determined by morphological examination of the base. The factors that lead to the development of cirrhosis are different, it is important to know their histological condition in order to identify them. If “tissue-vitreous” hepatocytes and “sandy nuclei” manifest cirrhosis as viral, fatty dystrophy with neutrophil infiltration, and Mellori’s hyaline bodies, this indicates an alcoholic etiology of cirrhosis. Decreased amount of bile ducts (pathways) and chronic cholestasis are indicative of primary biliary cirrhosis. Special staining methods are used for more information.

In cirrhosis of the liver, the pre-tumor status can be determined. Adenomatous hyperplasia-nodular formation occurs in cirrhosis, and the formation of hepatocellular carcinoma is exacerbated by an intermediate injury called adenomatous hyperplasia or dysplastic nodule. Atypical adenomatous hyperplasia occurs in cirrhosis nodes.

Etiology and pathogenesis. Etiological factors can be divided into exo and endogenous causes. **Exogenous** causes are primarily viruses (hepatitis B and C viruses), alcohol, hepatotropic drugs, lack of protein and vitamins in food or disorders of their absorption due to intestinal diseases, autoimmune mechanisms play an important role in the development of liver cirrhosis. has In rare cases, it is caused by Willson-Kanovalov disease, α-antitrypsin deficiency, primary sclerosing cholangitis, Baddi-Kiari syndrome, hemochromatosis. Primary biliary cirrhosis is distinguished. The listed damaging factors accelerate the formation of cirrhosis. Due to hepatocyte injury, they develop necrosis and then connective tissue and nodular regeneration in liver tissue. Toxic substances include carbon tetrachloride (CCl4), heliotrin, thioacetamide, and b. toxic substances lead to toxic-allergic cirrhosis of the liver.

Endogenous causes of liver cirrhosis may include cholestasis. Factors leading to cholestasis of the intrahepatic and external bile ducts may include blockage of gallstones, parasites in these pathways, or suppression by tumors, inflammation of these bile ducts, and consequent cholestasis. In addition, vascular thrombosis, metabolism and endocrine factors (thyrotoxicosis, diabetes mellitus, etc.) that supply blood to the liver can be included in endogenous causes. The connective tissue and scars formed under their influence change the structure of the liver and compress the blood vessels. This, in turn, does not meet the need for nutrients and oxygen in the liver, and the oxidation-reduction processes that take place here, the synthesis of proteins and enzymes lead to dysfunction. Dystrophic processes in the liver intensify, aggravate the course of the disease, and the pathological process becomes circular.

In the development of cirrhosis, morphological changes in the capillaries in the sinusoids are observed; The disse space expands, collagen is found in them, thickening and hardening of substances are observed, and from this the base membrane, endothelial fenestres are reduced in diameter and number, hepatocyte microvilli are flattened. The pathogenesis of cirrhosis, such as "sinusoidal capillary" and other changes, is explained by dysfunction of liver cells.

Clinical symptoms. The clinical manifestations of liver cirrhosis may not be long-lasting in most patients, or in rare cases - dyspepsia, undeveloped jaundice, hepatolienal syndrome, flatulence, and usually transient ascites. Ascites is also typical for portal hypertension syndrome with splenomegaly, varicose veins of the esophagus or rectum.

The terminal period is accompanied by progressive signs of functional insufficiency of the liver, leading to gastrointestinal bleeding and hepatic

coma in all forms of liver cirrhosis. Gastrointestinal bleeding is in the form of blood spitting or melena, which often results in the death of the patient or anemia due to rupture of the lower 1/3 of the esophagus or, in rare cases, gastric varicose nodes. will come.

In different variants of liver cirrhosis, although the Glisson capsule elongates as the liver enlarges and the necrotic tissue is located close to that capsule, the **pain syndrome is** disturbed and **occurs** mainly in the liver area, under the right rib, epigastrium, and throughout the abdomen. . This pain has a simmering (choking), groaning (uncomfortable) character and is exacerbated after eating (after a lot of fatty meals) and physical exertion. Sometimes the pain is associated with biliary tract injury.

Patients are mainly **diagnosed with dyspeptic syndromes** (anorexia), often with alcoholic cirrhosis, a feeling of heaviness in the epigastric region after a meal, nausea, flatulence, fatty foods. ng symptoms such as fluid in the stool (disorder), vomiting) are disturbing.

Astheno-neurotic syndromes (decreased ability to work, fatigue, rapid fatigue, insomnia, headache, etc.).

Hemorrhagic syndromes (**symptoms such as** bleeding from the gums, subcutaneous bleeding, hemorrhagic rash, bleeding from the intestines, nose, uterus and other places).

Jaundice syndrome (yellowing of the skin, sclera and other mucous membranes).

Fever (incorrect) is observed in postnecrotic and biliary cirrhosis. Weight loss (cachexia) is often seen in patients with portal cirrhosis. It is associated with disruption of digestion, absorption processes and disruption of protein synthesis in the digestive tract. In biliary cirrhosis, itchy skin is often observed.

Examination of patients . Patients are often thin, the skin and mucous membranes are yellow, the lips and tongue are dark red (raspberry), erythema (redness) of the cheek areas, enlargement of the abdomen due to ascites (Fig. 112 a and b) , thin and swollen legs can be seen (**swelling syndrome**). Jaundice is observed in the sublingual, palate area, sclera, and skin. Jaundice is accompanied by discoloration of feces and itchy skin. When examining patients, we may see itchy scars on the skin after itching. When the disease lasts a long time, the subcutaneous bilirubin turns into biliverdin, and the skin glows greenish-yellow. In biliary cirrhosis, due to the accumulation of melanin, a dark (brown) color appears. The main clinical signs that distinguish cirrhosis from hepatitis are enlarged, hard liver and spleen, signs of large liver. We may see **small liver symptoms in** patients . These include: vascular asterisks (telangiectasia), erythema of the palms (liver palms), dark red (raspberry) rand of the lips, oral mucosa and tongue, gynecomastia and other hormonal disorders in men due to impaired estrogen secretion,

a 112photo b

caps and other areas of the body ksantomatoz blyashkalarni photo (113) (pilakchalar) appears to be in violation of cholesterol metabolism, due to the formation of a drum shaped fingers. In most cases, cirrhosis can be accompanied by enlargement of the auricular salivary glands and Dupuytren's contracture as well as peripheral neuropathies.

Examination of the skin of the abdomen reveals dilated veins (Caput Medusae), hemorrhagic rashes, vascular asterisks, enlarged abdomen, and bulging of the umbilicus.

On palpation, the liver is hardened, the surface is **113 images**

The unevenness is determined by the sharpness of the lower edge. **In percussion, the** size of the liver is enlarged.

Laboratory data. In patients with compensated cirrhosis, biochemical parameters may be unchanged. However, serum transaminase activity may be increased. Laboratory examination of decompensated cirrhosis reveals anemia, leukopenia, thrombocytopenia, increased bilirubin and gamma globulin, as well as a decrease in albumin, prothrombin, cholesterol. If inflammation develops a 2 globulin, in jaundice syndrome the amount of b globulin increases. In biliary cirrhosis, the amount of lipids and cholesterol in the blood increases. Serum cholinesterase activity decreases. In cirrhosis, transaminases increase and in biliary cirrhosis, the amount of alkaline phosphatase increases. Large amounts of urobilin are excreted in the urine, and bilirubin is also excreted when jaundice syndrome is strongly developed. The excretion of stercobilin in feces is reduced.

Instrumental inspection . X-ray examinations can reveal dilated esophageal and hemorrhoidal veins. UTT (changes in liver and spleen size, heterogeneous liver parenchyma and signs of portal hypertension), CT (informative method), as well as liver puncture biopsy (determination of process variants and stages) are used.

Treatment. First of all, to eliminate the etiological factor, to organize a work regime, a diet rich in protein and vitamins. Spicy, canned, spicy, greasy, fried in fat, chocolate products are restricted from the diet. It is necessary to follow the general recommendations in the treatment of cirrhosis with low activity (excluding occupational and harmful factors, hepatotoxic drugs, alcohol restriction helps in the treatment of the disease). If cirrhosis of the liver has an active duration, treatment should be etiotropic. In the evening stages of ascites and edema, limited salt and liquid are used as diuretics, aldosterone antagonists (spironolactone) and other potassium-sparing diuretics (triamterone or amiloride). Although in the esophagus, hemorrhoidal veins are dilated, sclerosing treatment and b blockers and other treatments are used to prevent bleeding . To prevent itching in biliary cirrhosis, ursodeoxycholic acid is given in ursosan capsule, legalon (kartsil) 1-2 times 3 times a day, hafitol (tsinariks) per os 2 tx 3 m before meals, tikveol 1-2 tx 3 m 1-3 months. Heptral (L-adenosyl-L-methionine) 800 mg orally or

intravenously for 2 to 4 weeks, gepamerts (L-arnitine-L-aspartate) 10.0 to 100.0 glucose intravenously, α-lipoic acid intramuscularly will be sent. Interferon A is prescribed from 3 million 3 times a week (pegasis 180 mg per 1 m-week), Ribavirin (kapegus), Rabetol 800-1200 mg / day. In severe cases of cirrhosis, liver transplantation is used. Surgical treatment is effective if the bile ducts are blocked by stones and damaged by hangovers.

Dyskinesia of the biliary tract

Functional impairment of gallbladder motility and tone is biliary tract dyskinesia. 70% of gallbladder and tract diseases account for biliary tract dyskinesia. There are 2 different forms of biliary tract dyskinesia; 1. Hypertensive - hyperkinetic type - a hypertensive condition of the gallbladder, characterized by hypertension of the sphincter of Oddi and Lutkens, and occurs more often in young people. 2. hypotonic-hypokinetic type — characterized by hypotonia of the gallbladder and sphincter of Oddi — is more common in asthenics, adults.

Etiology - a disorder of neurohumoral control of the gallbladder. After neurosis, diencephalic syndrome, endocrine-humoral disorders (hypothyroidism, hyperthyroidism, menopause, testicles, ovaries, adrenal and other endocrine gland dysfunction) can also cause the disease. The hypertonic-hyperkinetic type occurs as a result of reflex effects (wound disease, colitis, enteritis, appendicitis, adnexitis). Asthenic conditions, severe infectious diseases, viral hepatitis, avitaminosis, products that irritate the body's constitution, acute, bitter gastrointestinal tract are also important in the pathogenesis of the disease. It leads to the development of hypotonic-hypokinetic type when the interval between diets is large.

Disease Clinic. Spastic contraction of the smooth muscles of the gallbladder and bile ducts in biliary (liver) colic of the hypertonic-hyperkinetic type. The latter occurs when the stone slides along the grass paths. These pains occur suddenly and are unbearable, often irradiating to the upper right shoulder area, right shoulder, and sometimes the heart area as well. It is followed by nausea and vomiting that do not relieve the patient. Pain may be felt when the gallbladder area is palpated. In the hypotonic-hypokinetic type of dyskinesia, the pain is slightly slower and less symptomatic. Palpation changes are less characteristic.

Diagnosis; the diagnosis is confirmed by laboratory and instrumental examinations. on radiography after stimulus delivery, oral cholecystography and intravenous cholegraphy (cholecystokinin, pancreozymin) showed rapid and strong contraction of the gallbladder in the hypertonic-hyperkinetic type and, conversely, slow contraction, enlargement and drooping in the hypotonic-hypokinetic type.

In multi-moment duodenal probing by dyeing (color), in hypertensive-hyperkinetic type of dyskinesia, phase II (when the sphincter of Oddi is closed) is prolonged or normal, phase IV (contraction of the gallbladder) is shortened and delayed.

In the hypotonic-hypokinetic type, phase II may not be present (the sphincter of Oddi is always empty), phase IV is prolonged and delayed.

Treatment; adherence to work and diet, improving the activity of the central nervous system (with tranquilizers and sleep-inducing drugs.

In the attack of **hypertensive-** hyperkinetic type diet 4, 5a and magnesium. It is advisable to give atropine sulfate from no-spa, papaverine, cholinolytic drugs.

In the hypotonic- hypokinetic type, diets 5 and 15 (Pevzner) are recommended. Drugs that improve the activity of the gallbladder - Strychnine, nitrate, caffeine, phenamine. In both cases, treatment in sanatoriums and resorts is effective.

Cholecystitis

Cholecystitis is an inflammation of the gallbladder. There are acute and chronic cholecystitis, which develops on the background of gallstone disease. Chronic cholecystitis can occur after acute cholecystitis, but often develops slowly independently.

Cause factors ; AC is made possible by biliary dyskinesia, gallstones (psixoemotsional cases, the influence of the endocrine and autonomic nervous system funk t bronchial disorders, gastrointestinal viral body has a number of pathological changes of the nervous reflex) due to the stagnation of grass in the gallbladder causes a tendency. The peculiarity of the anatomical structure of the gallbladder and bile ducts, the decline of internal organs, pregnancy, sedentary lifestyle, some foods can also cause cholecystitis.

Factors that cause cholecystitis - infection, viruses, vomiting (ascorbic acid, liabli), autolytic damage to the gallbladder mucosa from the pancreatic juice can also occur. The gallbladder can also be damaged as a result of toxic substances and allergic reactions .

Infection of the gallbladder occurs mainly through enterogenic (intestinal), hematogenous and lymphogenic routes.

Acute cholecystitis

Cholecystitis is an inflammation of the gallbladder. There are acute and chronic cholecystitis, which develops on the background of gallstone disease.

Various infections, damage to the mucous membrane of the gallbladder due to the secretion of sap from the pancreas, worm infestation play an important role in the origin of acute cholecystitis. Recently, the viral etiology of cholecystitis has been proven. In addition, cholecystitis of toxic and allergic nature is observed.

Clinic; strong, persistent pain under the right rib arch after sudden, often factors affecting it (mainly fatty, bitter, pungent food, alcohol, emotional distress beating) occurs with uncomfortable nausea and vomiting.

In patients with acute and chronic inflammation of the gallbladder, patients are palpated to the right of the point of the gallbladder and the neck area to the point called the diaphragmatic nerve between the

sternocleidomastoid muscles. complaining of groaning and suffocating pain under the arch of the right rib, which intensifies (Frenicus symptom).

Fever occurs, sometimes a small amount of jaundice can be seen . Abdominal palpation is painful, mainly under the right rib arch reveals specific symptoms characteristic of muscle tension and inflammation of the gallbladder. Symptoms of fever, pain, intoxication, which develop despite the use of therapeutic measures, indicate cholangitis, gallbladder empyema, its perforation, peritonitis.

Laboratory examination. Blood test - leukocytosis, left shift of leukocyte count. Biochemical analysis of blood: serum alkaline phosphatase, bilirubin, AST, amylase may increase,

Radiography provides little information. Most gallstones are made up of cholesterol, which is x-ray-negative, and only 10-15% of stones contain calcium to the extent that it is visible on X-rays.

Gallbladder ultrasound: indicates the presence of stones, may indicate the size of the organ, its wall thickness, anterior infiltration of the gallbladder, the concentration of gallbladder contents.

In acute cholecystitis must be hospitalized. In acute cholecystitis with purulent and gangrenous gallbladder is treated surgically. Patients with catarrhal cholecystitis are prescribed a strict bed rest, abstinence from food for 2 days after the initial attack, followed by a small portion diet 5-6 times a day, a wide range of antibiotics and antispasmodics.

Chronic cholecystitis

Chronic cholecystitis is an inflammation of the gallbladder that, in most cases, acute cholecystitis can develop as a result of incomplete treatment and independently. In chronic cholecystitis, gallstones are detected in 90-95% of cases. Chronic cholecystitis without stones occurs in only 5% of cases, and it also occurs in cases of severe stress.

Clinic. In rare cases the pain symptom is disturbing, in most cases the dyspeptic syndromes are disturbing. Re- exacerbation of the inflammatory process leads to thickening of the bladder wall, sclerosis, scarring, changes in its shape, and is effective in the diagnosis by ultrasound or cholecystography. Rest may be observed in the upper abdomen when patients are examined. On palpation, pain is felt in the projection of the gallbladder. Sometimes the symptoms of Myusse-Georgie v , Ortner, Vasilenko, Obratsov-Murphy are positive.

On laboratory examination, there is a slight increase in leukocytosis and ECG during the period of sensitivity. Duodenal probing (25% magnesium sulfate, 20 ml of olive oil, 10% peptone, 10% sodium chloride, 40% xylitol, 40% glucose solutions are given per os to enhance bile secretion) is obtained by taking three servings of fluid. Exactly the changes in the gallbladder portion i.e. portion **B** indicate the presence of cholecystitis and mucus, leukocytes and epithelium can be seen in the fluid composition.

The method of treatment is conservative in stoneless cholecystitis and surgical treatment in stone cholecystitis. In exacerbation of chronic

cholecystitis, treatment should be carried out in an inpatient setting (as in acute cholecystitis). In the non-acute period, anti-relapse treatment courses are conducted 1-2 times a year.

Periodic duodenal sounding or “soundless sounding” with mineral water, It will be tubed.

Herbal remedies are prescribed for 3-4 weeks .

Medal juice, profile sanatorium and spa treatment is recommended.

Practical training 9

Renal and urinary system. Methods of examination: interrogation, methods of physical examination (examination, palpation, percussion, laboratory-instrumental methods).

1. Practical training module .

Training time - 4 hours	The number of students 1 : 0 to 12
Form of training	Practical training
Practical training plan	Symptomatology of nephritis (acute and chronic). Methods of interrogation and examination of patients with blood diseases (complaints, collection of medical history, hereditary factors). Examination, palpation of lymph nodes. Palpation and percussion of the spleen
The purpose of practical training :	Symptomatology of nephritis (acute and chronic). To gain an understanding of the methods of interrogation and examination of patients with blood diseases. Palpation and percussion of the spleen .
Teaching style	Inquiry. Demonstration of patients, interactive teaching methods, practical skills.
Form of teaching	In small subgroups.
O ' unit equipment	Calls to O Training Guidebook , practical Typo content , projectors , computer .
Training mode	Methodically equipped auditorium.
Monitoring and evaluation	Oral control: questions and answers, tests, problem solving.

1.2 . ***Technological card of practical training***

See the stages of hearts and q ti.	Educator	Learners
Preparatory stage	22. See the audience to control the purity karîm 23. Checks students ' readiness for training 24. And control q karîm	

1. bullet and training g contain levels of intra q (10 da q i q a)	1. Preparation of educational content on the topic. 2. Preparation of presentation slides for the introductory speech 3. Develop a list of references used in the study of science	
2nd stage (160 minutes)	1. Divide students into small groups and ask questions on the topic. 2. Uses display posters 3. Uses slides, multimedia 4. Conducts treatment 5. Summarizes and summarizes the information provided on the basis of topics, encourages and actively evaluates the active participant students	They are divided into small groups They watch They participate They listen and answer questions
3 See the final stages of diarrhea (10 da q i q a)	1. Concludes 2. Provides independent work 3. Gives homework	He listens Takes notes Takes notes

3. Assessment of students' theoretical knowledge:

A) Frontal method:

1. 1. The concept of nephrites
2. Etiopathogenesis of acute glomerulonephritis
3. Classification of acute glomerulonephritis
4. Clinical manifestations of acute glomerulonephritis
5. Urinary changes in acute glomerulonephritis
6. Diagnostic criteria for acute glomerulonephritis
7. Modern methods of treatment and prevention of acute glomerulonephritis
8. Etiopathogenesis of chronic glomerulonephritis
9. Classification of chronic glomerulonephritis
10. Diagnostic criteria for chronic glomerulonephritis
11. Treatment and prevention of chronic glomerulonephritis
12. A general review of patients with chronic glomerulonephritis
13. Types of clinical course of chronic glomerulonephritis
14. Changes in urine in chronic glomerulonephritis
15. Comparative diagnosis of acute and chronic glomerulonephritis
16. Complaints of patients with blood diseases
17. What to look for when reviewing patients with blood disorders
18. Diagnostic significance of lymph node palpation
19. Techniques of palpation and percussion of the spleen
20. Diagnostic significance of spleen palpation and percussion

The "pen in the middle of the table" method

The whole group is asked questions (e.g., diabetes symptoms, beta-blocker medications, UIC propensity factors). Each student writes their answer on a piece of paper and sends it to a neighbor, placing the pen in the middle of the table.

The teacher checks the group work and writes the general option in the notebook.

Diseases of the urinary system

Kidney pair of parenchymal organ , on either side of the spine XI and III breast cancer b e l in the field of spine . Factors that cause kidney disease are divided into exogenous and endogenous factors. Exogenous infection is transmitted to the kidneys from the urinary tract through the retrograde route, in addition to viral hepatitis B and C, allergic reactions to drugs or vaccinations, post-pregnancy gestosis, some drugs (gentamicin, basal analgesics, gold drugs, penicillamine, aminoglycosides, antiepileptic drugs) nephrotoxic effects, exposure to toxic substances (including snake, scab, malaria and other venomous insect bites). Infection can lead to the development of alterative processes in the kidneys through the hematogenous route from other foci of infection. Endogenous causes play an important role in pathological processes such as kidney stones, hypertension, diabetes, systemic diseases, renal vascular thrombosis, HIV infection, syphilis, tuberculosis, as well as circulatory disorders.

Scheme of examination of patients with diseases of the urinary system:

I. Methods of subjective examination

1. Inquiry of the patient (primary and secondary complaints).
2 Medical history
3. Life history

II. Methods of objective inspection

1. Assess the patient's condition.
2. Review.
3. Palpation.
4. Percussion.
5. Auscultation.

III. Instrumental inspection methods.

1. Methods of radiological examination
2. Computed tomography
3. Magnetic resonance imaging
4. Endoscopic examination method
5. Ultrasound examination
6. Radioisotope testing methods

IV. Laboratory testing methods

1. Blood test;
1.1 The amount of urea in the blood;
1.2 The amount of creatinine in the blood;
2. General urinalysis;

2.1 The amount of protein in the urine;
2.2 The amount of sugar in the urine;
2.3 The amount of urea in the urine;
2.4 The amount of creatinine in the urine;
2.5 Transplantation of urine into microflora;
3. Puncture biopsy examination

V. Functional inspection methods.

1. To determine the function of concentration and liquefaction of the kidneys
2. Determine the relative density
3. Determine the filtration rate in the balls
4. Urine analysis by Nechiporenko method
5. Urine analysis by Addis-Kakovsky method
6. Urine analysis by Zimnitsky method
7 Urine analysis by Ambury method
8. Roberg test

B Emory questioning (interrogatio)

The following should be considered when questioning a patient.

1. The main complaints are identified.
2. Additional complaints will be identified.
3. Collection of medical history (anamnesis morbidity).
4. Collection of life history (anamnesis vitae).

Urinary system, patients seek b Ilan sick fail, mainly in patients with renal and kidney symptoms bother. Kidney symptoms include back pain and changes in urine (changes in urine color, volume, composition), pain associated with urination, edema, extra-renal, ie, general symptoms headache, headache dizziness, noise in the ears, pain in the heart area, decreased vision. These symptoms occur in kidney disease accompanied by hypertension. In patients with kidney disease, external symptoms of kidney failure include discomfort, decreased ability to work, impaired memory, sleep disturbances, itchy skin, bad breath, which are associated with urea poisoning and brain poisoning. In most patients, dyspeptic conditions such as suffocation, dry mouth, unpleasant fullness in the mouth, nausea, vomiting, and diarrhea are also disturbing when the disease is severe . The cause of these symptoms is mainly due to poisoning of the digestive tract with urea .

Urinary system, patients seek b vaccinated patients with kidney **pain,** frequent urination, siyganda strong burning or painful urination, change in urine color, body swelling appeared to be an complain . Additional complaints include general weakness, rapid fatigue, decreased ability to work, and other similar symptoms. These symptoms often confuse the doctor. In renal disease, enlargement of the renal capsule (in acute glomerulonephritis) - renal pelvis (acute and chronic pyelonephritis) - closure of the urinary tract is observed in various bending anomalies in the location of the kidney, renal tissue ischemia (renal infarction).

More attention is paid to the localization of pain. In most cases, the pain is in the lumbar region, sometimes unilateral, often characteristic of pyelonephritis, as well as kidney stones, paranephritis, and bilateral (in glomerulonephritis). It should be noted that in nephrological practice, the patient does not have severe pain in chronic glomerulonephritis or is painless. Kidney pain occurs mainly as a result of elongation of the kidney capsule.

Pain caused by strong contractions of the kidneys often spreads to the lower back and abdomen. As a result of the cold, the pain spreads to the urethra. The main pain symptoms determine the nature of the disease. Sometimes the pain in pyelonephritis is accompanied by groaning, the pain may increase as a result of obstruction of the urinary tract. The patient often behaves in a restless confusion, however, the patient has a general weakness, often varying from state to state. Patients may also vomit frequently when the pain becomes acute. In such patients, urinary incontinence is observed. In some cases, it should be noted that the origin of the disease is inextricably linked to certain factors. For example: kidney compression, agitation, pain in the bladder and urinary tract when urinating. When stones appear in the kidneys and stones in the urethra become clogged, a very strong sting is irritating and must be anesthetized. In this case, the cholinoblockers, angiospasm o litiklar and take a hot bath can improve the condition of the patient. In acute paranephritis, the patient assumes a compulsive position and slows down the pain by bending the thigh to the joint.

This is called hematuria. Hematuria can be macro (if the eye sees blood in the urine (urine color like meat wash water)) and microhematuria (when erythrocytes are found in the urine under a microscope).

By the origin of hematuria;

1. Prerenal hematuria - associated with impaired hemostasis, occurs in the overdose of thrombocytopenia, thrombocytopathy, hemophilia, DVS syndrome and anticoagulants.

2. Renal hematuria - **glomerulyar** -buyrak symptom of the disease, glomerular nephritis, kidney stones, heart disease, kidney disease, kidney vasculitis and neglomerulyar kidney tissue-destructive renal abscess, infarction, trauma, necrosis, nephropathy, gidronefrozda, cancer and renal tuberculosis decay stage, occurs.

3. Posrenal hematuria - occurs in diseases of the urinary tract, occurs in the urethra and gallstones, tumors and injuries.

The main clinical sign of diseases of the urinary system is a violation of urination (diuresis), its color, quantity , composition and frequency of urine . **Diuresis is the excretion of** urine over a period of time (normally 1000-1800-2000 ml per day). In tropical countries (hot), including Uzbekistan, this ratio varies depending on the seasons. In summer, the amount of urine excreted decreases due to the release of a lot of fluid due to heat through the tei and through the airways. Diuresis is positive and

negative. Negative when urine output is low compared to drinking fluid (vomiting, excessive diarrhea, excessive sweating, swelling during heart, kidney disease, excessive sweating when the air is too hot, etc.) . Positive diuresis is when there is more urinary excretion than when drinking fluid (in the reduction of edema when drinking diuretics, in diabetes mellitus and diabetes mellitus). Depending on the physiological condition and in some diseases, diuresis may increase and decrease.

The separation of the same amount of urine at the same time interval is called **isuria** . It **s** urunkali kidney lack of the amount of liquid and physical work environment, temperature and other factors affecting the balance of the separation of urine and the kidneys function kontsentirlash abuses occur.

Dysuria is a disorder of urinary excretion, aggravation. In dysuria, there is a violation of the amount of urine excretion, a violation of the frequency of urination and a violation of the composition of urine.

Enuresis is the inability to stop or hold urine. It is most common in children. Acute enuresis can also be caused by prostate disease.

Polyuria is an increase in daily urine by 1.8-2.0 (sometimes 10-20) l d a n . Polyuria (kidney) can occur not only in kidney disease but also in patients with diabetes due to a decrease in ambient temperature, exposure to something, excessive water intake, diabetes mellitus and diabetes mellitus. Polyuria is sometimes observed when diuretics are given to eliminate other tumors in the body. Polyuria leads to a decrease in reabsorption process in the kidneys, a decrease in water absorption and disruption. **Oliguria** is a decrease in the amount of urine per day from 500 ml, which is relative and absolute. Relative - excessive sweating, low fluid intake, high ambient temperature. Absolute- recurrence leads to excessive recording and diarrhea. Oliguria is caused by impaired renal function or chronic kidney disease. In addition, in heart failure, acute glomerulonephritis, poisoning of the kidneys with nephrotoxic substances, oliguria occurs during tumor development .

Anuria - urinary bladder Committee 's autobiography 50 ml am to acting or not to'planmasligini. This symptom occurs in the stage of decompensation of chronic glomerulonephritis, when the blood circulation in the kidneys is completely disrupted, and through the poisoning of the renal parenchyma with other chemicals and the formation of kidney stones. Anuria can be prerenal, renal, posrenal, and in very rare cases arenal (in the absence of kidneys). Prerenal renal renal failure (renal vascular thrombosis) and heart failure grade III, severe renal renal parenchyma, renal failure, obstruction or external compression of the posrenal upper urinary tract with stones and tumors, and urinary incontinence does not fall. Anuria does not produce secondary urine in the secretory-kidney and does not pass urine into the excretory bladder through the urinary tract (obstruction or external compression of the urinary tract with stones and tumors, spinal cord injury, coma). When the function of the central and peripheral nervous system is impaired, it leads to incessant urinary excretion. This condition is often

associated with acute cystitis or a tumor of the neck of the bladder. Shock, severe trauma, poisoning by heavy metal salts, stones from the kidneys and urinary tract, due to compression of the tumor by the tumor does not fall into the bladder. Anuria is a very acute disease that can be fatal if not treated in time.

Pollakiuria is the **excretion of** urine six or more times a day. Occurs as a result of reflex action on the musculature of the bladder or as a result of closure, constriction of the external urinary tract. This symptom occurs in cases associated with diseases of the urinary system (cystitis, urethritis, urinary stone disease, intoxication of this system) and unrelated (prostatitis, pregnancy in women, uterine fibroids and other pathological processes in it). In some cases, pollakiuria also plays a major role in the development of neurosis. Pollakiuria can also be due to the use of diuretics in a physiological state (stress and excitement) or in some diseases.

Ishuriya is an obstruction of the urethra that accumulates in the bladder, making it impossible for patients to empty the bladder. Ishuriya is organic and functional. In the first case, due to spinal cord injury, bladder atony (disruption of the bladder innervation is disrupted and its contractile activity is disrupted), tumors occur as a result of closure of the urinary tract due to prostate adenoma, in the second case, severe fear can be exemplified.

Stranguria is: a difficult, painful urination

Hypoisostenuria is a decrease in the relative density of urine from 1,009 to 1,011 (most often occurs in renal failure.

Shish. In renal disease, swelling is observed mainly in the upper and lower eyelids and face of the patient (Fig . 113a and b) and is most pronounced in the morning. Patients' facial skin was whitish, their faces, upper and lower lids were swollen, and their eye sockets were narrowed (facies nephritica 113 a and b) .

a 113 photo b

In the late stages of the disease, the tumor spreads throughout the body (anasarca).

In such cases, the color of the urine changes (micro-macrohematuria) as a result of the presence of trace elements in the blood.

Methods of detecting hidden tumors

1. McClure-Oldrich test - for this, 0.2 ml of physiological sodium chloride solution is injected subcutaneously into the upper third of the elbow and a bulge is formed. This swelling is absorbed in healthy people in about an hour, is quickly absorbed if there is a tendency to swelling, and the test is positive.

2. Measure the patient's weight daily.

3. Measure daily diuresis (comparison of drinking fluid and excreted fluid).

When patients are examined, first of all attention is paid to consciousness, and at the onset of the disease is active (in itself), in a state of uremic coma, a passive state, in paranephritis, renal colic. Patients should pay

attention to skin color, facial condition, and swelling. Attention is paid to the patient's urine (urine), its color, amount, frequency. Urinary changes in kidney disease are of diagnostic value.

Anamnesis of morbi. B kidney disease occurs primarily after a cold and often develops glomerulonephritis after angina, rheumatism, scarlet fever, otitis, sinusitis, diabetes, systemic diseases, hypertension and other diseases. The patient is asked if he has had acute glomerulonephritis, pyelonephritis, cystitis or edema syndrome, urinary incontinence, increased or no blood pressure, pain in the lumbar region or lower abdomen. The presence of mercury, bismuth, silver, sulema, phosphorus poisoning in the workplace or at home, a large overdose of drugs sulfanilamide drugs, gentamicin, whether or not blood and blood transfusions during life are also important in the diagnosis of kidney disease. Some medications (such as gentamicin have a nephrotoxic effect) , phenacetin, barbiturates, and camphor cause allergies in the kidneys. It is also important to ask about the nature of the disease (renal atherosclerosis, glomerulonephritis, renal amyloidosis. Inquiry into such factors is important in the diagnosis of kidney disease.

In the anamnesis vitae collection, more attention is paid to the factors that cause kidney disease. Dysuria has been observed throughout life (disorders of urinary excretion, frequency and amount of urinary excretion, impaired composition, changes in its color are of great importance in the diagnosis). Contact with toxic substances during work or throughout life leads to toxic alteration of the kidneys. Factors that lead to diseases of the urinary system often include pyelonephritis, glomerulonephritis, such as colds, living in a damp or cold place, freezing of the body, standing or working in a cold (skvoznyak) place. Diseases such as lifelong diseases such as inflammation of the genitals, tuberculosis, collagenosis, diabetes, blood diseases lead to kidney disease. Osteomyelitis, bronchiectasis diseases lead to renal amyloidosis. Pregnancy leads to nephropathy. You should be asked if you have had kidney or urinary tract surgery. Hereditary diseases and kidney diseases are asked to be recorded in the offspring. Then allergological and epidemiological anamnesis is collected.

Methods of objective inspection

When assessing the condition of I-Patient, attention is paid to the following;

1. The patient's consciousness (in himself, dizzy, not in himself, stupor, sopor, in a coma, etc.) Several times higher than normal urea in the blood can lead to memory loss or uremic coma.

2. Patient status (active, passive, compulsive condition). In the early stages of the disease, patients are able to self-serve, are active, sometimes in a coma, in a coma are in a passive state, can not serve themselves, someone has to help them . Patients with kidney and urinary stone disease or paranephritis feel compulsive and feel relieved.

Review-The following should be noted.

1. Skin color: pale-flowing, attention is paid to the presence of spots;

2. To the localization of tumors: local, general, diffuse, anasarca; starting from the squash field; attention is paid to the consistency-softness of the tumor.

3. Asthma type (Kussmaul in uremic coma).

4. Skin odor (coma smells of urea).

5. Body temperature (hypothermia, hyperthermia).

6. To the color and condition of the skin (it is white, you can see the traces of nails in a coma).

Examination of the renal area. It is difficult to diagnose kidney disease when examining the patient's lumbar and abdominal area, however, it is possible to have some information when the kidney size suddenly increases. In chronic and acute diseases of the kidneys can be seen swelling in the lower back and lower abdomen, swelling of the lower (upper) area of the abdomen as a result of frequent filling of the bladder. This condition occurs in most cases due to excessive filling of the bladder with urine as a result of urinary retention (functional and organic).

Kidney palpation (palpatio)

The following should be considered when palpating diseases of the urinary system. Palpation is used for objective examination of the kidneys .

1. The surface of the kidney is smooth, flat and soft, in pathological cases the surface is uneven, the consistency may be hard and enlarged.

2. Pain in the kidney area is detected.

In patients, lean people, they can palpate the kidney in the act of breathing on palpation of the kidney. When the kidney slides down (in nephroptosis) it is easy to feel, especially when it slides down when breathing. Bimanual palpation is used in renal palpation . After the hand is inserted, the patient is advised to breathe, at which time the diaphragm descends and the kidney also descends, at which time the kidney can be palpated. Palpation reveals that the left kidney is lower than the right kidney. On palpation of the kidneys, the patient lies in a horizontal position with the leg bent at the knee, the abdominal muscles relax after placing the hands on the chest. When palpating the kidney, the physician should stand on the right side and place the left hand on the patient's lumbar region with the fingertips near the spine and the index finger touching the 12th rib. When the abdominal muscles are relaxed, the abdomen is palpated using the fingers, while the lumbar region is pressed with the palm of the left hand. When the kidneys are palpated, their shape and boundaries can be determined. This means that the kidney is normally 12 cm long, 6 cm wide and has a smooth surface. On palpation of the kidney, the patient is unaffected, but may later return with discomfort. Abnormal kidney structure indicates the formation of tumors , tumors or other pathologies . It is known that the kidneys differ from other organs by their elastic structure, but in various diseases of the kidney (pyelonephritis, paranephritis, renal tumors) the kidneys lose their elasticity and vice versa.

The purpose of palpation of the kidney is to determine the size of the kidney, whether it is hard or soft, flat or uneven. Determine the level of pain, shifting the shape up and down and sideways. (114 photos)

a b

114 Figures **a** and **b** . The picture explains the technique of palpation of the kidneys.

Balatir push - palpation or (Gyuvon method).

A) Bimanual palpatsiyadagid e k is poured; B) Bel hand with the left hand, a n e cha short siltovchi in paragraph (Turkey); G) t e branadi under the influence of the kidney impulse ;

Level 1 if palpable: level 2 (ren mabilis); grade 3 (ren migrans) nephroptosis if it slides in all directions.

Diagnostic value. Determining the areas of pain, shape, displacement, hard-soft by palpation is of great importance in making an initial diagnosis.

Renal p e rcussion (percussion (Fig. 115 a and b)). In the percussion of diseases of the urinary system should pay attention to the positive, negative of Pasternadsky's symptom.

Low- e Refer to the method of the kidneys to check the entry of the flags k e ng. This t e kshim the use of the method, the doctor b e the right side of the morning, and left arm b e morning 12 instead of the ribs, spine debris field with his right usage. To'qillatib t e kshim the calculation method, the upper part of the bladder diagnostic significance. To do this, the doctor moves with his right hand b e morning starting from the umbilical cord and pointing up and down. If the bladder is empty, the tympanic sound is stored up to the area of the bladder . When the bladder is full, the tympanic sound decreases.

Kidney p e rkussiya method t e kshim in order to determine the pain.

115 a and b s urat

The picture explains the technique of detecting Pasternadsky's symptom

Auscultation. Heart tones and noises are evaluated on auscultation of urinary tract diseases.

Auscultation is useful in the diagnosis of renal artery stenosis. To do this, the patient should be in an upright position. The phonendoscope is placed on the abdominal wall 2-3 cm above the umbilicus and 2-3 cm outside, and if there is stenosis in the renal artery, a systolic murmur is heard at this point. The back can be auscultated to the patient's sitting position. To do this, a phonendoscope is placed in the corner of the rib-spine and audible.

Urinary system t e kshiruv basis. Urological practice, kidney disease t e z medical care, renal colic. Renal colic, urinary stone disease, caused ureter remain bent k e is located. Excreted through the urine due to distension kidney bowl measurement of blood pressure, kidney k e ngayadi and causes strong pain.

III. Instrum e ntal t e kshim methods.

The following instrumental methods of examination are performed in diseases of the urinary system.

1. Methods of radiological examination.

a) excretory urography;

b) retrograde pielography;

c) infusion urography

2. Computed tomography.

3. Magnetic resonance imaging

4. Renal artery angiography

5. Methods of endoscopic examination.

a) cystoscopy;

b) chromocystoscopy;

c) cystoscopic catheterization of the bladder, ureters and renal pelvis;

6. Ultrasound examination.

7. Radioisotope renography examination method.

8. Kidney scan

R e NTG e technology t e kshim. In the radiological examination method, patients are mentally and physically prepared before examination. In spiritual preparation, the patient is given an understanding of the diagnostic value of the examination method, the painlessness of the treatment, and the fact that it does not affect health. When physically preparing patients for the examination, they are prescribed a carbohydrate-free diet the day before the examination and their intestines are gutted. This test can determine the shape, size, number of kidneys , their location, and the presence of stones in them. The left kidney is normally 1.5-2 cm higher than the right kidney. Simple r e NTG e technology images of the kidneys, urinary tract and bladder diseases, it is difficult to determine, but thin people on either side of the spine XI and III breast cancer b e l oval-shaped shadow in the area of his spine. The location of oxalate and phosphate urinary stones in the kidney and the shape of the stone in the urinary tract are clearly visible. For more accurate information about the shape and location of the kidney, the method of sending air behind the peritoneum - r e tropn e vmop e riton e um or pn e vmor e n is used. In this method, the shadow of the kidney is clearly visible on a bright background.

Urinary tract contrast material t e kshirishning a number of ways.

1. **R e trograd pi e lografiya** . This method of examination is used in nephrology in the detection and diagnosis of tumors, tuberculosis, abnormal development of the kidneys, the condition of the urinary tract and urinary stones. This method is rarely used.

Prior to R e trograd pi e logography, the patient is explained the purpose of the examination and its importance in diagnosis and treatment, and before the examination, the bladder is emptied of urine through a catheter and then it is advisable to conduct an examination.

R e trograd pi e lografiya or urografiyada contrast agents (sergozin, kardiotrast, yodamid, triombrast, urography, c e rografin and bq) tube

directory e t e jacks using the urinary tract through the tsistoskop. T e x is only one-sided because it is not possible to do two-sided pi e logography at the same time . The contrast substance rises from the bottom up and fills the kidney cups. R e ntg e n image clearly shows the urinary tract, renal cavities, the shape of the vessels, the location of the kidneys. This method is less difficult and less commonly used because it is associated with many complications.

Antegrade pielography . In this method, the contrast agent is punctured through the skin into the renal pelvis. This method is used in the kidney, which has completely lost function.

2. intravenous e kskr e narrow urografiyasi photo (116 a) and (b) . This method of investigation by the kidney ekskr e police to be contrast b e the root of the morning have been sent to radiology, renal failure, urinary tubes and siuda box shadows. Not only does it assess its anatomical condition but also its functional state, and it is also possible to see how quickly and well the contrast material separates. An X-ray is taken every 1-10-15-20-30-60 minutes for a patient sent for contrast. This screening method is an effective way to diagnose chronic pyelonephritis. In particular, the condition of the calyx and cups of the kidney is assessed.

116 Figures a and b

a) X-ray image shows hesitation of the kidneys and urinary tract.

b) pyelouretral stenosis of the right kidney and enlargement of the vessels over the stenosis.

With the help of images, accurate information about the location of the kidneys, the permeability of the urinary tract is obtained. Kidney contours are also noticeable. They are located in the area of the XII ribs. The urinary tract sinks at an angle impenetrable to the renal pelvis, bends near the spine, and descends. At the entrance to the small pelvis, they bend again and attach to the bladder. R e ntg e n images also show stones in the kidneys and urinary tract very clearly. If the violation of the conductivity (stone, urinary tract tumors or other external influences), e saying contrast to breaks or narrowed. Renal circulatory disorders due to stenosis of the renal arteries, atherosclerosis can be detected due to **renal angiography** . Nephroangiography is a method of radiological examination in which contrast agents, such as diotrast or cardiotrast, are sent from the femoral artery to the aorta at the level of the renal arteries using special catheters.

Infusion urography. This method of examination is a method of X-ray examination of the kidneys, and urinary tract by injecting the substance into a vein within 10-15 minutes by intravenous drip X-ray contrast (more than a dose of 1 ml / kg mass, ie a simple excretory urography). This test method is better than a simple excretory urography test. If an adverse effect is felt, the contrast transmission is stopped immediately. For infusion urography, 60 ml of 65% urotrast or other contrast agent is added dropwise to 120 ml of saline or 5% glucose solution, and the first image is taken 5 minutes later and the

next image is taken. The conclusion is given by taking an image determined by the employee.

Contraindications to excretory urography - shock, collapse, decompensated renal failure, severe hyperazotemia, severe liver disease and its insufficiency, hyperthyroidism, hypersensitivity to iodine, hypertension, heart failure, decompensated stage it's not.

Computed tomography examination method. This method is an effective method for detecting pathological changes in kidney tissue, and it is possible to determine step by step in which area the pathological changes occur. Computed tomography can assess the nature of kidney damage. This makes it easy to find kidney tumors and determine if they have spread beyond the kidneys. Therefore , see Figure 117 a, b, c with high efficiency in complex diagnostic processes .

Magnetic resonance imaging. On magnetic resonance imaging **,** it is possible to describe the object of examination on three surfaces **, frontal** and **sagittal** . In this test method, it is possible to diagnose cases that cannot be diagnosed by other methods. Changes in the renal vessels and vascular circumference (aneurysm, arterio-venous swell (leakage), thrombosis and hanging) can be detected. The nature of renal cysts and cyst fluid can be assessed, and bleeding can be distinguished from infection.

117 a, b, c photo

A B

C 117 a, b, c photo

Kidney MRI. A kidney tumor is indicated by an arrow on the left kidney

Kidney and urinary tract using ultrasound t e kshim. T e kshim method k e issues of our times, k e ng used, and it is of great importance for the diagnosis. Exografiya kidney, large or small, shriveled, with the help of the form of the shell, jomlarining kidney, urinary tract skating e m e NGO, tumors, cysts, and the whole structure.

Radioisotope renography - helps to study kidney function. For examination, the patient is injected intravenously with a diotrast or hippuran, ^{131} J isotope, and a multi-channel radiograph is used to record each kidney function using separate curves. ^{131} **The** rate of blood clearance from the J isotope increases the secretory function of the kidney. Accumulation of the isotope in the bladder is a urodynamic sum of the upper urinary tract. Radioisotope renography is an effective method in the diagnosis of chronic glomerulonephritis, tuberculosis, pyelonephritis, amyloidosis, urinary incontinence in one of the kidneys, renal hyperplasia (detection).

Renal angiography. Angiography is an effective method of diagnosis of hydronephrosis, vasorenal hypertension, when the tumor process develops in the kidneys and adrenal glands. In clinical practice, images obtained from renal angiography are processed and evaluated on a computer. In this method of examination, the amount of contrast sent is 2-3 times less than in normal intravenous urography and is sent intravenously. In order to obtain kidney

images with a computer, sound signals are processed and data are obtained. **Guideline against inspection.** In renal insufficiency, when the body's sensitivity to iodine is increased, severe liver damage is a contraindication to this examination. Allergic reactions to anaphylactic shock, fainting, acute renal failure, acute liver failure, etc. may occur during these screening procedures, so be prepared to assist in these complications during the examination.

IV. Urine separation system l aborator methods

1. Blood test.

a) . General blood test (anemia is observed);

b). The amount of urea in the blood increases;

v). The amount of creatinine in the blood increases;

g). Hypoproteinemia

2. Urine analysis.

a) . general urinalysis (erythrocyturia, leukocyturia, proteinuria, bacteriuria, glucosuria are observed;

b). the amount of urea in the urine

v). the amount of creatinine in the urine

3. Biopsy materials are morphologically examined.

Get a kidney biopsy. A written consent form will be obtained from the patient for a biopsy. In the detection of kidney disease in special n e frology departments, a bioptat (small kidney tissue) is obtained from it. For a long syringe aspiration biopsy needle set, kidneys y e will be saying a small kidney tissue and morphological microscopic t e kshiriladi. If you have kidney disease, you need to identify the callers, planted in kidney tissue from a part of the food environment and the k e Yinchang native flora which antibacterial agents s e zgirligi. This method is very clear t e kshim method, but a small k e ng, because its own complications b e worldwide. **As an absolute contraindication to biopsy** ; 1. Single kidney; 2. Disorders of the blood coagulation system (hypocoagulation, thrombocytopenia, hemorrhagic diathesis); 3. Renal vein thrombosis; 4. Hydronephrosis, pionephrosis, polycystosis. **Relative contraindication;** 1. Severe arterial hypertension diastolic pressure 110 mm. sim. if above 2. In chronic renal failure, the creatinine level is higher than 0.44 mmol / l; 3. Pathological displacement of the kidneys; 4. Advanced atherosclerosis of the arteries. **Asoroti.** Bleeding into the renal pelvis, under the capsule, tissue, the formation of hematomas, pus in the biopsy site, injury to neighboring organs.

Urine separation systems laboratory t e kshim methods

In diseases of the urinary system, the composition and amount of urine changes. The amount of urine may decrease, increase. It contains trace elements of blood, protein, sugar, microorganisms, creatinine, uric acid in pathological conditions.

Protein. The excretion of protein in the urine is called proteinuria. **Proteinuria** can be **physiological and pathological** .

Urinary excretion of protein is one of the main symptoms of diseases of the urinary system. Normally 50-200 mg of protein can be excreted in the urine per day. Although the amount of protein in the urine is more than 0.033 g / l, it is indicative of a pathological process. The literature shows that protein is the same as urine during the day. Protein separation is more likely to occur in the horizontal position of the body and during the day. It is therefore advisable to detect daily proteinuria.

Depending on the amount of protein in the urine; **advanced** proteinuria more than 3 g / day; **undeveloped** proteinuria is less than 1–3 g / day and **partial** proteinuria is less than 1 g / day or less. Proteins in urine are small-molecule proteins, globulins, and albumins. Depending on the cause of proteinuria is divided into the following forms. Renal- **dependent proteinuria is** glomerular, tubular, and mixed. Non-renal proteinuria-hemodynamics occurs due to stagnation of blood in the pathways and ducts of the urinary tract. Proteinuria of this nature is observed in heart failure, thrombosis of the veins of the urinary tract, an increase in oncotic pressure of blood plasma due to protein overload (myeloma).

The mechanism of protein excretion in urine . In the outgoing part of the gene confusion occurs the secretion of a separate protein uroprotein. The kidney filter for protein filtration consists of 3 layers. The first layer is the epithelial layer, the second layer is the basal membrane (consisting of two layers of gel). The third layer consists of a layer of epithelial cells podocytes. The third layer has a body and legs and is located in the basement membrane. There are holes between the podocytes and through these holes partially pass albumins and small molecular proteins. The bulk of the albumins do not pass through the kidney filter because the kidney filter and the albumins are equally positively charged, they do not pass through the kidney filter because they push each other. In renal injury (immune complexes, inflammation, degenerative and sclerotic changes) the charge of the basement membrane, endothelium and padocytes changes and albumins pass freely through the renal filter and proteinuria occurs. Often the kidney filter is damaged in glomerulonephritis, amyloidosis, diabetic glomerulosclerosis and hypertension.

There may also be proteinuria in **physiological cases** . 1. Orthostatic proteinuria-Can be observed in people with asthenic and lordosis up to 30 years. 2. Proteinuria with elevated body temperature — most commonly observed in children and the elderly. 3. Stress proteinuria - occurs during strenuous physical activity, stress, stressful situations and excessive freezing of the body. In functional proteinuria, a partial protein (1 g / s) is excreted and then passes away and is not accompanied by erythrocyturia, leukocyturia, and bacteuria.

Proteinuria can also be a false positive. Myoglobinuria, erythrocyturia and leukocyturia and their breakdown are sometimes caused by the administration of an iodine contrast agent and the presence in the urine of penicillins, cephalosporins and sulfonamides.

Tubular proteinuria occurs due to a decrease in the reabsorption properties of the proximal tubules. In tubular proteinuria, $_2$β□-microglobulins predominate over albumins. Normally filtered $_2$β□-microglobulins from the vesicles are completely reabsorbed in the tubules. In pyelonephritis, acute tubular necrosis, renal transplantation, and congenital tubulopathy, tubular proteinuria is observed.

Sugar. The excretion of sugar in the urine is called glucosuria. Normally, there is no sugar in the urine, but glucosuria is observed when too much carbohydrate is consumed, sometimes in the daytime portion. Therefore, in order to determine the sugar content in the urine, an analysis is taken at breakfast in the morning. At the base, blood sugar levels may be normal and glucosuria may be observed in the urine. This is indicative of tubular dysfunction. This condition is more common in nephrotic syndrome and glomerulosclerosis. See the database and the amount of T e kshim b e defeated the urine of a k e cha-day gathering. At 8 o'clock in the morning b e mor should empty the bladder. Urine collected during a k e cha-day is collected in a single container. Urine from all containers is mixed well, 100-200 ml is poured into a smaller container and sent for analysis.

Urine t e kshim. Urine t e kshim only, but also to determine the functional state of the kidneys (urine and other clinical studies), the other members, for example, urinary bladder, gall bladder, liver diseases, as well as e k it possible to detect metabolic changes b e radi. T e kshim for Conte e ntrlangan morning urine is used, it will b e defeated hospital k e lib ground k e issues of our first morning. K e Yinchang 7-10 days in the hospital for at least 1 times the urine analysis is conducted.

During treatment, Op e k has been portable e - yin, blood transfusion to the patient before or k e yin and the other members of the system to identify pathological changes in urine t e h t e z clinical analysis is necessary. Before the urine of a woman's external sexual organs should be, and if she was menstruating, the directory e t e r urine with the help of k e Cancer. 150-200 ml of urine is sufficient for general analysis. Urine is taken in a well-washed dish or in a bottomed pan. Urine from the pan should be poured into a clean glass container to send to the laboratory. Bhakti e and, (planted) to determine the fungi and biological t e kshim urine prior to the external sexual organs d e zinf e ktsiyalovchi solution (for example, furatsilin or potassium p e rmanganat) with a solution of washing and st e ril directory e t e r special dish 15-20 ml of urine into his mouth at once b e rkitish. The urine Junk E Article ptik added. As a result of the influence of a variety of physical work, the body heat k e moving in a quantity of liquid, depending on the quality of food consumed, and even human emotions b e urine change the properties of their publication.

Urine collection for laboratory t e kshiruvi. K e AFFILIATES things: a ticket to a clean glass container, plastic tube. **Treatment algorithm:** b e morning genitals are thoroughly washed before urine

collection . It is not recommended to take a urine test during menstruation until the end of 2-3 days and 5-7 days after the cystoscope, and if necessary, urine is taken using a cat e t e r. **Urine collection for general analysis.** You will need a few ml of urine for this. For general analysis, the collected urine should **not** be **stored** for more than 1.5–2 h and should have a temperature no higher than 2–8 ° C. B e mor 100-200 ml of morning urine should be collected in a clean dry glass container. Pour on the analysis of the patient's name, and t e kshim and objectives on paper kleylanadi and sent to a laboratory. Bacteriological examination of urine. 118 green images with 12 ml for urine collection . **SARSTEDT** solution with filler part
and contains boric acid to prevent bacteria from multiplying. At room temperature, boric acid prevents bacteria from multiplying for 48 hours. **N chip e HKO urine collection method** . B e morga explains this method of urine collection. To do this, b e morning external genitalia are washed and the "middle part" of the urine is collected in a clean container. For analysis, it is taken in the morning after sleep and the middle part of the urine is taken into a container. Or you can take it at any time of the day. The first and last portions are sent to the toilet. A 12 ml yellow **SARSTEDT (urine)** test tube is used to collect the urine . To do this, urine is taken in a clean container and urine is drawn from the container through a syringe. The syringe is twisted and closed with a yellow cap, and this cap contains a piston. After the syringe is lowered into the urine, the piston is pulled and the syringe is inserted into the urine
is filled to the specified point, ie up to 12 ml. After the urine is taken, the cap is tightly closed and then broken (Fig . 119). -2-3 ml for analysis
118 photos (119 photos)
urine y e tadi. The referral is written and sent to the laboratory in warm urine. 1 ml of urine examination t e kshiriladi and normal cells in 1000, l e ykotsitlar 4000, these cylinders will be up to 220, kidney disease increases.

Urine collection in the Zimnitsky method . B e morgue is explained the rules of urine collection the day before. B e defeated at 6 o'clock in the morning empty the bladder and urinary this part is removed, and then pour it into a 3-hour break every time during the day nurse collects urine, a day before the k e gaining 8 clean glass container prepared for each b e morning, the name, the serial number of the bottle glued paper. B e mor should empty his bladder 8 times from 9 a.m. to 6 p.m. the next day. All of the urine is sent to the laboratory. Measure the daily amount of urine and determine the night and day diur e z. Daytime urine e z (9-18) in the morning the night k e Cancer. M e overall diurnal e zning about 2/3. The amount of urine from 22.00 to 6.00 is the night diur e z. In healthy people, this makes up 1/3 of the total diur e z. The total daily urine output is usually 65-75% of the fluid consumed per day. The functional capacity of the kidneys is considered good if the daytime diur e z is more than the night and the relative density of the urine varies from 1,008 to 1025. The relative density of urine decreased renal

function y e tishmovchiligidan mean b e worldwide. An increase in the relative density of urine occurs in diabetes.

Urine collection by Addis-Kakovsky method . B e morga explains how to collect urine the day before. B e defeated before collecting urine k e gaining at 10 am, urine and empty the box as possible to prevent him from urinating at night. At 8 in the morning of Europe - leaping clean glass bottles are collected and sent to a laboratory. Addis Kakovskiy b e morlardan 10-hour urine was collected and 12 minutes, the amount of 1/50 portion of c e ntrifuga will be under the microscope and gory e v e Rasim like the daily calculation of the amount. Normally, cells of 1 million liters e ykotsitlar, cylinders 2 million 20 thousand elementlarlar form of the disease increases. Urine t e kshim mainly in the morning. L e ykotsit, red blood cells and the number of cylinders (), the foreign e m e a handful for the collected urine to determine tahlilxonaga 1 hour. The referral records the total amount of urine in addition to the general explanatory information. Probation mon conduct e % of cases. If part of the amount of urine does not fit in the bottle, the rest is poured into a second bottle and marked on paper.

Ambyurj e urine collection method for the 3-hour urine is collected in the amount of blood the form of foreign e m e NGOs only 1 minute amount of urine.

V- Methods of functional examination of the kidneys

Kidney function . The kidneys perform the function of homeostasis in the body, regulating extracellular fluid and blood volume, acid-base balance in the body, blood pressure, erythropoiesis, and excreting the final products of nitrogen metabolism .

Its in the urine to determine renal function; 1. the relative density is determined, which can also be determined in the Zimnitsky test; 2. The amount of creatinine in the blood and urine is determined; 3. The filtration rate is determined in the balls; 4. The function of liquefaction and concentration of urine in the kidneys is determined. The relative density of urine is indicative of the liquefaction and concentration function of the kidney. It is the management of extracellular fluid from the cell. The relative density of urine is around 1005-1025. The relative density of urine depends on the amount of fluid that enters and leaves the body. The more fluid you drink, the more urine you have, the lower the density of the nib, and vice versa. if lost, the amount of urine is low and its relative density is high. The high relative density of urine is due to the presence of protein in the urine in nephrotic syndrome and the presence of sugar in the urine in diabetes.

If its relative density in the morning portion of urine is 1020-1018, the concentration function of the kidney can be considered as normal. When the relative density is checked again, if it is low, then the renal concentration function is reduced. Low relative density in urine is observed in chronic renal failure, chronic interstitial nephritis, pyelonephritis, ductal dysfunction, renal diabetes mellitus, polycystosis and hydronephrosis. To determine the concentration function of the kidneys, we use a special Zimnitsky test, in

which 8 servings of urine are collected in separate containers every 3 hours for a day. In healthy people, the daily amount of urine is 70-75% of the fluid consumed. Normally, 65-80% of the daily urine output should correspond to daytime urine. In the daily urine volume, the change in the relative density of urine in the Zimnitsky test should not be less than 12-16 (e.g., 1008-1020; 1006-1022). When the liquefaction function of the kidney is impaired (increased), its relative density in any portion of urine does not fall below 1011-1013, and conversely, when its concentration function decreases, its relative density in any portion does not exceed 1020. A low urine density of less than 1011-1013 is indicative of hypostenuria, which occurs in kidney disease. To determine the ability of the kidneys to concentrate and dilute, according to the Folgard test, dark food is consumed for 36 hours, liquid food is not consumed. Urine is collected just like the Zimnittsky test. When the concentration function of the kidneys is good, the amount of urine is reduced to 500-600 ml. In this case, the relative density of urine increases to 1028-1034. If the concentration of the kidney is reduced, the change in urine volume is small (the amount of urine is almost not reduced), and the relative density of urine does not exceed 1028. It is also possible to carry out a modified method by consuming dry food at the base. In this case, the patient does not drink fluids during the day from 2.00 to 8 am. At 8 o'clock in the morning the patient urinates and this urine is not analyzed and urine is collected for 1-1.5 hours, in which the relative density is determined, the average should be 1024. If it is lower, it indicates a decrease in kidney function.

Renal liquefaction test . In this method, when artificial hyperhydration is performed, the maximum ability of the kidney to dilute urine is determined. The examinee should drink plenty of water once or throughout the day. In a one-time test, the subject drinks fluid for 30-90 minutes . The amount of fluid should correspond to 20 ml / kg body mass. In healthy people, the relative density of urine decreases to 1003. In this case, more than 50% of the fluid consumed during the first 2 hours and more than 80% in 4 hours should be excreted. The relative density of urine does not fall below 1004 when renal function is impaired.

Determination of creatinine content. Creatinine is produced by muscle cells and filtered in the glomeruli, but is not reabsorbed and excreted in the urine. Therefore, with creatinine in the blood, it is possible to determine the excretory function of the kidneys, depending on the amount of it excreted in the urine. The amount of creatinine, like urea and residual nitrogen, does not depend on physical and physical exertion and diet. The amount of creatinine in the blood is 0.06-0.123 mmol / l. When the excretory function of the kidneys decreases, the amount of creatinine in the blood increases.

Determination of filtration rate in balls. In this case, the method of clearance (purification) of transportable filtered, non-reabsorbed substances is used. It is better to use creatinine and insulin and urea. In this method, the amount of creatinine in the urine and blood is determined and determined

according to the formula depending on the minute diuresis. The norm is 80-120 ml / min. Knowing the filtration rate (CFT) in the balls, the filtration is calculated in%. K FT - minute diuresis K FT%

In humans, the physiological state decreases CFT after the age of 40 years, by about 1% per year. After the age of 80, the CFT ranges from 40 to 80 ml / min. In kidney disease, filtration processes are reduced. SBE KFT can be reduced to 2-5 ml / min. This is due to a decrease in the number of nephrons performing the function, a decrease in the ability of the glomeruli to filter, a decrease in plasma flow in the kidney, obstruction of the renal tubules, damaged renal epithelium is associated with the process of multiple filtration. It occurs in chronic glomerulonephritis, chronic pyelonephritis, chronic interstitial nephritis, amyloidosis, nephrosclerosis, arterial hypertension. In addition, a decrease in CFT is observed in hypotension, shock, hypovolemia, advanced heart failure

In rare cases, hyperfiltration may also occur in some pathologies. In this case, the CFT can exceed 120 ml / min. This condition is observed in the early stages of diabetes, in hypertension, in the early stages of chronic glomerulonephritis.

Urine diastasis . T e kshiruv 50 ml conc e rvantsiz new urine has been sent to a laboratory.

R e b e rg test. The method of determining kr e atinin in blood and urine is used to determine how much plasma is filtered and reabsorbed by the renal nodes per minute . Kr e atinin is filtered only in the nodules and not reabsorbed in the renal tubules.

R e b e rg test b e Morgan cr e Athens and ichirmasdan.

The first method b e Morgan nahorga 3 g cr e Athens to birds and 500-1000 ml of water and bladder discomfort k e yin b e defined. K e yin urine is collected in separate vessels every hour and blood is drawn from v e nada every hour . The amount of kr e atin in the blood and urine is determined.

If you have one minute to plasma filtration F , urine e d d e s s e tints defined, F = SkrD P cr formula k e lib. S kr - urinary kr e atinin, P kr - plasma kr e atinin. The r e absorption of water in the tubules is determined by the following formula: (F - D) X 100 and in a healthy person it is 98.5-99%.

The second method b e defeated in the morning emptied the bladder k e Games 1 hour c e Nadal blood taken 2 hours k e yin urine is collected, then the blood and urine cr e Athens The amount is determined.

In a healthy person , nodal filtration on the R e b e rg test is 100–150 ml / min. will be. Kidney y e tishmovchiligida filtration 30-10 ml / min.gacha ur e decreased in the brain of 1-2 ml / min.gacha k e institution.

Practical training 10

Diseases of the kidneys and urinary system. Basic clinical syndromes.

N e frotic syndrome

N e Euphrates edema syndrome in many parts of the body k e prot include e inurl gipoprot e in e of the brain, GIP e rxol e h e e try to be a brain activation email to Ireland. This syndrome

chronic Grom e rolls e frit, pregnant n e fropatiya serving with diabetes Diab e t, amyloidosis, collages e spoiled, tuberculosis, s e psis b e zgak and other diseases. This syndrome is more common in chronic glom e roll e frit. The progression of N e frotic syndrome is mainly due to disruption of fat and protein metabolism. These patients will be large amounts of urine protein particles and lipoic kidney cells d e Survivor itself EPITO e remained cells, dystrophic changes in k e purified.

Clinic: The most obvious syndrome is tumors. They gradually s e animosity escalated in the face, torso, arms and legs spread. Tumor fluid accumulates in the abdominal cavity, pl e vra and p e ricardial cavity, and even in the cavities of large joints. Diur e z decreases to 300-500 ml and oliguria is observed. Urine contains a lot of protein in the amount of 8-20 g / l. Albumin-globulin coefficient e nti k e skin decreases. The cause of tumors is hypoprotein e in e brain. Blood speckled e h e rin increase in the amount of 20.67 mmol / l. N e Euphrates syndrome often inf e ktsion with complications.

Practical training 11

Clinic, diagnosis of acute and chronic glomerulonephritis .

Acute glomerulonephritis

Glomerulonephritis is an immunoallergic disease characterized by inflammation of the vessels of the glomerular apparatus of the kidneys. Pathogenic A gruhgagi b e g TMA E Molitor str e ptokokklar, g e e n chronic inf way through the fabric e ktsiya centers will be the kidneys (otitis media, sinusitis, skarlatina, tonsillitis, teeth, Caribbean e si, pn e vmonia, angina, upper respiratory tract, sometimes diphtheria, pneumonia, rash sweating, diarrhea and mainly streptococci, viral diseases and bq) disease develops. The effects of cold weather and humidity contribute to the development of the disease. Systemic diseases, radiation also play a key role in the development of glomerulonephritis (Fig . 120). Immunoallergic mechanisms play a role in the development of glomerulonephritis.

Clinic. The main complaints of patients are swelling of the face, eyelids, body, legs, headache, decreased ability to work, blurred vision, decreased urine output, discoloration, shortness of breath, suffocation
they complain.

Tumors on the body, on the eyelids, appear mainly in the morning. These tumors then spread to other areas of the body, **120 photos**
there is an increase in blood pressure, changes in the urine, ie hematuria, proteinuria. Acute nephritis can also have a sudden onset, but in most cases

can also be latent. At the onset of the disease, in addition to the known symptoms, the patient's face, eyes swell, body temperature rises, headache, weakness, decreased urine output, oliguria are observed. The disease begins with the appearance of eyelids, swelling and paleness in the face in 80-90% of patients. Due to the swelling on the patient's face in the morning, his eyes narrowed and his face flattened. Under the skin, between the lungs, heart membranes, fluid accumulated in the abdomen (up to 15-20 kg) increases body weight and gradually returns to its state as a result of treatment for 2-3 weeks. Tumor syndrome is caused by: 1) damage to the glomeruli, a decrease in their filtration activity, a decrease in sodium excretion, an increase in reabsorption (reabsorption); 2) accumulation of fluid in the body, an increase in the amount of circulating blood, ie hypervolemia; 3) secondary hyperaldosteronism and antidiuretic (ADG) hormone increase; 4) increased sensitivity of tissues to aldosterone, increased sensitivity of the distal parts of the nephron to ADG and more accumulation of fluid in the body; 5) a change in the hyaluronidase ridge occurs. The permeability of the capillary wall changes; 6) The liquid part of the blood exits the blood vessels and collects in the cavities and between the cells. In nephrotic syndrome, the oncotic pressure of plasma decreases. Hypoalbuminemia and tumors occur frequently and rapidly.

One of the main symptoms of acute glomerulonephritis is an increase in blood pressure. Blood pressure increases moderately in 70-90% of patients and is the result of circulatory disturbances in the kidneys. Accumulation of water in the body with sodium salt leads to hypervolemia. The increase in blood pressure is caused by a decrease in the activity of the renin-angiotensin-aldosterone chain and a decrease in the depressant activity of the kidneys (reduction of vascular tension). A sudden increase in pressure in the arteries complicates heart function, which is seen as acute heart failure (often left ventricular failure). This acute heart failure is manifested in manifestations such as inspiratory shortness of breath, cough, and cardiac asthma.

When examining patients, we pay attention to the appearance of patients, the skin color is flowing, it is possible to see that there is swelling on the face, eyelids, body, legs. In patients with hypertensive form, the capillaries in the retina of the eye narrow and the pressure in the capillary increases and ruptures and blood flows, resulting in decreased vision. **Palpation of** patients **reveals** the extent and nature of the tumors as well as the pulse and slowness. **Percussion** can detect transudate accumulation in the pleural cavity, pericardium, and other cavities. A hoarse-tympanic sound is heard in the lungs. Left ventricular dilatation occurs. It is found that the relative blunt limit of the heart is widened to the left. When we auscultate the lungs, there may be hard breathing in the lungs, wet wheezing, bradycardia in the heart, I tone decreases at the apex of the heart, gallop rhythm due to tension here systolic murmur, II tone accent in the aorta and pulmonary artery, . In most cases, a systolic murmur of a functional nature is heard at the apex of the heart. On the ECG, the amplitude R is high at

standard connections I, II, and III, and a change in T teeth is observed, in some cases a decrease in the voltage of the deep Q tooth and QRS complex. For X-ray examination of patients, a contrast agent sergozin, cardiotrast, which is excreted by the kidneys into the vein, is sent, and we see the exit through the kidneys on X-ray. During retrograde pyelography, sergosine, diotrast, cardiotrast substances are examined by cystoscope through urinary tubes using tubular catheters by sending them to the renal pelvis.

Patients have decreased urine output, reddening of color, erythrocytes and protein appear, and when viewed under a microscope, cylinders and renal epithelium can be seen. One of the first signs of acute nephritis is a decrease in daily urine (400-700 ml), in some cases anuria is observed. Decreased urination is due to changes in the bladder due to inflammation. Because inflammation leads to a decrease in filtration in the balls. The specific gravity of urine and its relative density vary. Acute nephritis is characterized by urinary syndrome-proteinuria, cylindruria, macro- and microhematuria. Not only finely dispersed albumins but also globulins and fibrinogen are released through the walls of the capillaries of the injured balls. There is almost no protein in the urine, although not more than 0.0033. However, the increase in protein in the urine is maintained only for the first 7-10 days, so the amount of albumin in the urine is not very high in late tests. Minor proteinuria sometimes occurs at the onset of the disease, while in other cases it may not occur at all. Low levels of protein in the urine can persist for a long time in acute nephritis, and this disappears altogether at 3-4-6, sometimes 9-12 months. Hematuria is a constant symptom of acute glomerulonephritis. In 13-15% of cases, macrohematuria is observed, in other cases, microhematuria is detected, in which the amount of erythrocytes in the urine sediment does not exceed 10-15 in the field of view. In acute glomerulonephritis, hyaline, granular, sometimes epithelial cylinders are found in 75% of patients, and cylindruria may not be observed in 25%. Deficiency of leukocytes from erythrocytes can be checked by urinary sedimentation by Addis-Kakovsky, Nechiporenko methods.

Due to decreased renal function, sometimes the amount of nitrogen residue in the blood, the amount of urea may increase slightly. Azotemia does not last long. In acute nephritis, a decrease in hemoglobin and erythrocytes in the blood is observed. The rate of erythrocyte sedimentation increases.

Chronic glomerulonephritis

Chronic glomerulonephritis is a long-lasting immunoallergic inflammation of both glomeruli. The disease is characterized by decreased renal function, increased arterial blood pressure and the development of renal failure due to gradual injury of the renal glomeruli.

Clinic. The clinical picture of chronic glomerulonephritis depends on its type, 1. Changes in urine: proteinuria, hematuria, cylindruria. 2. Changes that occur as a result of high blood pressure. 3. It is accompanied by changes in the body, such as swelling.

The following clinical types of chronic glomerulonephritis are distinguished:

1. Latent glomerulonephritis is a common type characterized by slight changes in the urine. In this type, blood pressure does not increase, and the tumor is not noticeably noticeable. The latent period can occur after 30-40 years. Patients do not lose their ability to work for many years, they do not feel sick. Often, the latent type of chronic glomerulonephritis is characterized by changes in urine during dispensation (decreased specific gravity, more red blood cells, more urine at night), a slight increase in blood pressure, accelerated ECG, decreased protein in the blood, determined on the basis of increased cholesterol. In most cases, it is detected when kidney failure develops. Renal insufficiency is caused by an increase in the amount of residual nitrogen and urea in the blood.

2. Nephrotic manifestations of chronic glomerulonephritis. Excessive protein excretion in the urine is more than 3-5 g per day, the amount of protein in the blood (hypoproteinemia) decreases, especially with a decrease in albumin, globulin ratio (dysproteinemia), an increase in cholesterol (hypercholesterolemia-600-800 mg%) represented by Hypoproteinemic tumors appear in the body of patients. Unlike tumors of the heart and other tumors, such tumors begin in the morning on the eyelids and gradually spread throughout the body. They have a permanent feature and even spread to the internal organs, heart, lungs and abdominal cavities. The skin, mucous membranes dry out, muscles atrophy, resulting in skin wrinkles. As protein is excreted in the urine, triglycerin and free cholesterol in the blood increase. The nephrotic appearance of chronic glomerulonephritis is accompanied by symptoms of nephrotic syndrome and nephritis (blood in the urine, decreased renal filtration). Blood pressure rises in the last period of the disease.

3. The hypertensive form of chronic glomerulonephritis is often characteristic of latent glomerulonephritis. Blood pressure is ischemia in the upper staglomerular apparatus in the kidney, which increases renin synthesis, which in turn leads to an increase in blood pressure. When renal function decreases, systolic and diastolic pressure increase steadily, gradually cardiac activity is also impaired, heart rate increases, I tone decreases in the apex area, systolic murmur occurs in aortic projection, and II tone accent is heard, three rhythmic tone i.e. the sound of a horse's trumpet is heard, then heart failure is added, a heart attack and asthma may occur. In chronic glomerulonephritis develops increased blood pressure, ischemic heart disease, angina. The patient may have myocardial infarction, cerebral hemorrhage. **When examining** patients **,** patients may see skin discoloration, swelling of the face, eyelids, body, legs, and scars on the body (see Fig. 121) . B e morler eye arterioles in the retina capillaries constrict and as the pressure in the capillary increases the venules dilate and bleed, there is exudation and swelling in the retina, at the end of the disease the optic nerve suckers change, due to which the ability to see decreases. Changes such as

neuroretinitis occur in the fundus of the eye, there is a change in the optic nerve suckers. Only in the last stage of the disease can there be serious changes in the retina that can not be treated. Palpation of patients reveals the spread and nature of the tumors, as well as a slowing of the pulse. Percussion can detect transudate accumulation in the pleural cavity, pericardium, and other cavities.

A hoarse-tympanic sound is heard in the lungs,

the relative blunt border of the heart shifts to the left.

4. Mixed appearance of chronic glomerulonephritis. This appearance is characterized by an increase in blood pressure and the appearance of symptoms of nephrotic syndrome. There are changes such as urinary incontinence syndrome (proteinuria, hematuria, cylindrnia), in which the patient's condition is satisfactory at the onset of the disease, and then develops renal failure after 2-5 years. The course of chronic glomerulonephritis consists of 2 stages: compensated and

Figure **121 is** decompensated. In the compensatory phase, the nitrogen-releasing function of the kidney is preserved. This stage is long, latent, and is manifested only by changes that occur in the urine. In the decompensated stage, the nitrogen-releasing function of the kidney is impaired. At this stage, urinary symptoms are insignificant but, with edema, hypertension is significant. This stage can begin within a period of more than 1 year (up to 30-40 years) after the onset of the disease. At this stage, kidney function decreases. Nitrogen excretion, urine thickening activity decreases. Changes in urine decrease, specific gravity decreases around 1007-1008. Protein is constantly excreted in the urine. Blood pressure is high. Swelling occurs in the skin, serum urea, creatinine, indigo increases, protein decreases.

The high number of leukocytes, the presence of active leukocytes, changes in the structure of the renal vessels on radiological examination of the kidney indicate the presence of chronic pyelonephritis. The nephrotic type of chronic nephritis should be compared with renal amyloidosis, diabetic glomerulosclerosis, liver cirrhosis. In the nephrotic type of chronic nephritis, signs of inflammation of the kidneys (hematuria, hypertension), decreased renal glomerular filtration, and decreased renal concentration activity play a key role in the diagnosis. A biopsy of the kidney confirms the diagnosis. To differentiate from amyloidosis, the oral mucosa is examined histologically. It is necessary to examine the renal and urinary tract using X-rays, take an X-ray of the kidney using an isotope and contrast agent, UTT examination, examination of the renal vessels (angiography). The use of renal tissue examination-puncture biopsy helps to diagnose the disease.

Practical training 12

O 'tkir and chronic pielonefritlarni symptom-matologiyasi. Acute and chronic renal failure. clinic, diagnostics .

Sharp pi e lon e frit

Acute pi e e announced frit directly to the right of kidney supplies and kidney tissue in connection with the microbes pass k e lib from the info, e ktsion disease.

Clinic: This disease is often accompanied by clinical symptoms of the Trinity: spasms, the K e yin heat, b e convene pain and often one-sided pain, dysuria events to try to e to Ireland. Headaches, change in urine color, turbidity b e zovta. B e ldagi pain, pain is usually simmilab b e radio, the man changed his status enhanced. Pregnancy pi e lon e frit is a separate form of the disease. It is characterized by subf e bril t e mp e ratura and a less symptomatic clinical picture. Pain and dysuria in the B e l area usually do not occur which makes diagnosis difficult. Analyzing the urine of pregnant again and again to see how to help diagnose b e worldwide. If the kidney is enlarged, it is palpable and painful on palpation. Low e rnatsky symptom is positive when percussion is performed. Examination of the blood of patients shows leukocytosis (30,000-40,000), aneosinophilia, an increase in the number of neutrophils, a shift to myelocytes. A large amount of urine (Piura, bacteriuria) l e ykotsituriya prot e inurl 0.5-1.0 g / l, myxoma e Jaafari, l e ykotsitlar and granular cylinders, a small amount of g e Mature found. During acute pi e lon e frit, intestinal bacilli, staphylococcus, vulgar prot e y, ent e rocococcus are found in the urine. B e Morley r e NTG email technology, ultrasound, computer e r tomography, radioisotope r e ethnographic (scanners) t e kshiruvlaridan transferred to it. Inflammation and enlargement of the kidneys can be detected in the renal par e .

Chronic pyelonephritis

Chronic pyelonephritis is a specific chronic inflammation of the intermediate (interstitial) t reading of the kidney due to bacterial exposure, in which the renal vessels and parenchyma are damaged.

Inquiry of patients. Symptoms of the disease depend on its type and stage, how it progresses, whether the pain has spread to one or both kidneys, how much urinary tract permeability is impaired. The following types of chronic pyelonephritis are: 1) latent; 2) sensitive; 3) accompanied by an increase in blood pressure; 4) azotemic species are distinguished. The latent type is characterized by very few clinical signs. Patients complain of general malaise, rapid fatigue, and sometimes subfebrile fever. Sometimes in patients, urine is excreted frequently (pollakiuria), rarely, irritably. Pasternatsky's symptom turns out to be positive, the pain in the kidney area intensifies when the disease progresses. Gradually the relative density of urine decreases (1006-1008). **Laboratory testing.** Patients have increased urea and uric acid in their blood, which results in uremia, with mainly pyuria (large numbers of leukocytes) and bacteriuria in the urine. Laboratory examination of urine reveals insignificant protein excretion, multiple white blood cell (leukocyturia) and bacterial excretion, Addis-Kakovsky, Nechiporenko method confirms leukocyturia. Recurrent type of pyelonephritis is the most common. It is characterized by a period of remission (recurrence of the disease) and an alternation of periods of exacerbation, pyelonephritis can

recur for various reasons. Pyelonephritis often develops after the flu, when there is a cold, when the urinary tract, the bladder is re-inflamed, when the patient is weak. Patients complain of a strange sensation in the lumbar region, changes in dysuric urine output, a rise in body temperature to 37-38 ° C. As the disease progresses, blood pressure rises (from 180/100 to 250/140 mm Hg). In particular, diastolic pressure remains high. The renin-angiotensin-aldosterone system plays a key role in the origin of hypertension. In the urine there is an increase in protein, leukocytes, cylinders, bacteria, erythrocytes. During the onset of the disease, moderate anemia develops, ECG accelerates and may be 20-30 mm / s. Examination of the blood reveals neutrophil leukocytosis. The type of increase in blood pressure is manifested by symptoms characteristic of suffocation. Headache, dizziness, shortness of breath, pain in the heart area, palpitations, sometimes changes in urine are manifested from time to time. High blood pressure during this period is the only sign of chronic pyelonephritis. Gradually begin to kidney failure, azotemia, residual nitrogen, urea, creatinine in the blood too much of pants, uric acid k o ' strut and as a result develop uremia, urine, mainly in Piura (k o p mi q ropes leukocytes) , etc. as well as bacteriuria b o takes the outbreak of chronic kidney failure. The data obtained from nausea, vomiting, weakness, weight loss, and laboratory tests are specific to renal failure. These include polyuria, hypoisostenuria, nocturia, pollakiuria. Proteinuria, hematuria, leukocyturia, cylindruria are unavoidable. Azotemia, on the other hand, is stable and multiplies. Patients should be examined by X-ray, ultrasound, computed tomography, radioisotope renography (scanning). Inflammation in the renal parenchyma and decreased kidney size can sometimes be detected, and sometimes kidney size can also be enlarged.

Practical training 13

Musculoskeletal and connective tissue system. Methods of examination: interrogation, methods of physical examination, methods of laboratory-instrumental examination. Basic clinical syndromes. TQB, scleroderma clinic, diagnosis .

. *Practical training module* .

Training time - 4 hours	The number of students 1 : 0 to 12
Form of training	Practical training
Practical training plan	Examination of patients with arthritis. Inquiry. Examination and palpation of the joints. Symptomatology of rheumatoid arthritis. The concept of osteoarthritis. Examination of patients with connective tissue disease. The concept of collagenoses.
The purpose of practical training :	Examination of patients with arthritis . Symptomatology of rheumatoid arthritis. The concept of osteoarthritis. Examination of patients with connective

	tissue disease .
Teaching style	Inquiry. Demonstration of patients, interactive teaching methods, practical skills.
Form of teaching	In small subgroups.
O ' unit equipment	Calls to O Training Guidebook , practical Typo content , projectors , computer .
Training mode	Methodically equipped auditorium.
Monitoring and evaluation	Oral control: questions and answers, tests, problem solving.

1.2 . ***Technological card of practical training***

Stages and timing of work.	Educator	Learners
In the preparatory stage of q	1Checks the cleanliness of the auditorium 2 Checks the readiness of students for training Controls the 3D machine	
1. Introductory stage of training (10 minutes)	1. Preparation of educational content on the topic. 2. Preparation of presentation slides for the introductory speech 3. Develop a list of references used in the study of science	
See the 2 basic levels of diarrhea (160 da q i q a)	1. Divide students into small groups and ask questions on the topic. 2. Uses display posters 3. Uses slides, multimedia 4. Conducts treatment 5. Summarizes and summarizes the information provided on the basis of topics, encourages and actively evaluates the active participant students	They are divided into small groups They watch They participate They listen and answer questions
4- final press q ich (10 da q i q a)	1. Concludes 2. Provides independent work 3. Gives homework	He listens Takes notes Takes notes

3. Assessment of students' theoretical knowledge:

A) Frontal method:

1. Complaints of patients with arthritis
2. Examination of patients with arthritis, palpation

3. Laboratory and diagnostic examination

4. General symptoms and localization of joint diseases

5. Rheumatoid arthritis. Describe arthritis

6. Clinical manifestations of rheumatoid arthritis

7. Activity levels of rheumatoid arthritis

8. Course, consequences and treatment of rheumatoid arthritis

9. Describe deforming osteoarthritis

10. Etiopathogenesis of osteoarthritis

11. Diagnosis of osteoarthritis, additional examination methods

12. The course of osteoarthritis, treatment, prevention

13. Etiopathogenesis of systemic lupus erythematosus, clinic

14. Diagnostic criteria of SQYU

15. Clinical course of etiopathogenesis of systemic scleroderma

16. Diagnostic criteria for systemic scleroderma

17. The concept of allergies

18. Causes of urticaria, clinic, treatment

19. Causes of Quincke's tumor, clinic, 1st aid

20. Causes of anaphylactic shock, clinical diagnosis, edema care

"Academic controversy" method

The group is divided into two groups, each of which is assigned a situational issue, for example, “consultation doctor-patient”. In each group, 1-2 students write down the pros and cons of the consultation - "lawyers", the other 2 students write down the disadvantages of the consultation - "prosecutors".

The findings of lawyers and prosecutors will be analyzed by the whole group.

Subject statement

Systemic diseases of connective tissue

Scheme of examination of patients with systemic diseases of connective tissue

I. Methods of subjective examination

a. Primary and secondary complaints

b. Collection of medical history

v. Collection of life history

II. Methods of objective inspection

a. Review;
b. Palpation;
v. Percussion;
g. Auscultation

III. Laboratory testing methods

a. General blood test.
b. Biochemical analysis of blood.
v. Urine analysis
g.Cytomorphological examination
g. Methods of morphological examination.

IV. Instrumental verification methods

a. X-ray examination.
b. Ultrasound examination.
v. Computed tomography
g.Angiography

The system is a red wolf

Li system lupus erythematosus (SKV) is a chronic systemic connective tissue autoimmune inflammatory disease of the internal organs (skin, joints, lungs, heart, kidneys, digestive system, liver, nervous system, etc.) and damage to blood vessels and the accumulation of fibrinoids in their walls, as well as the formation of autoantibodies, especially antinuclear antibodies, which circulate in the blood and have a wide range of effects. A sosan 80% of patients become ill before the age of 30 and 80-90% of women become ill. In 1875, M. Kaposhi recorded 141 photo injuries of the internal organs terry , along with rash butterfly syndrome on the nose in the facial area (see
Fig . 140) . **140 photos**

In 1895, B. Osler described arthritis with skin damage. In 1948, LE cells were found. This is why it is called a systemic disease.

1 41 photos

Figure 142 below shows damage to the oral cavity (a) and facial skin (b) .

a 142 photo b

Mesangial lupus nephritis is characterized by moderately expressed clinical symptoms. Occurs in 10% of cases and is associated with proliferation of mesangial cells. Deposits of O immunoglobulin and a third type of complement (SZ) are always found in the mesangial . Accumulation of deposits is characteristic of the first stage of mesangial lupus nephritis (volcanic nephritis) because the filtered immunocomplexes soon settle in the mesangial.

Focal glomerulonephritis is accompanied by partial (up to 50%) damage to the renal **glomeruli** , in which the renal glomeruli swell, endothelial and mesangial cells proliferate, focal necrosis in the capillaries, neutrophil infiltrations are observed. In some places fibrinoid deposits and intracapillary thrombi are found. Focal glomerulonephritis is clinically manifested in the form of microhematuria and proteinuria.

Diffuse proliferative glomerulonephritis is the most common type of volcanic nephritis, accounting for 40–50%. Morphological changes include proliferation of endothelial mesangial cells and thickening of the basement membrane. Sometimes it can be observed that the epithelial cells of the Shumlyansky-Bouman capsule proliferate, forming a characteristic crescent, and then the onset of sclerosis in the glomeruli. It is characteristic that the kidneys are symmetrically damaged and all the glomeruli are involved in the pathological process.

Membranous glomerulonephritis is characterized by thickening of the basal membrane of the capillaries and migration of the endothelium of the same vessels. Plasma proteins pass through the exposed basal membranes and are absorbed, leading to the onset of the phenomenon known as “wire loops”. Deposits of immune complexes accumulate in the basal membranes. Membranous glomerulonephritis does not have changes such as focal necrosis, neutrophil infiltration, mesangial cell proliferation. This type of nephritis occurs in 10% of cases and is very similar to idiopathic membranous glomerulopathy in terms of its clinic and morphology. In the pathogenesis of the described forms of lupus nephritis, the accumulation of DNA-anti-DNA complex (ie antigen - antibody complex) in the vascular bundles is important. Immune deposits are found in endothelial and subepithelial mesangials. In diffuse proliferative glomerulonephritis, the subendothelial location of immune complexes is characteristic of systemic lupus erythematosus, which enters the acute phase. As the endothelial cells proliferate and the basal membranes undergo alteration, the path of the capillaries narrows and sometimes becomes completely obliterated. These changes lead to damage to the renal tubules due to ischemia and the onset of interstitial fibrosis. The size of the kidney involved in the pathological process may be normal. Later it enlarges and begins to turn white, and a number of tiny blood clots appear in the crustal layer. Gradually, sclerotic processes begin in the kidney, leading to its secondary wrinkling. In the systemic lupus erythematosus, the tubules and interstitium are also damaged. The appearance of immune complexes between the ducts leads to the onset of diffuse interstitial fibrosis.

The joints also join the process, swelling occurs, and nonspecific mononuclear cell infiltration begins in the synovial membranes. Focal fibrinoid necrosis foci appear in the subepithelial connective tissue. Examination under a microscope reveals hyperemia and thickening of the synovial membranes. However, the phenomenon of destruction is not observed in the synovial membranes and

joint joints, due to which the shape of the joints does not change, i.e. there is no deformation event.

The spleen may be of normal size or slightly enlarged, the capsule thickened, and the follicles hyperplasia. The pulp contains many plasma cells containing immunoglobulins O and M. Around the central arteries of the spleen, a ring-shaped fibrous tissue that grows in the form of a lattice is visible, which is called ‘onion-like’ sclerosis.

Lymph nodes enlarge due to nonspecific changes initiated in the germinative center of the follicles. Plasma cells are visible in the perifollicular zone. In other organs and tissues , acute vasculitis, foci of mononuclear infiltration, and fibrinoid deposits beginning in capillaries are found. Necrosis that begins in the head and spinal cord leads to acute vasculitis and microinfiltrates, or microhemorrhages.

Clinical manifestations . Systemic lupus erythematosus most often begins in girls and women in their 20s and 30s, most often in adolescent girls. Fever, pain in the joints without changing the shape, pain in the chest (due to pleurisy), hypersensitivity to sunlight (photosensitization), facial skin the appearance of erythematous rashes ("butterfly" pattern), both on the hands and feet and mucous membranes
s The occurrence of such rashes is characteristic

(143 photos) .

Systemic lupus erythematosus is found in 100% of cases and to a lesser extent in other autoimmune diseases. The titer of the DNA-anti-DNA complex is a diagnostic marker of systemic lupus erythematosus and corresponds to the degree of kidney damage. The most characteristic clinical signs for lupus nephritis are: hematuria, proteinuria, and in some cases, classic nephrotic syndrome. In membranous and diffuse proliferative glomerulonephritis, persistent renal failure is observed with azotemia. Damage to the nervous system is manifested in the form of meningoencephalitis, meningoencephalomyelitis, which is accompanied by polyradiculoneuritis. The presence of psychoses is also described. Occasionally there are symptoms typical of damage to the gastrointestinal tract: vomiting, constipation, loss of appetite.

Diagnostic criteria . Butterfly symptom, discoid volcanic, Peyno syndrome, alopecia (hair loss), photosensitization (increased sensitivity to sunlight), painless sores in the mouth, throat and nose, arthritis without deformation, LE-cells (multinucleated neutrophils) , False positivity of the Wasserman reaction, presence of proteinuria, cylindruria, pericarditis clinic, psychosis and seizures , hemolytic anemia, leukocytopenia, thrombocytopenia (4 of them are grounds for diagnosis.

However, other laboratory changes, an increase in the amount of fibrinogen in the blood, an increase in the amount of globulins (alpha and gamma), an increase in ECG are observed.

The system li sklerodermia

Systemic scleroderma (systemic sclerosis) is an autoimmune disease characterized by the appearance of progressive diffuse fibrosis in the skin and internal organs of the body. Although skin lesions are the main symptom of systemic scleroderma (observed in 95%), damage to internal organs - gastrointestinal tract, lungs, kidneys, heart and transverse muscles - is life-threatening. is dangerous. The disease mainly 30-50 year-old women with the disease , but people see how y o countdown, including the perinatal period was also a chance to stay with the pain of the disease. Depending on the clinical manifestations, the following forms of systemic scleroderma are distinguished: 1) classic form of scleroderma, in which the skin of the body passes with a flat lesion, the process progresses rapidly and the internal organs are damaged in the early stages of the disease;

2) a form of limited damage to the skin of the body, mainly with damage to the skin of the fingers and face (CREST-syndrome). Calcinosis, Raynaud's syndrome , see Fig . 144 , is characterized by esophageal damage, sclerodactyly accompanied by telangiectasia. In this case, the internal organs are involved in the process much later. The disease is mild.

144 photos

Etiology and pathogenesis. The etiology of this disease is unknown, but systemic scleroderma is considered to belong to the group of autoimmune diseases. The following play an important role in the pathogenesis of the disease: impaired collagen synthesis, microcirculatory vascular damage is characterized first, followed by the onset of specific scleroderma angiopathy (obliterative endarteriolitis). The onset of progressive fibrosis in the skin is mainly due to the synthesis of collagen in the normal structure, which is forced by fibroblasts. At the same time, fibroblasts produce collagenase, which is involved in collagen degradation, in amounts that are characteristic of the norm, so that as collagen synthesis increases, it becomes redundant.

It is assumed that immunological factors play a role in the overgrowth of connective tissue and within the system. In the early stages of the disease, T-lymphocytes sensitized to collagen are found among the cells of the inflammatory infiltrate. Not surprisingly, a slow-onset hypersensitivity reaction to collagen initiates the release of lymphokines, which are known to attract fibroblasts and increase collagen synthesis.

System sklerodermii disease humoral immune dysfunction. Hypergammaglobulinemia, antinuclear antibodies, and rheumatoid factor are often found in blood serum . A Recent antinuklear two types of antibodies. Systemic scleroderma of one type, called 5 (1-70 (scleroderma-70)) is found in 30-40% of cases of diffuse type, whereas antisentromer antibodies (antibodies affecting the central chromatin) are detected in 50-70% of cases in CREST-syndrome .

Systemic scleroderma is also considered to be a specific diffuse vascular pathology that resembles obliterative endarteriolitis and leads to numerous vasospastic changes.

Damage to the microcirculatory system is thought to be due to primary damage to the vascular endothelium under the influence of cytotoxic factor present in the serum of patients. Damage to the endothelium leads to aggregation by platelet adhesion and the release of platelet factors leading to periadventional fibrosis. The vascular pathway narrows, causing damage to various organs due to ischemia.

Pathological anatomy. In systemic scleroderma, the pathological process develops in various organs, but the skin, musculoskeletal system, gastrointestinal tract, lungs, kidneys, heart are more affected . It starts with 'lims. The process then spreads to the upper parts of the arms, shoulders, neck and face. Three stages of morphological changes are distinguished: 1) solid tumor stage; 2) induration phase; 3) stage of atrophy. In the **first stage of the** disease there is swelling in **the** skin, in which the amount of the main substance increases, and inflammatory infiltration around the veins and skin products occurs. **In the second stage** , sclerosis begins in **the** dermis, sclerosis and hyalinosis appear in the vascular wall, collagen bundles thicken, hyalinosis begins, and subcutaneous tissue also joins the process. **The third stage** is skin atrophy, which begins several years after the onset of the disease. Examination under a microscope reveals hyaline tissue areas and diffuse epidermis atrophy in the skin and subcutaneous tissue, significant narrowing of the microcirculatory vessels, atrophy of skin products. These changes are accompanied by necrosis and trophic ulcers. In addition, the skin becomes pale (with vitiligo) and spots appear and darken. The man's face looks like a mask. In the subcutaneous tissue, there are areas of focal and scattered accumulation of lime, which is especially noticeable in CREST-syndrome.

In systemic scleroderma, changes that occur in the skin are often accompanied by damage to the joints, bones, and muscles. Nonspecific exudative synovitis begins with fibrosis of the synovial membranes, followed by resorption of the underlying bone. The fingers become thinner, the tip sharper, and look like bird claws. Skin fibrosis restricts their movement. Sclerotic atrophy of the fingertips may end with the disappearance of the phalanges of the fingertips. Recurrent wounds, chronic ischemia associated with vascular occlusion 145 a Fig . Can lead to spontaneous amputation of the fingers.

a) b)

1 45 A and B picture

a) Sclerodermic finger

b) appeared in the skin of the foot ulcer (ulcer went suyakkacha 145 b image)

Periarticular connective tissue sclerosis restricts joint movement. However, joint destruction is rarely observed. Focal inflammatory infiltrates that later lead to the onset of fibrosis can also occur in skeletal muscle. As a result, muscle atrophy begins in patients with systemic scleroderma.

Diffuse interstitial fibrosis begins in the alveolar barrier in the lungs , the onset of which is mainly due to the synthesis of collagen in the normal structure, which is forced by fibroblasts, leading to increased shortness of breath. The walls of the capillaries become thicker and thicker. Cysts can form as a result of the growth of connective tissue in the lungs.

Y symptoms of interstitial fibrosis around the vessels, lymphocytes and macrophages that infiltrate perivaskulyar. The walls of the small arteries and arterioles become thickened. Pneumosclerosis of the lungs leads to hypertrophy of the right side of the heart.

In the gastrointestinal tract, atrophy of the mucous membrane and smooth muscle, hyalinosis begins with sclerosis of the base and serous membrane between the mucous membranes, erosions and ulcers appear. Mainly the esophagus, stomach, small intestine are affected, the damage of the colon is less common. Periductal, perivascular sclerosis is observed in the liver. The onset of fibrosis within the lesions is less common. The walls of the vessels become hyaline and the hepatocytes undergo fatty dystrophy.

In the kidneys are found thrombosis, microinfarctions, necrosis of the cortex, called scleroderma kidney. Concentric proliferation occurs in the small arterial intima, deposits of glycosaminoglycans are found, reductation of the inner elastic membrane, and hyalinosis are observed. But these changes do not correspond to the level of hypertension. In addition, fibrinoid necrosis of arterioles is found in the kidneys, focal necrosis of the glomeruli, thickening of the basal membrane, enlargement of the mesangia, dystrophy and necrosis of the epithelium of the renal tubules. More than half of the patients die from kidney failure.

Changes in the nervous system, including the peripheral nervous system, are due to damage to the microcirculatory vessels.

Clinical manifestations. It reflects the growing nature of the disease within the system. In most cases, the disease begins with a vascular change that is characteristic of Raynaud’s disease. Changes in the blood vessels usually begin before changes occur in the skin. Increased collagenation of the skin leads to atrophy of the hands, limited vascular mobility , and then the appearance of contractures and disruption of muscle movements. Sclerotic processes in skeletal muscles and their atrophy exacerbate these changes. In all patients with systemic scleroderma, the musculoskeletal system is damaged, which is one of the reasons why patients become disabled. In 80-90% of patients, the skin is damaged in a characteristic way, which changes their appearance. The patient's face is shaped like a mask, wrinkles appear around the lips (Kiset's

symptom, see Fig. 146), and sclerodactyly is observed in the palms of the hands. The veins of the face and fingers are dilated. The skin of the fingers is tightened and tightened, mainly in such patients as Raynaud's syndrome. As the pain gets worse, the skin on the body becomes more and more damaged.

146 photos

Along with sclerotic processes in the skin in some places appear areas of discoloration, discoloration and blemishes. Damage **to the gastrointestinal tract** is manifested in the form of decreased esophageal peristalsis and indigestion (dysphagia). Absorption of nutrients is impaired due to atrophy and fibrosis of the base and muscle layer under the small intestinal mucosa.

Diffuse pneumosclerosis begins in the lungs and sometimes cysts appear. Pleural fibrosis is noticeable, which eventually leads to the onset of bronchiectasis, emphysema. Clinically , these changes are manifested in the form of persistent cough, symptoms characteristic of respiratory failure. Lung damage leads to the onset of failure in the right side of the heart.

The kidneys are usually involved in the process when systemic scleroderma is semi-acute and chronic, followed by glomerulonephritis and nephrotic syndrome. When the kidney becomes truly sclerodermic, it is characterized by the onset of renal failure soon: azotemia, oliguria, anuria, arterial hypertension, which can lead to the death of patients. In the nervous system there is a picture of neurocirculatory dystonia, polyneuritis.

Patients with CREST syndrome have five characteristic symptoms: calcinosis, sclerodactyly, esophagitis, Raynaud's syndrome, and telangiectasia (Figures 147 a and b and 148 a and b) .

148 Figures a and b

That is why the name of this syndrome is derived from the first letters of the Latin names of these symptoms that occur in it.

The outcome of the disease depends on how it progresses, whether it is identified in a timely manner and treated appropriately. It is usually somewhat better when the disease is chronic, as the process progresses slowly and sclerotic processes with the skin of the body begin slowly. In cases where systemic scleroderma is acute, especially in cases where true scleroderma renal status begins, the outcome of the disease is poor because renal failure can progress rapidly and lead to death.

Practical training 14

Endocrine glands and metabolic system. Control methods. Inquiry, physical examination methods. Methods of laboratory and instrumental examination.

1. Practical training module .

Training time - 4 hours	The number of students 1 : 0 to 12
Form of training	Practical training
Practical training plan	Symptomatology of thyroid disease (hypo and hyperthermia). Symptomatology of diabetes. The concept of diabetic and hypoglycemic coma. Basics of ambulance.
The purpose of practical training :	Study of symptomatology of thyroid disease . Examination of patients with diseases of the endocrine glands. Symptomatology of diabetes. The concept of diabetic and hypoglycemic coma. Basics of ambulance.
Teaching style	Inquiry. Demonstration of patients, interactive teaching methods, practical skills.
Form of teaching	In small subgroups.
O ' unit equipment	Calls to O Training Guidebook , practical Typo content , projectors , computer .
Training mode	Methodically equipped auditorium.
Monitoring and evaluation	Oral control: questions and answers, tests, problem solving.

1.2 . ***Technological card of practical training***

See the stages of hearts and q ti.	Educator	Learners
In the preparatory stage of q	25. See the audience to control the purity karîm 26. Checks students ' readiness for training 27. And control q karîm	
1. bullet and training g contain levels of intra q (10 da q i q a)	1. Preparation of educational content on the topic. 2. Preparation of presentation slides for the introductory speech 3. Develop a list of references used in the study of science	
2 - the main stages of diarrhea q (160 da q i q a)	1. Divide students into small groups and ask questions on the topic. 2. Uses display posters 3. Uses slides, multimedia 4. Conducts treatment 5. Summarizes and summarizes the information provided on the basis of topics, encourages and actively	They are divided into small groups They watch participate They listen and

	evaluates the active participant students	answer questions
1- final press q ich (10 da q i q a)	1. Closing Summary q karîm 2. Musta q il gives work 3. Gives homework	 He listens Takes notes Takes notes

3. Assessment of students' theoretical knowledge:

A) Frontal method:

1. 1. Complaints of patients with arthritis
2. Examination of patients with arthritis, palpation
3. Laboratory and diagnostic examination
4. General symptoms and localization of joint diseases
5. Rheumatoid arthritis. Describe arthritis
6. Clinical manifestations of rheumatoid arthritis
7. Activity levels of rheumatoid arthritis
8. Course, consequences and treatment of rheumatoid arthritis
9. Describe deforming osteoarthritis
10. Etiopathogenesis of osteoarthritis
11. Diagnosis of osteoarthritis, additional examination methods
12. The course of osteoarthritis, treatment and prevention
13. Etiopathogenesis of systemic lupus erythematosus, clinic
14. Diagnostic criteria of SQYU
15. Clinical course of etiopathogenesis of systemic scleroderma
16. Diagnostic criteria for systemic scleroderma
17. The concept of allergies
18. Causes of urticaria, clinic, treatment
19. Causes of Quincke's tumor, clinic, 1st aid
20. Causes of anaphylactic shock, clinical diagnosis, edema care

The “beehive” method

In this method, the problem is analyzed with the whole group or with two small groups. The assigned task can be assigned to one or two different groups for the whole group. Within 10-15 minutes, group participants analyze the task solution and report to each other. The best option will be selected from them.

Subject statement

Methods of diagnosis of diseases of the endocrine system

Scheme of examination of patients with diseases of the endocrine system.

1. Methods of subjective examination.

a. Primary and additional complaints are collected.

b. Medical history is collected.

v. Life history is collected.

2. **Methods of objective inspection.**

a. Review.
b. Palpation.
v. Percussion.
g. Auscultation

3. **Methods of laboratory testing.**

a. General blood test (hypochromic anemia, leukopenia with relative lymphocytosis, increased ESR) .
b. The amount of sugar in the blood is determined.
v. Ketone bodies are detected in the blood .
g. Protein-bound iodine is detected.
d. T riyodtironin (T3) , thyroxine (T4) to determine the amount.
e. C reading at urea, creatinine, calcium is determined.
j. The amount of sugar in the urine.
z. N is determined ajas calcium (increase) .

4. Methods of morphological examination: thyroid gland, adrenal gland, pancreas biopsy, cell composition.

5. Instrumental inspection methods.

a. X-ray examination.
b. Radioisotope testing method
v. Ultrasound examination.

1 22 photos

Practical training 15

Diseases of the endocrine glands and metabolic system. Diabetes mellitus, thyrotoxicosis, hypothyroidism clinic, diagnosis .

1. Practical training module .

Training time - 4 hours	The number of students 1 : 0 to 12
Form of training	Practical training
Practical training plan	Symptomatology of thyroid disease (hypo and hyperthermia). Symptomatology of diabetes. The concept of diabetic and hypoglycemic coma. Basics of ambulance.
The purpose of practical training :	Study of symptomatology of thyroid disease . Examination of patients with diseases of the endocrine glands. Symptomatology of diabetes. The concept of diabetic and hypoglycemic coma. Basics of ambulance.
Teaching style	Inquiry. Demonstration of patients, interactive teaching methods, practical skills.
Form of teaching	In small subgroups.
O ' unit equipment	Calls to O Training Guidebook , practical Typo content , projectors , computer .
Training mode	Methodically equipped auditorium.
Monitoring and evaluation	Oral control: questions and answers, tests, problem solving.

1.2 . ***Technological card of practical training***

See the stages of hearts and q ti.	Educator	Learners
In the preparatory stage of q	28. See the audience to control the purity karîm 29. Checks students ' readiness for training 30. And control q karîm	
1. bullet and training g contain levels of intra q (10 da q i q a)	1. Preparation of educational content on the topic. 2. Preparation of presentation slides for the introductory speech 3. Develop a list of references used in the study of science	
2 - the main stages of diarrhea q (160 da q i q a)	1. Divide students into small groups and ask questions on the topic. 2. Uses display posters	They are divided into small groups They watch

	3. Uses slides, multimedia 4. Conducts treatment 5. Summarizes and summarizes the information provided on the basis of topics , encourages and actively evaluates the active participant students	 participate They listen and answer questions
1- final press q ich (10 da q i q a)	1. Closing Summary q karîm 2. Musta q il gives work 3. Gives homework	 He listens Takes notes Takes notes

3. Assessment of students' theoretical knowledge:

A) Frontal method:

21. 1. Complaints of patients with arthritis
22. Examination of patients with arthritis, palpation
23. Laboratory and diagnostic examination
24. General symptoms and localization of joint diseases
25. Rheumatoid arthritis. Describe arthritis
26. Clinical manifestations of rheumatoid arthritis
27. Activity levels of rheumatoid arthritis
28. Course, consequences and treatment of rheumatoid arthritis
29. Describe deforming osteoarthritis
30. Etiopathogenesis of osteoarthritis
31. Diagnosis of osteoarthritis, additional examination methods
32. The course of osteoarthritis, treatment and prevention
33. Etiopathogenesis of systemic lupus erythematosus, clinic
34. Diagnostic criteria of SQYU
35. Clinical course of etiopathogenesis of systemic scleroderma
36. Diagnostic criteria for systemic scleroderma
37. The concept of allergies
38. Causes of urticaria, clinic, treatment
39. Causes of Quincke's tumor, clinic, 1st aid
40. Causes of anaphylactic shock, clinical diagnosis, edema care

The “beehive” method

In this method, the problem is analyzed with the whole group or with two small groups. The assigned task can be assigned to one or two different groups for the whole group. Within 10-15 minutes, group participants analyze the task solution and report to each other. The best option will be selected from them.

M iksedema

" Miksedema (hypothyroidism) shilimshi q " means the tumor , thyroid gland funk T Siyam said the disease caused by a lack of (122 photos) -123 . Women get sick more often than men. Hereditary

predisposition plays a role. Pathological and anatomical examination reveals atrophy of the glandular parenchyma, replacement of connective tissue, reduced organ size. Myxedema is characterized by shrinkage of the pituitary gland from other endocrine glands, insufficiency of the gonads, and enlargement of the anterior pituitary gland.

123 photos

Clinical picture of the disease (Fig . 123-124). Patients complain of inability to do physical work, poor intelligence and memory, voice becomes ugly, hoarse, speaks with difficulty, ears become heavy, eyes become dizzy, sighs heavily.

Examination of patients (125 photos) . Flesh tingling complains that the skin of the body has become different. Another characteristic sign is a mucous swelling, especially felt on the face and neck, and changes the overall appearance of the patient. The swollen face appears broad as it hardens, becomes yellowish, white, especially with thick wrinkles on the forehead, the eyelids become tense, and the eyelids become very narrow, the nose and lips thicken. The tumor descends into the neck and lumbar cavities, as if the head had penetrated the shoulder. The limbs are also enlarged (Pretibial myxedema). The tumor seen in myxedema is stiff compared to normal tumors and does not form pits when palpated.

124 photos

Tumors in myxedema are similar to tumors sometimes seen in nephrosis (in hypoproteinemia).

a 125 photo b

Facies myxoedematica Pretibial myxedema

Changes in the skin. The second important symptom of the disease is dystrophy of the skin, mucous membranes, the skin becomes dry, serous, peeling, sweat glands do not work, nails fall out, become dull, hair becomes dry and brittle, shedding Note that 126 photos of hair or eyelashes, eyebrows shed, sometimes overnight .

ENT organs. Voice and speech change (due to swelling of the mucous membrane that starts the larynx and vocal cords). The skin of the ear canal and the mucous membrane of the eustachian tube **126 photos**
the ear becomes heavier due to the swelling and thickening of the eardrum.

Metabolic disorders . A typical symptom of the disease is a sharp slowdown in metabolism. In any case, the basal metabolism is much lower than in other diseases (up to 50-60%). Weak decays of proteins, carbohydrates difficult to sing with the real one. In this regard, the body temperature decreases (up to 34 °). In patients with myxedema, acute

infections often pass with a normal or only insignificantly elevated temperature.

Cardiovascular system. In the internal organs, especially the symptoms of slowing and slowing of the cardiovascular system, there is a slowing of the pulse, a decrease in blood pressure, heart contraction and a decrease in tone (due to dystrophic changes in the myocardium and swelling of muscle fibers). contraction (on radiological examination) is accompanied by proliferation or even loss of teeth on the electrocardiogram. It should be noted that patients with hypothyroidism are more prone to atherosclerosis and obesity (so-called thyrogenic obesity) as a result of impaired cholesterol metabolism. The disease progresses slowly, lasting several years.

Digestive system. The mucous membranes thicken, so the tongue becomes large (so large that it does not even fit in the mouth, so patients open their mouths), Patients often suffer from constipation and flatulence due to intestinal atony. Gastric secretion decreases. A board is also reduced.

Laboratory changes. Anemia and lymphocytosis are exacerbated by neutropenia. The amount of iodine in the blood is greatly reduced.

Hyperthyroidism (Graves-Bazedov's disease)

Diffuse toxic goiter (Bazedov's disease-morbus Basedowi (thyrotoxicosis)) is a specific change in the neurohumoral system, a disease characterized by an increase in the amount of thyroid hormones in the blood . Hyperfunction of the thyroid gland increases the demand of tissues for oxygen, accelerates energy processes, affects the function of the nervous, vascular and other systems. Women are 5-10 times more likely to get sick than men.

Inquiry. Patients become irritable and restless, generally weak, sweat profusely, fever, sleep disturbances, and heart palpitations. The complete pronunciation of words is also distorted, sometimes words are spoken incompletely, and there are cases when one word is transferred to another without completion.

The picture of the disease is primarily a triad of known symptoms a) changes in the gland (in the strum); b) changes in the eye c) changes in the heart (tachycardia) and other organs.

K self-examination . Patients talk a lot and act a lot unnecessarily. The skin is moist and warm to the touch. Due to the high elasticity of the skin and the appearance of late wrinkles (wrinkles), patients look younger. The subcutaneous fat layer is poorly developed, the muscles atrophy due to increased weight loss. Some patients develop symptoms of myxedema as a result of autoimmune mechanisms. This is accompanied by a change in the skin of the front of the knee. The skin hardens and swells, the skin changes color. Enlargement of the thyroid gland is the main symptom of this. The enlargement of the gland can be diffuse and nodular and is mild on palpation, especially at the beginning of the disease, after which it usually becomes harder due to the appearance of connective tissue in the gland. There are 5 levels of enlargement of the thyroid gland. In grade I, the neck of the gland is

palpated when swallowed. In grade II, the gland is well palpated and looks good when swallowed. In grade III, the gland looks good (thick neck). A grade IV enlarged goiter deforms the neck. The V-level gland is very large in size.

Thyroid auscultation: A systolic murmur can be heard over the gland, which is amplified when pressed with a stethoscope and the patient bends his head.

It is also possible that only a portion of the thyroid gland is enlarged or that the struma is located retrosternally behind the chest, with only an X-ray examination yielding results. Eye blinking (Fig . 127) often occurs in both eyes, sometimes in one eye. At the same time, the human face seems to be frightened, sometimes completely terrified.

a 127 photo b

During the period when Bazedov's disease is not at its peak, it is typical for the patient to stare, especially with a twinkle in his eye.

Patients differ in the following eye symptoms according to the authors;

1. Grefe's symptom: when the eyelid moves down, the upper extremities are left behind, resulting in a scleral pathway over the pupil;

2. Mebius symptom - lack of convergence of the eyes: when any object (finger) is brought close to the patient's eyes, the eyes can not stand at a long point, and one or both eyelids involuntarily turn to one side ib goes;

3. Low and incomplete opening and closing of the eye, which is due to the fact that the upper eyelid is left behind, as well as the squinting of the eye;

4. Large opening of the eye (lagoftalm), accumulation of pigment in the eyelids-Elinek symptom;

5. Shtelvag symptom-limitation of eyelash movement (low eyelid),

6. Koxer's symptom - the formation of a white line between the upper lash line and the sclera of the eye when moving the eyeball upwards;

7. Marie's symptom — gentle tremor of the transmitted hands, exophthalmos-protrusion of the eyeball.

These eye symptoms are caused by increased function of the sympathetic nerves that innervate the corresponding eye muscles. It is believed that the twitching of the eye is due to the forced contraction of the orbital muscle of the eyeball, which can be filled with sympathetic nerves. is attached to the posterior pole, from which, when that muscle is forced to contract, the protrusion of the eyeball is observed; Grefe's symptom is due to the forced contraction of the muscle that lifts the upper eyelid.

An increase in thyroid hormones increases the permeability of the membrane of cells and mitochondria and disrupts the processes of oxidation and phosphorylation, leading to a deficiency of macroergic phosphates. To reduce such a deficiency, oxidation processes are intensified by increasing the activity of oxidizing enzymes. The increase in oxidation processes is due to the breakdown of fats and glycogen. These compensatory mechanisms cannot

overcome macroergic phosphate deficiencies. Protein is needed to keep the organs and azo functioning properly, and this process is disrupted due to the disruption of protein synthesis. In this regard, dystrophic changes occur in organs and tissues. These dystrophic changes lead to dysfunction of the heart, liver, kidneys and other organs.

-The vascular system of the heart (128 photos) . There will be tachycardia, sometimes as a symptom, the only sign of the disease. Patients feel their heart beating, the pulse is full and tense. Blood pressure i rises slightly, minimum blood pressure decreases. K apillyar thou pulse can be monitored. The rotation speed is accelerated, the blood becomes blood-volume mass-minute di multiply . In the early stages of the disease, the heart does not change, the pulse increases, the tones become much stronger. Later, symptoms of cardiac hypertrophy and dilatation appear, the rhythm is disturbed, and in the form of tremors and extrasystoles appear. Functional noises, as well as systolic and sometimes, diastolic noises, are heard in the mitral valve projection. Thyrotoxicosis is a disorder characterized by heart failure that results in symptoms of blood stasis. Thyrotoxic heart disease occurs due to severe impairment of cardiovascular function, ie symptoms of heart failure. In Bundy patients, breathing is shallow, accelerated, due to a decrease in muscle tone involved in this act of breathing . Patients develop shortness of breath, dry cough. This is due to stagnation in the small circulatory system, on the one hand, due to the constriction of the enlarged thyroid gland trachea and bronchi on the other hand (retrosternal goiter). In Bazedov's disease, the onset of cardiac symptoms is caused, on the one hand, by hyperexcitability of the autonomic nervous system and increased metabolism, on the other hand, by dystrophic changes in the myocardium (due to myocardial infarction and toxicity of excess glandular hormone). 'show mystery).

Changes in the nervous system . Excessive agitation, tremors, especially tremors of the arms extended forward, tremors of the legs, head, and some muscle groups are seen; patients become impatient, hurried, sometimes psychoses are observed. Pay reflexes are intensified, thyrotoxic neuralgia, neuritis develops.

Changes in the blood. Abundance of protein-bound iodine, a high level of absorption of **131** I in the thyroid gland , an increase in the amount of glandular hormones triiodothyronine (T3) and thyroxine (T4), an increase in basic metabolic parameters . Nonspecific changes in the blood include hypochromic anemia, leukopenia with relative lymphocytosis, increased ESR, urinary excretion of urea, creatinine, increased excretion of calcium in urine and feces.

128 photos

Proteins are broken down, sugar metabolism is disrupted (blood sugar levels increase, and after a meal, sugar is also formed in the urine), cholesterol

levels in the blood decrease, and dissimilation processes take precedence over assimilation processes. As a result, the person loses weight in a way typical of bazedov's disease. A person can lose weight too fast, but as the condition improves, the weight will increase more quickly.

An increase in thyroid hormones increases the permeability of cell and mitochondrial membranes and disrupts oxidation and phosphorylation processes, which in turn leads to heat production in the body and a deficiency of macroergic phosphates. Body temperature usually tends to rise to 37.5, and occasionally to 38 and above. Patients wear light clothing, which is difficult to keep warm and sweaty, and lie down at night. Sudden dermographism is observed when the skin becomes red, usually the skin is both thin and elastic, due to the intense heat separation.

If the function of the thyroid gland is checked against the iodine retention property of that gland, it is usually enhanced.

Increased gastric secretion **by the digestive organs,** increased appetite, abdominal pain, nausea, diarrhea and vomiting are characteristic, sometimes accompanied by constipation. Increased absorption of carbohydrates in the intestines gives the picture of diabetes. Due to the occurrence of fatty infiltration in the liver, it enlarges, toxic jaundice occurs due to impaired bilirubin conjugation function. This is one of the most severe manifestations of the disease . Sexual function is often impaired (menstruation is reduced); there is a good maturation of the breasts, a decrease in sexual desire, the course of the disease is varied and ranges from unknown mild forms (hyperthyroidism) to a severe process that takes place acutely. There is a risk of progressive changes in the heart and liver, which can lead to death. Osteoporosis is observed by the bones and muscles.

According to the course of the disease, mild, moderate and severe forms are distinguished. In the mild form, the pulse rate is up to 100 beats per minute, patients gain 3-5 kg, and the basal exchange rate increases by up to 30%. In the moderate form of the disease, the pulse rate is 100-120 times per minute, patients gain 6-10 kg, the basal exchange rate increases by 30-60% compared to the norm. In patients with severe disease, cachexia is observed, a lot of weight loss, severe tachycardia heart rate exceeds 120, the basal metabolic rate exceeds 60% of the norm. In this case, patients are accompanied by signs of atrial fibrillation, heart and adrenal insufficiency, liver damage.

Diabetes mellitus

Diabetes mellitus is a disease characterized by a metabolic disorder caused by a decrease in insulin production or its inactivation due to pancreatic b -cell deficiency or an increase in counterinsular hormones. Diabetes mellitus A disease caused by a decrease in insulin synthesis (absolute and relative) as a result of primary or secondary deficiency of b - cells in the islets of the pancreas and an increase in blood and urine glucose. According to the World Health Organization (WHO), the

disease is mainly caused by excessive food consumption, obesity, stress and inactivity.

Currently, the incidence of diabetes is increasing. The disease is more common in obese people and among the elderly, and it is not ruled out that it occurs among young people (children).

Disorders of carbohydrate metabolism. Insulin deficiency increases the processes of gluconeogenesis and glycogenolysis and decreases glycogen synthesis. Hyperglycemia and glucosuria occur as a result of impaired carbohydrate metabolism. This is due, on the one hand, to the slow passage of glucose into the muscles, and, on the other hand, to the slow passage of them into the adipose tissue. Slowing down their phosphorylation process slows down its breakdown, disrupts glycogen synthesis, slows down the conversion of carbohydrates into fats. Glycogen is broken down in the liver into glucose and passed into the bloodstream. Glucose is also formed from glycogenic amino acids.

In disorders of protein metabolism , the synthesis of glucose from glycogenic amino acids increases, the breakdown of proteins in the body increases, which leads to a violation of protein synthesis and the formation of trophic ulcers, making it difficult to heal wounds.

Disorders of fat metabolism. As a result of insulin deficiency, lipolysis processes in the body increase, leading to the accumulation of free fatty acids in the blood. Fats do not break down (burn) to the end, and the partially burned products of fat in the blood increase. Disruption of fat metabolism disrupts the formation of these high fatty acids and neutral fats from carbohydrates. This leads to weight loss in patients and fatty infiltration of the liver. This leads to ketosis, the acetone in the blood, B -oksimoy, atsetouksus leads to accumulation of acid in the urine can also be observed. Accumulation of ketone bodies in the blood leads to diabetic coma.

Disorders of water and electrolyte metabolism. Increased osmotic pressure in the renal tubules slows down the process of water reabsorption, leading to polyuria, which leads to disruption of water and electrolyte metabolism. Large amounts of sodium and potassium are excreted in the urine.

Insulin deficiency leads to disruption of the process of phosphorylation of vitamin B6, which leads to the development of neurological symptoms polyneuropathy.

Pathological and anatomical changes. We can see a decrease in b - cells in the pancreas , their degeneration, hyalinosis and fibrous changes. Morphological changes are observed in blood vessels, vascular angiopathy (retinopathy) in the retina, nerve cells (neuropathy), kidney (nodular glomerulosclerosis, tubular nephrosis).

Clinical landscape . The following clinical stages of diabetes are distinguished.

1. Pre-diabetes stage
2. Latent subclinical diabetes

3. Obvious (clinical) diabetes

It consists of three symptoms: polyuria - excessive urination, an increase in the amount and frequency of evening urine, ie nocturia, polydipsia (thirst), polyphagia - increased appetite. Hyperlipidemia (increase in the amount of unsaturated fatty acids in the blood, increase in the amount of lipids, cholesterol, phospholipids, triglycerides). The saturation of unsaturated fatty acids in the liver leads to their excessive oxidation, which in turn leads to the formation of acetyl-CoA, which in turn produces acetoux, b-oxymoic acid, acetone. Hyperketonemia is a severe disorder of lipid metabolism that reaches 300-400 mg / l in conditions of insulin deficiency. Patients complain of itchy skin, and women often complain of itching around the external genitalia. The general malaise is moderate. Decreased body mass, which results from malnutrition of the skin due to dehydration. In such patients, atherosclerotic processes develop more strongly than usual due to disorders of fat metabolism. Therefore, the course of diabetes can be aggravated by atherosclerotic symptoms in the form of damage to the coronary arteries (occurrence of myocardial infarction) and cerebral vessels (stroke). Decreased body mass is mainly due to water loss, catabolism of proteins, carbohydrates and fats. Due to the expansion of the network of peripheral blood vessels, the skin becomes pink, most of the skin develops scabs (due to itching in the itch), which are caused by malnutrition of the skin due to dehydration. In such patients, atherosclerotic processes develop more strongly than usual due to disorders of fat metabolism. Therefore, the course of diabetes can be aggravated by symptoms of atherosclerosis in the form of damage to the coronary arteries (myocardial infarction is likely to occur) and cerebral vessels (stroke). Vascular injury is very dangerous and is the leading cause of death and disability. There may be vascular injury, specific microangiopathies, and nonspecific macroangiopathies. In severe vascular injury, both conditions may coexist. As a result of damage to blood vessels, capillaries, there are changes in all internal organs.

Respiratory system . Diseases such as bronchitis, pneumonia, tuberculosis often develop due to the weakening of the body's immune system by the respiratory system.

Vascular system: atherosclerotic processes develop in the blood vessels, diseases such as cardiosclerosis, angina, myocardial infarction, capillary angiopathy, leg gangrene develop.

Increased appetite by the **digestive system** (bulemia), changes in the oral cavity, decreased gastric secretion, hypo and achlorhydria, fatty dystrophy in the liver, fatty infiltration, steatohepatitis, and cirrhosis. Inflammation of the gallbladder, the appearance of stones, changes in the secretory, motor function of the pancreas occur. Esophageal and intestinal dysfunction is observed.

Urinary system . Almost 30-35% of patients develop renal vascular atherosclerosis and intracapillary glomerulosclerosis Kimmelstil-Wilson. Renal capillaries are disturbed, renal ischemia results in increased

renin-angiotensin activity, increased blood pressure, urinalysis reveals proteinuria, cylindruria, microhematuria, isostenuria. When examining this category of patients, we may see swelling on the face, eyelids, legs, anemia, and an increase in ECG. Renal vascular glomerulosclerosis is exacerbated, patients with renal failure begin to die. Glucosuria is also observed at this time. There is leukocyturia, bacteriuria in the urine. Due to changes in the capillaries, the following symptoms develop: retinopathy, hypertension, albuminuria. Glomerulonephritis, pyelonephritis develops.

Visual organs: In occult diabetes, vascular injury often occurs before the diabetes clinic. Angiopathy of the fundus of the fundus of the eye is an example of a decrease in vision, blurred vision. Retinopathy, retinal blood vessels mikroanevrizmi, venulalarni the retina, the expansion of hemorrhage, 129 photos see and exudate was collected and hemorrhagic, anamaliyasi pigment yellow dog 'Bo' ladi. There will be changes such as scarring, retinal detachment, vascular proliferation in the vitreous body, hemorrhagic glaucoma. It often develops in cataracts.

Polyneuritis, polyneuropathy develops by the **nervous system** . Impairment of superficial and deep sensations, weakening of reflexes, impaired movement are observed, and as a result, pain occurs. Stroke develops due to cerebral vascular atherosclerosis . The nervous system damages the central nervous system, neurosis-like conditions, encephalopathies, myelopathy, as well as the peripheral nervous system, the innervation of the brain, spinal cord, autonomic neuropathy, cardiovascular, digestive system, urinary tract. Polyneuropathy of the leg nerves is observed. Clinical symptoms depend on the nature of the injury to the nervous system, paresis, paresthesia,
sensory disturbances, ataxic, vasomotor disorders, muscle relaxation, painful **129** Fig
syndromes, memory loss, etc. Vegetative disorders are manifested by changes such as impaired sweating, impaired intestinal motility, decreased sexual activity in men.

Vascular injury is mainly due to the **deposition** of **hyaline** in the inner intimate layer of the capillaries, which disrupts the permeability of the capillaries and results in vascular-related changes in all organs, leading to vascular-specific damage and vascular microangiopathy. For example, changes in the renal capillaries, nephropathy or angiopathy in many organs, including the fundus, ie when examined, the capillaries dilate, retinal capillary angiopathy, etc. Atheromatous changes in the veins enters.

Capillary angiopathy develops by capillaries. Sometimes there is damage to the arteries of the legs, atrophy of the skin, decreased pulsation in the arteries dorzalis pedis, pain in the legs, paresthesia, lameness in movement (lameness), followed by changes in the capillaries, the metabolism is completely disrupted to gangrene (diabetic compensation) will bring.

Bone musculoskeletal system : During the decompensation period of diabetes, muscle atrophy occurs and osteoparosis is observed.

When examining patients, the skin becomes red , mainly the face, cheek areas, and forehead area. Due to the enlargement of the peripheral vascular network, the skin becomes pink, the cheeks become red, the forehead and underarms become red (rumyanets), the capillaries dilate (rubeoz), the palms of the hands and soles of the feet become yellow due to vitamin A disorders. most skin develops rashes (due to itching in the itch), fungal diseases, women develop itching in the area of the external genitalia, and skin tension disappears, and we may see dry, cold , purulent rashes. Difficult trophic lesions on the body and legs as well as gangrene on the legs can be seen (Fig . 130 a and b) . **Changes in the oral cavity** of patients lips and the language I start fading, coating flows pus, inflammation of the mucous membrane of the mouth, gums, gingivitis and stomatitis develops, paradontozga drag down the teeth.

a) 130 photos b)

Basic data for **laboratory** diagnosis of **diabetes mellitus** : an increase in blood glucose above 6 mmol / l, an increase in blood sugar - hyperglycemia, the appearance of sugar in the urine - glucosuria (not the norm) , Occurs at 88 mmol / l (160mg%). There is an increase in the density and volume of urine, in which ketone bodies appear. Diabetes is divided into 3 levels depending on the severity: mild, moderate, severe. Only when you consume excessive amounts of light carbohydrates does its amount in the blood rise and it appears in the urine. At the average level, the patient complains of the above-mentioned complaints specific to diabetes, it is noted that the amount of sugar in the blood is increased, sugar is constantly present in the urine. In cases of severe diabetes, patients lose the ability to work. They may also develop diabetic coma.

Criteria for the diagnosis of diabetes. In randomized trials, patients had a plasma glucose level of 200 mg /% + if the classic symptoms of diabetes were polydipsia, polyuria, polyphagia, and weight loss;

• If the amount of glucose in the blood plasma at breakfast is 140 mg /% as a result of two tests;

• Plasma glucose is less than 140 mg /% at breakfast + 2 times when oral glucose tolerance test is performed. Blood glucose should be 200 mg /% or more after 2 hours of blood taken for analysis and 75 mg of glucose taken 1 hour after the intermediate test. There is no need to test for glucose tolerance if the patient's serum glucose is 140 mg /% or more.

The amount of glucose, insulin, S-peptide in the blood is determined. The S-peptide determines the functional status of the pancreas. The amount of glycosylated hemoglobin and fructosamine (glycosylated albumin) in the blood can be determined. The amount of this corresponds to the amount of glucose. Skin, muscle, cheek, and kidney

biopsies are used to diagnose diabetic microangiopathy, and these biopsies are examined. In addition, ophthalmological and functional tests of the kidneys are used. In addition to these methods, glucose tolerance is tested, for which 200 g of glucose is given orally at lunch, and after 2 hours, blood glucose levels are checked (normal in healthy people, 6.7 mmol / l (120 mg%) in capillary blood, and plasma). 7.8 mmol / l (140mg%)), if it exceeds the norm by more than 10 mmol / l (180mg%), the sample is positive and diagnosed as diabetes. This test is performed 2 times.

Practical training 16

Blood system. Control methods. Methods of physical examination. Methods of laboratory and instrumental examination.

Laboratory work: blood test. Clinical significance of general blood analysis. Symptomatology of anemia (iron deficiency, posthemorrhagic, hemolytic).

1. Practical training module .

Training time - 4 hours	The number of students 1 : 0 to 12
Form of training	Practical training
Practical training plan	Laboratory work: blood test. Clinical significance of general blood analysis. Symptomatology of anemia (iron deficiency, posthemorrhagic, hemolytic).
The purpose of practical training :	Lab: To gain an understanding of blood testing . Symptomatology of anemia (iron deficiency, posthemorrhagic, hemolytic).
Teaching style	Inquiry. Demonstration of patients, interactive teaching methods, practical skills.
Form of teaching	In small subgroups.
O ' unit equipment	Calls to O Training Guidebook , practical Typo content , projectors , computer .
Training mode	Methodically equipped auditorium.
Monitoring and evaluation	Oral control: questions and answers, tests, problem solving.

1.2 . ***Technological card of practical training***

Stages and timing of work.	Educator	Learners
Preparatory stage	31. See the audience to control the purity karîm 32. Checks students ' readiness for training 33. And control q karîm	
1. bullet and training g contain levels of intra q	1. Preparation of educational content on the topic. 2. Preparation of presentation slides for the introductory	

(10 da q i q a)	speech 3. Develop a list of references used in the study of science	
2 - the main stage (160 minutes)	1. Divide students into small groups and ask questions on the topic. 2. Uses display posters 3. Uses slides, multimedia 4. Conducts treatment 5. Summarizes and summarizes the information provided on the basis of topics, encourages and actively evaluates the active participant students	They are divided into small groups They watch They participate They listen and answer questions
3 See the final stages of diarrhea (10 da qiq a)	1. Concludes 2. Provides independent work 3. Gives homework	He listens Takes notes Takes notes

3. Assessment of students' theoretical knowledge:

A) Frontal method:

1. What does a general analysis of blood mean
2. Diagnostic value of general analysis of blood
3. Blood collection technique
4. Hemoglobin detection technique
5. Q form on elements y h REPORT
6. Determination of erythrocyte sedimentation rate
7. What is the leukocyte formula
8. Indications of normal blood flow
9. Puncture of blood-forming organs
10. Evaluation of hemolysis
11. The concept of anemia
12. Manifestations of anemia
13. Posthemorrhagic anemia, clinical manifestations
14. Describe the iron deficiency anemia
15. Causes of iron deficiency anemia
16. Clinical manifestations of iron deficiency anemia
17. Changes in blood analysis in iron deficiency anemia
18. Etiopathogenesis of V12 (folic acid) deficiency anemia
19. A general review of patients with V12 (folic acid) deficiency anemia

Laboratory diagnosis of blood in anemia

The "three-step interview" method

Three students will be selected in each group, and the roles of "doctor", "patient" and "expert-UASh" will be divided between them. The student who chooses the role of patient is told an anonymous diagnosis, and he or she makes complaints about that diagnosis, the doctor makes a diagnosis, and the expert checks the UASh complaints and the proportion of the diagnosis. Each group is consulted for 10-15 minutes, the expert examines the activity of the doctor on 3 points:

1. What was done right

2. What went wrong
3. How to do it

The conclusion of the group consultation is compared with the conclusion of the expert.

Another type: students are analyzed by the whole group, participating in the clinic in the role of an expert, in a real consultation.

Subject statement

Diseases of the hematopoietic system

Scheme of examination of patients with diseases of the hematopoietic system

1. Methods of subjective examination

a. Primary and additional complaints are collected.

b. Medical history is collected.

v. Life history is collected.

2. **Methods of objective inspection**

a. Review.

b. Palpation.

v. Percussion.

g. Auscultation

3. **Methods of laboratory testing**

a. General blood test.

b. The amount of erythrocytes in the blood is determined.

v. The amount of hemoglobin in the blood is determined.

g. The amount of leukocytes in the blood is determined by leukoformula .

d. ECG is detected in the blood .

e. Blood clotting time is determined.

j. S urinary analysis

4. Methods of morphological examination.

a. Bone marrow point.

b. Liver and spleen point.

5. Instrumental inspection methods.

a. X-ray examination.

b. Radioisotope testing method

v. Ultrasound examination.

g. Computed tomography

Inquiry of patients with diseases of the circulatory system

Complaints of patients with diseases of the hematopoietic system are diverse and general in nature. These are: weakness, rapid fatigue, drowsiness, headache, dizziness, shortness of breath during physical activity, frequent heartbeat, darkening of the eyes, sometimes fainting, and others. Such complaints are often observed in anemia (anemia) of various

causes, leukemia, myeloid hypoplasia. In addition, pain sensations on the tongue, irritation of the tip and edges of the tongue may be the first sign of anemia. Characteristic of necrotic angina, stomatitis, gum bleeding - leukemia and agranulocytosis.

In acute leukemia, patients experience pain in the throat when swallowing.

In many blood diseases, patients become very thin as a result of lack of appetite, and even go into a state of cachexia. This condition is especially noticeable in chronic leukemia, dangerous lymphomas. In anemia (chlorosis) caused by iron deficiency, complete cognition is often impaired. Patients tend to eat things that a healthy person does not consume, such as coal, mud, chalk, lime, etc. they like the smell of things like that.

Medical history. When patients are interviewed, they are asked about their pre-disease condition (weakness, fever, various hemorrhages) and whether the patient thinks there is a factor or condition that caused the disease (radiation, bleeding, menstrual cycle status, worm disease). , diet, pregnancy and its number, gestational interval, inflammatory diseases of the gastrointestinal tract, etc.), the time of onset of each symptom of the disease, the nature of the disappearance or exacerbation of these symptoms should be asked . Before the onset of the disease , it is important to determine if the patient's blood has been tested, if so, what changes have taken place, what medications (affecting the blood) the patient has taken due to other illnesses, and when. If a patient has been treated for a blood disorder, information about what medications they are taking and their effectiveness are also important in determining the diagnosis.

Life history of the patient. There are a number of things to consider when questioning the life history of a patient with circulatory system headaches. First of all, the patient is asked about his lifestyle, childhood and current living conditions, diet, quality, the impact of harmful factors (phenylhydrazine) in the workplace. In addition, the health of the parents, relatives, that is, whether they have blood diseases, the diseases that the patient has experienced throughout his life are inquired. Some of the diseases slowly or as a result of a large amount of bleeding, gastric mucosal atrophy, and a part of the resection is made of iron, vitamin B **12** absorption in violation may occur due to anemia. Chronic diseases of the liver and kidneys , systemic diseases can also cause anemia. Some drugs used in the treatment of various chronic diseases reduce the activity of bone marrow. Similarly, working with radiation or staying in radiation zones for long periods of time also impairs bone marrow activity.

Methods of objective examination of patients with diseases of the circulatory system

General examination of patients. The general condition of patients with diseases of the circulatory system can be mild, moderate and severe, depending on the type and duration of the disease. Patients are in a critical

condition in the later stages of anemia, in rapidly developing types of myeloid aplasia and leukemia. In such cases, their minds will also be distracted.

As a result of a decrease in erythrocytes and hemoglobin in the blood, the skin and mucous membranes become pale. While paleness develops slowly in chronic anemia, it appears suddenly in acute anemia, and this is often the main sign of bleeding. Depending on the type of anemia, the paleness also varies. For example, vitamin B **12** yetishmovchiligidan Addison-one patients with anemia caused by skin fair -sarg'ish, the skin becomes yellow, white curtain yellow eyes. In anemia caused by iron deficiency, especially in juvenile chlorosis, the skin turns a greenish color, sometimes reminiscent of the color of "alabaster". In hemolytic anemias, the skin is yellow in color. It is important to see the color of the eyes and gums at times when it is difficult to determine the color of the skin. In chronic leukemia, the skin color is gray.

Examination of the oral cavity is also of diagnostic importance. In anemia caused by vitamin B **12** deficiency, the nipples of the patient's tongue are flattened, shiny, dark red in color.

See the lymph nodes and spleen acoustical . In diseases of the circulatory system, lymph nodes, liver, spleen and bones are examined by palpation. Peripheral lymph nodes are palpated with both hands, first in the neck, then in the groin, armpits, and groin area. Their elasticity, mobility, surface smoothness or unevenness are determined. In lymphocytic leukemia, lymphogranulomatosis, and lymphosarcoma, the lymph nodes become particularly enlarged. Usually the lymph nodes in one part of the patient's body first and gradually the other group of nodes also enlarge. This enlarges not only the superficial lymph nodes, but also the deep ones.

In leukemia, lymphoma, they are hard, painless, not attached to the surrounding tissue, and never purulent. In lymphocytic leukemia, they have a dough-like elastic consistency. Only in lymphogranulomatosis, especially in lymphosarcoma, the lymph nodes become enlarged and stick together, forming large conglomerates. In many blood diseases, the spleen becomes enlarged. In Verl-Goff disease, Addison-Birmer anemia, lymphogranulomatosis, the spleen is slightly enlarged. Only in hemolytic anemia and chronic myeloid leukemia does the spleen have a very large, hard consistency, sometimes occupying the left side of the entire abdominal cavity and reaching the right as well as the small pelvis. Its edges are rounded and the surface is smooth.

To palpate the spleen, the patient lies on his back or on his right side. The examining physician sits on the patient's right side and presses the patient's chest slightly with his left hand. This limits breathing movements. Place the fingers of the right hand together and slightly bent under the arch of the left rib. If the spleen is much enlarged, he places his hand just below the lower edge found by his percussion. As the patient exhales slowly, insert his right hand inward, under the rib cage, and assign

deep breathing to the patient. If the spleen is enlarged, it will move down and hit the hand and slide over it. During palpation, attention is paid to the hardness or softness of the spleen, its pain, mobility, and whether there is a dent in the upper edge. An enlarged spleen often does not hurt. Only in splenic vein thrombosis, in perisplenit, the patient may experience severe pain under the left rib arch. In addition to blood diseases, the spleen is enlarged in many infectious diseases, cirrhosis of the liver. In the detection of diseases of the circulatory system, palpation of the flat bones as well as the epiphysis of the tubular bones, as a result of severe hyperplasia of the bone in these diseases, they become painful.

Recirculation. The spleen **is** located below the left rib, outside the upper part of the stomach, under the diaphragm, and moves in the act of breathing. Its projection is located on the left lateral surface of the thorax between the ribs IX-XI (in some literature IX-X). In diseases of the circulatory system, the percussion method is used to determine the size of the spleen. To do this, the patient is in a lying or standing position and is percussed very slowly because the spleen is surrounded by air-retaining organs and is itself much thinner. Percussion is performed with a very slow percussion stroke, from a clear percussion sound to a muffled sound.

To determine the longitudinal dimension of the spleen, percussion is performed on both sides over the X rib and it is 6-8 cm in a healthy person, the line to find the transverse dimension is 4 cm from the line of the costoarticularis sinistra percussion along the lateral line and it is 4-6 cm . The transverse dimension of the spleen corresponds to the IX-X rib range on the left.

Determining the size of the percussion on the kurlov . The upper and lower border of the first spleen is determined, then its posterior and anterior margin is determined. The percussion procedure should be in which the patient is lying on his right side or standing. The plexiglass finger is placed parallel to the test area. When percussion is conducted from top to bottom, the pleural sound is percussed from the tympanic sound in the Travbe area to the hoarse sound, and the plexiglass is determined by a clear sound (by the tympanic sound) from the outside of the finger. Percussion tattoos should be slow and slow. When the spleen size is normal, a more muffled percussion sound is produced because the gastric balloon and Traube area, as well as the small and small bowel gases, are located nearby. To determine the upper border of the spleen, the plethysmometer is placed in the transverse direction of the finger, the left lateral surface of the thorax at the level of the V rib. Percussion is performed to the left lateral bone wing until a hoarse percussion sound is heard, keeping the finger in a transverse position along the ribs or intercostal space. The place where the muffled percussion sound is heard is the upper border of the spleen, normally lying on the IX rib. The dermographer or left hand marks this point with the fifth finger, the plethysmometer finger is placed along the left iliac crest, and percussion is performed in the opposite direction along the mid-axillary line (from bottom

to top). . The transition of the tympanic sound to a muffled sound is the lower limit of the spleen, and this norm corresponds to the XI rib. To find the width of the spleen, the upper and lower boundary range is measured, which is normally 4-7 cm.

To determine the posterior edge of the spleen , it is necessary to palpate the left X rib and find the point of attachment to the spine. The finger is then placed on the left frontal line of the plexiglass, with the middle phalanx (middle bone) of the finger perpendicular to the X rib. The finger is percussed from the tympanic sound to a hoarse sound toward the spleen along the X rib, keeping the plethysmometer in this position. This is the posterior edge of the spleen and is marked with a dermograph. Normally, the posterior edge of the spleen does not extend beyond the left shoulder blade.

To determine the anterior **margin** of the spleen, the plethysmometer finger should be placed along the anterior line of the abdomen so that the middle phalanx of the finger is perpendicular to the umbilical line. Percussion is performed towards the spleen, from the point of intersection of the umbilicus and the left X rib to the left axillary line. The transition of the tympanic sound to a louder sound is the boundary of the anterior edge of the spleen. Normally this does not extend beyond the front underarm line. Then the distance between the anterior and posterior borders of the spleen is measured, and this is 6-8 cm. If the spleen is too large, the anterior border extends beyond the rib arch. In this case, the protruding part is measured. The limit of spleen determined by Kurlov is included in the history of the disease along the decimal line.

6 x 17/9 cm

This is where all the numbers (6) spleen fried part of a spread .

Photos (17) the length of the image ' g ' climate . Numerator (9) the width of the image ' g ' climate .

Divorce in size and can be detected by a simple method . If perkussuya condition of the patient at the time o ' fried ng side Bo is set to ' X ' Calls to clear the left secondary ltiq underground place of intersection with the line ' I like the sound of a liver kattaligicha Bo ' g ' Climate is defined , then the spleen is enlarged (ragon symptom) .

Splenic kattalashuvi is one of a number of important diagnostic signs of the disease . Liver , spleen and lymph nodes at once kattalashuvi some o ' tkir and chronic infections , sepsis , infectious end ocarditis , haemoblastosis and systematic immunopathological diseases . Liv er and spleen kattalashuvi chronic active hepatitis , liver cirrhosis , hemolyti c anemia , and metabolic disorders (chairperson of patients ' rights) (Goshen , something to be done) . Splenic kattalashuvi gate and the splenic vein thrombosis , spleen o ' sma injuries , cyst and other local develo pment . Enlarged spleen is bleeding chronic mieloleykozda , osteomielofibro

zda observed , and sometimes abdominal franchise occupies a large part of the placer may be even smaller chanoqqacha .

Laboratory tests in the blood. Determination of hemoglobin

Preparing patients for laboratory examination. Blood test **.** Blood counts vary throughout the day, so blood should be checked in the morning whenever possible, except in emergencies. All tests should also be obtained prior to radiography, ultrasound, fibrogastroduodenoscopic examinations, and physiotherapeutic treatment. This leads to a change in the analysis. Smoking is also prohibited for 1 hour before the test. It is important not to change your daily routine and diet during the day from physical exertion, alcohol. The analysis should be submitted at lunch, 12 hours after the last meal, water can be drunk. The results of the analysis are affected by the drug, the drug should not be taken during the day. It is preferable to take blood while lying down, dizziness may occur while standing. It is not possible to carry a load in a vein for 1 hour (carrying a bag). It is preferable to use a single reagent, taken in the same laboratory at the same time of day, to monitor laboratory readings. Because the results may be different if different reagents are used. It is necessary to use a vacuum system to draw blood from a vein. To avoid hematoma, the puncture site is tied with a sterile cloth for 15 minutes after the blood is drawn. For general analysis, blood is taken from the 4th finger at least 1 hour after the last feeding. Can be taken from the foot (compensation) in infants.

Blood is taken 12 hours after the last meal at lunch for **biochemical testing** . To check the lipidogram (cholesterol and its fractions) should be at least 14 hours after the last light meal. To test for glucose tolerance, it is recommended to follow a mixed diet for 3 days (the diet should contain 250 g of carbohydrates per day). It is strictly forbidden to take drugs and hormones that affect carbohydrate metabolism (except for diabetics, if life-threatening). The test lasts from 1 to 4 hours. This time the patient should be in the clinic. If hormones are to be checked in women of reproductive age, they should be taken blood on the appropriate day of the menstrual cycle. To test for blood clotting, patients should not have taken blood clotting drugs such as warfarin, heparin, fraxiparin, clexan, plavix, aspirin, curantil, thromboass, and blood clotting drugs such as aminocaproic acid, vitamin K (vicasol), dicinon.

Counting blood cells

Blood count is mainly counted in a special chamber. The counting of blood cells consists of a series of manipulations: 1) injection into the finger of the person being examined; 2) draw blood into the mixer; 3) dilute the obtained blood with an appropriate solution; 4) dripping a drop of diluted blood into the net of the counting chamber. The finger is usually injected with a separate tool -Frank needle (scarifier), the advantage of this needle over ordinary needles is that its tip penetrates the finger tissue to the desired depth, depending on the thickness of the skin; the injection can be done in other ways as well. First, wipe the needle with the skin of the finger

thoroughly with alcohol (for disinfection) and ether (for drying). The first drop of blood coming out of the injection site is wiped off and the next drop is taken. Do not pierce the needle too much (2-3 mm) to avoid repeating the injection. The finger should be warm.

Blood is drawn with the help of separate pipettes called mixers (melangers). The agitator consists of a capillary tube divided into levels, the tip of which widens and forms an ampoule; from the ampoule to the front of the capillary tube as before, and then slightly further with a slightly wider hole. Various mixers are used to count the red and white blood cells. Mixers designed to count erythrocytes have a slightly thinner caliber capillary and a much wider ampoule; it is marked: one mark 5 degrees after the sharp end (mouth) of the mixer - 0.5, the second 5 degrees before the entrance to the ampoule - 1.0 third, at the exit from the ampoule-101. Up to 0.5 or 1.0 characters of blood is taken, up to 101 characters of dilute solution is taken. The volume of liquid in the mixer up to 101 characters is 100 times larger than the volume up to 1.0 characters and 200 times larger than the volume up to 0.5 characters.

The hole of the mixing capillary intended for leukocytes is somewhat wider, and the ampoule is smaller; it is marked 0.5 or 1.0; the dilute liquid is absorbed up to 11 characters. This means that the volume of liquid in the mixer up to 11 characters is 10 times the volume up to 1.0 characters and 20 times the volume up to 0.5 characters. Both ampoules have a glass balloon to mix the blood with the diluent. At the blunt (wide) end of the mixer is placed a rubber tube with a small glass tip, which serves to suck the tube. The resulting blood is diluted with the following solutions.

1. To dilute red blood cells is usually used a 3% solution of common salt, recently Gayem prefers to use liquids (5 g. Sulema, 10 g. Sodium chloride, 37.5 g. Sodium sulfate is dissolved in water; and the volume of the liquid. Up to 1 l). In gamma fluid, erythrocytes are stored longer. It is also recommended to add dye to the diluent (e.g. methylviolet or gentianviolet): the dye stains several leukocytes, allowing the leukocytes not to be added to the erythrocytes. Typically, blood is drawn up to the 0.5 mark by sucking a little through a rubber tube attached to a stirrer to count red blood cells. The tip of the mixer is then lowered into a cup filled with one of the diluting liquids shown above, and the liquid is absorbed by filling the entire ampoule to the 101 mark (diluting 200 times); then the mixer is shaken rapidly with force in all directions for three minutes. If the blood of an anemic patient is being tested, it is preferable to suck up the blood to the 1.0 mark (dilute 100 times). **A** 5% solution of **acetic acid** is used **to dilute white blood cells** . Acetic acid dissolves red blood cells. However, at such a concentration it does not damage the white blood cells, which allows the white blood cells to count themselves. For a clearer view of the white blood cells, a little of a 1% solution of gentian violet in water is added to the diluent, and the gentian violet stains the nucleus of the leukocytes. To count white blood cells, blood is drawn from the hand up to the 1.0 mark, then a

dilute solution is drawn up to the 11 mark; thus the blood is diluted 10 times; If the amount of leukocytes is large, the blood is taken up to 0.5 mark. (Diluted 20 times). The mixer is shaken as described above. Blood drawing into the mixer requires great precision (it should be of the appropriate mark without air bubbles, etc.), the mixing liquid should be in a retained position (the tip should be in a horizontal position without touching the fabric or paper, a piece of liquid from the fabric or paper mixer can attract). The mixers should be extremely clean and dry. A drop of diluted blood is added to the net of the counting chamber only after the mixing liquid has been thoroughly mixed. The first few drops (liquid in the capillary part) should be drained from the mixer and only the liquid in the ampoule (only well-mixed liquid) should be used. The drop is dropped directly or directly (in a Tom-Tseiss-type camera), (the drop must be of the appropriate size in this case, only experience helps to determine the size of the drop) or not the drop is lowered into a special groove (ditch) next to the net, where the net fills itself (Burker-type camera). After the first type is dropped, a cover bottle is placed over it. In the second type, the cover is placed before the bottle, in which the drop taken from the mixer is brought to its edge, and the drop fills the net with a capillary property. The counting chamber consists of a thick piece of glass, on which a separate glass plate with a net is mounted or clamped, and a 0.1 mm thick glass plate is placed around (or on both sides) of the net. year The cover glass, which is placed on the mesh, is fastened to the same plate. The height of the gap between the net and the cover window, i.e. the depth of the net, is thus equal to 0.1 mm. It is necessary to use polished glass, the surface of which is finally flat (normal glass is often uneven, which distorts the required height of the camera). The cover windows should be tightly glued to the camera until Newtonian rings (arc rotates around the net) are visible. Tom-Zeiss and Burker-type account cameras are distinguished.

2. The Tom-Zeiss camera is shown. The mesh glued plate is round; around it is placed a second, slightly thicker plate, leaving a narrow circulating ion, the upper surface of which rises above the surface of the inner round plate, respectively. The height between the edges of the mesh plate and the slightly thicker plate that surrounds this plate is 0.1mm. The drop is dropped onto a mesh plate in the center. The glass is glued on top until a Newtonian ring appears. The size of the drop should be such that the net can cover a flat surface from head to toe; a little excess liquid partially falls into the tube.

3. The last, more perfect type of burker chamber consists of a thick piece of glass, in the center of which are placed two nets perpendicular to the glass, the bars of which are side-by-side and in the middle of each other. wrapped with; the outer edges of the side beams are raised, so that the height between the mesh surface and the top edges of the side beams rising above the Goryaevs is 0.1 mm (depth of the chamber). The cover glass is placed before the drop is dropped; a drop of the mixer is dropped onto the bar of the

outer beams protruding from under the cover glass . Due to the capillary, this drop fills the network. Both of the nets can be used in the same way, in which either the nets are filled with the same blood or with two different blood (each drop is dropped next to the first and second nets). The nets come in different looks. But their structure is the same as prin t sipi. All nets consist of ‘small’ and ‘large’ squares grouped differently. The sides of the small square are 10 mm, i.e. its face is 400 mm ² . Since the height of the camera is -5 mm, the small square size, so. A large square can consist of either 16 small squares, or 16 small squares in terms of surface area. The roof grid consists of 16 large squares, each consisting of 16 smaller squares; the large squares are separated by ‘three-story lines’, i.e. small squares divided by another line. Thus, the whole grid consists of 16 and 4 more small squares in each row, i.e., 20 rows (less than 20 squares in each row), for a total of 400 small squares. The tom net is enough to count the erythrocytes, but the smaller one to count the leukocytes accordingly has to fill the net several times to count the leukocytes and count and get the average numbers. The center of the Turk net is the Tom net, but the surface is much larger. For example, it has 8 more such surfaces. These additional surfaces are only divided into large squares. Grouchy old aunt h b o ' lib, 9 x 16, h er q number more than 12, a total of 144 square meters. Predtechensky net is very convenient This net has 100 large squares, some of which are divided into small squares, and even when divided, they are evenly divided in different sections of the net. The Goryaev net is a more advanced version of the Predtechensky net, the larger of which (15 in each of the 15 rows, for a total of 225 large squares) is currently used in our Burker-type cameras.

4. The Burker net is similar to the Turk net, which also consists of 144 large squares, when counting blood cells, a single evenly distributed small squares are used in the net cavity, which are the lines that separate the large squares. formed by cutting. Goryaev's net is the best of all. The shaped elements in the net are counted 2-3 minutes after the chamber is filled with the droplet (to allow the blood elements to settle). All cells lying in a square are also counted as cells standing in the boundary lines if more than half of them are within that square. Cells that cross half of the boundary lines are also taken into account. However, they are only counted as lying on the lines either at the top and left or at the bottom and right (i.e., cells in two of the 4 lines that limit the square). This is done so that single cells do not enumerate themselves twice. Cells that lie outside more than half of this square are not included in that square at all. Red blood cells are counted from small squares. In Goryaev’s nets, large squares of 16 small squares are selected, and usually 5 large squares (i.e., 80 small squares) are counted. It is best to select squares that lie in different parts of the net (to reduce the uncertainty caused by the uneven distribution of erythrocytes in the net. squares are used. There are 10 small squares in a row along the entire grid. Usually there

are 6 small squares in 6 rows and again two small squares in the seventh row. This also turns out to be a total of 80 squares.

This is done as follows: After counting the erythrocytes in 80 small squares equal to 16 large squares, the average number of erythrocytes in the small square is first calculated. Since the size of a small square is 1 mm ³ as mentioned above , in a mixture of 1 mm ³ **where** erythrocytes are counted, there will be 4,000 times more erythrocytes than one small square. Since the blood obtained is diluted in a mixer (usually 200 times), the resulting number must be multiplied by another 200. For example. Suppose we counted 400 red blood cells in all 80 small squares; so in one small square they are 400: 80 = 5; in a 1 mm ³ mixture obtained from the melange 5X 4000 = 20 000, 1 mm ³ **in the** blood. while there are 20,000 X 200 = 4,000,000 red blood cells. In practice, the number of erythrocytes listed in 80 small squares (or 5 large squares) is set to 4 zeros (i.e., that number is multiplied by 10,000). Leukocytes are counted in large squares. In the roof chamber are counted leukocytes lying on the entire surface of the net (i.e., 400 small squares); at least 3-4 drops are listed. There are 100 large squares in the burker grid (96 are taken from 8 rows and 4 from any row), the rows are not taken one after the other. It is best to enumerate the leukocytes lying within the net in the Burker net. The mesh size is 0.9 mm ³ . Therefore, to obtain the number of leukocytes in the blood of 1 mm ³ , it is sufficient to multiply the number of leukocytes counted from the whole network by 10/9 and the dilution number (10 or 20). Goryaev's network also lists more than 100 squares, usually "empty" (not divided into small squares) squares are selected - they consist of 4 rows. There are a total of 25 such squares in the grid. The number of leukocytes counted from 100 large squares is divided by 100, then by 16 (reduced to one small square). The average number of leukocytes per net in a small square is multiplied by 10 or 20 (depending on whether it is diluted 1:10 or 1:20) and 4000.

Determination of hemoglobin in the blood

To determine hemoglobin (Hb), blood is drawn from the tip of the fourth finger of the hand into a special pipette with a capacity of 20 mm ³ **up to the** mark of the pipette . The blood obtained is diluted in a special solution and the amount of hemoglobin is determined on a MiniGEM or on a hemometer.

Hemoglobinometer MiniGEM. MiniGEM is a new generation of hemoglobinometers that has a wide range of capabilities and detects hemoglobin at 540 nm operating wavelengths in all ways. The main method is the hemoglobin cyanide and hemichrome method (Fig . 134).

Hemoglobinometer MiniGEM 523 nm wavelength hemoglobin detection device is also available, which uses a 0.04% ammonia solution as a reagent (modified Derviz-Vorobyov method).

Determination of hemoglobin concentration. Blood sampling from the patient is traditional, with 20 µl of capillary venous blood being

drawn. Sample preparation time is 5-20 minutes and sample volume is 4-5 ml. Once the blood solution is prepared, it is placed in a cuvette and the hemoglobinometer is placed in the photometric cell of the MiniGEM and the concentration number is displayed on the MiniGEM display within 1 second. Automatically calculates the optical density of the solution to the hemoglobin concentration.

Hemoglobinometer The convenience of the MiniGEM is that it does not need to be heated and calibrated. Once the cuvette is dropped into the MiniGEM cell, 134 images are automatically taken
the apparatus lights up and starts measuring, and after the cuvette is removed from the cell, the hemoglobinometer goes into standby mode until the next test. A hemoglobinometer can detect hemoglobin every 2 seconds. Hemoglobinometer MiniGEM capability. At the optical density, the measuring range of the apparatus is 0 to 0.9 B, which corresponds to a hemoglobin concentration of 0 to 360 g / l.

Determination of hemoglobin in the blood by the method of hemiglobincyanide

Procedure for analysis . With red blood salt (celezosinerodist potassium) hemoglobin is oxidized to methemoglobin (hemiglobin). The resulting cyanmethemoglobin (hemiglobincyanide) stained with acetocyanhydrin is determined by calorimetric method.

Required reagents; 1. Transforming atetontsyanhydrin-0.5 mg ;. Red blood salt-0.2 g; Sodium bicarbonate-1 g; Distilled water-1 l; The solution is bright yellow.

2. A standard solution of hemiglobincyanide will be needed. 100 ml of solution-transformer solution required for 100 assays and 1 ampoule of hemiglobincyanide standard solution.

Procedure for analysis; Add 20 μl of blood to 5 ml of the transforming solution in the solution, mix well, stop for 10 minutes and test in a cuvette at MiniGEM.

This solution can also be detected by placing it on a 543 spectrophotometer in a 1 cm thick cuvette and placing it in front of the control test.

To create a calibration graph, a dilute solution is prepared from the standard solution as shown in the table;

t / r	Standard solution , ml	Transforming standard solution, ml	Hemoglobin concentration, g / l
1	–	6	–
2	2	4	50

3	4	2	100
4	6	–	150

The amount of hemoglobin is determined on the basis of a standard solution of standard hemiglobincyanide or on the basis of the following formula;

From here ; E_{op} . - experimental test extinktsiyasi ;

E_{st} . - standard solution extinction;

S is the concentration of hemiglobin cyanide in the standard solution mg /%;

K is the dilution coefficient of blood;

0.001 - recalculation coefficient mg / 100 ml g / 100ml;

10 - recalculation coefficient in g% g / l.

Determination of hemoglobin in Sali hemometry

Blood is inflated into a separate solution of the Sali hemometer. A hemometer is the simplest type of colorimeter. It consists of two small test tubes mounted on a tripod with a frosted mirror on the back of the tripod. One test tube of the apparatus contains a standard liquid, such as a one per cent solution of hematin chloride in glycerin; this solution is welded on both sides . Recently, this test tube has been replaced by brown glass rods that do not fade over time.

The second test tube, which is the same caliber as the first test tube, is empty; its wall is divided into 140. The blood to be tested is placed in the same test tube. Before decomposing the blood from the pipette into this solution, a decinormal solution of hydrochloric acid up to 10 degrees is poured into the solution. Precise measurements of blood with a pipette tip of the pipette, touch and the surface of the liquid and the liquid air, acid-down (try not to come to the last drops of hydrochloric acid slightly higher blowing into the tube wall, and then wash the tube bend a little, The blood in the solution is thoroughly mixed with hydrochloric acid and left for 10 minutes. After this time, water is poured into the solution until the color of the liquid in it is equal to the standard color. At the same time, the liquid is mixed either with a special glass rod or by shaking the solution. the level of the remaining liquid after dilution is determined according to the scale di; the corresponding level indicates the percentage of Hb relative to the amount assumed to be 100 in the norm.

1. The most accurate way to determine hemoglobin based on the oxygen capacity of the blood is 1.33 cm of oxygen per 1 g of hemoglobin. The norm is 14-16 g of Hb in 100 ml of blood in men and 12-14 g in women. The standard on the hemometer should be prepared so that the level set to 100 corresponds to 16 g Hb in 100 ml of blood. Unfortunately, the hemometers

produced are very diverse, often with an overly dark hue. In this regard, the amount of Hb turns out to be uncertain (often very low). It is necessary to make a "correction" to the readings of the hemometer, and the numerical values of the amount of hemoglobin in the blood, expressed in grams,

Determination of the number of erythrocytes in the blood

Erythrocytes are counted in the Burker or Goryaev chamber. To do this, the blood is first diluted 200 times with 3.5% saline solution in a special mixer. The mixer consists of a glass tube with an expanded area (reservoir) in the middle. To dilute the blood, draw up to 0.5 marks of blood in a mixer, then fill it to 101 marks with a 3.5% saline solution and shake for 3 minutes. The first drop of diluted blood is expelled and the next drop is carefully instilled into the counting chamber. The counting chamber will have a special mesh area and a drop of blood will spread to that mesh. Each of the nets will have several large squares, and inside them will be small squares. For example, Gorya e v kam e ras has 25 large squares, each containing 16 small squares. Less time, cells 5 is considered a small square (80 square). For example, more than 80 440 small square of red blood cells counted, it is a small square of 440: 80 = 5.5 m e e sluggish saying 1 mm ³ 5,5X400 = 22000, a mixture of 1 mm ³ p e e Rick Reef and the blood contains 22000x20 = 4400000 red blood cells. In healthy people, erythrocytes are around 4-5.10 in 1 liter of blood in men and 3.9-4.7.10 in women.

Determining the color index

The color k indicator is the ratio between the number of red blood cells in the blood and the hemoglobin mi q drug in them . When hemoglobin is 100% and erythrocytes are 5,000,000, the amount of hemoglobin corresponding to one erythrocyte is taken as a unit. Calculated by the formula: the amount of hemoglobin found expressed in grams is divided by 16 (or the amount of hemoglobin found expressed as a percentage is divided by 100), multiplied by the amount of erythrocytes found divided by 5,000,000. The color index is about 1.05 in the norm.

The color index indicates whether anemia in a particular case is due to a lack of more hemoglobin (low color index) or a low development of more red blood cells (high color index). However, this indicator determines how saturated each erythrocyte is in hemoglobin (on average). Under normal conditions, hemoglobin replenishes the erythrocyte until it is completely compressed. In order for the amount of hemoglobin in an erythrocyte to remain higher than normal and thus increase the color index to an equal level, the erythrocyte must increase its area or size. This is why when the red blood cells in such blood are larger than the average norm, the color index turns out to be large.

Determination of erythrocyte sedimentation rate

Rate of erythrocytes (Echt) Panchenko is determined by the apparatus of 135 pictures .

a b

135 Figures a and b

Because the erythrocytes in the peripheral blood have the same charge, they run away from each other and never stick together. Outside the vein, however, they begin to sink as a result of their weight and begin to stick together. However, this sinking rate should not exceed 2-15 mm per 1 hour in females and 2-1 0 mm / s in males . ECG depends on the amount of globulins, fibrinogen and mucopolysaccharides in the blood plasma.

In various diseases, the above substances that accelerate the adhesion of erythrocytes increase in the blood plasma and also accelerate ECG.

These diseases include various infectious collagen diseases, dangerous hangovers. The rate of erythrocyte sedimentation rate is a measure of the activity of a pathological condition. For many years, the Panchenkov instrument has been used to detect ECG.

This device consists of a tripod and 100 sections of glass tubes with a width of 1 mm. The glass is removed from the 5% sodium citrate solution up to the section 50 marked on the tube and lowered into the watch window. Blood is drawn from the patient's finger 2 more times up to 100 sections of the tube, which is also mixed with the reagent in the watch window and again filled up to 100 sections of the tube. The filled glass tube is placed in a precise vertical position on the tripod and after 1 hour it is determined how many mm the blood column has decreased.

Practical training 17

Diseases of the circulatory system. Clinic, diagnosis of anemia .

Laboratory work: blood test. Clinical significance of general blood analysis. Symptomatology of anemia (iron deficiency, posthemorrhagic, hemolytic).

1. Practical training module .

Training time - 4 hours	The number of students 1 : 0 to 12
Form of training	Practical training
Practical training plan	Laboratory work: blood test. Clinical significance of general blood analysis. Symptomatology of anemia (iron deficiency, posthemorrhagic, hemolytic).
The purpose of practical training :	Lab: To gain an understanding of blood testing . Symptomatology of anemia (iron deficiency, posthemorrhagic, hemolytic).
Teaching style	Inquiry. Demonstration of patients, interactive teaching methods, practical skills.
Form of teaching	In small subgroups.
O ' unit equipment	Calls to O Training Guidebook , practical Typo content , projectors , computer .
Training mode	Methodically equipped auditorium.
Monitoring and	Oral control: questions and answers, tests, problem solving.

evaluation	

1.2 . ***Technological card of practical training***

Stages and timing of work.	Educator	Learners
Preparatory stage	34. See the audience to control the purity karîm 35. Checks students ' readiness for training 36. And control q karîm	
1. bullet and training g contain levels of intra q (10 da q i q a)	1. Preparation of educational content on the topic. 2. Preparation of presentation slides for the introductory speech 3. Develop a list of references used in the study of science	
2 - the main stage (160 minutes)	1. Divide students into small groups and ask questions on the topic. 2. Uses display posters 3. Uses slides, multimedia 4. Conducts treatment 5. Summarizes and summarizes the information provided on the basis of topics, encourages and actively evaluates the active participant students	They are divided into small groups They watch They participate They listen and answer questions
3 See the final stages of diarrhea (10 da qiq a)	1. Concludes 2. Provides independent work 3. Gives homework	He listens Takes notes Takes notes

3. Assessment of students' theoretical knowledge:

A) Frontal method:

20. What does a general analysis of blood mean
21. Diagnostic value of general analysis of blood
22. Blood collection technique
23. Hemoglobin detection technique
24. Q form on elements y h REPORT
25. Determination of erythrocyte sedimentation rate
26. What is the leukocyte formula
27. Indications of normal blood flow
28. Puncture of blood-forming organs
29. Evaluation of hemolysis
30. The concept of anemia
31. Manifestations of anemia
32. Posthemorrhagic anemia, clinical manifestations
33. Describe the iron deficiency anemia
34. Causes of iron deficiency anemia
35. Clinical manifestations of iron deficiency anemia
36. Changes in blood analysis in iron deficiency anemia

37. Etiopathogenesis of V12 (folic acid) deficiency anemia
38. A general review of patients with V12 (folic acid) deficiency anemia

Laboratory diagnosis of blood in anemia

The "three-step interview" method

Three students will be selected in each group, and the roles of "doctor", "patient" and "expert-UASh" will be divided between them. The student who chooses the role of patient is told an anonymous diagnosis, and he or she makes complaints about that diagnosis, the doctor makes a diagnosis, and the expert checks the UASh complaints and the proportion of the diagnosis. Each group is consulted for 10-15 minutes, the expert examines the activity of the doctor on 3 points:

4. What was done right
5. What went wrong
6. How to do it

The conclusion of the group consultation is compared with the conclusion of the expert.

Another type: students are analyzed by the whole group, participating in the clinic in the role of an expert, in a real consultation.

Subject statement

Anemia (anemia) is a disease caused by a decrease in the amount of erythrocytes, hemoglobin and their content in erythrocytes per unit volume of blood, changes in their quality, which are hereditary and acquired (primary and secondary).

The causes of anemia are polyetiological and of various natures. Anemia is caused by the influence of exogenous and endogenous factors. Exogenous factors include infectious (malaria) and non-infectious (various toxic substances (phenylhydrazine), snake, scorpion bites, etc.), alimentary (vitamin B12 and folic acid, iron deficiency, etc.) factors. 'linadi. Examples of endogenous causes are systemic diseases. The causes of anemia also vary depending on the origin. In patients with anemia, first of all, there is a violation of the respiratory function of erythrocytes, ie the activity of oxygen delivery to tissues, and the consequences (if appropriate measures are not applied in a timely manner) are different from other diseases. At the same time, it should be noted that anemia is a very serious problem that is extremely comprehensive, not only medically-biological, but also ecologically-socially, economically, and so on. In anemias, various pathological forms of erythrocytes appear in the blood, which do not have normal functional capacity and have a low ability to carry oxygen. Some of them do not attach enough oxygen due to low hemoglobin retention, while others, such as young erythrocytes, on the one hand, attach less oxygen and, on the other hand, consume oxygen for their own life (they are resistant to various conditions and have short life cycles). ladi and so on).

Iron deficiency anemia

Distribution of the total amount of iron in the body (5 g):

In this type of anemia, the number of erythrocytes is almost unchanged, mainly due to a decrease in their saturation with

hemoglobin (from 30 to 35 pg). Hypochromic is characterized by hypoxemia, see Figure 131a and b .

a b

131 photos

Clinic: Subjective: Inquiry - interrogatio .

Complaints - **anemic, hypoxemic symptoms:** darkening of the eyes, decreased ability to work, rapid fatigue, shortness of breath, weakness, dizziness, palpitations, pain in the heart area, noise in the ears, decreased sensitivity of the skin, ant walking on the body, skin sensation, loss of appetite, nausea, loss of full cognition, nausea, consumption of chalk, smell of kerosene and gasoline (Pico chloratica), belching, discomfort in the epigastric area after a meal, ba intracranial fluid flow, hand and foot congestion, sideropenic dysphagia, taste disturbances (dysgysia), tendency to abnormal odors (Rossolimo-Bexterov syndrome was diagnosed in 1900-1901 and later, Plammer and Vinsons identified).

Hypoxic syndromes are accompanied by **central nervous** system - headache, dizziness, ghosts in the eyes, inability to concentrate for a long time, decreased thinking ability, memory loss, sleep disturbances, irritability, psychomotor in children developmental delay, crying, asthenovegetative disorders, and long-term subfebrile fever are disturbed by the nervous system.

Pulse lability by the **cardiovascular system** , tachycardia, in the arteries

" Syringe murmur ", a systolic murmur is heard at the apex of the heart. In severe cases, patients become very restless, weak, sleepy, crying. When patients are examined, they appear to be as pale or slightly yellowish as a pale puppet face. Patients have swollen face, swelling in the legs, knees, ecchymosis in the skin and mucous membranes. The skin is dry, the feet are cold, the muscles are loose.

Anamnesis of morbidity; attention should be paid to changes in the onset of the disease, how long it lasts, the condition of the hair, the condition of the nails-cologne, sideropenic syndromes, etc.

Anamnesis vitae; lifelong Inflammation of the gastrointestinal tract, the presence of worm diseases, breastfeeding, pregnancy, prolongation of the menstrual cycle and chronic excessive blood loss, gastric hyposecretion and other factors lead to the development of the disease.

Syndromes that occur in TTA

/ r	Disorders of complete cognition (Pico chloratica)	
	Impairment of complete cognition; signs such as chalk, lime, toothpaste and tooth	Dryness of the skin, decrease in skin tension (tur

	powder , eraser , sand, soil, raw meat, dough ist e goods, moisture, mud, aspiration to consume coal	
	Various unpleasant odors; The smell of kerosene, gasoline, acetone, fuel oil, rubber, cosmetic varnishes and paints, etc.	Hair thinning, dryness, graying, rapid breakage,
	Sudden desire to consume certain foods	Development of cologne - thinning and flattenin nail tissue, the appearance of transverse lines and de
		OG trail angle of rupture , MV trail Bo ' placer ' specific gingivitis , and should be the Reds ' ngach epithelium dystrophy
		Observation of atrophic gastritis and hypoacidity

Objective: - Symptoms of sideropenia: sideropenic dysphagia (Plammer-Vinson syndrome) is observed as a result of damage to the epithelium of the nasopharynx and upper esophagus. This is why dry and hard food swallowing is disrupted, food comes out again, and this is accompanied by pain in the upper third of the esophagus. Changes in the epithelium of the tongue lead to atrophy of its nipples, which is why the tongue is smooth, shiny, which leads to irritation and pain of the tongue. **Examination** - Inspectsio; children lag behind in growth, develop infantilism, pale skin and mucous membranes (waxy whitening of the skin), dry and mobile skin, thinning, flattening, brittle nails, deformity, spoon-shaped **cologne** (photo 132), gray hair,

132 photos

Spoon-shaped nail in the photo - **colonychia**

thinning, brittleness and shedding, dryness and brittleness are observed, muscle weakness. **Changes in the oral cavity** . The corners of the mouth rupture, sores appear, and changes in the oral cavity include angular stomatitis, gingivitis, glossitis, atrophy of the tongue, purulent inflammation of the gums (alveolar piorea), in the teeth The loss of luster darkens and leads to destruction as a result of taking iron supplements, which leads to rapid damage to the teeth, gastritis, duodenitis, colitis.

Palpation - Palpation; the expansion of the area of the heart impulse pulse can be detected. In children, the liver may be slightly enlarged.

Percussion - Percussio; The boundaries of the heart can be enlarged mainly in the left ventricle.

Auscultation - Auscultation; systolic murmur is heard in the apex of the heart and pulmonary artery, auscultation of blood vessels accelerates blood flow to the jugular veins mainly due to a decrease in the rheological properties of the blood, hence the murmur, mainly on the right side and this noise in the neck intensifies when turned in the opposite direction.

Laboratory: General analysis of blood; Hematocrit soni; The amount of iron in whey; It is necessary to determine the number of platelets .

The hemogram shows a sharp decrease in erythrocytes and more hemoglobin in the first place, and the color of the blood can be up to 0.6 (0.85-1.05 norm) and less. Hypochromia, microcytosis, anisocytosis, poikilocytosis are observed in the blood smear, and the degree of expression of microcytosis with hypochromia indicates a severe course of the disease. The number of reticulocytes is close to normal and may increase slightly during periods of bleeding. Thrombocytopenia is observed, relative lymphocytosis, monocytosis, eosinopenia can be observed. The decrease in serum iron is 1.5-2.5 times and the saturation of transferrin with iron is reduced by 15 times. Due to the decrease in the activity of iron- storing enzymes, atrophic processes take place in the gastric and intestinal mucosa, and a decrease in the production of achlorhydria and achilles, ie gastric juice, is observed.

Instrumental: Sterile puncture; On radiological examination, the folds of the mucous membranes of the esophagus, stomach, and intestines are flattened by atrophy, which can be seen more clearly when performing gastrofibroscopy. Morphological examination.

Vitamin B12 and folate deficiency anemia

Impairment of blood formation in this anemia occurs as a result of vitamin B **12** deficiency and is hyperchromic and megaloblastic type anemia (Fig. 133 a and b).

a b

133 Figures a and b

Inquiry. The clinical and blood changes of Addison-Birmer disease are most pronounced during relapse, and are more common in older people and more prevalent among men. The disease develops unnoticed, and the initial symptoms are weakness, rapid fatigue, the appearance of dryness of the tongue, dyspeptic conditions are disturbed. The heartbeat (tachycardia) is disturbed, the pulse on **palpation is** soft and rapid. The heart may shift to the left, on **auscultation** a systolic murmur is heard at the **apex of the** heart, and a "burr" murmur is heard in the jugular veins. On the ECG, the amplitude of the teeth decreases mainly T, the ST segment may shift below or above the isoline. Sometimes there are signs of funicular myelosis, accompanied by dysfunction of the nervous system. During the outbreak of Addison-Birmer disease, the clinic is characterized by a triad of syndromes: anemia, gastrointestinal disorders, neurological syndromes.

Anemic syndromes include : weakness, dizziness, tinnitus, shortness of breath, tingling in the heart area during tension, reminiscent of angina. When

patients are examined, the skin and visible mucous membranes become pale lemon yellow, and a subcutaneous fat layer is developed. In this category of patients, blood pressure is low, systolic murmur is heard on auscultation, and it is given to the veins. The ECG shows diffuse dystrophic changes in the myocardium.

Changes in the digestive system include irritability of the tongue, in many cases complete loss of sensation, pain in the tongue, loss of appetite, pain and discomfort in the epigastric area, disturbed fluid flow. Examination of patients shows a yellowish-yellow color of the skin and mucous membranes, as well as swelling of the face. **Changes in the oral cavity - the** tongue is dark red (raspberry color), shiny, the surface is smooth (atrophic Gunter glossitis), the tip of the tongue due to atrophy of the nipples, mainly on the tip and sides and on the sides the oral mucosa and the posterior wall are flattened by atrophy, sometimes sores appear on the mucous membranes of the oral cavity and at the tip of the tongue, around them, the teeth are prone to caries and become diseased.

Examination of gastric juice reveals the absence of gastromucoproteins and pepsin, as well as achlorhydria, which is maintained during remission. On palpation, the liver is painful and enlarged, soft. Sometimes the spleen also enlarges. In percussion, pain is felt in the flat and tubular (large calf) bones due to bone marrow hyperplasia. On fibrogastroduodenoscopy, we can see that the gastric mucosa is atrophied and thickened into polyps-like changes in the folds of the mucosa.

Neurological syndromes. As a result of vitamin B **12** deficiency, the lateral wall (columns) of the spinal cord are damaged, leading to dystrophic changes, which lead to **funicular myelosis** . Sometimes ischemic foci appear in the brain and nerve cells soften. The main clinical symptom is paresthesia, loss of depth and vibration sensitivity, mainly in the legs. As a result, ataxia is observed and movement is impaired. Weakness in the arms and legs, paresis, decreased or lost leg reflexes. At the same time Babinsky, Rossolimo pathological reflexes appear. Body temperature may rise during sensitivity.

When patients' blood is examined during an attack, there is a decrease in the number of erythrocytes and, in small amounts , an increase in the concentration of hemoglobin in them . In patients with this disease, the blood color index is higher than 1.05 and is hyperchromic. In the blood appear erythrocytes of different shapes and sizes (anisocytosis, poikilocytosis), leukopenia with neutropenia, eosinopenia, monocytopenia, thrombocytopenia, increased ECG. The number of reticulocytes increases after the start of treatment, which indicates an increase in erythropoiesis. In the blood smear, hemoglobin-rich erythrocytes, i.e. megalocytes, Jolly bodies, and Kebot rings, can be seen under a microscope.

G emolytic anemias

The main symptom of hemolytic sympathy is a high degree of breakdown of erythrocytes and a shortening of their life cycle. Under

physiological conditions, the lifespan of erythrocytes is 100-120 days. Aging erythrocytes are sequestrated (broken down) in the spleen sinuses and bone marrow. The bilirubin pigment formed as a result of physiological breakdown of erythrocytes is circulated in the blood in the form of free bilirubin and delivered to the liver cells. There it binds to glucuronic acid under the action of enzymes. The resulting bilirubin-glucuronide (bound bilirubin) passes from the liver cells into the bile ducts and is excreted in the intestine by the bile.

Hemolytic anemias are hereditary types and are associated with decreased erythrocyte resistance. There are 3 types of hemolytic anemia: 1. Decomposition - anemia in which the cause of hemolysis is the erythrocytes themselves; 2. (hematoxic) anemias caused by various toxins; 3. The cause of decomposition is antibodies associated with antibodies (autoimmune, immunohemolytic).

In one type of hemolytic anemia, hemolysis of erythrocytes occurs within the cell (macrophages), while in another type, hemolysis can occur in the blood itself, in which case hemoglobinuria is observed.

Hereditary hemolytic anemia

Classification. They are divided into 3 types:

Membranopathy. This anemia is mainly caused by a hereditary defect of the protein in the erythrocyte membrane. In this anemia, a genetic defect in the protein and a lack of ATF-aza, phospholipids, and cholesterol increase the permeability of the erythrocyte membrane and disrupt the potassium-sodium pump. As a result, sodium accumulates inside the cell and then water, the cell swells, changes shape (turns into a spherical shape). Such erythrocytes, i.e. spherocytes, do not have the ability to change their shape, it is difficult to pass through the sinuses in the spleen, and part of the erythrocyte separates (microspherocytes) becomes clogged and dies, eventually by spleen macrophages hemolysis occurs, swallowing - anemia occurs.

At the onset of the disease in the **clinic** , patients are mainly bothered by weakness, dizziness, fever. One of the main symptoms of the disease is yellowing of the skin and mucous membranes, the appearance of long-lasting, difficult-to-treat wounds on the knee. This depends on the extent to which the liver binds bilirubin. The color of the litter is dark yellow due to the abundance of stercobilin. Patients have developmental delays and changes in the structure of the facial skeleton in the form of "tower head", high palate, impaired development and placement of teeth, narrowing of the eyeballs, curved nose. On palpation of patients, we can detect enlargement of the spleen and sometimes the liver. The disease is accompanied by a crisis, accompanied by continuous hemolysis of the blood.

Enzymopathy . This type of anemia is mainly passed down from generation to generation through a recessive pathway. Mikrosferotsitar anemia, red blood cells in the normal form of the difference between the sky and the K reziztentligi high or normal blood cells. Examples of

enzymopathy are anemia due to deficiency of the enzymes glucose-6-phosphate dehydrogenase, glutathione reductase, pyruvate kinase, glutathione peroxidase in the membrane of erythrocytes. In this anemia, the return of NADF to NADFH is slowed, and the formation of reversible (restored) glutathione from oxidized glutathione is disrupted. As a result, along with the course of biochemical processes in the body and under the influence of various drugs, active forms of oxygen (atomic oxygen) and peroxide compounds damage the membrane of erythrocytes and cause hemolysis.

Clinic. In severe cases, patients experience fever, weakness, pain around the spine in the back, abdominal pain, shortness of breath accompanied by excessive vomiting, palpitations. Sometimes patients lose consciousness. Decomposed remnants of erythrocytes (hemoglobin) become clogged in the renal tubules and lead to renal failure. The skin and visible mucous membranes of the patients are yellowish in color and the color of the urine is dark , which is due to the separation of hemosiderin in the urine. On palpation we can detect enlargement of the spleen and sometimes the liver .

Hemoglobinopathy. Sickle- (crescent) cell anemia. This anemia is caused by a defect in the primary structure of hemoglobin. HbF is detected in the patient's blood (normally HbA), a decrease in the hypoxic resistance of hemoglobin to anemia due to the replacement of 6-hydrophilic glutamic acid by valine in one of the b chains of Hb in such a hemoglobin molecule.

Thalassemia. Thalassemia is due to a violation of the synthesis of polypeptide chains of hemoglobin, α-thalassemia occurs when the synthesis of the α chain is disrupted, and--thalassemia occurs when the synthesis of the b chain is disrupted.

a - thalassemia. During hemoglobin synthesis, the chromosome responsible for the α- chain is mutated, and the α- chain synthesis of Hb is disrupted. Bart Hb is synthesized in children and HbH in adults. Such hemoglobin-containing erythrocytes are rapidly oxidized, lose their elasticity, have a short lifespan, and they break down in the spleen.

b - in thalassemia . Excess HbA2 and HbF (Fetal) are synthesized in the b- chain of Hb-containing globin , and a -chain synthesis occurs. In the membrane of such erythrocytes, the permeability to calcium increases, resulting in the loss of elasticity of erythrocytes and their rapid degradation by enzymes.

In thalassemia, erythrocytes undergo hemolysis in macrophages in organs (spleen, liver, etc.). In hemolytic anemias, hemoglobin is often reduced in the same way as erythrocytes in the blood, so the color of the blood increases with the number of polychromatophilic normoblasts and sideroblasts. Poykilocytosis, hypochromia, and marked erythrocytes can be seen in stained blood smears. The diameter of erythrocytes is smaller than normal, the number of reticulocytes increases, and normoblasts can also be found in the blood. The number of leukocytes is slightly higher than normal.

According to the clinical course, thalassemia is a so-called "small" and "large", "small" occurs in heterozygotes and is a mild form. The heavy type of "adult" occurs in homozygotes and manifests itself in childhood. Homozygous b thalassemia (Culi anemia) in infants begins to show clinical signs 7 months after birth, which coincides with the period of HbF to HbA exchange.

Clinic. In such children, weakness, lethargy, paleness, mild jaundice in the whites of the eyes and mucous membranes are observed, along with hepatosplenomegaly and enlargement of the abdomen. Impairment of the development of the skull in such children leads to its minor shape and the appearance of a Mongolian face.

X-ray examination of the bones reveals changes such as osteoporosis, thinning of the tubular bones, and brush-like (hedgehog needles) in the skull.

On laboratory examination, patients have target erythrocytes, anisocytosis, paikilocytosis, multiple erythrocytes and normablasts, and reticulocytosis . Unlike other types of hemolytic anemia, Kuli anemia increases the osmotic resistance of erythrocytes.

In patients with homozygous--thalassemia, HbF is increased by up to 90%, HbA1 synthesis is decreased, and HbA2 may be decreased or increased in moderation.

Family-hereditary (genealogical) analysis suggests that the proband b for thalamic anemia received the thalassemia genes from both parents at the same time.

Acquired hemolytic anemia

Clinic: Acute and chronic autoimmune GA are differentiated according to the clinical course. In the acute form, the disease begins with sudden weakness, palpitations, shortness of breath, fever, yellowing of the skin and mucous membranes. In the chronic form, the disease begins gradually and less change in the condition of patients is observed. Although patients do not complain, palpation can reveal that their spleen and sometimes the liver are enlarged. The disease is accompanied by a hemolytic crisis, which occurs under the influence of both infection and cold. In the acute form of the disease, leukocytosis progresses to myelocytes with a shift to the left of the leukocyte count. In the chronic form, ECG is high even though the leukocyte count is normal. Hyperplasia is observed in the bone marrow. The amount of unbound bilirubin in the blood increases, the excretion of stercobilin in the feces increases.

GA (Markiafav - Mikel's disease) with paroxysmal evening hemoglobinuria and persistent hemosiderinuria

It was first identified at the same time by Markiafav-Mikel (1928), is a type of increased GA, and is accompanied by persistent intravenous hemolysis, in which hemosiderin is excreted in the urine. Every 1: 500,000 population, children and adults (30-40 years old) become ill.

Clinic. Patients mainly complain of weakness, dizziness, rapid fatigue, shortness of breath, palpitations. Abdominal pain is characteristic during a crisis, which is associated with thrombosis of the mesenteric vessels. Occasionally, thrombosis is also observed in peripheral vessels (arms, legs, brain, spleen, kidneys). Body temperature can also rise during a crisis. The color of urine is dark due to the presence of hemosiderin and hemoglobin. Hemoglobinuria sometimes occurs in the evening, which is why it is called evening paroxysmal hemoglobinuria. It is associated with acidosis and properdin activation in the evening . The most characteristic for this disease is hemoglobinuria.

The main factors in the diagnosis of evening paroxysmal hemoglobinuria are:

1. Reticulocytosis and hypochromia.
2. Neutropenia and thrombocytopenia
3. Increased levels of unbound bilirubin in the blood plasma and the presence of hemoglobinuria, hemosiderinuria.
4. Positiveness of hem (Gem) (acidic) and sucrose method.
5. Negativeness of the Coombs method.

The main modern diagnostic methods are the absence of CD55 and CD59 expression in erythrocytes and platelets by cytofluorimetry.

Aplastic anemia

In aplastic or hypoplastic anemia there is a decrease in the activity of dividing cells in the blood-forming tissues (bone marrow), pantsitopenia, ie a decrease in all types of blood cells. At the heart of this disease lies a violation of cell proliferation and differentiation. Blood production is more profoundly impaired in aplastic anemia than in hypoplastic anemia.

Clinic. Patients are mainly bothered by weakness, rapid fatigue, shortness of breath, palpitations, bleeding from the nose and gums in the acute and subacute course, prolongation and increase of the menstrual cycle in women, fever.

When examining patients, their skin becomes pale, lemon-colored, and yellow in color. Hemorrhagic syndromes develop in aplastic or hypoplastic anemia — petechiae, ecchymoses, subcutaneous hemorrhages, and hematomas occur mainly at the injection sites. Therefore, the tow symptom is positive in these patients. In patients, bleeding may sometimes occur in the mucous membranes of the oral cavity, in the retina of the eye, and sometimes in the brain. Blood pressure drops. On auscultation, a systolic murmur is heard at all points, and these murmurs are transmitted to the great blood vessels.

Laboratory changes. Normocytosis and normachromia are observed in blood smears. The disease is accompanied by leukopenia and the body's ability to protect itself from various biological factors is reduced. One of the specific manifestations of this anemia is a low number of platelets and, consequently, a violation of blood clotting, bleeding. When bone marrow is punctured, there is a decrease in nuclear-protective cells, inhibition of

erythroid, myeloid, megakaryocyte cell development. In this type of anemia, the bone marrow is completely replaced by fat cells.

Acute posthemorrhagic anemia

Acute posthemorrhagic anemia occurs for a variety of reasons. These include: trauma, trauma, surgery, vascular rupture, pathological conditions in pregnancy (postpartum placental abruption , (Shixan syndrome-see below)), fetal development in the fallopian tube, etc.

Shikhan syndrome is a postpartum necrosis of the pituitary gland. This condition is a complication that results from excessive acute blood loss after delivery and is accompanied by the development of arterial hypotension. This is because the size of the pituitary gland increases during pregnancy, but the blood supply remains at the same level. Acute blood loss and arterial hypotension after childbirth reduce the blood supply to the pituitary gland and lead to the development of hypoxia and necrosis.

After acute blood loss in the **clinic** , patients experience fatigue, dizziness, darkening of the eyes, nausea, vomiting, noise in the ears, palpitations, ghosts in the eyes, the feeling of itching does. Patients vomit blood when bleeding from internal organs (pure blood from the esophagus and coffee-like vomit from the stomach), bleeding with feces (melena, dark stomach and small intestine, and clean or thickened blood when bleeding from the intestine). Sometimes it is difficult to detect bleeding from internal organs when there is bleeding in the cavities. Examples of these are bleeding into the cavities, pleural cavity (hemothorax), trauma to the internal organs in the abdominal cavity, bleeding (due to rupture of the liver, spleen), etc.

On examination of patients, the skin and visible mucous membranes are pale, the skin is covered with cold sweat, respiration is rapid, shallow, pulse is accelerated, fibrous, soft, blood pressure drops both systolic and diastolic pressure.

Auscultation . When the heart is auscultated, we can hear that the tones are accelerated, the functional noise, and the I tone are amplified. On auscultation of blood vessels, blood flow is accelerated, mainly due to a decrease in the rheological properties of the blood in the jugular veins, so that the "Boricha" noise is heard, mainly on the right side, and this noise is amplified when turning the neck in the opposite direction.

There are almost no noticeable changes in the hemogram for 24-48 hours after blood loss. In this case, only the total volume of blood decreases, and the number of erythrocytes per unit of measurement, the amount of hemoglobin, color index and hematocrit may be normal. Examination of blood smear with a microscope reveals normocytosis, normochromia. Later (after 1-2 days) the volume of blood lost is replenished by the fluid passing through the tissues, the blood is diluted, the amount of erythrocytes and hemoglobin in its unit of measurement decreases, but the color of the blood does not change. After 3-4 days, the number of reticulocytes in the blood increases, and their peak of reproduction occurs in the last days of the week, during which nuclear erythrocytes may also appear. Now at the expense of

young erythrocytes the color of the blood decreases slightly. The bone marrow is an extremely rich tissue for the sphincters, which are supplied by the sphincters. Erythropoiesis lasts for a certain period of time, and the sphincter of the capillaries is closed until the erythrocytes mature. As the erythrocytes mature, the sphincters relax and the cells move into the general bloodstream. As regeneration intensifies, due to the constant presence of young cells in the bone marrow, the relaxation of the sphincter of the pelvis coincides with the immaturity of the cells, and the cells enter the bloodstream. Hence, acute posthemorrhagic anemia is hyperregenerative anemia. Along with erythropoiesis, leukopoiesis also increases (neutrophilia). Thrombopoiesis may also be exacerbated.

In this type of anemia, rapid and slow compensatory mechanisms are activated. These are; - In the first days - Acceleration of cardiac activity; Accelerated breathing; Narrowing of blood vessels; The release of blood that has stopped in the depot into the bloodstream; Transmission of interstitial fluid into the bloodstream;

- After 3-4 days; Increased blood clotting; Increased blood production (erythropoiesis) is observed in the bone marrow.

Chronic posthemorrhagic anemia

Chronic posthemorrhagic anemia is characterized by various chronic (eg, gastrointestinal ulcers, hemorrhoids, bleeding disorders in women, hemophilia, gastrointestinal, lung, kidney, and other organ tumors, bronchiectasis, hemorrhagic diathesis and bq) are observed in patients with small amounts of prolonged bleeding.

Clinically, after chronic blood loss, patients experience fatigue, dizziness, darkening of the eyes, nausea, vomiting, noise in the ears, palpitations, ghosts flying in front of the eyes. Patients experience changes such as bleeding with feces (melena, dark stomach and bleeding from the small intestine and clean or coagulated blood from the colon).

On examination of patients, the skin and visible mucous membranes are pale, the skin is moist, covered with sweat, breathing is rapid, shallow, pulse is accelerated, fibrous, soft, blood pressure decreases both systolic and diastolic pressure.

Auscultation . When the heart is auscultated, we can hear that the tones are accelerated, the functional noise, and the I tone are amplified. On auscultation of blood vessels, blood flow is accelerated, mainly due to a decrease in the rheological properties of the blood in the jugular veins, so that the "Boricha" noise is heard, mainly on the right side, and this noise is amplified when turning the neck in the opposite direction.

Hypochromia, poikilocytosis, anisocytosis, microcytosis are seen in the blood smear. Platelet counts are slightly below normal. The number of leukocytes is low or relative leukocytosis is observed. Slight reticulocytosis, leukocytosis (temporary) may be observed during blood loss. In chronic anemia caused by bleeding, white blood cells change slightly: in the regenerative phase the leukocytes increase slightly (shifted to the left), in the

hyporegenerative phase, in the next period the amount of leukocytes decreases slightly (shifts to the right). The prognosis of anemia that occurs after bleeding depends on the causes that caused it. If these causes cannot be eliminated, the anemia will worsen and take on an aplastic form. If these causes can be eliminated, the tumor may return to its original state over time, and the anemia may disappear.

Technological map of practical training

Practical training №1

Digestive system. Methods of examination of patients, interrogation, examination, palpation, percussion, laboratory and instrumental examination . **1.1. Technological model of practical training .**

Class time is 4 p.m.	Number of students up to 10
Place of training	An auditorium equipped with computers, multimedia and posters
Form of training	Practical training
Practical training plan	Digestive system. Methods of examination of patients, interrogation, examination, palpation, percussion, laboratory and instrumental examination
The purpose of practical training	To teach students methods of examination of patients, interrogation, examination, palpation, percussion, laboratory and instrumental examination
Teaching style	Inquiry, performance of practical skills, curation of patients
Form of teaching	Divide into small groups
Training equipment	Educational-methodical complex, Textbook, multimedia, posters, model
Evaluation procedure	On a 100-point scale with interactive games, assessment, Wenn diagram, problem solving and tests

1.2. Technological map of practical training .

Stages and timing of work	Educator	Learners
Preparatory stage (5 minutes)	1. The cleanliness of the auditorium is controlled 2. Student attendance is checked 3. To check the readiness of students for lessons	Be prepared to hear
O Training o ' the process of taking steps	1. Subject Expert liability 2. Preparation of presentation slides for the introductory lecture, preparation of the computer	Students are prepared

(1 0 minutes)	3. Putting multimedia, videos 4. Develop a list of references used in the study of science	
Theoretical training (Qiq a at 55)	1. Students are divided into small groups and given questions on the topic Assessment, Wenn diagram and bq. 2. Demonstration posters are used 3. Slides, multimedia are used 4. Summarizes and summarizes the information provided on the basis of the topics, encourages and actively evaluates the active participant students	A small group of the b h o ' Linas Q watch iladilar q participate t listen and answ questions
Clinical training (8 0 minutes)	1. Patients are curated together with the teacher 2. Students are assigned to each patient and they curate independently.	Students record the results of patient analysi and curation results in a notebook
Y akuniy bos q ich (10 da q i q a)	1. Concludes 2. Provides independent work 3. Gives homework	He listens Takes notes

Practical training №2

Main clinical syndromes: indigestion syndrome, intestinal absorption syndrome, acute abdomen, acute bleeding from the digestive system

1.1. Technological model of practical training .

Class time is 4 p.m.	Number of students up to 10
Place of training	An auditorium equipped with computers, multimedia and posters
Form of training	Practical training
Practical training plan	Basic clinical syndromes. Methods of examination of patients, interrogation, examination, palpation, percussion, laboratory and instrumental examination
The purpose of practical training	To teach students methods of examination of patients, interrogation, examination, palpation, percussion, laboratory and instrumental examination
Teaching style	Inquiry, performance of practical skills, curation of patients
Form of teaching	Divide into small groups
Training equipment	Educational-methodical complex, Textbook, multimedia, posters, model
Evaluation procedure	On a 100-point scale with interactive games, assessment, Wenn diagram, problem solving and tests

1.2. Technological map of practical training .

Stages and timing of work	Educator	Learners
Preparatory stage (5 minutes)	1. The cleanliness of the auditorium is controlled 2. Student attendance is checked 3. To check the readiness of students for lessons	Be prepared to hear
O Training o ' the process of taking steps (1 0 minutes)	1. Subject Expert liability 2. Preparation of presentation slides for the introductory lecture, preparation of the computer 3. Putting multimedia, videos 4. Develop a list of references used in the study of science	Students are prepared
Theoretical training (Qiq a at 55)	1. Students are divided into small groups and given questions on the topic Assessment, Wenn diagram and bq. 2. Demonstration posters are used 3. Slides, multimedia are used 4. Summarizes and summarizes the information provided on the basis of the topics, encourages and actively evaluates the active participant students	A small group of the b h o ' Linas Q watch iladilar q participate t listen and answ questions
Clinical training (8 0 minutes)	1. Patients are curated together with the teacher 2. Students are assigned to each patient and they curate independently.	Students record the results of patient analysi and curation results in a notebook
Y akuniy bos q ich (10 da q i q a)	1. Concludes 2. Provides independent work 3. Gives homework	He listens Takes notes

Practical training №3

Diseases of the digestive system: diseases of the esophagus , g astritis

1.1. Technological model of practical training .

Class time is 4 p.m.	Number of students up to 10
Place of training	An auditorium equipped with computers, multimedia and posters
Form of training	Practical training
Practical training plan	Diseases of the digestive system: diseases of the esophagus , g astritis . . Methods of examination of patients, interrogation, examination, palpation, percussion, laboratory and instrumental examination
The purpose of practical training	To teach students methods of examination of patients, interrogation, examination, palpation, percussion, laboratory and instrumental examination
Teaching style	Inquiry, performance of practical skills, curation of patients
Form of teaching	Divide into small groups
Training equipment	Educational-methodical complex, Textbook, multimedia, posters, model

Evaluation procedure	On a 100-point scale with interactive games, assessment, Wenn diagram, problem solving and tests

1.2. Technological map of practical training .

Stages and timing of work	Educator	Learners
Preparatory stage (5 minutes)	1. The cleanliness of the auditorium is controlled 2. Student attendance is checked 3. To check the readiness of students for lessons	Be prepared to hear
O Training o ' the process of taking steps (1 0 minutes)	1. Subject Expert liability 2. Preparation of presentation slides for the introductory lecture, preparation of the computer 3. Putting multimedia, videos 4. Develop a list of references used in the study of science	Students are prepared
Theoretical training (Qiq a at 55)	1. Students are divided into small groups and given questions on the topic Assessment, Wenn diagram and bq. 2. Demonstration posters are used 3. Slides, multimedia are used 4. Summarizes and summarizes the information provided on the basis of the topics, encourages and actively evaluates the active participant students	A small group of the b h o ' Linas Q watch iladilar q participate t listen and answ questions
Clinical training (8 0 minutes)	1. Patients are curated together with the teacher 2. Students are assigned to each patient and they curate independently.	Students record the results of patient analysi and curation results in a notebook
Y akuniy bos q ich (10 da q i q a)	1. Concludes 2. Provides independent work 3. Gives homework	He listens Takes notes

Practical training №4

Y ara disease, mal`absorbtsiya syndrome, chronic colitis

1.1. Technological model of practical training .

Class time is 4 p.m.	Number of students up to 10
Place of training	An auditorium equipped with computers, multimedia and posters
Form of training	Practical training
Practical training plan	Y ara disease, mal`absorbtsiya syndrome, chronic colitis . Methods of examination of patients, interrogation, examination, palpation, percussion, laboratory and instrumental examination
The purpose of practical training	To teach students methods of examination of patients, interrogation, examination, palpation, percussion, laboratory and instrumental examination

Teaching style	Inquiry, performance of practical skills, curation of patients
Form of teaching	Divide into small groups
Training equipment	Educational-methodical complex, Textbook, multimedia, posters, model
Evaluation procedure	On a 100-point scale with interactive games, assessment, Wenn diagram, problem solving and tests

1.2. Technological map of practical training .

Stages and timing of work	Educator	Learners
Preparatory stage (5 minutes)	1. The cleanliness of the auditorium is controlled 2. Student attendance is checked 3. To check the readiness of students for lessons	Be prepared to hear
O Training o ' the process of taking steps (1 0 minutes)	1. Subject Expert liability 2. Preparation of presentation slides for the introductory lecture, preparation of the computer 3. Putting multimedia, videos 4. Develop a list of references used in the study of science	Students are prepared
Theoretical training (Qiq a at 55)	1. Students are divided into small groups and given questions on the topic Assessment, Wenn diagram and bq. 2. Demonstration posters are used 3. Slides, multimedia are used 4. Summarizes and summarizes the information provided on the basis of the topics, encourages and actively evaluates the active participant students	A small grou of their b o h Linas Q watch iladilar q participate t listen and answ questions
Clinical training (8 0 minutes)	1. Patients are curated together with the teacher 2. Students are assigned to each patient and they curate independently.	Students record the results of patient analysi and curation results in a notebook
Y akuniy bos q ich (10 da q i q a)	1. Concludes 2. Provides independent work 3. Gives homework	He listens Takes notes

Practical training №5

Liver and bile ducts. Methods of examination: interrogation, physical examination (examination, palpation, percussion)

1.1. Technological model of practical training .

Class time is 4 p.m.	Number of students up to 10
Place of training	An auditorium equipped with computers, multimedia and posters
Form of training	Practical training

Practical training plan	Liver and bile ducts. Methods of inspection: interrogation, methods of physical inspection . Methods of examination of patients, interrogation, examination, palpation, percussion, laboratory and instrumental examination
The purpose of practical training	To teach students methods of examination of patients, interrogation, examination, palpation, percussion, laboratory and instrumental examination
Teaching style	Inquiry, performance of practical skills, curation of patients
Form of teaching	Divide into small groups
Training equipment	Educational-methodical complex, Textbook, multimedia, posters, model
Evaluation procedure	On a 100-point scale with interactive games, assessment, Wenn diagram, problem solving and tests

1.2. Technological map of practical training .

Stages and timing of work	Educator	Learners
Preparatory stage (5 minutes)	1. The cleanliness of the auditorium is controlled 2. Student attendance is checked 3. To check the readiness of students for lessons	Be prepared to hear
O Training o ' the process of taking steps (1 0 minutes)	1. Subject Expert liability 2. Preparation of presentation slides for the introductory lecture, preparation of the computer 3. Putting multimedia, videos 4. Develop a list of references used in the study of science	Students are prepared
Theoretical training (Qiq a at 55)	1. Students are divided into small groups and given questions on the topic Assessment, Wenn diagram and bq. 2. Demonstration posters are used 3. Slides, multimedia are used 4. Summarizes and summarizes the information provided on the basis of the topics, encourages and actively evaluates the active participant students	A small group of the b h o ' Linas Q watch iladilar q participate t listen and answ questions
Clinical training (8 0 minutes)	1. Patients are curated together with the teacher 2. Students are assigned to each patient and they curate independently.	Students record the results of patient analysi and curation results in a notebook
Y akuniy bos q ich (10 da q i q a)	1. Concludes 2. Provides independent work 3. Gives homework	He listens Takes notes

Practical training №6

Liver and bile ducts. Test methods: laboratory-instrumental test methods). Basic clinical syndromes

1.1. Technological model of practical training .

Class time is 4 p.m.	Number of students up to 10
Place of training	An auditorium equipped with computers, multimedia and posters
Form of training	Practical training
Practical training plan	Liver and bile ducts. Test methods: laboratory-instrumental test methods). Basic clinical syndromes **.** Methods of examination of patients, interrogation, examination, palpation, percussion, laboratory and instrumental examination
The purpose of practical training	To teach students methods of examination of patients, interrogation, examination, palpation, percussion, laboratory and instrumental examination
Teaching style	Inquiry, performance of practical skills, curation of patients
Form of teaching	Divide into small groups
Training equipment	Educational-methodical complex, Textbook, multimedia, posters, model
Evaluation procedure	On a 100-point scale with interactive games, assessment, Wenn diagram, problem solving and tests

1.2. Technological map of practical training .

Stages and timing of work	Educator	Learners
Preparatory stage (5 minutes)	1. The cleanliness of the auditorium is controlled 2. Student attendance is checked 3. To check the readiness of students for lessons	Be prepared to hear
O Training o ' the process of taking steps (1 0 minutes)	1. Subject Expert liability 2. Preparation of presentation slides for the introductory lecture, preparation of the computer 3. Putting multimedia, videos 4. Develop a list of references used in the study of science	Students are prepared
Theoretical training (Qiq a at 55)	1. Students are divided into small groups and given questions on the topic Assessment, Wenn diagram and bq. 2. Demonstration posters are used 3. Slides, multimedia are used 4. Summarizes and summarizes the information provided on the basis of the topics, encourages and actively evaluates the active participant students	A small group of the b h o ' Linas Q watch iladilar q participate t listen and answ questions

Clinical training (8 0 minutes)	1. Patients are curated together with the teacher 2. Students are assigned to each patient and they curate independently.	Students record the results of patient analysi and curation results in a notebook
Y akuniy bos q ich (10 da q i q a)	1. Concludes 2. Provides independent work 3. Gives homework	He listens Takes notes

Practical training №7

Liver and bile ducts. Chronic hepatitis

1.1. Technological model of practical training .

Class time is 4 p.m.	Number of students up to 10
Place of training	An auditorium equipped with computers, multimedia and posters
Form of training	Practical training
Practical training plan	Liver and bile ducts. Chronic hepatitis . Methods of examination of patients, interrogation, examination, palpation, percussion, laboratory and instrumental examination
The purpose of practical training	To teach students methods of examination of patients, interrogation, examination, palpation, percussion, laboratory and instrumental examination
Teaching style	Inquiry, performance of practical skills, curation of patients
Form of teaching	Divide into small groups
Training equipment	Educational-methodical complex, Textbook, multimedia, posters, model
Evaluation procedure	On a 100-point scale with interactive games, assessment, Wenn diagram, problem solving and tests

1.2. Technological map of practical training .

Stages and timing of work	Educator	Learners
Preparatory stage (5 minutes)	1. The cleanliness of the auditorium is controlled 2. Student attendance is checked 3. To check the readiness of students for lessons	Be prepared to hear
O Training o ' the process of taking steps (1 0 minutes)	1. Subject Expert liability 2. Preparation of presentation slides for the introductory lecture, preparation of the computer 3. Putting multimedia, videos 4. Develop a list of references used in the study of	Students are prepared

	science	
Theoretical training (Qiq a at 55)	1. Students are divided into small groups and given questions on the topic Assessment, Wenn diagram and bq. 2. Demonstration posters are used 3. Slides, multimedia are used 4. Summarizes and summarizes the information provided on the basis of the topics, encourages and actively evaluates the active participant students	A small group of the b h o ' Linas Q watch iladilar q participate t listen and answ questions
Clinical training (8 0 minutes)	1. Patients are curated together with the teacher 2. Students are assigned to each patient and they curate independently.	Students record the results of patient analysi and curation results in a notebook
Y akuniy bos q ich (10 da q i q a)	1. Concludes 2. Provides independent work 3. Gives homework	He listens Takes notes

Practical training №8

Cirrhosis of the liver, chronic cholecystitis. Intermediate control № 3

1.1. Technological model of practical training .

Class time is 4 p.m.	Number of students up to 10
Place of training	An auditorium equipped with computers, multimedia and posters
Form of training	Practical training
Practical training plan	Cirrhosis of the liver, chronic cholecystitis. Methods of examination of patients, interrogation, examination, palpation, percussion, laboratory and instrumental examination
The purpose of practical training	To teach students methods of examination of patients, interrogation, examination, palpation, percussion, laboratory and instrumental examination
Teaching style	Inquiry, performance of practical skills, curation of patients
Form of teaching	Divide into small groups
Training equipment	Educational-methodical complex, Textbook, multimedia, posters, model
Evaluation procedure	On a 100-point scale with interactive games, assessment, Wenn diagram, problem solving and tests

1.2. Technological map of practical training .

Stages and timing of work	Educator	Learners
Preparatory stage (5 minutes)	1. The cleanliness of the auditorium is controlled 2. Student attendance is checked 3. To check the readiness of students for lessons	Be prepared to hear
O Training o ' the process of taking steps (1 0 minutes)	1. Subject Expert liability 2. Preparation of presentation slides for the introductory lecture, preparation of the computer 3. Putting multimedia, videos 4. Develop a list of references used in the study of science	Students are prepared
Theoretical training (Qiq a at 55)	1. Students are divided into small groups and given questions on the topic Assessment, Wenn diagram and bq. 2. Demonstration posters are used 3. Slides, multimedia are used 4. Summarizes and summarizes the information provided on the basis of the topics, encourages and actively evaluates the active participant students	A small group of the b h o ' Linas Q watch iladilar q participate t listen and answ questions
Clinical training (8 0 minutes)	1. Patients are curated together with the teacher 2. Students are assigned to each patient and they curate independently.	Students record the results of patient analysi and curation results in a notebook
Y akuniy bos q ich (10 da q i q a)	1. Concludes 2. Provides independent work 3. Gives homework	He listens Takes notes

Practical training №9

Renal and urinary system. Methods of examination: interrogation, methods of physical examination (examination, palpation, percussion, laboratory-instrumental methods).

1.1. Technological model of practical training .

Class time is 4 p.m.	Number of students up to 10
Place of training	An auditorium equipped with computers, multimedia and posters
Form of training	Practical training
Practical training plan	Renal and urinary system. Control methods: . Methods of examination of patients, interrogation, examination, palpation, percussion, laboratory and instrumental examination
The purpose of practical training	To teach students methods of examination of patients, interrogation, examination, palpation, percussion, laboratory and instrumental examination
Teaching style	Inquiry, performance of practical skills, curation of patients

Form of teaching	Divide into small groups
Training equipment	Educational-methodical complex, Textbook, multimedia, posters, model
Evaluation procedure	On a 100-point scale with interactive games, assessment, Wenn diagram, problem solving and tests

1.2. Technological map of practical training .

Stages and timing of work	Educator	Learners
Preparatory stage (5 minutes)	1. The cleanliness of the auditorium is controlled 2. Student attendance is checked 3. To check the readiness of students for lessons	Be prepared to hear
O Training o ' the process of taking steps (1 0 minutes)	1. Subject Expert liability 2. Preparation of presentation slides for the introductory lecture, preparation of the computer 3. Putting multimedia, videos 4. Develop a list of references used in the study of science	Students are prepared
Theoretical training (Qiq a at 55)	1. Students are divided into small groups and given questions on the topic Assessment, Wenn diagram and bq. 2. Demonstration posters are used 3. Slides, multimedia are used 4. Summarizes and summarizes the information provided on the basis of the topics, encourages and actively evaluates the active participant students	A small group of the b h o ' Linas Q watch iladilar q participate t listen and answ questions
Clinical training (8 0 minutes)	1. Patients are curated together with the teacher 2. Students are assigned to each patient and they curate independently.	Students record the results of patient analysi and curation results in a notebook
Y akuniy bos q ich (10 da q i q a)	1. Concludes 2. Provides independent work 3. Gives homework	He listens Takes notes

Practical training №10

Diseases of the kidneys and urinary system. Basic clinical syndromes

1.1. Technological model of practical training .

Class time is 4 p.m.	Number of students up to 10
Place of training	An auditorium equipped with computers, multimedia and posters
Form of training	Practical training
Practical training plan	Diseases of the kidneys and urinary system . Basic clinical syndromes . Methods of examination of patients, interrogation, examination, palpation, percussion, laboratory and instrumental examination

The purpose of practical training	To teach students methods of examination of patients, interrogation, examination, palpation, percussion, laboratory and instrumental examination
Teaching style	Inquiry, performance of practical skills, curation of patients
Form of teaching	Divide into small groups
Training equipment	Educational-methodical complex, Textbook, multimedia, posters, model
Evaluation procedure	On a 100-point scale with interactive games, assessment, Wenn diagram, problem solving and tests

1.2. Technological map of practical training .

Stages and timing of work	Educator	Learners
Preparatory stage (5 minutes)	1. The cleanliness of the auditorium is controlled 2. Student attendance is checked 3. To check the readiness of students for lessons	Be prepared to hear
O Training o ' the process of taking steps (1 0 minutes)	1. Subject Expert liability 2. Preparation of presentation slides for the introductory lecture, preparation of the computer 3. Putting multimedia, videos 4. Develop a list of references used in the study of science	Students are prepared
Theoretical training (Qiq a at 55)	1. Students are divided into small groups and given questions on the topic Assessment, Wenn diagram and bq. 2. Demonstration posters are used 3. Slides, multimedia are used 4. Summarizes and summarizes the information provided on the basis of the topics, encourages and actively evaluates the active participant students	A small group of the b h o ' Linas Q watch iladilar q participate t listen and answ questions
Clinical training (8 0 minutes)	Supervision of patients with 1.O'qituvchi 2. Students are assigned to each patient and they curate independently.	Students record the results of patient analysi and curation results in a notebook
Y akuniy bos q ich (10 da q i q a)	1. Concludes 2. Provides independent work 3. Gives homework	He listens Takes notes

Practical training №11

Acute and chronic glomerulonephritis

1.1. Technological model of practical training .

Class time is 4 p.m.	Number of students up to 10

Place of training	An auditorium equipped with computers, multimedia and posters
Form of training	Practical training
Practical training plan	Acute and chronic glomerulonephritis . Methods of examination of patients, interrogation, examination, palpation, percussion, laboratory and instrumental examination
The purpose of practical training	To teach students methods of examination of patients, interrogation, examination, palpation, percussion, laboratory and instrumental examination
Teaching style	Inquiry, performance of practical skills, curation of patients
Form of teaching	Divide into small groups
Training equipment	Educational-methodical complex, Textbook, multimedia, posters, model
Evaluation procedure	On a 100-point scale with interactive games, assessment, Wenn diagram, problem solving and tests

1.2. Technological map of practical training .

Stages and timing of work	Educator	Learners
Preparatory stage (5 minutes)	1. The cleanliness of the auditorium is controlled 2. Student attendance is checked 3. To check the readiness of students for lessons	Be prepared to hear
O Training o ' the process of taking steps (1 0 minutes)	1. Subject Expert liability 2. Preparation of presentation slides for the introductory lecture, preparation of the computer 3. Putting multimedia, videos 4. Develop a list of references used in the study of science	Students are prepared
Theoretical training (Qiq a at 55)	1. Students are divided into small groups and given questions on the topic Assessment, Wenn diagram and bq. 2. Demonstration posters are used 3. Slides, multimedia are used 4. Summarizes and summarizes the information provided on the basis of the topics, encourages and actively evaluates the active participant students	A small grou of their b o h Linas Q watch iladilar q participate t listen and answ questions
Clinical training (8 0 minutes)	1. Patients are curated together with the teacher 2. Students are assigned to each patient and they curate independently.	Students record the results of patient analysi and curation results in a notebook
Y akuniy bos q ich (10 da q i q a)	1. Concludes 2. Provides independent work	He listens Takes notes

	3. Gives homework	

Practical training №12

O 'tkir and chronic pielonefritlarni symptom-matologiyasi. Acute and chronic renal failure

1.1. Technological model of practical training .

Class time is 4 p.m.	Number of students up to 10
Place of training	An auditorium equipped with computers, multimedia and posters
Form of training	Practical training
Practical training plan	O 'tkir and chronic pielonefritlarni symptom-matologiyasi. Acute and chronic renal failure . Methods of examination of patients, interrogation, examination, palpation, percussion, laboratory and instrumental examination
The purpose of practical training	To teach students methods of examination of patients, interrogation, examination, palpation, percussion, laboratory and instrumental examination
Teaching style	Inquiry, performance of practical skills, curation of patients
Form of teaching	Divide into small groups
Training equipment	Educational-methodical complex, Textbook, multimedia, posters, model
Evaluation procedure	On a 100-point scale with interactive games, assessment, Wenn diagram, problem solving and tests

1.2. Technological map of practical training .

Stages and timing of work	Educator	Learners
Preparatory stage (5 minutes)	1. The cleanliness of the auditorium is controlled 2. Student attendance is checked 3. To check the readiness of students for lessons	Be prepared to hear
O Training o ' the process of taking steps (1 0 minutes)	1. Subject Expert liability 2. Preparation of presentation slides for the introductory lecture, preparation of the computer 3. Putting multimedia, videos 4. Develop a list of references used in the study of science	Students are prepared
Theoretical training (Qiq a at 55)	1. Students are divided into small groups and given questions on the topic Assessment, Wenn diagram and bq. 2. Demonstration posters are used 3. Slides, multimedia are used	A small group of the b h o ' Linas Q watch iladilar q participate t listen and answ

	4. Summarizes and summarizes the information provided on the basis of the topics, encourages and actively evaluates the active participant students	questions
Clinical training (8 0 minutes)	1. Patients are curated together with the teacher 2. Students are assigned to each patient and they curate independently.	Students record the results of patient analysi and curation results in a notebook
Y akuniy bos q ich (10 da q i q a)	1. Concludes 2. Provides independent work 3. Gives homework	He listens Takes notes

Practical training №13

Musculoskeletal and connective tissue system. Methods of examination: interrogation, methods of physical examination, methods of laboratory-instrumental examination. Basic clinical syndromes.

1.1. Technological model of practical training .

Class time is 4 p.m.	Number of students up to 10
Place of training	An auditorium equipped with computers, multimedia and posters
Form of training	Practical training
Practical training plan	Musculoskeletal and connective tissue system. Methods of examination: interrogation, methods of physical examination, methods of laboratory-instrumental examination. Basic clinical syndromes . Methods of examination of patients, interrogation, examination, palpation, percussion, laboratory and instrumental examination
The purpose of practical training	To teach students methods of examination of patients, interrogation, examination, palpation, percussion, laboratory and instrumental examination
Teaching style	Inquiry, performance of practical skills, curation of patients
Form of teaching	Divide into small groups
Training equipment	Educational-methodical complex, Textbook, multimedia, posters, model
Evaluation procedure	On a 100-point scale with interactive games, assessment, Wenn diagram, problem solving and tests

1.2. Technological map of practical training .

Stages and timing of work	Educator	Learners
Preparatory stage (5 minutes)	1. The cleanliness of the auditorium is controlled 2. Student attendance is checked	Be prepared to hear

	3. To check the readiness of students for lessons	
O Training o ' the process of taking steps (1 0 minutes)	1. Subject Expert liability 2. Preparation of presentation slides for the introductory lecture, preparation of the computer 3. Putting multimedia, videos 4. Develop a list of references used in the study of science	Students are prepared
Theoretical training (Qiq a at 55)	1. Students are divided into small groups and given questions on the topic Assessment, Wenn diagram and bq. 2. Demonstration posters are used 3. Slides, multimedia are used 4. Summarizes and summarizes the information provided on the basis of the topics, encourages and actively evaluates the active participant students	A small group of the b h o ' Linas Q watch iladilar q participate t listen and answ questions
Clinical training (8 0 minutes)	1. Patients are curated together with the teacher 2. Students are assigned to each patient and they curate independently.	Students record the results of patient analysi and curation results in a notebook
Y akuniy bos q ich (10 da q i q a)	1. Concludes 2. Provides independent work 3. Gives homework	He listens Takes notes

Practical training №14

Endocrine glands and metabolic system. Control methods. Inquiry, physical examination methods. Methods of laboratory and instrumental examination

1.1. Technological model of practical training .

Class time is 4 p.m.	Number of students up to 10
Place of training	An auditorium equipped with computers, multimedia and posters
Form of training	Practical training
Practical training plan	Endocrine glands and metabolic system. Control methods. Inquiry, physical examination methods. Methods of laboratory and instrumental examination . Methods of examination of patients, interrogation, examination, palpation, percussion, laboratory and instrumental examination
The purpose of practical training	To teach students methods of examination of patients, interrogation, examination, palpation, percussion, laboratory and instrumental examination
Teaching style	Inquiry, performance of practical skills, curation of patients
Form of teaching	Divide into small groups
Training equipment	Educational-methodical complex, Textbook,

	multimedia, posters, model
Evaluation procedure	On a 100-point scale with interactive games, assessment, Wenn diagram, problem solving and tests

1.2. Technological map of practical training .

Stages and timing of work	Educator	Learners
Preparatory stage (5 minutes)	1. The cleanliness of the auditorium is controlled 2. Student attendance is checked 3. To check the readiness of students for lessons	Be prepared to hear
O Training o ' the process of taking steps (1 0 minutes)	1. Subject Expert liability 2. Preparation of presentation slides for the introductory lecture, preparation of the computer 3. Putting multimedia, videos 4. Develop a list of references used in the study of science	Students are prepared
Theoretical training (Qiq a at 55)	1. Students are divided into small groups and given questions on the topic Assessment, Wenn diagram and bq. 2. Demonstration posters are used 3. Slides, multimedia are used 4. Summarizes and summarizes the information provided on the basis of the topics, encourages and actively evaluates the active participant students	A small group of the b h o ' Linas Q watch iladilar q participate t listen and answ questions
Clinical training (8 0 minutes)	1. Patients are curated together with the teacher 2. Students are assigned to each patient and they curate independently.	Students record the results of patient analysi and curation results in a notebook
Y akuniy bos q ich (10 da q i q a)	1. Concludes 2. Provides independent work 3. Gives homework	He listens Takes notes

Practical training №15

Diseases of the endocrine glands and metabolic system

1.1. Technological model of practical training .

Class time is 4 p.m.	Number of students up to 10
Place of training	An auditorium equipped with computers, multimedia and posters
Form of training	Practical training
Practical training plan	Diseases of the endocrine glands and metabolic system . Methods of examination of patients, interrogation, examination, palpation, percussion, laboratory and instrumental examination
The purpose of practical training	To teach students methods of examination of patients, interrogation, examination, palpation, percussion, laboratory and instrumental

	examination
Teaching style	Inquiry, performance of practical skills, curation of patients
Form of teaching	Divide into small groups
Training equipment	Educational-methodical complex, Textbook, multimedia, posters, model
Evaluation procedure	On a 100-point scale with interactive games, assessment, Wenn diagram, problem solving and tests

1.2. Technological map of practical training .

Stages and timing of work	Educator	Learners
Preparatory stage (5 minutes)	1. The cleanliness of the auditorium is controlled 2. Student attendance is checked 3. To check the readiness of students for lessons	Be prepared to hear
O Training o ' the process of taking steps (1 0 minutes)	1. Subject Expert liability 2. Preparation of presentation slides for the introductory lecture, preparation of the computer 3. Putting multimedia, videos 4. Develop a list of references used in the study of science	Students are prepared
Theoretical training (Qiq a at 55)	1. Students are divided into small groups and given questions on the topic Assessment, Wenn diagram and bq. 2. Demonstration posters are used 3. Slides, multimedia are used 4. Summarizes and summarizes the information provided on the basis of the topics, encourages and actively evaluates the active participant students	A small group of the b h o ' Linas Q watch iladilar q participate t listen and answ questions
Clinical training (8 0 minutes)	1. Patients are curated together with the teacher 2. Students are assigned to each patient and they curate independently.	Students record the results of patient analysi and curation results in a notebook
Y akuniy bos q ich (10 da q i q a)	1. Concludes 2. Provides independent work 3. Gives homework	He listens Takes notes

Practical training №16

Blood system. Control methods. Methods of physical examination. Methods of laboratory and instrumental examination

1.1. Technological model of practical training .

Class time is 4 p.m.	Number of students up to 10
Place of training	An auditorium equipped with computers,

	multimedia and posters
Form of training	Practical training
Practical training plan	Blood system. Control methods. Methods of physical examination. Methods of laboratory and instrumental examination . Methods of examination of patients, interrogation, examination, palpation, percussion, laboratory and instrumental examination
The purpose of practical training	To teach students methods of examination of patients, interrogation, examination, palpation, percussion, laboratory and instrumental examination
Teaching style	Inquiry, performance of practical skills, curation of patients
Form of teaching	Divide into small groups
Training equipment	Educational-methodical complex, Textbook, multimedia, posters, model
Evaluation procedure	On a 100-point scale with interactive games, assessment, Wenn diagram, problem solving and tests

1.2. Technological map of practical training .

Stages and timing of work	Educator	Learners
Preparatory stage (5 minutes)	1. The cleanliness of the auditorium is controlled 2. Student attendance is checked 3. To check the readiness of students for lessons	Be prepared to hear
O Training o ' the process of taking steps (1 0 minutes)	1. Subject Expert liability 2. Preparation of presentation slides for the introductory lecture, preparation of the computer 3. Putting multimedia, videos 4. Develop a list of references used in the study of science	Students are prepared
Theoretical training (Qiq a at 55)	1. Students are divided into small groups and given questions on the topic Assessment, Wenn diagram and bq. 2. Demonstration posters are used 3. Slides, multimedia are used 4. Summarizes and summarizes the information provided on the basis of the topics, encourages and actively evaluates the active participant students	A small group of the b h o ' Linas Q watch iladilar q participate t listen and answ questions
Clinical training (8 0 minutes)	1. Patients are curated together with the teacher 2. Students are assigned to each patient and they curate independently.	Students record the results of patient analys and curation results in a

		notebook
Y akuniy bos q ich (10 da q i q a)	1. Concludes 2. Provides independent work 3. Gives homework	He listens Takes notes

Practical training №17

Diseases of the circulatory system . Anemias

1.1. Technological model of practical training .

Class time is 4 p.m.	Number of students up to 10
Place of training	An auditorium equipped with computers, multimedia and posters
Form of training	Practical training
Practical training plan	Diseases of the circulatory system . Anemias. Methods of examination of patients, interrogation, examination, palpation, percussion, laboratory and instrumental examination
The purpose of practical training	To teach students methods of examination of patients, interrogation, examination, palpation, percussion, laboratory and instrumental examination
Teaching style	Inquiry, performance of practical skills, curation of patients
Form of teaching	Divide into small groups
Training equipment	Educational-methodical complex, Textbook, multimedia, posters, model
Evaluation procedure	On a 100-point scale with interactive games, assessment, Wenn diagram, problem solving and tests

1.2. Technological map of practical training .

Stages and timing of work	Educator	Learners
Preparatory stage (5 minutes)	1. The cleanliness of the auditorium is controlled 2. Student attendance is checked 3. To check the readiness of students for lessons	Be prepared to hear
O Training o ' the process of taking steps	1. Subject Expert liability 2. Preparation of presentation slides for the introductory lecture, preparation of the computer 3. Putting multimedia, videos	Students are prepared

(1 0 minutes)	4. Develop a list of references used in the study of science	
Theoretical training (Qiq a at 55)	1. Students are divided into small groups and given questions on the topic Assessment, Wenn diagram and bq. 2. Demonstration posters are used 3. Slides, multimedia are used 4. Summarizes and summarizes the information provided on the basis of the topics, encourages and actively evaluates the active participant students	A small group of the b h o ' Linas Q watch iladilar q participate t listen and answ questions
Clinical training (8 0 minutes)	1. Patients are curated together with the teacher 2. Students are assigned to each patient and they curate independently.	Students record the results of patient analysi and curation results in a notebook
Y akuniy bos q ich (10 da q i q a)	1. Concludes 2. Provides independent work 3. Gives homework	He listens Takes notes

Procedure for performing practical skills

A financial skill № 1

Execution order standard.

№	*Sequence of actions (actions to be performed step by step):*	*Points*
1.	The student sterilizes the probe	6
2.	The patient is explained the purpose of the treatment, its safety	6
3.	The patient sits firmly on a chair, his back	6
4.	A togora or bucket is poured under his feet	6
5.	The student moves to the right side of the patient	6
6.	The patient's chest is covered with a cloth or apron	6
7.	Before sending the probe, the distance from the umbilicus to the molars is measured in each patient, the probe is lost.	6
8.	The patient opens his mouth wide, makes a "a" sound, and takes a deep breath through his nose	6
9.	The student quickly inserts the probe into the back of the root of the tongue	6
10.	The patient closes his mouth and makes several swallowing movements	6

11.	A 20 g syringe is placed on the bush end of the probe	6
12.	Gastric fluid is added to solution 4 at 15-minute intervals over 1 hour	6
13.	Another 200 ml of bone marrow is injected through the probe or the patient is given pentagastrin	7
14.	The gastric fluid is then injected into the 4th test tube every 15 minutes for one hour	7
15 .	The obtained gastric juice is taken in 8 test tubes and transferred to the laboratory	7
16.	At the end of the treatment, the tube is slowly pulled out	7
Total :		100

A financial skill № 2

Gastric lavage technique

Required equipment: gastric tube, tube, oilcloth, funnel, distilled water, 20 g syringe

	Movements k e bac k e tligi (which should be completed Kadambay step movement
1.	The patient is transferred to a chair, his ashes are tied to the back of the chair with sheet, and an apron is placed on his chest, and the nurse and junior nurse also hold apron.
2.	Standing on the patient's side, we first measure the length of the probe falling into medulla. To do this, the shovel is measured from the navel to the navel, adding 5 c to it.
3.	The patient is instructed to tilt his head back slightly and open his mouth, or open with a mouth opener, with his chest slightly bent forward.
4.	Holding the oval-shaped tip of the sterile probe in the ashes, the probe is moistene with marrow water, burned to the root of the patient's tongue, and instructed to swallow and take a deep breath through the nose.
5.	Yutinayotganda the patient , the tube should be gradual assumption with me , if th patient goes to bed , difficult breathing , Cook immediately intra need to get away from . This is what remains of the hall into the trachea tube or breathing Julius xikkildok lifetimes .
6.	Free end of the tube after the character you want , put the funnel , so the patient's knee tushiridadi crater at me ' juice drainage procedures , if developed , i xarakatlantiriladi .
7.	In this case penetrate a little kiyshaytirib , 1 liter of water simultaneously .
8.	The funnel is gradually raised until there is water at the bottom, the funnel is lowe and water begins to flow from it. Water from the stomach should be equal to burn

	water. If there is less water, the patient is asked to strain a little.
9.	This movement is repeated until clear water comes out of the funnel, ie the stoma
10.	Wash Me ' Daniel bulgach , the funnel can be taken away , intra- me ' Dad , thoroughly washed with distilled water and sewage sterilize the water to boil for 30 minutes .
Total :	

A financial skill № 3

Indicate the preparation of patients for X-ray examination of the digestive tract.

№	*Movements k f - k e tligi (Kadambay heating step which should be completed movements):*	*Poir*
1.	A strict diet is followed	10
2.	The intestines are free of fluid and gas	10
3.	The patient is prescribed easily digestible foods	10
4.	The patient is given chamomile tincture for up to three days	10
5.	The day before, the patient is forbidden to eat from 20 o'clock at night	15
6.	A cleansing enema will be done at 10 p.m.	15
7.	Gas transmission pipe for the collection of gas in the intestines simultaneously	15
8.	In the morning, the patient is given another cleansing enema	15
Total :		100

A financial skill № 4

Demonstrate duodenal probing technique

Required equipment: Duodenal tube, tube, oilcloth, distilled water, funnel

Execution order standard.

№	*Sequence of actions (actions to be performed step by step):*
1.	The patient is placed in a chair
2.	Towels are held in front of the patient on the legs and knees

3.	The patient psychologically prepared (deep breath and worry , the nurses said patient heavy case is assigned)
4.	The patient is instructed to open his mouth as “aaa” and stick out his tongue
5.	The patient is instructed to open his mouth as “aaa” and stick out his tongue
6.	The patient is instructed to close his mouth and swallow slowly without biting th
7.	It takes the patient to swallow this probe up to 3 characters, i.e. 20-30-45 minutes
8.	The patient is placed on a couch with his head down (without a pillow)
9.	A hard object is placed under the patient's right rib and a bone marrow burn is pe
10.	The duodenal tube is inserted into the 1st test tube on a tripod (the test tubes are l patient). The 1st portion is taken 12 fingers intestinal fluid
11.	Then the patient is placed on his back, 50-60 ml of 33% magnesium sulfate solut from the tip of the probe and the tip of the probe is cut for 10 minutes.
12.	Remove the pinch and collect the liquid from the gallbladder in test tube 2
13.	Depending on the color change of the liquid, the probe is transferred from the 2n 3rd test tube, ie the 3rd portion is collected.
14.	When the outflow of fluid from the tip of the duodenal tube is stopped, the patien transferred to a chair, and the probe is slowly pulled out, the resulting fluid is sen examination (laboratory).

A financial skill № 5

Show it as a cleansing enema.

Required equipment:

1. Esmarch mug, 2. Rubber tube with spout, 3. Vaseline, 4. Trinity, 5. Togora
6. Ilik suv, 7. Kleyonka, 8. Kushetka, 9. Thermometer

Execution order standard.

№	*Sequence of actions (actions to be performed step by step):*	*Points*
1.	Having prepared the necessary things, the patient pours it next to the couch	7
2.	The Esmarch mug is filled with marrow water, water 1-2 l , at 37-38 degrees	7
3.	Eroded into the air in the tube down to the end of the fieldworks , Esmarx cup in the exaltation of the queue	7
4.	When the air is expelled, the sung chumrak is closed	7
5.	Kleyonka falls on the couch	7

6.	The patient is placed on a couch, close to the edge, with the left side bent at the knees and pulled to the cornea.	7
7.	Sterile Vaseline is applied thinly on the tube	7
8.	The patient's buttocks are inserted using the left gray fingers, Vaseline is applied to the posterior exit hole	7
9.	Trinity spinal output port and the exaltation of 8-10 cm forward, karate , then tried to aim a little back , working with the	7
10.	The cup opens, and the Esmarch cup rises	7
11.	When there is a little water left in the mug, the tap closes	8
12.	The trio is removed from the rear outlet hole	6
13.	The patient goes to the toilet in a few more minutes and empties the bowels	8
14.	The trinity is separated from the rubber tube and unloaded	8
Total :		100

A financial skill № 6

Demonstrate the technique of liver percussion (according to Kurlov)

Execution order standard.

	Movements k e bac k e tligi (which should be completed Kadambay step movements):	*Points*
1.	The end of the patient's case until the middle Umran line on the PCI sound tumtoklashganga percussion short .	10
2.	This comes down to 6 ribs.	10
3.	Bottom line on the hub about the same until the tympanic percutaneous tumtoklashgunga the sound of percussion will be disqualified .	10
4.	This comes down to 10 ribs.	10
5.	1 vertical line of the staircase and the body moved to the front midline about drawing point cut is organized simultaneously .	15
6.	Depending on the exaltation of the umbilicus from the bottom until the cranial volume tumtoklashgunga percussion will be disqualified .	15

7.	This corresponds to the upper 3 to 1 part of the area between the umbilical cord and the umbilical cord.	15
8.	percussion is performed along the arch of the left rib cage from the area of the 10th rib cage to the dull sound.	15
9.	Normally, size 1 should be 10-12, size 2 8-10, size 3 5-7 cm.	
Total :		100

A financial skill № 7

Technique of examination of urine by Zimnistky method.
Required equipment: 8 clean jars, blanks

Execution order standard.

№	*Sequence of actions (actions to be performed step by step):*	*Points*
1.	Prepared in 8 clean jars	14
2.	A referral is written to each bank	14
3.	The patient empties his bladder at 6 a.m., which causes the urine to drain	14
4.	A 3-hour break is given, and the first portion of urine is taken at 6-9 p.m.	14
5.	The patient also empties the bladder 8 times a day	14
6.	The next portion is assembled at 6 o'clock the next morning	14
7.	8 portions of urine are sent to the laboratory	16
Total :		100

A financial skill № 8

Demonstrate the technique of testing urine by the Addis-Kakovsky method.
Required equipment: 3 clean jars, blanks

Execution order standard.

№	*Sequence of actions (actions to be performed step by step):*	*Points*
1.	The patient's external genitalia are washed	10
2.	A clean container for collecting urine should be larger	10
3.	Before collecting the urine, the container is filled with a preservative: a few drops of thymol crystals or a drop of farmalkdehyde	10
4.	Urine should be collected within three hours	10
5.	At 10 p.m., the patient empties the bladder	15

6.	Urine is collected from 10 pm to 8 am	15
7.	At 8 a.m., urine is collected and sent to the laboratory	15
8.	Substances that react to the patient	15
	Total :	100

Independent study topics

Independent work 1

Causes of development of dysphagia, risk factors etiology, pathogenesis, mechanism of pain formation.

1) Explain dysphagia
2) Causes of the development of dysphagia
3) Dysphagia x avf factors
4) Dysphagia etiology
5) Pathogenesis of dysphagia,
6) Disfagiya pain formation mechanism
7) Internal diseases that cause dysphagia
8) Diagnosis of dysphagia
9) Methods of diagnosis of dysphagia

The name of the topic	Ball	Baho	The level of knowledge of the student
T he medical deontology, euthanasia problems, iatrogeny.	86-100	A'lo	Able to draw conclusions and decisions, think creatively, observe independently, apply in practice, explain the essence, know, tell , have imagination.
	71-85	Good	Can observe independently, apply in practice, explain the essence, know, tell, have imagination.
	55-70	It's snowing	Explains the essence, knows, can tell, has imagination.
	0-54	Bloodless	He has no imagination, he does not know.

Topic 1

Independent work 2

Clinical manifestations of wound disease, clinical-laboratory criteria, complications and methods of treatment.

1) The concept of wound disease (tariff)
2) Clinical manifestations of ulcer disease
3) Etiology of ulcer disease
4) The mechanism of development of wound disease
5) ulcer disease clinical asi
6) Examination of patients with wound disease
7) Palpation of patients with wound disease
8) Laboratory examination of patients with wound disease
9) Examination of faeces by Gregerson method
10) radiological examination of patients with wound

11) Endoscopic examination of patients with wound disease

The name of the topic	Ball	Baho	The level of knowledge of the student
Comparative diagnosis of primary and secondary respiratory noise . Pneumonia	86-100	A'lo	Able to draw conclusions and decisions, think creatively, observe independently, apply in practice, explain the essence, know, tell, have imagination.
	71-85	Good	Can observe independently, apply in practice, explain the essence, know, tell, have imagination.
	55-70	It's snowing	Explains the essence, knows, can tell, has imagination.
	0-54	Bloodless	He has no imagination, he does not know.

Independent work 3

Etiology, pathogenesis, and classification of pancreatitis. Diagnostic tests for pancreatic tumors.

1 . Pancreatitis disease concept of the (tariff)
2. The clinical manifestations of the disease AP
3. pancreatic disease etiology
4. Development Mechanism, the diagnosis of acute pancreatitis
5. The clinical diagnosis of acute pancreatitis asi
6. Examination of patients with the diagnosis of acute pancreatitis
7. Palpation of patients with pancreatitis
8. The laboratory examination of patients with the diagnosis of acute pancreatitis
9. The diagnostic value of fecal examination in pancreatitis
10. The radiological examination of patients with the diagnosis of acute pancreatitis
11. The endoscopic examination of patients with the diagnosis of acute pancreatitis

Topic 3

The name of the topic	Ball	Baho	The level of knowledge of the student
Pleurisy. Bronchial asthma	86-100	A'lo	Able to draw conclusions and decisions, think creatively, observe independently, apply in practice, explain the essence, know, tell, have imagination.

	71-85	Good	Can observe independently, apply in practice, explain the essence, know, tell, have imagination.
	55-70	It's snowing	Explains the essence, knows, can tell, has imagination.
	0-54	Bloodless	He has no imagination, he does not know.

Independent work 4

Complications of nonspecific ulcerative colitis and diagnostic examination of the intestine.

1. The concept of non- specific ulcerative colitis (tariff)
2 of nonspecific ulcerative colitis disease diagnosis diferentsial
3. nonspecific ulcerative colitis disease aetiology
4 of nonspecific ulcerative colitis disease development mechanism
5. nonspecific ulcerative colitis disease clinical asi
6. Examination of patients with nonspecific ulcerative colitis
7. nonspecific ulcerative colitis disease patients acoustical
8. nonspecific ulcerative colitis disease patients with laboratory research
9. Diagnostic significance of fecal examination in nonspecific ulcerative colitis
10. nonspecific ulcerative colitis patients with radiographic evidence of disease
11. nonspecific ulcerative colitis disease patients endoscopic examination

Topic 4

The name of the topic	Ball	Baho	The level of knowledge of the student
Comparative diagnosis of organic and functional interactions	86-100	A'lo	Able to draw conclusions and decisions, think creatively, observe independently, apply in practice, explain the essence, know, tell, have imagination.
	71-85	Good	Can observe independently, apply in practice, explain the essence, know, tell, have imagination.
	55-70	It's snowing	Explains the essence, knows, can tell, has imagination.
	0-54	Bloodless	He has no imagination, he does not know.

Independent work 5

Etiopathogenesis of diseases associated with hepatomegaly, clinical manifestations, syndromic diagnosis of the disease .

1 . The concept of hepatomegaly
2. Diseases associated with hepatomegaly
3. Etiology of hepatomegaly
4 Mechanism of development of hepatomegaly
5. Hepatomegaly clinical asi
6 Examination of patients with hepatomegaly
7 Palpation of patients with hepatomegaly
8. hepatomegalia laboratory research

9. Instrumental examination of patients with hepatomegaly
10. Radioisotope examination of patients with hepatomegaly

Topic 5

The name of the topic	Ball	Baho	The level of knowledge of the student
Heart Defects Changes in heart rate norm and pathology	86-100	A'lo	Able to draw conclusions and decisions, think creatively, observe independently, apply in practice, explain the essence, know, tell, have imagination.
	71-85	Good	Can observe independently, apply in practice, explain the essence, know, tell, have imagination.
	55-70	It's snowing	Explains the essence, knows, can tell, has imagination.
	0-54	Bloodless	He has no imagination, he does not know.

Independent work 6

Etiopathogenesis, clinical manifestations, and diagnosis of liver cirrhosis.

1 . The concept of the etiopathogenesis of liver cirrhosis (tariff)
2. Clinical manifestations of liver cirrhosis
3. Etiology of liver cirrhosis
4. The mechanism of development of liver cirrhosis
5. liver cirrhosis clinical asi
6. Examination of patients with liver cirrhosis
7. Palpation of patients with liver cirrhosis
8 Liver cirrhosis , laboratory examination of patients
9. Diagnostic significance of liver cirrhosis , stool examination
10. Liver cirrhosis Liver palpation, percussion

Topic 6

The name of the topic	Ball	Baho	The level of knowledge of the student
Symptomatology of bacterial endocarditis	86-100	A'lo	Able to draw conclusions and decisions, think creatively, observe independently, apply in practice, explain the essence, know, tell, have imagination.
	71-85	Good	Can observe independently, apply in practice, explain the essence, know, tell, have imagination.
	55-70	It's snowing	Explains the essence, knows, can tell, has imagination.
	0-54	Bloodless	He has no imagination, he does not know.

Independent work 7

Etiopathogenesis of jaundice, clinical manifestations, syndromic diagnosis of the disease.

1 . The concept of the etiopathogenesis of jaundice (tariff)
2. Clinical manifestations of jaundice
3. Etiology of jaundice
4. The mechanism of development of jaundice
5. Jaundice clinical asi
6. Examination of jaundice patients
7. Palpation of patients with jaundice
8 Jaundice , laboratory examination of patients
9. The diagnostic value of jaundice stool examination
10. Jaundice liver palpation, percussion
11. Parenchymal jaundice
12. adrenal jaundice
13. submandibular jaundice

Topic 7

The name of the topic	Ball	Baho	The level of knowledge of the student
Symptomatology of postinfarction cardiosclerosis	86-100	A'lo	Able to draw conclusions and decisions, think creatively, observe independently, apply in practice, explain the essence, know, tell, have imagination.
	71-85	Good	Can observe independently, apply in practice, explain the essence, know, tell, have imagination.
	55-70	It's snowing	Explains the essence, knows, can tell, has imagination.
	0-54	Bloodless	He has no imagination, he does not know.

Independent work 8

Etiology, pathogenesis, clinical manifestations, diagnosis and activity criteria of inflammatory and immune inflammatory diseases of the kidneys.

1 . The concept of the etiopathogenesis of inflammation of the kidneys (tariff)
2. Clinical manifestations of inflammation of the kidneys
3. The etiology of inflammation of the kidneys
4. The mechanism of development of inflammation of the kidneys
5. kidney inflammation clinical asi
6. Examination of patients with inflammation of the kidneys
7. Palpation of patients with inflammation of the kidney
8 Inflammation of the kidneys , laboratory examination of patients
9. I immune inflammatory etiology, pathogenesis, kasalliknin clinical manifestations, diagnosis and asset values.
10. Inflammatory palpation of the kidney , percussion

Topic 8

The name of the topic	Ball	Baho	The level of knowledge of the student

Symptomatology of dilated cardiomyopathy	86-100	A'lo	Able to draw conclusions and decisions, think creatively, observe independently, apply in practice, explain the essence, know, tell, have imagination.
	71-85	Good	Can observe independently, apply in practice, explain the essence, know, tell, have imagination.
	55-70	It's snowing	Explains the essence, knows, can tell, has imagination.
	0-54	Bloodless	He has no imagination, he does not know.

Independent work 9

Etiology, pathogenesis and methods of diagnosis of nephropathy.

1. Etiology of nephropathy
2. Pathogenesis of nephropathy
3. Methods of diagnosis of nephropathy.
4. Clinical manifestations of nephropathy
5. Examination of patients with nephropathy
6. Palpation of patients with nephropathy
7 Laboratory examination of patients with nephropathy
8. nephropathy importance of instrumental investigation diagnastik
9. Methods of diagnosis of nephropathy.

Topic 9

The name of the topic	Ball	Baho	The level of knowledge of the student
Ischemic heart disease	86-100	A'lo	Able to draw conclusions and decisions, think creatively, observe independently, apply in practice, explain the essence, know, tell, have imagination.
	71-85	Good	Can observe independently, apply in practice, explain the essence, know, tell, have imagination.
	55-70	It's snowing	Explains the essence, knows, can tell, has imagination.
	0-54	Bloodless	He has no imagination, he does not know.

Independent work 10

Drugs used in the etiology, pathogenesis, classification, clinical syndromes, diagnosis and treatment of chronic renal failure

1 . The concept of the etiopathogenesis of chronic renal failure (tariff)
2. Clinical manifestations of chronic renal failure
3. Etiology of chronic renal failure
4. The mechanism of development of chronic renal failure
5. Chronic kidney failure clinical asi
6. Review of patients with chronic renal failure

7. Palpation of patients with chronic renal failure
8. Laboratory examination of patients with chronic renal failure
9. The diagnostic value of instrumental examination of chronic renal failure
10. Palpation, percussion of chronic renal failure
11. The diagnostic value of urinalysis in chronic renal failure

The name of the topic	Ball	Baho	The level of knowledge of the student
Ischemic heart disease	86-100	A'lo	Able to draw conclusions and decisions, think creatively, observe independently, apply in practice, explain the essence, know, tell, have imagination.
	71-85	Good	Can observe independently, apply in practice, explain the essence, know, tell, have imagination.
	55-70	It's snowing	Explains the essence, knows, can tell, has imagination.
	0-54	Bloodless	He has no imagination, he does not know.

Independent work 11

Etiology, pathogenesis, clinical manifestations and diagnosis of tumor syndrome.

1 . The concept of tumor syndrome (tariff)
2. Clinical manifestations of tumor syndrome
3. Etiology of tumor syndrome
4. The mechanism of development of tumor syndrome
5. The tumor syndrome asi
6. Examination of patients with tumor syndrome
7. Palpation of patients with tumor syndrome
8. Laboratory examination of patients with tumor syndrome
9. Diagnostic significance of fecal examination of tumor syndrome
10. Palpation, percussion of the tumor syndrome

The name of the topic	Ball	Baho	The level of knowledge of the student
Ischemic heart disease	86-100	A'lo	Able to draw conclusions and decisions, think creatively, observe independently, apply in practice, explain the essence, know, tell, have imagination.
	71-85	Good	Can observe independently, apply in practice, explain the essence, know, tell, have imagination.
	55-70	It's snowing	Explains the essence, knows, can tell, has imagination.
	0-54	Bloodless	He has no imagination, he does not know.

Independent work 12

Diagnostic criteria for rheumatism and rheumatoid arthritis.

1 . The concept of rheumatism and rheumatoid arthritis (tariff)
2. Clinical manifestations of rheumatism and rheumatoid arthritis
3. Etiology of rheumatism and rheumatoid arthritis
4. The mechanism of development of rheumatism and rheumatoid arthritis
5. rheumatism and rheumatoid arthritis clinical asi
6. Review of patients with rheumatism and rheumatoid arthritis
7. Palpation of patients with rheumatism and rheumatoid arthritis
8. Laboratory examination of patients with rheumatism and rheumatoid arthritis
9. Diagnostic significance of fecal examination for rheumatism and rheumatoid arthritis
10. Palpation, percussion of rheumatism and rheumatoid arthritis

The name of the topic	Ball	Baho	The level of knowledge of the student
Ischemic heart disease	86-100	A'lo	Able to draw conclusions and decisions, think creatively, observe independently, apply in practice, explain the essence, know, tell, have imagination.
	71-85	Good	Can observe independently, apply in practice, explain the essence, know, tell, have imagination.
	55-70	It's snowing	Explains the essence, knows, can tell, has imagination.
	0-54	Bloodless	He has no imagination, he does not know.

Independent work 13

Diagnostic criteria of seronegative spondyloarthritis (reactive arthritis, Bexterev's disease, psoriatic arthritis).

1 . Reactive arthritis, Bexterev's disease, psoriatic arthritis concept (tariff)
2. Clinical manifestations of reactive arthritis, Bexterev's disease, psoriatic arthritis
3. The etiology of reactive arthritis, Bexterev's disease, psoriatic arthritis
4. Mechanism of development of reactive arthritis, Bexterev's disease, psoriatic arthritis
5. reactive arthritis, Bexterev disease, psoriatic arthritis clinical asi
6. Examination of patients with reactive arthritis, Bexterev's disease, psoriatic arthritis
7. Palpation of patients with reactive arthritis, Bexterev's disease, psoriatic arthritis
8. Laboratory examination of patients with reactive arthritis, Bexterev's disease, psoriatic arthritis
9. Diagnostic value of fecal examination for reactive arthritis, Bexterev's disease, psoriatic arthritis
10. Reactive arthritis, Bexterev's disease, psoriatic arthritis palpation, percussion

The name of the topic	Ball	Baho	The level of knowledge of the student

Ischemic heart disease	86-100	A'lo	Able to draw conclusions and decisions, think creatively, observe independently, apply in practice, explain the essence, know, tell, have imagination.
	71-85	Good	Can observe independently, apply in practice, explain the essence, know, tell, have imagination.
	55-70	It's snowing	Explains the essence, knows, can tell, has imagination.
	0-54	Bloodless	He has no imagination, he does not know.

Independent work 14

Diagnostic criteria, activity levels and treatment methods of diffuse connective tissue diseases.

1 . The concept of diffuse connective tissue diseases (tariff)
2. Clinical manifestations of diffuse connective tissue diseases
3. Etiology of diffuse connective tissue diseases
4. The mechanism of development of diffuse connective tissue diseases
5. Clinical manifestations of diffuse connective tissue disease
6. Examination of patients with diffuse connective tissue diseases
7. Palpation of patients with diffuse connective tissue diseases
8. Laboratory examination of patients with diffuse connective tissue diseases
9. Intrumental examination of diffuse connective tissue diseases
10. Palpation, percussion of diffuse connective tissue diseases

The name of the topic	Ball	Baho	The level of knowledge of the student
Ischemic heart disease	86-100	A'lo	Able to draw conclusions and decisions, think creatively, observe independently, apply in practice, explain the essence, know, tell, have imagination.
	71-85	Good	Can observe independently, apply in practice, explain the essence, know, tell, have imagination.
	55-70	It's snowing	Explains the essence, knows, can tell, has imagination.
	0-54	Bloodless	He has no imagination, he does not know.

Independent work 15

Diagnostic criteria, activity levels, treatment methods and prevention of dermatomyositis and systemic vasculitis.

1 . The concept of dermatomyositis and systemic vasculitis (tariff)
2. Clinical manifestations of dermatomyositis and systemic vasculitis
3. Dermatomiozit and systemic pruritus aetiology
4. The mechanism of development of dermatomyositis and systemic vasculitis

5. Dermatomiozit and systemic pruritus clinical asi
6. Examination of patients with dermatomyositis and systemic vasculitis
7. Palpation of patients with dermatomyositis and systemic vasculitis
8. Laboratory examination of patients with dermatomyositis and systemic vasculitis
9. Diagnostic significance of instrumental examination of dermatomyositis and systemic vasculitis
10. Palpation , percussion of dermatomyositis and systemic vasculitis

The name of the topic	Ball	Baho	The level of knowledge of the student
Ischemic heart disease	86-100	A'lo	Able to draw conclusions and decisions, think creatively, observe independently, apply in practice, explain the essence, know, tell, have imagination.
	71-85	Good	Can observe independently, apply in practice, explain the essence, know, tell, have imagination.
	55-70	It's snowing	Explains the essence, knows, can tell, has imagination.
	0-54	Bloodless	He has no imagination, he does not know.

Independent work 16

Diagnostic criteria, activity levels, treatment methods and prevention of nodular periarthritis and nonspecific aortoarteritis.

1 . The concept (tariff) of nodular periarthritis and nonspecific aortoarteritis
2. Clinical manifestations of nodular periarthritis and nonspecific aortoarteritis
3. Etiology of nodular periarthritis and nonspecific aortoarteritis
4. The mechanism of development of nodular periarthritis and nonspecific aortoarteritis
5. Knots periartrit and non-clinical aortoarteriit asi
6. Examination of patients with nodular periarthritis and nonspecific aortoarteritis
7. Palpation of patients with nodular periarthritis and nonspecific aortoarteritis
8. Laboratory examination of patients with nodular periarthritis and nonspecific aortoarteritis
9. Diagnostic value of instrumental examination of nodular periarthritis and nonspecific aortoarteritis
10. Palpation , percussion of nodular periarthritis and nonspecific aortoarteritis

The name of the topic	Ball	Baho	The level of knowledge of the student
Ischemic heart disease	86-100	A'lo	Able to draw conclusions and decisions, think creatively, observe independently, apply in practice, explain the essence, know, tell, have imagination.
	71-85	Good	Can observe independently, apply in practice, explain the essence, know, tell, have imagination.

	55-70	It's snowing	Explains the essence, knows, can tell, has imagination.
	0-54	Bloodless	He has no imagination, he does not know.

Evaluation

THEMATIC RATING EVALUATION

IKP and the department of clinical pharmacology IKP science students' knowledge and assessment of students in higher educational institutions balimini Regulations on the monitoring and evaluation system, as well as medical higher educational institutions recommended by the Bureau of the ektorlari origin on the basis of the given statute.

Evaluation procedure and criteria.

Students' sa v iyasi, talent and skills of students on the basis of a rating system is characterized by the level of development of the science of IKP points.

IKP science semester student o ' z outstanding 100-point system will be evaluated .
These 100 points are distributed according to the types of control as follows :
Current assessment - 45 points ;
Intermediate assessment - 20 points;
Independent work - 5 points;
Final assessment - 30 points (15 points OSKE and 15 points test) ;
In assessing the knowledge of 3rd year students of the Faculty of Medicine and Medical Pedagogy, taking into account the specifics of teaching methods on the subject "ICP", the coefficient is used to take into account the value of DB, TMI, OB and YAB.

№	Type of assessment	Maximum score	Sorting score	coefficients
1.	Current evaluation	45	24.75	0.45
2.	TMI	5	2.75	0.05
3.	Interim evaluation	20	11.0	0.2
4.	Final evaluation	30	16.5	0.3
	TOTAL	100	55.0	1

The following standard criteria are recommended for the assessment of the student and the control of mastery of the 3rd year of medical pedagogy on the subject "IKP":

a) For 86-100 points the student's level of knowledge should correspond to :
conclusions and decision making ;
creative thinking;
to be able to observe independently;
q in practice what they learned o ' ill- ;
understanding the essence ;
to know, to tell;
to have an idea.

b) For 71-85 points, the student's level of knowledge must meet the following :
to be able to observe independently;
q in practice what they learned o ' ill- ;
understanding the essence ;
to know , to tell ;
to have imagination .
c) The level of knowledge for 55-70 points must meet the following :
understanding the essence ;
to know, to tell ;
to have imagination.
e) the student's level of knowledge should be assessed up to -54 points in the following cases :
not having a clear idea, not knowing .
In accordance with the standard criteria, the assignments of the evaluation criteria for the final control are developed by the Basic Educational Institution (TTA) in the specialty ICP, approved by the Scientific and Methodological Council of the institute and delivered to related higher education institutions.
Students' independent work on the subject of ICP is evaluated on the basis of the performance of the relevant tasks in the current, intermediate and final controls and the points allocated to it.

The student's IKP subject rating is determined :

$$R_f =$$

here:

The total workload for the subject of ICP in the V – V semester (306 hours).

- The level of mastery of the subject (in points).

In each of the current, intermediate and final controls on the subject of ICP, 55% of the allocated points are determined as qualifying points .
All students who score more than 55% of the qualifying score allocated in each of the current and intermediate controls will be included in the final control in this subject .
If a student scores higher than the qualifying score for this type of control in the final examination, this score is added to the points collected from the current and intermediate controls. Otherwise, the student is considered not mastered the subject of ICP.

Glossary

DIGESTIVE SYSTEM

Anaciditis	Absence of free hydrochloric acid in meda juice
Anorexia	Complete loss of appetite
Achilles	Absence of proteolytic enzymes and hydrochloric acid in meda juice
Axlorhydria	Absence of chloride kilos in gastric juice
Abdominal pain	Pain felt when the abdominal organs are damaged
Bryushnaya jaba	Occasional pain in the groin
Gastritis	Inflammation of the meda mucosa
Gastroptosis	The meda expands and elongates and descends
Gastroscopy	Examination of the mucous membrane of the medusa using a gastroscope
Gastroenterology	The science that studies the digestive organs
Hyperaciditis	Increased release of free chloride in meda juice
Hypersalivation	Excessive bone separation
Hypersecretion	Excessive secretion of meda juice

Gastrocardia l niy syndrome	Occurrence of congestion and pain in the heart after eating
Bolezn Gipshpringa	Constipation is the result of idiopathic enlargement and elongation of the colon
Hypoaciditis, hypochlorhydria	Decrease in free hydrochloric acid in meda juice
Hyposalivation	A small amount of bone separation
Hyposecretion	Low secretion of meda juice
Defecation	Diarrhea
Diaria	Diarrhea in liquid departure
Dyskenesia of the intestine	Impaired bowel movement
Dyspepsia brodilnaya	Bitter dyspepsia is diarrhea associated with impaired carbohydrate digestion in the gut. In this case, the patient has abdominal distension, edema in the abdomen, resulting in a liquid state and a bitter reaction, as well as an increase in plant tissue and starch granules.
Dempink syndrome	Me Are resected patients after cessation of food or feeding time, just to stay in power, the heart often clickthrough rates
Dyspepsia gnilostnaya	It is a pathological condition associated with insufficient digestion of proteins in the intestine due to the absence of hydrochloric acid in the meda juice. The reaction is characterized by diarrhea with indigestible pieces of food containing an alkaline odor,
Gastric dyspepsia	Impaired meda function, which is followed by pain, decreased appetite, wheezing, nausea, vomiting, vomiting.

Dysphagia	Difficulty in passing food through the esophagus
Duodenitis	Inflammation of the duodenum (duodenitis)
Dolixosigma	Congenital elongation of the sigmoid colon
Zapori	Constipation
Irrigoscopy	X-ray imaging of the colon
Izjoga	Heartburn
Cal	The mass that comes out of the lower part of the small intestine during respiration, defecation, deficiencies
Colitis	Inflammation of the mucous membrane of the colon
Collonoscopy	Examination of the mucous membrane of the colon using a colonoscope
Colloptosis	Dropping of the colon
Creatopia	The appearance of a large number of undigested muscle fibers in the stool
Melena	Black stools, this symptom indicates bleeding from the upper part of the intestine
Flatulence	Abdominal bloating, gas accumulation in the intestines
Pancreatitis	Inflammation of the pancreas. There are acute and chronic pancreatitis
Proctite	Inflammation of the mucous membrane of the

Peritonitis	rectum
	Inflammation of the peritoneum
Polyphagia	
	Eating too much
Toshnota	
	Nausea
Salivation (Ptializm)	
	Increased salivation
Pvota	
	Involuntary vomiting of food mixture through the esophagus (vomiting)
Recto romanasco p	
	And sigmoid colon mucosa are encouraged to test
Salivation	
	Saliva separation
Defective symptom	
	Defective symptom. In this case, an X-ray shows an image of a gastric tumor.
Symptom "nishi"	
	The “shelf” is a simon symptom. This symptom appears on X-ray in the form of a lump formed in the wall of the stomach and duodenum. It indicates that a wound has formed. It fills the stomach and duodenum with barium to diagnose the disease.
Srigivaniye	
	Quick return of food from the esophagus to the heavy cavity
Steator	
	Excess fat in the stool
Tenezmi	
	Strengthening. Inflammatory disease of the rectum. Frequent contractions of the bowel result in stiffness and pain. This symptom is

	most common in dysentery.
Fibroendoscopy	
	Examination of the mucous membrane of any internal organ using a fibroscope, marked biopsy, and imaging. Examination of the mucous membrane of the esophagus using an esophagoscope
Enteroptosis	
	Down fall into the small bowel
E n t e rit	
	Inflammation of the mucous membrane of the small intestine
Yazvennaya bolezn jeludka I dvenadsatiperstnoy kishki	
	It is a common chronic and recurrent disease, continuing with the appearance of a sore on the wall of the stomach and duodenum.

HEPATOBILIAR SYSTEM.

HEPATOBILIAR SYSTEM.

Axolichniy kal	Discoloration of feces occurs as a result of bile (bile) not entering the intestine .
	Separation of bilirubin with urine
Bilurubinuria	
	It caused damage to the central nervous system due to liver failure
Gapatargiya	
	Inflammation of the liver cells
Hepatitis	
	Radioisotope examination of the liver. This method is based on a graphical representation of the rate at which the isotope enters the liver, the rate of entry, the rate of accumulation, and

Hepatography	the rate at which the radioactive substance passes from the liver to the intestine. Bengal rose dye and labeled iodine 131 are used as radioactive substances
Hepatolienalniy syndrome	Simultaneous enlargement of the liver and spleen for various reasons. This syndrome occurs, for example, in blood diseases, hepatitis, cirrhosis, and portal vein thrombosis.
Hepatology	The science that studies diseases of the liver, gallbladder and bile ducts
Hepatomegaly	Enlargement of the liver
Hyperbilirubinemia	Increased bilirubin in the blood
Medusa head i	“Jellyfish head” is a scattering of dilated, swollen worm-shaped venous collaterals around the umbilicus. "Jellyfish head" occurs as a result of increased pressure in the vein and the anastomosis (fusion) of the portal vein network with the hollow veins. Medusa head is found in liver cirrhosis.
Dyskenesia	Impaired motor function of the gallbladder and bile ducts. There may be hypermotor and hypomotor dyskinesia
Interus	As a result of increased levels of bilirubin in the blood, the skin and mucous membranes turn yellow.
Bile ducts o bturasia	Blockage of bile ducts
Puncture biopsy	Examination of the structure of the liver. In this method, Vim-Silverman took a small piece of liver tissue with a Mengini needle and examined it under a microscope.
Portal hypertension	Increased blood pressure in the portal vein

Scanning cookies	Examination of the structure and functional status of the liver using a scanner (or gammatopograph). 198 and bengal roses were first used as radioisotopes.

MOCHEVIDELITELNAYA SYSTEM

URINARY SEPARATION SYSTEM

Azotemia	Increased nitrogen waste in the blood
Nitrogen slag	The final products of nitrogen (protein metabolism) excreted from the body along with urine
Anuria	Incomplete urination (anuria)
Amyloidosis	In the intermediate substances of the iron walls accumulate amyloid masses, which are composed of a special protein rich in diamond acids. These masses are formed as a result of severe disruption of protein metabolism. Amyloid kidneys appear in the form of enlarged, shiny, pale yellow. Renal amyloidosis often persists with amyloidosis of

	other organs (liver, spleen, intestines) (amyloidosis)
Acetonuria	Occurrence of ketone bodies in urine. These include acetone, acetoacetic acid, beta-oxymoic acid. Acetonuria is a symptom of diabetic coma.
Hematuria	Urinary excretion of blood (hematuria)
Hemodialysis	Purification of blood from nitrogen waste (hemodialysis)
Hyperstenuria	Increased specific gravity of urine (more than 1026) (hyperstenuria)
Hypostenuria	Decreased urinary specific gravity (less than 1015) (hypostenuria)
Hydronephrosis	Fluid in the kidneys due to obstruction of the urinary tract
	accumulation
Stranguria	Painful urination
Glycosuria	Kanal D with development of hardware damage caused B, urine glucose ajaralishi .
Diuresis	The amount of urine excreted over a period of time (daily diuresis normally varies from 1 to 2 l) diuresis)
Isosthenuria	Variable, almost identical specific gravity (isosthenuria) in different portions of urine equal to the specific gravity of blood plasma
Dysuria	Disorders of urinary excretion
A nuriya	Urinary retention (anuria) is an inflammation of the renal glomerular apparatus of infectious-allergic origin .
N efrit	Nephritis is accompanied by albuminuria, hematuria, hypoproteinemia, hypertension and edema (nephritis)

N efroz	Dystrophic changes of the renal tubular epithelium (nephrosis)
N ephralogy	The study of kidney disease (nephrology)
Nephrosclerosis	Syndrome that develops as a complication of atherosclerosis, hypertension, nephritis. Characterized by damage to the renal vascular system, ending in organ curvature and uremia (nephrosclerosis)
nephrotic syndrome	Syndrome characterized by the presence of expressed proteinuria and tumors (nephrotic syndrome).
Nephrocalcinosis	Accumulation of calcium salts in the renal tissue (parenchyma) (nephrocalcinosis)
Nephroptosis	Decreased renal function (nephroptosis)
Nikturia	Excretion of urine mainly at night (nocturia)
Oxalaturia	Excessive excretion of oxalate salts and calcium salts in the urine (oxalaturia)
Oliguria	A sharp decrease in the amount of urine excreted per day (oliguria)
Paranephritis	Inflammation of the tissues around the kidneys (paranephritis)
Pyelonephritis	Bacterial inflammation of the renal pelvis and parenchyma (pyelonephritis)
Pielit	Inflammation of the renal pelvis (pyelitis)
Pionephritis	Purulent inflammation of the kidneys (pionephritis)
Polyuria	Increased urine output per day (polyuria)
Pollakiuria	Frequent urination (pollakiuria)
Protenuria	Formation of protein in the urine (proteinuria)
Renin	Renin, a vasoconstrictor produced by the hypothalamic apparatus of the kidneys
Pasternaskogo symptom	Pain in the lumbar region when tapping the hand (pasternasky symptom)
	Excessive excretion of amorphous ammonium salts and uric acid in the urine (uraturia)

Uraturia Piuriya Glomerulonephritis Glucosuria	Urine pus (multiple leukocyte excretion with urine (pyuria)) Inflammation of the glomeruli (glomerulonephritis) Urinary excretion of sugar (glucosuria)
Uremia	Symptomatic complex (uremia) that develops as a result of accumulation and poisoning of nitrogen wastes in the body
Urolithiasis	Presence of stones in the bladder (urolithiasis)
Cystitis Cylindruria	Inflammation of the bladder (cystitis) Formation of cylindrical proteins in the patient's transverse urine Hyaline granular, epithelial and waxy cylinders (cylindruria)
Excretory urography	X-ray examination of the urinary tract by means of contrast agent delivery. The shapes and sizes of the renal pelvis and cups, the urinary tract are examined. Excretory urography allows to determine the rate of introduction of a contrast agent into the bladder (excretory urography)

BLOOD SYSTEM

Anaplasia	Loss of cell maturation (anaplasia)
Anemia	Anemia. A pathological condition characterized by a decrease in the number of erythrocytes in the blood and the amount of hemoglobin. 1 mm cubic blood erythrocyte count 4 000 to less than $ 000 (anemia)
Aniocytosis	Formation of erythrocytes of different sizes in the blood (anisocytosis)
Anisochramia	Erythrocytes of different colors (light and dark) due to different amounts of hemoglobin (anisochramia)
Anesinophilia	Absence of eosinophils in the blood (aneosinophilia)
Aplastic anemia	Decreased or damaged regenerative function of the bone marrow, atrophy (aplastic anemia)
Aleykemia	Decreased levels of leukocytes in the blood (aleukemia)
Bolezn Shenleyn Genoxa	Leakage of blood into the mucous membranes and skin as a result of impaired permeability of the vascular walls
Hepatomegaly	Enlargement of the liver (hepatomegaly)
Hematology	Science that studies blood and blood-forming tissue diseases (hematology)
Hemolysis	Rupture of the erythrocyte membrane, in which hemoglobin is released into the blood plasma and turns red (hemolysis)
Hemopoiesis	The process of development of blood cells in hematopoietic

	tissues (hypopoiesis)
Hemorrhagic capillary toxicosis	Hemorrhagic diathesis is a type of disease characterized by hemorrhage into the skin and mucous membranes due to excessive permeability of the vascular wall (hemorrhagic capillary toxicosis).
Hemorrhagic diathesis	A disease that is mainly caused by bleeding. verlrof, Shenlein-Genox, and hemophilia may be present (hemorrhagic diathesis)
Hemolytic anemia	Hemolytic anemia melting, rupture of erythrocyte membrane (erythrocyte breakdown)
Hemorrhage	Bleeding, hemorrhage (hemorrhage)
Hemophilia	Characterized by blood to xtamasligi , one of the diseases tug ' ma bo held on sus- this disease, blood clotting disorders , blood clotting factors 8, 9, 11, Bo ' lmasligidan from the (anemia)
Geophagy	The diagnosis of marbling , cuts sick , Bo ' consumer goods ' o ' l has a tendency to feel (geofagiya)
Hyperplasia	Rapid and abundant production of blood cells in blood-forming tissues (hyperplasia)
Hyperproteinemia	Excess protein in the serum (more than 8%) is called hyperproteinemia.
Hyperchromia	Excessive staining of erythrocytes (hyperchromia)
Hypoproteinemia	Serum protein deficiency (less than 6%) (hypoproteinemia)
Hypochromia	Decreased erythrocyte staining
Dysproteinemia	Changes in the number and quality of protein cells in the blood serum (similar to dysproteinemia (decreased albumin, increased globulins))
Ko y lonexii	Artificial spoon like shape (co lonexiyalar)
Leukoma (leukemia)	A systemic disease of the tissue that produces white blood cells, it is characterized by hyperplasia, metaplasia, anaplasia, and the appearance of immature forms of leukocytes in the blood.
Leukopenia	Decrease in the number of leukocytes, less than 5 00 (leukopenia)
Leukemia	The process of development of white blood cells in the bone marrow (leukemia)
Leukocyte formula	Present ratio of leukocytes a half forms
Leukocytosis	1 mm cubic in the number of blood leykositlar 9 exceed 000 (leykositoz)
Lymphopheny	Decrease in total number of lymphocytes in 1 mm3 of blood by 20%
Lymphocytosis	Excess of more than 35% of lymphocytes in 1 mm3 of blood (lymphocytosis)

Macrocytosis	Predominance of erythrocytes in the blood with a large diameter (9-10 microns) (macrocytosis)
Megalocytosis	In the blood, in contrast to erythrocytes, the formation of megalocytes in the form of ellipses and hemoglobin saturated with a large diameter (10-12 microns) (megalocytosis)
Medulla	Growth of myeloma tissue (medulla)
Metaplasia	Transformation of some types of tissue into other types of tissue (metaplasia)
Mielogram	Record the elements of bone marrow cells in the present ratio (myelogram)
Microcytosis	Excess of erythrocytes in the blood with a small diameter (6 microns and less) (microcytosis)
Myeloma	Growth of the coccyx.
	Bone marrow, bone marrow
Mieloz	Increased monocytes (more than 8% in 1 mm3 of blood).
Monocytosis	Decreased neutrophil count (less than 50% in 1 mm3 of blood)
Neutropenia	Increase in the number of neutrophils in the blood (more than 70% of neutrophils in 1 mm3 of blood)
Neutrophilia Neutrophilia	Rvation blood normoblastlarning Yavne nuclear applying red (normoblastoz)
Normoblastosis	Formation of various forms of erythrocytes in the blood (poikilocytosis)
Poikilocytosis	Occurrence of large numbers of erythrocytes in the blood, which turn gray-purple with acid and alkaline dyes (polychromatophilia)
Polychromatophilia	An increase of more than 1% of reticulocytes in 1 mm3 of blood (reticulocytosis)
Reticulocytosis	Enlargement of the spleen (splenomegaly)
Splenomegaly	Science that studies the methods of placement, preparation and storage of blood and blood substitutes (transfusiology)
Transfusiology	An increase of more than 400,000 platelets in 1 mm3 of blood (thrombocytosis)
Thrombocytosis	It is a type of hemorrhagic diathesis

Thrombocytopenic purpura (Verligofa disease)	characterized by a decrease in the number of platelets in the blood or the appearance of immature platelets (Werlhof's disease, thrombocytopenic purpura)
Thrombocytopenia	Less than 220,000 platelets in 1 mm3 of blood (thrombocytopenia)
Thrombocytopoiesis	Platelet formation and development in the bone marrow (thrombocytopoiesis)
Funukual	Thin cord, rope (funical)
Chlorosis	Iron deficiency anemia (chlorosis)
Schizocytosis	Formation of small, malformed erythrocytes in the blood (schizocytosis)
Eosinophilia	Increase in the number of eosinophils in the blood by more than 4% (eosinophilia)
Erythropoiesis	The process of formation and development of erythrocytes in the bone marrow (erythropoiesis)
Erythrocytosis	1 mm cubic blood cells number 5 000 from more than 000 (erythrocytosis polisitemiya)

ENDOCRINE AND METABOLISM.

Addisonova bolezn	This disease is caused by a lack of blood or total production of hormones in the adrenal glands (Addison's disease)
Adynamics	Severe weakness (adynamism)
Adinase-genital dystrophy	The disease is associated with damage to the hypothalamic-pituitary system and is characterized by underdevelopment and

	sebaceous glands.
Acromegaly	Acromegaly is a neuroendocrine disease associated with damage to the pituitary and hypothalamus, manifested by enlargement of the limbs, facial skeleton, internal organs, and metabolic disorders (acromegaly)
Bolezn Isenko Kushinga	The disease, which results from damage to the pituitary and adrenal glands, is characterized by overproduction of AKTG glucocorticoids and mineralocorticoids.
Bulimia	Constant hunger (bulimia)
Hypothyroidism	Decreased thyroid function (hypothyroidism)
Hyperthermia	Exacerbation of thyroid function (hyperthyroidism)
Hypotension	Decreased blood pressure (hypotension)
Hyperglycemia	Increased blood sugar above 120 mg% (hyperglycemia)
Hypoglycemia	Decreased blood sugar less than 80 mg% (hypoglycemia)
Hypertrichosis	Forced hair growth on the body and face in women (hypertrichosis)
Glucosuria	Urinary excretion of sugar (glucosuria)
Diaseptic rubeoz	Redness is observed on the face, forehead, upper eyelid area, and chin as a result of dilation of the skin capillary network.
Diastema	Enlargement of the interdental space (diastema)
Zob	Enlargement of the thyroid gland
Xantomatosis	Accumulation of histiocytes as a result of increased fat in the blood, resulting in accumulation of yellow nodules on the palms of the hands, feet, elbows and shoulders (xanthomatosis)
Klimaktericheskiy	Accumulation of fat on the neck, VII cervical vertebrae, climacteric bulge
Cushingoid teloslogeny	The patient's face (crescent face) is the accumulation of fat in the chest, abdomen, neck, where the limbs are relatively thin. Kushingsimon gavda.
Ellineka symptom	Occurrence of pigmentation on the eyelids
Krauss symptoms	Blurring of the whites of the eyes (Krauss's symptom)

Nanism	A disease caused by a lack of growth hormone in the body, it leads to stunted growth of skeletal, organ and tissue. Adult males are less than 130 cm tall and females less than 120 cm (nanism)
Hypothyroidism	The patient's tongue becomes enlarged and swollen, the labia thicken, and the vocal folds thicken and speech becomes slurred as a result of swelling of the vocal cords. Nerve-feeding vessels of the peripheral and

Neuropathy	autonomic nervous system (Neuropathy)
Diabetes mellitus	A disease that develops as a result of damage to the hypothalamic-pituitary area and a decrease in the release of antidiuretic hormone. Continues with polyuria and polydipsia (diabetes mellitus)
Polyuria	Excessive urination per day (polyuria)
Gi rsutizm	Mustache and hair growth in women, hair loss on the head (Gi rsutism)
Pigmentation	The appearance of dark gray and black spots on the lines of the palms of the hands, lips, gums, face, mucous membranes and in areas of high friction.
Polydipsia	Severe thirst and need to drink plenty of fluids (Polydipsia)
Pollakiuria	Frequent urination (Pollakiuria)
Prognatism	The lower jaw is enlarged and protrudes (Prognatism)
Symptom Da lrimpelia	Conjunctivitis (Da lrimpel's symptom)

SOEDINETELNAYA TKAN.

CONNECTIVE TISSUE.

Amylidosis	Disorders of protein metabolism due to the accumulation in tissues of proteins with characteristic physicochemical properties (amyloidosis)
Ankylosis	Lack of joint mobility (ankylosis)
Ant and gen lar	Iodine in the blood. Substances that enter the body and cause an immune response, produce specific antibodies (antigens)
Antibody	Protein immune substances (antibodies) formed in the blood and tissues when antigens enter the body
	Cutting a small piece of tissue or organ for examination under a microscope for diagnostic

Biopsy	purposes (biopsy)
Destruction	Destruction of an organ or tissue
Dermatomyositis	Inflammatory process (dermatomyositis) affecting muscles, skin, mucous membranes, and sometimes nerves and blood vessels, one of the types of DBST
Cachexia	Clinical anatomical syndrome (cachexia) characterized by excessive weight loss, physical weakness and general events
Pneumosclerosis	Growth of connective tissue in the lungs, in which the function of the lungs is impaired (Pneumosclerosis)
Puncture	Puncture of the tissue with a needle (or trocar) to diagnose or treat the disease
Scleroderma	In diseases of the skin and subcutaneous tissue - scleroderma
Soedinitelnaya tissue	It is a tissue that develops from the mesenchyme and performs basic, trophic and protective functions.
Phagocytosis	The process by which cells actively capture particles and digest them if these particles are organic (phagocytosis)
Xeylit	Inflammation of the red lips, mucous membranes and skin of the lips (cheilitis)
Eosinophilic	Increased number of eosinophils in the blood (Eosinophilia)

Criteria for current assessment of students' knowledge

Rating development and evaluation criteria on the subject "IKP"
(For 3rd year medical students)
Rating development.

№	Types of control	Soni	Maximum score	Coefficient	Total points
1.	JB				
	1.1. Practical training	35	100	0.45	45
	1.2. TMI	27	100	0.05	5
	1.3. ON	4	100	0.2	20

2.	Ya.B. 3.1. Ya.B. 3.2. OTKS	1 1	100	0.3	30
Total:		68	100	1.0	100

Student knowledge assessment system:

The following types of control are carried out to determine the level of mastery of students in the subject:

№	Type of assessment	Form of assessment	Max . ball
1.	Current b aholash	Oral, written, situational problem solving, assessment of practical skills	45
2.	TMI	Preparation of abstracts, abstracts, scientific articles, theses, reports, presentations	5
3.	Interval q ba h olash	YO zmayoki o g ' zakiso'rov , practical skills	20
4.	Concluding evaluation to assess the h	Completion of practical skills, solving situational problems, + oral questioning	30
	total		100

Evaluation criteria

1. Practical Exercise ' Typo by the department to assess each subject image produced on the basis of the evaluation criteria will be evaluated . Each train will contain a 100 -point system (86-100 a ' lo , 71-85 , 55-70 evaluated satisfactory) . O value of the average of 0.45 coefficient ko ' paytiriladi .
2. Students work independently , depending on the size and quality of the implementation of a 100 -point system will be evaluated o ' the value of the average received 0.05 coefficient ko ' paytiriladi .
3. Intermediate control is multiplied by a factor of 0.02.
4. The final assessment will be conducted in two stages:

1. OTKS - practical skills are assessed on a 100-point scale and multiplied by a factor of 0.3.
2. The student's mastery of each type of control is added to the total score and the mastery is assessed.

JB 0.45 + TMI0.05 + ON 0.2 + YaB 0.3 = UO '

In the current control, students' knowledge is based on the educational model in the following stages: theoretical, practical part and TMI (50:40:10)

	96-100	91-95	86-90	81-85	76-80	71-75	66-70	61-65	55-60	31-54	0-30
Theoretical part	52- 50	46-48	43-45	41-42	38-40	36-38	33-35	31-33	28-30	15-27	0-18
Practical part	35-40	36-38	34-36	32-34	31-32	28-30	26-28	24-26	22-24	12-22	0-12
TMI	9-10	9	9	8-9	7-8	7	7	6	5-6	4-5	0

Resident Evil ba h Assessment Criteria

Assimilation%	Bah o	The level of knowledge of the student
96-100 %	A'lo “5”	The student’s answer to all the questions on the ticket is complete, confident. Thinks creatively in solving a situational problem on the syndrome, gives a clear answer and logically substantiates the answer. In response, he summarizes his knowledge of human anatomy, normal physiology and biochemistry, is able to draw independent conclusions and decisions. Demonstrated practical skills

		step by step. Correctly answered questions about the respiratory organs in the theoretical part. Solved the tests correctly. Correctly selected the answer to the compatibility tests. Completed and mastered independent study assignments in a timely, quality manner.
91-95 %	A'lo “5”	The student’s answer to all the questions on the ticket is complete, confident. Thinks creatively in solving a situational problem on the syndrome, gives clear answers, and logically substantiates the answer. In response, he summarizes his knowledge of human anatomy, normal physiology and biochemistry, is able to draw independent conclusions and decisions. Demonstrated practical skills step by step. Correctly answered questions about the respiratory organs in the theoretical part. Solved the tests correctly. Correctly selected the answer to the compatibility tests. Completed and mastered independent study assignments in a timely, quality manner.
86- 90 %	A'lo “5”	The student’s answer to all the questions on the ticket is complete, confident. Thinks creatively in solving a situational problem on the syndrome, gives a clear answer and logically substantiates the answer. In response, he summarizes his knowledge of human anatomy, normal physiology and biochemistry, is able to draw independent conclusions and decisions. Demonstrated practical skills step by step. Correctly answered questions about the respiratory organs in the theoretical part . Solved the tests correctly. Correctly selected the answer to the compatibility tests. Completed and mastered independent study assignments in a timely, quality manner.
81-85 %	GOOD “4”	The student’s answer to all the questions on the ticket is complete, confident. He thinks creatively in solving a situational problem on the syndrome, but finds it somewhat difficult to justify. In response, he summarizes his knowledge of human anatomy, normal physiology and biochemistry, is able to draw independent conclusions and decisions. He demonstrated the practical skills step by step, but with a bit of difficulty. He answered questions about the respiratory organs in the theoretical part. He solved the tests correctly. There are some shortcomings in the response to compatibility tests. Completed and mastered independent study assignments in a timely, quality manner.
76-80 %	Y a xshi “4”	The student’s answer to all the questions on the ticket is complete, confident. He thinks creatively in solving a situational problem on the syndrome, but finds it somewhat difficult to justify. In response, he summarizes his knowledge of human anatomy, normal physiology and biochemistry, is able to draw independent conclusions and decisions. Demonstrated step-by-step practical skills, could not justify changes in the syndrome diagnosis. He answered questions about the respiratory organs in the theoretical part. He solved the tests correctly. There are some shortcomings in the response to compliance tests. Completed and mastered independent learning tasks in a timely, quality manner.
71-75 %	Y a xshi “4”	The student’s answer to all the questions on the ticket is complete, confident. He thinks creatively in solving a situational problem on the syndrome, but finds it somewhat difficult to justify. In response, he summarizes his knowledge of human anatomy, normal physiology and biochemistry, is able to draw independent conclusions and decisions. Demonstrated step-by-step practical skills, could not justify changes in the syndrome diagnosis. He answered questions about the respiratory organs in the theoretical part. He solved the tests correctly. There are some shortcomings in the response to compatibility tests. Completed and mastered independent study assignments in a timely, quality manner.

66-70 %	Satisfactory " 3 "	The student answers 65-70% of the questions asked on the ticket. Makes some mistakes in solving a situational problem. K o ' the implementation of nikmalarni kamchilliklar bor.Mustaqil fulfillment of the tasks of education is of poor quality, nutrition is not enough.
61-65 %	Satisfactory " 3 "	The student answers 60% of the questions on the ticket. Makes some mistakes in solving a situational problem. K o ' the implementation of nikmalarni kamchilliklar bor.Mustaqil fulfillment of the tasks of education is of poor quality, nutrition is not enough.
55-60%	Satisfactory "3"	Analytical q name, applied k o ' nikmalarni gross errors in the implementation. Independent study assignments were performed poorly.
54 %	Satisfactory s i z " 2 "	K o ' performance nikmalarni t o ' exploration does not know the issue, the situation can not solve.

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