MINISTRY OF HIGHER AND SECONDARY EDUCATION OF THE REPUBLIC OF UZBEKISTAN

MINISTRY OF HEALTH OF THE REPUBLIC OF UZBEKISTAN

BUKHARA STATE MEDICAL INSTITUTE NAMED AFTER ABU ALI IBN SINO

"APPROVED"
Prorector of Educational work of
Bukhara State Medical Institute, c.m.s
G.I.Jarilkasinova
"__" _____ 2020 year

CHAIR OF "FACULTY AND HOSPITAL SURGERY, UROLOGY"

Training and metodology complex

ON THE SUBJECT "HOSPITAL SURGERY"

COMPILED BY:

ScD., Professor
R.M.AXMEDOV
PhD., Associate professor
S.O.KOMILOV
Assistant
U.X.YULDOSHEV

Bukhara - 2020 y.

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CHAIR OF "FACULTY AND HOSPITAL SURGERY, UROLOGY"

720000 - Healthcare, 5720100 - Educational-methodical complex was developed for students of medical and medical-pedagogical faculties on the basis of the curriculum.

Compiled by:
ScD., Professor R.M.AXMEDOV
PhD., Associate professor S.O.KOMILOV
Assistant U.X.YULDOSHEV

Reviewers:
Sh.T. Urakov - Head of the Chair of Surgical Diseases and Reanimation, Doctor of Medical Sciences, Associate professor
V.R..Akramov - Head of the Department of Traumatology and Orthopedics, PhD, Associate professor t

The educational and methodological complex was considered and approved at the meeting of the Central Medical Research Center of the Bukhara State Medical Institute by the protocol of
"_____" _____ 2020.

ANNOTATION

Hospital surgery is a special clinical discipline dealing with etiology, pathogenesis, clinic, research methods, prognosis and complications, principles of treatment and prevention of surgical diseases, emergency care in case of emergency. In the conditions of RMS and CMS, from up to 1/3 of patients are treated for surgical diseases and they often need urgent measures to save the patient's life. Therefore, the study of Hospital Second Surgery of the GP program is a prerequisite for students of medical and medico-pedagogical faculties.

Methodical complex of andone second surgery is made to conformity Standard Program for hospital surgery in view of the educational standard, qualifying characteristics of general practice doctor and volume of an academic load, according to the curriculum.

The program is, enclosed volume of theoretical and practical skills that students must master the medical and medical-pedagogical faculties in the study of surgical diseases of the GP program.

Typical program by discipline "Hospital surgery"

MINISTRY OF HIGHER AND SECONDARY SPECIAL EDUCATION OF THE REPUBLIC OF UZBEKISTAN

Registered "Approved"
DB number - 57202-210 Ministry in s with u him and medium
" 28" August 2008 g . Special education
Republic of Uzbekistan
Order No. 263
«23» August 2008 g .

TYPICAL PROGRAM

BY DISCIPLINE

HOSPITAL SURGERY

Area of expertise: 720,000 - "Healthcare"

By direction (specialty):

5720100 - "General Medicine"

5140900 - "Professional education"

Tashkent - 2008

CREATED BY:

Karimov Sh.I. - Head of the Department faculty and hospital surgery medical faculty of the Tashkent Medical Academy, MD, etc. on fessor.

Nazyrov F.G. - Head of the Department of Faculty and Hospital Surgery of the Medical and Pedagogical Faculty of the Tashkent Medical Academy, Doctor of Medical Sciences, Professor.

A.A. Imomov - responsible for the educational process of the department faculty and hospital Hira p ology of the medical faculty of the Tashkent Medical Academy, MD, assistant professor.

B.A. Ugarov - responsible for the educational process of the department fakulte t tion and hospital surgery medical and pedagogical faculty of the Tashkent Medical Acad e mission, Ph.D. assistant.

REVIEWERS:

Ataliev A.E. - Professor of the Department of Surgery of the GP of the TMA, Doctor of Medical Sciences, pr o fessor

Soatov RR . - Associate Professor of the Department of Surgery TashIUV, Ph.D. sciences.

The program was discussed and recommended at the Academic Council of the Tashkent Medical Academy.

Minutes No. ___ dated "___" _____ 2008.

The program was discussed at the interuniversity coordination council (protocol No. ___ dated "___" _____ 2008) and recommended for approval by the Ministry of Higher and Secondary Specialized Education of the Republic of Uzbekistan and the country

INTRODUCTION

Hospital surgery is a special clinical discipline dealing with etiology, pathogenesis, clinic, research methods, prognosis and complications, principles of treatment and prevention of surgical diseases, emergency care in case of emergency. In the conditions of SVP and SHP, from up to 1/3 of patients are treated for surgical diseases and they often need urgent measures to save the patient's life. Therefore, the study of the hospital under the GP program is a necessary requirement for students of medical and medical-pedagogical faculties.

A typical program for hospital surgery is drawn up taking into account the State Educational Standard, the qualification characteristics of a general practitioner and the volume of the study load, in accordance with the curriculum. The program invested the amount of theoretical and practical skills that students must master the medical and health teacher in the Faculty - Tetovo, in the study of surgical diseases of the GP program.

Purpose of the item:

The purpose of the training is: teaching 5-year students on the basis of theoretical knowledge , providing practical medical care to surgical patients under the program of hospital surgery in outpatient and inpatient settings. general practitioner on an outpatient basis. On the provision of self-help to the patient, or referral to a specialized hospital, depending on the category of the disease.

Objectives of the subject:

- Teaching students the etiology, pathogenesis, clinical picture, diagnosis and treatment of widespread and rare surgical diseases.
- Teaching self-supervision of patients.
- Filling out medical history, outpatient cards and other documents.
- Providing emergency medical care and diagnostics of acute surgical diseases .
- Planning diagnostics and treatment of patients according to the diagnostic and treatment standards of surgical diseases.
- Teaching students to practical skills.

- Training in methods of prevention and clinical examination of surgical patients in a polyclinic . (SVP, GVP, etc.)

Hospital medicine is taught to 5th year students of the medical faculty and medical pedagogy. The lesson is held in the departments of the surgical profile of the clinic using a new pedagogical technology of slides, multimedia, tables and demonstration of case patients. Students' knowledge is assessed based on the ranking.

Students participate in seminars and lectures. Classes chairs e conducted in cycles. During the cycle, each student every day takes part in the supervision of the treated patients on the topic. At the same time they are in the Department of Abdominal Surgery, thoraco-to osudisto second surgery, resuscitation, points transfusion of blood , contaminated surgery. If there are no patients on the topic, for the purpose of diff. diagnostics examine patients with similar diseases.

Each student ATS is 2 and night duty pischet one history. This is all taken into account when assessing the student.

For students 5 -Course allocated 1 to 90 hours, including 36 hours lectures (18th order), 108 -hr practical component (2 1-ones we) and 46 hours of independent operation (1 8 -Themes s).

Innovation in Learning in the first is that used in the classroom interactive methods: multimedia, test questions, and case studies , etc.. . All students are assessed based on their ranking throughout the year. Based on a 100-point rating system, students are assessed in TO, PO, IO.

Current assessment (TO) 0.45 coefficient, students' independent work (IWS) 0.05 coefficient intermediate grade (PO) 0.2 coefficient, final control (IC) consists of two parts - OSKI-0.15 coefficient and test control-0.15 coefficient. Practical studies 5 0% n p s on d GSI interactive method.

The student should know:

- Etiology, pathogenesis, clinics, diagnostics, treatment and prevention of surgical diseases according to the program.
- Clinical symptoms in surgical diseases of internal organs (chest and abdominal cavity)
- Clinical symptoms with injuries of internal organs (chest and abdominal cavity)
- Clinical symptoms in acute and chronic surgical diseases.
- Methods of modern rehabilitation in the postoperative period.

Skills that the student must possess .

1. The imposition of a tourniquet for arterial bleeding
2. Applying a hemostatic forceps to a bleeding vessel
3. Applying a pressure bandage for venous bleeding
4. First aid for arterial bleeding from the extremities
5. Anatomical points for stopping bleeding from large vessels by finger pressure
6. Venesection technique
7. Venous tests :
8. Outpatient care for bleeding from the esophagus and stomach
9. Blackmore Probe Placing Technique
10. Technique of laparocentesis
11. Outpatient care for bleeding from the lungs
12. Perirenal blockade
13. Vagosympathetic blockade
14. First aid for chest injuries
15. Outpatient care for pneumothorax
16. Outpatient care for closed pneumothorax
17. Outpatient care for valvular pneumothorax
18. Pleural puncture
19. Thoracocentesis
20. Feeding the patient through a nasogastric tube
21. Technique of flushing CCHS.

The volume of academic hours of the subject

Total scope of work:	**Auditorium clock**		**Independent work**
Total	Lecture	Practice	4 6
19 0	36	10 8	

MAIN PART

1. **Introduction. Understanding the subject of Hospital Surgery and writing a medical history.** Familiarization with specialized departments. Patient supervision. Anamnesis and objective examination. Anatomical and physiological concept of the esophagus. Special examination methods, classification of diseases of the esophagus.
2. **The main manifestations of diseases of the esophagus. Chemistry esophagus burns** **chemical substances. Diverticulum of the esophagus.** Burns of the esophagus with chemicals. and Cicatricial narrowing, esophageal diverticulum. Clinic, diagnostics, conservative and operative new treatment. Types of outcomes of surgical interventions.
3. Disease of the esophagus. The concept of cardiospasm . Clinic, diagnostics and differential diagnostics. Technique of cardio - dilation. Types of operations foreign body of the esophagus. Endoscopic and surgical methods of treatment.
4. Portal hypertension and its complication . Etiology, pathogenesis, clinical picture and diagnosis of portal hypertension. Conservative and surgical methods of treatment. Indication for surgery. Types of operations. Buddy-Chiari syndrome. Clinic, diagnosis and treatment.
5. Echinococcosis. The concept of parasitic diseases . Parasite characteristics. Echinococcosis and alveococcosis of the liver. Clinic, diagnostics and differential diagnostics. Types of surgical treatment and their outcomes. Surgical treatment of combined echinococcus-goose of the liver and lungs and its sequence.
6. Varicose veins. Anatomical and physiological data of the veins of the lower extremities. Special new types of surveys. Varicose veins of the lower extremities tei. Clinic and diagnostics. Etiopathogenesis and treatment. Syndrome of Parks-Weber-Ruba-seams, Klipel-Trenonne Paget-Schrötter.
7. Thrombosis and thrombophlebitis of superficial and deep veins of the lower extremities. Clinic and diagnostics. Postthrombophlebitis syndrome. (PTFS). Concept, etiopathogenesis, clinical picture and treatment, prevention of PTFS.
8. Anatomical and physiological data on the aorta and arterial system. Etiology of pathogenesis of occlusive lesions of arterial vessels. Special methods of examination of patients with arterial pathology and stages. Nespetsifiches - ki nd aortoarteriit. Types operative the intervention STV (sympathectomy, endartektomiya, prosthetics and surgery).
9. Occlusive disease of the aortic arch. Takayasu syndrome. Etiopathogenesis, clinical forms, methods of treatment. Surgical s treatments.

10. Occlusive disease of the abrasive aorta. High, middle and inferior occlusion of the abdominal aorta. Etiopathogenesis .Sindrom chronic abdominal ischemia (SKHAI) .Etiologiya, classification I and methods of surgical leche Nia
11. Symptomatic arterial hypertension. Vase o-real hypertension syndrome . Etiopathogenesis , clinic, diagnostics and surgical treatment. Pheochromositoma. Coma syndrome. Itsenko-Cushing syndrome. Diagnostics, treatment methods. New technologies. ...
12. Occlusive diseases of the lower extremities. Leriche syndrome. Jurger 's disease B , Raynaud 's disease . Diabetic angiopathy. Klee nickname, diagnosis, differential. diagnostics. Surgical tactics, methods of treatment. Klerosis obliterans atheres. On-literiruyuschie endoart rit .
13. Acute arterial insufficiency. Acute thrombosis and embolism. Etiopathogenesis. Thrombosis formation factors. Clinical course . Con - conservative and surgical treatment. Principles of anticoagulant therapy.
14. Congenital heart defects. Anatomical and physiological information. "White" and "Blue" vices. Open arterial defect Triad, Tet-rada Fallot and pentad. Luthenbacher's syndrome. Cortation of the aorta. Clinic, diagnostics.
15. Acquired heart defects. Defects of the mitral , aortic and three cuspid valves. Etiopathogenesis, circulatory disorders. Clinic, diagnostics and indications and contraindications for surgical treatment. Open and closed methods of surgical treatment
16. Anatomy and physiology of the lungs. Survey methods . Acute nonspecific abscess and gangrene of the lungs. Etiology, pathogenesis, clinical picture, diagnosis and methods of treatment.
17. Nonspecific lung diseases . Chronic nonspecific lung abscess . Etiology, Clinic, diagnostics, conservative and surgical methods of treatment. Bron choectatic disease. Clinic and methods of treatment.
18. Disease of the pleura. Anatomical and physiological information about the pleura. Acute pleural empyema and types. These are opathognosis and treatments. Chronic pleural empyema. Etiopathognosis, clinical picture, differential diagnosis. Bronhople vralny and broncho plevrotoro Calne communication seek. Surgical treatment methods . Thoracoplasty methods. "Incident"
19.Lung cysts. Classification .. Pneumothorax and pyopneumothorax. Concept, types, reasons. Clinic and treatment. Echinococcus of the lungs. Etiopathogenesis, complications. Operation methods and outcomes.
20. Disease of the mediastinum . Anatomical and physiological information of the mediastinal organs. Service methods. Benign tumors of coli and

mediastinal cysts. Classification, diagnosis, treatment. Mediastenitis. Etiology, clinic, diagnosis and treatment. Myasthenia , diagnostics, methods of treatment .
21. Disease of the diaphragm. Diaphragmatic hernia. Anatomophysi-ological properties of the diaphragm. Clinical manifestations of various diaphragmatic hernias. Hernia of the esophageal opening of the diaphragm. Relaxation of the diaphragm. Types of plastic diaphragms.
22.Diseases of the operated stomach. Post-gastrointestinal resection syndrome . Classification, clinical picture, causes, diagnostics . Indication for surgical treatment. Wee dy reconstructive surgery. Postvagotomic syndrome. Le chenie.
23. Postcholecystectomy syndrome. Concept. Classification. Etiopia is togenesis. Diagnostics and diff. Diagnostics in postcholecystectomy syndrome. Types rekonstruktvnyh and operative - GOVERNMENTAL interventions
24. Obstructive jaundice. Types and reason. Methods and diagnostics. Surgical treatment methods. Cholangitis. Treatment methods.

Main literature on the subject:

1. Casaliklar surgeon. Sh.I. Karimov, Toshkent, 2005
2. Kasaliklar surgeon. Sh.I. Karimov, N.H. Shamirzaev, Toshkent, 1995
3. Surgical diseases. Edited by M. I. Kuzin., Medicine., 2002
4. Methodological manual for hospital surgery. Nazyrov F.G. with co-workers Tashkent 2004
5. Clinical surgery. Ed. Pantsyreva Yu.M. M. "Medicine", 1988
6. Vorobiev A Handbook of a practical doctor in 3 volumes. 1990
7. Conden R., Neuhus L. Clinical surgery Moscow. Practice 1998
8. Nazirov F.G., Denisov I.I., Ulumbekov E.G. A guidebook for practitioners and forging doctors. Moscow 2000
9. Petrovsky B.V. ed. Guide to surgery (in 12 volumes) M. Medicine 1959-1966 .

Additional literature:

1. Alperovich B.I. Surgery of the liver and biliary tract. M. Medicine 1997
2. Astapenko V.G. A practical guide to surgical diseases. Minsk 1984 Part 2.
3. Bedensky K.N. Varicose disease. Leningrad, "Medicine", 1983
4. Burakovsky V.I., Bockeria L.A. Cardiovascular surgery, M., "Medicine," 1989 .
5. Vasilenko V.Kh, Grebnev A.L. Hernia of the esophageal opening of the diaphragm., M., "Medits and na", 1978.

6. Vakhidov V.V., Khadzhibaev A.M. Postvagotomnye syndromes and surgical correlator to tsym. V., Tashkent, 1993.
7. Vakhidov. V.V., Gambarin B.L. Surgical treatment of chronic Zabol e vany veins. Tashkent, "Medicine" 1979.
8. Vilyavin G.D., Soloviev V.I., Timofeev T.A. Cardiospasm, M., "Medicine", 1971
9. Vishnevsky A.A., Adamyan A.A. "Surgery cf. e dosteniya" Moscow, 1977.
10. Galperin E.I., Kuzovlev N.O., Karagulyan S.R. Prot biliary stricture scar on Cove. M., "Medicine", 1982.
11. Goldberg V., Lavnikova G.A. "Tumors and cysts of the mediastinum" M., 1965
12. Grippy M, A. Lung pathophysiology Moscow, St. Petersburg, Binome 2000
13. Dauderis I.N. Diseases of the veins and the lymphatic system of the extremities. M. "Medic and na", 1984
14. Erokhin I.A., Gelfand B.R., Shlyapnikov S.A. Surgical infections. Sank t .Petersburg 2003
15. Klementyev A.A., Bedensky A.N. Surgical treatment of diseases of veins of the extremities of stey. Leningrad, "Medicine", 1983.
16. Kozyrev M.A. Komarov B.D., Kanshin N.N., Abakumov M.M. Damage to the esophagus. M., "Medic and na", 1981.
17. I. S. Kolesnikov, B. S. Vikhriev Lung abscesses. Leningrad. Medicine 1973
18. Kolesnikov I.S., Lytkin M.I. - "Surgery of the lungs and pleura" L., 1988
19. Kolesov V.I., Surgery of the coronary arteries. Leningrad "Medicine," 1977.
20. Loginov A.S., Blok Yu.E. Chronic hepatitis and cirrhosis of liver diseases and des h GOVERNMENTAL paths. M. 2002
21. Mayer K.P. Hepatitis and the consequences of hepatitis. M. 1999 .
22. Nazirov F.G., Akilov Kh.A., Devyatov A.V. Surgery complications of portal w Perth n sion in patients with cirrhosis of the liver. "GEOTAR-MED", 2002
23. Orlova N.V., Parinskaya T.V. Acquired heart defects in children. L., 1979
24. Pantsyrev YM, Galinger YI .. Operative endoscopy of the gastrointestinal tract to that. M., "Medicine", 1980
25. Patient MD Surgery of portal hypertension .. Tashkent, "Medicine", 1984.
26. Petrovsky B.V., Knyazev M.D., Shabalkin B.V. Surgery of chronic coronary artery disease M. "Medits and na," 1973
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29. Pokrovsky A.V. Diseases of the aorta and its branches. M., "Medic and na," 1979
30. Pokrovsky A.V., Zotikov A.E., Yudin V.I. Nonspecific aorto-arteritis (T a kayasu's disease) M. 2000
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32. Rabkin Ch.Kh. Guidelines for angiography. M., "Medicine," 1977
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34. Rozanov B.S., Foreign bodies and esophageal injuries and associated complications. M. "Me Dr. Guise," 1961.
35. Savelyev VS, Speridonov IV acute mesenteric circulatory disorders e Nia., M., "Medicine", 1979.
36. Saveliev V.S. 50 lectures on surgery. Moscow 2004.
37. Saveliev V.S., Dumpe Z.I., Yablokov Z.S. Disease of the great veins. M. "Medic and na," 1992.
38. Savelyev VS, Zatevakhin I.I Stepanov NV, acute obstruction bifurcation AB p you and the main arteries of the limbs. M., "Medicine," 1987
39. Saveliev V.S., Yablokov E.G., Kirienko A.I. Massive pulmonary embolism. M "M e Diqing", 1990.
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43. V. I. Struchkov Purulent diseases of the lungs and pleura. Leningrad. 1967.
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Hospital surgery
Training Work Program

INTRODUCTION

Hospital surgery is a special clinical discipline dealing with etiology, pathogenesis, clinic, research methods, prognosis and complications, principles of treatment and prevention of surgical diseases, emergency care in case of emergency. In the conditions of SVP and SHP, from up to 1/3 of patients are treated for surgical diseases and they often need urgent measures to save the patient's life. Therefore, the study of the hospital under the GP program is a necessary requirement for students of medical and medical-pedagogical faculties.

A typical program for hospital surgery is compiled taking into account the State educational standard, the qualification characteristics of a general practitioner and the volume of the study load, in accordance with the curriculum. The program includes a volume of theoretical and practical skills that students of medical and medical-pedagogical faculties must master when studying surgical diseases under the GP program.

Purpose of the item:

The purpose of the training is: teaching 5-year students on the basis of theoretical knowledge, providing practical medical care to surgical patients under the program of hospital surgery in outpatient and inpatient settings. general practitioner on an outpatient basis. Providing self-help to the patient, or referral to a specialized hospital, depending on the category of the disease.

Objectives of the subject:

- Teaching students the etiology, pathogenesis, clinical picture, diagnosis and treatment of widespread and rare surgical diseases.
- Teaching self-supervision of patients.
- Filling out medical history, outpatient cards and other documents.
- Providing emergency medical care and diagnostics of acute surgical diseases.
- Planning diagnostics and treatment of patients according to the diagnostic and treatment standards of surgical diseases.
- Teaching students to practical skills.
- Training in methods of prevention and clinical examination of surgical patients in a polyclinic. (SVP, GVP, etc.)

Hospital surgery is taught to 5th year students of the medical faculty and medical pedagogy. The lesson is held in the departments of the surgical profile of the clinic using a new pedagogical technology of

slides, multimedia, tables and demonstration of case patients. Students' knowledge is assessed based on the ranking.

Students participate in seminars and lectures. Classes of the department are conducted in the form of cycles. During the cycle, each student every day takes part in the supervision of the treated patients on the topic. At the same time, they are in the departments of abdominal surgery, thoraco-vascular surgery, resuscitation, blood transfusion point, purulent surgery. If there are no patients on the topic, for the purpose of diff. diagnostics examine patients with similar diseases.

Each student hands over 2 night shifts and beeps one medical history. This is all taken into account when assessing the student.

For 5-year students, 190 hours are allocated, of which 36 hours of lectures (18 topics), 108 hours of practical classes (21 topics) and 46 hours of independent work (18 topics).

The innovation in teaching lessons is that the lessons use interactive methods: multimedia, test questions, and situational tasks, etc. All students are assessed based on the rating throughout the year. Based on a 100-point rating system, students are assessed in TO, PO, IO.

Current grade (TO) 0.45 coefficient, student independent work (IWS) 0.05 coefficient intermediate grade (PO) 0.2 coefficient, final control (IC) consists of two parts - OSKI-0.15 coefficient and test control-0.15 coefficient. Practical lessons 50% are conducted interactively.

The student should know:

- Etiology, pathogenesis, clinics, diagnostics, treatment and prevention of surgical diseases according to the program.
- Clinical symptoms in surgical diseases of internal organs (chest and abdominal cavity)
- Clinical symptoms with injuries of internal organs (chest and abdominal cavity)
- Clinical symptoms in acute and chronic surgical diseases.
- Methods of modern rehabilitation in the postoperative period.

Skills that the student must possess .

22. The imposition of a tourniquet for arterial bleeding
23. Applying a hemostatic forceps to a bleeding vessel
24. Applying a pressure bandage for venous bleeding
25. First aid for arterial bleeding from the extremities

26. Anatomical points for stopping bleeding from large vessels by finger pressure
27. Venesection technique
28. Venous tests :
29. Outpatient care for bleeding from the esophagus and stomach
30. Blackmore Probe Placing Technique
31. Technique of laparocentesis
32. Outpatient care for bleeding from the lungs
33. Perirenal blockade
34. Vagosympathetic blockade
35. First aid for chest injuries
36. Outpatient care for pneumothorax
37. Outpatient care for closed pneumothorax
38. Outpatient care for valvular pneumothorax
39. Pleural puncture
40. Thoracocentesis
41. Feeding the patient through a nasogastric tube
42. Technique of flushing CCHS.

The volume of academic hours of the subject

Total scope of work:	**Auditorium clock**		**Independent work**
Total	Lecture	Practice	46
190	36	108	

CALENDAR O- THEMATIC PLAN

LECTURES ON HOSPITAL SURGERY

No.	The name of the s	Clock	Group	Time passed.	Visual material	Used literature
1	Disease of the esophagus. Diverticulum of the esophagus. Chemical burns of the esophagus.	2			Table 1,2.3.4. Multimedia radiographs s Educational film	1. "Kasallik surgeon - liklar " , Sh. I. Karimov T oshkent . 2005. 2. "Surgeon Kasalliklar", Sh.I.Karimov N.H. Shamirzaev T. 1995. 3. "Surgical diseases" n od ed. M.I. Kuzina, Medicine 2002. 4. Methodological manual for hospital surgery. Nazyrov F.G. et al. T - 2004. 5. Clinical surgery. Edited by Yu.M. Pantsyrev, M. "Medicine" 1988. 6. A.A. Shalimov "Surgery food - water" 7. G. L. Ratner "Burns of the

						esophagus" . VS Savelyev , AI Kirienko " Surgical diseases ", M .; 2006; T- II ; 13 - 54 betlar. 8. "Diverticulum of the esophagus" BV Petrovsky, EN Vantsyan, M. Meditsina, 1968. 9. "Қizilўngach kasalliklari", R.M. Akhmedov; S.O.Komilov va b., Conv. Қўl. 2010y.
2.	Disease of the esophagus. Car diospasm .	2			Tables 1.5. Multimedia R -grams	1. "Kasallik surgeon - liklar " , Sh. I. Karimov T oshkent . 2005. 2. "Surgeon Kasalliklar", Sh.I.Karimov N.H. Shamirzaev T. 1995. 3. "Surgical diseases" n od ed. M.I. Kuzina, Medicine 2002. 4. Methodical Pozo - Bie for hospital surgery. Nazyrov F.G. et al. T - 2004. 5. Clinical hirur - ogy. Edited by Yu.M. Pantsyrev, M. "Medicine" 1988. 6. A.A. Shalimov "Surgery food - water" 7. "Cardiospasm", GD Vilyavin, VI Soloviev et al., M. 1971. 8. "Foreign body and trauma of the esophagus " BS Rozanov, M. "Medgiz", 1961.
3.	Portal hypertension and its complication.	2			Tables 6.7. Multimedia CT, UTT	1. "Kasallik surgeon - liklar " , Sh. I. Karimov T oshkent . 2005. 2. "Surgeon Kasalliklar", Sh.I.Karimov N.H. Shamirzaev T. 1995. 3. "Surgical diseases" n od ed. M.I. Kuzina, Medicine 2002. 4. Methodical Pozo - Bie for hospital surgery. Nazyrov F.G. et al. T - 2004. 5. Clinical surgery. Edited by Yu.M. Pantsyrev, M. "Medicine" 1988. 6. "Surgery of portal hypertension" by M.D. Patsiora, Moscow, 1984. 7. "Surgery of complications of portal hypertension in patients with liver cirrhosis." F.G.Nazyrov, Kh.A. Akilov et al., "GZOTARMED", 2002. 8. "Chronic hepatitis and cirrhosis" AS Loginov, Yu.E. Blok, M.2002. 9. "Portal hypertension", R.M. Akhmedov, S.O.Komilov va b. conv- llanma, 2010.
4.	Echinococcosis . The concept of parasitic diseases. Parasite characteristics. Echinococcosis and	2			Tables 8,9,10. MultimediaUTT , CT	1. "Kasallik surgeon - liklar " , Sh. I. Karimov T oshkent . 2005. 2. "Surgeon Kasalliklar", Sh.I.Karimov N.H. Shamirzaev T. 1995. 3. "Surgical diseases" n od ed. M.I. Kuzina, Medicine 2002. 4. Methodological manual for hospital surgery. Nazyrov F.G. et al. T -

	alveococcosis of the liver and lung.					2004. 5. Clinical surgery. Edited by Yu.M. Pantsyrev, M. "Medicine" 1988. 6. "Surgery of echinococcosis", BV Petrovsky, OB Milonov et al., M. Medicine, 1985. 7. "Surgery of the liver and bile ducts " , AA Shalimov et al., Kiev, 1993. 8. "Surgery of the liver and biliary tract" BI Alperovich, M.1997. 9. "Zhigar echinococcosis", R.M. Akhmedov, I.A.Mirkhzhaev va b. usl-llan, 2010.
five .	Varicose veins . Diseases Parks -Weber-Rubashev a , Klippel-Trenone.	2			Tables 11,12,13, 14.15. Phlebogram Multimedia	1. "Surgeon Kasalliklar" , Sh. I. Karimov T oshkent . 2005. 2. "Surgeon Kasalliklar", Sh.I.Karimov N.H. Shamirzaev T. 1995. 3. "Surgical diseases" n od ed. M.I. Kuzina, Medicine 2002. 4. Methodological manual for hospital surgery. Nazyrov F.G. et al. T - 2004. 5. Clinical hirur - ogy. Edited by Yu.M. Pantsyrev, M. "Medicine" 1988. 6. "Varicose veins" KN Vedensky, Leningrad, 1983. 7. "Surgical treatment of veins disease of the extremities", AA Klementyev, AN Vedensky, Leningrad "Medicine", 1983. 8. "Surgical treatment of chronic venous insufficiency" V.Ya. Yablokov, M. 2001. 9. "Diseases Backbone s x veins" V.S.Savelev, Z.I.Dumpe et al. M.Meditsina 1992. 10. " Оуқ venalari kasalliklari " R.M.Ahmedov, S.O.Komilov va b., Standard llanma, 2010.
6.	Post - thrombophlebitis scary syndrome. T rhombophlebitis of the superficial and deep veins of the extremities . The disease Paget and - Shretter well .	2			Tables s 11,12,13, 14.15. Flebog Ramm Multimedia	1. "Surgeon Kasalliklar" , Sh. I. Karimov T oshkent . 2005. 2. "Surgeon Kasalliklar", Sh.I.Karimov N.H. Shamirzaev T. 1995. 3. "Surgical diseases" n od ed. M.I. Kuzina, Medicine 2002. 4. Methodological manual for hospital surgery. Nazyrov F.G. et al. T - 2004. 5. Clinical hirur - ogy. Edited by Yu.M. Pantsyrev, M. "Medicine" 1988. 6. "Varicose veins" KN Vedensky, Leningrad, 1983. 7. "Surgical treatment of veins disease of the extremities", AA Klementyev, AN Vedensky, Leningrad "Medicine", 1983. 8. "Surgical treatment of chronic venous insufficiency" V.Ya. Yablokov, M. 2001. 9. "Diseases Backbone s x veins" V.S.Savelev, Z.I.Dumpe et al. M.Meditsina 1992.

						10. " Oyқ venalari kasalliklari " R.M.Ahmedov, S.O.Komilov va b., Standard llanma, 2010.
7.	About occlusive disease of the aortic arch. H e Spec ches t o s aortas o- arteritis. Takayasu syndrome .	2			Tables 11,12,13, 14.15. Multimedia angiograms Educational film	1. "Surgeon Kasalliklar" , Sh. I. Karimov T oshkent . 2005. 2. "Surgeon Kasalliklar", Sh.I.Karimov N.H. Shamirzaev T. 1995. 3. "Surgical diseases" n od ed. M.I. Kuzina, Medicine 2002. 4. Methodological manual for hospital surgery. Nazyrov F.G. et al. T - 2004. 5. Clinical hirur - ogy. Edited by Yu.M. Pantsyrev, M. "Medicine" 1988. 6. "Cardiovascular surgery" V. I. Burakovsky, L. A. Bokeria, M; Medicine, 1989. 7. "Diseases of the aorta and its branches" AV Pokrovsky, M. Medicine, 1979. 8. "Nonspecific aorto- arteritis" (Takayasu's disease) AV Pokrovsky, AE Zotikov, M. 2000. 9. "Guide to angiography" Ch.Kh. Rapkin, M. Meditsina, 1977. 10. "Surgery of the aorta and main arteries", A. Shalimov, N.F.Dryuk, Kiev, "Health", 1977. 11. "Takayasu syndrome" R.M. Akhmedov, S.O.Komilov va b. conv. Allanma, 1910.
eig ht.	About occlusive diseases of the abdominal aorta. Chronic abdominal ischemia syndrome. (SHAI). Simptomati Sa nchez to s Arte rial te a giperten - Zia. Vazorenal te a hypertension.	2			Tables 20,21,22,23. Angiograms	1. Sh. I. Karimov T oshkent . 2005. 2. "Surgeon Kasalliklar", Sh.I.Karimov N.H. Shamirzaev T. 1995. 3. "Surgical diseases" n od ed. M.I. Kuzina, Medicine 2002. 4. Methodological manual for hospital surgery. Nazyrov F.G. et al. T - 2004. 5. Clinical surgery. Edited by Yu.M. Pantsyrev, M. "Medicine" 1988. 6. "Cardiovascular surgery" V. I. Burakovsky, L. A. Bokeria, M; Medicine, 1989. 7. "Ischemic disease of the digestive organs " L.V. Potashev, M.D. Knyazrov et al., Leningrad, 1985. 8. "Acute violation of mesenteric circulation", VS Savelyev, IVSperidonov, I.Meditsina, 1979. 9. "Guide to angiography" Ch.Kh. Rapkin, M. Meditsina, 1977. 10. "Surunkali abdominal ischemia syndromes" R.M.Ahmedov, S.O.Komilov va b. conv. қўllanma , 20 10.
nin e.	Lower limb artery occlusive				Tables s 26,27,28 Echotomo Megaphone	1. Sh. I. Karimov T oshkent . 2005. 2. "Surgeon Kasalliklar", Sh.I.Karimov N.H. Shamirzaev T. 1995. 3. "Surgical

	disease. Lerich e syndrome. Ate obliterating - rosclerosis . O bliter iruyusch y endarteritis. Disease Burger and , disease Reynaud diabetes Sanch ez to th angiop athy.	2			Angiogram s Doppler M ultimedia	diseases" n od ed. M.I. Kuzina, Medicine 2002. 4. Methodological manual for hospital surgery. Nazyrov F.G. et al. T - 2004. 5. Clinical surgery. Edited by Yu.M. Pantsyrev, M. "Medicine" 1988. 6. "Cardiovascular surgery" V. I. Burakovsky, L. A. Bokeria, M; Medicine, 1989. 7. "Surgery of the aorta and main arteries" A.A. Shalimov, N.F.Dryuk, Kiev "Health" 1977. 8. "Guide to angiography" Ch.Kh.Rabkin, M. Medicine, 1977. 9. "Acute obstruction of the aorta bifurcation of the main arteries of the extremities" VS Savelyev, II Zatevakhin et al., M. Medicine, 1987. 10. "Disease of the aorta and its veins" AV Pokrovsky, M. Medicine, 1979. 11. "Oyo κ arterialarning occlusionalovchi casalliklari " R.M.Ahmedov, S.O.Komilov va b. Ullanma, 2010.
1 0.	Acute arterial insufficiency. Acute arterial thrombosis and embolism.	2			Tables 26,27,28 Angiograms Multimedia	1. Sh. I. Karimov T oshkent . 2005. 2. "Surgeon Kasalliklar", Sh.I.Karimov N.H. Shamirzaev T. 1995. 3. "Surgical diseases" n od ed. M.I. Kuzina, Medicine 2002. 4. Methodological manual for hospital surgery. Nazyrov F.G. et al. T - 2004. 5. Clinical hirur - ogy. Edited by Yu.M. Pantsyrev, M. "Medicine" 1988. 6. "Cardiovascular surgery" V. I. Burakovsky, L. A. Bokeria, M; Medicine, 1989. 7. "Surgery of the aorta and main arteries" A.A. Shalimov, N.F.Dryuk, Kiev "Health" 1977. 8. "Guide to angiography" Ch.Kh.Rabkin, M. Medicine, 1977. 9. "Acute obstruction of the aorta bifurcation of the main arteries of the extremities" VS Savelyev, II Zatevakhin et al., M. Medicine, 1987. 10. "Disease of the aorta and its branches" AV Pokrovsky, M. medicine, 1979. 11 . “ Acute disorders of mesenteric circulation ” , VS Savelyev, IVSperidonov, M.Meditsina, 1979. 12. "Acute obstruction of the aorta bifurcation of the main arteries of the extremities" VS Savelyev, II Zatevakhin et al., M. Medicine, 1987. 13. “ Etkir thrombosis va embolia-lar ” R.M. Akhmedov, S.O.Komilov va b. Ullanma, 2010.

1 1.	Congenital malformations hearts	2			Tables 29,30,31,32,33 34 ECG FCG Educational films Multimedia	1. Sh. I. Karimov T oshkent . 2005. 2. "Surgeon Kasalliklar", Sh.I.Karimov N.H. Shamirzaev T. 1995. 3. "Surgical diseases" n od ed. M.I. Kuzina, Medicine 2002. 4. Methodological manual for hospital surgery. Nazyrov F.G. et al. T - 2004. 5. Clinical surgery. Edited by Yu.M. Pantsyrev, M. "Medicine" 1988. 6. "Cardiovascular surgery" V.I.Bura - kovskiy, L.A. Bokeria, M; Medicine, 1989. 8. “Yurakning tuuma poroklari” R.M. Akhmedov, S.O.Komilov va b. Ullanma, 2010.
1 2	Acquired vices hearts	2			Tables 29,30,31,32,33 34 Educational films Multimedia	1. Sh. I. Karimov T oshkent . 2005. 2. "Surgeon Kasalliklar", Sh.I.Karimov N.H. Shamirzaev T. 1995. 3. "Surgical diseases" n od ed. M.I. Kuzina, Medicine 2002. 4. Method - e manual for hospital surgery. Nazyrov F.G. et al. T - 2004. 5. Clinical hirur - ogy. Edited by Yu.M. Pantsyrev, M. "Medicine" 1988. 6. "Cardiovascular surgery" V. I. Burakovsky, L. A. Bokeria, M; Medicine, 1989. 7. "Acquired heart defects" NV Orlova, TV Parenskaya, L. 1979. 8 . "Juracking orttirilgan poroklari" R.M.Akhmedov, S.O.Komilov va b. Ullanma, 2010.
1 3.	Acute and chronic nonspecific purulent lung diseases.	2			Tables 35,36,37,38,39 40.41. Radiographs Educational films Multimedia CT.	1. Sh .. I. Karimov Toshkent. 2005. 2. "Surgeon Kasalliklar", Sh.I.Karimov N.H. Shamirzaev T. 1995. 3. "Surgical diseases" n od ed. M.I. Kuzina, Medicine 2002. 4. Method - e manual for hospital surgery. Nazyrov F.G. et al. T - 2004. 5. Clinical surgery. Edited by Yu.M. Pantsyrev, M. "Medicine" 1988. 6. "Abscesses of the lungs" IS Kolesnikov, BS Vikhriev, L. Medicine, 1973. 7. "Purulent diseases of the lungs and pleura" VI Struchkov, Leningrad, 1967. 8. "Purulent-septic surgery" Stoyan Popkirov, Sofia, 1974. 9. "Purulent surgery" V.I. Struchkov, Moscow, 1962. 10. “pka va pleura yiringli kasal-liklari” R.M.Ahmedov, S.O.Komilov va b. Ullanma, 2010.
fou rtee n.	Diseases of p levra . Acute	2			Tables 37,41,42,43, 44.45.46.47.	1. Sh. I. Karimov T oshkent . 2005. 2. "Surgeon Kasalliklar", Sh.I.Karimov N.H. Shamirzaev T. 1995. 3. "Surgical

	and chronic empyema p levre Bronchopleural and bronchopl eural fistulas .				Uch. films Multimedia Radiographs Bronchoscope Drainage	diseases" n od ed. M.I. Kuzina, Medicine 2002. 4. Toolkit Hospital - Noah surgery. Nazyrov F.G. , T - 2004. 5. Clinical surgery. Edited by Yu.M. Pantsyrev, M. "Medicine" 1988. 6. "Abscesses of the lungs" IS Kolesnikov, BS Vikhriev, L. Medicine, 1973. 7. "Purulent diseases of the lungs and pleura" VI Struchkov, Leningrad, 1967. 8. "Purulent-septic surgery" Stoyan Popkirov, Sofia, 1974. 9. "Purulent surgery" V.I. Struchkov, Moscow, 1962. 10. “pka va pleura yiringli kasal-liklari” R.M. Akhmedov, S.O.Komilov va boshqalar usl.kўllanma, 2010.
fift een	Diseases of the mediastinum. Cysts of the lung and mediastinum. Benign tumors of the mediastinum. Myasthenia gravis. Mediast enitis.	2			Tables 42.43.44.45 46.47 X-ray Computed tomography Bronchoscope	1. Sh. I. Karimov T oshkent . 2005. 2. "Surgeon Kasalliklar", Sh.I.Karimov N.H. Shamirzaev T. 1995. 3. "Surgical diseases" n od ed. M.I. Kuzina, Medicine 2002. 4. Methodological manual for hospital surgery. Nazyrov F.G. et al. T - 2004. 5. Clinical surgery. Edited by Yu.M. Pantsyrev, M. "Medicine" 1988. 6. "Surgery of the mediastinum" A.A. Vishnevsky, A.A. Adamyan, Moscow 1977. 7. "Tumors cysts s mediastinal" V.Goldberg; G.A. Lavnikova, Moscow, 1965. 8 . Ўpkaning havfsiz ўsmalari va kistalari ”R.M.Ahmedov, S.O.Komilov va b. Ullanma, 2010.
sixt een .	Disease of diaphragm s . ... Diaphragm fla x hernia. Relaxation of diaphragms .	2			Tables 42.43.44.45 46.47 Radiographs Computed tomography Multimedia	1. Sh. I. Karimov T oshkent . 2005. 2. "Surgeon Kasalliklar", Sh.I.Karimov N.H. Shamirzaev T. 1995. 3. "Surgical diseases" n od ed. M.I. Kuzina, Medicine 2002. 4. Methodological manual for hospital surgery. Nazyrov F.G. et al. T - 2004. 5. Clinical surgery. Edited by Yu.M. Pantsyrev, M. "Medicine" 1988. 6. “ Hernia of the esophageal opening of the diaphragm ” V.Kh. Vasilenko, A.L. Grebnev, M.Meditsina, 1978. 7. "Guide to surgery" BV Petrovsky, M. Medicine, 1966. 8. “ Churra lari diaphragm ” R.M.Ahmedov, S.O.Komilov va b. Ullanma, 2010.
17.	Postgastroreze ktsion nye syn drome s .	2			Table 42.43.44.45 46.47 Multimedia	1. Sh. I. Karimov T oshkent . 2005. 2. "Surgeon Kasalliklar", Sh.I.Karimov N.H. Shamirzaev T. 1995. 3. "Surgical diseases" n od ed. M.I. Kuzina, Medicine 2002.

						4. Methodological manual for hospital surgery. Nazyrov F.G. et al. T - 2004. 5. Clinical surgery. Edited by Yu.M. Pantsyrev, M. "Medicine" 1988. 6. “Postvagotomy syndromes and their chir. correction "V.V.Vakhidov, A.M. Khodzhibaev, Tashkent, 1993. 7. "Surgery of peptic ulcer and 12 p.p. intestines" AF Chernousov et al., M.1996. 8. "Guide to surgery" BV Petrovsky, M. Medicine, 1966. 9 . “ Osh κ ozonning operationdan keyings casalliklari” R.M.Ahmedov, S.O.Komilov va b. Ullanma, 2010.
18.	Postcholecystectomy syndrome. Obstructive jaundice.	2			Tables 52,53,54,55, 56.57. Multimedia Uch. films CT, UTT, RPHG, CPHG, Cholangiograms, Bouzhy	1. Sh. I. Karimov T oshkent . 2005. 2. "Surgeon Kasalliklar", Sh.I.Karimov N.H. Shamirzaev T. 1995. 3. "Surgical diseases" n od ed. M.I. Kuzina, Medicine 2002. 4. Method - e manual for hospital surgery. Nazyrov F.G. et al. T - 2004. 5. Clinical surgery. Edited by Yu.M. Pantsyrev, M. "Medicine" 1988. 6. "Guide to surgery" BV Petrovsky, M. Medicine, 1966. 7. "Postcholecystectomy syndrome and repeated operations on the biliary tract " VM Sitenko, AI Nechai, Lehning. " Medicine ", 1977. 8. "Surgery of the liver and biliary tract" A.A. Shalimov et al. Kiev 1993. 9. "Disease of the liver and biliary tract" N. Sherdock, J. Dooley, M. Medicine, 1999. 10 . "Postcholecystectomyadan keyingi syndrome" R.M.Akhmedov, I.A.Mirkhuzhaev va b. Ullanma, 2010.
	AND T ABOUT:	**36 hours**				

CALEDAR-THEMATIC PLAN OF PRACTICAL HOSPITAL SURGERY CLASSES

No.	Topic name	Clock	Group - py	Time	Visual material	Used literature
1	Introduction. Understanding the subject of Hospital Surgery and writing a medical history. Acquaintance with the special lysed department niyami. Pain management them. Anamnesis and objective	4			Tables 1,2.3.4. Multimedia X - ray - gram s	1. "Kasallik surgeon - liklar " , Sh. I. Karimov T oshkent . 2005. 2. "Surgeon Kasalliklar", Sh.I.Karimov N.H. Shamirzaev T. 1995. 3. "Surgical diseases" n od ed. M.I. Kuzina, Medicine 2002. 4. Methodological manual for

	examination. Anatomical and physiological concept of the esophagus. Special methods of examination, classification of food-water diseases. **Interactive method:** "Incident"					hospital surgery. Nazyrov F.G. et al. T - 2004. 5. Clinical surgery. Edited by Yu.M. Pantsyrev, M. "Medicine" 1988. 6. A.A. Shalimov "Surgery food - water" 7. G. L. Ratner "Burns of the esophagus" . VS Savelyev , AI Kirienko " Surgical diseases ", M .; 2006; T- II ; 13 - 54 betlar. 8. "Қizilчngach kasalliklari", R.M. Akhmedov; S.O.Komilov va b., Conv. Қўl. 2010y.
2	The main manifestations of diseases of the esophagus. Chemistry esophagus burns chemical substances and Cicatricial narrowing, di-the esophagus vertical. Kli nickname, diagnostics, con servative and operative new treatment. Types of outcomes of surgical interventions. **Interactive method:** "Situational task"	4			Table 1,2.3.4. Multimedia X - ray - gram s Educational film	1. "Kasallik surgeon - liklar " , Sh. I. Karimov T oshkent . 2005. 2. "Surgeon Kasalliklar", Sh.I.Karimov N.H. Shamirzaev T. 1995. 3. "Surgical diseases" n od ed. M.I. Kuzi na, Medicine 2002. 4. Methodological manual for hospital surgery. Nazyrov F.G. et al. T - 2004. 5. Clinical surgery. Edited by Yu.M. Pantsyrev, M. "Medicine" 1988. 6. A.A. Shalimov "Surgery food - water" 7. G. L. Ratner "Burns of the esophagus" . VS Savelyev , AI Kirienko " Surgical diseases ", M .; 2006; T- II ; 13 - 54 betlar. 8. "Diverticulum of the esophagus" BV Petrovsky, EN Vantsyan, M. Meditsina, 1968. 9 . "Қizilўngach kasalliklari", R.M. Akhmedov; S.O.Komilov va b., Conventional l. 2010y.
3	Disease of the esophagus. The concept of cardiospasm Clinic, diagnostics and differential diagnostics. Technique of cardiodilation. Types of operations foreign body of the esophagus. Endoscopic and surgical methods of treatment. **Interactive method:**	4			Tables 1.5. Multimedia R -gram lar	1. "Kasallik surgeon - liklar " , Sh. I. Karimov T oshkent . 2005. 2. "Surgeon Kasalliklar", Sh.I.Karimov N.H. Shamirzaev T. 1995. 3. "Surgical diseases" n od ed. M.I. Kuzi na, Medicine 2002. 4. Methodical Pozo - Bie for hospital surgery. Nazyrov F.G. et al. T - 2004. 5. Clinical hirur - ogy. Edited by Yu.M.

	"Incident"					Pantsyrev, M. "Medicine" 1988. 6. A.A. Shalimov "Surgery food - water" 7. “Cardiospasm”, GD Vilyavin, VI Soloviev et al., M. 1971. 8. "Foreign body and food-water trauma " BS Rozanov, M. "Medgiz", 1961.
4	Portal hypertensive zia and its complication. Etiology, pathogenesis, clinical picture and diagnosis of portal hypertension. Conservativ e and surgical methods of treatment. Indication for surgery. Types of operations. Buddy-Chiari syndrome. Clinic, diagnosis and treatment. **Interactive method:** "Situational task"	4			Tables 6.7. Multimedia CT, UTT	1. "Kasallik surgeon - liklar " , Sh. I. Karimov T oshkent . 2005. 2. "Surgeon Kasalliklar", Sh.I.Karimov N.H. Shamirzaev T. 1995. 3. "Surgical diseases" n od ed. M.I. Kuzi na, Medicine 2002. 4. Methodical Pozo - Bie for hospital surgery. Nazyrov F.G. et al. T - 2004. 5. Clinical surgery. Edited by Yu.M. Pantsyrev, M. "Medicine" 1988. 6. "Surgery of portal hypertension" by M.D. Patsiora, Moscow, 1984. 7. "Surgery of complications of portal hypertension in patients with liver cirrhosis." F.G.Nazyrov, Kh.A. Akilov et al., "GZOTARMED", 2002. 8. "Chronic hepatitis and cirrhosis" AS Loginov, Yu.E. Blok, M.2002. 9. "Portal hypertension", R.M. Akhmedov, S.O.Komilov va b. conv- llanma, 2010.
five	Echinococcosis. The concept of parasitic diseases. Parasite characteristics. Echinococ cosis and alveococcosis of the liver. Clinic, diagnostics and differential diagnostics. Types of surgical treatment and their outcomes. Surgical treatment of combined echinococcus-goose of the liver and lungs and its sequence. **Interactive method:** "Storming the Brain"	4			Tables 8,9,10. MultimediaUTT , CT	1. "Kasallik surgeon - liklar " , Sh. I. Karimov T oshkent . 2005. 2. "Surgeon Kasalliklar", Sh.I.Karimov N.H. Shamirzaev T. 1995. 3. "Surgical diseases" n od ed. M.I. Kuzi na, Medicine 2002. 4. Methodological manual for hospital surgery. Nazyrov F.G. et al. T - 2004. 5. Clinical surgery. Edited by Yu.M. Pantsyrev, M. "Medicine" 1988. 6. "Surgery of echinococcosis", BV Petrovsky, OB Milonov et al., M. Medicine, 1985. 7. "Surgery of the liver and

						bile ducts " , AA Shalimov et al., Kiev, 1993. 8. "Surgery of the liver and biliary tract" BI Alperovich, M.1997.
6	Varicose veins. Anatomical and physiological data of the veins of the lower extremities. Special new types of surveys. Varicose veins veins of the lower extremities tei. Clinic and diagnosis tick. Etiopathogenesis and treatment. Syndrome of Parks-Weber-Ruba-seams, Klipel-Trenonne Paget-Schrötter. **Interactive method:** "Research problem"	4			Tables 11,12,13, 14.15. Phlebogram Multimedia	1. "Surgeon Kasalliklar" , Sh. I. Karimov T oshkent . 2005. 2. "Surgeon Kasalliklar", Sh.I.Karimov N.H. Shamirzaev T. 1995. 3. "Surgical diseases" n od ed. M.I. Kuzi na, Medicine 2002. 4. Methodological manual for hospital surgery. Nazyrov F.G. et al. T - 2004. 5. Clinical hirur - ogy. Edited by Yu.M. Pantsyrev, M. "Medicine" 1988. 6. "Varicose veins" KN Vedensky, Leningrad, 1983. 7. "Surgical treatment of veins disease of the extremities", AA Klementyev, AN Vedensky, Leningrad "Medicine", 1983. 8. " Oyқ venalari kasalliklari " R.M.Ahmedov, S.O.Komilov va b., Standard llanma, 2010.
7	Thrombosis and thrombosis bits of superficial and deep veins of the lower extremities. Clinic and diagnostics. Postthrom bophlebitic syndrome. (PTFS). Conce pt, etiopathogenesis, clinical picture and treatment, prevention of PTFS. **Interactive method:** "Storming the Brain"	4			Table 11,12,13, 14.15. Phlebogram Multimedia	1. "Surgeon Kasalliklar" , Sh. I. Karimov T oshkent . 2005. 2. "Surgeon Kasalliklar", Sh.I.Karimov N.H. Shamirzaev T. 1995. 3. "Surgical diseases" n od ed. M.I. Kuzi na, Medicine 2002. 4. Methodological manual for hospital surgery. Nazyrov F.G. et al. T - 2004. 5. Clinical hirur - ogy. Edited by Yu.M. Pantsyrev, M. "Medicine" 1988. 6. "Varicose veins" KN Vedensky, Leningrad, 1983. 7. "Surgical treatment of veins disease of the extremities", AA Klementyev, AN Vedensky, Leningrad "Medicine", 1983. 8. "Surgical treatment of chronic venous insufficiency" V.Ya. Yablokov, M. 2001.

						9. "Diseases Backbone s x veins" V.S.Savelev, Z.I.Dumpe et al. M.Meditsina 1992. 10. " Oyқ venalari kasalliklari " R.M.Ahmedov, S.O.Komilov va b., Standard llanma, 2010.
eig ht	Anatomical and physiological information about the aorta and arterial system. Etiology pathogenesis occlusion arterial lesions alial vessels. Specific optimal methods of examination arterial patients mental pathology and stages. Nespetsifiches - ki nd aortoarteriit. Types of surgical interventions (sympathect omy, end artectomy, prosthetics and shunting). **Interactive method:** "Incident"	4			Table 11,12,13, 14.15. Multimedia Angiogram Educational film	1. "Surgeon Kasalliklar" , Sh. I. Karimov T oshkent . 2005. 2. "Surgeon Kasalliklar", Sh.I.Karimov N.H. Shamirzaev T. 1995. 3. "Surgical diseases" n od ed. M.I. Kuzi na, Medicine 2002. 4. Methodological manual for hospital surgery. Nazyrov F.G. et al. T - 2004. 5. Clinical hirur - ogy. Edited by Yu.M. Pantsyrev, M. "Medicine" 1988. 6. "Cardiovascular surgery" V. I. Burakovsky, L. A. Bokeria, M; Medicine, 1989. 7. "Diseases of the aorta and its branches" AV Pokrovsky, M. Medicine, 1979. 8. "Nonspecific aorto-arteritis" AV Pokrovsky, AE Zotikov, M. 2000. 9. "Guide to angiography" Ch.Kh. Rapkin, M. Meditsina, 1977. 10. "Surgery of the aorta and main arteries", AA Shalimov, NF Dryuk, Kiev, "Health", 1977.
nin e	Occlusive disease of the aortic arch. Takayasu Syndrome. Etiopathogene sis, clinical forms, methods of treatment. Surgical treatments. **Interactive method:** "Storming the Brain"	4			Table 16.1718.19. Multimedia Angiogram Educational film	1. "Surgeon Kasalliklar" , Sh. I. Karimov T oshkent . 2005. 2. "Surgeon Kasalliklar", Sh.I.Karimov N.H. Shamirzaev T. 1995. 3. "Surgical diseases" n od ed. M.I. Kuzi na, Medicine 2002. 4. Methodological manual for hospital surgery. Nazyrov F.G. et al. T - 2004. 5. Clinical hirur - ogy. Edited by Yu.M. Pantsyrev, M. "Medicine" 1988. 6. "Cardiovascular surgery" V. I. Burakovsky, L. A. Bokeria, M; Medicine, 1989. 7. "Diseases of the aorta and its branches" AV Pokrovsky,

						M. Medicine, 1979. 8. "Nonspecific aorto-arteritis" (Takayasu's disease) AV Pokrovsky, AE Zotikov, M. 2000. 9. "Guide to angiography" Ch.Kh. Rapkin, M. Meditsina, 1977. 10. "Surgery of the aorta and main arteries", AA Shalimov, NF Dryuk, Kiev, "Health", 1977. 11. "Takayasu syndrome" R.M. Akhmedov, S.O.Komilov va b. conv. Allanma, 1910.
ten	Occlusive disease of the abrasive aorta. High, middle and bottom ny occlusion of the abrasive aorta. Etiopathogenesis. Chronic abdominal ischemia syndrome (ACAI). Etiology, classification and methods of surgical treatment **Interactive method:** "Situational tasks".	4			Tables 20,21,22,23. Angiogram	1. Sh. I. Karimov T oshkent . 2005. 2. "Surgeon Kasalliklar", Sh.I.Karimov N.H. Shamirzaev T. 1995. 3. "Surgical diseases" n od ed. M.I. Kuzi na, Medicine 2002. 4. Methodological manual for hospital surgery. Nazyrov F.G. et al. T - 2004. 5. Clinical hirur - ogy. Edited by Yu.M. Pantsyrev, M. "Medicine" 1988. 6. "Cardiovascular surgery" V. I. Burakovsky, L. A. Bokeria, M; Medicine, 1989. 7. "Ischemic disease of the digestive organs " L.V. Potashev, M.D. Knyazrov et al., Leningrad, 1985. 8. "Acute violation of mesenteric circulation", VS Savelyev, IVSperidonov, I.Meditsina, 1979. 9. "Guide to angiography" Ch.Kh. Rapkin, M. Meditsina, 1977. 10. "Surunkali abdominal ischemia syndromes" R.M.Ahmedov, S.O.Komilov va b. conv. Allanma, 20 10.
ele ven	Symptomatic arterial hypertension. Vasoreal hypertension syndrome. Etiopathogenesis, clinical presentation, diagnosis and surgical treatment. Pheochromosit oma. Coma syndrome. Itsenko-Cushing	4			Tables 20,21,22,23. Angiogram Multimedia	1. Sh. I. Karimov T oshkent . 2005. 2. "Surgeon Kasalliklar", Sh.I.Karimov N.H. Shamirzaev T. 1995. 3. "Surgical diseases" n od ed. M.I. Kuzi na, Medicine 2002. 4. Methodological manual for hospital surgery. Nazyrov F.G. et al. T - 2004.

	syndrome. Diagnostics, treatment methods. New technologies. **Interactive method:** Brain storm					5. Clinical hirur - ogy. Edited by Yu.M. Pantsyrev, M. "Medicine" 1988. 6. "Cardiovascular surgery" V. I. Burakovsky, L. A. Bokeria, M; Medicine, 1989. 7. "Ischemic disease of the digestive organs " L.V. Potashev, M.D. Knyazrov et al., Leningrad, 1985. 8. "Surgery of the aorta and main arteries" A.A. Shalimov, N.F.Dryuk, Kiev "Health" 1977.
12	Occlusive diseases of the lower extremities. Leriche syndrome. Burger's disease, Raynaud's disease. Diabetic angiopathy. Clinic, diagnostics, differential. diagnostics. S urgical tactics, treatment methods. Atherosclerosis obliterans. Ob-literating endoarthritis. **Interactive method:** Brain storm	4			Tables 26,27,28 Echotomo Megaphone Angiograms Doppler Multimedia	1. Sh. I. Karimov T oshkent . 2005. 2. "Surgeon Kasalliklar", Sh.I.Karimov N.H. Shamirzaev T. 1995. 3. "Surgical diseases" n od ed. M.I. Kuzi na, Medicine 2002. 4. Methodological manual for hospital surgery. Nazyrov F.G. et al. T - 2004. 5. Clinical surgery. Edited by Yu.M. Pantsyrev, M. "Medicine" 1988. 6. "Cardiovascular surgery" V. I. Burakovsky, L. A. Bokeria, M; Medicine, 1989. 7. "Surgery of the aorta and main arteries" A.A. Shalimov, N.F.Dryuk, Kiev "Health" 1977. 8. "Guide to angiography" Ch.Kh.Rabkin, M. Medicine, 1977. 9. "Acute obstruction of the aorta bifurcation of the main arteries of the extremities" VS Savelyev, II Zatevakhin et al., M. Medicine, 1987. 10. "Disease of the aorta and its veins" AV Pokrovsky, M. Medicine, 1979. 11. "Oyo қ arterialarning occlusi onalovchi casalliklari " R.M.Ahmedov, S.O.Komilov va b. Ullanma, 2010.
13	Acute arterial insufficiency. Acute thrombosis and embo Leah. Etiopathogenesis. F ak thrombus formation tori bose. Clinical leak nie. Conservative and	five			Tables 26,27,28 Angiogram Multimedia	1. Sh. I. Karimov T oshkent . 2005. 2. "Surgeon Kasalliklar", Sh.I.Karimov N.H. Shamirzaev T. 1995. 3. "Surgical diseases" n od ed. M.I. Kuzi na, Medicine 2002. 4. Methodological manual

	surgical treatment. Principles of anticoagulant therapy. **Interactive method:** "Situational task"					for hospital surgery. Nazyrov F.G. et al. T - 2004. 5. Clinical hirur - ogy. Edited by Yu.M. Pantsyrev, M. "Medicine" 1988. 6. "Cardiovascular surgery" V. I. Burakovsky, L. A. Bokeria, M; Medicine, 1989. 7. "Surgery of the aorta and main arteries" AA Shalimov, NF Dryuk, Kiev "Health" 1977. 8. "Guide to angiography" Ch.H. Rabkin, M. Medicine, 1977. 9. "Acute obstruction of the aorta bifurcation of the main arteries of the extremities" VS Savelyev, II Zatevakhin et al., M. Medicine, 1987. 10. "Disease of the aorta and its branches" AV Pokrovsky, M. medicine, 1979. 11 . “ Acute disorders of mesenteric circulation ” , VS Savelyev, IVSperidonov, M.Meditsina, 1979. 12. "Acute obstruction of the aorta bifurcation of the main arteries of the extremities" VS Savelyev, II Zatevakhin et al., M. Medicine, 1987. 13. “ Etkir thrombosis va embolia-lar ” R.M. Akhmedov, S.O.Komilov va b. Ullanma, 2010.
fou rtee n	Congenital heart defects. Anatomical-physio- logical information. "White" and "Blue" poros Ki. Open artery nye vice Triad, Tet-rada Fallot and pentad. Luthenbacher's syndrome. Cortation of the aorta. Clinic, diagnostics. **Interactive method:** "Incident"	five			Tables 29,30,31,32,33 34 ECG FCG Educational film Multimedia	1. Sh. I. Karimov T oshkent . 2005. 2. "Surgeon Kasalliklar", Sh.I.Karimov N.H. Shamirzaev T. 1995. 3. "Surgical diseases" n od ed. M.I. Kuzi na, Medicine 2002. 4. Methodological manual for hospital surgery. Nazyrov F.G. et al. T - 2004. 5. Clinical hirur - ogy. Edited by Yu.M. Pantsyrev, M. "Medicine" 1988. 6. "Cardiovascular surgery" V. I. Burakovsky, L. A. Bokeria, M; Medicine, 1989. 8. “Yurakning tuuma poroklari” R.M. Akhmedov, S.O.Komilov va b. Ullanma, 2010.

fift een	Acquired heart defects. Vices mitral leg, aortic and three-cuspid valve. Etiopathogenesis, impaired blood circulation. Clinical picture, diagnostics and indications and contraindications for surgical treatment. Open and closed methods of surgical treatment **Interactive method:** "Brain storm".	five			Table 29,30,31,32,33 34 Educational film Multimedia	1. Sh. I. Karimov T oshkent . 2005. 2. "Surgeon Kasalliklar", Sh.I.Karimov N.H. Shamirzaev T. 1995. 3. "Surgical diseases" n od ed. M.I. Kuzi na, Medicine 2002. 4. Methodological manual for hospital surgery. Nazyrov F.G. et al. T - 2004. 5. Clinical hirur - ogy. Edited by Yu.M. Pantsyrev, M. "Medicine" 1988. 6. "Cardiovascular surgery" V. I. Burakovsky, L. A. Bokeria, M; Medicine, 1989. 7. "Acquired heart defects" NV Orlova, TV Parenskaya, L. 1979. 8 . "Juracking orttirilgan poroklari" R.M.Akhmedov, S.O.Komilov va b. Ullanma, 2010.
sixt een	Anatomy and physiology of the lungs. Survey methods. Acute nonspecific abscess and gangrene of the lungs. Etiology, pathogenesis, clinical picture, diagnosis and methods of treatment. **Interactive method:** Brain storm	five			Tables 35,36,37,38,39 40.41. X-ray Educational film Multimedia CT.	1. Sh .. I. Karimov Toshkent. 2005. 2. "Surgeon Kasalliklar", Sh.I.Karimov N.H. Shamirzaev T. 1995. 3. "Surgical diseases" n od ed. M.I. Kuzi na, Medicine 2002. 4. Methodological manual for hospital surgery. Nazyrov F.G. et al. T - 2004. 5. Clinical surgery. Edited by Yu.M. Pantsyrev, M. "Medicine" 1988. 6. "Abscesses of the lungs" IS Kolesnikov, BS Vikhriev, L. Medicine, 1973. 7. "Purulent diseases of the lungs and pleura" VI Struchkov, Leningrad, 1967. 8. "Purulent-septic surgery" Stoyan Popkirov, Sofia, 1974. 9. "Purulent surgery" V.I. Struchkov, Moscow, 1962. 10. “pka va pleura yiringli kasal-liklari” R.M.Ahmedov, S.O.Komilov va b. Ullanma, 2010.
17	Non-specific lung diseases. Chronic nonspecific lung abscess. Etiology, Clinical ka, diagnostics,	five			Tables 35,36,37,38,39 40.41. X-ray Bronchoscope	1. Sh .. I. Karimov Toshkent. 2005. 2. "Surgeon Kasalliklar", Sh.I.Karimov N.H. Shamirzaev T. 1995. 3. "Surgical

	conservation active and operational methods of treatment. Bronchoectatic disease. Clinic and methods of treatment. **Interactive method:** "Situational task"				CT scan	diseases" n od ed. M.I. Kuzi na, Medicine 2002. 4. Methodological manual for hospital surgery. Nazyrov F.G. et al. T - 2004. 5. Clinical surgery. Edited by Yu.M. Pantsyrev, M. "Medicine" 1988. 6. "Abscesses of the lungs" IS Kolesnikov, BS Vikhriev, L. Medicine, 1973. 7. "Purulent diseases of the lungs and pleura" VI Struchkov, Leningrad, 1967. 8. "Purulent-septic surgery" Stoyan Popkirov, Sofia, 1974. 9. "Purulent surgery" V.I. Struchkov, Moscow, 1962. 10. “pka va pleura yiringli kasal-liklari” R.M.Ahmedov, S.O.Komilov va b. Ullanma, 2010.
18	Pleural disease. Anatomical and physiological information about the pleura. Acute pleural empyema and types. Etiopathogenesis and treatment methods. Chro- nic empyema of the pleura. Etiopathognosis, clinical picture, differential diagnosis. Bronchopleural and broncho-pleurotoro-fecal fistulas. Ways of surgical treatment. Thoraco-plasty methods. **Interactive method:** "Incident"	five			Tables 37,41,42,43, 44.45.46.47. Educational film Multimedia X-ray Bronchoscope	1. Sh. I. Karimov T oshkent . 2005. 2. "Surgeon Kasalliklar", Sh.I.Karimov N.H. Shamirzaev T. 1995. 3. "Surgical diseases" n od ed. M.I. Kuzi na, Medicine 2002. 4. Methodological manual for hospital surgery. Nazyrov F.G. et al. T - 2004. 5. Clinical surgery. Edited by Yu.M. Pantsyrev, M. "Medicine" 1988. 6. "Abscesses of the lungs" IS Kolesnikov, BS Vikhriev, L. Medicine, 1973. 7. "Purulent diseases of the lungs and pleura" VI Struchkov, Leningrad, 1967. 8. "Purulent-septic surgery" Stoyan Popkirov, Sofia, 1974. 9. "Purulent surgery" V.I. Struchkov, Moscow, 1962. 10. “pka va pleura yiringli kasal-liklari” R.M.Ahmedov, S.O.Komilov va b. Ullanma, 2010.
nin ete en	Lung cysts. Classify cation .. Pneumothorax and pyopneumoto rax. Concept, types, at ranks. Clinic and treatment	five			Tables 37,41,42,43, 44.45.46.47. X-ray gram Multimedia Computer	1. Sh. I. Karimov T oshkent . 2005. 2. "Surgeon Kasalliklar", Sh.I.Karimov N.H. Shamirzaev T. 1995. 3. "Surgical diseases" n od ed. M.I. Kuzi

	nie. Echinococcus of the lungs. Etiopathogenesis, complications. Operation methods and outcomes. **Interactive method:** Brain storm				tomography	na, Medicine 2002. 4. Methodological manual for hospital surgery. Nazyrov F.G. et al. T - 2004. 5. Clinical surgery. Edited by Yu.M. Pantsyrev, M. "Medicine" 1988. 6. "Abscesses of the lungs" IS Kolesnikov, BS Vikhriev, L. Medicine, 1973. 7. "Purulent diseases of the lungs and pleura" VI Struchkov, Leningrad, 1967. 8. "Purulent surgery" V.I. Struchkov, Moscow, 1962. 9. "Surgery of echinococcosis" BV Petrovsky, OB Milonov and others M. Meditsina, 1985. 10. "канирkaning havfsiz ўsmalari va kistalari" R.M.Ahmedov, S.O.Komilov va b. Ullanma, 2010.
2 0	Mediastinal disease. Anatomical and physiological information of the mediastinal organs. Would about the survey. Benign tumors and cysts of the mediastinum. Classificati on, diagnostics, treatment. Mediastinitis. E tiology, clinic, diagnosis and treatment. Myasthenia gravis, diagnostics, methods of treatment. **Interactive method:** "Incident"	five			Tables 42.43.44.45 46.47 X-ray Compu- thorny tomography Bronchoscope Drainage	1. Sh. I. Karimov T oshkent . 2005. 2. "Surgeon Kasalliklar", Sh.I.Karimov N.H. Shamirzaev T. 1995. 3. "Surgical diseases" n od ed. M.I. Kuzi na, Medicine 2002. 4. Methodological manual for hospital surgery. Nazyrov F.G. et al. T - 2004. 5. Clinical surgery. Edited by Yu.M. Pantsyrev, M. "Medicine" 1988. 6. "Surgery of the mediastinum" A.A. Vishnevsky, A.A. Adamyan, Moscow 1977. 7. "Tumors cysts s mediastinal" V.Goldb erg; G.A. Lavnikova, Moscow, 1965. 8 . "Ўpka va k ў ks yelling ning havfsiz ўsmalari va kistalari" R.M.Ahmedov, S.O.Komilov va b. Ullanma, 2010.
2 1	Diaphragm disease. Diaphragmatic hernia. Anatomical and physiological properties of the diaphragm. Clinical manifestations different diaphragms small hernias. Hernia of the esophageal opening of	five			Tables 42.43.44.45 46.47 X-ray Computed tomography Multimedia	1. Sh. I. Karimov T oshkent . 2005. 2. "Surgeon Kasalliklar", Sh.I.Karimov N.H. Shamirzaev T. 1995. 3. "Surgical diseases" n od ed. M.I. Kuzi na, Medicine 2002. 4. Methodological manual for

	the diaphragm. Relaxation of the diaphragm. Types of plastic diaphragms. **Interactive method:** "Incident"					hospital surgery. Nazyrov F.G. et al. T - 2004. 5. Clinical surgery. Edited by Yu.M. Pantsyrev, M. "Medicine" 1988. 6. " Hernia of the esophageal opening of the diaphragm " V.Kh. Vasilenko, A.L. Grebnev, M.Meditsina, 1978. 7. "Guide to surgery" BV Petrovsky, M. Medicine, 1966. 8. " Churra lari diaphragm " R.M.Ahmedov, S.O.Komilov va b. Ullanma, 2010.
2 2	Diseases operated on th stomach. Postgastro-resection syndrome. Classification, clinics, causes, diagnostics. Indication for surgical treatment. Types of reconstructive operations. Postvagotomic syndrome. Treatment. **Interactive method:** "Incident"	five			Tables 42.43.44.45 46.47 Computer tomography Diashgrams Multimedia	1. Sh. I. Karimov T oshkent . 2005. 2. "Surgeon Kasalliklar", Sh.I.Karimov N.H. Shamirzaev T. 1995. 3. "Surgical diseases" n od ed. M.I. Kuzi na, Medicine 2002. 4. Methodological manual for hospital surgery. Nazyrov F.G. et al. T - 2004. 5. Clinical surgery. Edited by Yu.M. Pantsyrev, M. "Medicine" 1988. 6. "Postvagotomy syndromes and their chir. correction "V.V.Vakhidov, A.M. Khodzhibaev, Tashkent, 1993. 7. "Surgery of peptic ulcer and 12 p.p. intestines" AF Chernousov et al., M.1996. 8. "Guide to surgery" BV Petrovsky, M. Medicine, 1966. 9 . " Osh қ ozonning operationdan keyings casalliklari" R.M.Ahmedov, S.O.Komilov va b. Ullanma, 2010.
2 3	Postcholecystectomy syndrome. Concept. Class ification. Etiopia is togene sis. Diagnostics and diff. Diagnosis in post - cholecystectomy syndrome. Types recon - struktvnyh and operative - GOVERNMENTAL inte rventions	five			Tables 52,53,54,55, 56.57. Multimedia Educational film, CT, UTT, RPHG, CPHG, Cholangiogram, bougie	1. Sh. I. Karimov T oshkent . 2005. 2. "Surgeon Kasalliklar", Sh.I.Karimov N.H. Shamirzaev T. 1995. 3. "Surgical diseases" n od ed. M.I. Kuzina, Medicine 2002. 4. Methodological manual for hospital surgery. Nazyrov F.G. et al. T - 2004. 5. Clinical surgery. Edited by Yu.M. Pantsyrev, M. "Medicine" 1988. 6. "Guide to surgery" BV

	Interactive method: "Incident"					Petrovsky, M. Medicine, 1966. 7. "Postcholecystectomy syndrome and repeated operations on the biliary tract " VM Sitenko, AI Nechai, Lehning. " Medicine ", 1977. 8. "Surgery of the liver and biliary tract" AA Shalimov et al. Kiev 1993. 9. "Disease of the liver and biliary tract" N. Sherdock, J. Dooley, M. Medicine, 1999. 10 . "Postcholecystectomyadan keyingi syndrome" R.M.Akhmedov, I.A.Mirkhuzhaev va b. conventional allanma,
24.	Obstructive jaundice. Types and reason. Methods and diagnostics. Surgical treatment methods. Cholangitis. Tre atment methods. **Interactive method:** "Incident"	five			Multimedia Educational film CT, UTT, RPHG, CPHG, Cholangiogram, buzhy	1. Sh. I. Karimov T oshkent . 2005. 2. "Surgeon Kasalliklar", Sh.I.Karimov N.H. Shamirzaev T. 1995. 3. "Surgical diseases" n od ed. M.I. Kuzina, Medicine 2002. 4. Methodological manual for hospital surgery. Nazyrov F.G. et al. T - 2004. 5. Clinical surgery. Edited by Yu.M. Pantsyrev, M. "Medicine" 1988. 6. "Guide to surgery" BV Petrovsky, M. Medicine, 1966. 7. "Postcholecystectomy syndrome and repeated operations on the biliary tract " VM Sitenko, AI Nechai, Lehning. " Medicine ", 1977. 8. "Surgery of the liver and biliary tract" AA Shalimov et al. Kiev 1993. 9. "Disease of the liver and biliary tract" N. Sherdock, J. Dooley, M. Medicine, 1999. 10. "Practical guide to surgical diseases" VG Astapenko, Minsk, 1984, 2-part 11. "Surgery of the liver and biliary tract" B. I. Alperovich, M. Med. 1997. 10 . "Postcholecystectomyadan keyingi syndrome" R.M.Akhmedov, I.A.Mirkhuzhaev va b. Ullanma, 2010.
	TOTAL:	**108 hours**				

LIST independence 's WORK IN HOSPITAL SURGERY

11111 No.	Topic name	Clock	Brief annotation
1	Operating principles of the surgical department	2	Organization of activities in the surgical department. Otlichatelnye signs surgical separation I

			operationally dressings s m unit therapeutic. Administration of patients in the postoperative period. Inpatient and spa treatment. The principles of rehabilitation therapy. Principles of VKK and VTEK.
2	Damage to the vertebral arteries.	2	Damage to the vertebral arteries. Their role in the impaired blood circulation in the brain. Clinical signs, diagnosis and differential diagnosis. The role of angiography in the diagnosis of brain diseases. The principles of surgical treatment. Principles of " Steal " syndrome.
3	Critical ischemia of the upper and lower extremities.	2	Critical ischemia of the upper and lower extremities. Etiopathogenesis. The principles of conservative treatment. Types of surgical treatment and indications for it.
4	Raynaud's disease. Burger's disease.	2	Raynaud's disease. Burger's disease . Etiopathogenesis, clinical signs, diagnosis and diff. diagnostics. Methods of instrumental diagnostics and their informational content of treatment. Surgical treatment methods. Outcomes of conservative and surgical treatment.
five	Modern sclerotherapy.	2	Modern sclerotherapy. Kinds. Indications. Methodology. The effectiveness of the treatment. Possible complications.
6 .	Paget-Schrötter disease.	2	Paget-Schrötter disease. Etiopathogenesis, clinical signs, diagnosis and diff. diagnostics. The role of phlebography in the diagnosis of the disease. Treatment. Results of conservative and surgical treatment.
7.	Ileofemoral venous thrombosis.	2	Ileofemoral venous thrombosis. Types of conservative and surgical treatment (antithrombolytic therapy). The concept of a floating thrombus. Diagnostics and treatment.
eight.	Myxoma of the heart.	2	Myxoma of the heart. Etiology and pathogenesis., Types and forms of clinical signs, diagnosis and treatment.
nine.	Pericarditis.	2	Pericarditis. Concept, etiology and pathogenesis. Treatment of various types of pericarditis. Compressive pericarditis. Hemodynamic changes.
ten.	P ak and gastritis molecules ti stomach .	2	The role and gastritis of the gastric stump. Clinical signs, diagnosis, differential. diagnostics, the role of endoscopic diagnostic methods and differential. diagnostics of surgical tactics.
eleven	Postcholecystectomy syndrome. Extrabiliary principles.	2	Postcholecystectomy syndrome (PCES). Extra-biliary principles, characteristics, etiopathogenesis. Modern instrumental diagnostic methods and their role in diagnostics and differential diagnostics. Treatment methods and methods.
1 2	Obstructive jaundice of malignant etiology.	2	Obstructive jaundice of malignant etiology. The reasons are clinical. Diagnostic measures. Differential diagnostics. New technologies in treatment.
13	Alveococcosis.	2	Alveococcosis. Etiopathogenesis, clinical picture, diagnosis and diff. diagnostics. Surgical treatment methods.

fourteen	Liver transplant.	2	Liver transplant. Orthotic and gitotopic transplantation. Indications. Surgical technique.
1 5	Modern types of treatment of nonspecific purulent lung diseases.	3	Modern types of treatment for non-specific purulent lung diseases. The concept of non-specific purulent lung diseases. Methods of therapeutic measures and the role of torocoscopy in this disease.
1 6	Chest damage.	3	Chest damage. Characteristic. Clinical manifestations of dependence on the damaged organ. Diagnosis of traumatic pleurisy, diagnosis of pleurisy depending on the type of pleurisy.
1 7	W abolevaniya mediastinal.	3	Chest damage. Purulent diseases of the mediastinal organs. Definition from mediasthenitis. Etiopathogenesis, clinical signs, diagnosis. Conservative treatment methods. Indication for surgical treatment. Types of surgical treatment. Treatment results.
1 8	Foreign bodies of the esophagus.	3	Foreign bodies of the esophagus. The reasons for the ingress of foreign bodies into the esophagus. Clinical signs and diagnosis. Role and possibility of X-ray and endoscopic methods in case of ingress of foreign bodies. Types of surgical and endoscopic treatment.
nineteen	Relaxation of the diaphragm.	3	Relaxation of the diaphragm. Etiology. Types of clinical signs. Diagnostics and diff. diagnostics, surgical tactics. Types of surgical treatment.
20	Nonparasitic lung cysts. Spontaneous pneumothorax.	3	Nonparasitic lung cysts. Spontaneous pneumothorax. Etiopathogenesis. Surgical treatment. Clinical signs, diagnostics. Types of complications. Treatment methods. Complicated and uncomplicated forms. Methods for the treatment of spontaneous pneumothorax.
	F and:	**4 6 h**	

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List of teaching films by subject "Hospital surgery"

No.	Educational film s
1.	Thrombectomy surgery.
2.	Contomirlard's artery operationlar
3.	Fallot tetradacida operation aldar
4.	Jurak aneurismasida operationlar
five.	Myocardium infarctini surgeon Yul Bilan davolash operation
6.	Bracheocephalus arterialarida (Takayasu syndromy) operationlar.
7.	Corinne aortasining aneurysmiside prosthetic operationlari.
eight.	Kizilungachda bajariladigan operationlar.
nine.	Zhigar resection.
ten.	Kukrak kafasida bajariladigan operationlar.
eleven.	Upkada bajariladigan operationlar.
12.	Kizilungachdan profuz kon ketganda tukhtatish usullari.
13.	Zhigar echinococcni diagnostics va davolash usullari.
fourteen.	Diabetic tovon sindromini zamonaviy davolash usullari.

List of Hospital Surgery Multimedia

No.	Media name
1.	Diseases pi ni Euodias. Burns of the esophagus .
2.	Cardiospasm
3.	Portal hypertension
4.	Parasitic liver disease
five.	Phlebeurysm
6.	Occlusive diseases of the aortic arch .
7	Aortic occlusive disease.
eight.	Venous thrombosis and thrombophlebitis.
nine.	Acute purulent destructive lung disease.
ten.	Pleural disease
eleven.	Renovascular hypertension
12.	Congenital heart disease
13.	Acquired heart defects
fourteen.	Diaphragm disease
fifteen.	Post-gastro-resection diseases
sixteen.	Postcholecystectomy syndrome . Obstructive jaundice
17.	Occlusive diseases of the arteries of the lower extremities.

18.	Acute thrombosis and embolism of limb arteries

Timing of practical lessons

(lesson duration - 4 hours)

No.	The main parts of the classes	Place of classes	Instructional method aids	Time distribution. (minutes) _	Observation and evaluation
1	Checking student attendance	Study room		five	
2	Introduction, homework and synopsis	Study room		ten	
3	Assessment of students' primary knowledge	Study room	Tables, slides, pictures, disks and a computer.	20	Current test control. Poll.
4	Patient supervision	In departments, wards	Tonometer, Fonen-doskop	35	
five	Discussion of supervised patients	In departments, wards	Disease history.	20	
6	Session in the operating unit and dressing room	X-ray, ultrasound, and endoscopy rooms.	Endoscopic devices.	thirty	
7	Application of new methods of pedagogical technologies and assessment of students' knowledge	Study room		40	100 point rating system.
eight	Lesson summary. Setting rating points. Homework.	Study room		20	

TOPIC : . ESOPHAGAL DISEASE. DIVERTICULE of the ESOPHAGUS. CHEMICAL BURNS OF THE ESOPHAGUS.

The purpose of the lecture: To acquaint students with pathological conditions associated with diseases of the esophagus, the reasons for their development, clinical course, their complication, differential diagnosis, and possible methods of treatment of these diseases.

The educational goals of the lecture is to expand the range of students' knowledge about the cause of the development of pathological conditions in diseases of the esophagus, preventive measures. Development of clinical thinking, preparation of students for practical training.

Lecture objectives:

1. Give an understanding of diseases of the esophagus.
2. Explain the causes and mechanisms of development of these pathological conditions.
3. Give the clinical characteristics of these diseases, possible variants of their course.
4. Give differential diagnosis with other diseases.
5. Communicate the available methods and methods of treatment.
6. For better assimilation of the material, give vivid examples, demonstration material, establish a live two-way communication with the audience.
7. All the material should be given in the aspect of high-quality preparation of a general practitioner.

PLAN OF THE LECTURE.

1. anatomical and physiological sketch. - 5 minutes
2. general symptomatology. - 5 minutes
3. Research methods of the esophagus. - 5 minutes
4. Burns of the esophagus. - 25 minutes
    - Etiopathogenesis
    - Pathological anatomy
    - Clinic
    - Course and complications
    - Diagnosis
    - Treatment
    - Prevention
5. Diverticula of the esophagus. - 20 minutes
    - Classification
    - Etiopathogenesis
    - Pathological anatomy
    - Clinic

- Treatment

6. Achalasia of the cardia. - 25 minutes
   - Disease frequency
   - Etiopathogenesis
   - Classification
   - Clinic
   - Treatment

7. List of literature required by students for a deeper acquaintance with the pathologies of the esophagus

Burns of the esophagus. Chemical burns of the esophagus. Damage to the esophagus caused nye reception acids (usually acetic acid) or alkalis more ammonia, caustic soda), are called toxic corrosive esophagitis. These substances are taken for the purpose of suicide or by mistake. Severe damage divisions nutritional ritelnogo tract is also at reception of a solution of potassium permanganate, perhydrol, acetone. The mechanism of their action on tissue is different from that of acids and alkalis. Thus, potassium permanganate and perhydrol act on tissues as oxidants. When chemical burns esophagus can simultaneously fuss Katyas burns of the mouth, larynx, lung edema, acute res Nye failure. This happens when taking acetic acid and ammonia due to their volatility and when exposed to vapors of concentrated acids and alkalis.

With a burn, four stages can be conditionally distinguished. Stage I - hyperemia and edema of the mucous membrane, stage II - necrosis and ulceration, stage III - formation of granulations, stage IV - scarring. When chemical burns of the esophagus ingested substance has, in addition to the local and general toxic effect on the organs of Lowland, which primarily affects the heart, liver, kidneys. Severe renal failure may develop.

Conventionally, there are four stages of clinical manifestations slaughtering Levan: I step - sharp (acute period corrosivity aiseau fagita); Stage II - the stage of chronic esophagitis (stage of "imaginary well-being"); III stage - stage stricture formation from 2-3 months to 2-3 years (organic food restriction water); Stage IV - the stage of late complications (obliteration of the lumen, perforation of the esophageal wall, cancer development).

According to the severity of injury in the acute stage are three degree or esophageal burns: easy (first), moderate (second) and severe (third). The first degree of a burn occurs as a result of ingestion of a small amount of a corrosive substance in low concentration or hot food. In this case, the surface layers of the epithelium are damaged in a larger or smaller part of the esophagus. The second degree of a burn is characterized by more extensive necrosis of the epithelium throughout the depth of the mucous membrane. Third degree burn - mucosal necrosis captures about span of, submucosal and muscular layers extend to paraesophageal tissue and neighboring organs. The defeat of esophageal acid or alkali may be accom panied by the defeat of the stomach, duodenum and jejunum entry with the appearance of necrotic areas and per perforation them, which leads to the development of acute peritonitis in

perio de, as well as scar deformity of the stomach afterwards. I In the acute phase (5-10 days) patients ispy TYVA severe suffering. Following the reception of a caustic alkali or acid appear severe pain in the mouth, pharynx, chest, epigastric, abundant slyunootdele set, frequent vomiting, dysphagia due to esophageal spasm in a burn and edema of the mucosa. Swallowing impossible possible. The patients are agitated, frightened. The skin is pale, moist. Breathing is quickened, there is tachycardia. Note again the personal degree of shock phenomena: agitation or retardation, fatigue, a bad reaction to the environment, drowsiness, akrozianoz, tachycardia, decreased blood pressure, Glu Chi heart sounds, decreased amount of urine until anu Rhee. A few hours after the burn along with symptoms of burn shock symptoms of toxemia: increasing pace perature body to 39 °, retardation replaced excitation sometimes arise delirium, muscle twitching. Respiration is frequent, shallow, pulse rate up to 120-130 per minute, blood pressure is reduced due to hypovolemia. Patients develop excruciating thirst as a result of dehydration, dyselectrolithemia. The study of blood leukocytosis point, lei shift kotsitarnoy to the left, increased erythrocyte sedimentation rate, increased hematocrit, hypo- and Dysproteinemia. In severe cases, the Supervisory etsya hyperkalemia, hyponatremia and chloropenia metabolite cal acidosis. With a concomitant burn of the larynx and vocal cords, there may be hoarseness, difficulty breathing, asphyxia. In tyazhe mated cases develops toxic damage of parenchymal organs (acute liver and renal failure). Poisoning with vinegar essence can cause the development of intravascular hemolysis. Patients develop jaundice, urine acquires the color of meat slops, bilirubinemia, hemoglobinuria are noted, anemia increases. As a result of aspiration, patients may develop tracheobronchitis, pneumonia. In direct vascular damage in the area of the burn occur early blood flow. Sometimes on the 5-7th day, psychosis develops (mental trauma, stress, pain, burn toxemia). Stage II - the stage of "imaginary well-being" - lasts from 7 to 30 days. By the end of the 1st week, rejection of necrotic tissues of the esophagus begins. Swallowing liquid food becomes somewhat freer, bleeding is possible. With deep necrosis occurs with the development of esophageal perforation Thieme symptoms mediastinitis, pericarditis, empyema, esophago-bronchial fistulae. Pains in the chest and back intensify, shortness of breath and tachycardia increase, body temperature becomes hectic, chills are replaced by torrential sweats, subcutaneous emphysema may form, and coughing occurs when eating liquid food. Aspiration that occurs in the acute period can manifest itself as symptoms of acute tracheobronchitis, pneumonia, and lung abscess. In severe cases, in the presence of extensive wound surfaces on the walls of the esophagus, sepsis may develop. The latency period is characterized by dysfunction of parenchymal organs, which is caused by dystrophy cal changes, protein starvation. By the end of the month (the stage of stricture formation), under the influence of treatment, the burned areas of the esophagus heal. In 10-15% of patients during radiological examination reveal no longitudinal folding of the mucous membrane, single or multiple portions of different narrowing of the esophagus against the field intensity. In 20% of patients by the end of the month phenomenon "interspersing yuscheysya" dysphagia increases. At esophagoscopy

celebrate different length sections, which wound prois goes slack. The wound surfaces are covered with dense scab, bleed easily. Scar formation is possible within the last few months. In the long term (up to 2--3 years) after a burn (stage of late complications), dysphagia is in the first place in the clinical course. From starvation, the patient's condition progressively worsens. In addition to strictures, the development of cancer, perforation of the esophageal wall and complications such as pneumonia, lung abscess, bronchiectasis, diverticula, esophageal-bronchial fistulas are possible.

In fresh cases of burns of the esophagus, which are rarely the object of X-ray examination, it is possible to reveal swelling of the folds of the mucous membrane and local spasms. In more distant terms, X-ray examination accurately establishes the length, degree of narrowing and deformation of the esophagus.

ESOPHAGOSCOPY - with annular (the most common form) narrowing reveals an annulus fibrosus, the color of which is paler than the surrounding mucous membrane. The narrowed prostate is usually centrally located and often surrounded by a star-shaped scar.

Emergency assistance at the scene. To relieve pain, patients are shown the introduction of drugs (promedol, morphine, etc.). To reduce salivation and relieve esophageal spasm, atropine, papaverine, ganglion blockers are administered. Cele according lavage oral assignment antihistamines (diphenhydramine, Suprastinum, Promethazine et al.). An important measure aimed at removing and neutralizing the poison is gastric lavage using a rubber probe. Depending on the nature of the substance taken, weak solutions of alkali or acid are used for gastric lavage. When the drink is expedient burn acids 2% - solution hydrocarbonyl is the sodium, magnesium oxide (magnesia), Almagelum at Poisoning alkalis - 1--1,5% acetic acid solution. In the first 6-7 hours, antidotes are administered.

Treatment: stationary. Patients undergo anti-shock and detoxification therapy - the introduction of a glucose-novocaine mixture, neuroleptics (droperidol), rheopolyglucin, hemodez, albumin, gelatinol, plasma, electrolyte solutions. To normalize the activity of the cardiovascular system, cardiac glycosides, corticosteroids are prescribed. Correction of metabolic acidosis is carried out by introducing 5% sodium bicarbonate solution. To combat hypovolemia about lead parenteral nutrition of patients within 3-4 days. For the prevention of secondary

infections and pneumonia, broad-spectrum antibiotics are prescribed. In order to prevent the development of rumen narrowing nutritionally and patients with 1st day given every 30-40 min 1--2 pharynx medicine comprising sunflower oil, benzocaine, novocaine solution of 5%, an antibiotic, a 3rd of the day they are fed oh cool the food. Early intake of medicine and food, as it were, realizes "soft" bougienage of the esophagus. The formation of scars is also prevented by the appointment of corticosteroids (cortisone, prednisolone, etc.), which delay the development of fibroblasts and reduce inflammatory changes in the esophagus. At present, it is considered expedient to carry out an early (from the 9-11th day) bougienage of the esophagus for 1-1 1/2 months in combination with subcutaneous

administration of lidase or ronidase for 2 weeks. Too early, before this time, started bougienage has the opposite effect, causing an exacerbation of the inflammatory process in the esophagus and increased scarring. Before you start you need to make bougienage ezofagosko Pius, which will establish the extent of the burn, the absence neottorgnuvshihsya necrotic tissue.

Esophageal diverticulum is a limited protrusion of the esophageal wall. Distinguish between pulsating and traction diverticula. Pulsionnye diverticulum formed due to protrusions pi schevodnoy wall under high intraesophageal prevent Lenia arising during its contraction. Development suit tional diverticula associated with inflammation in okra zhayuschih tissue and scar formation that pull the esophageal wall to the side of the affected organ (mediastinal lymphadenitis, chronic mediastinitis, pleurisy) Traction IU mechanism of observed at the beginning of the development of a diverticulum, then join pulsionnye factors, resulting the diverticulum becomes pulsation-traction. Diverticula subdivided depending on the location on pharyngoesophageal (Zenker) epibronhialnye (bifurcation, srednepischevodnye) epifrenalnyu (epidiafragmalnye) Distinguish true Deaver Ticul, the wall of which comprises all the layers of the esophageal wall and dozhna, in which there is no wall muscle layer. The vast present most diverticula are acquired, congenital Deaver Ticul are extremely rare. When dysmotility pi schevoda (esophagism) pseudodiverticulum observed that arise Suitable
only at the moment of contraction of the esophagus, when the esophagus relaxes, they disappear. Diverticula are rare before the age of 30 and often after 50; among the patients, men predominate. Most often, diverticula occur in the thoracic esophagus.

Clinical picture and diagnosis: a small pharyngeal-esophageal diverticulum is manifested by a feeling of perspiration, scratching in the throat, dry cough, sensation of a foreign body in the pharynx, increased salivation, and sometimes spastic dysphalia. As Uwe lichenie diverticulum filling it with food may be accompanied by a gurgling noise when swallowing, lead to the development of dysphagia of varying severity, to the appearance of protrusions on the neck with abduction head back. Bulging has a soft konsisten tion decreases with pressure, after receiving water at lane cussions above it is possible to determine splashing. The possibility of spontaneous naya regurgitation of undigested food from the lumen of the diverticulum at a certain position of the patient, difficulty in breathing due to compression of the trachea, the occurrence of hoarseness when compression of the recurrent laryngeal nerve during a meal in patients may develop a "phenomenon of blockade", which appears red face, the feeling of it short of breath, dizziness, faintness, which disappears after a long delay emesis When food Deaver Ticul appears putrid breath.

The clinical picture. Most pain GOVERNMENTAL disturbed nutrition, which leads them to exhaustion. Epibronhialnye diverticula often characterizes asymptomatic, possible effects of dysphagia, pain in the chest , Noah or in the back, in chronic diverticulitis - a breakthrough in the trachea, aspiration pneumonia development of lung abscess. Epifrenalnye diverticula as the majority of patients

are asymptomatic, but may present with pain behind the lower hour Tew sternum, aerophagia, nausea, vomiting, dyspnea reflex Coy, palpitations, bronchospasm, symptoms of compression of the food water and cardiospasm. Course of the disease is slow, with no existing member venous progression. Zenker's diverticulum can be complicated by the development of diverticulitis, which in turn can cause FLEG Mona's neck, mediastinitis, development of esophageal fistula, sepsis. Regurgitation and aspiration of contents diverticulum lead to chronic bronchitis, repeated pneumonias, pulmonary abscesses possible bleeding eroded mucosa di vertikula, development therein polyps, malignancy its wall. With a prolonged delay of food masses in the epibronchial and epiphrenal diverticula, complications may occur: diverticulitis, mediastinal abscess with a breakthrough into the bronchus, esophagus, pericardium and other mediastinal organs, massive bleeding. Chronic diverticulitis predisposes to cancer. Pharyngeal-esophageal diverticula can sometimes be found on examination and palpation of the neck. The basic method of diagnosing di vertikulov esophagus is a radiographic contrast uc adherence establishing presence diverticulum, cervical width, the delay time therein barium degree of impairment Pass Mosti esophagus, features of a polyp and cancer diverticulum, esophageal-forming bronchial and esophageal fistulas mediasti onal. Endoscopic examination to establish the presence of a diverticulum, detect mucosal ulceration it is about span of, the presence of bleeding, diagnose a polyp or cancer in di vertikule. The study must be very careful due to the possibility of perforation of the diverticulum. Treatment diverticula at small sizes, there are no complications, the absolute contraindications to surgery le cheniyu performed conservative therapy to the pro galaxies, delay food masses in diverticulum and to reduce the possibility of developing diverticulitis. Food must be fully valuable, mechanically, chemically and thermally sparing. Patients recommend eating good food After eating chopped follows blows to drink a few sips of water, taking a position that promotes emptying of the diverticulum. For large amounts of diverticula sometimes necessary washing strips minute diverticulum.

Treatment. Indications for surgical treatment of esophageal diverticula: complications (perforation, penetration, bleeding, stenosis, food water, cancer, development of fistulas), large diverticula complicated with at least a short-term delay in their food masses, Constant naya delay food diverticulum regardless of its size. Depending on the location of the diverticulum choose Operating ny access: when pharyngoesophageal - cervical, when epibronhialnyh - sided transthoracic, with a left-epifrenalnyh transthoracic. Diverticulectomy is used: a diverticulum is isolated from the surrounding tissues up to the neck, a myotomy is performed, it is excised and the hole in the esophagus wall is sutured. With a significant muscle atrophy or defect we antiplaque esophagus fibers produce plastic reconstruction of its wall diaphragm flap pleura. Intussusception is used only for small diverticula. Mortality after surgery is 1-1.5%.

Achalasia (cardiospasm) neuromuscular Zabolev of esophagus, passing violation manifested food mass in the stomach due to persistent violations reflex of covering of the cardia in swallowing, modify peristalsis and weakening Nia tone esophageal

wall. The incidence in relation to other diseases pi schevoda from 3 to 20%. The first symptoms of the disease often appear at the age of 20-40. More often women are ill.

Etiology and pathogenesis: etiological factors of achalasia of the cardia - congenital anomalies in the development of the nervous apparatus of the esophagus, degeneration of the intermuscular (Auerbach) plexus); constitutional neurasthenia with the onset of neurogenic discoordination of esophageal motility; reflex dysfunction of the esophagus; infectious toxic lesions of the nerve plexuses of the esophagus and cardia. Stress or prolonged emotional stress is a resolving factor. Pathogenesis: when examining the intraesophageal pressure in the region of the esophageal-gastric junction, a sphincter (physiological cardia) was found. In healthy people, he alone is in a state of tonic contraction after swallowing relaxed etsya. The main disorder that determines the symptoms of the disease is the lack of relaxation or insufficient relaxation of the cardia after swallowing. Various reactions of the cardia (incomplete opening when swallowing, incomplete opening and spasm, complete achalasia, achalasia and spasm, initial
hypertonicity, etc.) have one initial mechanism of violation of the innervation of the esophageal wall. Cases of achalasia occurring with cardiac hyper sfin ktera, can not be regarded as a true "cardiospasm", as the main mechanism for violating the patency of the cardia is not hypertonicity sphincter relaxation and the absence of his swallowing. Increasing the pressure in a physiological cardia wherein a secondary reaction and due to its constant yannoe pressure filling esophagus content scar-inflammatory changes in the tissues of the terminal part of the food water and loss of elasticity.

With achalasia of the cardia, the tone and peri esophageal steel. Instead of spreading to the stomach ne ristalticheskih cuts appear nepropulsivnye (has not been ensured ensures, passage) waves, they are joined by segmental contraction of the esophagus wall. Food long delayed food water and enters the stomach due to mechanical cardia opening under the influence of the hydrostatic pressure of the liquid column above it. Prolonged stagnation food masses, saliva and mucus in the food water leads to a significant expansion of its lumen, and the development of esophagitis periezofagita, which in turn aggravates disrupt Nia esophageal peristalsis. Pathological anatomy: in severe cases of the disease, an expansion of the esophagus up to 15-18 cm in diameter is noted , its elongation, as a result of which it can take an S-shape. Its capacity reaches 2-3 liters instead of 50-100 ml in healthy people. The distal part of the esophagus is sharply narrowed, it reveals dystrophy of ganglion cells and fibers of intramural nerve plexuses up to their death. In myshech prefecture layer observed dystrophy muscle fibers proliferation
connective tissue, especially in the wall of the narrowed segment
fib endoneurium roses, vasodilatation, the appearance around infil tration of lymphoid and plasma cells. In all layers of the esophageal wall and surrounding tissues exhibit Prizna ki inflammation. The mucous membrane of the esophagus is hyperemic, edematous, ulcerated in places. Over expressed from Menenius near the narrowed portion of the esophagus.

Clinic and diagnostics: for achalasia characterized thorns triad of symptoms: dysphagia, regurgitation, pain. Dysphagia - basic and in most cases the first symptom zabole Bani. In some patients, it occurs suddenly, as if in full health, in others it develops gradually. Strengthening of dysphagia in most patients say after nervous excitation Denia during hasty
food, when eating dense, dry and poorly chewed food. Sometimes there is a paradox disfa Gia: dense food passes into the stomach rather than the liquid and semi-liquid.
In a number of patients with achalasia, dysphagia depends on the temperature of the food: warm food passes poorly or does not pass, and cold food passes, or vice versa. Patients gradually adapted to facilitate the passage of food into the stomach through a series of stages (walking, gymnastic exercises, air ingestion and mica us, receiving a large amount of warm water, etc.). Severe cachexia with cardia achalasia is rarely observed.
Regurgitation with a small expansion of the esophagus occurs after a few mouthfuls of food, at a significantly extended pi schevode is more rare, but abundant and caused severe spastic contractions of the esophagus that occur when nepepolnenii. Regurgitation in the supine position and strong to clone the body caused by mechanical pressure of the contents of the esophagus in the pharyngeal-esophageal sphincter and stretching. Nocturnal regurgitation is associated with a slight decrease in the tone of the glo-oesophageal sphincter. Pain behind the sternum with achalasia of the cardia are of a varied nature. They can be associated with spasm of the esophageal muscles and are eliminated by taking nitroglycerin, amyl nitrite and atropine. However, the majority of patients pain occur overflow of the esophagus and disappear after regurgitation or passers Denia food into the stomach. Some patients have spontaneous pain attacks in the chest on the type of pain crises Such pain is more often observed in the initial period of the disease, sometimes before the onset of dysphagia and regurgitation, which is not always SNI toil atropine or nitroglycerin, which allows assumption live their relationship with progressive dystrophic process in intramural nerve plexus of the esophagus. Pain on an empty stomach or after vomiting is more often caused by esophagitis and is relieved
eating. Air belching, nausea, increased salivation, burning along the esophagus, halitosis also OCU by previously esophagitis. In patients with both acute and gradual beginning zabole Bani symptoms progress over time: enhanced dysphagia, regurgitation often occurs. Many patients are ashamed of their lack, become withdrawn, painfully resentful. Nai more frequent complication disease is congestive esophagitis, which occurs when a long delay in mass of food in the esophagus. In mild cases, it appears hyperemia and edema of the mucosa, in more severe - the presence of coarse and HEPA -dimensional folds, erosions, ulcers, which usually are not arranged as above narrowed portion. In the future, bleeding, perforation of the esophagus, peri-esophagitis may develop. Chronic esophagitis can cause cancer of the esophagus and cardia.
Frequent complications of achalasia are repeated aspiration bronchopneumonia, lung abscesses, pneumosclerosis. Oso cially often these complications occur in

children. Described oslozh neniya caused by compression of the advanced recurrent nerve esophagus, the right main bronchus, superior vena cava, the vagus nerve, etc. B. In Petrov sky allocates four hundred di- disease: I phase-changeable functional cardia spasm, expand pi schevoda not observed;

Stage II - stable spasm cardia with soft extension esophagus, III stage - scarring s cardia muscle ate with a marked expansion of the esophagus, IV stage sharply expressed adjoint stenosis dilatation with esophageal achalasia often S-shaped, and esophagitis. The main methods of diagnosing cardia achalasia are X-ray examination, esophagoscopy, esophagotonokymography, pharmacological tests ..

When beskontrastnom uc radiological follow chest roll stands ki Patients with achalasia identify additional bulging right contour mediastinum, the presence of the liquid level in the posterior mediastinum projection The absence of a gas bubble stomach. The main radiological signs of achalasia - the narrowing of the terminal esophagus with clear, smooth and elastic loops ("the flame will move one candle", "mouse tail") folds mucosa is about span of constriction in the throat to maintain the first barium can flow freely into the stomach, then the contrast mass lingers in the esophagus for a long time. Above the barium suspension, a layer of liquid and food residues are determined. The expansion of the esophagus over the site of its narrowing is expressed in varying degrees. In a number of patients, lengthening and curvature of the esophagus are noted.

The endoscopic picture depends on the duration of the disease. At the beginning of bo existing illness esophagus widened slightly, as the disease progresses the lumen increasingly expanding and some pain GOVERNMENTAL become crimped. The mucosa has features Sun Singe: folds thickened, arteries and veins dilated, sometimes visible portions flushing, erosion, leukoplakia, ulceration. Typically, the end esophagoscope possible to carry out through the narrowed portion, which confirms the functional advantageously ha rakter changes in the esophagus. The mucous membrane at the site of narrowing is most often not changed. Ezofagotonokimograficheskoe study - the main method for early diagnosis of esophageal achalasia, as violations of juice ratitelnoy ability of the esophagus and cardia physiological occur much earlier clinical symptoms Zabolev Niya. The study was conducted with a special multichannel Nogo probe with rubber cartridges or "open" catheters ramie, registering esophageal contractions and changes in the intra-esophageal pressure. Normally, after swallowing the esophagus races uted peristaltic wave, at this point the cardia from opened or pressure is reduced. After passing peristal cal wave cardia is closed again. With achalasia of the cardia, there is no reflex relaxation of the cardiac sphincter when swallowing, and the intraluminal pressure remains at the same figures. Another characteristic feature is the violation of the ne ristaltiki esophagus: various shapes and swallowing vneglotatelnye spastic contraction, a large number of local - secondary contractions of the esophagus, which indicates esophagitis. In all patients, along with spastic contractions We mention chayut large amount of propulsive peristaltic contractions of the esophagus.

QUESTIONS

To the audience to establish feedback and clarify the achievement of the lecture goal

1. List 3 anatomical parts of the esophagus
2. What are the 3 narrowing of the esophagus is normal
3. Why do varicose veins raspologue in n / 3 esophagus
4. Tactics of GPs for burns of the esophagus
5. What types of diverticula do you know
6. What is achalasia of cardia
7. Complications of bougienage of the esophagus
8. Types of operations for the reconstruction of the esophagus after its burns
9. Complications of diverticula
10. What are the most common and common symptoms in diseases of the esophagus?

T THEME: CARDIOSPASM. ETIOLOGY, CLINIC, DIAGNOSTICS AND TREATMENT

The value of studying the topic. Despite the rapid development of clinical gastroenterology over the past decades, he touched little on clinical esophagology. Many issues of physiology and pathology, morphology and function, diagnosis, prevention and treatment of diseases of the esophagus are not well known to a wide range of doctors. The pathology of the esophagus, which causes dysphagia syndrome, includes a number of diseases: cardiospasm, cicatricial stenosis of the esophagus, neoplasms, phlebectoid veins of the esophagus, hiatus hernia, divertculosis, polyposis, foreign bodies, trauma to the esophagus, etc. Untimely diagnosis and treatment lead to severe general and local complications that threaten the patient's life.

The purpose of the lesson:

1. To study the main clinical, radiological and endoscopic
features of diseases of the esophagus with dysphagia syndrome.
2. Conduct differential diagnostics most often
occurring diseases of the esophagus:
and. Cardiospasm.
b. Esophageal carcinoma.
in. Benign neoplasms.
city of Diverticulosis.
e. Foreign bodies of the esophagus.
e. Hernia of the esophageal opening of the esophagus.
g. Lymphogranulomatosis. Mediastinal form.
h. Tumors and cysts of the mediastinum.
and. Ulcerative esophagitis.
3. To familiarize with possible local and general complications.
4. To acquaint practically with the methods of modern diagnostics of diseases
levanisation of the esophagus:
and. X-ray method

b. Endoscopic method.

in. Cytological method.

e. Sample with methylene blue solution.

5. To study the basic principles of complex conservative treatment and types of surgical interventions for various diseases of the esophagus.

and. Know the indications for conservative and surgical methods of treatment.

b. Know the basics of preoperative and postoperative treatment.

in. Know the main stages of surgical procedures on the esophagus.

Test questions:

1. Topographic characteristics of the esophagus
2. Basic theories of the etiopathogenesis of esophageal diseases
3. Patnanatomy of diseases of the esophagus
4. Development of methods for examining patients
5. Dif. diagnostics
   - Cardiospasm
   - Cicatricial strictures of the esophagus
   - Esophageal cancer
   - Diverticulosis
6. Treatment tactics for patients with esophageal pathology
7. Conservative therapy
8. Pre- and postoperative treatment of patients
9. Types and stages of typical surgical interventions.

Practical skills

1. Reading radiographs of the esophagus.
2. Nasogastric intubation
3. Have an idea of the course and stages of surgery for diseases of the esophagus.
4. Carrying out diagnostic and therapeutic procedures for dysphagia.

THEORETICAL PART

Dysphagia - difficulty swallowing - a symptom of diseases of the esophagus, adjacent organs or neurogenic disorders of the act of swallowing. Sometimes the swallowing disorder reaches the degree of aphagia, i.e. complete impossibility of swallowing. All causes of dysphagia can be combined into four groups: ***traumatic and inflammatory diseases of the pharynx*** (acute tonsillitis, paratonsillar abscess, allergic edema of the tissues of the pharynx, fracture of the hyoid bone), ***damage to the nervous system and muscles involved in the act of swallowing*** (bulbar paralysis, rabies, botulism, tetany , hypoglossal nerve neuritis, dermatomyositis, neuroses), ***compression of the esophagus by adjacent abnormal or pathological formations*** (tumors, sclerosing mediastinitis, abnormal location of the right subclavian artery, double aortic arch, aortic aneurysm, etc.), ***various diseases and lesions of the esophagus*** (trauma, burns, tumor, inflammatory and degenerative processes).

DIFFERENTIAL DIAGNOSTICS OF DYSPHAGIA

The main reasons for the development of dysphagia are:

- a foreign body of the esophagus;
- trauma to the esophagus;
- esophageal diverticulum;
- achalasia of the cardia;
- chemical burn of the esophagus;

- cicatricial narrowing of the esophagus;
- swelling of the esophagus;
- reflux esophagitis;
- peptic ulcer of the esophagus;
- diaphragmatic hernia.

Normal transport of the food bolus depends on:
- the size of the swallowed lump,
- the diameter of the lumen of the swallowing canal,
- peristaltic contraction of the esophagus,
- the function of the swallowing center, which includes the normal relaxation of the upper and lower sphincters of the esophagus during swallowing and suppression of persistent contractions in the body of the esophagus.

Depending on the violation of certain components of the normal transport of the food bolus, i.e. mechanism of formation, allocate mechanical (organic) dysphagia and motor (functional).

Dysphagia caused by too large a lump of food, a foreign body or organic narrowing of the lumen of the esophagus of various etiologies is called ***mechanical.***

Dysphagia, caused by uncoordinated, weak peristaltic contractions of the walls of the esophagus, inhibition of the swallowing center with impaired functioning of the esophageal sphincters, is called ***motor dysphagia.***

Complaints arising from dysphagia will vary depending on the mechanism of its formation. So, the difficulties that arise when eating only solid food indicate the presence of mechanical dysphagia, in which the lumen is not narrowed so much and fluid intake can improve the passage of food through the narrowed area. With a pronounced decrease in the lumen, dysphagia develops with the use of both solid and liquid food (Table 4.1.).

In contrast, motor dysphagia due to achalasia and diffuse esophageal spasm is equally affected by the consumption of solid and liquid food from the very beginning of the disease. Patients suffering from scleroderma are susceptible to the development of dysphagia when eating solid food that is not related to the position of the body, while when eating liquid food, dysphagia is observed in them in the supine position, but is absent when the body is upright.

Information about the duration and nature of the course of dysphagia can help in making a diagnosis. Short-term transient dysphagia may be caused by any inflammatory processes. Progressive dysphagia over several weeks to several months is characteristic **of esophageal cancer.** Occasional dysphagia when eating solid food, noted for several years, indicates a benign disease and is characteristic of the lesion of the lower esophageal ring.

Concomitant symptoms are of great diagnostic value. Regurgitation in the nose and tracheobronchial aspiration during swallowing are signs of paralysis of the pharyngeal muscles or the presence of a **tracheoesophageal fistula.** Tracheobronchial aspiration not associated with swallowing may be secondary in the presence of **achalasia, Zenker's diverticulum,** or **gastroesophageal reflux.** A pronounced decrease in body weight, not proportional to the severity of dysphagia, is characteristic **of esophageal cancer.** If dysphagia is preceded by hoarseness, the primary lesion is usually located in the larynx. Hoarseness that occurs after the development of dysphagia may indicate involvement of the laryngeal recurrent nerve in the process through the spread of esophageal cancer beyond the walls of the esophagus. A combination of symptoms of damage to the larynx with dysphagia is also observed in various neuromuscular disorders. Hiccups suggest a lesion in the distal esophagus. Unilateral wheezing, combined with dysphagia, indicates a process in the mediastinum, affecting the esophagus and large bronchus. Pain in the chest area, combined with dysphagia, develops with **diffuse spasm of the esophagus** and associated movement disorders. Pain in the chest area, similar to the pain experienced with diffuse spasms of the esophagus, can also occur with **acute aphagia** due to too large a food lump. A history of prolonged heartburn and reflux preceding

dysphagia indicates **peptic esophageal stricture.** Similarly, a history of corrosive swallowing, radiation therapy, or concomitant mucocutaneous diseases may indicate **mechanical esophageal stricture.**

The final diagnosis is based on endoscopic and radiopaque studies.

The sudden, acute development of dysphagia is typical, as a rule, for foreign bodies of the esophagus or its damage.

Suddenly onset dysphagia, accompanied by pain during swallowing or constant pain, drooling, sometimes coughing, attacks of suffocation, without evidence of any traumatic effects, indicate the presence of a **foreign body** in the lumen of the esophagus. For the purpose of diagnostics, a multi-axis X-ray examination is shown, which, in the case of X-ray contrast foreign bodies, makes it possible to diagnose and determine their location. Foreign bodies can be localized at any level, however, they are more often located in the upper third of the thoracic esophagus. In all cases, esophagoscopy is necessary. The latter is used not only to clarify the diagnosis, but also to remove a foreign body. Caution must be exercised during esophagoscopy as rough manipulation can damage the esophageal wall.

Acute dysphagia, accompanied by pain, the appearance of subcutaneous emphysema in the neck, is possible with **damage to the esophagus.** With damage to the intrathoracic esophagus, signs of periesophagitis and mediastinitis are characteristic. Damage to the esophagus is diagnosed using an X-ray contrast study using water-soluble contrasts - a contrast spot outside the esophagus is a direct indication of a violation of the integrity of its wall. In a conventional X-ray examination, the expansion of the mediastinal shadow is determined (the presence of air in the mediastinum, the phenomenon of mediastinitis). During esophagoscopy, a defect in the esophageal wall with edges imbibed by blood (in the later stages - pus) is determined.

Diseases accompanied by the gradual appearance of dysphagia and its progression:

- esophageal diverticulum;
- achalasia of the cardia;
- cicatricial narrowing of the esophagus;
- swelling of the esophagus;
- reflux esophagitis;
- peptic ulcer of the esophagus;
- diaphragmatic hernia.

For the differential diagnosis of the listed diseases, more information than clinical data is provided by X-ray examination with contrast and endoscopic examination. On the basis of radiography, it is possible to diagnose and differential diagnose diseases such as diverticula of the esophagus, cicatricial narrowing of the esophagus, tumors of the esophagus, diaphragmatic hernia, and achalasia of the cardia. Fiber-optic endoscopy allows diagnosing the following diseases: reflux esophagitis, peptic ulcer of the esophagus, tumors of the esophagus, diverticula of the esophagus, achalasia of the cardia, hernia of the esophageal opening of the diaphragm.

With **diverticula of the esophagus**, a filling defect with clear contours extending beyond the walls of the esophagus is determined during radiography. The clinical picture depends mainly on the size of the diverticulum. In the initial stages of the disease, there may be only vague discomfort - unpleasant sensations when swallowing, a feeling of "scratching" or "stuck" food when swallowing. As the bag of the diverticulum grows, typical symptoms appear - dysphagia associated with the ingress of food masses into the bag of the diverticulum and compression of the esophagus. If the diverticulum is large, there may be pain behind the sternum, swelling of the veins of the neck, and the appearance of other signs that are characteristic of masses of the mediastinum. Regurgitation of food masses from the diverticulum sac into the oral cavity is often observed, which is especially pronounced when the patient is lying down. It is important to find out if there are phenomena of diverticulitis, esophagitis, and also to exclude the presence of a polyp or cancer in the area of the diverticulum. Radiographically, the diagnosis of diverticulitis is established on the basis of studying the relief of the mucous membrane (blurred contours) and the degree of retention of the

contrast agent in the diverticulum bag, the delay of barium suspension up to 2 minutes. is an indirect sign of diverticulitis. Endoscopic examination is advisable only after radiography if a tumor or fistula in the diverticulum is suspected.

Cicatricial stricture of the esophagus is radiographically defined as a narrowing of a funnel-shaped shape, without any depressions and niches. A distinctive feature of a malignant tumor is the presence of smooth contours. Strictures in length are short (up to 3 cm) and long (more than 3 cm), often capturing the esophagus throughout (total lesion). The degree of narrowing is determined by the diameter of the barium stream, as well as by the speed of passage of the contrast and the duration of its delay over the site of narrowing. Above the place of the stricture, depending on the duration of its existence, suprastenotic expansion develops of varying degrees of severity. The clinical picture depends on the degree of cicatricial stricture and the main symptom is dysphagia, which can reach the degree of complete obstruction of the esophagus. At the same time, regurgitation, increased salivation, esophageal vomiting, and esophagitis develop. In cases where differential diagnosis with tumors is necessary, endoscopic examination with biopsy is indicated.

X-ray can diagnose both benign and malignant **tumors of the esophagus .** The characteristic radiological signs of ***benign tumors*** are: a well-defined defect of filling (usually on one of the walls of the esophagus), oval, preserved relief of the mucous membrane and preserved elasticity of the walls in the area of the filling defect. Typical for a benign tumor (especially for intramural), the X-ray symptom is the "peak" symptom - a clear angle between the esophageal wall and the edge of the tumor, detected in an oblique position. Although the folds of the esophageal mucosa over the tumor look flattened, unlike cancer, they never break off in the area of the filling defect, but go around it. The esophagus in the area of the tumor may be moderately dilated, but, as a rule, there is no retention of the barium suspension (the elasticity of the esophageal walls remains). To clarify the diagnosis of a benign tumor, an endoscopic examination is used.

The polyp of the esophagus, which belongs to intraluminal tumors, during X-ray examination is determined as a filling defect with clear, more often rounded contours, located centrally. Sometimes the filling defect has a cellular structure, the contrast mass seems to flow around the intraluminal formation. With polyps on a thin stem, the filling defect changes its position during the examination. The x-ray picture of a polyp on a broad basis is very similar to the picture of exophytic esophageal cancer.

The main X-ray symptom in a ***malignant tumor of the esophagus*** is the loss of elasticity of the esophageal wall in the area of the neoplasm. Early radiological symptoms in esophageal cancer are uneven contours, a small marginal filling defect, and deformation of longitudinal folds. Mandatory polypositional radiography, because narrowing of the esophagus can be detected only in one of the oblique positions. There is no enlargement of the esophagus above the tumor in the early stages. In the later stages of the disease, a significant narrowing of the lumen of the esophagus is seen, the lumen is contrasted in the form of a convoluted and rigid canal with corroded contours. Above the tumor, a suprastenotic expansion of the esophagus develops, the degree of which depends on the duration of the stenosis. Esophagoscopy is necessary not only for the purpose of endoscopic diagnosis, but also for biopsy for the purpose of morphological verification of the diagnosis.

X-ray changes in different stages of **cardia achalasia are** different (Fig. 4.1 a, b, c, d, respectively, I , II, III , IV century). There are 4 stages of the disease: Stage I (early, or initial) is characterized by an unstable spasm of the cardia of a functional nature, there is no narrowing of the cardia and no expansion of the esophagus; Stage II - stable spasm of the cardia with a mild expansion of the esophagus; Stage III - cicatricial changes in the muscle layers of the cardia with pronounced expansion of the esophagus; Stage IV - pronounced cicatricial stenosis of the cardia with dilatation of the esophagus, often having an S- shaped form, esophagitis, often accompanied by peri-esophagitis and fibrous mediastinitis.

In patients with ***stage*** I of the disease, the diameter of the esophagus and cardia remains normal, but violations of the passage of the food lump in the direction of the cardia, which does not open immediately after the barium suspension enters the esophagus, is already clearly visible. The relief of the esophageal mucosa is not changed.

In the II ***stage of the*** disease, a more pronounced dysfunction of the cardia is noted. The intervals between the passage of the contrast suspension through this part of the esophagus are longer, and the periods of the opening of the cardia are very short. There is a slight uniform expansion of the esophagus and a significant violation of its evacuation function. The relief of the mucous membrane is not changed. The contrast agent can remain in the esophagus for several hours.

In ***stage*** III, ***the*** X - ray picture indicates the appearance of organic changes in the esophagus and cardia. A significant expansion of the esophagus is determined. In addition to spastic changes, the cardiac canal has a narrowing of an organic nature, which is expressed in the form of a cone-shaped or coracoid sharpening (a symptom of a "mouse tail"). The peristalsis of the esophagus is disturbed and irregular. Significant violations of the evacuation function of the esophagus: the contrast suspension through the cardia enters the stomach at rare moments when its patency appears and in small portions, therefore it lingers in the esophagus for a long time.

In ***stage*** IV, as a result of a decrease in the tone of the esophageal wall, the expansion becomes more significant throughout its entire length. The esophagus is elongated and S - shaped. The evacuation of contrast suspension from it is slowed down. The relief of the mucous membrane changes sharply, the thickening of the folds is clearly defined. A characteristic x-ray sign of the III- IV stage of cardia achalasia is the absence of a gas bubble of the stomach.

FOREIGN BODIES OF THE ESOPHAGUS. The ingress of foreign bodies into the esophagus is quite common and can lead to dangerous consequences.

Most often, fish, meat and bird bones (70-90%), dentures, coins, buttons, pins, and nails get stuck in the esophagus. The reasons for the ingress of foreign bodies into the esophagus are different. Children literally take everything by mouth, older people are often inattentive or have bad teeth, which contributes to the penetration of foreign bodies. A common reason for the ingress of foreign bodies is carelessness while eating, wearing ill-fitting dentures, eating while intoxicated. The mania to swallow all sorts of objects is known in many mental patients. The retention of foreign bodies most often occurs at the level of the jugular notch of the sternum (about 50% of all foreign bodies), at the level of the tracheal bifurcation (18%) and in the area of cricopharyngeal narrowing (11%).

If the foreign body has sharp edges, early perforation of the esophagus is possible, and if the foreign body is large, late due to wall necrosis.

Without treatment, foreign bodies of the esophagus quickly lead to the development of periesophagitis, mediastinitis. Typical symptoms are pain, dysphagia, regurgitation, salivation.

One of the most severe complications is bleeding from large-caliber vessels (more often the aorta, carotid artery). Cases are described when massive foreign bodies caused closure of the entrance to the larynx and rapid death due to asphyxiation. With this in mind, treatment in such situations should begin with the imposition of a tracheostomy.

Diagnostics and treatment. The examination of the patient begins with an instrumental examination of the pharynx and pharynx using a laryngeal mirror. After this, an x-ray of the esophagus can be performed. However, the most informative diagnostic method is fibroesophagoscopy, which in most cases allows you to immediately remove a foreign body. If esophagoscopy fails to remove a foreign body, surgical methods are used. When a foreign body is localized in the cervical region (25 cm from the incisors), cervical esophagotomy is used. Foreign bodies of the mid-thoracic region are removed using thoracotomy or dorsal esophagotomy according to Nasilov. However, the need to perform such operations is rare.

DAMAGE OF THE ESOPHAGUS. The most common cause of damage to the esophagus is diagnostic and treatment manipulations (FEGDS, blockage of the esophagus), foreign bodies.

Penetrating wounds of the thoracic esophagus are less common than the cervical. As a special form of rupture of the esophagus, the so-called spontaneous rupture of a previously healthy esophagus is described.

Typical symptoms of damage to the cervical esophagus are pain, aggravated by swallowing, dysphagia, fever, the presence of subcutaneous emphysema in the neck. Subsequently, there is swelling and stiffness of the neck. With penetrating wounds from the outside, saliva or food particles appear in the wound.

With fluoroscopy, a layer of gas in the soft tissues of the neck and the presence of a horizontal liquid level can be observed, and when contrast is given, it flows into the soft tissues of the neck.

Injury to the intrathoracic esophagus is particularly severe because trauma and infection of the mediastinum, as well as the pleural cavity with saliva, food leads to shock, cardiopulmonary failure, dehydration, progression of intoxication and sepsis. Typical signs of damage to the intrathoracic esophagus are chest pain, fever, tachycardia, shortness of breath, subcutaneous emphysema. Patients occupy a forced sitting position. There are three phases of the clinical course of perforation of the thoracic esophagus.

The initial phase **-** the shock phase lasts up to 4-5 hours.

The second phase - the phase of false calm, lasts up to 18-36 hours. In this phase, the pain subsides, but the body temperature remains high, and dehydration increases.

The third phase - the phase of mediastinitis and other inflammatory complications - develops a septic state.

Diagnosis is based on clinical findings and X-ray findings. A tetrad of radiological signs of perforation of the intrathoracic esophagus is proposed: mediastinal emphysema; subcutaneous emphysema; hydropneumothorax; the exit of the contrast agent beyond the esophagus. However, the most informative diagnostic method is FEGDS.

Treatment of injuries of the cervical esophagus is operative. The wound of the esophagus is sutured with a two-row suture, drainage tubes are also brought here.

In advanced cases, with a formed peri-esophageal abscess, the abscess is opened and drained. Isolation in these cases of the esophagus and the search for the site of perforation is impractical, because the destruction of adhesions can lead to the spread of the inflammatory process into the mediastinum. The patient is fed through a thin tube. From 7 days, liquid is allowed by mouth.

The choice of a method for treating injuries of the thoracic esophagus depends on the time elapsed since the moment of perforation, the size of the perforated opening, patency of the esophagus, and the general condition of the patient.

Conservative treatment is possible in the early stages from the moment of perforation with the general satisfactory condition of the patient, a small perforated hole, good patency of the esophagus. It consists in carrying out tube feeding, antibiotic therapy, infusion therapy.

The main method of surgical treatment of penetrating damage to the esophagus is surgery - suturing of the perforated opening, aimed at restoring the integrity of the esophagus and eliminating the source of infection.

The operations used to treat injuries of the intrathoracic esophagus are divided into three groups:

1. Operations to turn off the esophagus: gastrostomy, jejunostomy, transection of the esophagus in the cervical region, transection of the stomach in the cardiac region.
2. Drainage operations: cervical mediastinotomy, posterior extrapleural mediastinotomy, transphrenic mediastinotomy, transepleural mediastinotomy, their combination, pleural drainage.
3. Operations directly on the esophagus: suturing of the perforated hole, resection of the esophagus.

PRACTICAL PART

Thematic tests

1

1. With a chemical burn of the esophagus in the acute stage, it is shown:
a) rinsing the mouth, esophagus, stomach with water
b) prescribing morphine preparations and sedatives
c) drinking milk
d) everything is correct
e) everything is not correct

2. The main methods of treating cardiospasm are:
a) cardioplasty according to Geller
b) fundoplication according to Nissen
c) cardiodillation
d) various methods of extramucous plastic
e) bougienage

3. The main method for diagnosing esophageal diverticulum is:
a) esophagoscopy
b) contrast X-ray examination
c) ultrasound
d) radionuclide examination

4. In cancer of the middle thoracic esophagus, the following is most often observed:
a) regurgitation
b) pain behind the sternum
c) cough, shortness of breath
d) dysphagia
e) salivation

5. Zenker's diverticulum of the esophagus is localized:
a) in the area of the tracheal bifurcation
b) above the diaphragm
c) in the esophagus in / 3
d) in the pharyngeal-esophageal junction
e) above the cardia

6. For the diagnosis of neoplasms of the mediastinum can be used:
a) pneumothorax
b) pneumoperitoneum
c) retropneumoperitoneum
d) bronchography
e) pneumomediastinography

7. With a diverticulum of the cervical esophagus, it is shown:
a) intussusception of the diverticulum
b) tube feeding
c) removal of the diverticulum

d) endoscopic dissection at the site of narrowing, below the diverticulum
e) all of the above
8. The earliest manifestation of esophageal cancer is:
a) dysphagia
b) pain behind the breastbone and in the back
c) regurgitation of stagnant contents
d) increased salivation
e) weight loss
9. In the diagnosis of foreign bodies of the esophagus, they do not use:
a) collection of complaints and anamnesis
b) probing of the esophagus
c) esophagography
d) esophagoscopy
e) laryngoscopy

Situational tasks

Problem number 1
Patient D., was admitted to the hospital with poisoning of one of the strong inorganic acids. In the places of contact of acid with the esophageal mucosa, crusts with a yellowish color were found.
1. What acid caused the poisoning?
Answer: 1. The color of the crusts is characteristic of inorganic acid (nitric) and
is due to the formation of xanthoproteins. In case of burns with sulfuric acid, the color is greyish, and with hydrochloric acid, it is whitish.

Problem No. 2
A young woman came to the doctor's office with complaints of impaired
passage of food, regurgitation of unchanged food. Thick food goes better than liquid food. The disease manifested itself after a difficult family drama.
When esophagography is determined by the narrowing of the cardiac part of the esophagus in the form of a "pen" and suprastenotic significant expansion. The
evacuation of barium from the esophagus is significantly slowed down.
1. Diagnosis? 2. What research methods need to be carried out? 3. Tactics?
Answer: 1. Cardiospasm of the 2nd degree. 2. Fibroesophagoscopy with targeted
biopsy of the cardiac mucosa. 3. Cardiodilation

Problem number 3.
Patient V., 36 years old, was admitted to the hospital with complaints of progressive dysphagia. Has been sick for three years. For the passage of food, he washed it down with liquid, swallowed air. I lost weight, vomiting with little changed
food. On X-ray examination, the narrowing of the esophagus in the cardiac region and its suprastenotic expansion.
1. Diagnosis? 2. What diagnostic measures need to be performed? 3. Treatment tactics?
Answer: 1. Cadiospasm of the 3rd degree. 2. Fibroesophagoscopy with targeted
biopsy of the cardiac mucosa. 3. Surgical treatment.

Problem number 4.
Patient Sh., 36 years old, was admitted to the hospital with complaints of difficulty in swallowing while intoxicated. A year and a half ago, I drank some liquid by mistake. Within 10 days he was treated in the
surgical department. In a specialized department,

bougienage of the esophagus was performed . After about a month, signs of dysphagia appeared, which gradually increased. After the next course of bougienage, the symptoms of dysphagia decreased. However, then they began to grow again. Repeated bougienage courses were not effective. Due to dehydration and exhaustion, a gastrostomy tube was placed. Can only swallow water.
1. Diagnosis? 2. What diagnostic measures should be taken
? 3. Possible methods of surgical treatment?
Answer: 1. Cicatricial narrowing of the esophagus. 2. Esophagography, fibroesophagoscopy
. X-ray of the stomach. 3. Esophago-esophagoanastomosis, esophagofundoanastomosis
, plastic surgery of the esophagus in the stomach, small or large
intestine.

TOPIC: PORTAL HYPERTENSION. ETIOLOGY, PATHOGENESIS, CLINIC, CLASSIFICATION, DIAGNOSTICS AND TREATMENT

The purpose of the lesson:

1. To study the etiology, pathogenesis, clinic of liver cirrhosis and its complications - ascites.
2. Learn the correct diagnosis according to the modern classification.
3. Be able to make a differential diagnosis.
4. Know the basic principles of treatment.
5. Study the concept, etiology, diagnostics, carry out differential. diagnostics of the tactics of treating uncontrolled protrusions in the abdominal wall, groin and femoral region.
6. Establish the basic principles of prevention, labor expertise.

Test questions:

1. Etiology and patogenesis of liver cirrhosis.
2. Clinical picture, differential diagnosis of liver cirrhosis.
3. Treatment of liver cirrhosis.
4. Etiology and patogenesis of ascites.
5. Clinic of ascites.
6. Differential diagnosis and treatment of ascites.

THEORETICAL PART

ASCITES SYNDROME

Ascites means the presence of free fluid in the abdomen. On the title comes from the Greek Askites (which translates as "pocket", "scrip").

Pathological physiology. Normally abdominal IME is a slight amount of peritoneal fluid volume which preserves nyaetsya unchanged as a result of a complex mechanism, support vayuschego equal to the level of production and absorption.

According to the hypothesis The Starling , in the phenomenon of physiological produ tsirovaniya peritoneal fluid, as well as in its absorption are important

capillary permeability and osmotic pressure of blood. The drop of plasma albumin below 2.59% causes the tendency to trans sudatsii in all serosal cavities. In theory Starling , in the capillary loop, depending on the relation between hydrostatic pressure (filtration) and colloid osmotic pressure (suction) We establish INDICATES and the ratio between the length of the respective zones TRANSCEM dations (near edge metarteriolyarnogo) and absorption (near venular region). Sphincters forming these metarteriolyarnye ring regularity liruyut hemodynamic pressure in capillary loops, playing the role of protective barriers that for each increase in arterial pressure GOVERNMENTAL boxes prevent the occurrence repercussions in the capillaries. Venular capillaries on the edge, on the contrary, due to the lack of such sfink ters, for any change in venous pressure (portal) network immediately direct mode change occurs in the circulation Kapil lyarah portal area. In other words, as a result of the increase in portal pressure, the filtration zone directly increases to the detriment of the suction zone in the internal organs of the abdominal cavity served by the portal vein.

An important addition to this hypothesis is the recognition of the role of capillary permeabilities: under certain conditions, an increase in the permeability of the capillary endothelial membrane can transform capillary mechanism of extravasation, without resorption.

For the peritoneal-portal zone, which is the subject of our research, an important reason for the increase in the penetrating ability of the capillaries is inflammation and irritation of the peritoneum. In the liver, as in any organ or tissue, the filter portion of the capillary should limfati cal way. Thus, the lymphatic vessels fill differently depending on the volume of fluid around the capillaries, which, as has been seen, increases simultaneously with the increase in venous pressure. In patients with cirrhosis with ascites, a doubling of the thoracic canal is established, with an increase in the volume of lymphatic fluid by 3-6 times. For hepatic venous capillary pressure of the capillary region is not more Portal nym (in other intraabdominal organs), and is the pressure of the inferior vena cava or the suprarenal veins. In such capillaries, any intrahepatic morphological disorder, entailing deviations of the suprahepatic veins, can lead to difficulty in draining blood to the suprahepatic veins with an increase in capillary filtrate. Thus, there are conditions that are realized both in the Budd-Chiari syndrome (suprahepatic barrier) and in ordinary cirrhosis.

Under these circumstances, the liver itself becomes the site of significant fluid excretion, or, in other words, the liver is sometimes an important cause of ascites. The above authors argue that it is possible to observe the appearance of ascitic fluid in the form of drops on the surface and in the gates of the organ.

However, if the pathogenesis of cirrhotic ascites reduced only to the recognition of equilibrium capillary filtration - resorption, it means not to take into account Vat most specific element of cirrhosis, namely the complex nature of metabolic disorders, circulatory complicates Modifying the katsiyami. So, already giposerinemiya cirrhotic patients is a rather complicated mechanism of a result of participating in this supply shortage phenomenon, food preliminarily failure and, finally, the very failure of metabolic liver processes that entail subnormal synthesizing serine liver cells damaged or regenerated morphologically and impaired functional. Consequently, and especially giposerinemiya op -determination general serinemii (in total plasma) from the start of cirrhosis indicates the status of functional liver failure. Respectively governmental raising globulin resulting functional hut accuracy HPP or autoimmunity phenomena, even if it is in grams and equalizes serine deficiency, not nearly enough to keep the normal colloid osmotic (oncotic) pressure, due to the fact that, based on the one grams, there are fewer large globulin molecules than small serine molecules.

In this regard, it should be noted the violation with cirrhosis and water and electrolyte metabolism, which is an important factor in the postoperative term treatment.

For a long time in patients with cirrhosis showed an increase in plasma volume , we, the volume of blood remained in the normal range by reducing the volume of red blood cells - a fact Confirm flown to and when we carried out the definitions using radiolabeled iodine serum albumin. Determination of the volume of extracellular water using radioactive. They do not lead

to the establishment of a significant increase in this zone, however, the interpretation of this phenomenon is a very difficult matter, in view of the fact that with cirrhosis it is sodium metabolism that greatly deviates from the norm.

Normally, the liver breaks down (metabolizes) excess adrenal cortical hormones that would otherwise cause excess sodium retention at the level of the renal tubes. Functionally deficient cirrhotic liver contains excessive amounts of certain sterol fractions wasp fected sodium significant delay (apparently, aldosterone). A recent study it was found that there is a mechanism in the liver remote STI emulation aldosterone secretion by the adrenal cortex. With increasing intrahepatic vascular pressure (as in cirrhosis with portal hypertension) thereby arises secondary hyperaldosteronism bli with a nearest its consequence - Water + sodium delayed, then the appearance relative hypoproteinemia. In fact, sodium is the total delay deviation of metabolism in patients with cirrhosis, even if patho physiologists concerned prospects therapy found out heads nym, the phenomena taking place in the kidneys. This is evidenced by the fact that in patients with cirrhosis, the sodium content also decreases in saliva, lacrimal fluid, sweating, etc.

Finally; in at lzu this type of secondary kortikosuprarenalizma (point her secondary aldosteronism) patients with cirrhosis of the following facts:

- diurnal cycle diuresis in patients with cirrhosis of the back, that can be obtained in the experiment at the Introduction SRI extracts of the adrenal cortex;
- Bilateral adrenalectomy allowed for long-term control of some recurrent ascites.

Practical application, due to these observations, anti-aldosterone treatment has been very effective. An important role in the disturbance of water-electrolyte metabolism is played by the relationship between the posterior lobe of the pituitary gland and the kidney.

It is proved that by increasing the plasma concentration of antidiuretic hormone secreted by the posterior lobe of hypo Fiza (vasopressin) osmoreceptors while being in the vascular bed of the internal carotids. A normal person subjected gipergidra tation, antidiuretic hormones no longer produced; in patients with cirrhosis, despite the increase in plasma hypotension as a result of a water test, this hormone still differs in the urine. Among the various hypotheses trying to explain the presence of the parallel delay of water and sodium, the most convincing allocated hypothesis that patients with cirrhosis adapted to a specific urs nude hyponatremia. In this case any intake of salt (delayed organisms IOM due to hyperaldosteronism) leads to the relative increase of sodium level in the internal environment by stimulating osmoreceptors to gipersekre tion antidiuretic hormone.

It should be noted that in a decompensated patient with cirrhosis with ascites, respiratory disorders are noticeably manifested, which exist latently in all chronic patients with hepatitis and compensated patients with cirrhosis. We are talking about hyperventilation, venous mixing (through intrapulmonary shunts) and arterial hypoxia. In post operative period the patient with cirrhosis threatening lung infection and "wet lung".

Thus, these concepts of pathological physiology come to help the surgeon for the correct interpretation of clinical facts and logical orientation in the pre- and postoperative treatment of patients with cirrhosis with ascites.

Clinical research and drug treatment. It has already been noted above that in almost 28% of patients with cirrhosis, ascites is the first sign of a clear establishment of cirrhosis. It is assumed that more than 50% of patients with advanced stage of cirrhosis also have ascites. In practice sometimes difficult to deny the presence of fluid in the abdominal cavity due to the fact that to 1500 ml of free liquid can uskol znut by percussion or percussion hardly detected by a knee-pectoral position.

Until the stage at which ascites becomes clinically detectable, this is preceded (or noted) by the appearance of significant flatulence, with a feeling of unpleasant sensation, less often with an unpleasant sensation in the abdominal area, and most often as constant overcrowding. This subjective Nome sensation corresponds objective abdominal enlargement in the circle and the stress state of the abdominal wall. At the same time, the patient observed given daily urine

volume reduction with increase in night-time and delay the increase in diuresis after absorption of large quantities of water.

Only with very careful and detailed observation by specialists can one detect a phase consisting in a too strong increase in diuresis, in excess of the volume of water drunk once - an early phase of a disorder of water and electrolyte metabolism, which takes place simultaneously with the establishment of obvious decompensation (ascites). Internal medicine treatises detail clinical and complementary studies of ascites. Of these, we note the following:

- The patient is pale, with emaciated limbs as a result of muscle atrophy; the abdomen is enlarged, there are often small supra-malleolar edema (large ascites).
- Belly: froglike - in an inclined position or in the form of a saddle bag in an upright position with an average sagging; spherical with large ascites.
- On percussion: dullness moving along an inclined line with upward concavity with moderate ascites; sub-umbilical and lateral dullness of less mobility with ascites of a very large volume (fluid has nowhere to move).
- Wave-like sign with medium-sized ascites.
- Trial puncture: gray-lemon transudate liquid (first puncture) or orange with the presence of granulocytes (after several punctures).
- Subcutaneous porto-caval circulation can be replaced with long venous ducts on the sides (cava-caval type), while in the case of significant accumulations of fluid, the inferior vena cava is compressed.

In other respects the form and volume of the liver and spleen are not relevant to step astsi one and are specific to cirrhosis. Only giposerinemiya, with a return ratio of serine-globulins, indicating hepatocellular DEKOM pensation, and gives the key to one of the sides of the pathogenesis of cirrhosis.

Being present in a latent form or erased many of the patients with cirrhosis, ascites becomes in some parts dominant symptom, assuming the character of complications due to a significant nature vyzy ously disorders.

Ascites large amount (more than 12 liters) leads to distension of weakly -singular points of the abdominal wall and more pronounced exudation latent hernias, especially at the umbilical ring, wherein the elevation protrudes a - sign that the formed small hernial sac stretched liquid. Sometimes here the thinned wall does not withstand, it cracks and a significant part of the liquid is poured out. But soon the sense of relieve th cheniya patient is replaced by the worsening evolution of peritonitis as a result of infection with ascites.

Surgery. As a result of pathological and physiological studies it was found that ascites occurs as a consequence to popular Denia between production and resorption of the peritoneal fluid. Various proposed methods are tried and surgical vmesha stances affect either the first or the second phenomenon. In the case when it is possible to bring them into line, the ascites disappears. Success is temporary or long-term, depending on the duration of the operation act, as well as on the possible addition of other ascytogenic factors, in addition to those that existed before the operation. Methods to promote resorption or drainage of ascitic fluid. These methods differ from pumping puncture in that they are used to recuperate fluid, a fact that is important due to the saving of proteins more than water or salts. In general, considering the issue of surgical treatment of ascites, it can be stated that there are a large number of surgical techniques, which reflects the state of the issue at the moment, not yet fully resolved. At present, quite satisfactory results have been achieved, namely, various methods of "internal drainage of ascitic fluid" have been proposed to replace the exhausting pumping puncture.

Wide drainage methods had very limited effectiveness, as a result of the fact that they soon found themselves covered with a large omentum and isolated from the large cavity, ceasing to have a drainage effect.

It was also proposed to carry out the capillary drainage of ascites using non-absorbable thread drawn from the stomach into kletoch hydrochloric tissue of the abdominal wall. Soon around such thread (made even from the well-tolerated plastics) develops fibrous tissue that

surrounds and penetrates them in their structure, filling the capillary nye space. Thus, the effectiveness of this method is limited in time. Omentopexy has found wider application .

PRACTICAL PART

THEMATIC TESTS

1

1. Select a pathology that cannot be the cause of extrahepatic portal hypertension:
a) portal vein atresia
b) cavernous transformation of the portal vein
c) phlebosclerosis of the portal vein
d) portal vein thrombosis caused by various inflammatory diseases
e) block of hepatic capillaries of the portal vein network.

2. Select a rare symptom of portal hypertension from the following:
a) the occurrence of collateral circulation
b) splenomegaly
c) hemorrhagic manifestations
d) ascites
e) jaundice

3. Select the study with the greatest information content in determining the level of the portal circulation block:
a) EGDS
b) laparoscopy
c) ultrasound of the liver
d) celiacography
e) endoscopic retrograde pancreatography

4. What is Budd-Chiari disease?
a) endophlebitis of the hepatic veins
b) liver amyloidosis
c) dropsy of the gallbladder
d) hemochromatosis
e) persistent hepatitis.

5. Select a common type of portal hypertension:
a) intrahepatic
b) suprahepatic
c) subhepatic
d) mixed
e) dynamic

6. What is the normal pressure in the portal system?
a) 50-150 mm water column
b) 50-150 mm Hg.
c) 150-250 mm of water column
d) 10-50 mm of water column

e) 250-500 mm of water column

7. What is the absolute indication for surgical treatment?
a) bleeding from varicose veins of the esophagus
b) ascites
c) jaundice
d) liver cirrhosis
e) splenomegaly

8. What is the laboratory indicator of hypersplenism?
a) anemia
b) thrombocythemia
c) leukopenia
d) thrombocytopenia
e) erythrocythemia

9. What is the basis of primary biliary cirrhosis?
a) intrahepatic cholestasis
b) extrahepatic cholestasis
c) impaired blood supply to liver tissue
d) portal hypertension
e) hyperbilirubinemia

10. The purpose of omentohepatophrenicopexy in liver cirrhosis:
a) improving the blood supply to the liver tissue, reducing portal pressure
b) a decrease in the level of bilirubin in the blood
c) improving the blood supply to the spleen tissue
d) fixation of the diaphragm
e) increased urine output, decreased renal pressure

1

SITUATION TASKS

1. A 40-year-old patient consulted a polyclinic doctor complaining of pain in the upper abdomen. The skin is of normal color. The condition is relatively satisfactory. At the age of 20, the patient suffered from viral hepatitis. At present, an abuse alkogo Lemma. Clinically revealed signs of portal hypertension, splenomegaly. What can you think about?
a) about the suprahepatic block
b) about the intrahepatic block
c) about the subhepatic block
d) about the mixed block
e) about hypersplenism

2. A 50-year-old patient turned to a therapist of an admission department of a hospital with complaints of an acute onset of the disease (acute pain in the right and left hypochondrium, a sudden increase in body temperature. From the anamnesis it is known that before the present visit to the doctor, the patient was hospitalized and examined. The physical and instrumental

studies niyah at the time of initial hospitalization liver and spleen were enlarged in size liver and spleen is not significantly increased, dullness in sloping areas the abdomen ultrasonography revealed hepatosplenomegaly, ascites diagnosis...:

a) Chiari disease
b) liver cirrhosis
c) pylephlebitis
d) atresia of the branches of the portal vein
e) chronic indurative pancreatitis

3. The patient is 42 years old enrolled in the surgical department of the hospital with the sting Bami to vomiting blood, weakness, loss of consciousness. From the anamnesis it is known that 5 years ago he was hospitalized in an infectious diseases hospital for viral hepatitis, notes alcohol abuse. On physical examination, the patient's condition is of moderate severity, pulse 105 beats per minute, rhythmic, BP - 90/60 mm Hg. The tongue is dry, the stomach is swollen, palpation reveals a painful enlarged liver, an enlarged spleen. With rectal examination - melena. Define your actions as a surgeon on duty:

a) hemostatic conservative therapy, dynamic observation
b) gastric lavage with ice water to prepare for emergency EGDS
c) emergency operation
d) ultrasound of the abdominal organs
e) emergency EGDS

4. A 44-year-old patient was admitted to the hospital in order to provide emergency care. Suddenly fell ill. Singing wound abundant repeated bloody vomiting. The patient was sharply weakened. From the anamnesis it was possible to find out that he abused alcohol for many years. When obsl dovanii patient drew the attention of the pain Shai dense spleen. The liver was not palpable. Which can serve as sources whom bleeding? Between what diseases follows blowing conduct the differential diagnosis?

5. A 42-year-old patient was admitted to the hospital in order to provide emergency care. Hematemesis arose la suddenly. The patient said that during the 2 years that some bleeding starts in the third time. History of Botkin's disease. On examination, there is a pronounced venous network on the abdomen in the form of a "jellyfish head". Palpates Xia large spleen and dense edge of the liver in the ribs Noah arc. You have been diagnosed with bleeding from varicose veins of the esophagus. What will be the emergency and subsequent pain relief Nome?

THEORETICAL PART

Portal bleeding

Bleeding from varicose veins of the esophagus and stomach is the most dramatic complication of portal hypertension.

The risk of first bleeding among patients with liver cirrhosis is 5% per year. Mortality at the first bleeding is about 50%. Even more pessimistic is the prognosis for patients who have had bleeding in the past. The incidence of recurrent hemorrhages is 50–90%. The mechanisms of development and progression of portal hypertensive changes in the cardioesophageal zone, increased portal pressure leads to the spontaneous formation of portosystemic collaterals. Despite the fact that any venous vessel of the gastrointestinal tract can undergo varicose veins, changes in the veins of the cardioesophageal region are of the greatest clinical importance.

The vessels of the submucosal layer and the external venous plexus are connected by perforating veins that penetrate the muscle layer of the esophagus. With an increase in portal pressure, the vessels in this area undergo varicose veins. It is assumed that bleeding of moderate

severity occurs due to rupture of intraepithelial vessels, while rupture of the veins of the submucosal plexus leads to hemorrhages with massive blood loss.

There are several theories for the development of bleeding due to rupture of varicose veins. One of the earliest is reflux.

It was believed that varicose veins lead to a violation of the closure function of the cardiac pulp, the throwing of the acidic contents of the stomach into the lumen of the esophagus, the formation of erosion and rupture of blood vessels. Autopsy data from patients who died from complications of portal hypertension indicate that in 50% of cases it is possible to identify inflammatory changes in the esophageal mucosa.

Portohypertensive changes can create preconditions for diffuse bleeding. In such clinical situations, endoscopic signs resemble inflammatory changes in the mucous membrane (hemorrhagic gastritis, multiple acute stomach ulcers). By their pathogenetic essence, these changes are portal - portal hypertensive gastropathy.

At the heart of the development of gastropathy are not inflammatory, but portohypertensive changes - ectasia of capillaries and venules of the mucous and submucous layer, which in severe cases are accompanied by edema and hyperplasia of the mucosa.

There are two degrees of severity of portal gastropathy: - a mild degree is characterized by the presence of pinkish-red spots on the mucous membrane, superficial redness, mosaic-like edema of the mucosa; - severe degree is characterized by the presence of diffuse dark red spots or hemorrhages (as in hemorrhagic gastritis).

It is advisable to use a point-based assessment of the severity of portal gastropathy.

0 - no gastropathy

1 - mild

2 - severe

3 - portal gastropathy complicated by bleeding.

The pathogenesis of portal gastropathy has not been fully explained.

It should be noted that there was no correlation between the severity of hypertensive gastropathy and the degree of portal hypertension or the functional state of the liver.

Endoscopic diagnosis of bleeding. The period has not yet been forgotten when, with suspicion of esophageal varicose veins, endoscopic examination was considered contraindicated due to fears of provoking bleeding.

However, with the accumulation of experience and the evolution of surgical methodology, endoscopy gradually took a key place in the diagnosis and prognosis of the course of complications of portal hypertension. If no one doubts the dominant role of endoscopy in the diagnosis and assessment of the degree of varicose veins, then there is no consensus regarding the advisability of a study at the height of bleeding. Concerns are expressed in connection with possible complications of the diagnostic procedure on an emergency basis, the danger of aspiration or the provocation of more massive bleeding.

The prospect of widespread use of endoscopic examination is determined by the possibility of:

-Combination of diagnostic and therapeutic stages in acute bleeding;

- identification of risk factors for bleeding in order to address the need for primary prevention of hemorrhage;

-estimation of the likelihood of recurrent bleeding and the choice of therapeutic tactics to prevent recurrence.

The aim of the study in acute bleeding is not only to detect the rupture of varicose veins, but also to exclude the source of bleeding of a different genesis, as well as to plan the subsequent therapeutic intervention. The presence of "fresh" blood clots in the lumen of the esophagus and stomach reflects the ongoing pattern of bleeding. Visualization of the site of rupture of the varicose wall with jet discharge of blood or its insignificant outflow allows you to accurately localize the place of bleeding. In the case when the endoscopic examination is performed after

the bleeding has stopped, the main sign indicating the place of hemorrhage is the presence of a fixed parietal thrombus in the projection of the varicose vein.

During endoscopy, in addition to direct signs of varicose bleeding, the prognostic significance of all local factors should be assessed:

- the size, shape and color of varicose veins;
- the presence and prevalence of "red markers" of the wall (spots of "red cherry", hematocyst spots, telangiectasia);
- severity of portal gastropathy.

Varicose veins of the esophagus:

I degree - the diameter of the veins does not exceed 5 mm, elongated, located only in the lower third of the esophagus;

II degree - varicose veins with a diameter of 5 to 10 mm, convoluted, spread in the middle third of the esophagus;

III degree - the size of the veins is more than 10 mm, tense, with a thin wall, located close to each other, on the surface of the veins "red markers".

Varicose veins of the stomach:

I degree - the diameter of the veins is not more than 5 mm, barely visible above the gastric mucosa;

II degree - varicose veins ranging in size from 5 to 10 mm, solitary-polypoid character;

III degree - veins with a diameter of more than 10 mm, represent an extensive conglomerate of nodes, thin-walled, polypoid in nature.

Non-operative methods of treatment for acute gastroesophageal bleeding

Obturator probe (Sengteiken-Blackmore). Installation of an obturator probe is an effective method for stopping varicose esophageal-gastric bleeding. However, after the end of venous compression, the frequency of recurrent bleeding in the early post-hemorrhagic period reaches 50% or more. Blackmore probe consists of rubber triple lumen tube with two circular cylinders and cylindrical shapes two channels serve as a probe for inflating balloons, the third (open vayuschiysya in the distal portion of the probe) for the aspiration of gastric contents (control efficiency hemostasis).

Probe Blackmore administered through the nose into the stomach is inflated distal (zhelu zling) balloon pumping 60-70 mL of air. Then the probe podtyagi vayut until resistance is felt, which occurs when the balloon localization in the cardia. After that, 100-150 ml of air is injected into the esophageal balloon. In this state, elastic wall Nogo cylinder exert a uniform pressure over the entire circumference of the esophagus, squeezing the vein bleeding distal food water and cardia of the stomach. After a few hours, the pressure in the balloons is released, monitoring the hemostasis effect using the probe. The duration of the stay of the probe in the esophagus should not exceed 2 days due to the risk of pressure ulcers.

The use of phlebosclerosing drugs (varicocide, thrombovar, etc.) in the treatment of bleeding from varicose veins of the esophagus is carried out through an esophagoscope. Special needle through an endoscope into a vein or paravenozno administered drug call conductive venous intima damage, obliteration of its sticking to about light. In order to create favorable conditions for adhesion of the walls of varicose veins, a Blackmore probe is introduced for 1 day.

Endoscopic methods

The poor tolerance of patients with cirrhosis of the liver of extensive, traumatic surgical interventions has always dictated the need to search and improve low-traumatic methods of treatment. One of the most important minimally invasive areas of treatment for this category of patients is endoscopic interventions, which are recognized as the "first line method" both at the height of bleeding and at a high risk of its development.

At the height of bleeding, endoscopic sclerotherapy is associated with better hemostasis control and survival compared to vasopressin therapy. When comparing sclerotherapy and therapy with somatostatin or sandostatin, there are no significant differences in indicators.

Emergency endoscopic sclerotherapy has advantages over esophageal balloon tamponade.

The recurrence rate of bleeding after using sclerotherapy reaches 40%. In 20%, relapses of bleeding are associated with the development of post-injection ulcers. The incidence of complications ranges from 1% to 20%.

To date, the range of drugs used for sclerotherapy includes: ethanolaminoleate, sodium morruate, sodium tetradecyl sulfate (thrombovar), polidocanol (ethoxysclerol), ethyl alcohol, phenol, tissucol (beriplast), histoacryl (bucrylate).

Surgery

According to the main pathogenetic orientation, portosystemic shunting and portoasigal separation are distinguished.

Reducing the portal pressure by portosystemic shunting eliminates one of the decisive pathogenetic factors of bleeding from varicose veins of the esophagus and stomach.

The use of vascular portosystemic shunts in clinical practice has raised a number of problems. Operational trauma often becomes intolerable for the majority of decompensated patients with liver cirrhosis. Portosystemic encephalopathy syndrome occurs in 5–52% of operated patients.

After portosystemic shunting, the processes of liver regeneration are reduced. In choosing the type of shunt, there are a kind of "scissors": the larger the diameter of the shunt, the more pronounced the decompressive effect of the intervention, but at the same time the higher the postoperative lethality, more often encephalopathy occurs, the shorter the duration and lower quality of life of patients. Conversely, a small diameter of the shunt is accompanied by lower mortality, encephalopathy, and moderate suppression of hepatic regeneration, but it is often ineffective in reducing portal pressure. The main disadvantage of portosystemic anastomoses is associated with a significant decrease in portal liver perfusion and an increase in the volume of shunting blood.

Selective drainage of the gastrolienal zone is pathogenetically substantiated in liver cirrhosis. In this case, the goals of both decompression of the cardioesophageal zone and the preservation of slight hypertension in the portal vein system, which ensure the maintenance of portal perfusion, are pursued. One of the options for selective portosystemic shunting that meets the above conditions is the *Warren* distal splenorenal anastomosis . Lack of distal splenorenal shunting is associated with a risk of injury to the pancreas.

Currently, more attention is paid not to bypass selectivity, but to the degree of portal decompression. The dominant position is occupied by the concept of partial (dosed, partial) portal decompression. The optimal diameter of the anastomosis is 6–8 mm.

At the same time, the risk of bleeding is significantly reduced, and a decrease in liver function and aggravation of portosystemic encephalopathy are not expressed.

The procedure for *extrahepatic intrahepatic portosystemic shunting* is one of the latest innovative technologies to perform efficient decompression of the portal system.

In the English-language scientific literature, it has become generally accepted to use the abbreviation TIPS (Transjugular intrahepatic portosystemic shunt) to denote this intervention.

PRACTICAL PART

Thematic tests

1. A 26-year-old patient who was admitted to the clinic with gastrointestinal bleeding, during emergency gastroduodenoscopy, revealed a chronic duodenal ulcer with a thrombosed vessel and the threat of recurrent bleeding. Your tactics:
* a) urgent operation;
b) conservative treatment;
c) embolization of the vessels of the stomach;
d) surgery for recurrent bleeding;
e) everything is correct.

2. In Mallory - Weiss syndrome, the mucous membrane is affected:
a) the esophagus;
* b) the cardiac part of the stomach;
c) antrum of the stomach;
d) small intestine;
e) rectum.

3. Stress stomach ulcer manifests itself more often:
a) perforation;
* b) bleeding;

c) penetration;
d) malignancy;
e) none of the above.

4. To establish whether the patient's gastrointestinal bleeding continues or has stopped, it is possible with the help of: 1) fluoroscopy of the stomach; 2) laparoscopy; 3) a nasogastric tube; 4) a blood test for serum iron content; 5) gastroduodenoscopy. Choose the correct combination of answers:
a) 1, 2, 3, 4;
b) 3, 4, 5;
c) 1, 2, 3;
d) 4, 5;
* e) 3, 5.

5. For a bleeding duodenal ulcer, the following symptoms are characteristic: 1) increased abdominal pain; 2) vomiting of the "coffee grounds" type; 3) reduction of pain syndrome; 4) Shchetkin's symptom - Blumberg; 5) melena. Choose the correct combination of answers:
a) 1, 3, 5;
b) 1, 2, 5;
* c) 2, 3, 5;
d) 2, 3, 4;
e) 1, 2.

6. What complication of duodenal ulcer disease is characterized by the disappearance of pain and the appearance of melena;
a) pyloroduodenal stenosis;
b) perforation of the ulcer;
c) malignancy of the ulcer;
* d) bleeding;
e) penetration into the pancreas.

THEME. ECHINOCOCCAL DISEASE

The purpose of the lecture : to familiarize students with echinococcal disease, *the* methods of transmission, with methods of diagnosis and treatment.

Educational goals: Lecture enhances students' knowledge about the range of echinococcosis, its possible complications and methods of prevention and treatment is a preparatory material *for* Practical and cal studies.

Lecture Objectives: To give the concept of hydatid disease, epidemiology and etiopathogenesis echinococcosis, familiarize with clinical manifestations and complications of the disease, disclose methods of diagnosis and differential e differential diagnosis, to introduce modern methods of treatment of echinococcosis.

Lecture plan.

1. Echinococcosis and its epidemiology - 15 min

2. Etiopathogenesis of echinococcosis - 15 min

3. Methods of research of echinococcosis of the lungs and liver - 20 min

4. Clinic, complications, differential diagnosis of echinococcosis - 20 min.

5. Treatment of echinococcosis - 20 min

Hydatid disease of the liver, according to current opinion of issledovat e lei, including lesions in various organs and tissues occurs most cha with it, ranging from 44.2% to 84.3% of cases.

It is generally accepted that human infection with echinococcus is carried out by o n cosphers, secreted by tapeworms, which parasitize the dog's intestines. R.P.Askerhanov indicates that infection Split timing of the first host, including humans, can occur not only as a result of ingestion of eggs vomvta dog, but also by eating raw or Paulus s swarm .pecheni containing elements of hydatid cysts.

Human infection with canine alista embryos can occur in three ways: through the mucous membranes of the gastrointestinal tract, through the mucous membranes of the respiratory tract and through the wound surface. On and more often penetrate through the gastrointestinal tract.

Possibility aerogenic way germ infestation of tapeworm in by previously detecting hexacanth in the air while maintaining their zhiznesposo b Nosta in these conditions.

Three ways of penetration of echinococcus embryos are through the wound surface when a dog bites, on whose teeth there are viable oncospheres, or when the embryos enter the operating wound.

Unilocular echinococcus of the liver develops slowly. According to A.V. Lvov, the value reached per year by individual echinococcal cysts is different. It varies from 2-3 to 20-30 mm in diameter. In the presence of optimal and malignant conditions, cyst development occurs rather quickly. In other conditions infiltrated embryo may die while undergoing total p e resorption or encapsulation type of a foreign body.

According to the characteristics of the pathogenesis, R. Askerkhanov distinguishes five varieties of the disease in humans:

1. Primary hydatid disease, growing in one or more of the p ganah after penetration into the organism viable oncospheres.

2. Secondary multiple echinococcosis in the abdominal or plevral v hydrochloric cavity cysts developing after perforation.

3. Metastatic echinococcosis resulting from the opening of echo and nococcal cysts into the heart cavity or into a large blood vessel.

4. Implantation echinococci, appeared after exposure scoleces or oncospheres in operating (during operation) or other p as well.

5. Heterotopic echinococcosis, perceived mainly as a theoretical possibility. For example, a cyst, is located in the subpleural lung, after the rupture of the fibrous capsule without breaking the n th Lost chitin and cuticular membranes enters the pleural cavity, where its development continues in the new environment and s.

Clinic of liver echinococcosis. The clinical picture echinococcosis n e Cheney diverse and largely depends on the phase of development of the parasite, and on other factors. Often, this is accompanied by weak on Stu, general malaise, appetite loss, fatigue with or without weight loss, dyspnea. Periodically Observed and are allergic reactions such as repeating urticaria, diarrhea, nausea, and vomiting. An objective examination reveals an increase in the liver (more often its right sections or all sizes of an organ with multiple cysts).

When localization anteroinferior parasitic cysts on the liver surface can be determined by the visible bulging anterior abdominal ste n ki, and at the location of its lateral - deformation costal arch and rib-like hump. The enlarged liver is dense on palpation. On its surface can be palpable protrusion having a smooth power metal x NOSTA hemispherical shape and typically plotnoelasticheskoy woody consistency or density (for calcified cysts walls). the symptom of fluctuation is determined more closely.

Palpation are usually painless, but pain can be regex and wife with festering cysts or inflammation of the tissue around it. In some cases, effleurage over the area location ki with you reveals a symptom of "gidatid shake" typical for echinococcosis.

The third stage is characterized by a variety of complications Echinococcus about over. They can develop as a result of changes taking place not only in the parasitic cyst abscess, perforation, obyzvestvl e of), but also in the affected organ or the body as a whole. Examples of such a complication in the occurrence of ascites are progressively during compression of uv e lichivayuscheysya cyst inferior vena cava or jaundice vsleds m Wier obturation inside or extrahepatic bile ducts.

The most common among the complications of liver echinococcosis meets Mr. and suppuration hydatid cysts in patients .If this suddenly appear severe pain in the swelling, the latter increases, it becomes more intense, sharply .boleznennoy palpation. Temperature dressing s creases to 49-41 ^{0}C and takes hectic nature. I grow rapidly in the intoxication Lenie, accompanied by shaking chills and

shed in nym then. In the future, a septic condition may develop. Poss of wives as a breakthrough abscess into the pleural or peritoneal cavity, BOAD th bus or perirenal tissue.

It is also a serious complication gap neinfitsirov as hydrochloric hepatic hydatid cysts, occurring in 6-9 cases. This typically occurs at high superficial bladder with ISTO n chennymi walls. Cyst rupture can occur during a medical with Mothra - with abdominal palpation, pelvic exam. In these cases, the contents of the cyst along with its subsidiaries and bubbles of Lebanon into the abdominal cavity, causing local spread of echinococcosis. In s account may also break hydatid cysts and empty the gallbladder, vnutrip e chenochnye bile ducts, intestine or stomach, and when lock and tion of it on the diaphragmatic surface of the liver in plevral v hydrochloric cavity, lung, bronchus. In the first case, wherein b develops severe attack on leu as in cholelithiasis, cholangitis and jaundice, in the second - to a strong and cough with sputum containing hydatid bladders, torn cuticle p hydrochloric shell purulent pleuritis. breakthrough in the gastrointestinal tract of the elements of hydatid cyst B has also been observed in the feces.

Calcified echinococcosis of the liver is observed in patients. Zab about Levan is often asymptomatic and is found mainly in the X-ray examination. Only in cases where there are large cysts, dull pain and heaviness appear in the right hypochondrium. Occasionally, cysts compress the bile ducts and cause obstructive jaundice. The timely detection of these or other at GIH hepatic echinococcosis complications is very important, since it allows to work out the appropriate treatment strategy. Therefore, the clinician neo b walk to know the clinical course of both uncomplicated and complicated echinus on Kokkoz liver.

Very characteristic symptoms of this localization is a feeling of heaviness and raspivaniya in the liver, as well as the feeling of pen e complements epigastric, worse after eating, when running and walking fast.

The objective signs are the increase in the upper abdomen, extending intercostal spaces and asymmetry or vypyachena and of the lower parts of the chest, enlarged liver with a percussion extension dullness borders both downwards and upwards, weakening or otsuts m Wier respiratory noise at the bottom of hydrochloric cells chest. more often on the right, Mr. and presence of palpable tumor formation in right or left on the d edges in the epigastric region.

It should be emphasized that despite the presence of these symptoms, the state of the majority of patients remained quite satisfactory.

When complicated forms poddiagfragmalnogo hepatic echinococcosis, occurring much more frequently than other localizations appears t Xia symptoms characteristic of the presence of festering oslozhneniya.Pri in sheysya hydatid cysts on fore INTOX signs and katsii sometimes flowing chronically and at the perforation and breakthroughs of a Rouge authorities of the symptoms of damage

(piopnevmot of raks, purulent sputum with elements of tapeworm, the bile peritonitis, the MBT in the diet jaundice etc.).

Most frequently hydatid cysts of the liver, localized in at d phrenic space, break toward the chest cavity and its organs to form empyema, purulent cavity in the lung, bronchial and even bile-bronchial fistulas.

In the latter case, patients expectorate bile-colored phlegm with elements of echinococcus and a characteristic bitter taste of bile.

DIAGNOSTICS OF ECHINOCOCCOSIS OF THE LIVER

Early diagnosis of liver echinococcosis, despite the success of present-day e mennoy surgical hepatology, often more difficult. This St. I associated with a lack of clear symptomatology of the disease, especially in the early period of its development, as well as the location of the cysts in the body depth. Therefore, timely and correct diagnosis can significantly reduce the number of postoperative complications and improve the outcomes of surgical treatment.

CLINICAL AND LABORATORY STUDIES

Clinical and laboratory methods of blood testing in hydatid echinococcosis of the liver make it possible to obtain additional, sometimes very useful information to clarify the diagnosis and the stage of development of the parasite in the preoperative period, first of all, this refers to an increase in the number of eosinophils. However, n is not eosinophilia and reliably con- stant and hepatic echinococcosis feature, since an increase in the percentage of eosinophils observed for other parasitic diseases. In addition, eosinophilia is characteristic only with a living parasite. When both of vestvlennyh, dead or festering cysts when pas reeks is killed, this symptom is absent. After removal of the echonococcal cyst, eosinophilia and lia , as a rule, are eliminated after 4-6 months.

X-RAY STUDY

A role in the recognition of echinococcosis liver Acc e INH radiographic studies, through which Representat a wish to set up possible to identify a number of characteristic features of the disease: obyzvestvl e NIJ walls parasitic cysts, increased liver size, height about some distance or different deformation of the diaphragm dome, constraint d mobilities the latter, and the displacement bodies disposed al. informative in Nye value radiographic examination depends on ra with cysts position and its state, that determines the occurrence of direct or indirect radiographic symptoms of hepatic echinococcosis.

Application of artificial pneumoperitoneum allows Differen n -skilled hydatid cyst diaphragmatic surface of the liver from p e relaxation aperture or neoplasms of the lower lobes of the right lung. Introduced gas, located between the liver and diaphragm, is easily visible as light bands against which clearly

determines I etsya generally oval rounded bulging dome enlarged liver cysts or shade.

A suppurating echinococcal cyst of the liver is radiologically difficult to distinguish from a liver abscess. However, in the presence of calcification around the cavity with a liquid level and, especially, when determining floating daughter cysts and chitinous membranes, the diagnosis of echinococcosis is facilitated.

RADIO ISOTOPE HEPATOSCANING

Radioisotope gepatoskanirovanie based on a polling n of absorption liver cells radioactive dyes, rose bengal or radioactive colloidal gold solution EXPERIMENTAL n experimentally been developed and first used in the clinic for diagnostic Novo b mations liver.

In the most common hepatic echinococcosis skanograficheskim Prizna and com is the presence of one or more defects accumulation radio to tive drug. Sometimes the defect accumulating isotope occupies the entire area of the right or the left lobe of the liver, and the border areas of functioning river gana move down, down and left or just left.

Most of the authors emphasize the opportunities computer f p tomography in the detection of morphological changes of the liver cystic on the first character, in particular the differential diagnosis of echinococcosis. Computed tomography certainly has great advantages n th ed other methods and identifying smaller and single foci. Ehin of coccoid cysts detected on a computer tomogram as okra g mated zones with reduced absorption coefficient, thus it is possible to identify the formation of I cm in diameter. In addition, to the m computed tomography can be successfully used in the post-operative period to control p and dikalnosti the operation, detection of complications in vmesh zone and stances and relapse.

SELECTIVE CELLIACOGRAPHY

In the preoperative diagnosis of liver echinococcosis , selective celiacography is of great importance , which makes it possible to clarify the localization, size and nature of the pathological focus. The district and worth the time becoming increasingly widespread use of the village to tive angiography of the abdominal cavity. Proposed author m e Tod is that the probe is introduced into the aorta by percutaneous puncture of peripheral limb arteries. for this purpose uses a special needle with manna d Wren, after removing of which is inserted a flexible metal Provo d nick on which probe is inserted into the aorta. Thereafter, the needle guide and ud and lyayutsya. for contrasting vessels, 603 solution of verograph and on or triombrast is used. When echinococcosis convergence characteristic vessels to have voltage and their arcuate offset circumferentially

cysts, which is formed in the corresponding shape avascular portion. In parenhim and toznoy phase angiogepatogramme defect determined to n opacification in the cysts and accumulation of contrast medium around its periphery between kultilyarnoy fibrous capsule shell and - positive and -negative symptom Rusik. When Alveococcosis position and cancer of the liver, unlike Ah and nokokkoza except avascular zones marked amputated of bathrooms, like ragged branch vessels, as well as new plots melkopetlistyh crimped about the formed vessels (hypervascularization symptom), but unusual for N hydrochloric area.

Tseliakografiya - one of the most reliable and modern meth about rows hepatic echinococcosis diagnosis. Despite this, tseliakografiya should not be leading in all cases required by issl e dovaniya with liver echinococcosis due to its technical complexity and with complements. Selective tseliakografiya liver echinococcosis should be performed on strict conditions when more simple and less layer w GOVERNMENTAL research methods can not determine the location, nature and extent of the pathological process.

LAPAROSCOPY

Laparoscopically relatively easy to install hydatid Ehin about coccosis liver and other abdominal organs, except when the cysts are located inside the body in the region of its dome or for d her surface. When an echinococcal cyst is located on the surface of the liver through a laparoscope, a spherical

forms a formation with a smooth matte surface of pinkish-blue color. Liver tissue surrounding the cyst, and more often than not of Menen, clearly mark the boundaries of the fibrous capsule, which is one of characteristic features of echinococcosis Id. Great difficulty watching tons of camping with laparoscopic diagnosis of recurrent echinococcosis, as after previous surgery in the abdominal cavity develops adhesions, prevent the carrying out of this study. In these cases it is necessary to use scan and tseliakografiyu and serologich e skie reaction. In order differential diagnosis when swollen and cysts of the liver Lyakh contact carried laparoscopy combined with biopsy, palpation, and moving the tool bodies. For n on mere suspicion hydatid combined laparoscopy is suitable under anesthesia in the operating room, so if necessary, immediately after lapar of microscopy urgently make surgery on the abdominal organs.

Percutaneous transhepatic holangiography

Transcutaneous transhepatic cholangiography is to puncture the intrahepatic ducts, introduction into them of contrast agents and the e blowing radiological examination. Typically, transdermal cholangitis about graphy is performed under mechanical jaundice: this method allows ACK p dit presence obturation bile prophets specify its localization and s to reveal the cause.

Sometimes, with echinococcosis of the liver, there is compression by a cyst of both intrahepatic and extrahepatic bile ducts, which is accompanied by obstructive jaundice. In such cases, it becomes necessary to perform percutaneous-transhepatic cholangiography.

This method makes it possible to determine the place of compression and select the appropriate type of surgical intervention.

DIAGNOSIS OF LIVER ECHINOCOCCOSIS RECURRENCES

The diagnosis of recurrence of echo and nococcosis of the liver, which, as you know, burns to be true and false, is often very difficult . The first includes cases that have developed as a result of the second invasion of the parasite. They are formed mostly in patients who continue to live and work under the previous conditions of close contact with animals, and many years after operations, which is explained by the slow growth of the parasite.

False relapses usually occur soon after surgery, sometimes several months later. They are conditioned by leaving undetected cysts in the liver during primary plural pores and zhenii last or due to contamination of the germinal parasite elements occurring during operation tackle art.

Diagnostic recurrence difficulties echinococcosis of the liver, especially in the early stages, due to the fact that a number of methods of investigation and Niya, securities in the primary echinococcosis, is little effective in repeated e SRI disease, and laparoscopy, for example, generally can not be due to adhesions formed in the abdominal cavities after surgery. Therefore, while it and lovazhnoe importance is the reaction of latex agglutination. In the case of disease recurrence reaction latex agglutination is again so Xia positive.

TREATMENT OF ECHINOCOCCOSIS OF THE LIVER

1. Echinococcctomy:
a) two-story,
b) one-stage.
2. Echinococcectomy:
a) after formalin treatment,
b) after aspiration of echinococcal fluid,
c) without breaking the integrity of the chitinous membrane.
3. Ideal echinococcectomy.
4. Echinococcectomy:
a) with organ resection,
b) with the removal of the organ,
P. In relation to the residual cavity.
After echinococcectomy:
a) drainage,
b) marsupialization.

A. Open methods:

a) external drainage,

b) marsupialization,

c) semi-closed method,

d) leaving the residual cavity open while restoring the integrity of the covering tissues.

B. Closed methods.

I. With preservation of the residual cavity:

a) covered marsupialization,

b) restoration of only the cut area.

2.filling the residual cavity:

a) antiseptic solutions,

b) vivicol,

c) an omentum or muscle.

3. Elimination of the residual cavity:

a) capitonage;

b) invagination of the free fibrous capsule into the cavity,

c) the release of a part of the fibrous capsule and its invagination,

d) gluing with fabric glue,

e) removal of most of the fibrous capsule.

Sh. Reoperation for biliary fistula.

I. Excision of the fistula course and closure of the residual cavity. 2. Tamping of the residual cavity.

3. Organ resection.

4. removal of an organ.

Treatment

Treatment of echinococcosis of the liver, like other human organs and tissues, is only surgical. In establishing this diagnosis should be no prospect of assign surgical guide slowly, taking

into account the possibility of at any time of life-threatening complications: festering cysts, break it into adjacent organs, rupture ka n Sula and massive infestation tapeworm in the abdominal cavity. In GOVERNMENTAL A similar cases may need to be urgent surgical intervention Český due to severe suppurative processes, the development of b turatsisnnoy jaundice, suppurative cholangitis, peritonitis, kishech Noah neproh about gence, etc. Contraindications to surgery are

only severe cardiovascular insufficiency and deep functional disorders of the liver, often develop in multiple echinococcosis due to the shutdown of a significant part of the liver tissue.

Surgical treatment of patients with liver echinococcosis is the only method that allows them to achieve their complete cure.

Operation with liver echinococcosis should consist of all parasitic cyst removal of the contents (liquid, scolexes, subsidiaries and corolla y give nearly

linear bubbles) and germinal and cuticular membranes (chitin). The fibrous capsule formed in the liver around walls stork in response to the toxic effect of the parasite is not a member larvae Echinococcus and can be left in situ removal of the fibrous capsule always tra in matichno due intimate spayaniya it with the surrounding liver tissue, and usually accompanied by significant and sometimes massive the blood flow e cheniem due to the presence in the zone of a large amount novoobrazova n GOVERNMENTAL blood vessels. If the bile ducts of the liver are damaged, bile flow can be observed simultaneously. Such operations often lead to serious complications and are accompanied by high mortality of Stu. The need to remove the fibrous capsule occurs when it Oba of vestvlenii. Ideal echinococcectomy is the removal of the entire echinococcal cyst without damaging its membranes. This operation can usually in s complete for dangling leg or cysts on the location of its edge in the liver. The ideal is the most optimal echinococcectomy m e Tod operation because cyst removal without breaking the shell chitin serves as prevention of contamination of the abdominal cavity. It should be noted that after performing an ideal echinococcectomy, the postoperative period is short and proceeds smoothly.

Liver resection together with an unopened echinococcal cyst, despite its radical nature, was not widespread due to the complexity and relatively high mortality rate.

Echinococcectomy by opening cysts when properly performed its e SRI - a relatively simple, low-impact and radical vmesh and ments, leading to a stable recovery of patients. This operation, according to the literature, is performed most often. The essence of the operation bookmark th chaetsya that after aspirating the contents of cyst we reveal the cyst cavity and remove the chitin shell. Fibro cavity of hydrochloric capsule, which remains in the liver after removal chitinous Obolo h ki, to be drained and treated with 2% formalin solution in glyceryl and not Deplete scolexes. After drying it is necessary to re-L and kvidirovat, given that the remaining cavities are formed neparaz and tare cysts that often (I2-24%), followed by about abscess b mation purulent, purulent bile fistula.

After removal of echinococcus, it is necessary to eliminate the residual cavity in the liver. It is like a second moment of surgical im e -interference, the final Echinococcectomy.

Elimination of the residual cavity is achieved in the liver serial- s nym overlay catgut purse string sutures on the wall of fibrous capsules in Proceedings of the last lumen. This method was proposed by Delbe in 1998. Yes n ny method is insecure due to the possibility of blood vessels injured printed e audio and bile ducts.

Method of tamponade of the residual cavity with an omentum on the pedicle. After filling poaasti gland is fixed to the individual sutures naru w Nome edge fibrous capsule. The pronounced reparative properties of the omentum contribute to the rapid obliteration of the residual cavity. Note e nyaetsya adhesive method and in combination with other methods of elimination with tatochnoy cavity (kapitonazh by Delbo, tamponade gland, fibro invagination of hydrochloric capsules), which is adding to them.

Drainage of the residual cavity is typically used when festering in shihsya hydatid cysts, parasites deaths involving PLAYBACK and Li--inflammatory changes in the cuticular and fibrous casings, when bol s Shih to a brush with buhtoobraznymi and protrusions and volvulus, when there is no confidence in the complete removal of all the germ cells of the parasite ...

This method makes it possible to regularly wash the residual cavity with antiseptic solutions (dioxidin, furagin, furatsilin) and individually sensitive antibiotics. After stopping fin Incoming e trolled from the cavity drvnazhposle control tightened and subsequently th present is removed. Marsupialization operation. It consists in suturing binding of mean free edge of the fibrous capsule to a wound of abdominal wall, this cavity that m ponir gauze to fill it from the bottom of granulation occurred.

QUESTIONS

to the audience to establish feedback and clarify the achievement of the lecture goal

1. Tell us about the mechanism of human echinococcus infection.

2. Tell about the most typical complaints presented by patients with echinococcus of the liver and lungs in stage 1

3. What is a basic diagnostic method with echinococcosis printed e no 4. List echinococcosis complications of liver and lung

5. In what state of echinococcus is the "sail" symptom observed.

TOPIC: DISEASE OF THE VEINS OF THE LOWER EXTREMITY.

The purpose of the lesson:

1. To study x irurgical anatomy and normal hemodynamics of the veins of the lower extremities.
2. To study research methods, types of operations performed on the veins
3. To study the etiology, pathogenesis, clinical picture and diagnosis of patients with thrombophlebitis of the superficial and deep veins of the lower extremities. Learn the correct diagnosis according to the modern classification. Be able to make a differential diagnosis.

4. Know the basic principles of treatment of patients with thrombophlebitis of superficial and deep veins of the lower extremities, indications for surgical treatment, management in the postoperative period.
5. To study the clinic, diagnosis, methods of treatment of ileofemoral thrombosis.
6. Establish the basic principles of prevention, labor expertise.

Test questions:

1. Anatomy and normal hemodynamics of the veins of the lower extremities.
2. Methods for examining the veins of the lower extremities.
3. Etiology and patogenesis of thrombophlebitis of the superficial and deep veins of the lower extremities.
4. Clinical picture, differential diagnosis of thrombophlebitis of superficial and deep veins of the lower extremities.
5. Principles of postoperative patient administration.
6. Study of a patient with deep vein thrombophlebitis of the lower leg
7. Etiology and patogenesis of ileofemoral thrombosis
8. Clinical picture , differential diagnosis, treatment methods and leofemoral thrombosis.
9. Conservative treatment of thrombophlebitis
10. Surgical treatment of thrombophlebitis of superficial and deep veins of the lower extremities.

THEORETICAL PART

Acute thrombophlebitis of superficial veins

Acute trobophlebitis is understood as inflammation of the vein wall, accompanied by the formation of a blood clot in its lumen.

Etiology and pathogenesis. Among the reasons contributing to the development of acute process are important infectious disease, traumatic injury, malignant novoob mations (paraneoplastic syndrome), allergic Zabolev Nia. Thrombophlebitis often complicates the course of varicose veins of the lower extremities. Acute thrombosis of the superficial veins of the upper extremities is relatively rare, and typically is a corollary intravenous injection, catheterization, continuous infusions of medicaments.

In the pathogenesis of thrombotic disorders have meaning structure venous wall tours, and increasing blood flow slowing rolling emosti blood due to increased activity of procoagulant and platelet hemostasis.

Clinical presentation and diagnosis. The main SIM ptom disease - pain along the vein thrombosed, amplifies schayasya during movement, physical exertion. On examination, hyperemia and edema of the skin, painful induration along the vein, usually clearly delimited from the surrounding tissues, are noted. The circumference of the affected limb card is unchanged or slightly increasing etsya (1-2 cm in diameter).

The general condition of the patients is usually satisfactory, the perature often low-grade body. Only in rare cases does a purulent fusion of a thrombus occur with the development of sepsis.

With progressive course of the disease can thrombosis races voluminous system of the great saphenous vein in the proximal direction beyond a junction saphenofemoral float conductive (the floating) thrombus posing a real threat embolism le of pulmonary artery. The development of this complication is also possible for propagation roubleshooting deep vein thrombosis in the mouth through the small saphenous vein or incompetent perforating veins.

Extremely difficult runs purulent septic thrombotic phlebitis, which can be complicated by cellulitis course, the development of metastatic abscesses in the lungs, kidneys, and brain.

Treatment. The method of treatment is determined by the localization of pathologies Cesky process. Conservative treatment in outpatient settings is possible in cases where the pathological process does not go beyond the lower leg or occupies a limited portion of the upper finite Nost. The complex of drug therapy include drugs, seizing depleting blood rheology, microcirculation, exerting guides inhibitory effect on the adhesive-platelet aggregation function (acetylsalicylic acid, Trental, troksevazin, xantinol nicotinate, nikoshpan) drugs with nonspecific skim antiinflammatory action (reopirin, butadiene , ibuprofen) and giving a hyposensitizing effect (diazolin, tavegil, diphenhydramine, suprastin). It is advisable to apply locally heparin, butadion ointment; obligatory bandaging of the limb with an elastic bandage and dosed walking.

In a hospital setting, this treatment is complemented by the appointment of anticoagulants. As stihanija acute inflammation prescribed physiotherapy: shorter dia Terme electrophoresis trypsin (himopsina), potassium iodide, etc. heparin.

Surgical treatment is indicated when the process extends to the saphenous vein of the thigh (to the border of its lower and middle third). To prevent the femoral vein thrombosis uplink is urgent shown Nye ligation of vein - Troyanova operation - Trendelenburg. If the patient's condition permits, it is advisable to remove the thrombosed vein together with the area of the skin covering it and the infiltrated subcutaneous fatty tissue.

Currently, a surgical method is usually used to treat acute thrombophlebitis of varicose veins. This is due to the fact that the operation removes the underlying disease, reducing disability periods patients and prevents the development of wasps threatening complication in the.

ACUTE THROMBOSES OF THE DEEP VINS OF THE LOWER LIMBS

Etiology and pathogenesis. Deep vein thrombosis is most commonly seen in the lower extremities. Global Developing thrombosis often are patients suffering from cardiovascular diseases, diabetes, obesity, elderly and cancer patients. Deep vein thrombosis often occur after trauma, accompanied yuschihsya fractures, pathological and after a difficult birth, after a difficult and lengthy operations. They can complicate the course of infectious and purulent diseases.

In the development of post-surgical and post-traumatic venous thrombosis plays an important role of tissue thromboplastin, which is in excess comes from the damaged tissue in the shelter -bearing channel.

Acute vein thrombosis is divided into phlebothrombosis and thrombophlebitis. In acute thrombophlebitis, a thrombus forms on an altered section of the vascular wall as a result of exposure to infectious agents, toxins, and trauma. The thrombus is early and rather tightly fixed to the intima. With phlebothrombosis, a thrombus forms in the lumen of an intact vein, is weakly or not at all fixed to its wall, can easily break off and cause pulmonary embolism. Such a thrombus is normally not completely obturate the lumen of the vein, and therefore the clinical cal manifestations of acute phlebothrombosis scarce. However, after 2-3 days in the vessel wall at the site of thrombus occur secondary PLAYBACK -inflammatory changes caused damaging effect BIOL cally active substances, the fixation of the thrombus. By this time, the differences between phlebothrombosis and thrombophlebitis are erased.

Clinical presentation and diagnosis. The most common localization of deep vein thrombosis of the lower extremities is the leg veins. The clinical picture is often erased. General with standing patients remains satisfactory, marked insignificant Tel'nykh pain in the calf muscles, aggravated by movement, slight swelling lower third of the leg, gastrocnemius muscle tenderness to palpation. Pathognomonic signs of disease are pain in the calf muscles of the foot dorsiflexion (romansa sign) or during compression the middle third of tibia mana zhetkoy mercury sphygmomanometer, in which air is injected slowly. While in healthy

people, an increase in pressure in the cuff up to 150-180 mm Hg. Art. does not cause any pain, patients with deep vein thrombosis begin to experience sharp pain in the calf muscles even with a slight increase in pressure.

The clinical picture becomes pronounced when all three paired deep veins of the leg are thrombosed. This is accompanied by sharp pain, a feeling of fullness, tension, swelling of the lower leg, often combined with cyanosis of the skin. The body temperature rises.

With ascending thrombosis spreading to the femoral vein, femoral edema appears, which is never significant unless the orifice of the deep femoral vein is blocked, which has a rich network of anastomoses with the branches of the femoral vein. Palpation along the thrombosed vein is painful. When combined thrombosis of femoral and popliteal veins sometimes have swelling, pain, limitation of motion zheny in the knee joint. The spread process on a proximal segment of the femoral vein (above the mouth of the deep femoral vein) with the accompanied by an increase in volume of the entire affected limb, increased pain, cyanosis of the skin.

With ileofemoral thrombosis, patients are worried about pain along the anterior-inner surface of the thigh, in the calf muscles, and sometimes in the groin. Finiteness increases in volume, swelling propagation tends from the groin to the foot, sometimes proceeds to buttock. The color of the limb varies from pale to cyanotic. When Pal patsii determined tenderness along the main veins on the thigh and the groin. After 3-4 days from the onset of the disease, the edema decreases slightly and an enhanced pattern of cutaneous veins appears.

Sometimes the disease begins suddenly with acute throbbing pain in the limb, coldness and numbness, as in arterial embolism. Swelling increases rapidly, the movement of the toes becomes limited, reduced temperature sensitivity and cutaneous distal limb segments weakens or disappears PERIPHERAL -parameter pulsation. This form of ileofemoral thrombosis is called "pseudoembolic" or white painful phlebitis (phlegmasia alba dolens). It is based on a combination of deep vein thrombosis with expression -adjoint arterial spasm.

With widespread thrombosis of the deep veins of the lower finite Nosta and pelvic limb dramatically increases in volume, it becomes edematous, thick. The skin becomes dark blue or almost black in color. Blisters with serous or hemorrhagic fluid appear on it - blue painful phlebitis (phlegmasia coerulea dolens). Characterized by severe pain, tearing, no pulsation periphery iCal arteries. In severe cases shock, venous gan Gren limbs.

Ascending thrombosis of the inferior vena cava is a complication of thrombosis of the main pelvic veins. Edema and cyanosis grab healthy finite Nosta and spread to the lower half of the body. Pain in the lumbar and hypogastric regions is accompanied by protective tension of the muscles of the anterior abdominal wall.

Diagnosis of acute thrombosis of the main veins of the lower finite Nosta based on data from clinical disease. The most simple and safe method of detection is phlebothrombosis radioindikatsiya via fibrinogen labeled with a radio active isotope 125I, having a long period poluras pad (60 days). For intravenous administration of the drug it nakapli INDICATES in thrombi, after which the radioactivity was measured in thrombus locations along the deep veins of the leg and thigh. Povy shenie radioactivity in the study areas indicates the presence of blood clots in the lumen of the vein.

Treatment. Surgical treatment is a radical method for acute thrombosis of the great veins: thrombectomy performed with a Fogerty catheter allows to restore blood flow in the vessels, preserve their valve apparatus and thereby prevent the development of post-thrombophlebitic syndrome.

Thrombectomy is shown at floating thrombus, venous thrombosis ileofemoralnom bulky when blue painful phlebitis, thrombosis embologenic, in segmental femoral thrombosis or under vzdoshnoy vein thrombosis distributing on the inferior vena cava. Radical thrombectomy is feasible only in the early stages of bolevaniya when thrombotic mass fixed to the intima of the vessel unstable. Operations are possible only on veins of medium and large diameter (popliteal, femoral, iliac, inferior vena cava). Retrograde removal of a thrombus from the left iliac veins through the phlebotomy opening in the femoral vein is not always feasible due to compression

by the right iliac artery, the presence of intravascular septa and adhesions in the lumen of the common iliac vein. Thrombectomy from the right iliac veins is associated with the risk of pulmonary embolism. Thrombosis of the great veins is often ascending. It originates in the veins of the lower leg, from which the removal of blood clots is impracticable. Therefore, after thrombectomy from large veins, early postoperative retrombosis often develops. Bypass surgery is not widespread due to the complexity of their implementation and frequent thrombosis.

Conservative treatment of acute deep vein thrombosis of the lower extremities is similar to that carried out for arterial thrombosis (see "Diseases of peripheral arteries. Thrombosis and embolism"). The inclusion of pathogenetic thrombolytic drugs in drug therapy is justified. Primary thrombolytic therapy is particularly indicated for the uplink vein thrombosis tibia with parted wound on the thigh (due to the low efficiency thrombectomy) and at high risk of surgery (e.g., open myocardial infarction). The effectiveness of thrombolysis is greatest in the early stages of the disease.

Prevention. Prevention of thrombosis Glu bokih veins is of great importance, because it saves patients from such severe complications of the disease such as pulmonary embolism, post-trombophlebitic syndrome. The need for prevention of thrombosis is especially great in the elderly, in patients with cancer and severe diseases of the cardiovascular system; with obesity; after surgery, CCA cially gynecological, cancer and trauma. AUC zannomu contingent patients should be administered drugs that improve the flow properties of blood and microcirculation (reopoligljukin, komplamin, xantinol nicotinate, etc.), Inhibiting the adhesive-platelet aggregation function (Trental, acetylsalicylic acid, Curantylum) reducing koagulyatsionnyi of blood potential of Nonspecific thrombosis prophylaxis comprises bandaging limbs elastic bandages, electrical incentives lation lower leg muscles, a complex of gymnastic uprazh neny improving venous outflow, early ambulation in postoperative diet period, timely correction of water-electrolyte imbalance, eliminating anemia, a correction of cardiovascular and respiratory disorders.

PRACTICAL PART

THEMATIC TESTS

1

1. The small saphenous (hidden) vein is usually located:
   a) in the subcutaneous fat in the lower half of the leg and between the leaves of the deep fascia in the upper
   b) in the subcutaneous tissue throughout the posterior surface of the lower leg
   c) between the leaves of the deep fascia throughout
   d) subfascial
   e) between the leaves of the deep fascia in the lower half and subfascial in the upper
2. When assessing the patency of deep veins, a functional test is used:
   a) Mayo-Pretta
   b) Talman
   c) Sheinis
   d) Hackenbruch
   e) Brody-Troyanov-Trendelenburg
3. For phlebothrombosis of the ilio-femoral segment is not typical
   a) hyperemia of the thigh skin in the area of vein passage
   b) swelling of the foot and lower leg
   c) bursting pain in the hip

d) an increase in the volume of the thigh and lower leg
e) cyanotic skin of the thigh

4. The most dangerous complication of deep vein thrombosis is:
a) pulmonary embolism
b) trophic leg ulcer
c) thrombophlebitis of superficial veins
d) deep vein obliteration
e) elephantiasis

5. The spread of phlebothrombosis in the veins of the lower extremities is prevented by:
a) all of the above
b) strict bed rest
c) anticoagulant therapy
d) antiplatelet therapy
e) elastic bandage

6. The most common cause of thromboembolism is thrombophlebitis:
a) Deep veins of the lower extremities and veins of the small pelvis
b) Facial veins
c) Deep veins of the upper limbs
d) Superficial veins of the lower extremities
e) Superficial veins of the upper extremities

7. What are the most common symptoms for acute thrombophlebitis of the femoral vein
a) answers b , c , e
b) sharp swelling of the lower limb
c) soreness along the hunter's canal
d) necrosis of toes
e) distention of the superficial veins on the thigh in the basin of the great saphenous vein

8. The most typical symptoms of post-thrombophlebitis syndrome are:
a) All of the above
b) Pain and swelling of the lower limb
c) Secondary varicose veins on the lower leg and thigh
d) Induction of subcutaneous tissue
e) Dermatitis and lesions on the lower leg

9. With ileofemoral venous thrombosis in a pregnant woman, it is necessary:
a) be hospitalized in the vascular department
b) hospitalized in a maternity hospital
c) carry out conservative therapy at home
d) determine the nature of treatment after determining the prothrombin index on an outpatient basis
e) send the patient to the antenatal clinic

10. For phlebothrombosis of subclavian-axillary venous thrombosis is not typical
a) reduction of pulsation on the radial artery
b) fatigue in the arm when working
c) hand swelling
d) cyanosis of the skin of the forearm and shoulder
e) bursting pain in the limb

11. The modern method for diagnosing thrombosis of the inferior vena cava is
a) radiolabelled fibrinogen
b) retrograde iliocavagraphy
c) distal ascending functional phlebography
d) sphygmography
e) retrograde femoral phlebography

Tasks

1. A patient, 30 years old, was hospitalized with complaints of pain in the right lower limb, intermittent claudication. Pain constant yannye. At times, marked fever, swelling, hyper mia, seals, and tenderness along the veins. There is edema of the right lower limb. Starting from the foot to the lower third of the leg, to Ms bluish-pink diet. Thrombosed veins are palpated. Ripple artery pas foot missing, and popliteal and troubles rennoy clear.

Your diagnosis, to confirm what has been said any additional nye studies should be carried out and the treatment regimen?

... A 35-year-old female patient was hospitalized with severe pain and a bursting left lower leg, edema of the left lower limb. The process started sharply and accompanied by the high temperatures. The left lower limb compared with the right led away Chen twice, there is soreness and the calf muscles. The movements are painful.

What diagnosis do you suggest, what research is needed and the treatment plan?

... A 42-year-old patient is diagnosed with acute deep vein thrombophlebitis of the left leg. Complex conservative treatment is used in combination with anticoagulants.

What anticoagulants can be used, the right to appoint Nia and control them during the treatment period?

4. The patient, 30 years old, diagnosed with acute thrombophlebitis hypodermis GOVERNMENTAL veins right shin was appointed heparin. A few days later, during the bypass, signs were revealed that indicate an overdose of anticoagulants.

What signs indicate this and how should the treatment be structured in this observation?

5. The patient, 45 years old, was admitted to hospital with complaints of feeling aches, especially in the evenings and a slight swelling of the left foot and leg, 5 years ago, suffered acute thrombophlebitis deep into the FIR veins of the left lower leg. Conservative treatment was carried out and was discharged from the hospital with improvement. Left shin compared to. Right somewhat enlarged veins are not dilated and the skin without of Change. Muscles are painful on palpation, symptoms of Opitz-Rami-pesa and Homans are positive.

What diagnosis do you suggest, what tests should be carried out to confirm and your treatment tactics?

6. The patient, 40 years old, with a degree in the bus conductor, was admitted with complaints of pain along the veins of the left lower leg, making it difficult to Niya walk and fever. She suffers from varicose veins of the lower extremities for several years. The latter are not Delhi appeared above complaints. On examination, varicose veins of the lower extremities of the main type are noted. On the left lower leg, the varicose veins are swollen and sharply painful on palpation. The skin over them is hyperemic.

In this case, what complication of varicose veins of the lower extremities has developed, the plan of examination and treatment?

7. The patient has ascending ileofemoral thrombosis. Your tactics?

8. A patient with ileofemoral thrombosis has difficulty breathing, cyanosis, hemoptysis. What is this complication? Prescribe treatment

9. Thrombosis of the common iliac vein. The operation did not completely remove the clot. What should I do?

10. There is a suspicion of deep vein thrombosis of the leg. How to confirm the diagnosis?

TOPIC: POST-THROMBOPHLEBITIS SYNDROME

Purpose of the lesson: To study the etiology, diagnosis and differential diagnosis, research methods and treatment tactics for post-thrombophlebitic syndrome.

What the student SHOULD KNOW:

1. The causes of post-thrombophlebitic syndrome.
2. Clinic of post-thrombophlebitic syndrome
3. Diagnostics of the post-thrombophlebitic syndrome
4. Conduct differential diagnosis of peripheral venous diseases.
5. Modern instrumental methods of vein examination.
6. Conservative and surgical methods for the treatment of post-thrombophlebitic syndrome.

Having studied the topic, the student MUST BE ABLE TO:

1. Correctly conduct a survey of patients with post-thrombophlebitic syndrome.
2. Conduct an objective examination of patients.
3. Palpation of peripheral veins.
4. Analyze the results of objective and laboratory examination methods.
5. Technique of elastic leg bandaging.
6. Appoint an additional plan of examination of patients.
7. Based on the data obtained about the patient, formulate a clinical diagnosis.
8. Conduct differential diagnostics.
9. Determine s tactics and the amount of therapeutic measures.

THEORETICAL PART

Post-thrombophlebitic syndrome (post- *thrombotic disease; chronic venous insufficiency)*

Post-thrombophlebitic syndrome (PTFS) is a symptom complex that arises as a result of postponed thrombosis of the main veins of the lower extremities, manifested by venous insufficiency.

Etiology and pathogenesis The immediate cause of this disease is various deep vein thrombophlebitis of the lower extremities. In connection with insufficient recanalization after resorption of thrombi, sclerosing of the inflamed vessel wall, ineffectiveness of the valve apparatus, patients develop various variants of lymphovenous insufficiency, up to the development of trophic ulcers.

Clinic of post-thrombotic disease. Depending on the prevalence of the clinical picture of venous insufficiency, the following forms of PTFS are distinguished:

1. Painful;
2. Varicose veins (secondary varicose veins);
3. Edematous-necrotic (ulcerative);
4. Mixed

The painful form of PTFS is characterized by a feeling of heaviness and bursting pains in the ankle, sole or calf muscles, especially with prolonged standing and hard work.

Performance decreases, fatigue appears.

By the end of the day, swelling usually appears in the feet and ankles. After a night's rest, the swelling disappears or decreases significantly. Often at night there are cramps in the calf muscles. An elevated position, combined with light foot movements, will usually resolve the cramps.

The varicose form occurs in about 70% of patients with PTFS. The expansion of superficial veins can be predominantly of the main type or loose. Saccular enlargement is most often formed in the area of perforating veins that have valvular insufficiency. These veins are palpable as rounded fascia defects.

The varicose form is almost always accompanied by swelling of the limb (lymphovenous stasis).

The ulcerative form of PTFS is characterized by constant indurative edema of the tissues of the lower third of the lower leg and foot. The skin and subcutaneous tissue become dense, painful. Severe pigmentation, weeping eczema in the area of the inner ankle and the antero-inner surface of the lower third of the lower leg. Over time, skin necrosis occurs in this area, trophic ulcers are formed, which have a flat bottom, covered with necrotic tissues and flaccid granulations. As a rule, a putrefactive infection develops in such ulcers, therefore the discharge is dirty gray in color, an unpleasant or fetid odor.

Diagnostics

1. A history of acute deep vein thrombophlebitis; fracture of the limb bones, accompanied by persistent edema of the lower leg.

2. Marching test.

3. Doppler ultrasonography.

4. Phlebography, phleboscopy.

5. Thermography.

6. Rheography.

PTFS treatment

By about n c e p in atm and in yet e l e h e n s

1. Improving the rheological properties of blood

2. Anti-inflammatory drugs

3. Antihistamines

4. Curiosin

5. Suction massage

6. Exercise therapy

7. Zinc-gelatin dressing (Unna dressing). It is applied for 12 ... 14 days 3 … 4 times. Correctly applied (in the morning, after sleeping with a raised limb) zinc-gelatin dressing creates an elastic frame.

During walking, the function of the lymphovenous pump improves, the gradient of venous pressure between the distal and proximal vessels increases, which helps to restore blood flow in extraorgan deep veins, and to form new valves.

Bandages are usually applied to the lower leg and even in the presence of ulcers. When changing the dressing, the skin is treated, massage and exercise therapy are performed within 3-4 days. 8. Wearing elastic stockings.

Operative treatment

Indications

- varicose form of PTFS with incompetent commutative veins;
- PTFS with trophic disorders on their limbs.

Surgical intervention for PTFS is undertaken after the elimination of the acute phenomena of deep vein thrombophlebitis and the completion of the recanalization process. The principle of the operation is to improve blood flow in the affected limb.

1. With a predominantly varicose type of PTFS with sufficient patency of deep veins, phlebectomy of superficial varicose veins is performed in combination with subfascial ligation of perforating veins according to Linton (Fig. 12.4) or suprafascial ligation of perforating veins according to Cockett.

Subfascial ligation of the perforating veins of the lower leg - Linton's operation.

2. In case of unilateral lesion with occlusion of the iliac veins, the Palma operation is performed: suprapubic saphenofemoral bypass surgery using a vein of a healthy leg.

3. Restoration of the function of the valves of the recanalized deep vein.

4. In case of edematous-necrotic form of PTFS, dermal plasty is used, the most effective is dermal-muscular plasty on the vascular pedicle with complete excision of trophic ulcers and sclerosed tissues.

PRACTICAL PART
Thematic tests

1

1. Specify the most dangerous complication of deep vein thrombosis of the lower extremities:
a) trophic ulcer of the leg;
* b) pulmonary embolism;
c) phlebitis of varicose veins;
d) obliteration of deep veins;
e) elephantiasis

2. Indicate the factors that do not contribute to the improvement of venous blood flow in the lower extremities in the postoperative period:
a) early getting up;
b) elastic compression of the legs;
c) elevated position of the limbs;
* d) long and strict bed rest;
e) contraction of the calf muscles.

3. A young woman on a background of uncomplicated pregnancy proceeds STI appeared varicose veins on the right leg. Hirur by God revealed that the deep veins of the right shin passable, having etsya insufficient perforating veins in the lower third. When Pal patsii veins soft, painless, skin over them is not changed. Based on this clinical situation, the patient should be recommended

a) planned surgical treatment before childbirth
* b) wearing elastic bandages, radical phlebectomy in the postpartum period
c) sclerotherapy sessions before - and after childbirth
d) Troyanov-Trendelenburg operation before childbirth, radical phlebectomy after childbirth
e) treatment with nonspecific anti-inflammatory drugs (butadion, troxevasin, escuzan)

4. The development of pronounced edema of one limb is observed in:
a) varicose veins of the lower limbs;
* b) acute deep vein thrombosis of the lower extremities;
c) obliterating atherosclerosis of the vessels of the lower extremities;
d) arterial thrombosis;
e) heart failure.

5. Varicose veins of the lower extremities are characterized by: 1) expansion of the superficial veins of the lower extremities; 2) heaviness in the legs; 3) symptom of intermittent claudication; 4) the formation of trophic ulcers on the toes; 5) chilliness of the limbs. Choose the correct combination of answers:
a) 2, 5;
b) 2, 3, 4;
*at 12;
d) everything is correct;
e) everything is wrong.

6. Chronic venous insufficiency is characterized by: 1) cold extremity; 2) heaviness in the legs; 3) intermittent claudication; 4) limb edema; 5) lack of pulse in the arteries of the dorsum of the foot. Choose the correct combination of answers:
a) everything is correct;
b) 1, 2, 3;
c) 2, 4, 5;
* d) 2, 4;
e) 4, 5.

7. Acute deep vein thrombosis is characterized by: 1) pain in the affected limb; 2) cyanosis of the skin; 3) hyperemia along the saphenous vein; 4) sharp swelling of the limb; 5) intermittent claudication. Choose the correct combination of answers:
* a) 1, 2, 4;
b) 1, 2, 3;
c) 2, 3, 5;
d) 3, 4, 5;
e) everything is correct.

8. Name the symptoms of acute thrombosis of the ilio-femoral venous segment: 1) hyperemia of the skin of the thigh in the area of veins; 2) swelling of the entire limb; 3) bursting pain in the hip; 4) an increase in the volume of the thigh and lower leg; 5) pallor of the skin of the thigh. Choose the correct combination of answers:
a) 1, 2;
b) 2;
* c) 2, 3, 4;
d) 3, 4;
e) 3, 4, 5.

9. Name the most modern, most informative method used for topical diagnosis of inferior vena cava thrombosis:
a) radioindication with labeled fibrinogen;
* b) ileocavagraphy;
c) distal ascending functional phlebography;
d) sphygmography;
e) retrograde femoral phlebography.

10. The development of thrombophlebitis is promoted by: 1) slowing down the blood flow; 2) hypocoagulation; 3) hypercoagudation; 4) increased fibrinolytic activity of the blood; 5) damage to the intima of blood vessels. Choose the correct combination of answers:
a) 2, 4;
* b) 1, 3, 5;
c) 3, 4, 5;
d) 4, 5;
e) everything is correct.

11. Chronic venous insufficiency can lead to: 1) gangrene of the limb; 2) the development of trophic ulcers; 3) varicose veins; 4) flat feet; 5) hallux valgus. Choose the correct combination of answers:
a) 1;
b) 4, 5;
c) 2, 4, 5;
* d) 2, 3;
e) 3, 4, 5.

12. Acute violation of regional venous circulation develops in: 1) thrombophlebitis of superficial veins; 2) deep vein thrombophlebitis; 3) embolism; 4) varicose veins; 5) traumatic deep vein injury. Choose the correct combination of answers:
a) 1, 3;
b) 3, 4, 5;
* c) 2, 5;
d) 4, 5;
e) everything is true.

13. List the complications of primary varicose veins of the lower extremities: 1) acute thrombophlebitis; 2) bleeding from varicose veins; 3) trophic ulcer of the leg; 4) pulmonary embolism, 5) elephantiasis. Choose the correct combination of answers:
* a 1, 2; b) 2, 4 c) 1, 3, 4, 5 d) 2, 3, 5; D) everything is correct.

14. The main symptoms of subclavian - axillary venous thrombosis include: 1) chronic fatigue in the arm during work; 2) hand swelling; 3) cyanosis of the skin of the forearm and shoulder; 4) bursting pain in the limb; 5) arterial hypertension on the affected side. Choose the correct combination of answers:
a) 1, 2, 5;
b) 1, 2, 3;
c) 1, 4, 5;
* d) 2, 3, 4;

e) everything is correct.

15. Give the main clinical symptoms of massive pulmonary embolism: 1) chest pain; 2) collapse; 3) choking; 4) cyanosis of the face and upper half of the body; 5) swelling and pulsation of the jugular veins; 6) emphasis of the second tone on the pulmonary artery. Choose the right combination of arteries:
a) 1, 2, 5;
b) 1, 3, 4.6;
c) 2, 3, 5, 6;
d) 3, 4, 6;
* e) everything is correct.

16. What characterizes post-thrombophlebitis syndrome of the lower extremities? 1) skin pigmentation; 2) congestive dermatosis and sclerosis; 3) skin atrophy; 4) pale "marbled" skin; 5) secondary varicose veins of superficial veins. Choose the correct combination of answers:
a) 2, 3;
b) 4, 5;
c) 1, 2, 4;
* d) 1, 2, 5;
e) 2, 3, 4, 5.

THEME: OCCLUSIONAL DISEASES OF THE ARCH BRANCHES. SYNDROME TAKAYASU.

The purpose of the lecture : To acquaint students with occlusive diseases of the aortic arch, the causes leading to them, to give an idea of acute arterial thrombosis and embolism, clinical manifestations, with optimal methods of diagnosis and treatment, management of the postoperative period.

Educational purposes of the lecture : Students should know about occlusive diseases of the branches of the aortic arch, that the causes of acute cerebrovascular accidents are mainly these lesions. Students should know about embologic diseases, thrombotic patients, remember that in 80-95% of cases, the causes of arterial thrombosis are heart disease. Untimely diagnosis of these diseases is the cause of mutilation and death of patients.

Lecture objectives:

1. Give the concept of occlusive lesions of the branches of the aortic arch
2. Explain the reasons for the development of occlusive lesions of the branches of the aortic arch
3. Explain the clinical manifestations of the disease
4. Conduct differential diagnosis with other diseases
5. To acquaint students with the most modern and informative methods of examination and treatment of patients
6. Demonstration of examples from surgical practice: patients, angiograms, slides

Lecture plan:

1. Background - 5 min
2. The concept of occlusive diseases of the branches of the aortic arch - 5 min
3. Clinic - 15 min
4. Diagnostics and treatment of occlusive diseases of the branches of the aortic arch - 20 min
5. Differential diagnosis - 15 min
6. Treatment - 15 min
7. Complications and their prevention - 15 min

OCCLUSIONAL LESIONS OF THE AORTIC ARCH BRANCHES

One of the most important problems in medicine is the treatment of cerebrovascular accidents. This is due to the very high prevalence of cerebrovascular diseases, in the structure of which ischemic circulatory disorders, caused by atherosclerosis, take the leading place.

Mortality from ischemic stroke in all economically developed countries ranges from 12 to 20% of the total mortality, second only to mortality from heart disease and tumors of all localizations. In the United States, stroke is the third leading cause of death in adults. Patients with acute stroke make up 5% of emergency hospital beds in the United States. Among the white population in the United States, stroke mortality is 31 per 100 thousand men and 27 per 100 thousand women. It is interesting to note that the mortality rate from stroke among the black population in the United States is higher than among the white population, namely 59 per 100 thousand men and 48 per 100 thousand women, but mainly due to untreated hypertension. At the same time, the black population of the United States has a significantly lower incidence of stroke due to damage to the extracranial segments of the carotid arteries.

Ischemic stroke is also a leading cause of disability, causing enormous economic damage. In the United States alone, $ 20 million is spent on stroke from the health budget. From 40 to 60% of patients after ischemic stroke become disabled, persistent residual changes are observed in 30% of patients, and working capacity is restored in 10% of patients. The incidence of stroke increases markedly with age, doubling every 10 years of life from 45 to 85 years and rising to 1,440 per 100 thousand of the population between 75 and 84 years of age. The severity of stroke also increases over the years: among patients over 65 with stroke, only 35% could be independent in five daily functions. About 50% remained totally dependent on those around them.

Therefore, the problem of prevention and treatment of disorders of cerebral circulation, being a serious medical problem, is of great socio - economic importance, especially since the growth rate of mortality from ischemic stroke is the highest in patients aged 30 to 50 years.

The devastating consequences of thrombotic / ischemic stroke have influenced the course of history. World leaders are exposed to disease in the same way as ordinary people. James F. Tole, in his book on cerebrovascular disease, wrote:

"In February 1945, before the end of World War II, Franklin Delano Roosevelt, Winston Churchill and Joseph Stalin gathered in Yalta to conclude a treaty on the post-war division of the world. The painful effects of this treaty have remained to this day and, to one degree or another, have played a role in the conflicts in Korea. , Vietnam, Czechoslovakia and Poland. At the same time, the leaders of the great powers, who gathered in Yalta, suffered from cerebrovascular diseases. So, F. Roosevelt died 3 months later from massive cerebral hemorrhage. J. Stalin later died from the same cause. Churchill had a series of minor strokes that later led to dementia.

Roosevelt apparently suffered several cerebrovascular episodes during his second term as president. As for Yalta in 1945, his condition was dire. Lord Moran, the physician of W. Churchill, describes Roosevelt's condition as follows:

"He had a drape or shawl that covered his shoulders, which made him look like a shrike bird. He sat looking straight ahead with his mouth open as if he had something there. Everyone was shocked by his sight and seemed to agree with it. that the President is heading for physical decrepitude. It was not only his physical destruction that gave this impression. He took very little part in the discussions. When in the past he was sometimes not sufficiently aware of the subject of discussion, he was rescued by extraordinary insight. Now they say that his insight is gone and never will not come back".

Woodrow Wilson, President of the United States during the First World War, suffered a stroke during a re-election campaign and was declared disabled for almost two years. He practically did not lead his Cabinet, and Mrs. Wilson was the intermediary between the government and the President, who eventually became involved in the movement of the President's administration. It was at this critical time that the creation of the League of Nations was completed. Wilson, as an active advocate of this organization and seeing it as the main obstacle to preventing future wars, was unable to convince the United States Congress of the value of this organization.

Of the fifteen US Presidents since 1900, from Theodore Roosevelt to Ronald Reagan, thirteen have died of stroke or coronary artery disease, excluding Herbert Hoover and John F. Kennedy.

Although a number of diagnostic details of the death of Vladimir Lenin are unknown, the most common opinion is that it happened from cerebrovascular disease.

So, behavioral changes as a consequence of cerebrovascular disease are likely widespread. This affects not only our daily life, but also international affairs. In many cases, this remains unknown to most of us.

The main cause of ischemic disorders of cerebral circulation is atherosclerosis of the vessels of the brain, which is confirmed by a large statistical material.

Among all the causes of ischemic cerebral stroke, extracranial lesions (stenosis and thrombosis) of the internal carotid artery (ICA) account for at least 40%. In one of the American studies of 1000 patients with new-onset stroke, it was shown that 89% of patients had cerebral infarctions and 68% of them had a stroke in the carotid artery basin. Among patients with infarction in the carotid system, 34% had stenosis of more than 50% of the ipsilateral internal carotid artery (ICA). Thus, in 206 out of 1000 cases, heart attacks had a cause that could theoretically be prevented by carotid endarterectomy (EAE).

The dependence of the functions of the brain on the state of the carotid arteries has long been known to mankind. Garrison cites Hippocrates, who probably gave the first description of what can now be called a transient ischemic attack: "Unusual attacks of numbness and anesthesia are signs of impending apoplexy." Hippocrates also described paralysis and sensory disturbances in the limbs on the side opposite to the brain injury; much later (in the 7th century) this description was given by Galen (12). The Greeks knew the carotid artery as "the artery of asphyxia and deep sleep." One of the marble sculptures brought from the Parthenon in Athens to the London Albert and Victoria Museum depicts a centaur compressing the carotid artery of a warrior.

One of the exhibits of the Hunter Museum of the Royal College of Surgeons (England) is a preparation of the carotid artery with ulcerative atheromatosis, which was identified by J.Hunter as "ossification" (ossification) about 200 years ago. The term "atherosclerosis" was not known. Early surgical procedures on the carotid arteries were limited to bandaging to stop bleeding during trauma, as described in 1803 by Fleming and in 1805 by Sir A. Cooper. However, even then there were timid assumptions about a possible connection between the lesion of the carotid artery and cerebral symptoms, which was stated in isolated clinical reports in France, Germany, England and the United States.

In England, in 1856, a clinical observation of a young woman with neurological and visual impairments with a weak pulsation of the right carotid artery and the presence of a systolic murmur above it was published. Autopsy revealed stenosis and thinning of the wall of the right common carotid artery on the right side; the right external carotid artery was occluded with clinical consequences in the form of necrosis on the scalp and skull in the basin of this artery.

Perhaps the first clinical description of thrombosis of the internal carotid and vertebral arteries was provided by Penzoldt of Germany in 1881. Chiari in 1905, during a postmortem examination of a patient with cerebral embolism and, finding

no other paths for embolism, dissected the carotid artery along its entire length and found an ulcer with thrombosis in the bifurcation of the carotid artery. Even then, he strongly advised examining the carotid arteries in patients with "apoplectic stroke". Nine years later, in 1914, the New York internist R. Hunt, on the basis of a postmortem examination, found a connection between occlusive lesions of the arteries of the lower extremities and the carotid arteries, and thus, for the first time, indicated to clinicians the systemic nature and prevalence of atherosclerosis. He recommended an examination of the carotid arteries as mandatory in postmortem examination. For patients with neurologic signs and symptoms of vascular involvement, Hunt wrote, physicians should take "the same stance as for groups of patients with intermittent claudication, gangrene and other vascular symptoms ..." In two fundamental studies in 1951 and 1954, C. Miller Fisher once and for all proved the connection between occlusive disease of the cervical carotid arteries and, as he wrote, "the main causes of stroke." In his extensive studies of the carotid arteries in the neck, he identified 45 cases of total or subtotal obstruction of one or both carotid arteries among 432 autopsies. In 4 patients with proven cerebral embolism, atheromatous material was found in the carotid bifurcation. He also recommended compulsory examination of the cervical carotid arteries.

Following the work of Fisher, a whole series of studies appeared very quickly, confirming the close relationship of atherosclerosis of the cervical carotid arteries with cerebral atherosclerotic infarction. At the Mayo Clinic, Milliken and Seikert described carotid and vertebral insufficiency syndromes. In London, Jates and Hatchinson noted that nearly all patients suffering from cerebral ischemia had occlusive lesions of the extracranial arteries at autopsy. Hollenhorst drew attention to the bright cholesterol emboli observed by him in the retinal arterioles in a number of patients with atherosclerotic lesions of the carotid arteries.

In connection with these works, a vigorous debate has arisen about the ways and possibilities of differentiation between cerebral symptoms caused by carotid artery obstruction, true ischemia and symptoms determined by embolic occlusion of intracranial arteries or arterioles with transfer of emboli from atheromatous changes in the cervical arteries. Many clinicians found it difficult to accept the thesis that focal transient signs and symptoms of intracranial ischemia are associated with simple narrowing of the proximal vessel. Not all clinicians admitted that recurrent neurological symptoms may be the result of embolism in the basin of the same cerebral territory. The debate continued until the 1960s. Only the accumulation of significant experience in surgical treatment has made it possible to clarify the relative role of these two mechanisms in the development of cerebrovascular accidents. Research by Julian, Moore, and Imparato has largely resolved this problem. In the first two studies, clinical and morphological comparisons have convincingly proved a clear relationship between ulcerated atheroma in the cervical carotid arteries and clinical symptoms of transient ischemic attacks. The third work revealed a correlation between the development of intra-plaque hemorrhages with cerebrovascular symptoms. Continuing these studies, Lusby et al. Showed that not only intra-plaque hemorrhage, but also

erosion on its surface, atheromatosis of the arterial walls are sources of embolism and the cause of subsequent recurrent neurological symptoms.

One cannot fail to note the great contribution of domestic neuropathologists to this problem, and especially E.V. Schmidt and the school.

Pathomorphological studies were also of great importance, showing that isolated extracranial lesions of the carotid arteries are not uncommon, but a rule, and occur much more often intracranial and combined extra-intracranial lesions. The widespread occurrence of this type of ICA lesion in the general structure of atherosclerotic lesions of the cerebral vessels, the relatively easy access to the arteries of the neck and the relationship of extracranial lesions of the carotid arteries with the clinical manifestations of cerebrovascular insufficiency immediately attracted the attention of vascular surgeons. Nevertheless, the development of the surgical treatment of carotid insufficiency was rather slow, gradually approaching the volume of operations on the aortoiliac and femoropopliteal segments. The beginning of the carotid artery surgery consisted of bandaging it for trauma or cervical tumors. It should be noted that in the era of Leriche, the French surgical school was not limited only to ligation of the carotid arteries, but performed resection of the stellate ganglion and cervical sympathectomy to increase cerebral blood flow. To block repeated cerebral embolisms, silver clips were used to clip the intracranial portion of the internal carotid artery. In 1918, the French surgeon le Fevre reported an anastomosis between the branches of the external carotid artery and the distal portion of the internal carotid artery in order to restore intracranial blood flow after resection of a tumor in the neck. Conley from New York repeated this operation forty years later, but used the vena safena, or superficial femoral vein, for prosthetics after tumor removal.

Carrea, Molins and Murphy, respectively neurosurgeon, vascular surgeon and general practitioner in 1951 in Buenos Aires, performed the first successful reconstruction of the common and internal carotid artery after resection of the stenotic site in the bifurcation, while also performing cervical sympathectomy. This 41-year-old patient experienced recurrent episodes of aphasia, right-sided hemiplegia, and blindness in the left eye. For the next 23 years, he was neurologically healthy. The authors emphasized that the idea for this operation was inspired by the work of Fisher.

The standardization of surgery for carotid artery lesions became possible after the development of the thromboendarterectomy technique proposed by the Portuguese surgeon J.Cid Dos Santos in 1951. Dos Santos suggested performing thromboendarterectomy on the arteries of the lower extremities. In the USA, this operation was used by Wylie from San Francisco for the defeat of the abdominal aorta and iliac arteries.

In 1953, thromboendarterectomy was successfully used for carotid artery disease. This surgeon was M. DeBakey of Houston, Texas, who, however, did not publish the operation until 1975.

Therefore, and in this regard, the priority of the first successful operation is given to Eastcott, Pickering and Rob from London. Their patient was a 66-year-old woman with multiple episodes of transient left-sided blindness, right-sided hemiplegia, and aphasia. Puncture left-sided carotid angiography revealed severe stenosis of the left internal carotid artery. During the operation, to protect the brain from ischemia, a lot of ice and an open window in the operating room (!) Were used, the operation was performed under general anesthesia. The bifurcation of the common carotid artery was resected and an anastomosis was created between the common and internal carotid arteries. Dr. Rob writes that this patient recovered, no longer had transient cerebrovascular accidents, and lived to be 86 years old.

Gradually, carotid endarthrectomy has become a standard procedure. Complications and mortality declined sharply as more experience gained in the technical details of surgery, better patient selection, more careful consideration of the risk of comorbidities, and improvements in the prevention of cerebral ischemia and improved anesthesia.

Over the past 40 years, carotid artery surgery has developed rapidly. Hundreds of thousands of operations on the carotid arteries, tens of thousands of reconstructions of the vertebral and subclavian arteries have been performed. Unfortunately, such impressive figures characterizing the development of this branch of vascular surgery do not in any way apply to our country. The obvious lag in surgery of the brachiocephalic arteries has a number of reasons, including, above all, a low level of functional communication between neuropathologists and vascular surgeons, which, in essence, is the key point in organizing angiosurgical care for this category of patients. We are sure that a significant part of neuropathologists, primarily of the outpatient clinic, are not sufficiently aware of the diagnosis and clinical manifestations of lesions of the brachiocephalic arteries, and, consequently, of the indications for surgical treatment.

An important role in determining the indications for surgical treatment of carotid artery stenosis is played by the precise determination of the mechanism of stroke or TIA development. It can be considered proven that among them the most important is the hemodynamic mechanism of the development of cerebral dysgemia and the mechanism of arterio-arterial embolism.

The development of pronounced stenosis of the carotid artery with a narrowing of the lumen of more than 70-75% or with thrombosis can lead to a decrease in perfusion pressure in the carotid artery distal to the stenosis or occlusion and, as a consequence, to low perfusion in the branches of the ICA - the middle and anterior cerebral arteries. Exposure to extracerebral factors (lowering blood pressure, peripheral vasodilation, etc.), leading even to a short-term

additional decrease in the level of perfusion, can lead to the development of ischemia. Exactly the same developmental mechanism is possible with a sharp stenosis or occlusion of the vertebral artery (PA), only with a decrease in perfusion pressure in the main artery system. The state of the collateral circulation, the coagulation and anticoagulation systems of the blood, and a number of other factors affect the rate of perfusion recovery: at a fast rate, clinical manifestations may be limited to TIA, with slow or incomplete recovery of the perfusion level, ischemic stroke develops.

The detachment of a fragment of an atherosclerotic plaque in the carotid plaque, its movement into the distal vascular bed, most often into one of the branches of the middle cerebral artery with its thrombosis and the development of transient or persistent ischemia underlies the mechanism of arterio-arterial embolism.

The mechanism of arterio-arterial embolism is more universal, since it can be realized with any degree of ICA stenosis, including very small and even so-called. ulcerated plaques (from the Latin ulcera - ulcer), when the lumen of the artery is practically not narrowed, but the existing ulcerative surface can be a source of emboli. On the other hand, in case of carotid artery thrombosis (the more accepted term in angiosurgery is occlusion), the mechanism of arterio-arterial embolism, of course, does not work - this situation is purely hemodynamic.

A number of researchers assert that the mechanism of arterio-arterial embolism can act in patients with PA stenosis, which is proved by single morphological studies. Nevertheless, the peculiar structure of the vertebrobasilar basin (fusion of both PAs into one main artery, branching of the branches of the main artery to the brain stem almost at right angles), much less in comparison with ICA, the role of atherosclerosis in the development of PA lesion puts arterio-arterial embolisms far from first place among the reasons for the development of vertebrobasilar insufficiency (VBI). Most researchers are inclined to believe that the hemodynamic type of development of VBI predominates in case of PA lesion, not only with occlusion of this artery, but also with its stenosis, tortuosity and bends.

Taking these data into account, it is quite obvious that with occlusion of the ICA or PA, hemodynamically significant stenosis or tortuosity of these arteries, the main task of surgical revascularization is to increase blood perfusion into the vascular basin (bypass grafting in cases of occlusion, plaque removal in hemodynamically significant stenoses - endarterectomy "straightening" tortuosity), and in case of hemodynamically insignificant stenosis, but with the potential (or already realized) possibility of the development of arterio-arterial embolism - in the removal of plaque from this artery.

The clinical course of stroke with extracranial lesions of the carotid arteries is well studied. Pathognomonic for him is the development of TIA, transient disorders of the retinal circulation (amaurosus fugax) or contralateral optic-

pyramidal syndrome and minor strokes. At the same time, the proportion of these syndromes in the overall structure of clinical manifestations is no more than 20-25%. It is also well known that TIA is characteristic of lesions of the carotid arteries in the neck, but only in 30% of patients they precede the development of a stroke, and in 70% of patients, a stroke occurs suddenly without any precursors. The VBI clinic practically does not depend on the type of PA lesion (stenosis, tortuosity, extravasal compression). Moreover, other causes of VBI development (arterial hypertension, cervical osteochondrosis) do not have supporting symptoms that differ from the clinical picture of organic lesions of PA.

Thus, in the differential diagnosis of lesions of the carotid and vertebral arteries, instrumental research methods are highlighted, among which Doppler ultrasound diagnostics takes the leading position. In the "pyramid" of Doppler methods of examination, all its components are important and complement each other. The combination of periorbital Doppler ultrasonography with spectral analysis and duplex scanning gives a completely complete picture of the ICA lesion with an accuracy (in relation to angiography) up to 95-96%. The state of intracranial cerebral vessels can be diagnosed using transcranial Doppler ultrasound. Overall, the introduction and improvement of non-invasive testing is a tremendous achievement. Its development is constantly improving for all vascular regions and has made angiography, with its potential risk and high cost, almost unnecessary. Intraoperative monitoring using ultrasound methods is widely used.

Thus, diagnostic problems in occlusive lesions of the carotid arteries can be considered resolved.

The main question that the clinician faces when faced with this pathology is the question of treatment tactics. What determines the solution of this issue? Probably, first of all, on what is the risk of the natural course of occlusive lesions of the brachiocephalic arteries. Secondly, it depends on the effectiveness of surgical treatment. To the fullest extent, these questions are answered by the so-called. cooperative studies, the results of which are based on large, statistically significant, homogeneous clinical material. Currently, several foreign cooperative studies have been completed on the comparative assessment of carotid endarterectomy for ICA stenosis and the natural course of this pathology. The results of these studies (NASCET, ECST, VASCET) unambiguously showed that with hemodynamically significant ICA stenosis more than 70% in the so-called. For "symptomatic" patients (that is, those who have suffered a stroke or TIA), surgical treatment is significantly superior in its effectiveness to the results of drug treatment. Comparative assessment of the results of surgical and drug treatment in patients with hemodynamically insignificant ICA stenosis (i.e. less than 70%) in these cooperative studies is at the stage of accumulation of clinical material, however, according to the literature, indications for carotid EAE in this case depend on the morphological state of atherosclerotic plaque. determined by duplex scanning. In the presence of diagnostic signs of ulceration of the plaque,

hemorrhage under it, that is, with obvious signs of its embolism, carotid EAE is indicated

The question of indications for surgical treatment of patients with ICA occlusion stands somewhat apart. Revascularization of the brain in this pathology has long remained an insoluble problem. The first thrombectomy operations from the ICA in most cases were ineffective or impossible, especially when thrombosis spread to the intracranial sections of the ICA, which subsequently led to the rejection of these operations. Numerous studies have shown that with occlusion of the ICA, the severity of cerebrovascular insufficiency is to a greater extent the result of inadequate collateral circulation than the actual occlusion of the ICA.

In this regard, the creation of an extra-intracranial microanastomosis (EICMA) between the superficial temporal artery (PVA) and the cortical branches of the middle or anterior cerebral arteries (MCA, PMA) with occlusion of the ICA is the most adequate operation aimed at revascularization of the brain and an increase in perfusion pressure (PD) in the basin of the occluded ICA.

The idea of creating EIKMA was first expressed in 1912 by Crutrie and was implemented in 1967 by Donaghy and Yazargil. The introduction of this type of surgical correction into practice was regarded as a significant progress in the treatment of cerebrovascular diseases, since before that, patients with ICA occlusion were considered inoperable and had a high risk of recurrent strokes. So, in the first year after the disease, recurrent strokes occur in 9-12% of cases, and over the next 5 years - in 20-50%, with a mortality rate of 16-40%. At the same time, in the group of operated patients with completed stroke, these indicators were significantly lower.

Already in the first years of using the EIKMA operation, good results were obtained in the treatment of patients with TIA.

In a number of studies, it was also noted that EIKMA led, in addition to a pronounced preventive effect, to a regression of neurological symptoms in patients with mild and moderate neurological deficits after a completed stroke.

Against the background of the widespread use of the operation to create an EICMA with a good hemodynamic and clinical effect in occlusive lesions of the ICA, the results and conclusions of the International Cooperative Study in 1985 (ICT) on the study of the effectiveness of EICMA, initiated in 1977, were completely unexpected . came the American National Institute of Neurological Diseases. The MCI results showed that EICMA is no more effective than conservative treatment for reducing the incidence of stroke or death from it, does not affect clinical results, and therefore is not justified for revascularization in patients with atherosclerotic lesions of the cerebral vessels. In accordance with the recommendations of the ICI, the number of EICMA operations in most clinics around the world has sharply decreased, which significantly limited the possibility of continuing the study of the hemodynamic and clinical effect of EICMA.

The conclusions of the ICI gave rise to a very intense controversy, since they largely contradicted the experience accumulated by neurosurgeons and suffered from a number of methodological errors. A number of surgeons agreed with the findings of the ICI, and many criticized the study. Methodological errors in the selection criteria for patients and indications for surgery were noted. Thus, when selecting groups of patients for analysis, the MCI did not take into account the initial state of collateral compensation, did not study cerebral blood flow, and did not assess the state of the great vessels of the head and cardiac cavities for embolism. Without such consideration, it was impossible to find out the nature of the stroke and determine the indications for the creation of EICMA. Thus, the analysis of the results was given before the development of objective indications for the creation of EICMA, proving only that the presence of occlusion or stenosis of one of the cerebral arteries is not an indisputable indication for the creation of EICMA.

That is why research on EICMA in a number of clinics did not stop, but on the contrary, continued intensively, and at a new level of study of cerebral hemodynamics in occlusion of the ICA before and after surgery, using modern research methods such as transcranial Doppler ultrasonography (TCD), dynamic computer scanning (DCS) , positron emission tomography (PET), radionuclide study of regional cerebral blood flow (RCC). As a result of these studies, the main indications for the creation of EICMA were formulated. They are based on the objectively proven position that EICMA only makes sense if there is a low cerebral perfusion reserve (CPR) in the basin of the occluded ICA, when the collateral circulation into the basin of this artery through the connecting arteries of the Willis circle and other collaterals is so "tense", that the need for additional enhancement of blood flow (for example, with any unfavorable extracerebral factors) is not realized. At the same time, when collateral circulation is well developed, the CRR is high enough to respond with increased blood flow in any unfavorable situation.

How is CPR determined? To determine this important indicator, a situation is clinically modeled in which an increase in blood flow is necessary. A number of researchers use drug tests (diamox, nitroglycerin), others - an activation test with inhalation of 5-6% of a mixture of carbon dioxide with air. Control of changes in blood flow in the middle cerebral artery from the side of ICA occlusion is most often carried out using TCD.

Thus, hemodynamic indications for EIKMA are formulated as follows:

- the state of cerebral hemodynamics in patients with ICA occlusion is a determining factor for hemodynamic indications for the creation of an extra-intracranial anastomosis;
- the creation of EICMA is not indicated with a high perfusion cerebral reserve;
- the creation of EICMA is shown with a low perfusion cerebral reserve, in which the method of mathematical multivariate analysis gives a

confident preoperative forecast of a hemodynamically significant anastomosis;

- when extracranial lesions of the carotid artery (stenosis of the external carotid artery, occlusion or stenosis of the common carotid artery) are combined with occlusion of the ICA, the primary reconstruction of these segments is necessary to ensure normal hemodynamic conditions for EICMA.

At the same time, as our studies have shown, the achievement of significant positive clinical results is possible only with mild neurological deficits (TIA, completed stroke with mild residual events) with a minimum amount of brain tissue damage according to computed tomography. It is only the coincidence of the conditions of hemodynamic and clinical efficacy that determines the overall indications for brain revascularization in chronic ICA occlusion.

Now it seems quite clear that it is equally wrong to completely abandon the use of this operation, as declared in the International Cooperative Study, as well as to widely apply EIKMA, based only on the fact of ICA occlusion, as the only criterion. Only careful selection for hemodynamic and clinical signs is the most correct way to determine the indications for this operation.

Another problem is the subject of growing attention of angiosurgeons - the pathology of the vertebral arteries. The high prevalence of chronic vertebrobasilar insufficiency, especially at the polyclinic level, is well known, which is described by well-known diagnoses - "vertebral artery syndrome", "cervical osteochondrosis". Establishing indications for surgical treatment in pathology of the vertebral arteries is a difficult task. This is due to the fact that very often VBI can be the result of the summation of several reasons, while organic lesions of the PA (stenosis, extravasal compression, tortuosity) can be combined with others (cervical osteochondrosis and other vertebral lesions, arterial hypertension, clinically manifesting VBI syndrome, intracranial hypertension). In addition, if ultrasound Doppler diagnostics is practically unmistakable in case of lesions of the carotid arteries, then in case of PA pathology its accuracy hardly reaches 70%. Therefore, in this case, angiography becomes the main diagnostic method. Finally, studies have shown that the pathology of the vertebral arteries rarely leads to the development of stroke. Thus, the goal of surgical treatment in PA pathology is to achieve, rather, not prophylactic, but rather clinical efficacy. Consequently, the indications for surgical treatment of PA depend on the solution of three problems: firstly, a thorough diagnosis of PA lesion, confirmed by angiography, is necessary, and secondly, it is very important to determine exactly what the PA pathology is the main cause of clinical manifestation, and, third, that VBI is resistant to drug therapy.

Having emerged more than 40 years ago at the intersection of angioneurology, neurosurgery and vascular surgery, surgery of the brachiocephalic arteries has now taken a firm place as one of the most effective methods of treating ischemic disorders of cerebral circulation. The development of this promising direction in

our country depends on how much practical doctors of different specialties, primarily neuropathologists, fully understand the need for a modern solution to this issue.

A number of problems require further research, including the study of the diagnostic value of magnetic resonance angiography, a deeper study of the role of asymptomatic plaques in the risk of stroke, the study of indications for combined reconstructions of the coronary and carotid arteries and the problem of restenosis, as well as the development of intraoperative monitoring for the purpose of diagnosing intraoperative embolism, which plays a major role in the development of complications such as perioperative strokes. Finally, the main problem remains to find ways to prevent and regress atherosclerosis.

QUESTIONS

to the audience to establish feedback and find out that the goal of the lecture has been achieved

I. What is "acute arterial obstruction"?

2. What are the causes of acute arterial obstruction?

3. What is thrombosis?

4. What is embolism? How it differs from thrombosis

5. What is the main cause of arterial embolism?

b. List 3 etiological factors of OAN - Virchow triad

7. Tactics of GPs in diagnosing OAN

8. What operations are performed with OAN?

9. Name the absolute contraindications in OAN to surgery

10. Measures for the prevention of acute arterial thrombosis and embolism

TOPIC: SYMPTOMATIC HYPERTENSION. RENOVASCULAR HYPERTENSION AND I. ETIOLOGY, PATHOGENESIS, DIAGNOSIS AND TREATMENT.

The purpose of the lesson:

1. To study the etiology, pathogenesis, clinic of hypertension.
2. Learn the correct diagnosis according to the modern classification.
3. Be able to make a differential diagnosis.
4. Know the basic principles of treatment.
5. Establish the basic principles of prevention, labor expertise.

Test questions:

1. Etiology of hypertension.

2. The pathogenesis of hypertension.
3. Modern classification of hypertension.
4. Clinic of hypertension.
5. Differential diagnosis.
6. Symptomatic hypertension.
7. Basic principles of hypertension treatment.
8. Clinical characteristics of hypertensive crises.
9. Treatment of hypertension.
10. Hypertensive crisis treatment.
11. The main groups of antihypertensive drugs.
12. Principles of prevention of hypertension.
13. Labor examination for hypertension.

ARTERIAL HYPERTENSION

The term "arterial hypertension" (AH) is increasingly used in the medical literature instead of such names as essential hypertension, essential hypertension (HD) and primary arterial hypertension.

In many countries, hypertension remains the most common disease of the cardiovascular system and among the population of developed countries at the age of 18 - 74 years it is detected in 29.8%. In some regions, the incidence of the disease among men of working age reaches 44%. With age, the number of patients increases. So, among people under 30 years old it is 4-10%, 50-60 years old - 44%, 61-69 even - 54%, over 70 years old - 65%. At the medical department, out of 2000 served residents, hypertension is detected in 100 - 500. Hypertension accounts for 90% of all cases of arterial hypertension.

The development of hypertension is due to many interacting hemodynamic, neurohumoral, metabolic and other factors. Among the main pathogenetic mechanisms of its development and progression, the leading ones are the activation of neurohumoral systems, a decrease in the distensibility of the aorta, endothelial dysfunction, nephrosclerosis, obesity (metabolic syndrome), increased sensitivity to table salt and the use of large amounts of it, chronic stress, genetic predisposition, etc.

The formation of the level of blood pressure (BP) is directly influenced by 3 factors: stroke volume (SV), total peripheral vascular resistance (TPV) and circulating blood volume (CBV): BP = SV + TPV + CBV. Consequently, with a relative constancy of the BCC, the blood pressure level can increase due to an increase in SV and / or TPVR.

In addition to these main factors, other mechanisms are involved in the formation of blood pressure. In particular, the value of systolic blood pressure (SBP) is a derivative of three parameters: SV, maximum blood expulsion rate, elasticity (extensibility) of the aorta. The value of diastolic blood pressure (DBP) is a derivative of two parameters - systemic vascular resistance and heart rate.

According to the nervous theory of the origin of hypertension, developed by G.F. Lang, the central link in the occurrence of hypertension is a change in the tone of smooth muscles of the arterioles of the systemic circulation. Excessive nervous and emotional stress creates foci of stagnant excitement in the cerebral cortex. Involvement in this process of the reticular formation and the vasomotor center of the medulla oblongata leads to depletion of the central vasomotor mechanisms, to their inability to adequately respond to peripheral neurogenic stimuli, which causes an increase in vascular tone and spasm of arterioles. In addition, nervous and emotional stress through the sympathetic nerve centers contribute to the release of large amounts of catecholamines from the adrenal medulla, which also lead to generalized or regional (in the brain, heart and kidneys) vasospasm. An increase in sympathetic influences, in addition, leads to an increase in heart rate and myocardial contractility, which, in turn, causes an increase in stroke volume. The consequences of sympathetic activation also include metabolic disorders (insulin resistance), increased platelet aggregation ability, and acceleration of organ damage.

COMPLICATIONS OF ARTERIAL HYPERTENSION

Hypertension is dangerous because it is one of the main risk factors for the development of cardiovascular diseases of atherosclerotic origin, which are associated with about half of all deaths in the developed countries of the world.

The main vascular complications of hypertension can be divided into 2 groups:

1. Hypertensive, ie directly associated with overloading the cardiovascular system, pressure. These complications include hemorrhagic stroke, congestive heart failure, dissecting aortic aneurysm, and nephrosclerosis.

2. Atherosclerotic, ie, associated with the accelerated development of atherosclerotic lesions of the aorta and its large branches in conditions of high blood pressure. This group includes ischemic heart disease (IHD), sudden death, other arrhythmias, ischemic stroke, obliterating atherosclerosis of the vessels of the lower extremities.

The most common complication of hypertension is coronary artery disease. In recent years, a new term "continuum" has been introduced into the medical literature, which means the inevitability of the outcome of hypertension, inevitably ending in heart failure through hypertrophy and left ventricular dysfunction, or after the development of myocardial infarction.

Of the organ lesions that precede the development of congestive heart failure in hypertensive patients, the most studied is left ventricular hypertrophy. ECG signs of it are found in 3-8% of patients with hypertension I degree. Echocardiography reveals myocardial hypertrophy in 20-60% of hypertensive patients.

Detection of left ventricular hypertrophy is important in hypertension, since in patients with ECG signs of left ventricular hypertrophy, the risk of developing cardiovascular complications is 3-6 times higher than in patients of the same age and sex, but without signs of hypertrophy.

In terms of the degree of study of target organs in patients with hypertension, the kidneys are in second place. Kidney function is usually judged primarily by the level of protein loss. Only the loss of albumin molecules reflects the true destruction of the renal filter. It is known that the diameter of albumin molecules is significantly less than the pore diameter of the renal filter (3.6 and 7.0 nm, respectively). However, the same name of the electric charge of albumin molecules and the cells that form the renal filter leads to their mutual repulsion and prevents the loss of albumin. The change in the charge of the renal filter, which occurs during its destruction under the influence of various factors (hypertension, diabetes mellitus), contributes to a progressive increase in the loss of albumin.

The rate of increase in blood pressure (WHO, 1999)

Index	BP, mm Hg Art.	
HELL	systolic	diastolic
Optimal	<120	<80
Normal	<130	<85
Increased normal	130-139	85-89
AG Degree 1	140-159	90-99
AG Degree 2	160-179	100-109
AG Degree 3	> 180	> 110

The degree of hypertension is determined when hypertension is detected for the first time or during the period when the patient is not taking an antihypertensive drug. The crisis increase in blood pressure is also not taken into account. If SBP and DBP correspond to different degrees of AH, then a higher one is assigned. Example: HELL 135/100 mm Hg. Art. - AG degree 2; BP 165/90 mm Hg. Art. - AG degree 2; BP 170/115 mm Hg. Art. - AH degree 3.

AGE RISK CRITERIA

1. Risk factors.
   - ➢·Men over 55.
   - ➢·Women over 65.
   - ➢·Smoking.
   - ➢·Cholesterol level> 6.5 mmol / L.
   - ➢·Family history of early cardiovascular disease (in women under 65 and in men under 55).
2. Damage to target organs.
   - ➢·Left ventricular hypertrophy (ECG, echocardiography, or radiography).
   - ➢·Protenuria and / or creatininemia (105.6-176 μmol / l).
   - ➢·Ultrasound or radiological signs of an atherosclerotic plaque.
   - ➢·Generalized or focal narrowing of the retinal arteries.
3. Associated (concomitant) clinical conditions.

Vascular diseases of the brain:
   - ➢·ischemic stroke
   - ➢·hemorrhagic stroke
   - ➢·tranny ischemic attacks

Heart disease:
   - ➢·myocardial infarction
   - ➢·angina
   - ➢·congestive heart failure.

Kidney disease:
   - ➢·diabetic nephropathy
   - ➢·renal failure with creatininemia> 176 μmol / L

Vascular diseases:
   - ➢·dissecting aortic aneurysm
   - ➢·clinically severe peripheral arterial disease

Retina:
   - ➢·hypertensive retinopathy
   - ➢·hemorrhages or exudates
   - ➢·swelling of the optic nipple.

Diabetes.

MAIN DIRECTIONS OF PRIMARY PREVENTION AND NON-DRUG TREATMENT OF AH

1. Struggle with excess body weight. Its decrease by 5 extra kg leads to a decrease in SBP by 5.4 mm Hg, DBP - by 2.4 mm Hg. Art.

2. Quitting smoking. Nicotine is a strong stimulant of the sympathetic nervous system, causes the release of vascular catecholamines into the bloodstream, increases vascular permeability, promotes cholesterol deposition and increases platelet aggregation.

3 Limiting consumption of table salt to 5 g per day.

4. Limiting consumption of alcoholic beverages.

5. Increase in physical activity, fight against physical inactivity. Physical exercise in the open air of moderate intensity and lasting at least 30-60 minutes 3-4 times a week.

6. Adding calcium, magnesium and fish oil to food.
7. Relaxing exercises.
8. Limiting consumption of caffeine (tea, coffee, etc.).
9. Increase the duration of sleep up to 9-10 hours.
10. Evasion of psycho-emotional stress.

PRACTICAL PART

THEMATIC TESTS

1

1. Choose the correct statement: BP is primarily determined by:
   a) force of heart contraction and shock release.
   b) elasticity of blood vessels.
   c) peripheral resistance to blood flow.
   d) bypassing blood in the lungs
   e) deposition of blood in the spleen

2. Correctly match the type of hypertension and the disease causing it:
   a) renal hypertension
   b) hormonal hypertension
   c) hypertension due to changes in hemodynamics
      1. Itsenko-Cushing syndrome
      2. aortic isthmus stenosis
      3. renal artery stenosis

3. You prescribe saluretics to the patient to reduce hypertension. On the ECG, you find: flattening of the T wave, depression of the ST segment , an increase in the Q - F interval . What complication is this picture?
   a) hypokalemia
   b) hyperkalemia
   c) hypernatremia
   d) hypocalcemia
   e) hypercalcemia

4.In case of hypertension, the content of renin in the blood (choose the correct solution):
   a) all have significantly increased
   b) all have slightly increased
   c) within normal limits
   d) all have reduced
   e) in some it is increased, in others it is within the normal range

5. To diagnose the vasorenal form of hypertension, it is necessary to perform the following studies, except for one:
   a) urography
   b) plasma renin level
   c) renal vein angiography
   d) kidney scintigraphy

e) Rehberg's test

6. Rational therapy for renovascular hypertension - application:
a) diuretics
b) β-blockers
c) salt-free diet
d) surgical treatment
e) apressin

7. What should be limited in the diet of a patient with essential hypertension:
a) water
b) sugar

SYMPTOMATIC ARTERIAL HYPERTENSION

The purpose of the lecture : Introduction to students with symptomatic arterial hypertension, the causes and E for their development, clinical features, course of complicated forms, differential diagnosis, optimal range s GOVERNMENTAL therapies, postoperative care, reabilit a tion patients.

Educational purposes lectures: Suggestion students need for timely adequate operation to the development of serious complications , and in their development - meeting the most informative and modern m e todami diagnostics, surgical treatment, familiarity with the in of possible complications is the operation and the operational period of Prof. and lactics. Development of students' clinical thinking. Development of present-day e alternating view of the problem the issue from the perspective of world medicine and general practitioners.

Lecture objectives:

1. To give an understanding of symptomatic arterial hypertension.
2. Explain the causes and mechanisms of complications.
3. Give a clinical description and possible variants of the course of the disease.
4. Make a differential diagnosis with other diseases I mi.
5. To acquaint students with modern and most informative s methods, which examination of patients
6. Demonstration of examples of their surgical practice: patients, slides, phlebograms.
7. All the lecture material for students to prepare and present, in an b Birmingham, necessary for quality training of general practitioners.

PLAN OF THE LECTURE.

1. The urgency of the problem - 5 min
2. Etiopathogenesis of symptomatic hypertension.
3. Clinical picture - 10 min

a) Etiopathogenesis;
b) Clinic and diagnostics.
c) Differential diagnosis.

d) Treatment.

4. Diagnostics. - 10 min
5. Differential diagnostics. - 10 min
6. Treatment - 15 min
7. Disease prevention - 10 min

Diseases of the cardiovascular system occupy the first place in the overall morbidity structure of the population, being one of the causes of disability, premature disability and death. Most camshaft on roubleshooting among cardiovascular diseases are hypertension.

Symptomatic arterial hypertension - a very heterogeneous group of diseases, joinable one feature - high Arterial s nym pressure (Goghin EE et al 1978.). Included in this group Klinichev e skie forms not only represent different nosological units and Nitsa with dissimilar etiology, pathogenesis of different, but related to ra s personal health professions - medicine, surgery, urology, e n dokrinologii etc. Allocate following forms simptomati iCal-art e 'an hypertension:

- parenchymal renal, diseases caused by renal parenchyma (pyelonephritis, glomerulonephritis, urolithiasis, Polikom with renal inos, diabetic nephropathy, etc.)
- adrenal due to adrenal disease (fe on hromotsitoma, Conn's syndrome, Cushing's syndrome)
- central origin, due to diseases of the brain (encephalitis, tumors, trauma)
- malformations of large vessels (coarctation of the aorta, congenital hypoplasia and aplasia of the aorta)
- renovascular hypertension

Renovascular hypertension (VRG) - one of the forms of secondary hypertension, which develops due to violation MAGIS t eral blood flow in the kidney of the primary lesion without renal parenchyma and urinary tract. C mong all forms of hypertension vases of renal hypertension is 2-5%.

Renal hypertension is always based on one- or two - sided narrowing of the lumen of the renal artery or one or more of its large branches. As a result, less blood enters the kidney per unit of time through a section of an artery with a pathologically with a hole in the kidney. This leads to the development of renal tissue ischemia, the severity of which the head and the sieve on the degree of stenosis of the affected artery.

Etiology. Atherosclerosis is the main cause of renovascular p Noah hypertension in persons over 40 years and is 60-85% of cases. Ater about Sclerotiniose cally plaques localized mainly in the mouth or in the forward of ksimalnoy third renal artery. In the overwhelming majority of cases, there is a unilateral lesion of the renal artery, while its bilateral lesion occurs in about 1/3 of cases and leads to a more severe course of renovascular hypertension. The disease is more common (2-3 times) in men.

Fibromuscular dysplasia as the cause of renovascular giperte n sion is second only to atherosclerosis. Fibromusk found in polar dysplasia predominantly in young and even children's age (12 to 44 years); the average age is 28-29. In women, she found a etsya 4-5 times more often than men. Morphologically di fibromuscular with plasia proyav - it possible in the form of dystrophic changes and sclerosing, advantageously gripping the inner casing and the average kidney and p Theurillat and their branches. In this case, hyperplasia of the muscular elements of the wall can be combined with the formation of microaneurysms. As a result, there is an alternation of areas of narrowing and widening (aneurysms), which gives the art e Riyam original form - in the form of strings of pearls or beads. Pathological forth about process of, though, and has a common, but in 2/3 of the cases is about Dr. nostoronnim.

Renovascular hypertension may develop due to extra and knitting compression renal arteries from thrombosis or embolism of the renal artery, aneurysm formation, the main renal artery hypoplasia, Nephroptosis, tumors, cysts, abnormal kidney development and others.

Pathogenesis. Narrowing or occlusion of the kidney results in decreased renal blood flow and decreased perfusion pressure. Development ish e mission kidney tissue leads to hyperplasia juxtaglomerular cells (SOUTH) of the vessel, resulting in the hypersecretion of renin. Renin (this - enzyme) coming from the liver converts angiotensinogen into a n angiotensin I , which is under the influence of ACE fe p ment converting - etsya to angiotensin II . Angiotensin II of - one of the most powerful vasoconstrictor, which is directly affecting the systemic arterioles, causing them to spasm and dramatically increases peripheral interfacing to tance. In addition, angiotensin-aldosterone stimulates the adrenal cortex, resulting in the development of secondary giperaldost e ronizma, with sodium and water retention. Peripheral vasoconstriction, Gypea p natriemiya hypervolemia and exacerbate arterial giperte n sion.

The natural course of atherosclerotic CPH is characterized by a progressive decrease in renal blood flow, which ultimately leads to a complete loss of renal function ("ischemic nephropathy"). This disease manifests itself in middle or old age. On the contrary, fi b romuskulyarnaya dysplasia usually manifests at a young age, is more common in women, has not progressive course and rarely pref on dit to ischemic nephropathy.

Clinic. Patognomichnyh renovascular hypertension symptoms characteristic of certain forms of hypertension (Conn's syndrome, Cushing's Syndrome, Fairy of hromotsitoma) no.

Patient complaints can be divided as follows:

1. Complaints specific to cerebral hypertension, - headaches used to know, a feeling of heaviness in the head, tinnitus, pain in eyeballs, memory loss, poor sleep.

2. Complaints related to the overload of the left heart and crown p Noah failure - pain in the area and the heart palpitations, so I tin behind the breastbone.

3. A feeling of heaviness in the lumbar region, not intensive pain and hematoma at the dence in the case of renal infarction.

4. Complaints characteristic ischemia other organs backbone and p terii, which struck simultaneously with the renal arteries.

5. Complaints characteristic common syndrome in inflammation (nesp e cific aortoarteriit).

6. Complaints characteristic of secondary hyperaldosteronism: myshe h Nye weakness, paresthesias, tetany seizures izogipostenuriya, polyuria, n of lidipsiya, nocturia.

However, it should be noted that about 25% of patients with vasorenal hypertension are asymptomatic.

Diagnostics. For statement of the diagnosis the following are important anamnest and cal data:

1. Development of stable hypertension in children and adolescents.

2. Stabilization and refractory hypertension treatment in persons over 40 years, in whom the disease before a benign and hypothetical n zivnaya therapy to be effective, the identification of these patients are interleaved th conductive lameness or \ and symptoms of chronic cerebrovascular insufficient and accuracy.

3. Communication develop hypertension of pregnancy and childbirth (without nefrop and TII)

4. Connection of the onset of hypertension with instrumental studies or manipulations in the kidney area, with kidney and abdominal aorta operations.

5. The development of hypertension after an attack of pain in the lumbar region and hematuria in patients with heart disease, arrhythmias or in patients with post - napark cardiosclerosis and episodes of embolism in other arterial basins.

When viewed measure the pressure on the upper and lower extremities that would eliminate koarktatsionny syndrome and identify hitting e Nia arteries of the upper and lower extremities, as well as in the horizontal and vertical position. If orthostatic position Arterial s pressure was higher, then you can think about Nephroptosis.

Necessary auscultation abdominal aorta and renal arteries - straight and approximately 40% of patients auscultated systolic murmur in the projection renal and p Theurillat or abdominal aorta. Diagnosis can help auscultation systolic murmur of surfactant arranged arteries: with n GOVERNMENTAL, subclavian and femoral - as a sign of systemic lesions in atherosclerosis and aortitis

On the basis of examination and a number of studies, the following signs can be identified that allow one to suspect renovascular hypertension:

- arterial hypertension resistant to two or more hypothetical n zivnym drugs and diuretics;

- the appearance of arterial hypertension before the age of 20 in women or after 55 years;
- accelerated progressive or malignant hypertension;
- the presence of various manifestations of atherosclerotic disease;
- azotemia, especially developing with the use of ACE inhibitors or angiotensin II receptor blockers;
- systolic murmur over the abdominal aorta and renal arteries;
- differences in kidney size exceeding 1.5 cm (based on ultrasound);

The above mentioned features allow only suspect, assume renovascular hypertension, is often sufficient substantiation Mr. Noah, but they do not make it impossible to fully confirm this diagnosis. Additional studies are needed to confirm or exclude the diagnosis of renovascular hypertension. The most reliable and easy method for diagnosing renovascular hypertension is angas of renal graphy, which can be performed in specialized vessel and grained centers. Angiography allows you to determine the cause of the stenotic process, to assess the degree of stenosis and its localization, which is crucial for resolving the issue of surgical treatment.

However, there are a number of minimally invasive, screening method is studied on the Island - steps that may reveal the defeat of the renal arteries and to determine indications for angiography and avoid it to those patients who have other d e ab hypertension. In particular, high sensitivity have stsintigr and raphy with ACE inhibitors, doppler - ultrasonography, magnetic resonance and CT angiography, and they can be used in combination or t separately to achieve adequate screening patients prior to revascularization or conventional angiography.

Renoscintigraphy with angiotensin-converting enzyme (ACE) inhibitors. The use of ACE inhibitors in functionally significant item e noze renal artery leads to a decrease in glomerular rate fil m radios, due to the removal or substantial weakening of constriction of the efferent arteriole. This results in characteristic changes in the renogram.

Angiotensin-converting enzyme (ACE) inhibitor scintigrams should be interpreted consistently with low, moderate and high likelihood of renovascular hypertension. The most specific diagnostic criterion for renovascular p hydrochloric scintigraphy hypertension is ACE inhibitor-induced edited e neniya.

These criteria are:

1. A normal scintigram using ACE inhibitors shows a low probability of HRH less than 10%.
2. Reduced poorly functioning kidney (capture less than 30% over time the maximum activity of [Tmax] 2 minutes, which is not dormancy and binds changes scintigraphy using ACE inhibitors and DUPLEX n of symmetrical disorders such as cortical delay tubular agent indicates the average probability VRG.

3. Criteria associated high probability VRG include deteriorated e of stsintitgraficheskoy curve decrease in relative seizure, etc. of dlenie renal and parenhimalnog transit time increase over 20 minutes / peak capture rate coefficient and extending T-max.

Doppler - ultrasound examination. This research has advantages in terms of its non-invasiveness and low cost. Two methods are used to detect VRG using Doppler - ultrasound Resea and lines: The direct visualization of the renal arteries and the analysis of Doppler waveforms.

Direct visualization of the renal arteries. The first method includes direct viewing main renal arteries with color or dopler- ultrasound energy with an analysis of the renal arteries rate using spectral dopler- ultrasonograph Signal enhancement can be achieved by taking contras t Nogo substance which facilitates the visual image of the renal art e ry.

Three-dimensional ultrasound angiography provides a d e tal visualization of the renal arteries and the accuracy of the image yoy and presentable with a three-dimensional magnetic resonance angiography.

Four criteria are used to diagnose significant proximal renal artery stenosis or occlusion:

(a) an increase in peak systolic velocity in the renal and p terii (in the literature, the threshold for significant renal artery stenosis - 100-200 cm / sec);

(b) the renal-aortic ratio of the maximum systolic velocity is greater than 3.5;

(c) turbulent flow in the post-stenotic region of the region;

(d) visual observation of the renal artery without detectable d of plerovskogo signal indicating an occlusion.

In addition, with the help of ultrasound, it is possible to identify indirect signs of VRH, in particular, a decrease in the size of the kidneys due to its ischemic atrophy. Kidneys less than 7-8 cm in length are usually severely damaged by ischemia. In these circumstances, revascularization is not usually so forth and leads to restoration of function or hypertension, and the patient is shown nephrectomy.

Magnetic resonance angiography. Magnetic angiogr and raphy with gadolinium contrast is now available as a high discharge e Collapsing imaging system with high image quality, to about Thoraya capable of forming a three-dimensional image. Angiographic contrast - vapor and magnetic, reduces the T1-effect. Blood before with nent bright, while the fabric still remain dark. Subtraction images nekontrastirovannyh removes all background Sig and ly and improves vascular signals.

Spiral computed tomographic angiography . Cn and eral computer tomographic angiography (CTA) is a noninvasive method, however, requires the introduction of 150 ml of a counter and stnogo substance. It can be adapted to measure renal cr on votoka patients renovascular hypertension, as well as three-dimensional roluchit of a vascular mapping

The first step in diagnosing renovascular hypertension are Klinichev e Skye diagnosis and selection of patients with moderate and high probability of this disease by clinical criteria. Non-invasive screening tests provide sighting selection of patients with a high probability of renal artery stenosis, thereby reducing the frequency of potential v GOVERNMENTAL adverse effects of X-ray angiography when it is widely used as research institutes. In patients with a high probability of disease must be pre d taken X-rays to determine the intended item e Noz renal artery. Spiral CT can provide excellent visualization and visualization of the renal vessels, but requires a lot of contrast. Currently MRA gives a good image of the renal vessels without risk to the patient, but, with their higher cost and shorter until a tupnostyu, it should be reserved for patients with undecided n nymy funktsionalnymy results images but high Klinichev e skim suspected VRG, and patients who have a contraindication to standard angiography: renal failure or allergies to iodine and oral medications

Treatment. The following types of treatment can be distinguished:

1. Conservative - with contraindications to the intervention s stvu.

2. Surgical methods:

- Reconstructive surgery: transaortic endarterectomy, p e plantation renal artery, renal artery resection prosthesis renal artery.
- Organ-carrying operations - nephrectomy.

3. X-ray endovascular methods: transluminal angioplasty of the renal artery (or X-ray endovascular balloon dilatation - RED) with or without stenting; simultaneous REV on the adrenal glands for the correction of secondary hyperaldosteronism.

The most effective treatment for renovascular hypertension - surgery aimed at removing the causes of stenosis n of Chechnya of the arteries and the restoration of normal renal blood flow. Up to 1952 g . the only method of surgical treatment was nephrectomy, which was used for deliberately unilateral lesions and in an advanced stage of the disease. Nephrectomy is used and at the moment, if dominated by narrowing intrarenal vessels or by regex and zhennoy hypoplasia of the affected kidney and significant violation of her fe to tion. The indication for nephrectomy is to reduce the size of the kidney to 8 cm or less. In other cases, organ-preserving operations are widely used to restore renal blood flow. The earlier the diagnosis of vasorenal hypertension and the cause of its appearance are established, the more effective the results of surgical treatment.

At the same time in patients with renovascular hypertension, even when zlok and qualitatively during the sometimes possible to achieve a good effect with individually selected antihypertensives. However, in the proof n stenosis of the renal artery is not recommended to perform the conservative therapy, as blood pressure reduction leads to further deterioration poche h Nogo flow and

development in a short time secondary smo p schivaniya kidney and loss of its function.

Depending on the etiology of the disease, PTCA or stenting can be successful in 80% of cases. However, these procedures are invasive and can lead to rupture or dissection of an artery, ateromato of hydrochloric kidney embolism or lower extremities, acute renal Insufficient hours density due to contrast induced nephropathy, bleeding on the side of puncture and (rarely) the patient's death.

Surgical revascularization remains the reserve method for those patients who have failed CHTPA and stenting, as well as for bol s GOVERNMENTAL with concomitant abdominal aortic lesion requiring operative on the first intervention. Patients with high and poorly controlled Arterial v hydrochloric hypertension if thus reduced dimensions kidney and significantly reduced its function is shown more nephrectomy.

Adrenal hypertension is most often caused by its tumors. The most common: aldosteroma, pheochromocytoma, mixed tumors of the adrenal cortex, corticosteroma, androsteroma, corticoest e Roma. All these types OPU Hawley can be either benign or PLN on quality.

Aldosteronoma (primary hyperaldosteronism, Conn syndrome) develops the zona glomerulosa of the adrenal cortex at the feed -governing bol s shinstva patients the tumor is benign and only 5% detect malignant growth pattern. The tumor tissue produces an excessive amount of aldosterone.

Pathogenesis. Excessive production of aldosterone leads to various biochemical and morphological changes in the body. First of all, this disease is characterized by pronounced electrolyte disturbances. Aldosterone, acting on the renal tubules, leads to a decrease in the reabsorption of potassium and water and, conversely, to an increase in sodium reabsorption. Enhanced excretion of potassium in the urine leads to the development of hypokalemia (less than 3.0 mmol / l.) Potassium ions in the cell are replaced by sodium ions and in the body. A decrease in natriuresis leads to an increase in the content of sodium ions in the intra- and extracellular space. Sodium being hydrophile s charged ion retains and attracts water. As a result, edema tissue, particularly vascular wall decreases its inner etc. of light at the arterioles, increased vascular tone and peripheral vascular resistance, hypertension develops.

The disease most often affects women mature vozra hundred. Symptoms of ald o steroma can be divided into 3 groups:

1) neuromuscular
2) renal
3) associated with increased blood pressure

Myshech neuropsychiatric symptoms were caused by hypokalemia and tied s mi this impaired neuromuscular conduction. Patients liking so camping on severe muscle weakness, the degree of which varies - from the b s operation fatigue to flaccid paralysis, covering most of the leg muscles. Paresthesias and convulsions are very common.

Among the renal symptoms, the most common are polyuria, nocturia, hypostenuria . Thirst develops due to the loss of a large amount of fluid in the urine.

Hypertension - the main, sometimes the only si m ptom aldosteroma. The course of hypertension is usually stable. The level of blood pressure increase ranges from mild (160/100 mm Hg) to the expression n Nogo (220-250 / 120- 140 mm .rt.st.). Most patients complain of severe headaches caused by high blood pressure. Hypertension leads to severe hypertrophy of the left w e ludochka, the ECG shows signs of hypokalemia. Very common is the defeat of the vessels of the fundus with impaired visual function.

Diagnostics is based on the analysis of clinical manifestations of diseases and laboratory data. Radioimmunoassay reveals an increase in the concentration of aldosterone in plasma, in basal conditions and its paradoxical decrease after a test with a 4-hour walk, a decrease in plasma renin activity. Biochemical studies reveal gipok and Liem, hypernatremia. An alkaline urine reaction may have a certain diagnostic value. Among the instrumental methods, ultrasound and CT are important. Due to the fact that have small aldosteroma pa of steps (1.5 cm -2) by means of ultrasound can reveal approximately 60% bol s GOVERNMENTAL. The most accurate method of diagnosis is a computer tomogr and FFL. CT scan reveals formations of low density (12-14 Hn units).

Treatment: surgical - adrenalectomy

Pheochromocytoma - a tumor of neuroectodermal origin of the chromaffin tissue, producing catecholamines (adrenaline, noradr e Nalin, dopamine). Most often it develops from the adrenal medulla and ka (in 90% of cases). In 10%, a pheochromocytoma (paraganglioma) of extra-adrenal localization (more often in the sympathetic paraaortic ganglia, bladder, posterior mediastinum) is found. The tumor may be ML and a night, and multiple, dobroka -quality and malignant. Ill and of often arises in middle age men about equally often. There are reports of a familial nature of pheochromocytoma.

In the pathogenesis of the disorders developing in patients with pheochromocytoma, hypersecretion of catecholamines and their periodic burst release into the systemic circulation are of primary importance. The level of catecholamines during crisis, particularly noradrenaline, ten times higher than normal, and their excess causes excitation of alpha- and beta-adrenoceptors, which pref about dit a pronounced arteriolar spasms level and sharp increase commonly e th peripheral resistance, whereby increases as a system for cyclically and diastolic blood pressure.

The clinical picture. Kar dinalnym pheochromocytoma symptom is hypertension, koto paradise can be of three types - a stable, paroxysmal and mixed, in connection with which secrete sootvets m vuyuschie clinical types ones cheniya disease. In the paroxysmal form, hypertensive crises are noted with an increase in blood pressure to 250 - 300 mm Hg and above. A sudden increase in blood pressure is accompanied by a sudden headaches, palpitations, fear of death, chills, fever t e la, sweating. Shortness of breath, pain in the lumbar region, in the

abdomen, behind the breastbone are often noted. Nausea and vomiting may occur. Prolonged s NOSTA kriza from several minutes to several hours. For catecholamine of first hyperleukocytosis crisis characterized by hyperglycemia and glycosuria. Outside the crisis, blood pressure is normal and patients do not present complaints.

With a stable form of arterial hypertension, there is a persistent increase in blood pressure without crises. In the mixed form, catecholamine crises are observed against the background of increased blood pressure (160 / 100-180 / 120 mm Hg). Nekupiro-vanny catecholamine crisis can result in death, with and hydrochloric which may be congestive heart failure, pulmonary edema, hemorrhage in the brain.

Diagnostics. a leading role in establishing the diagnosis feohromo tsitomy, along with the clinical picture, belongs to the study of concentrations of catecholamines in the urine (daily or collected after a crisis). Giperprodu to tion norepinephrine and increasing excretion of the hormone in the urine but when p mal concentrations of adrenaline characterized thorns for extra-adrenal tumor localization. The simultaneous increase in the concentration of both p Mon in urine is more typical for adrenal tumor localization in practice quite often used to determine the concentration-vanillyl mandelic acid in urine. This acid is a metabolite of both P Mon, and its concentration in the urine of a few tens of times to n centration of adrenaline and noradrenaline. For feohromotsi toms characterized by a significant increase in the concentration of m-vanillyl a distant acid m of Th. Given the large size of the tumor, they can be easily detected by ultrasound and CT.

Feohrotsitomy only surgical treatment - removal of the tumor (Fairy of chromium tsitomy).

Among other adrenal diseases is necessary to allocate a symptom of the mo complex endogenous giperkortizma that combines different claim and pathogenesis, but similar clinical manifestations of the disease. A similar clinical picture is caused due to the overproduction of glyukoko p tikoidnyh hormones, especially cortisol. Distinguish between Itsenko-Cushing's syndrome and Itsenko-Cushing's disease (tumor-free form). Cushing's syndrome is caused by a tumor that develops from cortex zona fasciculata nadpochech nick (benign tumor - corticosteroma, zlokach e governmental - kortikoblastoma). Tumor tissue cells are overproduced to p Tizol. Zabol vayut more often women (almost 80%) aged 20--40 years. Kleene Cesky picture syndrome and Cushing's disease is quite typical. The most persistent symptoms are obesity and arterial g and pertension. Fatigue and muscle weakness, decreased performance, and sexual dysfunction appear early. At a later date, osteoporosis joins. Obesity is associated with excessive production of cortisol and ACTH, which inhibit the fat-mobilizing effect of the growth hormone and on. Arterial hypertension in Cushing's syndrome has Art and stably over without crises, there is a proportional increase in B with tolicheskogo and diastolic pressure, is resistant to antihypertensive ter and FDI. Characterized by the appearance of patients - moon face, purplish-bluish complexion and upper chest, the presence of "red stretch

marks" - purple-bluish stripes on the abdomen skin, waist, breast w e climb, thighs. The skin becomes dry, the limbs acquire a bluish-marble color.

Diagnosis: decisive role belongs study concen tration level 17 of corticosteroids (CS-17) in blood and urine. When corticosteroma this dormancy and ence significantly increased, especially in malignant nature of op in Hawley.

Instrumental diagnostics - ultrasound, CT.

Treatment: surgical - adrenalectomy - removal of the tumor (corticosteroma) together with the adrenal gland.

Androsteroma develops from the reticular area of the adrenal cortex. The clinical picture is due to the excessive production of andro-genes. Zabol e vanie occurs at a young age and mature. More often women are ill. In childhood, girls appear hyper trihoz accelerated growth chre of uniformly developed musculature of Los becomes low, rough. In small s tors comes first time puberty, characterized by also strengthening the devel ment muscles, short stature, short of the lower limb of the STI. In women, the disease is manifested symptoms of masculinization with clause about the phenomenon of male sexual characteristics - reduction hypodermis Foot layer of fat, gain muscle development, atrophy of the breasts, menstrual dysfunction; often fuss repents hirsutism.

In the study of the patient's hormonal profile, attention is drawn to the huge content of 17-KC in the urine. For defined Lenia tumor localization applied ultrasound and CT.

Treatment: surgical - adrenalectomy.

QUESTIONS

to the audience to provide feedback and to determine Ven and zheniya lectures goal **.**

1. What is "essential hypertension"?
2. What is symptomatic hypertension?
3. The main etiological causes of renovascular hypertension (VRH).
4. The main etiological causes of adrenal hypertension.
5. What is aldosteroma?
6. What is pheochromacytoma?
7. Angiosemiotics in FMD.
8. Types of operations with VRG.
9. Types of operations for adrenal hypertension.
10. Tactics of GPs in diagnosing SAG.

THEME . OCCLUSIVE AND STENOSING DISEASES OF THE ARTERIES OF THE LOWER LIMBS. LERICHE SYNDROME. OBLETERATING ATHEROSCLEROSIS .. BUGER'S DISEASE.

Intermittent claudication. Etiology, diagnosis and differential diagnosis, research methods and treatment tactics. Obliterating atherosclerosis, endoarteritis. Buerger's disease, congenital vascular disease, lower limb ischemia, classification.

Leriche syndrome. Etiology, diagnosis and differential diagnosis, research methods and treatment tactics. Indications for surgical treatment.

Chronic arterial insufficiency. Etiology, diagnosis and differential diagnosis, research methods and treatment tactics.

The purpose of the lesson:

1. Study the surgical anatomy of the lower limb arteries
2. Vascular research methods.
3. To study the etiology, pathogenesis, clinic and diagnosis of patients with obliterating endarteritis. Learn the correct diagnosis according to the modern classification. Be able to make a differential diagnosis.
4. Know the basic principles of treatment of patients with lower limb artery disease.
5. Establish the basic principles of prevention, labor expertise.

Test questions:

1. Classification of peripheral vascular lesions of the lower extremities
2. Anatomy and normal hemodynamics of the vessels of the legs.
3. Special methods for the study of peripheral circulation
4. Etiology and pathogenesis of diseases of occlusive lesions of the arteries of the lower extremities.
5. Clinic, differential diagnosis of these pathologies.
6. Determination of the level of vascular occlusion
7. Conservative treatment.
8. Surgical treatment .
9. Determination of the level of limb amputation in gangrene.

THEORETICAL PART

Research methods:

Inquiry. Usually, upon questioning, symptoms associated with circulatory failure of a particular organ are revealed. So, complaints about the appearance of pain in the legs when walking and their disappearance at rest (intermittent claudication) suggest the presence of stenosis or occlusion of the arteries of the lower extremities. Manifestation niyami peripheral circulatory disorders also are muscle weakness, numbness and "pins and needles." Headaches can be caused by cerebrovascular insufficiency, especially when combined with episodic vertigo or loss of vision, and pain in the abdomen at the height pischevare Nia - a symptom of chronic circulatory disorders visceral scheniya.

Physical research methods. Examination, in some cases gives erate information on the nature of the pathological pro cession. In chronic limb ischemia in patients usual but develops muscle hypotrophy, reduced content under cutaneous veins varies skin coloration (pallor, marbling and t. D.) Appear trophic disorders in the form of hair loss, dry skin, thickening and brittle nails, etc. In severe peripheral ischemia, blisters filled with serous fluid, dry (mummification) or wet (wet gangrene) necrosis of the distal limb segments are determined. The presence of a pulsating formation in sync with the pulse in one or another area (neck, abdomen, limb) allows one to suspect a vessel aneurysm.

Auskulypatsiya vessels is an essential component obsl dovaniya patients. Normally, over the main arteries vyslushi INDICATES pin tone pulse wave noise occurs when systolic stenosis or aneurysmal enlargement arteries. Auscultation is performed over the projection of the carotid

and subclavian arteries, brachiocephalic trunk, vertebral arteries, ascending and abdominal aorta, celiac trunk, renal, iliac and femoral arteries. In this case, the noise from the left subclavian artery listen behind sternocleidomastoid muscle at the point of its attachment to the Clew chitse; on the right at the same point, you can determine the noise from the brachiocephalic trunk. Noises from the vertebral arteries are projected 2 cm proximal to the middle of the clavicle, from the ascending aorta - in the second intercostal space to the right of the sternum. According to the midline of the abdomen under the sword prominent appendage auscultated with celiac artery stenosis of the aorta when. Along the pararectal line, in the middle of the distance between the horizontal lines, mentally drawn through the xiphoid process and the navel, a noise is heard from the renal arteries. In the midline at the level of the navel and above, noises from the abdominal aorta are localized. The noise from the iliac arteries is projected along the line connecting the abdominal aorta with a point located on the border of the inner and middle third of the inguinal ligament. Auscultation and total sleep femoral artery is produced in locations where they ripple determined. When listening to the neck vessels should be distinguished from noise artery stenosis and heart murmur, the intensity of which increases as when approximation to the heart. When aneurysms and arteriovenous fistula vascular maximum noise intensity noted in the places of their locale tions.

Functional tests. Among the functional tests used in the diagnosis of chronic arterial insufficient STI lower limbs, the most practical application have Oppel symptom Goldflama sample Samuels, knee Panchenko phenomenon, a symptom of compression of the nail bed.

Oppel's symptom (plantar ischemia) is the pallor of the sole of the foot of the affected limb, raised up at an angle of 45 °. Depending on the speed of blanching can judge the degree of circulatory disorders in the limbs: in severe Ishe mission it occurs over the next 4-6 seconds.

Goldflam test: position of the patient on his back with legs raised above the bed. It performs flexion and extension in Gol nostopnyh joints. If blood circulation is impaired, after 10-20 movements, the patient experiences fatigue in the leg. Simultaneously monitored for coloring the plantar surface of the foot (Ca sample myuelsa). With severe lack of blood supply, the feet turn pale within a few seconds.

Panchenko's knee phenomenon is determined while sitting. Sick, injured leg thrown back to a healthy, soon begins uc experience t pain in the calf muscles, numbness in the foot, oschu schenie crawling in the fingertips of the affected limb.

Symptom compression of the nail bed is that the compression of the terminal phalanx of the I toe in the anteroposterior direction lenii for 5-10 sec in healthy people formed the nail bed blanching immediately replaced by a normal color. When at Rushen blood circulation in the limb is kept blanching length tion time, in cases where the nail plate to change, watching the color is not the nail bed and the nail shaft. In pain GOVERNMENTAL with peripheral circulatory disturbance formed as a result of compression of a white patch on the skin over an extended period of time.

Special instrumental methods of investigation of Bani. The most informative are ultrasound research methods, computed tomography, rheovasography, thermography and angiography.

The ultrasonic flowmetry (Doppler) based on the ef fect is Doppler ultrasound beam registration to oscillations reflected from the surface of a moving blood vessel. Dopplerograms recorded with peripheral arterial disease, provide valuable information on the values of the regional blood pressure, if the linear velocity of blood flow, which gives an indication of the degree of vascular lesions and status of collateral circulation. With advanced new generation of devices equipped with micro rokompyuterami, conduct spectral analysis of Doppler sig catch obtained display image on the screen (or Jun color but white) receptacles define a diameter of the lumen and outer con tour, calculated volumetric flow rate.

Computed tomography is based on obtaining layer-by-layer cross-sectional images of the human body using an X-ray tube rotating around it. It allows you to visualize the cross-sections of the aorta and the mouths of its branches (iliac, ruff echnyh, the renal arteries, the celiac trunk,

brachiocephalic arte ry), to judge the state of the walls, the relationship with the surrounding tissue structures.

Magnetic resonance imaging (MRI) provides WHO possibility to carry out research vessels without the introduction of contrast agents in several mutually perpendicular planes, and thereby obtain a detailed picture of the state of the vascular bed.

Rheography based on registration of electric oscillations with disobedience tissue, changing depending on the limb blood supply. Segmented curve normal (Fig. 26) is characterized by a steep and rapid rise in the pulse wave (AB), clear Ver bus (B), the presence of two additional teeth (D, F) in the descending portion (catacrota). It can be used to determine the time parted injured pulse wave (β□), the speed of the maximum blood filling investigated seg ment α□and a number of other parameters, among which the most informative is the quantity rheographic index - about derivative on the ratio s plitudy fundamental wave rheographic curve to the height of the calibration signal.

Thermography is based on the registration of own infrared radiation of a particular part of the human body and its transformation into electronic pulses. With the help of modern equipment made possible to obtain an objective idea of the intensity Nosta thermal radiation on the color scale. Currently, thermography has lost its importance, as more and more informative ultrasound examination is being used more and more.

Angiography is required for accurate topical diagnosis pas ontological processes. There are: 1) puncture arteriography, in which a contrast agent is injected directly into one of the peripheral arteries (femoral, brachial) by puncturing them through the skin; 2) Seldinger aortoarteriography, in which a special radiopaque probe is inserted into one or another part of the aorta retrogradely through a peripheral artery (femoral, brachial), a contrast agent is injected through it, and a series of images is taken to study changes in the aorta and its branches; 3) transluminal aortography, in which the aorta is punctured with a special needle at the level of the XII thoracic or I lumbar vertebrae, a contrast agent is injected and X-rays are taken.

With the new generation plants via ethnographic can receive digital (digital) image substraktsionnye ap Theurillat after intravenous administration of relatively low doses of the contrast agent.

In most cases of vascular disease, the correct diagnosis can be made using routine clinical examination. Special methods, as a rule, only detail it. Therefore, at certain stages of the examination, with the correct use of clinical methods, a number of instrumental studies can be abandoned. Diagnostics is undeniable when ority during preoperative preparation and then after the operation observation.

Obliterating endarteritis is a vascular disease of a neurohumoral nature, manifested initially by spasm, and later by thrombosis and obliteration of small and then larger arteries. The distal arteries of the lower extremities are usually affected. Zabo Levan observed mainly in men aged 20-30 years.

Etiology and pathogenesis. Development endarteriita sposobst exist prolonged hypothermia, frostbite, injuries to the lower extremities; smoking, vitamin deficiency; severe emotional shocked Niya, mental disorders; infectious diseases, disrupt Nia autoimmune processes, and other factors that cause persistent vasospasm. It is believed that vasospasm is maintained by hyperadrenalinemia due to increased adrenal function. A long-term spasm of arteries and their accompanying vasa vasorum leads to chronic ischemia of the vascular wall, thereby advancing intimal hyperplasia, fibrosis of the adventitia and degenerative tive changes in their own neural apparatus of the vascular wall. Against the background of the altered intima, a thrombus forms, narrowing and obliteration of the vessel lumen occurs. If at the beginning of the disease mainly affects the distal lower extremities vessels, in particular arteries leg and foot, then subsequently in the pathological process involved and the larger arteries (popliteal, femoral Nye, iliac).

"Leriche syndrome"

X ronicheskie obliterating diseases of the aorta and lower limb arteries (in most cases due to atherosclerosis) account for over 20% of all types of cardiovascular disease, which corresponds to 2-3% of the total population. So, in the Edinburgh study (1990), patients with intermittent claudication accounted for 4.5% in the age group from 55 to 74 years, and asymptomatic lesions were noted in 8% of cases. It is significant that the attending physicians only 30–50% of patients knew about the presence of intermittent claudication in the latter.

Modern diagnostic methods

Modern methods of diagnosing disorders of peripheral arterial circulation are characterized by a wide range - some are used to clarify the clinical diagnosis, the nature and degree of vascular lesions, while others are used to assess the effectiveness of treatment or follow-up of the patient. In order to study hemodynamics in the lower extremities and topical diagnosis of lesions of the arterial bed, the following ***instrumental research methods*** are used ***:*** ultrasound Doppler sphygmomanometry, treadmill test, ultrasound angioscanning, including duplex scanning, and X-ray contrast aortoarteriography. In addition, it is necessary to determine the indicators of lipid metabolism, coagulation system and rheological properties of blood.

As the first stage, all patients with suspected occlusive stenotic lesions of the aorta or lower limb arteries ***undergo Doppler ultrasound with measurement of the ankle-brachial index*** .

This index is the ratio of the maximum pressure on one of the tibial arteries to the pressure on the brachial artery. A decrease in this indicator to less than 0.9 requires closer attention to the patient. In this regard, one of the most promising at present is the combined use of ***ultrasonic Doppler and standard treadmill test*** . Non-invasive research methods also include ***ultrasound angioscanning*** , thanks to which it is possible to determine the degree of stenotic lesion with a high degree of certainty. Recently, ***duplex ultrasound angioscanning*** in the algorithm of the diagnostic program has taken one of the leading places due to non-invasiveness and safety, as well as high sensitivity and specificity. According to the data of duplex scanning, not only the structure of the atherosclerotic plaque is determined, but also the hemodynamic degree of stenosis is assessed, which is of fundamental importance. ***Radiopaque angiographic examination*** currently remains the main method for diagnosing obliterating diseases of the vascular bed. Using this method, it is possible to accurately determine the localization, length, degree and nature of stenosis, the multiplicity of occlusive lesions of the main arteries of the lower extremities, to assess the state of the collateral bed, to predict the nature and volume of reconstructive surgery, and to monitor the effectiveness of treatment and surgical intervention. In the arsenal of angiologists and vascular surgeons, there are also diagnostic methods such as laser Doppler flowmetry, transcutaneous O_2 monitoring , photoplethysmography, radioisotope studies, computed tomography and nuclear magnetic resonance.

PRACTICAL PART

THEMATIC TESTS

1.
1

2. When obliterating endarteritis is most often affected
   a) Arteries of the foot and lower leg
   b) Large caliber artery
   c) Aorta
   d) Veins
   e) All vessels to the same extent

3. A 26-year-old patient complains of constant pain in the right foot, sleeps with the foot down, the objectively right foot of a purple cyanotic color is cold to the touch, the pulsation of the arteries of the feet is not detected. Your diagnosis
   a) Obliterating endarteritis - stage 3 ischemia
   b) Obliterating atherosclerosis
   c) Obliterating endarteritis-stage 4 ischemia
   d) Leriche syndrome
   e) Raynaud's disease
4. A 24-year-old patient complains of excruciating constant pain in his right foot, sleeps with his leg down. Objectively: the right foot is purple-cyanotic, cold to the touch, pulsation of the foot arteries is not detected. Your diagnosis
   a) Obliterating endarteritis
   b) Obliterating atherosclerosis
   c) Diabetic angiopathy
   d) Deep vein thrombophlebitis
   e) Blue phlegmas
5. The most common cause of acute arterial thrombosis is:
   a) Obliterating atherosclerosis
   b) Thromboangiitis obliterans
   c) Puncture and catheterization of arteries
   d) Extravasal compression of arteries
   e) polycythemia

5. For acute arterial insufficiency of the extremities is not typical
   a) Paralysis of the limbs
   b) Lack of pulse
   c) Paresthesias
   d) Trophic leg ulcers
   e) Pale skin and pain
6. The main factor that determines the treatment tactics for acute arterial insufficiency is:
   a) degree of limb ischemia
   b) the patient's age
   c) the severity of the general condition
   d) the presence of concomitant atherosclerotic stenosis or occlusion of the affected artery
   e) localization of thrombus or embolism
7. For acute obstruction of the main arteries of the limb is not typical
   a) epidural edema
   b) paresthesia
   c) pallor
   d) pain
   e) disappearance of pulse
8. In the differential diagnosis between acute venous and arterial thrombosis, everything except
   a) sex of the patient
   b) the nature of the edema
   c) skin coloration
   d) pulsation of arteries
   e) condition of superficial veins
9. The thought of mesenteric thrombosis can be suggested by:
   a) abdominal pain not consistent with clinical findings that would explain its greater intensity
   b) colicky abdominal pain

c) cramping abdominal pain
d) intermittent abdominal pain
e) all of the above is true

10. Contraindications to reconstructive surgery on the arteries are
a) Purulent infection in the wound
b) Gangrene of the limb
c) subfascial limb edema
d) hypo and areflexia in the affected area
e) Lack of active finger and hand movements

1

Thematic tasks

1. The patient, 32 years old, was admitted to hospital with complaints pas unbearable pain in the limbs, weakness, fatigue when walking, chill fingers, feet, and various sensations povy shennuyu sweating. A history of frostbite of the toes, a heavy smoker. Along with the above patient notes Move zhayuschaya lameness. The o time gain indicated the absence of pain stop arterial pulsations.

Your diagnosis, what stage of the disease, plan of examination and treatment?

2. Entered a patient 30 years with complaints of pain in the pas ikronozh muscle activation and intermittent claudication every 2030 met the ditch. Suffering for several years, he was treated repeatedly in a hospital. Of bad habits -
smokes. Despite the PA treatment, the above symptoms of the disease increased. The patient pale, but did not sleep at cham due to pain in the extremities. There is incipient necrosis on the feet. The pulsation of arterial vessels on the foot is absent, on the popliteal, femoral is weak,

What is your diagnosis, stages of the disease, plan of examination and treatment?

3. The patient is 38 years old. the diagnosis of obliterating endarteritis of the left lower extremity was established 3 years ago. Along with the classic symptoms of the disease has not healing lang wa 1 left toe. After repeated conservative treatment (according to Silbert, iontophoresis with novocaine and heparin), the improvement came in a short time. What other treatments can you add and make a treatment plan?

4. Patient 43 years old, diagnosed with endteritis obliterans of the left lower extremity, complicated by gangrene. Disease duration 4 years. Despite the repeated complex conservative treatment, the patient's condition has deteriorated sharply in recent months . The patient is sharply haggard, does not sleep, and even drugs do not relieve pain. Toe dead, the skin Bled naya and celebrated pastoznost shin. The arterial vessels of the left moan do not pulsate. Operation is recommended.

How is amputation performed in such cases?

6. The patient has pain in the calf muscles when walking, cold feet, fatigue, pale skin with a cyanotic tinge, no pulse in the femoral arteries.

What is your diagnosis and treatment?

7. In a patient, the pulsation on the vessels of the lower extremities is reduced. Gangrene of the nail phalanx of the first toe developed. What is your diagnosis and tactics?

8. A 60-year-old patient has a circulatory disorder of the right leg. An arteriogram showed occlusion at the level of the popliteal artery. What is your diagnosis and tactics?

9. A 35-year-old patient was admitted to the hospital with complaints of pain in the calf muscles of the right lower limb. MULTI suffering to years before the disease got frostbite toes and besides hard-core smokers. In the initial period of the disease, he could walk 700 meters without rest or stopping , and now no more than 100 200.

Notes the feeling of chilliness and crawling of the flies on the groan. On examination, the nails of the hoop of the right foot are deformed, the skin is dry. The right foot is cold to the touch. There is no pulsation of the dorsal artery of the left foot, and the posterior tibial arteries on both limbs are weakened. Under the knee and femoral arteries pulsating dissolved well.

What is your diagnosis? Examination and treatment plan.

9. The patient, 35 years old, was admitted to hospital with gangrene end O of the phalanges of the upper limb. Ill for several years, to give the disease la exacerbation during the cold season. During the attack the patient noted a lot of pain in the fingers of the upper extremity, which wasps melt cold. On examination, there is necrosis of the terminal phalanges of the type of dry gangrene. The pulsation in the arteries of the upper limb is preserved.

What is your diagnosis and treatment strategy?

10. A 40-year-old patient has a symptom of claudication, pain in the calf muscles. The pulse on the femoral artery is preserved, on the popliteal artery - absent, on the artery of the foot the pulse is reduced. What is your diagnosis and tactics?

BLOTHERING ATHEROSCLEROSIS .. BUGER'S DISEASE.

The purpose of the lesson:

Based on knowledge about the pathophysiological processes that occur in the patient's body with muscle injuries, vascular pathology of the lower extremities (obliterating atherosclerosis, thrombosis and embolism, Buerger's disease) , the clinic and the principles of treatment of patients with this pathology, be able to make and substantiate a complete clinical diagnosis, conduct a preoperative preparation, and in the future, depending on the clinic and operational findings, to determine the tactics of surgical treatment.

Test questions:

1. Anatomy of the venous system of the lower extremities (superficial, deep and perforating veins, valve apparatus), physiology of venous outflow.
2. The concept of varicose veins, frequency, predisposing and producing factors.
3. Pathological forms of varicose veins and the nature of changes in the walls.
4. Clinical manifestations of the disease.
5. Stages of the course (preclinical, compensation, decompensation).
6. Functional tests to identify the state of the valve apparatus (Troyanov-Trendelenburg, cough Hackenbruch, palpation-percussion Schwartz); patency of deep veins (marching - Delbe-Perthes); the consistency of the communicating veins (Pratt's two-cord test, Sheinis's three-cord test).

7. Instrumental research methods (phlebomanometry, thermometry, capillaroscopy, rheovasography, ultrasound duplex scanning).

8. Doppler ultrasound examination of blood flow in the veins. Ultrasound of veins.

9. Laboratory research methods (coagulation and anti-coagulation blood systems, platelet aggregation and adhesion, analysis of venous blood for oxygen and carbon dioxide content).

10. Complications of varicose veins.

11. Surgical treatment methods: a) phlebectemia - operations of Troyanov-Trendelenburg, Bebkokk, Madelung, Prat; b) turning off the veins - dressing according to Shede-Kocher, Klapp, Sokolov; c) ligation of insolvent perforated veins according to Cockett and Linton.

12. Phlebosclerotherapy, indications and contraindications to it. Endovascular electrocoagulation, endoscopic phlebectomy.

13. Conservative treatment.

14. The concept of thrombophlebitis and phlebothromboeach. Classification by etiology, localization, clinical course.

15. Deep vein thrombophlebitis.

16. Thrombophlebitis of superficial veins.

17. Migratory thrombophlebitis.

18. Complications of thrombophlebitis.

19. Conservative treatment of thrombophlebitis.

20. Surgical treatment of acute thrombophlebitis and indications for it.

21. The concept of post-thrombophlebitis syndrome and its treatment.

22. Prevention of embolic complications

23. Prevention of embolic complications.

Practical skills

1. Inspection.
2. Troyanov-Trendelenburg test.
3. Hackenbruch's cough test.
4. Marching test of Delbe-Perthes.
5. Sheinis' three-bundle test.
6. Examination for thrombophlebitis.
7. Interpretation of these indicators of the blood coagulation system.
8. Interpretation of phlebography data.
9. Interpretation of ultrasound research data.

THEORETICAL PART

PAIN IN THE LIMBS. DISEASES OF THE ARTERIES AND VENES OF THE LIMBS.

Diseases of the vessels of the lower extremities are a group of diseases that include pathology of arteries (acute thrombosis and embolism of the main arteries, obliterating atherosclerosis, obliterating thromboangiitis, nonspecific aortoarteritis, Raynaud's disease) and veins (acute venous thrombosis, Paget-Schroetter syndrome, varicose veins, post-mortem congenital arteriovenous dysplasias) of the upper and lower extremities These diseases are different in etiology and pathogenesis However, they have one of the clinical manifestations in common - pain in the extremities It should be noted that vascular diseases of the extremities are a

very common pathology, which, in the first place, is often encountered family and district doctors, as well as emergency and emergency doctors.

DIFFERENTIAL DIAGNOSTICS OF ACUTE AND CHRONIC DISEASES OF ARTERIES AND VENES OF THE LIMBS.

Pain in the extremities is observed in a variety of diseases Moreover, this pain can come from any part of the limb.However, in some cases, the cause of pain in the extremities are diseases of the internal organs with pain radiating to the arms and legs, as, for example, with myocardial infarction, acute cholecystitis , an attack of renal colic, etc. In other cases, pain in the extremities can be caused by diseases of the spine, arteries and veins, nerves, joints, bones, muscles, etc.

In this regard, they are distinguished (R Hagglin, 1997).

1.Radiating pain:

- diseases of the chest cavity organs (diseases of the heart and large vessels (almost always in the arm and back), lung diseases (in the arm and back).
- diseases of the liver, gallbladder and bile ducts (in the arm, back, rarely - in the legs).
- diseases of the spleen (in the arm, back, rarely - in the legs).
- diseases of the genitourinary system (in the back and legs).
- diseases of the digestive tract (mainly in the back and legs).
- diseases of the spine (arms and legs).
- lesions of the shoulder girdle with neurovascular syndrome (in the hands) ***2. Pain in diseases localized in the spine and extremities :.***
- lesions of the spine.
- diseases of arteries, veins, capillaries, lymphatic vessels.
- nerve diseases.
- joint diseases.
- diseases of the periarterial tissues and subcutaneous tissue.
- bone diseases.
- muscle diseases.
- skin diseases.

We will not dwell on the first group of diseases, since along with radiating pain in the limb, manifestations of the underlying pathology prevail in their clinical picture. Let us consider the second group of diseases in more detail.

Spinal lesions are limited mainly to diseases of the vertebrae, intervertebral joints and other elements connecting individual parts of the spine (intervertebral discs, ligaments). With all diseases of the spine, pain in the region of the spinal column is noted. Along with this, they may be accompanied by pain radiating to the lower extremities. In this case, pain in the limbs is often the leading symptom in the quality of the so-called radicular pain. The latter are characterized by segmental spread, increased by coughing and sneezing, increased or decreased after certain movements of the spine, increased at night.

The most common diseases of the spine include deforming spondylosis, which is characterized by changes in the intervertebral discs and vertebral bodies, and spondolarthrosis, caused by degenerative, non-inflammatory changes in the small intervertebral joints. Both of these processes often develop at the same time.

In *spondylosis deformans* pain, as a rule, are vague in nature and are most intense after a long period of rest, ie, with a "run-up", as well as after prolonged immobility of the joints (at night, early in the morning). There is an increase in pain with injuries, prolonged bed rest, cooling and infections. Arthrosis of the shoulder joint is especially typical in myocardial infarction. In contrast to inflammatory arthritis, complete immobility (with the exception of the hip joint) and pronounced restrictions on mobility are not observed. With pain in the hands, one should think about the possibility of degenerative changes in the cervical spine. The main diagnostic method is spinal x-ray (narrowing of the cracks of small joints with sclerosis of the

articular edges and atrophic deformity, narrowing of the intervertebral discs with sclerosis of the intervertebral plates).

Ankylosing spondylarthritis (*ankylosing* spondylitis - Strumpel - Pierre Marie) is characterized by intense pain, often at night and especially severe after a short period of immobility - painful getting up. Pain is felt mainly in the spine, often radiating to the sacrum and legs. Therefore, at the beginning of the disease, this disease is often mistaken for sciatica. In addition, early immobility of the lumbar and cervical spine, the typical position of the patient's body, are characteristic. Restriction of the respiratory mobility of the chest is also noted. The diagnosis is based on a typical clinical picture, especially with early onset of immobility, as well as on x-ray examination (damage to both sacroiliac joints, etc.).

Focal spondylosis can be observed in various infectious diseases, such as tuberculosis, brucellosis, typhoid fever.

A herniated disc is characterized by pain in the lumbar region and lower extremities, previously suffered more or less prolonged lumbodynia, usually occurring after "unsuccessful" movement, weight lifting ("lumbago") or other physical activity. The pain increases significantly with movement, especially when turning and bending the trunk, as well as with all movements accompanied by raising the legs. Pain can increase with coughing, sneezing, laughing. Diagnostic methods - MRI, radiography.

Neurovascular syndrome with lesions of the shoulder girdle, even with congenital changes, usually occurs at the age of 30-50 years.Its clinical manifestations are caused by compression of the arteries and nerves in certain positions of the arm.These pathological conditions are characterized by prolonged pain in the shoulder, the entire arm, or in the hand. in some cases, patients experience only unpleasant sensations in the hand, manifested by discomfort and the inability to find a suitable more comfortable position.Most patients complain of a feeling of numbness, and more often only the hand or forearm is affected. Quite often, paresthesias, redness and swelling at the site of pain are noted, as well as muscle weakness.

There can be various reasons for the neurovascular syndrome.

- *costal - clavicular syndrome* - narrowing of the gap between the clavicle and the 1st rib, compression occurs when the shoulder is abducted downward and posteriorly.

- cervical rib *syndrome - the* presence of a cervical rib is established radiographically.

- *excessive abduction syndrome - a* sharp decrease in the pulsation of the radial artery and blood flow with the maximum abduction of the arm.

- *syndrome of the scalene muscle* - increased pain and disappearance of the pulse with a sharp turn of the head in the opposite direction or with a deep breath.

- *scapular-rib syndrome* - pain emanating from the shoulder blades and radiating to the back of the head, lower part of the head, shoulder girdle, shoulder, forearm.

- *Putnam's disease* - sharp pains in the area of both forearms that occur in the second half of the night in women over 30 years old, intensifying before menstruation and accompanied by a feeling of immobility, limitation of movement. The pain decreases when hands are lowered or rubbed.

In cervical-costal syndrome, the main diagnostic method is radiography, in other cases - anamnesis data and an objective examination.

Pain radiating to the extremities is observed **in diseases of the spinal cord** - dorsal tabes and tumors of the spinal cord The main diagnostic method is magnetic resonance imaging.

Pain in the extremities can also be observed **with neuralgia.They** are characterized by the sudden appearance of pain, respectively, the area of distribution of peripheral nerves. Motor and sensory phenomena of prolapse are absent.In men, the disease occurs more often than in women.When neuralgia, pains of a tearing or pulling character of varying duration are noted - seconds, minutes Characteristically complete the absence of pain or a decrease in their intensity between attacks Fundamental in the diagnosis is sensitivity to pressure on the affected nerves, especially at the Balle points - the places where nerves exit from the bone canals, fascia, etc.

Pain in the limbs can be caused **by joint lesions,** which are observed in various diseases.

1 *In rheumatism* - rheumatic arthritis.

2 *For degenerative joint lesions* - arthrosis, coxarthrosis.

3 *In metabolic diseases* - gout, alcaptonuria, lipoidosis (Schüller-Christian disease), lipoid gout (Buerger's disease), Gaucher disease.

4 *In collagen diseases* - rheumatoid arthritis, systemic lupus erythematosus, periarteritis nodosa, scleroderma, dermatomyositis.

5 *In infectious diseases* (especially viral) - hepatitis, scarlet fever, tuberculosis, brucellosis, gonorrhea, Reiter's syndrome.

Common to all these diseases is local damage to the joints (swelling, hyperemia, soreness) Moreover, in some cases, small joints are affected, in others - large ones.However, these manifestations are almost never leading.The main symptoms of these pathologies always come to the fore.

In the **pathology of bones,** pain in the extremities is observed relatively rarely. Apparently, this is one of the reasons for the untimely detection of initial bone lesions. In the differential diagnosis of bone lesions, one should take into account, first of all, age and radiological data.

Limited bone lesions are observed in bone tumors, bone tuberculosis, osteomyelitis, and multiple *lesions are* observed in inflammatory lesions (osteomyelitis, tuberculosis, syphilis, fungal lesions, sarcoidosis), tumors (myeloma, primary bone marrow tumors), bone metastases (hemogranulomatosis)), storage diseases (Gaucher disease, Niemann-Pick disease, Hand-Scheller-Christian disease) It should be noted that with multiple bone foci and diffuse bone changes (osteoporosis, osteosclerosis), especially in aged patients, it is primarily necessary think not about a local disease, but about secondary bone changes due to some general disease.

Pain in the extremity is the leading symptom of various **diseases of the arteries and veins of the extremities** . First of all, they should be attributed.

- *acute arterial obstruction* (acute thrombosis and embolism of the main arteries).

- *acute venous thrombosis* (acute superficial vein thrombophlebitis, acute deep vein thrombosis, Paget-Schrötter syndrome).

- *obliterating arterial lesions* (obliterating atherosclerosis, thromboangiitis obliterans, nonspecific aortoarteritis, Raynaud's disease).

- *chronic venous insufficiency* (varicose veins of the lower extremities, post-thrombophlebitic syndrome).

In this case, both **acute** and **chronic pain** in the extremities can be observed .

Acute pain in the hands is most often caused by Paget-Schrötter syndrome, in the legs - by acute arterial obstruction and acute venous thrombosis.

Paget-Schrötter syndrome is characterized by acute bursting pain in the arm, edema, cyanosis of the skin, a feeling of heaviness in the limb, and enlargement of the saphenous veins.

More often, unilateral lesion is noted Most often, Paget-Schrötter syndrome occurs after physical exertion or with strong tension of the muscles of the shoulder girdle.In some cases, it becomes necessary to carry out differential diagnostics with lymphostasis of the upper extremities, metastases of malignant tumors of the chest, compressing veins In addition, in recent years, thrombosis of the subclavian and axillary veins began to occur as complications of subclavian vein catheterization, which is widely used for long-term intravenous infusions.

The main diagnostic methods are Doppler ultrasound. and radiopaque phlebography, less often CT and chest x-ray.

For **acute arterial obstruction** is characterized by severe, persistent pain in the affected limb, do not respond to use of narcotic analgesics, severe pallor, lack of pulsations distal to the occlusion, limb numbness and muscle weakness. In the future, violations of tactile, pain, temperature and deep sensitivity join, active movements are lost. With embolism of the aortic bifurcation, along with the listed symptoms, severe pain in both legs and lower abdomen is noted. With embolism of the main arteries, clinical manifestations are more pronounced than

with thrombosis. It should be noted that embolism is more common between the ages of 40 and 60.

The most common causes of arterial thrombosis are cardiovascular diseases, atherosclerosis obliterans, thromboangiitis, diabetes mellitus, arterial injuries, soft tissue bruises, dislocations and fractures of limb bones, compression of the vascular bundle by a tumor or hematoma, as well as various surgical interventions - angiographic studies, endovascular interventions , reconstructive vascular surgery and other interventional procedures. Thrombosis is often observed against the background of certain hematological (erythrocytosis) and infectious (typhus) diseases.

The causes of arterial embolism are usually: myocardial infarction, especially complicated by severe heart rhythm disturbances, acute or chronic left ventricular aneurysm; rheumatic combined heart disease with a predominance of stenosis, atrial fibrillation, subacute aseptic endocarditis, congenital heart defects, aneurysms of the abdominal aorta and large main arteries.

The main diagnostic methods are Doppler ultrasound and angiography.

Acute thrombophlebitis of the superficial veins of the lower extremities is manifested by pain, induration, edema, and flushing of the skin along the inflamed vein Palpation of this zone is painful Swelling of the tissues is usually limited to the area of the lesion of the vein. This pathology is more often observed in varicose veins.

Recognition of acute thrombophlebitis of the superficial veins of the lower extremities does not present any particular difficulties.However, if it is accompanied by lymphangitis and lymphadenitis, it must be differentiated from erysipelas, cellulitis and phlegmon of the subcutaneous tissue.

Erysipelas is characterized by the formation of a small red spot at first, which then gradually increases. The boundaries between healthy and affected skin are sharply delineated, and the area of the affected skin looks as if raised above the healthy one.

With ***subcutaneous phlegmon*** , significant swelling and redness is locally determined with the formation of edema in the surrounding tissues.In this case, there is usually a pronounced general reaction in the form of chills and high body temperature There are severe lymphangitis and an increase in regional lymph nodes.

Acute form of deep vein thrombosis is characterized by sharp pain in the limb (or extremities), rapidly increasing edema of the foot and distal parts of the leg, cyanosis of the skin, a feeling of distention in the lower leg The level of edema of the limb always corresponds to the level of thrombosis Peripheral arterial pulsation, as a rule, is not impaired. the temperature of the affected limb is 1.5-2 ° C compared to the healthy one. In the future, the expansion of the superficial veins of the limbs is added.

With white painful phlegmas, there is a sharp pain, cold snap, numbness of the limb ***Swelling*** rapidly increases, finger movements become limited, sensitivity and temperature decrease.

Blue (blue) phlegmasia is characterized by a pronounced diffuse edema of the limb, spreading to the genitals, buttocks, and the anterior abdominal wall. The skin is bluish in color, and the distal parts are purple or even black. Within 1-3 days, the skin and subcutaneous tissue of the back and lower legs appear hemorrhagic rashes, there is a detachment of the epidermis, bubbles form.

Risk factors for the development of thrombosis are elderly and senile age, surgical interventions, especially orthopedic, trauma, cancer, acute myocardial infarction, infections, acute and chronic heart and respiratory failure, stroke, arterial hypertension, obesity, the presence of varicose veins, pregnancy, childbirth ...

For deep vein thrombosis, positive symptoms of Moses, Lovenberg, Homans, Payr, Pratt, Sperling are characteristic. However, the leading role in the diagnosis belongs to ultrasound Doppler ultrasound, magnetic resonance phlebography and X-ray contrast phlebography.

Most often, acute deep vein thrombosis of the lower extremities has to be differentiated with arterial thrombosis and embolism, neuritis of the femoral and sciatic nerves, lymphostasis.

Acute disturbance of blood flow in the main arteries is characterized by pain in the affected limb, which is difficult to remove with narcotics.The skin of the limb is pale, cold to the touch, there is no edema In this case, there is a violation of all types of sensitivity The pain does not decrease with an elevated position of the limb In acute thrombophlebitis, the pain decreases with an elevated position of the limb and increases when the limb is lowered or in a standing position.

With neuritis of the femoral or sciatic nerve, pain is localized on the anterior or posterior surface of the extremity along the corresponding nerve trunks The occurrence of ***lymphostasis of the*** lower extremities is usually associated with an inflammatory process and lymphadenitis There is no pain syndrome and a network of dilated superficial veins The same signs are characterized by edema of the lower extremities associated with the development of cardiac and renal failure.

Chronic pain in the upper limbs is most often caused by Raynaud's disease, in the upper (more often) and lower limbs - nonspecific aortoarteritis, in the lower limbs - obliterating arterial lesions, varicose veins, post-thrombophlebitis syndrome (Table 1.2.).

For **Raynaud's disease** characterized by a symmetrical and bilateral lesion of the upper extremities More common in women younger noted spasm, cold end phalanges, most II and III fingers which become pale, cold to the touch and insensitive After some time spasm may be replaced by vasodilatation, whereby occurs redness of the skin and warm fingers Diagnosis is mainly based on clinical findings.

For **nonspecific aortoarteritis** characteristic defeat both the upper (more often) and lower extremities The acute phase of the disease is more common in young adults Moreover, the disease occurs in women under the age of 30 years The most characteristic clinical manifestation is the absence of a pulse at the radial artery and its asymmetry.

There are no specific diagnostic methods. Doppler ultrasound and angiography can only establish the nature of blood flow, localization and extent of the lesion.

There are three degrees of acute arterial ischemia (V.S.Saveliev).

I degree :.

I A - numbness, coldness, paresthesia of the limb ,.

IB - the same signs, but with pain syndrome.

II degree :.

PA - impaired sensitivity and active movements in the joints of the limb, paresthesia.

II B - complete plegia of the limb.

III degree :.

III A - the symptoms listed above with subfascial edema of the limb.

III B - ischemic contracture, beginning tissue necrosis.

The clinical picture. Symptoms of acute arterial obstruction are most pronounced in embolism The onset of the disease is characterized by the appearance of sudden severe, acute and constant pain that captures the entire distal part of the limb.In its origin, spasm of both the main artery and collaterals is of paramount importance. After 2-4 hours, the spasm decreases, and the intensity of pain decreases slightly. A feeling of numbness, pallor and coldness of the limb joins the pain, and then muscle strength decreases, active movements are lost.

In some cases, pain initially appears in the distal parts of the limb, then spreading in the proximal direction. Severe pallor of the skin also first appears in the distal parts, and then spreads to the proximal segments.Disorders of tactile, pain, temperature and deep sensitivity may appear earlier than movement disorders.

OBLITERATING ATHEROSCLEROSIS is a chronic lesion of arteries of large and medium caliber, characterized by primary deposition in their inner membrane (intima) of plasma

lipoproteins and lipids contained in them.This leads to complex structural and cellular changes in the intima, which culminate in the proliferation of connective tissue with the formation of fibrous plaques.

The etiology of atherosclerosis is still not fully known. At the same time, a number of risk factors have been identified that contribute to the development of atherosclerosis. According to the degree of probability of exposure, these factors can be divided into three groups.

Identified risk factors:

- atherogenic dyslipoproteinemia.
- high concentration of cholesterol in blood plasma.
- high concentration of triglycerides in blood plasma.
- high concentration of intermediate density lipoproteins in blood plasma.
- low concentration | 3 - lipoprotein cholesterol in blood plasma ,.
- arterial hypertension (hypertension).
- smoking.

Possible risk factors:

- diabetes mellitus or decreased glucose tolerance.
- emotional stress and individual characteristics of personality behavior, - hereditary factors (this or that type of lipoprotein metabolism disorder is transmitted, manifested mainly by an increase in the level of lipoproteins in the blood).

Estimated risk factors:

- obesity ,.
- insufficient physical activity.

Pathogenesis. Atherosclerotic vascular lesions take place in the following stages.

- stage of lipid spots.
- the stage of atherosclerotic plaque formation.
- stage of complications.

Recently, there is also a dolipid stage, in which swelling of the intima is observed. The severity of these changes increases as more and more plasma lipoproteins penetrate into the subendothelial space. Low density lipoproteins are the most atherogenic.

The main changes develop in the intima of the arteries There are the following pathological changes in the intima - fatty strips, fibrous plaques and complicated lesions - ulceration of plaques, the formation of blood clots.The earliest manifestation of atherosclerosis is fatty strips, characterized by focal accumulation in the intima of macrophages filled with lipids (smooth muscle cells cells) and fibrous tissue.

The formation of an atherosclerotic plaque begins with the accumulation of lipids in the intima - the stage of lipoidosis. Around the foci of lipoidosis, the proliferation of intima and smooth muscle fibers develops, young connective tissue appears, the maturation of which leads to the formation of fibrous atherosclerotic plaque - the stage of liposclerosis.

Fibrous atherosclerotic plaque rises above the surface of the intima and represents its thickening It has a dome-shaped shape, dense consistency, protrudes into the lumen of the artery and narrows it The main components of the plaque are extracellular fat located in the central part, the remains of necrotic cells (detritus) covered with fibromuscular a layer or a canopy containing a large number of smooth muscle cells, macrophages and collagens. At the same time, the thickness of the plaque significantly exceeds the normal thickness of the intima. The composition of extracellular fat of the plaques resembles plasma lipoproteins.

Abundant accumulation of lipids disrupts blood circulation in the tissue membrane of the plaque, which leads to the development of necrosis of the cells included in its structure.Hemorrhage occurs in the thickness of the plaque, cavities filled with amorphous fat and tissue detritus appear.This is often accompanied by the formation of ulceration on the surface of the intima and atheromatous masses, as well as parietal thrombotic overlays are rejected into the lumen of the vessel. Getting into the distal vascular bed with the blood flow, they can cause microembolism.In the future, calcium salts are deposited in the tissue elements of

the plaque (atherocalcinosis). The listed processes proceed in waves and lead to thrombosis and obliteration of the vessel.

The most frequent localization of atherosclerotic lesions are the sites of division of the main arteries, the brachiocephalic trunk, the mouth of the vertebral arteries, bifurcation of the aorta, common carotid, common iliac, femoral and popliteal arteries. blood flow Here, there is a certain slowdown and separation of the blood flow along the arterial branches, as well as the deviation of the main blood flow from a rectilinear trajectory As a result of this, vortices are formed that damage the intima and contribute to the formation of plaques In this regard, atherosclerotic damage to the walls of the arteries can be considered as a chronic regenerative process in response to chronic trauma of the intima by turbulent and direct blood flow.

The pathological process is most often localized in the aorta (usually distal to the renal arteries) and the iliac arteries or the femoral and popliteal arteries, that is, in the large and medium arteries of the elastic type Moreover, one or another artery is not affected totally, but in separate areas, more often the bifurcation region is involved in the process, branches and narrowing Narrowing and obliteration of these arteries cause severe ischemia of the extremities. Approximately 1/3 of patients are affected by the aorto-iliac (Leriche syndrome) segment, and in 2/3 of patients - the femoral-popliteal segment.

Classification. Until recently, the most common classification of the degree of ischemia was **the Fontaine classification** (1954), which includes 4 degrees.

I - full compensation (symptoms of chilliness, fatigue, paresthesia).

II - circulatory failure during functional stress (the leading symptom is intermittent claudication).

III - arterial insufficiency at rest (the main symptom is constant night pain).

IV - severe tissue destruction of the distal limb (ulcers, necrosis, gangrene).

In the CIS countries, the AB Pokrovsky classification was adopted (1979), which somewhat supplemented and refined the Fontaine classification. In the literature, it is better known as the **Fontaine- Pokrovsky** classification **.**

According to the **Russian consensus** in 2001, the **classification of the degree of ischemia by Fontaine Pokrovsky** was supplemented with some objective indicators (Table 1 3).

Table 1 3.

Improved classification of Fontaine- Pokrovsky (2001)

Power	**Symptoms**	
1	intermittent claudication that occurs when walking more than 1000 m	Ankle-brachial index (ABI), normal treadmill test **
2a	Intermittent claudication that occurs when walking 200 m to 1000 m	Resting ABI 0.7-0.9, patient walks more than 200 m on standard treadmill test, and ABI recovery time to baseline after treadmill test is less than 15.5 minutes
26	Intermittent claudication that occurs when walking up to 200 m	Resting ABI less than 0.7, patient walking less than 200 m with standard treadmill test and / or ABI recovery time to baseline after treadmill test greater than 15.5 minutes
3		Art, a collateral type of blood flow is recorded along the arteries of the foot or arteries are not visualized at all, finger pressure < 30 mm Hg
4	Stage of trophic disorders	Art, a collateral type of blood flow is recorded along the arteries of the foot or

		arteries are not visualized at all, finger pressure < 30 mm Hg

PRACTICAL PART

Thematic tests

1

1. Specify the most dangerous complication of deep vein thrombosis of the lower extremities:
a) trophic ulcer of the leg;
* b) pulmonary embolism;
c) phlebitis of varicose veins;
d) obliteration of deep veins;
e) elephantiasis

2. Indicate the factors that do not contribute to the improvement of venous blood flow in the lower extremities in the postoperative period:
a) early getting up;
b) elastic compression of the legs;
c) elevated position of the limbs;
* d) long and strict bed rest;
e) contraction of the calf muscles.

3. A young woman on a background of uncomplicated pregnancy proceeds STI appeared varicose veins on the right leg. Hirur by God revealed that the deep veins of the right shin passable, having etsya insufficient perforating veins in the lower third. When Pal patsii veins soft, painless, skin over them is not changed. Based on this clinical situation, the patient should be recommended
a) planned surgical treatment before childbirth
* b) wearing elastic bandages, radical phlebectomy in the postpartum period
c) sclerotherapy sessions before - and after childbirth
d) Troyanov-Trendelenburg operation before childbirth, radical phlebectomy after childbirth
e) treatment with nonspecific anti-inflammatory drugs (butadion, troxevasin, escuzan)

4. The development of pronounced edema of one limb is observed in:
a) varicose veins of the lower limbs;
* b) acute deep vein thrombosis of the lower extremities;
c) obliterating atherosclerosis of the vessels of the lower extremities;
d) arterial thrombosis;
e) heart failure.

5. Varicose veins of the lower extremities are characterized by: 1) expansion of the superficial veins of the lower extremities; 2) heaviness in the legs; 3) symptom of intermittent claudication; 4) the formation of trophic ulcers on the toes; 5) chilliness of the limbs. Choose the correct combination of answers:
a) 2, 5;
b) 2, 3, 4;
*at 12;

d) everything is correct;
e) everything is wrong.

6. Chronic venous insufficiency is characterized by: 1) cold extremity; 2) heaviness in the legs; 3) intermittent claudication; 4) limb edema; 5) lack of pulse in the arteries of the dorsum of the foot. Choose the correct combination of answers:
a) everything is correct;
b) 1, 2, 3;
c) 2, 4, 5;
* d) 2, 4;
e) 4, 5.

7. Acute deep vein thrombosis is characterized by: 1) pain in the affected limb; 2) cyanosis of the skin; 3) hyperemia along the saphenous vein; 4) sharp swelling of the limb; 5) intermittent claudication. Choose the correct combination of answers:
* a) 1, 2, 4;
b) 1, 2, 3;
c) 2, 3, 5;
d) 3, 4, 5;
e) everything is correct.

8. Name the symptoms of acute thrombosis of the ilio-femoral venous segment: 1) hyperemia of the skin of the thigh in the area of veins; 2) swelling of the entire limb; 3) bursting pain in the hip; 4) an increase in the volume of the thigh and lower leg; 5) pallor of the skin of the thigh. Choose the correct combination of answers:
a) 1, 2;
b) 2;
* c) 2, 3, 4;
d) 3, 4;
e) 3, 4, 5.

9. Name the most modern, most informative method used for topical diagnosis of inferior vena cava thrombosis:
a) radioindication with labeled fibrinogen;
* b) ileocavagraphy;
c) distal ascending functional phlebography;
d) sphygmography;
e) retrograde femoral phlebography.

10. The development of thrombophlebitis is promoted by: 1) slowing down the blood flow; 2) hypocoagulation; 3) hypercoagudation; 4) increased fibrinolytic activity of the blood; 5) damage to the intima of blood vessels. Choose the correct combination of answers:
a) 2, 4;
* b) 1, 3, 5;
c) 3, 4, 5;
d) 4, 5;
e) everything is correct.

1. Leriche's syndrome is:
a) brachiocephalic nonspecific arteritis;
* b) atherosclerotic occlusion of the bifurcation of the abdominal aorta;

c) capillaropathy of the distal extremities;
d) migratory thromboangiitis;
e) occlusion of the inferior vena cava.

2. Obliterating atherosclerosis of the lower extremities is characterized by:
a) flying pains in the joints of the extremities;
* b) intermittent claudication;
c) fulminant necrosis of the foot;
d) the occurrence of trophic ulcers in the knee joints;
e) concomitant deep vein thrombophlebitis.

3. Chronic arterial insufficiency develops when: 1) thromboangiitis obliterans; 2) post-thrombophlebitic syndrome; 3) angiospasm; 4) arterial aneurysm; 5) obliterating atherosclerosis. Choose the correct combination of answers:
a) 2, 3;
b) 3, 4:
* c) 1, 5;
d) 2, 3, 4;
e) 2, 5.

4. What drugs are pathogenetically justified for the treatment of patients with chronic arterial insufficiency? 1) desensitizing; 2) vasoconstrictor agents; 3) vasodilators; 4) antiaggregants; 5) antibiotics. Choose the correct combination of answers:
* a) 1, 3, 4;
b) 1, 3, 5;
c) 2, 4, 5;
d) 3, 4, 5;
e) everything is correct.

5. The most perfect instrument for embolectomy from the main artery is:
a) Volmar's vascular ring;
b) vacuum - suction;
* c) Fogarty balloon catheter;
d) Dormia catheter;
e) fenestrated forceps.

6. Arterial embolism of the vessels of the lower extremities is not characterized by:
a) no pulsation;
b) parasthesia;
* c) trophic ulcers of the lower extremity;
d) pain;
e) pallor of the skin.

7. What can not be expected with arterial aneurysm:
a) rupture;
b) compression of adjacent organs;
c) pain in acute rupture;
d) arterial thromboembolism;
* e) spontaneous healing.

8. In acute ischemia of the extremity IIb degree, which developed as a result of femoral artery embolism, it is uncharacteristic:

a) pain in the extremity;
b) "marble" pattern of the skin;
c) limb cooling;
d) lack of active movements in the joints of the limb;
* e) muscle contracture.

SUBJECT : ACUTE THROMBOSIS AND EMBOLISM ARTERIES AND .

Purpose of the lesson: To study etiology, diagnostics and differential diagnostics, research methods and treatment tactics (determination of the pulsation of the main arteries). Acute arterial thrombosis and embolism. Clinic, conservative and surgical treatment.

What the student SHOULD KNOW:

1. Causes of acute arterial obstruction.
2. Clinic of diseases in which acute arterial obstruction occurs.
3. Diagnostics of thrombosis and embolism of arterial vessels
4. Conduct differential diagnosis of arterial and venous obstruction.
5. Modern instrumental methods for examining the patency of arterial vessels.
6. Conservative and surgical methods for the treatment of acute arterial obstruction.

Having studied the topic, the student MUST BE ABLE TO:

1. Correctly conduct a survey of patients with acute arterial obstruction.
2. Conduct an objective examination of patients.
3. Bandaging of the lower extremities with elastic jersey.
4. Be able to palpate and auscultate peripheral arteries.
5. Analyze the results of objective and laboratory examination methods.
6. Appoint an additional plan of examination of patients.
7. Based on the data obtained about the patient, formulate a clinical diagnosis.
8. Conduct differential diagnostics.
9. Determine s tactics and the amount of therapeutic measures.

THEORETICAL PART

Arterial thrombosis and embolism.

Narrowing or obstruction of the vessel lumen with a blood clot or an embolus leads to acute arterial obstruction, accompanied by ischemia of tissues deprived of blood supply.

Thrombosis is a pathological condition characterized by the formation of a blood clot in one or another part of the vascular bed.

Etiology and pathogenesis. Indispensable conditions for the occurrence of arterial thrombosis are a violation of the integrity of the vascular wall, a change in the hemostatic system and a slowdown in blood flow. This explains the high incidence of thrombosis in persons suffering from cardiovascular diseases, obliterating atherosclerosis, thromboangiitis, and diabetes mellitus. Often, the development of thrombosis is facilitated by damage to the walls of the arteries with soft tissue bruises, dislocations and fractures of the limb bones, compression of the vascular bundle by a tumor or hematoma. Acute arterial thrombosis may be preceded by angiographic studies, endovascular interventions, vascular reconstructive surgeries, and other interventional procedures. Thrombosis also occurs against the background of some hematological (erythrocytosis) and infectious (typhus) diseases.

In all these cases, the response to damage to the endothelium of the vascular wall is adhesion and subsequent aggregation of platelets. The resulting aggregates tend to grow further,

which is associated with the effect of physiologically active substances, cytokines released from endothelial cells, macrophages, neutrophilic leukocytes and platelets. The intensity of the formation of platelet aggregates also depends on the ability of the endothelium to produce inhibitors of aggregation, in particular nitric oxide (NO), prostacyclin. The platelet factors and biologically active substances released from the platelets not only promote platelet aggregation, but also lead to the activation of the blood coagulation system, a decrease in its fibrinolytic activity. As a result, fibrin filaments are adsorbed on the surface of the aggregate, forming a reticular structure, which, retaining the blood cells, contributes to the formation of a blood clot - a thrombus. With a significant inhibition of the lytic link of the hemostasis system, thrombosis can become widespread.

Embolism is a blockage of the lumen of a blood vessel by an embolus, which is usually represented by a part of a thrombus or plaque that has "detached" from the vascular wall, migrating with the bloodstream through the bloodstream.

Etiology and pathogenesis. In 92-95 *% of* patients, the causes of arterial embolism are heart disease and primarily myocardial infarction (especially in the first 2-3 weeks of the disease), complicated by severe heart rhythm disturbances, acute or chronic left ventricular aneurysm.

The cause of embolism may be intra-atrial thrombosis, which is often observed in rheumatic combined mitral heart disease with a predominance of stenosis, atrial fibrillation. Arterial embolism also occurs with subacute septic endocarditis and congenital heart disease.

Clinical presentation and diagnosis. The symptoms of acute arterial obstruction are most pronounced with embolism. The onset of the disease is characterized by the appearance of sudden pain in the affected limb. In its origin, spasm is of paramount importance - both of the main artery and collaterals. After 2-4 hours, the spasm decreases, and the intensity of the pain decreases slightly. The pain is joined by a feeling of numbness, coldness and severe weakness in the limb.

The skin of the affected limb acquires a deathly pale color, which is later replaced by a characteristic marbling. The veins are desolate, in the course of them depressions are formed (a symptom of a groove or a dry river bed). Pulsation of the artery distal to the localization of the embolus is absent, above the embolus it is usually increased. The skin temperature is significantly reduced, especially in the distal extremities. At the same time, pain and tactile sensitivity is disturbed, and at first superficial, and then deep sensitivity decreases. Patients with severe ischemic disorders often develop complete anesthesia. Limb function is impaired up to flaccid paralysis. In severe cases, there is a sharp restriction of passive movements in the joints, sometimes muscle contracture develops. Subfascial muscle edema causes painful sensations experienced by the patient on palpation. With the progression of local symptoms, the general condition of patients also worsens.

The level of occlusion, the intensity of arterial spasm, the degree of obstruction of the lumen of the artery with an embolus, the features of collateral circulation and the size of the continued thrombus have a significant effect on the clinical picture of the disease. Extremely severe clinical symptoms are observed with embolism of the aortic bifurcation. It is manifested by sudden intense pain in the lower extremities and hypogastric region, radiating to the lumbar region and perineum. "Marble" skin pattern within the next 1-2 hours extends to the skin of the buttocks and the lower sections of the anterior abdominal wall. Due to impaired blood circulation in the pelvic organs, dysuric phenomena and tenesmus are possible. Ripple in the femoral arteries is not detected, and the zone of impaired sensitivity reaches the lower abdomen. The motor function of the limb quickly disappears, muscle contracture develops, and irreversible changes in the tissues occur.

The clinical picture of acute arterial thrombosis resembles that of embolism, but it is characterized by the gradual development of symptoms. This especially applies to patients suffering from obliterating diseases of the peripheral arteries, in whom vascular thrombosis often

occurs against the background of a developed network of collaterals. Only as thrombosis progresses, there are pronounced symptoms of persistent ischemia of the affected limb.

There are three degrees of ischemia of the affected limb in acute arterial insufficiency, each of which is divided into two forms (V.S.Saveliev). With ischemia of the IA degree, a feeling of numbness and coldness, paresthesia appears; with grade 1B, pain is added. Ischemia of the II degree is characterized by disturbances of sensitivity and active movements in the joints of the extremities from paresis (PA degree) to paraplegia (PB degree). Grade III ischemia is characterized by incipient necrosis, as evidenced by subfascial edema in grade III and muscle contracture in grade III ischemia. The end result of ischemia can be limb gangrene.

Embolism and thrombosis of mesenteric vessels

Acute disturbance of blood circulation in the mesenteric vessels develops as a result of arterial embolism or thrombosis of the mesenteric arteries and veins. The most often affected is the upper (90 %), less often - the lower mesenteric artery (10 %).

Etiology. The main cause of embolism is heart disease, complicated by the formation of blood clots (rheumatic defects, rhythm disturbances, myocardial infarction, cardiosclerosis, endocarditis). The source of embolism can be atherosclerotic plaques of the aorta, as well as thrombotic masses of the aneurysmal sac. Changes in the vascular wall (atherosclerosis or arteritis) predispose to the occurrence of thrombosis of the mesenteric arteries. The development of mesenteric vein thrombosis is possible in the presence of purulent processes in the abdominal cavity (pylephlebitis), portal hypertension, accompanied by stagnation of blood in the portal vein, with sepsis, trauma, compression of blood vessels by neoplasms. The disease is equally common in men and women, it develops mainly in middle and old age.

Pathological picture. Due to the violation of mesenteric blood circulation, ischemia of the intestinal wall occurs, in which severe destructive-necrotic changes develop, ranging from ischemic to hemorrhagic infarction. When a small arterial branch is occluded, only a limited section of the intestine suffers; when the main trunk is blocked, all intestinal loops in the zone of impaired blood supply die.

Clinical presentation and diagnosis. Thrombosis and embolism of mesenteric vessels have similar clinical symptoms. The disease, as a rule, begins suddenly with an attack of intense abdominal pain, the localization of which depends on the level of vessel occlusion. With the defeat of the main trunk of the superior mesenteric artery, pain is localized in the epigastric or peri-umbilical region, or spread throughout the abdomen. With embolism of the ilio-colonic artery, which is involved in the blood supply to the terminal ileum and the ileocecal angle, pain often occurs in the right iliac region, simulating a picture of acute appendicitis. For thrombosis and embolism of the lower mesenteric artery, pain in the left lower quadrant of the abdomen is characteristic. The pain is often constant, sometimes cramping, resembling those with intestinal obstruction. For fear of their strengthening, patients try to lie still, on their backs, bending their legs at the knee and hip joints.

Nausea and vomiting are observed already in the first hours of the disease in 50% of patients. Subsequently, these symptoms become permanent. Frequent loose stools appear in 20% of patients, often in the feces there is an admixture of unchanged blood. At the onset of the disease, the pulse is usually quickened, the tongue is moist, the abdomen is usually soft, not swollen, and slightly painful.

As the disease progresses, a picture of paralytic intestinal obstruction develops, characterized by bloating, lack of peristalsis, stool and gas retention, and frequent vomiting. The tongue becomes dry, the abdomen is painful, there is tension in the muscles of the abdominal wall. With a digital examination of the rectum, traces of blood are sometimes found on the glove. The end of the disease is peritonitis.

Acute disorders of mesenteric circulation are characterized by pronounced leukocytosis (20-30-10 ^{6}/ l), which is rare in other acute surgical diseases of the abdominal organs.

X-ray examination of the abdominal cavity gives certain information only in the last stage of the pathological process, when there is paralytic intestinal obstruction. When the main trunk of the superior mesenteric artery is occluded, X-ray examination reveals swollen loops of the small and right half of the large intestine. At the same time, horizontal fluid levels are determined in the lumen of the small intestine, which, unlike levels with mechanical intestinal obstruction, do not move from one knee of the intestine to another.

It is necessary to carry out patients with lateroscopy, paying attention to changes in the X-ray picture when turning the body: swollen intestinal loops in patients with mechanical intestinal obstruction remain fixed when turning the body from one side to another; in patients with paralytic intestinal obstruction caused by acute thrombosis or mesenteric embolism, they easily move to the overlying parts of the abdomen.

Selective angiography has the greatest diagnostic value. A reliable sign of mesenteric artery thrombosis is the absence of contrasting of the main arterial trunk or its branches on angiograms; venous thrombosis is characterized by the absence of the venous phase and prolongation of the arterial phase. In connection with the lengthening of the capillary phase of the study, a longer and more intense contrasting of the intestinal wall is determined.

Differential diagnostics. Acute disturbance of mesenteric circulation should be differentiated from acute surgical diseases of the abdominal organs, in particular from mechanical intestinal obstruction, perforated gastric and duodenal ulcers, acute pancreatitis, acute cholecystitis and acute appendicitis. When conducting a differential diagnosis, laparoscopy can be of great help.

Sometimes a clinical picture similar to acute obstruction of mesenteric vessels is observed in myocardial infarction with atypical localization of pain. In these cases, a carefully collected anamnesis and data from electrocardiographic studies acquire diagnostic value.

Treatment. Only surgical treatment is effective, which can save the patient's life. In the absence of intestinal necrosis, a reconstructive operation on the mesenteric vessels can be performed (embolectomy, endarterectomy, resection of the superior mesenteric artery with prosthetics or implantation of its stump into the aorta). With gangrene of the intestine, its resection is indicated within the limits of healthy tissues. In some cases, it is advisable to combine resection with vascular reconstructive surgery.

Forecast. Postoperative mortality reaches almost 80 %, which is caused not only by the difficulties of diagnosis and the severity of the operation, but also by the presence of an underlying disease that led to an acute disturbance of the mesenteric circulation.

PRACTICAL PART

Thematic tests

1

1. The most perfect instrument for embolectomy from the main artery is:
a) Volmar's vascular ring;
b) vacuum - suction;
* c) Fogarty balloon catheter;
d) Dormia catheter;
e) fenestrated forceps.

2. For arterial embolism of the vessels of the lower extremities, it is not typical:
a) no pulsation;
b) parasthesia;

* c) trophic ulcers of the lower limb;
d) pain;
e) pallor of the skin.

3. What cannot be expected with arterial aneurysm:
a) rupture;
b) compression of adjacent organs;
c) pain in acute rupture;
d) arterial thromboembolism;
* e) spontaneous healing.

4. In acute ischemia of the extremity IIb degree, developed as a result of femoral artery embolism, it is uncharacteristic:
a) pain in the extremity;
b) "marble" pattern of the skin;
c) limb cooling;
d) lack of active movements in the joints of the limb;
* e) muscle contracture.

5. Operation embolism and thrombectomy in acute obstruction of the bifurcation of the aorta and limb arteries in severe ischemia is not indicated in the development of:
a) acute myocardial infarction;
b) acute ischemic stroke;
c) cases of abscessed pneumonia;
* d) total contracture of the affected limb;
e) everything is wrong.

6. In the diagnosis of acute arterial obstruction, the most informative research method is:
a) sphygmography;
b) rheovasography;
* c) aorto - arteriography;
d) plethysmography;
e) thermography.

7. The characteristic signs of acute disturbance of arterial blood flow in the limb are: 1) a sharp edema of the limb; 2) sudden onset of sharp pain; 3) limb warming; 4) pallor of the limb; 5) limb cooling. Choose the correct combination of answers:
a) 1, 3, 5;
b) 2, 3, 5;
* c) 2, 4, 5;
d) 1, 3;
e) 2, 5.

8. For acute ischemia of the extremity IIIB degree, which developed with femoral artery embolism, the following symptoms are characteristic: 1) pain in the extremity; 2) limb cooling; 3) lack of active movements in the joints of the limb; 4) sharp pain when trying to passive movements in the joints of the limbs; 5) total muscle contracture. Choose the correct combination of answers:
a) 1, 2, 3;
b) 3, 4, 5;
c) 1, 2, 4;
* d) everything is correct;

e) everything is wrong.

9. The most common cause of acute thrombosis of the arteries of the lower extremities is:
a) thromboangiitis obliterans;
* b) obliterating atherosclerosis;
c) puncture and catheterization of the artery:
d) extravasal compression of the artery;
e) polycythemia.
1

TOPIC: CONGENITAL HEART DISEASES

The purpose of the lecture : Acquaintance of students with congenital heart defects, the reasons for their development, the peculiarities of the clinical course, the course of complicated forms, differential diagnosis, optimal methods of treatment, postoperative management, rehabilitation of patients.

Educational purposes of the lecture: To instill in students the need for a timely adequate operation before the development of formidable complications , and with their development - acquaintance with the most informative and modern methods of diagnostics, surgical treatment of patients, acquaintance with possible complications outside the operation and the operating period, their prevention. Development of students' clinical thinking. Development of a modern view of the problem from the perspective of world medicine and general practitioner.

Lecture objectives:

8. Give an idea of congenital heart disease.
9. Explain the causes and mechanisms of complications.
10. Give a clinical description and possible variants of the course of the disease.
11. Conduct differential diagnosis with other diseases.
12. To acquaint students with modern and most informative methods, examination of patients
13. Demonstration of examples of their surgical practice: patients, slides, coronagraphs.
14. To prepare and present all the material of the lecture to the students, in the amount necessary for the high-quality training of a general practitioner.

PLAN OF THE LECTURE.

7. The urgency of the problem - 5 min
8. Etiopathogenesis of congenital heart defects.
9. Clinical picture - 10 min
   a) Etiopathogenesis;
   b) Clinic and diagnostics.
   c) Differential diagnosis.
   d) Treatment.

10. Diagnostics. - 10 min
11. Differential diagnostics. - 10 min
12. Treatment - 15 min

7. Disease prevention - 10 min

Many classifications of congenital heart defects have been proposed, many of them are complex and intended for specialists. Conventionally congenital heart defects can be divided into 3 groups

The first group - heart disease with intracardiac pathology cal messages, causes reset arterial KRO vie in the venous bed (from left to right, primary pale) - defect (cleft), atrial septal (ASD), ventricular septal defect (VSD), patent ductus arteriosus, aortolegochny fistula,
mitral stenosis with atrial septal defect, etc.

The second group - with intracardiac defects abnormal messages, reset causes of venous blood in arte ble direction (from right to left, the primary blue). This group includes the triad, tetrad and pentad of Fallot, artesia of the tricuspid valve, displacement of the tricuspid valve towards the right ventricle with an atrial septal defect, transposition of the great vessels, etc.

The third group - congenital heart disease in which Naru sheniya circulation caused by narrowing of the main blood vessels of the heart: the isolated pulmonary artery stenosis, aortic stenosis, coarctation of the aorta.

Patent ductus arteriosus

Patent ductus arteriosus (PDA) is one of the most common CHDs (in clinical practice, its frequency is 11-20%). The defect is almost 2 times more common in girls. PDA is a vessel through which, after birth, the pathological communication between the aorta and the pulmonary artery is preserved. Patent ductus arteriosus (or arterial canal) located between the barrel pulmonary arte Ria and initial department descending aorta It opens into the aorta with 1- 2 cm below the discharge of the left subclavian artery. Duct length about 1 cm , diameter 0.5-1 cm

Hemodynamics. If the ductus arteriosus does not clog due to the pressure difference in the aorta and the pulmonary artery, part of the arterial blood from the aorta enters the pulmonary artery and further into the lungs. The circulation of additional blood volumes in the lungs leads to overfilling of its vascular bed and causes increased work of the left heart, causing their hypertophy. The amount of arteriovenous shunt of blood depends on the diameter of the duct and the pressure difference in the aorta and pulmonary artery. Long-term hypervolemia of the pulmonary circulation leads to irreversible sclerotic changes in the small branches of the pulmonary artery and an increase in peripheral vascular resistance of the lungs and the development of pulmonary hypertension. The increase in pressure in the pulmonary artery leads to a gradual decrease in the discharge of blood from the aorta into the pulmonary artery. Over time, the pressure in the pulmonary artery rises so that it becomes equal to the system or even exceeds it Shunt becomes bi-directional, ie. E., Along with discharge from the aorta to the pulmonary artery appears reset from the pulmonary artery to the aorta In advanced cases, discharge

of blood takes place in the OS from the pulmonary artery to the aorta. The volumetric load on the left ventricle decreases and systolic overload of the right ventricle develops. At this stage of the disease, the characteristic symptoms of the defect gradually disappear and the symptoms of pulmonary hypertension begin to prevail. Gradually, the heart loses its pumping function and the death of patients occurs as a result of heart failure. In the natural course of the defect, the average life expectancy of patients with PDA is 25 years.

Clinical picture and diagnostics: there are no pathognomonic symptoms characteristic of PDA. The most frequent complaints are rapid fatigue, shortness of breath, aggravated by exertion, palpitations. The child lags behind in phi phe- development, often sick with pneumonia. The skin is pale, but in young children, when straining, screaming, cyanosis of the lower half of the body may appear, and the cyanosis disappears immediately after the load is stopped. Persistent cyanosis occurs only in adults and is caused by a reverse shunt of blood due to high pulmonary hypertension.

On examination, it is often possible to reveal a deformation of the chest in the form of a heart hump. On palpation of the chest, systolic-diastolic or systolic tremor in the projection of the heart base is determined. The borders of the heart are expanded. Measurement of blood pressure reveals a large pulse difference due to a decrease in diastolic blood pressure. The auscultatory picture of the defect is characteristic: in the second intercostal space to the left of the sternum, a rough systolic-diastolic murmur is heard, conducted on the vessels of the neck and in the interscapular space. As pulmonary hypertension develops and the discharge of blood from the aorta into the pulmonary artery decreases, the diastolic component of the murmur disappears and only a short systolic murmur is heard. With the same pressure in the aorta and pulmonary artery, the defect is practically "aphonic". Heart sounds are clear, there is an increase in 2 sounds over the pulmonary artery, in most cases. 2 tone is not only strengthened, but also split. On the ECG, specific changes are not detected. Chest X-ray reveals an increase in the vascular pattern of the lungs. The heart is moderately enlarged due to left ventricular and atrial hypertrophy. With the development of pulmonary hypertension, the right ventricle also increases. The arch of the pulmonary artery swells. Cardiac catheterization can reveal a number of signs of a defect. First, the passage of a catheter from the pulmonary artery into the descending aorta through the duct is an absolute sign of malformation; secondly, with thoracic aortography, the aorta and pulmonary artery are simultaneously contrasted. When probing the heart, along with an increase in pressure in the right ventricle and in the pulmonary artery, an increase in the degree of blood saturation with oxygen is found in the latter in comparison with its saturation in the cavity of the ventricle. ...

Complications of the CAP should be noted tank ter endocarditis and endarteritis (5% of patients), and very rarely observed spontaneous rupture of the aneurysm flow. Treatment: only operative. The optimal age for surgery is 2-5 years. However, in case of a complicated course of the disease, early age is not a contraindication for surgery. The operation is performed from the left-sided transthoracic access. The duct is closed either by bandaging it with a double

ligature, or by crossing it, followed by suturing at both ends. A contraindication to surgery is pulmonary hypertension 3b - 4 tbsp. It should be noted that the method of X-ray endovascular embolization or duct filling has also been proposed and implemented, but despite the good results of the method, it has not found wide application in clinical practice.

Ventricular septal defect (VSD)

Congenital heart disease, in which there is a communication between the right and left chambers of the heart at the level of the ventricles. VSD - the hour th congenital heart disease. In clinical practice, it occurs in 15-25% of cases.

Clinic and diagnostics. For large defects of the disease is difficult events decompensation, which is manifested by shortness of breath, an increase in Pec nor malnutrition. Children often get pneumonia. With small defects, the symptoms of the disease are not so pronounced. Some children have symptoms of slight fatigue and shortness of breath during exercise.

The examination noteworthy pallor, delay into the physics skom development chest wall deformation due to the presence of "serdech Nogo hump". On palpation of the chest, systolic tremor is determined along the left edge of the sternum, and, with large defects, the tremor is less pronounced, and in some patients it is not determined at all. Auscultation over the region of the heart is heard a rough systolic murmur with the greatest intensity in the third or fourth intercostal space to the left of the sternum. It is not performed on the vessels of the neck and back. With severe pulmonary hypertension, the noise is insignificant or is not heard at all, the 2nd tone above the pulmonary artery is accented and unfinished. In about half of the patients, a diastolic murmur can be heard above the apex.

On chest x-ray, there is an increase in the pulmonary pattern. The heart is enlarged due to both ventricles and atria, the pulmonary artery bulges. The electrocardiogram shows signs of hypertrophy of both ventricles and atria. Echocadiography is the most informative in detecting VSD.

Angiocardiography allows one to judge the size and localization of the defect, as well as exclude concomitant pathology. With catheterization of the cavities of the heart in the right ventricle and pulmonary artery, an increase in the oxygen content in the blood and a significant increase in pressure are detected, right ventriculography is completed to assess the state of the right ventricle and pulmonary artery. Left ventriculography is necessary to determine the location of the defect, its size, completes its thoracic aortography to exclude PDA, which is often combined with VSD.

With large VSDs, the clinical course is severe and can lead to the death of the patient in the first months of life. Therefore, all children with suspected VSD should be under constant supervision of a cardiologist. Insufficiency of blood circulation that cannot be controlled by drugs and the development of signs of a critical condition are indications for targeted examination in specialized cardiac surgery clinics. Treatment of the disease is only surgical. Distinguish between palliative and radical surgery for VSD. A radical operation is to close the defect. For defects with a diameter of not more than 1 cm . suturing with U-stitches

can be performed. In case of large defects, they can only be closed with a patch made of synthetic (Dacron, Teflon) or biological (autopericardium or canned xenopericardium) materials. Radical surgeries are performed under conditions of artificial circulation or hypothermia. The essence of palliative surgery is to narrow the pulmonary artery with a cuff to reduce the discharge of blood through the defect and reduce pulmonary blood flow and pressure in the pulmonary artery. The operation should be performed before the onset of Eisenmenger's syndrome, in which the operative closure of the defect, as a rule, does not give an effect and leads to rapid failure of the right ventricle and death as soon as possible after the operation, since the right ventricle cannot cope with the high pressure in the vessels of the lung

The development of X-ray endovascular surgery has led to the emergence of alternative methods of radical surgery for the treatment of septal defects. The essence of the method lies in the closure of septal defects by the transcatheter method using the so-called occluders (patches). The "patch" looks like two interconnected wire discs made of a nickel-titanium alloy. Both discs are connected by a neck that automatically centers the patch in the baffle hole. The mechanical properties of the base wire are such that they can be “packed” into a 2.5 mm catheter . The material from which the occluder is made has “memory, ie. after being pulled out of the catheter, the patch takes on the shape that was given to it during production. With the help of transcatheter implantation of occluders, it is possible to treat defects of the atrial and interventricular septa, open oval window, and patent ductus arteriosus.

Atrial septal defect

Atrial septal defect (ASD) is a congenital heart defect characterized by the presence of a message in the septum between the atria through which blood is released. According to V.I. Burakovsky in isolation given ny defect was detected in 7.8% of patients with congenital heart disease. According to their embryological origin, ASDs are divided into 3 groups: primary, secondary defects, and a single atrium. Primary ASD in isolated form is extremely rare; it occurs due to non-closure of the primary communication between the atria. Located in the lower part of the interatrial septum directly above the atrioventricular openings. A secondary defect is the most common form of defect. With them, the edge of the interatrial septum in the lower section is always preserved. The location of the defect can be different, most often it is located in the center of the interatrial septum. 0% of patients.

Hemodynamic disturbances in ASD are caused by the discharge of arterial blood from the left atrium to the right. The amount of discharge can be different, on average - 10-15 l / min. If there is a defect in the partition large part of arterial blood is excluded from the normal blood flow because, bypassing the left ventricle, it arrives from the left to the right before the angry, the right ventricle, into the vessels of the lungs and then re cart rotates in the left atrium. Congestion occurs right zhelu daughter, leading to hypertrophy, dilation and subsequent overfilling the vascular system of lungs excessive amount of blood, which leads to the

development of pulmonary hypertension. Last in the early stages of a functional nature and is caused by reflexive Thorne spasm of arterioles. Over time, vasospasm is replaced by their obliteration. Pulmonary hypertension becomes irreversible and progressive

The clinical picture of the defect depends on the degree of hemodynamic disturbance and the patient's age. With an uncomplicated course of the disease, patients complain of shortness of breath and attacks of palpitations, with physical exertion, rapid fatigue. Lagging behind in growth, susceptibility to pneumatic niyama and colds characteristic auscultatory picture. Over the heart of the second and third intercostal space left of the sternum listen systolic murmur due to increased of pulmonary Foot blood relative. pulmonary artery stenosis. The murmur is not as harsh as in VSD or pulmonary stenosis. Above the pulmonary artery II, the tone is split and its pulmonary component is enhanced. The electrocardiogram reflects the overload of the right heart.

On chest radiographs in frontal projection, there is an increase in the pulmonary arterial pattern. A rather specific symptom is the increased "pulsation of the roots of the lungs." The heart is enlarged due to the right sections. The arch of the pulmonary artery swells.

Echocardiographic examination allows visualizing the location and size of the defect. Angiocardiography is of secondary importance when examining patients with ASD.

In early childhood, the defect is usually benign. The first clinical symptoms usually appear at 2 to 3 years of age. The average life expectancy of patients with ASD is 37-40 years. The cause of death is heart failure.

Treatment: surgical only. If the size of the defect is less than 3 cm , it is sutured; in case of a large defect, the septum is plasticized with a patch.

Fallot's tetrad

One of the most common primary blue heart defects. Among patients with CHD, it occurs in 14-15% of all observations. Fallot's tetrad is characterized by 4 anatomical features:

1) narrowing of the pulmonary artery,

2) a defect of the interventricular septum,

3) offset to the right of the aorta (aortic dekstrapozitsiya) and races position its mouth above the defect in the interventricular Negotia rodke;

4) hypertrophy of the wall of the right ventricle.

There may be the following options for narrowing the pulmonary artery:

1) infundibular stenosis - narrowing of the outlet section of the right ventricle,

2) valvular stenosis (fusion of the leaflets, malformation of the valve),

3) a combination of valvular and infundibular stenosis,

4) hypoplasia of the main trunk or atresia of the orifice of the pulmonary artery.

Defect interventricular Negotia rodki at tetralogy of Fallot large size, it is equal to the diameter of the mouth of the aorta and is located in the membranous part of the partition under the septal tricuspid valve flap Dekstrapozitsiya aortic may be varying severity. Very often a large part of the area of its cross section is

over mezhzheludoch kovoy septum and the aortic lumen in communication with the right and left ventricles.

Hemodynamic disturbances in Fallot's tetrad are primarily determined by the degree of narrowing of the pulmonary artery. As a result of resistance in the path of blood flow from the right ventricle of pulmonary hydrochloric artery right ventricle performs extensive work, resulting in hypertrophy. A significant part of the venous blood, bypassing the pulmonary circulation, enters the left ventricle and aorta. In severe patients, the discharge of venous blood through the defect into the aorta can reach 70-80% Hypovolemia of the pulmonary circulation develops, and the systemic circulation is overloaded with venous blood Discharge of venous blood into the arterial leads to the development of hypoxemia

Clinic and diagnostics. The first signs of defect appear in the first months of life. Parents notice the appearance of cyanosis of the lips during the crying and crying of the baby or during feeding periods. In most children, by the age of one year, persistent cyanosis of the skin and mucous membranes is revealed, aggravated by physical exertion or crying. The disease takes the most severe form with the development of the so-called dyspnea-cyanotic attacks. Seizures can also occur in infants, but most often appear between 2 and 5 years of age. During an attack, cyanosis and shortness of breath sharply increase, tachycardia develops, a sharp weakness appears, the patient often loses consciousness or even falls into a coma. Cases of death or the development of severe forms of acute cerebrovascular accident are described. The occurrence of dyspnea-cyanotic seizures is associated with a sharp spasm of the muscles of the outlet of the right ventricle, as a result of which blood practically does not enter the pulmonary artery, and all the blood from the right ventricle enters the aorta. A large amount of venous blood entering the systemic circulation leads to a sharp increase in oxygen deprivation, up to loss of consciousness.

When examining patients, the skin and visible mucous membranes are cyanotic. Cyanosis is especially pronounced on the lips, conjunctiva, auricles, and nail phalanges of the hands and feet. Cyanosis is a consequence of an increase in the content of reduced hemoglobin. Fingers are kind of "drumsticks", the child behind in physics Český development. For patients with tetralogy of Fallot, a forced position during rest is characteristic: they squat or lie down with their legs brought to the stomach. Patients with tetralogy of Fallot are often cachectic, lethargic. Deformation of the chest rarely occurs, since the size of the heart with Fallot's tetrad is slightly increased. With auscultation of the heart in the second - third intercostal space at the left edge of the sternum, a rough systolic

A murmur that occupies the entire systole. Its intensity depends on the degree of pulmonary artery stenosis. The second tone over the pulmonary artery is weakened. In blood tests, there is an increase in the number of erythrocytes up to b-10 in mm 3., an increase in the level of hemoglobin to 130-160 g / l. On the phonocardiogram, two murmurs are often recorded: above the pulmonary artery and above the defect area in the interventricular septum.

The ECG reveals right ventricular hypertrophy. X-ray examination reveals signs characteristic of

tetralogy of Fallot: amelioration ri sunka roots of the lungs, pulmonary pattern depletion due to hypovolemia small circle. Due to hypertrophy of the right ventricle and moderate enlargement of the walls of its cavity heart takes the form of "dere vyannogo shoe" .Very valuable technique is echocardiography, which allows to establish the degree dekstrapozitsii aorta to evaluate the extent and severity of narrowing of the right ventricle and the pulmonary trunk output card, to determine the size of the right ventricle.

Cardiac catheterization with measurement of pressure in the pulmonary artery and right ventricle is of great diagnostic value. Usually the pressure in the pulmonary artery is 20-25 mm Hg, and in the right ventricle it is sharply increased and is equal to the pressure in the aorta. With the introduction of contrast medium into the right ventricle simultaneously contrasted blood fills pulmonary arte tory and ascending aorta. On radiographs hundred novitsya well prominent pulmonary artery stenosis

With the natural course of Fallot's tetrad, 25% of sick children die during the first year of life, 40% die by 3 years, 70% by 10 years and 95% by 40 years. Establishing a diagnosis of Fallot's tetrad is an absolute indication for surgical treatment. Distinguish between radical and palliative methods of defect correction. Radical elimination of the defect is carried out in conditions of artificial circulation. SUMMARY operation is for adjournment of interventricular defect eliminating stenosis and pulmonary artery. Depending on the type of stenosis, dissection of the fused leaflets of the pulmonary artery valve is performed along their commissures, excision of the fibromuscular ridge of the excretory section of the right ventricle, insertion of a patch of synthetic material into the longitudinal section of the excretory tract of the right ventricle and the trunk of the pulmonary artery. The defect of the interventricular septum is eliminated by sewing in a patch. The essence of palliative surgery is to restore pulmonary blood flow and eliminate hypoxemia. This is achieved by the imposition of bypass inter-arterial anastomoses. The most common is the Bllok-Tausig operation - an end-to-side anastomosis between the right subclavian artery and the right branch of the pulmonary artery. For a long time, the Vishnevsky-Donetsky operation was used - bypassing the subclavian and pulmonary arteries using a vascular graft. At the age of up to 3 years, operations are performed according to strict indications, only with frequent dyspnea-cyanotic attacks, severe cyanosis and impaired physical development. Preference is given to the Blloka-Tausig operation. The purpose of the operation - to give children the opportunity to experience a critical period in order to subsequently produce radical hydrochloric operation. In recent years, for the treatment of patients with tetralogy of Fallot, the method of balloon valvuloplasty has been introduced into clinical practice to eliminate valve stenosis of the pulmonary artery. With the successful completion of this procedure, there is no need to perform palliative operations.

Isolated valvular pulmonary stenosis

Isolated pulmonary valve stenosis (ICSLA) **is a** congenital defect characterized by the presence of an obstruction to blood flow at the level of the pulmonary artery valve. Isolated ste ERA pulmonary artery is 9 - 12% of all

patients with vrozh dennymi heart defects. Valvular stenosis of the pulmonary artery is formed as a result of fusion of the valve leaflets without any disturbances in the development of the outflow tract of the right ventricle. The valve is a funnel with an opening, turning schennym into the lumen of the pulmonary artery. A rounded hole is located in the center of the funnel or somewhat on the side. Its diameter ranges from 2 to 10 mm is always a poststenotic races extension of the main trunk of the pulmonary artery, often reaching large diameter.

Pulmonary artery stenosis is an obstacle to blood flow from the right ventricle to the pulmonary circulation. To maintain a normal minute volume, the right ventricle has to do a lot of work. This leads to a pronounced Noah hypertrophy of the right ventricle and then to tonogennoy and myogenic dilatation failure develops over time great O of the heart with decompensation in a large circle krovoob rashchenija.

Clinic and diagnostics. One characteristic IKSLA complaints of patients is shortness of breath, increasing with exertion, also marked heart beating, fatigue. Older children often complain of pain in the region of the heart, the development of which is caused by a deficiency of the coronary circulation. Cyanosis is not a characteristic feature of the defect. On examination, note Nali Chiyo cardiac hump, swelling and pulsation of the jugular veins. With auscultation of the heart in the second or third intercostal space at the left edge of the sternum, a rough systolic murmur is heard, which is conducted into the interscapular space and the side of the left clavicle. There is an increase in 1 tone, 2 tone above the pulmonary artery is not heard or is sharply weakened

The electrocardiogram reveals right ventricular hypertrophy and signs of overload in the right heart. When X-ray has an increase in the shadow of the heart due to the right ventricle, bulging and the elongation of the second arc of le vomu contour of the heart due to poststenotic expansion of the pulmonary artery. In 1 oblique projection, hypertrophy of the right ventricle is revealed, and in 2 - the right atrium occupies a significant part of the retrocardial space. A characteristic radiological sign of an isolated pulmonary artery stenosis is an enlargement of the pulmonary trunk with a normal or poor pulmonary pattern.

The final diagnosis is based on defect give the results comrade cardiac catheterization, in which the detected pressure increase in the right ventricular cavity and having a pressure gradient between it and the pulmonary artery. With angiocardiography, direct signs of pulmonary artery narrowing are revealed.

The prognosis without surgery is poor

Treatment is only surgical and consists in the elimination of valvular stenosis - pulmonary valvotomy. Distinguish between open and closed operations. In recent years, good results have been obtained after performing X-ray endovascular balloon valvuloplasty.

V O P R O S S

to the audience to establish feedback and find out the achievement objectives of the lecture

1. What is congenital blue heart disease?
2. What is white congenital heart disease?
3. List congenital heart defects related to blue defects.
4. List the congenital heart defects referred to as white defects.
5. List the congenital heart defects that characterize hypovolemia in the pulmonary circulation.
6. List the congenital heart defects that characterize normovolemia in the pulmonary circulation.
7. List the congenital heart defects that characterize hypervolemia in the pulmonary circulation.
8. What is the characteristic of Falo's tetrad.

TOPIC: ACQUIRED HEART DISEASES

The purpose of the lecture : Acquaintance of students with acquired heart defects, the reasons for their development, peculiarities of the clinical course, the course of complicated forms, differential diagnosis, optimal methods of treatment, postoperative management, rehabilitation of patients.

Educational purposes of the lecture: To instill in students the need for a timely adequate operation before the development of formidable complications ,and with their development - acquaintance with the most informative and modern methods of diagnostics, surgical treatment of patients, acquaintance with possible complications outside the operation and the operating period, their prevention. Development of students' clinical thinking. Development of a modern view of the problem from the perspective of world medicine and general practitioner.

Lecture objectives:

15. Give an understanding of acquired heart defects.
16. Explain the causes and mechanisms of complications.
17. Give a clinical description and possible variants of the course of the disease.
18. Conduct differential diagnosis with other diseases.
19. To acquaint students with modern and most informative methods, examination of patients
20. Demonstration of examples of their surgical practice: patients, slides, coronagraphs.
21. To prepare and present all the material of the lecture to the students, in the amount necessary for the high-quality training of a general practitioner.

PLAN OF THE LECTURE.

13. The urgency of the problem - 5 min
14. Etiopathogenesis of acquired heart defects.
15. Clinical picture - 10 min

a) Etiopathogenesis;
b) Clinic and diagnostics.
c) Differential diagnosis.
d) Treatment.

16. Diagnostics. - 10 min
17. Differential diagnostics. - 10 min
18. Treatment - 15 min

7. Disease prevention - 10 min

ACQUIRED HEART DEFECTS

The most common cause of valvular heart disease and once vice Vitia is rheumatic fever, followed by infective endo carditis, coronary heart disease to warrant postinfarction for Rocky (ventricular septal defect, mitral insufficient accuracy, cardiac aneurysm, blockades), chest trauma. A rare cause is atherosclerosis, which can lead to calcified aortic valve stenosis in the elderly.

Due to destruction of the connective tissue rheumatic mitral, aortic, tricuspid valves thicken fused, resulting in a stenosis, or due to thinning, deformation corroded edges and sediments Nia calcium salts occurs valve insufficiency. Distinguish stenosis and insufficiency of the valves, and to the GDS has flaps and their fusion failure at the same time, talk about the combined vice. In the latter case, it may be prevalence of stenosis or under sufficiency. The pulmonary valve is rarely affected by the rheumatic process.

Before starting the presentation of the material on acquired heart defects, it should be noted that the outcome of any heart disease is chronic heart failure caused by a violation of the pumping function of one or both ventricles of the heart. Many classifications of chronic heart failure have been proposed, including with valvular heart disease. For the purpose of a unified approach, cardiac surgeons around the world have adopted the well-known classification of the New York Heart Association (NYHA), according to which 4 functional classes (FC) are distinguished. It is based on the signs of heart failure, determined at rest and physical activity.

FC 1 - normal physical activity does not cause noticeable fatigue, palpitations, shortness of breath, pain, i.e. physical activity is tolerated in the same way as before the illness.

FC 11 - heart disease causes a slight restriction of physical activity; at rest no complaints. Normal physical activity causes fatigue, shortness of breath, palpitations, or sore throats.

FC 111 - there is a noticeable restriction of physical activity, when minor physical activity causes fatigue, pain, shortness of breath and palpitations. At rest, patients feel good.

FC 1U - Any physical activity is difficult. Subjective symptoms of heart failure are present even at rest.

In the CIS countries, the classification of circulatory insufficiency (NK), proposed by G.F. Lang, V.Kh. Vasilenko and N.Kh. Strazhesko, is also used. There are 3 stages of NK.

Stage I - initial, latent circulatory failure is manifested by shortness of breath, palpitations and fatigue during exercise. At rest, these symptoms disappear. Hemodynamics is not impaired.

Stage II - in this stage there are 2 periods. Period A - signs of NK at rest are moderately expressed, exercise tolerance is reduced; there are moderate hemodynamic disturbances in the systemic and pulmonary circulation. Period B - pronounced signs of circulatory failure at rest; there are severe hemodynamic disturbances in both the large and the pulmonary circulation.

Stage III - the final, dystrophic stage with severe hemodynamic disturbances, metabolic disorders and irreversible changes in the structure of organs and tissues.

Practice shows that the guarantee of good results of surgical treatment of patients with valvular heart disease is the early referral of patients to specialized clinics, before the appearance of a detailed picture of chronic insufficiency.

Mitral stenosis

Mitral stenosis is the most common rheumatic heart disease and is characterized by fusion of the edges of the mitral valve leaflets. Isolated mitral stenosis occurs in 1/3 of all mitral valve defects. According to V.Kh. Vasilenko, there are 50-80 patients with mitral stenosis per 100,000 population.

At the heart of the defect are sclerotic processes, which involve the cusps, annulus fibrosus, chords and papillary muscles. Stenosis of the mitral foramen begins with adhesion of the edges of the valves in contact with each other. Two commissures are formed, which, spreading from the ends of the valves to the center, cause an ever greater narrowing of the opening

Normally PLO schad left atrioventricular (atrioventriku-lar) holes is 4-6 cm2. Clinical symptoms of mitral stenosis begin to appear when the area of the mitral opening is less than 2 cm2.

Narrowed mitral orifice of an obstacle to Gnanu blood from the left atrium into the left ventricle due to an overflow of blood in the left atrial pressure is increased by several times. Compensatory hypertrophy and hyperfunction of the left atrium develops. However, due to the fact that the left atrium is a rather weak part of the heart, it early ceases to cope with increased stress. The increase in pressure in it is transmitted to the pulmonary veins, and then to the pulmonary capillaries and terminal branches of the pulmonary artery. If the capillary pressure exceeds the oncotic blood pressure, pulmonary edema develops. Spasm pulmonary arterioles system arte Rhee pulmonary capillaries prevents excessive pressure increase and increases the resistance in the pulmonary artery. Such a neuro-reflex spasm of the arteries helps to protect the capillary network of the lungs from overflowing with blood, although it does not reduce the pressure in the pulmonary veins and the left atrium. However, prolonged vascular spasm contributes to the development of sclerotic changes in the small branches of the pulmonary artery and a persistent pulmonary "second barrier" arises.

As a result of an increase in pressure in the pulmonary artery, compensatory hypertrophy of the right ventricle develops, and then the right atrium. A significant load on the right ventricle with mitral ste noze leads to incomplete emptying it during systole, increase in diastolic blood pressure and development relative tricuspid valve. Stagnation of blood in the venous part of the systemic circulation leads to an enlargement of the liver and the appearance of edema, i.e. the formation of right ventricular failure. Due to overstretching of the walls of the left atrium and its dilatation, damage to the conductive pathways of the heart by the rheumatic process, the normal rhythm of the heart is often disrupted and atrial fibrillation occurs. As a result, atrial contractions become completely ineffective, and there is an even greater expansion of the cavities, which creates conditions for thrombus formation in the left atrial cavity. With rhythm disturbances (from atrial extrasystoles to atrial fibrillation), some patients develop thromboembolic complications.

Clinic and diagnostics. With slight narrowing of the mitral valve normal hemodynamics supported enhanced operation of the left atrium, and patients may not predyav lyat no complaints. With the progression of narrowing and increasing pressure in the pulmonary circulation, shortness of breath and palpitations appear during exercise. Patients complain of cough, dry or with sputum containing streaks of blood, weakness, increased fatigue, less often pain in the heart and interruptions in the work of the heart. With the dramatic increase in pressure in the pulmonary capillaries develop in the mortar cardiac asthma, lung edema.

An objective study draws attention to the nature of ny "mitral" glow with a purple tinge on his pale face, cyanosis con chica nose, lips and fingers. Palpation of the region of the heart can determine the diastolic tremor in the apex ("cat's purr"). On auscultation, the I tone is enhanced (clapping). At the apex, the tone of the mitral valve opening is heard. Clapping I tone in conjunction with the II tone, and the tone of the opening created at the apex of the heart character of hydrochloric trinomial melody - "the rhythm of a quail." In patients with pulmonary hypertension in the second intercostal space, to the left of the sternum, an accent of the II tone is heard. The characteristic symptoms auscultatory mitral stenosis include diastolic noise that mo Jette occur in different periods of diastole. The ECG shows signs of atrial overvoltage, overload and hypertrophy of the right ventricle, the electrical axis of the heart is deviated to the right, the P wave is enlarged and split, which is regarded as a sign of left atrial overload. A phonocardiographic study registers an increase in the I tone and a diastolic murmur above the apex of the heart, which increases during the presystole period with sinus rhythm. X-ray examination of the heart in the anteroposterior projection shows a smoothing of the waist of the heart, bulging of the third arc of the left contour of the heart due to an increase in the left atrium. With high pulmonary hypertension, an increase in the second arch of the left contour is revealed due to the bulging of the pulmonary artery arch. A characteristic sign of the defect is the expansion of the left atrium, detected in the second oblique projection.

Echocardiography is a reliable diagnostic method. Characteristic echocardiographic features blemish are: a) a unidirectional diastolic movement of

the flaps mit eral valve, b) a reduction rate of early diastolic for covering of anterior mitral leaflet, c) reduction of the total excursion of movement of the mitral valve, g) reduction of diastolic race walking mitral leaflets and d) increase in size cavity left of the atrium. Cardiac catheterization is indicated for combined mitral defect to determine the degree of mitral regurgitation, with combined heart defects, with severe pulmonary hypertension - to determine its degree.

During mitral stenosis is dependent on the degree of narrowing of the mitral orifice and significantly deteriorates the development oslozh neny: atrial fibrillation, coarse fibrosis and mitral valve calcification, thrombus formation in the left front angry episodes with arterial embolism, pulmonary hypertension, relative or organic tricuspid valve. Death occurs from progressive heart failure, pulmonary edema, exhaustion.

The choice of method of treatment of mitral stenosis determined wish to set up the severity of the patients, the degree of violation gemodi Namiki.

In functional class 1, patients do not need surgical treatment. A gentle regimen and seasonal prevention of recurrent rheumatic attacks allow maintaining blood circulation in a state of stable compensation. In the 11th functional class, indications for surgery are relative. In 111 and 1U functional class indications for surgery are absolute.

The choice of a surgical method depends on many factors. Closed mitral commissurotomy is indicated for isolated mitral stenosis without gross changes in valve structures, as well as with concomitant grade 1 mitral insufficiency or grade 1 mitral valve calcification. Reconstructive operations on the mitral valve are indicated for patients with combined mitral defect with a predominance of insufficiency in the absence of valve calcification and without gross changes in the cusps, chords and papillary muscles.

With pronounced changes in valve structures, calcification of 2-3 degrees and concomitant mitral regurgitation, valve replacement is performed using mechanical or biological prostheses

Insufficiency of the mitral valve

The main causes of orga nical shape mitral insufficiency are rheumatism and sepsis (infection) endocarditis. In rheumatism, the tissues of the mitral valve leaflets are destroyed and marginal defects are formed, as a result of which the valve leaflets do not close during left ventricular systole. In rheumatism, pure mitral insufficiency is less common, more often it is combined with mitral stenosis or other valvular defects. With septic endocarditis, there are marginal defects in the valve tissue, as well as defects located in the body of the valves. Chord separation is often found. Incomplete closure of the mitral valve leaflets causes back blood flow (regurgitation) from the ventricle to the atrium during ventricular systole. The amount of regurgitation determines the severity of mitral regurgitation. The left ventricle is forced to constantly eject more blood, since part of it returns to the left atrium during systole and re-enters the left ventricle. The increased blood flow to the left ventricle causes hypertrophy and subsequent dilatation. Defect length tion time is compensated by a powerful left ventricle. Fasting foam develops a significant increase in the left atrium and ventricle. The pressure in the cavity of

the left atrium rises and then is retrogradely transmitted to the pulmonary veins; giving rise Lenie in the pulmonary artery, develops hypertrophy right zhelu daughter.

Clinic and diagnostics. The blemish compensation stage patients can perform significant physical burden and bolevanie often detected accidentally during the maintenance inspection. With a decrease in the contractile function of the left ventricle and an increase in pressure in the pulmonary circulation, patients complain of shortness of breath during exercise and palpitations. In some patients, a dry cough or cough with mucous sputum, sometimes mixed with blood, appears. More often than with mitral stenosis, patients complain of pain in the heart. With an increase in stagnation in the pulmonary circulation, dyspnea at rest and attacks of cardiac asthma may appear.

The appearance of patients usually does not change. Sometimes the chest deformity is revealed - "heart hump". On palpation and by eye, an increased apical impulse is determined, displaced to the left and down. On auscultation, the I tone is weakened or absent, the accent of the II tone over the pulmonary artery is moderately pronounced. Often a III tone is heard at the apex of the heart. The most characteristic symptom of auscultatory when mitral regurgitation is etsya systolic sound over the top, which takes place in the left armpit and along the left sternal border. The ECG shows signs of left ventricular and atrial hypertrophy. Revealing hypertrophy of the right heart is a sign of pulmonary hypertension. On phonocardiogram amplitude I tone greatly reduce on. Systolic murmur begins immediately after the I tone and occupies the entire systole or most of it.

When X-ray in a direct projection tion observed IV curve of the arc on the left cardiac contour due to dilatation and hypertrophy of the left ventricle. In addition, an increase in the left atrium causes bulging of the third arch. The increase in the left atrium is especially clearly detected in the first oblique lateral projection, where this part of the heart displaces the contrasted esophagus along an arc of a large radius (more than 6 cm). With a large increase in the left atrium shadow latter may extend beyond the contour of the right heart in the form of Doba paid-shade. When X-rays in cases of severe mitral insufficiency can be observed systolic bulging of the left atrium. Isolated mitral regurgitation on an echocardiogram is characterized by dilation of the left heart, excessive excursion interventricular septum diastolic multidirectional motion Niemi thickened mitral leaflets and a noticeable lack of systolic closure.

Intracardial examination determines the volume of regurgitation from the left ventricle to the left atrium, pressure in the cavities of the heart and in the pulmonary artery.

With moderate insufficiency of the mitral valve, patients remain able to work for a long time. Severe mitral insufficiency quickly leads to the development of severe circulatory failure and death of patients

The choice of treatment for mitral insufficiency is determined by the stage of development of circulatory failure. (see Treatment of mitral stenosis).

Aortic heart disease

The cause of aortic heart defects can be rheumatism, bacterial endocarditis, atherosclerosis. In terms of the frequency of lesions by the rheumatic process, the aortic valve is in second place after the mitral valve. The disease occurs in males are 3-5 times more likely than wives Shin. The leaflets of the aortic valve undergo calcification, often massive, with the transition of calcification to the fibrous ring of the valve, the wall of the aorta, and the myocardium of the left ventricle. Distinguish aortic valve stenosis, insufficiency aor Talnoe valve and combined lesions when concurrently Menno has stenosis and insufficiency.

Clinical picture and diagnosis: patients complain of shortness of breath, angina pain in the heart, palpitations and interruptions, dizziness and fainting. Dyspnea mo Jette wear paroxysmal (cardiac asthma) and completed the development of pulmonary edema. When aortic defects sometimes death comes suddenly on the background of apparent blagopo -being. On examination, patients show a spilled lifts the apex beat of the heart, which is offset down and to the left at a severe aortic valve regurgitation observed Wuxi fief pulsation of the arteries; carotid pulsation is clearly visible. When aortic valve sistoliche skoe pressure is raised, characterized by the decrease in diastolic pressure (often zero) and respectively considerable withdrawn chenie pulse pressure. When aortic valve and listen registers of riruyut fonokardiograficheski diastolic noise which follows blowing directly behind II tone and can occupy the full diastole. This noise generally decreasing, spreading along the left sternal border, formed by the blood stream, returning from the aorta into the cavity les Vågå ventricle during diastole. In the projection of the aortic valve with aortic stenosis, a rough systolic murmur is heard, which spreads to the carotid arteries.

X-ray reveals an increase in the size of the heart due to an increase in the left ventricle, the ascending aorta and its arch. Waist well defined heart, the heart becomes so-called directly aortic configuration

Echocardiography helps to identify CTE stump enlargement of the aorta and the left ventricle, the prevalence protses sa hypertrophy or dilatation of the myocardium, to evaluate its contractility, diagnose calcification of the valve and its spread to neighboring heart structures. Cardiac catheterization and cavities Angiocardiography Prima nyayut to clarify the degree of stenosis or insufficiency and assessment of myocardial contractility, identify areas of akinesia with his left ventricle. With angina pectoris, coronary angiography is performed to identify concomitant violations of the patency of the coronary arteries. When aortic defects, progressive hypertrophy myo infarction of the left ventricle leads to relative coronary insufficiency, angina pectoris, myocardial focal scarring and death from acute left ventricular failure.

Treatment is prompt. With isolated stenosis, surgery is indicated when the pressure gradient between the left ventricle and the aorta exceeds 30 mm Hg. Art. In case of aortic stenosis, if the valve leaflets are slightly changed, a valve-preserving operation is possible - the separation of the fused leaflets by commissures. In aortic regurgitation, surgery is indicated for grade II regurgitation. With calcification of the leaflets, aortic insufficiency, combination of stenosis and valve insufficiency, aortic valve replacement is performed.

V O P R O S S

to the audience to establish feedback and find out the achievement objectives of the lecture

1. List the acquired heart defects.
2. Describe the first functional class of chronic heart failure.
3. Describe the second functional class of chronic heart failure.
4. Describe the third functional class of chronic heart failure.
5. Describe the fourth functional class of chronic heart failure.
6. Describe the first degree of circulatory failure.
7. Describe the second degree of circulatory failure.
8. Describe the third degree of circulatory failure.
9. Describe the degree of mitral stenosis.
10. Preventive measures for acquired heart defects.

TOPIC : ACUTE AND CHRONIC NON-SPECIFIC DISEASES OF THE LUNG AND PLEURA .

The purpose of the lesson:

7. To study the etiology, pathogenesis, clinic of diseases of the lungs and pleura.
8. Learn the correct diagnosis according to the modern classification.
9. Be able to make a differential diagnosis.
10. Know the basic principles of treatment.
11. Establish the basic principles of prevention, labor expertise.

Test questions:

1. Anatomy and physiology of the lungs and pleura.
2. Research methods of the lungs and pleura.
3. Malformations of the lungs and pleura.
4. Damage to the lungs and pleura. Pneumothorax.
5. Specific inflammatory diseases of the lungs and pleura (tuberculosis, actinomycosis).
6. Purulent and chronic inflammatory diseases of the pleura (purulent pleurisy, pyopneumothorax, pleural empyema).
7. Purulent and chronic inflammatory diseases of the lungs (lung abscess, lung gangrene).
8. Malignant tumors of the lung. Crayfish.

THEORETICAL PART
ANATOMY OF THE LUNGS AND PLEURA.

The right and left lungs occupy most of the chest and are similar in shape to a cone dissected in the sagittal plane. Each lung is enclosed in a pleural sac. The outer (parietal) leaf of it lines the chest from the inside, and the inner (visceral) is tightly fused from all sides with the lung tissue. In the region of the lung root, both pleural layers pass into one another, and the pulmonary ligament is formed downward from the lung root in the form of a pleural duplication . At the bottom, with its free edge, it almost reaches the diaphragm.

In the parietal pleura, costal, diaphragmatic and mediastinal (mediastinal) parts are distinguished . Costal pleura covers the inside edges intercostal space nye muscle and fascia, with which it is associated layer of loose fiber. This will facilitate the separation of the lung pleura together with at seam. In the lower parts of the chest cavity there are spaces, the volume of which changes during breathing. These spaces are formed by the sheets of the parietal pleura. These include the costal-diaphragmatic sinus, located at the junction of the diaphragmatic pleura into the costal, cost-median sinus, which lies at the junction of the costal pleura into the mediastinal.

Between pleura normally contains 20-25 ml of fluid, to provide vayuschey smooth movement of the lung act of breathing. Complex structures, located between the pleural bags on the sides, sternum in front, the spine and the back part of the tendon of the diaphragm from the bottom, is called among the wall.

T pax Her and bronchi. At the level VI-VII of the cervical vertebrae, the larynx passes into the trachea. The trachea is a section of the respiratory tube from the larynx to the main bronchi. The larynx, in addition to phonation, prevents aspiration of foreign particles into the trachea, plays an important role in the mechanism of coughing. The trachea is a fibromuscular tube with an average length of 10-12 cm and a width of 13-22 mm. Retained its lumen due to the presence in the wall of 15--20 tracheal cartilage, occupy boiling 2/3 -4/5 its circumference and forming the front and side sections. The posterior part is called the membranous part. Ring-shaped ligaments are located between the cartilages. Outside trachea covered connective tissue sheath, inside - fusion Zist shell, located on loose submucosal layer. The latter are lymphoid follicles and alveolar-tubular glands that produce conductive protein and mucous secretion. The submucosal layer without a sharp border passes into the dense perichondrium of the tracheal cartilage.

Mucosal epithelium - pseudostratified cylindrical surfaces with nym ciliated layer. Constant oscillatory motion of cilia spo its proper advancement of fine dust particles and formed in the direction of mucus lenii larynx.

The blood supply to the trachea is carried out by the branches of the lower thyroid arteries and the branches of the bronchial arteries originating from the descending aorta or the upper intercostal arteries.

Venous blood flowing mainly in the venous plexus location with conjugated around the trachea and the esophagus, where it comes to the lower venous plexus of the thyroid gland, and unpaired hemiazygos veins.

Lymphatic drainage of the trachea is in a centripetal ventrodorsalnom direction lenii. The pathways for the outflow of lymph from the trachea are closely related to the lymphatic pathways of the esophagus, larynx, thyroid gland.

The trachea is innervated by the branches of the recurrent and superior laryngeal nerves. In addition, the trachea has a sympathetic and parasympathetic innerva tion. Part of the nerve fibers - spinal (from $_{1}$ to D $_{6}$).

Distinguish between the cervical and thoracic trachea. Last is divided into the upper chest and nadbifurkatsionnuyu part. The location of the trachea on the neck and in the chest cavity determines its relationship with the organs of the neck and chest. On its front neck cover isthmus of the thyroid gland, thyroid vein front, side - thyroid lobe and carotid arteries (aa. Carotis), rear - with return esophagus nerves lying in the groove between the esophagus and tra heey. In the thoracic region, in front of the trachea, there is the beginning of the anonymous artery (a.anonyma) and the left carotid artery, behind - the esophagus, on the left - the aortic arch, left common carotid artery, left recurrent nerve, on the right - anonymous artery, right vagus nerve.

In older people, calcium salts are deposited in the trachea. She rents at genologicheskom study gives a denser shadow. Extended aorta, Aneva ism, tumors can shift it aside.

The right main bronchus departs from the trachea at a sharper angle than the left, and is, as it were, its continuation. This leads to more frequent ingress of foreign bodies into the branches of the right main bronchus, the flow of vomit. Lim phatic nodes located at the bifurcation of the trachea, the presence of these metastases do bronchus dividing angle a blunt, round out its vertices have . Above the left bronchus is the aortic arch, above the right, spreading over it, is the azygos vein (v. Azygos), which flows into the superior vena cava.

The main bronchi are divided into lobes, the right one into three, and the left one into two branches. Continuing to divide into segmental and subsegmental, they decrease in diameter, pass into small bronchi, and then into bronchioles.

Lungs . The lungs are divided by a deep groove into two unequal lobes - upper and lower. In addition, in the right lung, the upper lobe is further divided into the upper and middle. In the left lung there is no middle lobe, but accordingly it has a reed segment of the upper lobe not separated by a clear interlobar groove. The pulmonary segment is the main morphological unit of lung tissue. It includes an anatomical complex with a separate bronchus, artery, veins, nerves and lymphatic vessels. In shape, the segment resembles an irregularly shaped cone or a polyhedral pyramid with the apex directed towards the root and the base towards the periphery of the lung. Table 1 is a segmental classification comrade lungs.

Edge bronchioles and pneumatic (terminal and respiratory) vet vyatsya within lung lobes. Dividing Zack n Chiva alveolar passages, passing into alveolar bags whose walls protrude to form the alveoli.

Table 1. Classification of lung segments

Right lung		Left lung	
Share	Segment	share	segment
Upper	1. Upper	Upper	1-2. Posterior upper
	2. Rear		3. Front
	3. Front		4. Upper
Average	4. Lateral	Reed	5 . Lower
	5. Medial	segment	6. Upper
Lower	6. Upper	Lower	7. None
	7. Mediobasal (cardiac)		8. Anterobasal
	8. Anterobasal		9. Laterally
	9. A ateral s but basal- ny		basal
	10. Posterior basal		10. Posterior basal

The walls of the alveoli have three layers. From the inside, the alveolus is lined with a single-layer alveolar epithelium located on the basement membrane; the basis of the wall is the interstitial tissue of elastic collagen fibers. Nar uzhn s m layer is basal capillary membrane and epithelium. Thus, the blood flowing through the vessels is separated from the air in the alveoli by a very thin partition through which gas exchange takes place .

Blood circulation in the lungs: venous blood from the superior and inferior vena cava enters the right ventricle, and then through the pulmonary artery and its branches into the lungs. By and channels on each of the bronchi with The appropriate branch of the pulmonary artery, which is together with the bronchi and to a NCU all falls into the pulmonary capillaries, shrouding the alveoli. At this level there is an exchange of gases, whereupon about ogaschennaya sour native arterial blood goes from the capillaries into the veins of the lung. Each lung has two pulmonary veins, an upper and a lower, that flow into the left atrium, from where oxygen-rich blood enters the left ventricle and then into the systemic circulation. The walls of the bronchi, and interstitial lung tissue supplied ar t erialnoy blood from the bronchial arteries. The outflow of blood vessels of large b p onchia occurs bronchial veins flowing into unpaired hemiazygos and veins, and capillaries of small bronchi A direct venno in the branch of the pulmonary veins. Between the branches of the small pulmonary

arteries and veins are arteriovenous shunts, which normally do not function and are opened only in the presence , of a number of pathological conditions. As of pulmonary vessels in Bron hialnye, and vice versa, - of bronchial arteries to the pulmonary vessels, possible discharge of blood.

The lymphatic system of the lung is well developed and consists of *:* 1) initial networks of lymphatic capillaries, 2) intraorgan plexuses, 3) discharge vessels, which flow into the lymphatic ducts.

There are four groups of tracheobronchial lymph nodes: 1) intrapulmonary, located in the lung parenchyma and in the field of separation Bron Hove, 2), bronchopulmonary, lying in the region of the gate and lung root at the corners separating the main and lobar bronchi, 3) tracheobronchial, positioned at the bifurcation trachea, 4) paratracheal, located along the trachea.

Lymph flow goes from the periphery to the root of the lung and to the confluence of limfatiches cue duct passes through at least one of the lymph nodes. Most of the lymph flows from the lungs into the right lymphatic duct, only from the upper part of the left lung from about to lymph flows through the left paratracheal chain of lymph nodes. From the lower lobes of both lungs, lymph also flows through the vessels that go down in the sheets of the pulmonary ligaments to the node located in the posterior mediastinum behind the esophagus and diaphragm.

Lungs are parasympathetic, sympathetic and sensitive innerva tion. The sensory innervation of the lungs occurs mainly through the spinal nerves. All of these nerves form powerful anterior and posterior pulmonary plexuses at the root of the lungs.

Lung expansion during inhalation provided by the presence of negative prevent Lenia in the pleural cavity (2-5 m m Hg. V.).

During a decrease in the volume of the chest, the alveoli collapse, the pressure in them exceeds atmospheric and exhalation occurs. As a result, the pulmonary valve tion is updated in the alveolar of spirit. For normal gas exchange, it is required that the oxygen pressure in the alveolar air is 110 mm Hg. Art., and carbon dioxide 40 mm Hg. Art.

Gas exchange in the alveoli occurs according to the physical laws of diffusion. Gases pass through the alveolar wall under the influence of the difference in pressure of gases in the blood and alveolar air.

The diffusion capacity of carbon dioxide is much higher (25-30 times) than that of oxygen. As part of the alveolar oxygen contained in the air if honors 15 vol. %, and carbon dioxide - 6 vol. %. The difference in oxygen tension al Véolia and blood is also small (6-9 mm Hg. V.). In this regard, the absorption of sour kind of broken lighter than carbon dioxide output.

Normally, the absorption of oxygen in the blood is almost equal to its consumption in the tissues. Quite significant respiratory and circulatory disorders can be compensated and do not manifest themselves in anything when the patient is calm. However, a load due to surgery on the lungs, an exacerbation renie diseases cause disorders of respiratory function, leading to a breach of hemodynamics.

RESEARCH METHODS

Instrumental research methods

In order to establish a definitive diagnosis and choice of treatment, in addition to a thorough clinical examination, have considerable tion of the patients with diseases of the respiratory system req Dimo implementation of a number of special methods of investigation.

X-ray examination is indicated for all patients. Obja optionally and perform fluoroscopy and radiography, wherein at least two planes - frontal and lateral. Often, they make it possible to put a definitive diagnosis of pathological process (presence of abscess cavity, pleural effusion, and others.), Determined to share the location of the lesion directed research.

Tomography - layering radiological research went FIR - allows you to set the change of the lumen of the trachea and bronchi (for this purpose, in addition to imaging in the frontal projection, an is also necessary shadow formation. The diagnostic capabilities of the method is increased smiling when applying so-called direct imaging withdrawn cheniem.

Tracheobronchoscopy - *a* study using a bronchoscope - along with tomography, is the main research method for diseases of the trachea and bronchi. Absolute indications for its use are diseases of the trachea and bronchi, beneath vision for lung cancer, foreign bodies in the trachea and bronchi.

When bronchoscopy using special forceps mozh but take a piece of tumor or suspicious her fabric piece for histological examination using a special schetoch ki receive scraping the mucosa bronchi (so-called brush-biopsy) for histological and cytological explores Bani, moist swab to take a smear for cytology The investigations have been concerned. Special bronchoscopes with fiber optics (from "Olimpus" or similar systems) make it possible to examine bronchial branches of 4-5 orders and obtain material from them for cytological research.

Tsitologiches to th study. Cytology shall undergo sputum, pleural exudate material obtained brush-biopsy and using the tampon with bronhosko FDI mediastinoscopy.

Other methods used for certain research indications for diagnosis, determination of communication swelling whether light with the surrounding tissues, detection of metastases, obtaining n Ia biopsy material.

By ompyuternaya tomography it provides a cross-sectional X-ray image of the chest and its organs with a very high-definition and high-resolution. On transverse sections, depending on the density of the tissue, i.e. , on the degree of absorption of X-ray radiation, it is possible to clearly distinguish tumors (denser areas), cysts, abscesses, fluid accumulation in the pleural cavity, the presence of changes in the mediastinum when a lung tumor grows into it or lymph node metastases.

Bronchography - radiography after Zapolle neniya bronchi contrast agent - is important for the diagnosis and localization refinement bronchiectasis, residual GOVERNMENTAL cavities after suffering a lung abscess, bronchopleural fistula, cicatricial stenoses bronchi. Simultaneous vi deozapis or filming (bronhokinematografiya) greatly extend the capabilities of the method. If necessary, the study details chit only a certain portion of the bronchial tree is used aiming bronchography, which is produced using a special catheter meter or "controlled" catheter. In order to prevent the so-called yodolipolnyh pneumonia, koto rye may develop after an investigation, it is advisable to Use Vat lipiodol mixed with sulfonamide drug or water - soluble contrast agents.

Angiography - contrast research vessels lay anyone. It may be common and selective when certain cannulated vessel and after administration of small doses of contrast Nogo material (15-20 ml) make a radiograph of a limited region of the lung. Angiopulmonography is used mainly to clarify the operability in lung cancer. A symptom of amputation of a large branch of the pulmonary artery or a defect in its filling indicates that the lung is not removable. The method is also used for the diagnosis of pulmonary ar terii, vascular malformations of the lung, arteriovenous Aneva ism.

Upper cavography is a contrast study of the superior vena cava. It is used in cases of suspected sprouting in her OPU holy lung or mediastinal metastases of lung cancer there is in the mediastinum.

Arteriography of bronchial arteries - X explores vanie performed when completing the bronchial arteries pin gain in contrast substance. It is shown mainly to clarify the localization of the source of bleeding and as a research method, prior embolization artery in pulmonary krovotech Research Institute.

Pnevmomediastinografiya - chest X-ray after the introduction of gas mediastinal tissue (usual but 150-200 ml of oxygen) through the puncture on the sternum or second intercostal space to the left. The method allows to diagnose lung tumor invasion into the mediastinum, to

determine the increase limfati cal nodes in the hilar and mediastinal metastases in the mediastinum.

Pleurography is an X-ray examination of the pleural cavity after the introduction of a contrast agent into it. Using are only water-soluble contrast agents. For definition Lenia cavity size must study at least three positions: a vertical position, and on the back side (on the side of the lesion).

For certain indications, the primary purpose of the floor cheniya material for cytological or histological The investigations dovaniya, apply thoracoscopy and mediastinoscopy.

Thoracoscopy is performed with a special instrument - a thoracoscope. It gives the opportunity to study for a trifling eral content, see the parietal and visceral Pleven ry tumor sprouting up lung surface, take a piece for histological examination of the pathological education Niya. A thoracoscope is usually introduced along the midaxillary line in the fourth - fifth intercostal space.

Mediastinoscopy - examination of the anterior surface of the trachea (before bifurcation) using a special instrument. Mediastinoscopy applied in order to obtain for histological explores Bani punctates from the lymph nodes, which are located along the trachea with suspected for the presence of metastases of lung cancer, diagnosis of systemic diseases (Hodgkin et al.).

Radioisotope scanning with xenon is used for the definition Niya blood flow and the participation of various departments in the light breeze. The study is carried out using a VAIME T multi - detector or a similar device with 16 sensors, each of which gives information from a portion of the lung field, which is 1/8 of its part. Inhalation ^{133}Xe last distributed in lay FIR on the bronchi that enables to determine the proportion of each lung field participate in breathing. By the half-life of the drug, the state of bronchial patency is judged. An increase of the half-life of the isotope evidence of obstructive tive process in the bronchi.

When administered intravenously, the drug register its races EFINITIONS (perfusion) in the lungs and removing time, which indicates the state kapillyaroalveolyarnogo barrier. The method specifies the true volume of the affected lung tissue.

ABSCESS AND LUNG GANGREN

Abscess and gangrene - a qualitatively different Patolo -meteorological processes.

With an **abscess, there** is a limited purulent-destructive process in the lung tissue. Limiting the inflammatory focus and transition ichorization in suppuration indicate expression zhennoy protective reaction of the organism, while Prevalence nennaya Gangrene is the result of progressive necrosis resulting weak reactivity or complete unresponsiveness op - organisms.

Among the patients, men aged 30-35 years prevail, women get sick 6-7 times less often, which is associated with the peculiarities of the production activity of men, the more common abuse of alcohol and smoking among them, leading to a violation of the drainage function of the upper respiratory tract.

Etiology and Pathogenesis: the main factors OCU tributes abscesses and gangrene of lung development are airless lung tissue (due to obstruction of the bronchus, atelectasis and inflammation), circulatory disorders in it , a direct effect of toxins on the airless lung tissue with impaired circulation.

There are bronchopulmonary, hematogenous-embolic, lymphogenous and traumatic ways of occurrence of pulmonary abscesses and gangrene.

When single pyogenic abscesses cavity rather quickly released by the pus its wall progressively cleansed from necrotic masses and coated granulations, in place of abs cession formed scar or lined with epithelium narrow cavity. With large, poorly draining cavities, prolonged purulent fusion of necrotic tissues, the presence of an inflammatory process in the surrounding parts of the lung, the release of the cavity from necrotic masses occurs slowly,

dense scar tissue forms in the wall of the abscess, which interferes with healing. A chronic abscess is formed.

Multiple abscesses usually preceded by widespread ny inflammatory process in the lung. Against this background, lung tissue necrosis occurs in several areas. Areas of necrosis under jected purulent , melt at different times, pus breakthrough nicks in the bronchial tree does not occur simultaneously.

When multiple abscesses outcome of acute period is a smiling formation of several cavities, surrounded by a thick shell of necrotic and granulation tissue. Easily between Cloth from abscesses does not recover its normal structure

Bronchopulmonary tract . One of the most frequent causes originated novena abscesses and gangrene is a disturbance patency segmental and lobar bronchi due to their getting into the lumen of infected material from the oropharynx. When bessozna Tel'nykh state (due to alcoholic intoxication, after an opera tion) for severe infections function esting chatogo bronchial epithelium is disturbed, the cough reflex is suppressed and infected material (food particles , plaque, saliva) can be fixed in the bronchus as much time as necessary to the development of atelectasis and inflammation in the corresponding area of the lung. As a rule, abscesses in these cases are localized in the posterior segments (II, VI) and more often in the right lung.

Similar conditions occur in bronchial occlusion GTC holyu, foreign body, the narrowing of its lumen scar (obstructive abscesses). Removal of foreign body and the reduction e patency bronchus in these cases often lead to rapid cure of the patient. Metapneumonic abscesses occur in 1.2-1.5 % of patients with pneumonia. Their development is favored by a decrease in the reactivity of the organism, pronounced disturbances in ventilation and blood supply to the lung, often caused by previous lung diseases, insufficiently active treatment of the pulmonary process.

Hematogenous-embolic pathway. This way 7-9 % of lung abscesses develop . Contact with the lung infection is due to transfer current blood infected emboli from extrapulmonary infection foci at septicopyemia, osteomyelitis, septic thrombophlebitis, etc. The infected. Emboli occlude vayut lung vessels, - developing pulmonary infarction, which first undergoes purulent melt. Abscesses of hematogenous -embolic origin are more often localized in the lower lobes; they are plural.

The lymphogenous pathway of development of pulmonary abscesses and gangrene is rare. Skid infection to the lungs can angina, mediastinitis, subdiaphragmatic abscess et al.

Abscesses and gangrene of traumatic origin are the result of closed chest trauma with damage to lung tissue and penetrating wounds.

P Athol ogicheskaya anatomy: at the time of abscess formation in the lung tissue on a background of morphological changes, characterized Terna for pneumonia, there is one or more areas of necrosis. Under the influence of bacterial proteolytic enzymes occurs purulent fusion of necrotic masses, - forms a camping cavity filled with pus. Disruption of one of the walls of the bronchi located in necrotic area leads will do of pus into the bronchial tree. Subsequently morphological e skie changes determined by the state of the patient reactivity condition viyami abscess drainage and its dimensions over the inflammatory - inflammatory process in lung tissue surrounding...

Lung gangrene is characterized by the absence of restriction of altered lung tissue from healthy one. An area of gangrenous tissue without sharp boundaries turns into softened lung tissue of a dark color, which also without clear boundaries turns into healthy tissue.

Clinical picture and diagnosis: *with typical forms of the* disease in the clinical picture, two periods can be distinguished; 1) the period before opening the abscess in the bronchus, 2) the period after opening the bronchus.

The disease usually begins with symptoms characteristic of pneumonia: the body temperature rises, pains in the side appear with a deep breath, and cough. Physical examination reveals lag in respiration of the chest, respectively stvuyuschey the affected sections of the lungs, tenderness Pal patsii; here the shortening of the percussion sound is determined. The X-ray and

CT scan is visible pain Shih or smaller dense shade. Despite treatment, pneumonia is not allowed and becomes protracted characterized ter. The high temperature is accompanied by chills and torrential sweat. Sometimes patients notice bad breath. In the study of blood, high leukocytosis is revealed, a sharp shift in the white blood formula to the left.

The second period begins with the breakthrough of the abscess into the bronchial tree. In cases where it is emptied through a large bronchus, a large amount of pus immediately leaves, sometimes mixed with blood. The patient's condition is improving rapidly. However, more often the emptying of the abscess occurs not in a direct way, through a large bronchus, but through a tortuous course formed by a small bronchus, starting in the upper part of the abscess. Accordingly, the release of pus from it is slow, the state of the patient remains etsya heavy. Pus, getting into the bronchi, causes the development of pus Nogo bronchitis with copious mucus formation (up to several hundred milliliters per day). Sputum with a lung abscess has an unpleasant odor, and when standing in a jar, it is divided into three layers. The lower one consists of pus, the middle one is of serous fluid and the upper one is foamy. Sometimes the sputum may be seen on the small jerks changes in lung tissue (pulmonary sequestrations). When E -macroscopic study show its great quantitative of leukocytes, elastic fibers, a plurality of bacteria.

The data of the physical examination changes compared to the first period. As release oral abscess with pus and permits perifocal inflammation uc vanishes zone shortening percussion sound. In the presence of a large cavity, free of pus, a tympanic sound can be determined above it, more clearly detected if the patient opens his mouth during percussion. With significant amounts abscess auscultated amforicheskoe breath over the cavity and raznokali Bernier rales, preferably at adjacent regions of the lung.

Heavier flow *multiple abscesses* lay anyone. They are usually metapnevmonicheskimi and WHO Nick against the backdrop of the inflammatory tion infiltration of the Schirn areas of lung tissue. Break one of an abscess in the bronchial tree is not at leads to a significant reduction in toxicity and improvement of pain Foot as foci of necrosis purulent n remain in the lung tissue of melting. The condition is aggravated by developing purulent bronchitis with profuse discharge of fetid sputum. Physical examination determines the lag in breathing of the chest on the side of the lesion, dullness during percussion, respectively, of one or two lobes of the lung: auscultatory - a lot of wheezing of different caliber.

X-ray examination first reveals extensive opacification in the lung; as ulcers emptying the contents of the first against the backdrop of darkening become visible cavity levels Jew bones. As a rule, the patient does not recover. Zabol vanie progresses. Develop pulmonary heart failure Nost, stagnation in the pulmonary circulation, degenerative of Menenius parenchymal organs. All this quickly leads to death.

Gangrene is the most severe form of purulent lung lesions. Absorption of the products of putrefactive decay formed during gangrene of the lung, and bacterial toxins leads to the sharpest intoxication of the patient. In early lung gangrene nachi naet separate a large amount of stinking foamy sputum, having the form of "meat slops" impurities from the blood due arrozirovannyh pulmonary vessels. In the process, usually involving etsya pleura, leading to the development of putrid empyema or pneumoempyema. When examining the patient, attention is drawn to pronounced shortness of breath, anemia, cyanosis, a significant zone of shortening of the percussion sound over the affected lung; on auscultation, a lot of moist rales of various sizes are heard. Radiological findings reveal an extensive blackout in an easy to Thoroe increases with each passing day. Before the advent of antibiotics, patients with lung gangrene usually died during the first days of the disease.

Treatment : acute suppurative lung disease should be treated with a complex: it is aimed at improving the resilience of STI of the body, improving the drainage of the abscess, infection control, normalization of cardiac function, the function of internal organs.

Increasing the resistance of the organism is achieved: a) from Resp hygienic mode, b) power supply. Patients with sputum lose a large amount of protein and primarily albumin. Total

caloric intake should be part lyat 3500-4000. Food should be protein; to reimburse energy costs, it is advisable to complete parenteral and enteral (including tube) nutrition.

Improvements conditions abscess drainage manages reached pull the: a) applying expectorants, b) introducing into the bronchial tree solutions of proteolytic enzymes, mucolytic agents as an aerosol, by pouring into the abscess cavity through the bronchoscope, the puncture ulcer through the chest wall in the case subpleurally its location, c) assign Niemi physical therapy in combination with postural drainage (giving the patient position, wherein the contents ab stsessa will flow away due to gravity).

Rational antibacterial therapy should be tailored to the sensitivity of the flora, seeded wet you. In the absence of sensitivity flora expediency data but to use broad spectrum antibiotics (aminoglycosides, cephalosporins, etc.), In combination with sulfonamides, metronidazole (Trichopolum). Aside from administration of antibiotics, intravenously, intramuscularly or orally, it is necessary to introduce them into the bronchial tree or abscess cavity (in the form of an aerosol through the bronchoscope when broncho scopy, abscess cavity in the puncture abscess).

Normalization of cardiac reach-Application Niemi heart means. For detoxification and improve the micro- circulation should be used gemodez, reopoligljukin.

Immunocorrective therapy is of great importance. Repeated transfusions of blood, plasma, administration of IgG (gamma globulin), increase the therapeutic sera reactivity organisms ma. Some medications also contribute to this: levamisole, thymosin, prodigiosan, etc.

In case of staphylococcal destruction, it is also necessary to administer lipofundin or other fat emulsions used for parenteral nutrition. Injected into the bloodstream fat binder Vaeth bacterial enzymes and reduce their destructive dei Corollary to the lung tissue.

Surgical intervention is indicated for pulmonary gangrene (pulmonary or lobectomy); in an acute abscess, it is resorted to when there are extensive foci of destruction of the lung tissue in the absence of a sufficiently satisfactory drainage. In these patients, one-stage (in the presence of adhesions between the visceral and parietal pleura) or two-stage (in the absence of adhesions) pneumotomy is performed. In recent years, these operations produce less and less, as the good drainage of the abscess can be achieved by Use Vania puncture it through the chest wall, the introduction into the cavity of the abscess drainage via a trocar. Follow schaya aspiration of pus and the introduction of proteolytic enzymes and antibiotics are usually given a good effect.

Conservative treatment futile when abscesses diameters rum more than 6 cm, very thick capsule of abscess, detectable by X-ray study of intoxication, not inferior grade complex therapy. In these cases, you can rec Dowa lung resection is already in the acute period.

Outcomes of acute lung abscess: 1) complete recovery, in which, along with the disappearance of clinical symptoms, the radiological symptoms of a lung abscess also disappear; 2) clinical improvement which is characterized by the complete disappearance of clinical symptoms of the disease, but rents genologicheski lightweight dry taped cavity; 3) clinical improvement: to discharge the patient's body temperature remains subfebrialnaya, the patient releases a small amount of fusion Zist-purulent sputum. X-ray reveals a cavity with infiltration of lung tissue in its circumference; 4) without improve Nia: in these patients without any remission acute form zabole Bani becomes chronic. Rapidly growing intoxication, developing pulmonary heart disease, degeneration guy himatoznyh bodies; 5) death.

The most severe complications in the acute period, often causes death, are as follows: a) breakthrough abscess sa into the pleural cavity with the development of an intense pnevmotorak ca, b) bleeding into the bronchial tree, as a result of which the mo Jette occur asphyxia, c) aspiration of pus in the unaffected areas of the bronchial wood and the development of new abscesses, g) of the transform of ulcers in distant organs, most commonly in the brain.

Therapeutic measures are determined by the nature of the complications: a) with the development of a tense pneumothorax, urgent drainage of the pleural cavity is necessary; b) in case of bleeding in the bronchi cial tree in an emergency event shows is urgent naya double-lumen endotracheal tube that can prevent blood from flowing into the bronchi unaffected lung. In

the future, hemostatic therapy is carried out. In the presence of the corresponding boiling conditions suitable endovascular surgery - embolization bronchial arteries affected lung arrosion koto ryh most often causes bleeding in the respiratory ny minute; c) the newly formed abscesses in the lung treated in soot sponds with the above principles of therapy of lung abscess; d) metastatic abscess treated by the conventional scheme (early abscess autopsy, rational antibacterial Nye therapy, immunotherapy, and others.).

PRACTICAL PART

THEMATIC TESTS

1

1. Active drainage of the pleural cavity is not shown:
a) with thoracotomy
b) with hemothorax due to rib fracture
c) with recurrent hemothorax
d) with pleural empyema
e) with lower lobe pneumonia.

2. The gangrene of the lung is characterized by:
a) the development of the disease when the body is unresponsive
b) the absence of a granulation shaft at the border of the lesion
c) advanced necrosis of the lung tissue
d) severe intoxication
e) all of the above is true.

3. Chronic pleural empyema is considered:
a) from the second week
b) from 4 weeks
c) from 6 weeks
d) from 8 weeks
e) from 3 months.

4. A complication of an acute lung abscess cannot be:
a) abscess breakthrough into the pleural cavity
b) bleeding
c) aspiration of pus into a healthy lung
d) sepsis
e) the formation of a dry cavity in the lung.

5. With gangrene affecting one of the lobes of the lung, it is recommended:
a) daily sanitation of the bronchial tree through a bronchoscope
b) administration of antibiotics to the pulmonary artery
c) lobectomy
d) intensive therapy with endobronchial antibiotics
e) complex therapy, including all of the above-mentioned conservative treatment methods.

6. The reason for the transition of acute pleural empyema to chronic cannot be:
a) failed attempt to obliterate the cavity in the acute period

b) premature drainage removal
c) large primary cavity
d) tuberculosis and other specific flora
e) broncho-pleural fistula.

7. With a large air cyst, the following are possible:
a) pneumothorax
b) pulmonary hemorrhage
c) cyst suppuration
d) all of the above is true
e) all of the above is incorrect.

8. Acute pneumothorax is not characterized by:
a) dyspnea at rest
b) pain syndrome up to shock
c) horizontal fluid level in the pleural cavity
d) tachycardia
e) change in percussion sound.

9. The main early symptoms of central endobronchial lung cancer are:
a) cancerous pneumonitis
b) recurrent pneumothorax
c) atelectasis of the lung area
d) shortness of breath and pain on breathing
e) hacking cough and hemoptysis.

10. Pencost's cancer is:
a) central cancer of the middle lobe
b) peripheral cancer of the lower lobe
c) central cancer of the upper lobe
d) peripheral apex cancer
e) cavity form of peripheral lung cancer.

1

SITUATION TASKS

1. A 40-year-old man, after being drunk, slept on the street for 4-5 hours. After 2 days, he had a fever and pains in the chest. Subsequently, the temperature rises to 39 degrees. After 2 weeks, suddenly when coughing, about 200 ml of pus with an unpleasant odor left.

What is your preliminary diagnosis?

2. A patient with an abscess of the right lung developed sharp pains in the chest, shortness of breath. R - logically: collapse of the right lung, a wide horizontal fluid level, a sharp shift of the mediastinal shadow to the left.

Diagnosis?

3. A 70-year-old patient with an acute lung abscess with a diameter of up to 10 cm, located near the chest wall, has signs of severe intoxication. Which treatment should you prioritize?

4. Patient 42 years old, complains of paroxysmal dry cough, periodic hemoptysis. Patient for 8 years. On tomograms in the lumen of the right main bronchus, a rounded shadow with a diameter of 1.2 cm with clear contours is determined. What is your diagnosis?

4. A patient has peripheral cancer of the lower lobe of the lung with parietal germination. Morphologically - squamous cell carcinoma. Intraoperatively, no metastases were detected in the lymph nodes of the lung root. What kind of treatment does the patient need?

TOPIC: DISEASE OF THE PLEURA. ACUTE AND CHRONIC EMPIEMAS OF THE PLEURA

EMPIEMA OF THE PLEURA

Inflammation of the pleura is called pleurisy. Purulent inflammation of the pleura or accumulation of pus in the pleural cavity is called pleural empyema.

Empyema reasons:

Empyema of the pleura is, as a rule, a complication of some other disease.

I. 1. Lung disease (abscess, festering EB) is smiling cause empyema 90% - secondary empyema.

P. 1. After a penetrating wound of the chest, especially during the war.

2. After operations on the lungs, as well as in other organs of the chest. Empyema may also develop.

This is the primary pleural empyema.

Bacterial microflora:

- in the old days, Staphylococcus aureus occupied the 1st place in the etiology of empyema. And currently it causes empyema in 25-30% of cases.

- currently dominated by:

Gr (-) Klebsiella pneumonia,

Pseudomonas aeruginosa,

enterobacteriaceae, Escherichia coli.

non-clostridial non-spore-forming anaerobes (bacteraids, fusobacteria, peptococci, peptostreptococci). Non-clostridial anaerobes are more likely to cause empyema.

Pathogenesis

The ways of implementation are different:

- at length - parapneumonic (simultaneously with pneumonia moniey)

- metapnevmonicheskie - after purulent pneumonia and Zabolev Nij mediastinum and chest

- hematogenous and lymphogenous pathways (in 5% of cases).

When pneumonia and OIDL, per continetatum, infection directly by falls in the pleura. According to S.I. Spasokukotsky with pneumonia, when there is no

obvious destruction, even then there is a small focus of destruction of the subcortical location, which breaks into the pleura.

In wounds - infective gets into the wound and chest hydrochloric cavity with the projectile, bullet, shrapnel wounding subject.

With p / o complications, the genesis of empyema is as follows:

a) no operation takes place under absolutely aseptic conditions;

b) adhesions, mooring lines, as a rule, contains a dormant infection.

c) the infection comes from the culture of the bronchus, with the primary failure of the bronchial stump.

d) the development of pleural empyema is facilitated by the dissociation of the pleural sheets - i.e. the presence of a residual cavity filled with air, blood or other exudate when the lung collapses (collapse).

PS Serous (or sympathetic, in the old way) pleurisy in pneumonia is also a good nutrient for microbes.

Pneumo- or hemothorax with a penetrating wound also contributes to the development of empyema.

e) coagulated hemothorax is a good ground for empyema, as it does not dissolve without surgery.

The elastic resistance of the lung (traction) contributes to the collapse of the lung and conditions for the residual cavity and empyema.

The residual cavity must be eliminated on the very first day - then there will be no suppuration.

If the lung is not straightened, for example, with an abscess, when it is folded by a pleurobronchial fistula, then yaibrin settles on the pleura, it thickens, moorings are formed over the entire surface and pleural empyema is formed.

Bronchial fistulas:

- broncho-pleural fistula
- pleuro-cutaneous fistula
- broncho-pleuro-cutaneous fistula

They give an empyema discovery of the form.

Length characteristics: limited - involvement of one wall of the pleural cavity; common - the involvement of 2 or more walls of the pleural cavity; total - involving all Pleven tral cavity from the diaphragm to the pleura dome.

Characteristics of the degree of lung collapse:

I degree - collapse of lung tissue within the cloak.

II degree - collapse of the lung tissue within the trunk.

III degree - collapse of the lung tissue up to the nucleus.

Clinical and symptomatology

The initial manifestations of the disease are often masked by the clinic of the disease, which is complicated by empyema. Prop Twain empyema seen complaints: pain in his side.

- inability to take a deep breath
- cough with a little phlegm,
- temperature rise to high numbers (39-40 °), with large daily ranges (subfebrile temperature in the morning).

- shortness of breath.
- loss of appetite.
- sweating at rest.

Objectively:

- shortness of breath at rest.
- cyanosis, facial features are pointed.
- intoxication of the body with the contents of the pleural cavity with pus.
- the intercostal spaces are smoothed.
- the skin is thickened.
- heat.
- pulse - 110-120 in 1 min.
- the affected side lags behind in breathing.
- adynamia is noted.

Physical signs: marked blunting of roll stands in the thoracic ki if exudate over 250-300 ml.

Garland - a dull-tympanic sound due to a purged lung exudate.

Rauchfus-Grokko - dullness due to displacement of the mediastinum to the healthy side.

The phenomenon of Skoda - timbrels, and with open mouth - noise split of the pot.

When pleural empyema fluid level horizontal and spe mong it is not visible (shaded) of the thin layer of liquid in these areas.

Puncture data of the pleural cavity with cytology and microflora

Usually up to 0.5-1 liters of exudate is produced in the pleural cavity (purulent). In addition, the light goes suppurative about the process of

- therefore, protein losses are large, hypoproteinemia develops
- up to 3.5 - 4 gr% and dysproteinemia - coefficient of alb / glob. at empyema becomes 0,7-0,6 (1.5 vs. normal) and led Chiva diuresis, as water poorly retained due gipopro-teinemii and disproteienemii decreases oncotic pressure in the blood. Albumin is also known to retain water more than gamma globulins.

Hypokalemia develops. Erythropoiesis is impaired and anemia develops rapidly. Reduced immunity.

From special studies:

- fluoroscopy and chest x-ray are performed.
- pleurography of the residual cavity.

Prevention

1) creating conditions for the contact of 2 sheets of the pleura.

2) elimination of residual cavities (puncture, aspiration, suction)

3) elimination of pneumatic and hemothorax (draining and ASPI walkie from the pleural cavity).

With coagulated hemothorax fibrinolytics: streptokinase, streptase dissolves blood clots.

4) maximum hemostasis during surgery.

5) Good vacuum suction to the pleural cavity of executions Lenia lung.

Treatment

Acute empyema - conservative or semi-conservative, le chenie OIDL.

Principle I - evacuation of pus from the pleural cavity (puncture and or drainage).

- puncture for closed empyema (for pneumonia, etc.).

- thoracostomy aspiration and straightening of the lung under vacuum (80 ml Hg). In the past, the Bulau underwater drainage was widely used.

Method II of temporary occlusion of bronchial fistulas with a foam sponge.

III principle - for antibacterial treatment chuvstvitelnos five, large doses.

Principle IV - normalization of homeostasis:

iv proteins, albumin, prep. K hemosorbtion (detoxification ex trakorporalnaya).

Chronic empyema treatment

In chronic empyema, pleural tolycin reaches 1-2 cm .

The treatment of chronic empyema is based on 2 principles:

I - Creation of the chest wall resection mobility by D Azur.

Thoracoplasty - intraplevralnaya (Schede) currently BPE on me does not apply.

- extrapleural.

Stair thoracoplasty according to Geller-Limberg.

It is used most often intra-muscular extrapleural plastic (Kirchner) is used for small wasps tatochnyh cavities.

With bronchopleural fistulas - bronchial tamponade with a muscle on the leg - Abrazhanov's operation.

II - release of the lung from the mooring - decortication of the lung (Delorme).

Pleuropulmonectomy is the removal of the costal pleura, moorings and visceral pleura with a pathologically altered lung.

Pleurectomy is the removal of conditionally all sheets of the empyema cavity wall.

QUESTIONS

to the audience to establish feedback and clarify the achievement of the lecture goal

1. What is the difference between abscess and lung gangrene?
2. What are the main diagnostic methods for OBDL?
3. Clinic of an undrained abscess.
4. Clinic of a drained abscess.
5. Complications of OBDL.
6. Methods for stopping pulmonary bleeding.
7. First aid for valve pneumothorax?
8. Indications for surgery for lung abscess.
9. Types of pleural empyema encapsulated.
10. Preoperative preparation of pleural empyema.

SUBJECT : DISEASE MEDIASTINUM. CYSTS OF THE LUNG AND MEDIASTINUM. PNEUMOTHORAX. MEDIASTENITIS. REASONS, CLASSIFICATION, CLINIC, DIAGNOSTICS AND TREATMENT.

The value of studying the topic. In the structure of surgical and infectious diseases, diseases that occur with pneumothorax occupy a significant place. This is primarily the opening and closing of lung damage and lung disease. Sometimes difficulties arise in their differential diagnosis. Specialists of different profiles are faced with different types of pneumorax.

All of the above dictates the need to study the principles of differential diagnosis of pneumothorax by 5-year students.

The purpose of the lesson:

1. To teach students the etiology, pathogenesis, clinical picture, diagnosis and differential diagnosis of pneumothorax, as well as the hand of the lung.

Test questions:

1. Normal anatomy of the chest, pleural cavity.
2. The concept of hemothorax and pneumothorax
3. Classification of pneumothorax
4. X-ray diagnostics of pneumothorax and hemothorax
5. Clinic and features of the course of diseases in which there is pneumothorax and hemothorax. Know the mechanism of development of pneumothorax.
6. Modern methods of treatment of pneumothorax and hemothorax.

Practical skills

1. Correctly examine the patient.
2. Registration of medical history.
3. Correctly interpret the results of radiographic images.
4. Correctly palpate the chest.
5. Assess the severity of the disease.
6. Correctly make appointments to the patient, calculate the dose of drugs.
7. Determine the patient's blood group.
8. Learn how to perform a pleural puncture.
9. Learn to conduct intercostal blockades.

THEORETICAL PART

Pneumothorax

Pneumothorax is a pathological condition of the body, due to the presence (intake) of air in the pleural cavity.

Etiology

1. Penetrating chest wounds
2. Damage to the lung (bronchus)

Penetrating chest wounds may be due to mechanical damage or surgical trauma

Damage to the lung (bronchus) is possible with a closed injury and spontaneous (spontaneous for no apparent reason).

Possible causes of spontaneous pneumothorax:

- Bullous emphysema
- Lung cysts
- Pneumosclerosis
- Pleural adhesions
- Pulmonary tuberculosis
- Lung abscess associated with bronchus.

Pneumothorax can be unilateral or bilateral.

Pathogenesis of homeostasis disorders in pneumothorax.

1. Collapse (collapse) of the lung
2. Violation of pulmonary ventilation - deterioration of blood oxygenation conditions
3. Violation of oxygen transport, the development of hypoxemia and hypoxia

Depending on the nature of the communication of the pleural cavity with the external environment, there are **closed, open and valve pneumothorax** . All of them are found in open lesions, when the integrity of the skin and parietal pleura is disturbed.

Closed pneumothorax is formed when a wound hole in the soft tissues of the chest and lung quickly closes as a result of tissue displacement and their traumatic edema; in the further, air does not enter the pleural cavity through it. The volume of air trapped in the pleural cavity can be significant or very small, practically undetectable by conventional research methods.

Open pneumothorax. If the wound of the chest wall gapes, then there is always a chain of unfavorable anatomical and functional changes. When inhaling, a portion of air, entering the pleural cavity, squeezes the lung, the pliable parts of the heart and the hollow veins, sharply pushes the mediastinum to the healthy side, and the diaphragm downward. When you exhale, air is pushed out of the pleural cavity: the lung, devoid of elastic traction of the chest, partially straightens. Paradoxical breathing appears, in which, on inhalation, part of the air saturated with carbon dioxide enters the healthy lung from the affected lung, and on exhalation it rushes in the opposite direction. As a result, not only the lung on the side of damage is turned off from gas exchange, but the efficiency of the respiratory function of a healthy lung is noticeably reduced, general and pulmonary hemodynamics are significantly impaired, hypoxemia develops, which, in combination with irritation of the nerve formations of the mediastinum during flotation, leads to an increase in functional disorders.

Valvular pneumothorax. Severe respiratory and circulatory disorders also occur with valvular pneumothorax. With each inhalation, air on the side of the injury is forced into the pleural cavity through the wound of the chest wall or bronchus, more and more squeezing the lung and pushing back the mediastinum, since as a result of the valve mechanism it cannot go out (Fig.). Thus, intra pleural compression occurs, rapidly leading to severe respiratory and cardiovascular failure.

Hemothorax is a consequence of damage to the vessels of the chest wall (intercostal, internal chest, etc.) and the lung. Less often, dangerous wounds of the heart, aorta, hollow and pulmonary veins are detected. Depending on the amount of blood poured into the pleural cavity, small (in the pleural sinuses), medium (to the level of the scapula angle) and large hemothorax are distinguished (Kupriyanov P.A., 1946). By the time the victim is examined, the bleeding may either stop or continue. Disorders of gas exchange and cardiac activity depend on the volume of blood loss and on the degree of collapse of the lung on the affected side.

With closed chest injuries, accompanied by rib fractures, their fragments can penetrate into the lung tissue, tearing it. In such cases, a closed or valvular hemopneumothorax occurs, and through the torn parietal pleura, air spreads into the cellular spaces of the chest wall (subcutaneous emphysema).

Clinical presentation and diagnosis. Diagnosis of closed trauma and chest wounds is often difficult due to the severity of the condition of the victims and the dynamism of clinical manifestations caused by the growth of pathological changes; therefore, it is very important to know the mechanism of injury, the time elapsed since the moment of injury, and the nature of care at the prehospital stage.

Breast lesions have a number of common diagnostic features:

- pain of varying intensity on the side of the wound, aggravated by inhalation, coughing, changes in body position, often with a sharp restriction of respiratory movements, especially if the skeleton is damaged;
- shortness of breath and shortness of breath, also aggravated by movements, which, together with pain, forces the victim to take a forced body position;
- hemodynamic changes of varying severity;
- hemoptysis of varying intensity and duration;
- emphysema in the tissues of the chest wall, mediastinum and adjacent areas;
- displacement of the mediastinum in the opposite direction from the wound site;
- other physical changes.

Some of them are noted in the vast majority of victims (pain, shortness of breath), others are much less common (emphysema, hemoptysis).

A systematic physical examination, including examination, palpation, percussion, auscultation, examination of the nature and localization of wounds, etc., is always essential in assessing the condition of the victim, even in the absence of other research methods. damage and take urgent remedial action. The data obtained also serve as the basis for choosing the type and sequence of clarifying diagnostic techniques. In emergency situations, to detect hemopneumothorax and pneumothorax, ongoing bleeding or hemopericardium, a diagnostic and treatment puncture is very useful.

Methodically correctly performed, it can easily establish the presence of air or blood in the cavity of the pleura and pericardium, and, if necessary, remove them.

Despite the rather high information content of the physical examination, the main role in clarifying the nature of the lesion belongs to the radiation method of research, the implementation of which should be considered mandatory for all chest injuries. Sound echolocation is a promising method that significantly complements the data of other studies. It can be used to establish the thickness of the pleura, the contents of the pleural cavity, the mobility and airiness of the lung, foreign bodies that block X-rays.

Symptoms for closed chest trauma depend on the severity of the chest wall damage, the severity of concomitant pneumo - and hemothorax, the degree and extent of damage to the lung, heart, bronchi and other organs.

With relatively small chest bruises, the clinical picture is usually mild. The main complaint of the victims is pain in the bruised area, aggravated by deep breathing and movement.

In cases of more serious damage, pronounced disorders of the general condition are usually observed. Severe pain in the area of injury, forced position of the victim, numerous abrasions and subcutaneous hemorrhages, chest wall wounds that do not penetrate the pleural cavity, severe shortness of breath, cyanosis, increased and weakened pulse, chest deformity, paradoxical movements of its individual fragments or lagging behind breathing one or the other half indicate the severity of the closed injury. Physical examination provides an additional basis for clarifying the diagnosis. Palpation of the breast allows you to identify subcutaneous emphysema, sites of rib fractures, to determine the intensity of voice tremors. A shortened percussion sound indicates the presence of hemothorax or lung atelectasis. Tympanitis is characteristic of pneumothorax. Percussion also manages to establish the boundaries of the lungs, heart, mediastinal displacement, etc., and with auscultation to note the absence or weakening of breathing.

Plain radiographs reveal fractures of the breast skeleton, the presence of free gas and fluid in the pleural cavity, displacement of the mediastinal organs, diaphragm, collapse or atelectasis of the lung, mediastinal emphysema and other signs.

Symptoms of non-penetrating chest wounds depend on the nature and extent of the injury. In cases of blind, through or tangential wounds of the chest wall, the general condition usually suffers little, respiratory and cardiovascular disorders are not significantly expressed.

It is important to remember that with tangential injuries to the chest as a result of the force of the lateral impact of the injuring projectile, severe bruises of the internal organs and, first of all, the heart and lungs can occur.

Penetrating chest wounds are usually accompanied by damage to internal organs, primarily the lungs, heart, large vessels, etc. Quite often there are associated wounds, when other areas of the body are simultaneously damaged. These types of injuries are distinguished by the severity of the course and high mortality, even despite the timely provision of medical care.

In general, the clinical picture of penetrating chest wounds depends mainly on the nature of the destruction of the intrathoracic organs and the massiveness of hemothorax and pneumothorax.

Diagnosis of open chest injuries with perforating wounds does not cause serious difficulties. Comparison of the entrance and exit holes of the wound channel creates an idea of the possible movement of the wounding projectile and the involvement of certain organs in the pathological process. With blind wounds, diagnosis can be difficult. By the time the victim is admitted to the hospital, respiratory disorders that have arisen after the injury remain insignificant, shortness of breath is noticeable only during physical exertion.

On radiographs, respectively, the affected side is determined by areas of enlightenment in the form of a larger or smaller zone devoid of pulmonary pattern. The lung is collapsed, the mediastinum is displaced in the opposite direction.

When lung tissue or blood vessels of the chest wall, less often the mediastinum are injured, the clinical picture depends on the amount of blood loss and the amount of blood accumulated in the pleural cavity.

Small hemothorax is manifested by minor clinical signs. Dysfunctions of the cardiovascular and respiratory systems are minimal and short-lived or absent at all.

With medium and especially large hemothorax, the clinical picture is more severe: the victims complain of general weakness, severe chest pain, shortness of breath. An objective examination reveals signs of respiratory failure and hemodynamic disorders (cyanosis, pallor of the skin, cold sweat, rapid breathing, small and rapid pulse, drop in blood pressure). With percussion, signs of fluid accumulation in the corresponding pleural cavity are found. X-ray is determined by

uniform intense shading of the greater part or even the side opposite from the wound. A decrease in the number of erythrocytes, hemoglobin content and hematocrit value reflect the degree of exsanguination of the body (Fig. ??).

When puncture of the pleural cavity, 1 liter of blood or more is evacuated, and often it accumulates again. In cases of continued bleeding, the blood obtained during the puncture coagulates, since it does not have time to undergo the fibrinolizing effect of the pleura.

The victims are agitated, frightened, suffer from sharp pain in the wound and excruciating cough. Those who do not have an occlusive dressing on the wound tend to close the gaping defect of the chest wall with their hand. Respiratory disorders clearly predominate in the general clinical picture. Cyanosis of the skin, cold sweat, severe shortness of breath, lowering blood pressure, a small and frequent pulse indicate the severity of the condition of the wounded. Examination of a gaping chest wound, communicating with the pleural cavity, through which air passes with noise in both directions, provides the basis for establishing a definitive diagnosis. When coughing or changing position of the body, frothy blood may flow out.

Physically determined pneumothorax with almost complete collapse of the lung and mediastinal displacement in the opposite direction. In most observations, hemothorax, expressed to a greater or lesser extent, can also be detected, collapse of the lung, displacement of the mediastinum in the opposite direction, and horizontal fluid level can also be detected (Fig. 106).

Establish the nature of bone damage, localization of foreign bodies. noticeable changes in blood (anemia) can be detected: a significant decrease in the hemoglobin content, hematocrit number and the number of erythrocytes, valvular pneumothorax are observed in a small group (1-2%) of the total number of victims, but differ in a significant severity of functional changes. In these cases, during the examination, most of the symptoms that occur in other patients are observed, along with signs of hypoxia and hemodynamic disorders, a pronounced growing subcutaneous emphysema of the chest wall is striking, often extending to the neck, head, limbs, and abdomen.

The wounded to the valve pneumothorax requires urgent surgical care, without which they quickly die in connection with the progression of respiratory and cardiovascular disorders. In cases of very dangerous mediastinal emphysema, the subcutaneous air cushion first of all appears on the neck, in the region of the jugular notch, and from there spreads symmetrically to both halves of the body.

Basic principles of surgical care. Improving the outcomes of treatment of victims with breast injury largely depends on the clarity of the organizational support for the provision of care at all stages of medical evacuation. The measures carried out at the scene of the accident, during the period of transportation and further in the hospital, should always be pathogenetically justified, aimed at reducing the period of acute functional disorders and possibly quickly removing the wounded from shock.

In general, the staged treatment of victims with chest injuries is as follows. At the scene (in the lesion focus), a protective aseptic bandage is applied to the chest wound. Stop external bleeding with a pressure bandage. In open pneumothorax, the gaping chest wound is sealed with an occlusive dressing. In cases of asphyxiation, the oral cavity is cleaned of blood, mucus and foreign bodies; if indicated, artificial respiration is performed using an S- shaped air duct. All victims are administered analgesics, cardiac drugs and carried out on a stretcher, preferably in a half-sitting position.

With a tense pneumothorax, the pleural cavity is punctured with a thick needle (Dufo type) in the second intercostal space along the midclavicular line, with its fixation to the skin with a plaster. A rubber flap made from the finger of a surgical glove is attached to the free end of the needle. If necessary, resort to artificial or assisted breathing.

In the cold season, the victim should be surrounded by heating pads and wrapped in a blanket. If there are signs of exsanguination and a drop in blood pressure due to vital indications, infusion therapy (polyglucin, saline solutions, glucose) is performed, which, however, should not interfere with the transportation of the wounded.

After first aid has been provided, those wounded in the chest always need emergency evacuation to a hospital.

The general schematic diagram of the treatment of patients with chest injuries in a surgical hospital includes:

- early and complete drainage of the pleural cavity;
- replenishment of blood loss;
- effective maintenance of airway patency;
- elimination of pain;
- sealing and stabilization of the chest wall;
- antimicrobial and supportive therapy.

Clinical experience shows that each of the groups of victims with chest injuries, while maintaining the concept of providing assistance to them, has its own characteristic features.

In complex therapy, in addition to draining the pleural cavity, almost all victims are shown the appointment of painkillers, antibiotics, oxygen therapy and respiratory gymnastics. At the same time, the use of non-narcotic analgesics simultaneously with intercostal or paravertebral novocaine blockade provides quite satisfactory anesthesia.

In most cases of penetrating chest wounds, initial surgical debridement is required. It consists of a layer-by-layer dissection of tissues through the wound canal, excision of non-viable and contaminated areas of subcutaneous fat, fascia and especially muscles, resection of damaged ribs, removal of spilled blood and clots, foreign bodies, ensuring thorough hemostasis. With through and blind wounds without torn edges, when the diameter of the inlet does not exceed 2-2.5 cm, there is no open pneumothorax and damage to large vessels, surgical treatment can be omitted, limiting the wound to the toilet.

Patients with closed pneumothorax with a generally satisfactory condition are punctured or, more often, the pleural cavity is drained with a thin plastic tube with a diameter of 0.5-0.6 cm in the second intercostal space along the midclavicular line, followed by active aspiration by a vacuum system at a constant vacuum of 30-40 mm of water. Art. (fig. ??). During active aspiration, it is necessary to ensure constant airway patency, timely elimination of atelectasis, which often prevents the full expansion of the lung. According to indications, surgical treatment of wounds is performed, analgesics are administered, and breathing exercises are prescribed. There is usually no need for wide thoracotomy in this group of wounded.

In cases of hemothorax and hemopneumothorax, the features of treatment largely depend on the severity of the condition of the wounded and the amount of blood loss. The common thing is the obligatory drainage of the pleural cavity with wide-lumen drainage tubes with a diameter of 14-15 cm.

Technique of pleural cavity drainage .

In the seventh to eighth intercostal space along the middle axillary line, soft tissues are anesthetized with a 2% solution of trimecaine. The integument in the intercostal space is pierced with a scalpel, focusing on the upper edge of the underlying rib in order to avoid damage to the intercostal nerves and blood vessels. A prepared wide-lumen tube with an additionally cut lateral opening is grasped with a curved clamp or forceps and inserted through the incision into the pleural cavity after removing the scalpel (Fig. ??). The edge of the skin wound next to the drain is stitched with one fixing suture and the tube is fixed with it. The outer end of the tube, equipped with a valve, is lowered into a vessel with an antiseptic solution. Often, in patients with hemopneumothorax, to remove air and more quickly and reliably expand the lung, it is necessary to install a second tube from the blood transfusion system (5-6 mm in diameter). It is introduced using a trocar in the second intercostal space along the midclavicular line and connected to a water-jet or electric suction.

In wounded with small hemothorax with minor and short-term respiratory and circulatory disturbances, therapeutic measures include puncture or drainage of the pleural cavity with wide-lumen tubes, prescription of painkillers, antibiotics and breathing exercises. Infusion therapy is carried out in a volume of 500-800 ml. Usually, there is no need for surgical treatment of skin wounds, which heal well under the scab. In cases of medium and especially large hemothorax, a more extended amount of care is used. In addition to draining the pleural cavity, the contents of the respiratory tract are persistently removed due to the frequent excessive accumulation of secretions in the tracheobronchial tree, and inhalation of humidified oxygen is widely used, thus improving blood oxygenation.

When signs of heart failure appear, cardiac glycosides and steroid hormones are prescribed, bronchodilators and antihistamines are simultaneously administered. Clinical experience shows that the majority of patients with secondary hemothorax require intravenous infusion of predominantly plasma-substituting fluids, less often blood, only on the day of admission. It is advisable to use saline solutions, 5% glucose solution, low molecular weight dextrans (total volume of liquids 1300-1500 ml).

With a large hemothorax with a blood loss of more than 1000-1500 ml, infusion-transfusion agents, together with a complex of respiratory therapy, play a particularly important role. On the day of admission, 2000-2500 ml of fluid is poured into each victim, including mandatory blood transfusions. Intravenous administration of plasma-substituting fluids is also necessary in the next 2-3 days (total volume 4500-5500 ml).

In 8-10% of patients with medium and large hemothorax, there is a need for wide thoracotomies. In the first hours and days after injury, in 55-60% of them, the indication for surgery is ongoing intrapleural bleeding. At a later date, the cause of the intervention is coagulated hemothorax, persistent collapse of the lung, or pleural empyema.

The amount of surgical care for wounded with open pneumothorax depends on the size and nature of the injury. Most of the victims need urgent operative closure of the wound defect of the chest wall and mandatory drainage of the pleural cavity, the main task of which is to fully expand the lung. To shorten the duration of the period of functional disorders and possibly quickly remove the wounded from shock, in the order of preoperative preparation, short-term (within 40-60 minutes) anti-shock therapy is always justified. It is aimed at combating pain, respiratory failure and blood loss. Preoperative therapeutic measures should include control over the tightness of the pleural cavity, the introduction of analgesics, the implementation of intercostal, paravertebral and subpleural novocaine blockades, effective maintenance of airway patency, inhalation of humidified oxygen, replenishment of blood loss, the introduction of cardiotonic drugs, bronchodilators, steroid hormones. It is required to drain the pleural cavity with wide-lumen tubes before the operation, and not after the surgical treatment of the chest wound.

PRACTICAL PART Thematic tests

1

1. The patient has multiple rib fractures, acute respiratory
failure. After tracheal intubation and transfer to mechanical ventilation, the state is sharply worsened, hypoxia is increasing, blood pressure dropped to 80 mm Hg, deaf heart sounds. The most likely reason for the deterioration is:
Due to rupture of the thoracic aorta
Incorrect positioning of the endotracheal tube
Aspiration of gastric contents
+ Tension pneumothorax
Severe contusion of the lungs and heart
2. Puncture of the pleural cavity with closed pneumothorax is carried out:
In the 6th intercostal space along the posterior axillary line
In the 6th intercostal space along the midclavicular line
In the 2nd intercostal space along the posterior axillary line
+ In the 2nd intercostal space along the midclavicular line
3. Mark the intercostal space in which the puncture should be performed in case of valvular pneumothorax:
+ In II
In III
In IV
In V
In IX
4. Specify along which edge of the rib the needle should be inserted when puncture of the pleural cavity:
On the bottom
Vertebral
+ Top
Sternal

5. For what purpose is the Ruvillois-Gregoire test intended?
To determine the degree of respiratory failure
Festering hematrax
+ Definitions of ongoing bleeding
The amount of air in the pleural cavity
6. Specify the methods of sealing the pleural cavity with open pneumothorax at the stage of medical care
Applying aseptic dressing
+ Sealing occlusive dressing
Suturing a skin wound
7. Should drainage of pleural aid be performed with closed pneumothorax at the stage of qualified medical care?
Yes
+ No
8. Should an injured person with internal valvular pneumothorax and subcutaneous emphysema wear a chest bandage during the skilled care stage?
Yes
+ No
9. What measures should be taken immediately in case of tension pneumothorax?
+ Transfer it to open by puncture of the pleural space

Mechanical ventilation
Tracheal intubation
10. Specify the types of hemothorax:
Limited
Welterweight
Maximum

Total

1

DISEASES OF THE ORGANS OF THE MEDIUM, MEDIASTENITIS

The purpose of the lecture : Introduction of students with diseases of the mediastinal organs, medistanitami, reasons and E for their development, clinical features, complications over n GOVERNMENTAL forms, differential diagnosis, best practices Leche e Nia, postoperative care, rehabilitation patients.

Educational purposes lectures: Suggestion students need for timely adequate operation to the development of serious complications , and in their development - meeting the most informative and modern methods of diagnosis and surgical treatment of patients, familiarity with in s possible complications is the operation and the operational period of Prof. and galaxies, ... Development of students' clinical thinking. Development of present-day e alternating view of the problem the issue from the perspective of world medicine and general practitioners.

Lecture objectives:

22. To give an idea about diseases of the mediastinal organs, mediastinitis.
23. Explain the causes and mechanisms of complications.
24. Give a clinical description and possible variants of the course of the disease.
25. Make a differential diagnosis with other diseases I mi.
26. To acquaint students with modern and most informative s methods, which examination of patients
27. Demonstration of examples of their surgical practice: patients, slides, phlebograms.
28. All the lecture material for students to prepare and present, in an b Birmingham, necessary for quality training of general practitioners.

PLAN OF THE LECTURE.

19. The urgency of the problem - 5 min
20. Etiopathogenesis of diseases of the mediastinal organs, mediastinitis.
21. Clinical picture - 10 min

a) Etiopathogenesis;
b) Clinic and diagnostics.
c) Differential diagnosis.
d) Treatment.

22. Diagnostics. - 10 min
23. Differential diagnostics. - 10 min
24. Treatment - 15 min

7. Disease prevention - 10 min

Anatomical and physiological features

The mediastinum is the part of the thoracic cavity, bounded from below by the diaphragm, in front of the sternum, behind the thoracic spine and neck of the ribs, from the sides by the pleural sheets (right and left mediastinal pleura). Above the handle of the sternum, the mediastinum passes into the cellular spaces of the neck. As a result, the upper border of the mediastinum is considered to be a conditional horizontal plane passing along the upper edge of the sternum handle. In clinical practice, it is convenient to distinguish four sections of the mediastinum. A conventional line drawn from the place of attachment of the handle of the sternum to the body of the sternum towards the IV thoracic vertebra divides the mediastinum into superior and inferior. Heart Bag divides the lower mediastinum on the front, middle and back Nij departments. In the upper mediastinum are the proximal trachea and esophagus, the thymus gland, the aortic arch and its main branches, the thoracic lymphatic duct, the left and right brachiocephalic veins. In the anterior mediastinum, between the pericardium and the sternum, the distal thymus, adipose tissue, and lymph nodes are located. The middle mediastinum contains the pericardium, heart, intrapericardial sections of large vessels, tracheal bifurcation and main bronchi, pulmonary arteries and veins, bifurcation lymph nodes. In the posterior mediastinum, bounded in front by the bifurcation of the trachea and the pericardium, and behind the lower thoracic spine, there is the esophagus, the descending thoracic aorta, the thoracic lymphatic duct, sympathetic and parasympathic (vagus) nerves, and lymph nodes.

MEDIASTINAL DAMAGE

Allocate open and closed damage to the mediastinum.

Klee nickname and diagnostic ka : about clinical phenomena injury depend FNF on what op gan mediastinum on vrezhden, the intensity of the internal or ruzhnogo bleeding. When closed harmed Denia mediastinal prac cally are always internal hemorrhage with hematoma formation, which mo Jette cause compression of vital organs (especially thin-walled veins mediastinum). When the recurrent nerve is compressed, a dry cough, hoarseness of the voice occurs; when compression of the boundary sympathy Cesky barrel - Horner syndrome (ptosis, miosis, endophthalmitis).

Diagnosis is based on anamnesis data (character elucidation injury), disease symptoms dynamics (evolution under cutaneous emphysema, respiratory disorders, and others.).

When rentgenolo based surveys, seen in the displacement of the mediastinum or Dru any direction, its extension due to hemorrhage. Significant enlightenment of the mediastinal shadow is a symptom of mediastinal emphysema. Treatment is aimed at normalizing the functions of vital organs (heart, lungs). Anti-shock therapy is performed; in violation framing functions thorax (due to the inability of active respiratory movements) for changing ventilator. Indications for surgical treatment: compression of vital organs with sharp violation of their functions, esophageal tears, trachea, main bronchi, the large krovenos GOVERNMENTAL vessels with continued bleeding. Open injuries are usually combined with damage to the mediastinal organs, which is accompanied by appropriate symptoms, as well as bleeding, the development of mediastinal emphysema. For open injuries of the mediastinum, surgical treatment is indicated. The choice of the operation method depends on the nature of the damage, the degree of wound infection, the general condition of the patient.

INFLAMMATORY DISEASES

Acute mediastinitis is an acute purulent inflammation of the mediastinal tissue, which occurs in most cases in the form of phlegmon and much less often in the form of a limited abscess - an abscess. Most often, acute mediastinitis occurs as a result of open injuries of the mediastinum, perforation of the esophagus by a foreign body, with instrumental examination of the trachea and main bronchi, with incompetence of the sutures after operations on the esophagus, with the spread of deep phlegmon of the neck to the mediastinal tissue.

Clinical picture and diagnosis : acute purulent mediastinitis develops rapidly, leading to a serious condition of patients. Chills, high body temperature, tachycardia, shock, shortness of breath, stitching and bursting pains in the chest and neck are characteristic. The localization of pain depends on the localization of the inflammatory process. The pain intensifies when the neck is extended and the head is abducted posteriorly, when the sternum is pressed (with anterior mediastinitis), and the posterior ribs are pressed (with posterior mediastinitis). Because of the pain, patients take a forced position (half-sitting with the head tilted forward), which reduces pain. With perforation of the esophagus, trachea or bronchi, mediastinal and then subcutaneous emphysema occurs. A general blood test reveals a high leukocytosis with a shift of the formula to the left, an increase in ESR. X-ray examination determines the expansion of the mediastinal shadow, with perforation of the hollow organs - gas against the background of the mediastinal shadow.

Treatment : surgical, aimed primarily at eliminating the cause that caused acute mediastinitis. In case of open injuries with the presence of a foreign body in the mediastinum, ruptures of the esophagus, trachea or main bronchi, failure of the sutures of the esophageal anastomoses, emergency operations are indicated in order to eliminate the source of infection of the cellular spaces of the mediastinum. Surgery is completed by draining the mediastinum. Some surgeons recommend treating acute mediastinitis with active aspiration from a purulent focus through double-lumen drains introduced into the mediastinum. An antiseptic solution (furacilin, dioxidine, chlorhexidine) in combination with broad-spectrum antibiotics, proteolytic enzymes is brought to the purulent focus through a thin channel of the drainage tube. Exudate is

aspirated through a wider channel. Prolonged washing of the purulent cavity with active aspiration allows you to remove non-viable tissues, pus, and suppress the growth of microbial flora. Depending on the localization of the abscess, drainage can be carried out through the cervical, transthoracic, transsternal or laparotomic access. An important role in the treatment of patients with mediastinitis belongs to massive general antibiotic therapy, detoxification, infusion therapy, parenteral and enteral ("tube") nutrition. "Probe" food - drip of liquid nutrients and food on thin probe about REFERENCE duodenum or jejunum via an endoscope. Chronic mediastinitis (and mediastinal fibrosis) can be the outcome of acute mediastinitis, as well as tuberculosis, actinomycosis, and syphilis. There is also an unclear etiology of sclerosing (idiopathic) mediastinitis, characterized by the proliferation of connective tissue along the formations of the mediastinum. Chronic mediastinitis is observed much less often than acute.

Clinic and diagnostics : symptoms usually ACS naya - body temperature is normal or subfebrialnaya, chest pain, not intensive, although the general condition of patients is gradually deteriorating. A consequence of chronic prolonged IU diastinita can be mediastinal fibrosis tissue, growing of intrathoracic lymph nodes, followed by compression of vital organs - the superior vena cava, esophagus, trachea, large bronchi. Diagnosis is based on data anam Nez detectable by X-ray examination, extension rhenium shadow of the mediastinum, serological blood tests for tuberculosis, actinomycosis, syphilis. Also used rentgenolo based surveys with contrast of the esophagus, the top of Loi veins.

Treatment : Most patients with drug on the Management Board to suppress the inflammatory process that caused the development of chronic mediastinitis (TB Tera Pius treatment of actinomycosis aktinolizatom and antibiotics, and others.). Idiopathic sclerosing mediastinitis used lu chevuyu therapy corticosteroids. In the case of compression of vital formations of the mediastinum, one has to resort to surgical treatment - removal of mediastinal lymph nodes, elimination of cords and adhesions that caused compression of organs.

QUESTIONS

to the audience to establish feedback and clarify the achievement of the lecture goal

1. Tell the anatomical features of the mediastinum.
2. Classification of mediastinal damage.
3. Clinical features of mediastinal damage.
4. Classification of mediasthenitis.
5. Features of surgical treatment of mediastinal cysts.
6. Clinical picture of medistanitis.
7. Prevention of mediasthenitis.

TOPIC : DISEASE OF THE DIAPHRAGM. DIAPHRAGMAL HERNIA. RELAXATION OF THE IRIS.

The purpose of the lecture : to familiarize students with the anatomical and physiological features of the diaphragm, with the main clinical signs of various types of diaphragmatic hernias, hernias of the esophageal opening of the

diaphragm, congenital and traumatic hernias of Lorrey and Bohdalek, relaxation of the diaphragm, sliding, paraesophageal and other types of hernias. To familiarize with the diagnostic methods and indications for surgery.

Educational purposes: the etiology, pathogenesis, clinic, and radiological data given in the lecture allow students to learn how to diagnose diseases of the diaphragm, choose tactics and methods of treating patients.

Substantiation of the topic: diaphragmatic hernias are a common pathology of the abdominal septum and arise as a result of an anomaly in the embryonic development of the diaphragm, its traumatic injuries. They are internal hernias, as they exit from one cavity to another, which leads to respiratory and cardiovascular disorders. Entrapment of organs in a diaphragm defect can cause life-threatening complications. Hernia of the esophageal opening of the diaphragm is one of the common diseases, insufficient knowledge of this pathology can lead to a diagnostic error.

Lecture plan

1. anatomical and physiological information - 15 minutes
2. hernia and relaxation of the diaphragm (clinic, diagnosis, treatment) - 35 minutes
3. hernia of the esophageal opening of the diaphragm - 30 minutes
4. GP tactics for diaphragm diseases - 10 minutes

ANATOMO-PHYSIOLOGICAL INFORMATION

The diaphragm is the abdominal muscular septum that separates the chest cavity from the abdominal cavity. The muscular part of the diaphragm begins along the circumference of the lower aperture of the chest from the sternum, the inner surface of the cartilage of 7-12 ribs and lumbar vertebrae (sternal, costal and lumbar diaphragms). Muscle bundles go up, radially and end in the tendon center, forming dome-shaped bulges on the left and right. Between the sternal and costal sections there is a sternocostal space (Morgagni's, Larrey's triangle), filled with fiber. The lumbar-costal space is represented by the Bogdalek triangle. The lumbar diaphragm consists of 3 legs: external (lateral), internal (medial) and intermediate.

The tendon edges of both medial legs at the level of 1 lumbar vertebra to the left of the midline form an arc that delimits the opening for the aorta and the thoracic lymphatic duct.

The esophageal opening of the diaphragm is most often formed by the right inner leg. The vagus nerves also pass through this opening. Sympathetic trunks, celiac nerves, unpaired and semi-unpaired veins pass through the muscle gaps of the lumbar diaphragm. The opening for the inferior vena cava is located in the tendon center of the diaphragm.

The diaphragm is covered from below by the intra-abdominal fascia and peritoneum, and from above by the intrathoracic fascia, pleura and pericardium. Adjacent to the retroperitoneal part of the diaphragm are the kidneys and adrenal glands, the pancreas, and the duodenum surrounded by a fatty capsule.

The left lobe of the liver, the spleen, the fundus of the stomach, and the liver are adjacent to the left dome of the diaphragm. There are corresponding ligaments between these organs and the diaphragm. The standing height of the diaphragm depends on the constitution, age, the presence of various pathological processes in the chest and abdominal cavities. The left dome of the diaphragm is located lower (fifth intercostal space) than the right (fourth intercostal space).

The blood supply to the diaphragm is carried out by the upper and lower diaphragmatic arteries extending from the aorta, the musculophrenic and pericardial-diaphragmatic arteries extending from the internal thoracic arteries, as well as six lower intercostal arteries. The outflow of venous blood occurs through the veins of the same name, through the semi-unpaired and unpaired veins, as well as the veins of the esophagus.

The lymphatic vessels of the diaphragm are represented by several networks: peritoneal, subperitoneal, intrapleural, subpleural, pleural.

HERNIA AND DIAPHRAGM RELAXATION

Among the various surgical diseases of the diaphragm, the most common are various hernias and relaxation of the diaphragm. However, practical

Doctors are not sufficiently familiar with these diseases, which often leads to severe diagnostic and treatment errors.

With a diaphragmatic hernia, the abdominal organs move: into the chest through the formed defect, the weak zones of the diaphragm, or the expanded natural opening of the diaphragm. Unlike hernias, during relaxation of the diaphragm, there is a sharp thinning of it and the absence of muscles in the whole dome or in any part. This area or the entire dome protrudes high into the chest along with the adjacent organs of the abdominal cavity, while there are no pronounced hernial orifices, therefore, infringement with this disease is impossible.

To date, a large number of different classifications of hernia and diaphragm relaxations have been proposed, based on very different principles and subdividing these diseases according to etiological signs, nature, localization and size of the hernial orifice, the presence or absence of a hernial sac, and during relaxation - for the reasons for its occurrence and the size of the affected area.

Hernias of the diaphragm can be subdivided according to their origin into traumatic, resulting from various injuries of the diaphragm (open and closed) and non-traumatic, which has a different origin.

Depending on the presence or absence of a hernial sac, hernial hernias are respectively divided into true and false. Traumatic ones are almost always false, being the result of a rupture or injury to the diaphragm, and only extremely rarely are true.

Among non-traumatic hernias of the diaphragm, one should separate false congenital hernias, or defects of the diaphragm, which are the result of non-closure of the messages between the thoracic and abdominal cavities that exist in the embryonic period. The other nontraumatic diaphragm hernia are true and can be divided into diaphragm hernia weak zones hernia atypical localization and hernia diaphragm orifices, each of which in turn is subdivided into smaller groups nN. So among congenital hernias, depending on the size of the hernial orifice, aplasia of

the entire diaphragm is distinguished - a defect usually incompatible with life, or one of its domes, as well as partial defects of the diaphragm. The latter in localization can be posterolateral, anterolateral, less often central, phreno-pericardial, and esophageal-aortic.

TRUE HERNIA OF THE WEAK ZONES OF THE DIAPHRAGM

They arise as a result of certain conditions that contribute to an increase in intra-abdominal pressure, a weakening of the tone of those parts of the diaphragm, which are the area of connection of its various parts. This is the zone of the Larrey gap - the sternocostal triangle formed at the junction of the sternum and costal parts of the muscular part of the diaphragm.

and slit Bogdaleka - lumbocostal triangle located Nogo between its respective departments, in the region of these triangles have muscle and it is only more or less thin unite Tel'nykh - Cloth plate adjacent thereto with the pleura and peritoneum.

In addition, hernias often occur in the area of the underdeveloped sternal part of the diaphragm. These hernias which are located directly behind the breastbone, called the retrosternal Unlike hernias sterno-costal triangle, called a retro-kostosternalnymi, since they are located a few side of the sternum and are adjacent to the edge hrya scham. Both types can be combined under the term "parasternal hernia".

True hernia atypical localization are extremely rare and are characterized by the presence of a limited relaxation expressed hernia in his mouth, and hence the possibility of infringement.

Among hernias, natural openings of the diaphragm, hiatal hernias are the most common. Rare hernias of natural openings of the diaphragm include hernias of the sympathetic nerve cleft, the opening of the inferior vena cava, and also the aortic cleft. However, they meet MULTI to rarely that their practical value is small.

The relaxation of the diaphragm was described for the first time in the book of Jeanne Petit in 1774. At the beginning, relaxation was found only at autopsy. The number of such studies has rapidly increased with the use of the X-ray method of research. The relaxation of the diaphragm is congenital and acquired. Evidence for the existence of congenital relaxation can be found in fetuses, newborns and infants in combination with various abnormalities of development: transposition of the abdominal aorta, tetralogy of Fallot, non-closure of the ductus arteriosus, etc.

With congenital relaxation, primary underdevelopment or complete aplasia of the muscles of the abdominal obstruction is observed, which can be caused by a vicious laying of the diaphragm myotomes or a violation of the differentiation of muscle elements, as well as intrauterine aplasia or trauma of the abdominal nerve.

Much more common acquired relaxation occurring at extrauterine period in these cases already formed diaphragm muscle due to various reasons arise expressed degenerative and atrophic changes vplot until complete disappearance myshech by the elements, quite rarely acquired relaxation occurs on the basis of direct damage to the diaphragm itself in trauma or inflammatory processes.

The most common cause of acquired relaxation is damage to the phrenic nerve with the development of secondary neurotic muscle atrophy. These injuries

can be traumatic, surgical, inflammatory, and the result of invasion or compression of the nerve by a tumor or scarring.

Relaxation of the diaphragm can be complete, if the entire dome is thinned or moved into the chest, and partial, if any of its parts is displaced.

The clinic of hernia of the diaphragm depends on compression and kinks in the hernial orifice of the abdominal organs, mixed into the chest, compression of the lung and displacement of the mediastinum by organs that have fallen out through the diaphragm openings and on the dysfunction of the diaphragm itself, therefore all symptoms can be divided into gastrointestinal, depending on the disruption of the activity organs of the abdominal cavity, and cardio-respiratory, associated with compression of the lungs by displacement of the mediastinum. During relaxation, the causes of the onset of symptoms are the same, but the absence of a hernial orifice makes it impossible to infringe or even pronounced compression of the displaced organs.The severity of clinical symptoms depends mainly on the nature, volume and degree of filling of the abdominal organs, and in hernias, in addition, on the size, shape and the nature of the hernia orifice. So the movement of the stomach is often accompanied by a picture of acute or chronic volvulus or phenomena associated with its compression., In particular, the development of hemorrhagic gastritis or even ulcers.

With kinks of the esophagus, dysphagic symptoms often occur. Liver movement in some cases, is accompanied with jaundice The greater the volume has shifted bodies, the more pronounced symptoms of compression of the lung and the displacement of the mediastinum, therefore all points contributing povy sheniyu abdominal pressure, such as weight lifting, and pregnancy., Constipation, and meal causes increased or the appearance of symptoms.

Large defects of the diaphragm are often accompanied by the prolapse of a large number of abdominal organs, but with them less often than with narrow gates, infringement occurs, which sharply changes the clinical picture of the disease and causes a sharp deterioration in the general condition of the patient.

The most characteristic symptoms of diaphragmatic hernias are: the appearance or intensification of pain in the epigastric region, corresponding to the half of the chest or hypochondrium, as well as a feeling of heaviness, shortness of breath and palpitations immediately after eating, especially profuse. This makes the patients, who often feel practically healthy, sharply limit their food intake, which in some cases leads to exhaustion. Comparative but often occurs after eating vomiting (sometimes with blood), after which usually comes relief, which is also noted by patients after a bowel movement. A very common symptom is a feeling of "gurgle" and rumbling in the chest on the side of the hernia, as well as a significant increase in shortness of breath when moving patients to the horizontal of decomposition. The relationship between the severity of the above symptoms and the degree of filling of the gastrointestinal tract is very essential her diagnostic value.

Physical examination can note the presence of the scar on the chest or abdominal wall hernia of the diaphragm, which arose after open injuries, reducing the mobility of the chest on the affected side with smoothing intercostal spaces,

and in congenital hernias with a significant shift of the mediastinum and heart often observed Ser dechny hump. When long-existing hernia with stepping into stubble -sectoral cavity of a large part of the abdominal organs may be noted abdominal retraction, described for the first time NI Pirogov.

Percussion of the chest on the affected side is marked blunted tympanic sound askultativno in this area - the weakening or absence of respiratory noise, which is heard instead of intestinal peristalsis, or splashing, characterized by change auscultation and percussion of data depending on the degree of filling zhelud ka and intestines. No less characteristic displacement of the heart dullness and sredoste Nia in a healthy way, expressed generally greater, the higher is the blunting zone and tympanitic Such complaints and physical evidence found in the relaxation of the dome of the diaphragm, only when it lacks the phenomena associated with compression of the bodies in the hernial scrying max, since they are absent during relaxation.

The above signs usually make it possible to ascertain the movement of the abdominal organs into the chest and to suspect a hernia or relaxation of the diaphragm, the exact diagnosis of which is possible only with an X-ray examination.

A characteristic feature of diaphragm hernias is the variability of the X-ray picture depending on the degree of filling of the gastrointestinal tract. It also matters which organs have moved to the chest. So, with a survey fluoroscopy, with a prolapse of the stomach, a large horizontal level with air above it is visible, as in hydropneumothorax, and its height increases after eating or drinking. Aude neous loss of small bowel loops accompanied by the appearance of diffuse alveolar infiltrates with rounded portions pros vetleniya, and if the fall of the large intestine often hinges on the background of hectares of haustration can be seen. Moving the liver or spleen gives ct mud dimming pulmonary relevant department field.

Often, the dome of the diaphragm is clearly contoured and it can be seen that the abdominal organs are located above it, but in some cases the shadow of the diaphragm is not visible and the level of its location has to be judged on the basis of indirect signs obtained during a contrast study of the gastrointestinal tract. In this case, on contrasting bodies, respectively but the place they pass through the aperture, observed or depressions, called "flow symptom or" symptom of hernia gate. "This allows you to determine the location and defect sizes diafrag IU, as well as the nature and condition of the fallen bodies.

For large defects, diaphragm hernia symptoms gate absence is at the top, and is expressed by a bow-shaped line, dubbed the border, which can be organized as a shadow of the diaphragm during the relaxation and the stomach wall with a diaphragmatic hernia. Relaxation in these cases is to say the location of the medial contour of the gas bubble stomach and lateral location of the splenic angle colon under general arc formed by the diaphragm and Obra mations their characteristic triangle Dyuvalya- Kenyu-Fatou. In cases where the nature of the boundary line can not be established, differentiating hernia and relaxation of the diaphragm is necessary to impose diag nostichesky pneumoperitoneum. When relaxation

aperture shadow typically extends upwardly from the located underneath gastric and intestinal loops, with herniation air can pass into the pleural cavity and to give a picture pneumo thorax or placed in the abdominal cavity and contoured sides of the diaphragm in hernial ring. However, it should be emphasized that the indications for the imposition of a diagnostic pneumoperitoneum are very limited, since with a correct assessment of the data of a radiopaque examination, in most cases it is possible to make the correct diagnosis.

TREATMENT. The question of the treatment of various lesions of the diaphragm is solved differently. In patients with a hernia of the diaphragm, the possibility of infringement is a direct indication for surgery. When relaxation of the diaphragm operation proved expressed only in the case of health problems, if symptoms are mild, it is advisable to carry out the conservative therapy.

Preoperative preparation for hernias and relaxation of the diaphragm consists in prescribing a diet that is low in toxins 2 - 3 days before the operation, taking laxatives and prescribing on the eve of the operation and early in the morning on the day of the intervention of cleansing enemas.

The choice of access depends on the location and size of the defect, the age and condition of the patient, and the expected nature of the operation. So, for parasternal access, transabdominal access is more convenient, in particular, the upper median laparotomy; for operations on all other parts of the right dome of the diaphragm, transthoracic access is used in the seventh or eighth intercostal space, which is also most preferable for manipulations in all parts of the left dome in adult patients. In children with congenital hernia false when- usually no seam after falling Shih organs in the chest wall, as well as palliative surgery for intra-abdominal relaxation used transabdominal approach.

The purpose of surgery for diaphragmatic hernias is to bring down the displaced organs and suture the defect in the diaphragm. Often, especially with traumatic hernias, it is necessary to separate numerous battles and adhesions between the prolapsed organs and the chest wall, as well as the hernial orifice. This manipulation should be done with caution as dressing rezhdenie, such as prolapsed spleen, makes her pay in the hut zhanie postoperative bleeding.

After bringing down the fallen organs into the abdominal cavity, it is necessary

suture the defect of the diaphragm, Usually the edges of the defect are sutured with separate interrupted thick silk sutures, trying, if possible, to create a duplication if the tissue of the diaphragm is not strong enough. With very large sizes of the edge defect, it is not possible to pull it off and one has to resort to various plastic methods.

Of the various options for organoplasty, only hepatopexy remains of some importance. Using other bodies wasps Tavlya due to the danger of creating a fixed diaphragmatic hernia. It is also very traumatic to use various autoplastic methods with cutting out muscle, muscle-pleural and muscle-periosteal flaps. Undesirable and various embodiments torakop erasers aimed at the approach of the edges of the chest wall defect, due to the advancing with the latter expressed strain.

There was widespread Meto various alloplastic replacement rows Various prostheses are used for this purpose.

For the surgical treatment of the diaphragm relaxation times suggested different methods that can be divided into two main groups:

1. Interventions aimed at eliminating individual symptoms - palliative surgery on the stomach and large intestine.
2. Operations in the diaphragm itself, the purpose is to relegation of the aperture and displaced whelping abdominal organs in the normal position and strengthen the thinned diaphragm.

PALLIATIVE SURGERY: gastropexy; gastro-gastrostomy; gastroenterostomy; resection of the stomach; 5. resection of the large intestine.

OPERATIONS ON THE IRIS :

Plasty of the diaphragm at the expense of its own tissues: resection of the thinned area with suturing of the edges of the diaphragm; dissection of the diaphragm with the formation of duplication; phrenoplection.

Plastic yk p eplenie aperture (combined with a type of plastics due to own mk and n s aperture) autotransplantation (skin graft ; muscle flap ; musculo-na d braid T -boundary-pleural flap) alloplastica (mesh of tantalum ; grid plate or cloth made of nylon , polyvinyl l kogoln th g ubkoy).

DIAPHRAGM HERNIA

Hiatal hernia are very Prevalence nennym disease. Sliding hernia of the esophageal opening of the diaphragm is more common. The back wall of the upper part of the cardiac part of the stomach is not covered by the peritoneum, as a result of which, when the cardia is displaced upward into the mediastinum, this part of the gastric wall participates in the formation of the hernial sac. On this basis, the hernia is classified as sliding.

With paraesophageal hernias, the cardia remains fixed under the diaphragm, and one or another organ is displaced into the mediastinum next to the esophagus. Therefore, the hernia is called paraesophageal. Sliding hernias can be fixed and non-fixed.

In the latter case, they adjust themselves with the patient's vertical position. Large hernias are always fixed due to the suction action of the chest. Hernia fixation is associated with a shortening of the esophagus. The latter may be congenital. In adults, the shortening of the esophagus in the vast majority of cases is acquired. The shortening of the esophagus is subdivided into 2 degrees. At grade 1, the cardia is fixed no higher than 4 cm above the diaphragm. More significant shortening refers to the 2nd degree.

As for the origin of hernia of the esophageal opening of the diaphragm, then, like hernias of other localizations, they can be congenital and acquired. Acquired hernias are much more common, and age-related changes play a significant role in their origin. In the vast majority of cases, these hernias are observed after the age of 40 years. In addition to the expansion of the esophageal opening of the diaphragm, due to age-related involution, and the weakening of the connection

between the esophagus and the diaphragm, a hereditary constitutional predisposition to herniation is of great importance. It depends on the congenital weakness of the mesenchymal tissue.

The weakening of the connection between the esophagus and the diaphragm caused by one reason or another is the background from which the hernia develops. In the direct mechanism of formation, two kinds of factors can be distinguished: pulsatory and traction.

The pulse factor is an increase in intraperitoneal pressure, with heavy physical exertion, overeating, flatulence, constipation, pregnancy, wearing tight belts and corsets., With large tumors and cysts of the abdominal cavity.

Traction factor - associated with increased contraction of the longitudinal muscles of the esophagus.

It is the spastic longitudinal contraction of the esophagus that is of particular importance in the origin of the acquired shortening of this organ. The second reason for the shortening of the esophagus is the development of scar tissue in its wall as a result of peptic reflux zzophagitis.

The clinic of sliding hiatal hernias depends mainly on the onset of a disorder of the valvular function of the cardia. When the cardia is displaced upward, the angle of the His becomes obtuse, which leads to smoothing of the folds of the mucous membrane, which act as a valve. The weak tone of the esophagocardial pulp becomes unable to withstand intragastric pressure, and the contents of the stomach begin to flow into the esophagus, that is, gastroesophageal reflux occurs. In this case, the gastric mucosa is burned. Such patients complain of excruciating heartburn, belching, regurgitation. These symptoms are aggravated after eating and with postures that promote reflux, which also causes pain in the chest or high in the epigastric region and in the left hypochondrium. Constant exposure of the esophageal mucosa to peptic active gastric juice leads to the development of reflux esophagitis, which in some cases becomes erosive and even ulcerative. The inflamed mucous membrane is easily injured, which is accompanied by bleeding.

Reflux most often occurs with a cardiac hernia, especially if it is combined with a shortening of the esophagus.

Sliding hiatal hernias are never pinched. It depends on the fact that the cardia shifts above the diaphragm, and if there is some compression of the stomach in the hernial orifice, then full venous stasis in the supraphrenic part of the stomach occurs along the esophagus. Thus, with sliding hiatal hernias, the conditions necessary for the development of infringement are absent, while paraesophageal hernias can cause infringements as much as conventional ventral hernias.

DIAGNOSIS. When recognizing hiatal hernias, attention should be paid to the patient's complaints with features of gastroesophageal reflux.

X-ray examination is necessary to diagnose hernias.

When cardiac-gastric fundus, or the giant hernia Detect alive part of the gas bubble of the stomach in the posterior mediastinum. The contrast study convinces that the detected enlightenment refers to the stomach displaced upward.

Cardiac herniation can be detected only when transferring patients go into a horizontal position, Hoti and in this position, she revealed a number of indirect signs.

TREATMENT. Treatment should start with conservative measures. First of all, these include the appropriate regimen and diet.

Patients should take food often, in small portions. In no case should you eat at night. The last meal should be 3 to 4 hours before bedtime. You can't go to bed afterwards. Patients should also sleep with the head end of the bed raised, on two pillows. When you drastically expressions symptoms of reflux esophagitis can be assigned to medical therapy.

Only in the absence of the effect of conservative therapy should patients be recommended to operate. Surgical treatment is indicated in OC De complications with bleeding peptic stenosis of the esophagus, the twisting of the stomach and a suspected tumor.

With paraesophageal hernias due to infringement, surgical treatment is also indicated.

Surgical access: transperitoneal and transthoracic. There are several types of operations.

Lowering the stomach into the abdominal cavity and plastic hernial orifice The main stage of the intervention is stitching behind the esophagus with each other the medial legs of the diaphragm, due to which the hernial orifice narrows - cruraphia. But after this operation is not always disappear reflux due to the remaining straight angle of His and dekompensa tion ezofagokardialnogo pulp.

To restore the cardiac valve, zzophagofundography is performed, that is, the stitching of the fundus of the stomach with the esophagus, due to which the acute angle of His is restored.

In the presence of pronounced decompensation of pulp, the operation is used - Nissen fundoplication. Operation is fully enveloping the terminal part of the esophagus wall bottom zhelud ka. At the same time, a valve valve is formed in the cardia region, which functions even with complete decompensation of the esophagocardial pulp.

With the shortening of the II degree esophagus, valve gastric plication is used, which consists in creating a valve from the gastric wall with a simultaneous lengthening of the esophagus due to the stomach.

Questions to the audience to establish feedback and clarify the achievement of the goal of the lecture

1. What is a true hernia of the diaphragm?
2. A false hernia of the diaphragm is ...?
3. Define the concept, relaxation of the diaphragm
4. List the operations performed on the diaphragm
5. Name 3 types of hernias according to the classification of B.V. Petrovsky
6. Complications of hiatal hernias
7. The main methods of treating hiatal hernia
8. Types of online access

9. Direct indications for surgery
10. Where is the Bogdalek triangle

TOPIC : DISEASES OF THE OPERATED STOMACH

Purpose of the lesson: To study about the complexity of stomach ulcers and 12 p.c.: Penetration. Concept. The reasons. Diagnostics and differential diagnostics, research methods and treatment tactics.

Malignancy. Concept. Causes, diagnosis and differential diagnosis, cytological and histological examination, treatment tactics.

What the student SHOULD KNOW:

1. Clinic of penetration and malignancy of gastroduodenal ulcers.
2. To carry out differential diagnosis of penetration and malignancy of gastroduodenal ulcers.
3. Know the characteristic differences between the penetration of gastric ulcers and ulcers 12 bp.
4. Modern instrumental examination methods to detect penetration and malignancy of gastroduodenal ulcers.
5. Laboratory methods of examination in the diagnosis and penetration and malignancy of gastroduodenal ulcers.

Having studied the topic, the student MUST BE ABLE TO:

1. Correctly conduct a survey of patients with penetration and malignancy of gastroduodenal ulcers.
2. Conduct an objective examination of patients.
3. Analyze the data of X-ray, endoscopic and laboratory examination methods.
4. Appoint an additional plan of examination of patients.
5. Based on the data obtained about the patient, formulate a clinical diagnosis.
6. Conduct differential diagnostics.
7. Determine s tactics and the amount of therapeutic measures.

THEORETICAL PART

Penetration of gastroduodenal ulcers

Penetration ulcer - entering it into contact with the stomach or lu duodenum kovitsey organs: liver, pancreas, ma ly packing. Among patients with penetrating gastroduodenal ulcers bo Lee 70% of people of working age. In men, this complication occurs 13 times more often than in women.

When the ulcer penetrates, a wall defect is formed due to necrosis of all its layers in the ulceration zone. As a result, the bottom of the ulcer are adjacent to the stomach organs and tissues associated with stomach or duodenum fibrosis GOVERNMENTAL seam. In most cases,

penetrating ulcers YaV lyayutsya callous. Their edges and the walls are represented coarse fiber unite tion tissue and therefore do not have ulcers tendency to heal. Most frequently ulcers penetration occurs in the pancreas (67.8%), the liver (10.1%), ma ly seal (10.1%), hepatic-duodenal ligament (10.1%). Less common is penet radio in the gallbladder, mesentery of transverse colon Division, the wall of the small intestine.

Penetrating ulcers most often localized in the same department piloroantralnom ludka and in the duodenum (90%). At the same time, as a rule, has expressed zhennoe periultseroznoe inflammation. Often combined with a penetration of an ulcer drank roduodenalnym stenosis perigastritom and periduodenitom. In addition, with penetrating ulcers, there is a great threat of profuse bleeding from arosed large arteries. Penetrating stomach ulcers are prone to malignancy.

Constant pain, characteristic of penetrating ulcers subtree alive in constant tension cortico-adrenal system. In this regard, there are disorders of portal blood flow, liver hypoxia. This UD ments often detectable increase in the concentration of lactic and pyruvic acid in the blood and urine.

The clinical picture of gastroduodenal ulcers with penetration development as a great rule, varies considerably. The pain is no longer seasonal. They become vyatsya practically constant, strong and resistant. Irradiation is characteristic depending on the zone of penetration: in the back, right hypochondrium, right arm. Signs of damage to the organ where penetration occurs quickly join.

When the ulcer penetrates into the head of the pancreas, symptoms of chronic pancreatitis dominate. The pains are of a girdle nature, localized in the projection of the pancreas and the left hypochondrium. The secretory function of the pancreas is impaired. Due to the transition of the inflammatory infiltration that at the large duodenal papilla can be determined ikterichnost sclera and light yellowness of the skin. In this case, an erroneous diagnosis of acute cholecystitis, chronic pancreatitis, obstructive jaundice is often established. The pain is long-duration time are unjustified treatment for these diseases.

With the penetration of the ulcer into the hepatic-duodenal ligament, yellowness of the skin can also be detected due to compression of the common bile duct by an inflammatory infiltrate. Clinical manifestations of foam tration in the liver and small omentum are scarce. Pain localized in the IU chevidnogo process. There may be soreness on palpation of the edge of the liver, its increase. Typically, the penetration into the liver and the small oil seal detects smiling during surgery.

In the study of the peripheral blood revealed moderate constant but marked leukocytosis, can be detected by biochemical markers Auto ary pancreatitis and hepatitis.

The final judgment on the presence of the patient penetrating ulcers can be done only after radiographic and endoscopic studies, and with placing their results.

The most informative method radiological diagnosis is camping polypositional study esophagus, stomach and duodenum kish ki and the assessment of relief pnevmorelefa mucosa, as well as the study of these organs in a tight filling. In recognition penetrating duodenal ulcers are based on a combination of a number of characteristics, primarily such as a rough initial deformation and fixation of delov dvenaadtsatiperegnoy intestine, local retraction and contraction around ulcerative hydrochloric niche. Then the dimensions of the latter are estimated.

Radiological examination revealed gastric limited in mobility of his contrast depots in the form of a niche, goes far beyond the contour zhelud ka.

X-ray of patient L. Ulcerative niche outside the contour of the stomach.

In contrast, penetrating ulcers of the stomach in which large depot is often defined dimensions contrast with the horizontal and the air bubble above him, when penetrating ulcers duodenal ulcer size and shape of the niche is not always of paramount diagnostic values of. Penetrating duodenal ulcers may be very small, however, they are always deep. During the operation, the size of the ulcer often do not coincide with the definition of fissionable X-ray examination, which is due to close at the rank. These include swelling of the mucous membrane, the ulcer clot clotting mi blood, an abundance of mucus and spasmodic contraction of the muscular layer around the ulcer. Organs and tissues that penetrates in ulcer, often are con glomerat formed by growth of connective tissue and seal when lying to ulcers organ walls, which determines the radiographic signs retractions. In contrast to the inflammatory shaft, local retraction is not caused by edema and infiltration of the mucous membrane around the ulcer, but by the inflammatory and cicatricial-adhesive process in the organs into which the ulcer penetrates. In this case, the contours of the ulcer niche are uneven and indistinct. Penetrating duodenal ulcers are characterized by low variability in the time of tight first filling and after emptying from the contrast mass. Barium slurry can linger long in the ulcer crater, impregnate it for at how many hours of research. With penetrating ulcers, the ulcerative surface sometimes occupies the entire wall or even both walls, including the mucous membrane of the bulb or the bulbous sections of the duodenum. In such cases, the X-ray colon looks uneven record structured Channel. Such ulcers are difficult to distinguish from cicatricial changes. An important difference -inflammatory signs are lack of mucosa and relief of variability pathological section at different points of the study. In 50% of SLE teas offset notes duodenum upward. X-ray examination allows not only to establish signs of ulcer penetration, but in most cases to determine its direction. Penetration ulcers hepato-duodenal ligament ends can form the pigs holedohoduodenalnogo conductive, which can also be detected by X-ray examination.

When endoscopy penetrating ulcer appears CCA cially deep and extensive. When such an ulcer is localized in the stomach, a biopsy must be performed to exclude malignancy.

With ultrasound echography, thickening and fibrotic changes in the pancreas are often found.

According to modern concepts of ulcer penetration it is irreversible about the process and the soprvozhdaetsya profound metabolic and circulatory dist roystvami tissue. This circumstance gives grounds to consider operative intervention as the only possible method of radical treatment. The same changes determine the high risk of surgery. Patients with penetrating ulcers need intensive preoperative preparation for 10-12 days. This training includes anti-ulcer and protivovospa inflammatory therapy, correction of disorders of homeostasis. The prescription of rheological drugs that improve microcirculation and tissue blood flow is of great importance.

Surgical intervention in patients with a penetrating ulcer presents serious difficulties for the surgeon and a great risk for the patient. Therefore, the operation should be as gentle and pathogenetically grounded as possible. As a rule lo, 2/3 gastric resection performed Billroth-1 or Billroth -11 modifi- tion Hofmeister-Finsterer. Technically more difficult is the intervention for

duodenal ulcers that penetrate into the head of the pancreas. This is due to the difficulty of closing the duodenal stump. The operation of choice and in these cases remains resection of the stomach. However, if penetri ruyuschey Postbulbarnye ulcers with massive inflammatory infiltration okra zhayuschih organs and tissues less risky intervention may be one or another variant of vagotomy.

Malignant ulcer

Malignancy of an ulcer is the transformation of an ulcer into cancer. As a rule, gastric ulcers undergo blast transformation (6 ... 8%).

Typical signs of the degeneration of an ulcer into cancer:

1. Pain syndrome becomes less pronounced, constant and does not depend on food intake.
2. Loss of appetite.
3. Aversion to meat foods.
4. Progressive weight loss
5. Deterioration of the general condition (decreased performance, increased fatigue, discomfort, etc.).
6. The size of the ulcer is more than 2.5 - 3 cm .
7. Localization of the ulcer (long-term ulcers of the greater curvature of the stomach are malignant in 90% of patients, the lower third of the stomach - in 86%, the cardiac part of the stomach - in 48%).
8. X-ray signs (the ulcer defect has an irregular, trapezoidal shape, high undermined uneven edges, wall rigidity
stomach, lack of peristalsis, etc.).
9. Endoscopic signs (ulcer defect larger than 2.5-3 cm , blurred outline of the edges of the ulcer, granular mucosa around it, tumor-like growths in the ulcer itself or along its edges, etc.).

Especially valuable data can be obtained by the method of gastrobiopsy (6-7 pieces of biopsies from different zones of the ulcer) with subsequent morphological examination.

Treatment of malignant stomach ulcers is operative, subtotal or total removal of the stomach (gastrectomy) is performed as in primary cancer of this organ.

PRACTICAL PART

Thematic tests

1. Which of the following is not true for stomach cancer?
a) three times more common in men than in women;
b) develops against the background of chronic atrophic gastritis;
c) can develop from a polyp;
d) rarely localized on the greater curvature;
* e) early diagnosed by determining the content of α - fetoprotein.

2. In case of a malignant ulcer of the antrum of the stomach, the patient is shown the operation:
a) truncular vagotomy with pyloroplasty and excision of the ulcer;
b) resection $^{2}/_{3}$ of the stomach;
* c) subtotal gastric resection;
d) antrumectomy;
e) excision of the ulcer.

3. What are the criteria for early gastric cancer?
a) low degree of morphological differentiation of the tumor;
* b) the depth of invasion, limited to the mucous and submucous layer;
c) the depth of invasion is limited by the muscle layer of the gastric wall;
d) tumors measuring at least 2 cm ;

e) tumors without regional metastasis.

4. Signs indicating the degeneration of a stomach ulcer into cancer are:
1 persistent abdominal pain
2.The appearance of pain in the epigastrium 40 minutes after eating
3 heartburn
4 anemia
5.Zero acidity of gastric juice
1, 3, 2
2, 3, 4
3, 4, 5
+ 1, 4, 5
Only 1 and 4

5. The most typical complication of ulcers of the anterior wall of the duodenum are:
1. Malignancy
2. Perforation
3. Bleeding
4. Penetration into the head of the pancreas or hepatoduodenal ligament
12
2, 3
fourteen

6. Surgical treatment for a patient with a duodenal ulcer is indicated in those cases when:
1. Relapses of the disease often occur
2. The disease is complicated by profuse bleeding
3. Pyloroduodenal stenosis occurs
4. Ulcer perforation occurs
5. The ulcer penetrates into the head of the pancreas, giving frequent exacerbations and symptoms of pancreatitis
Only 1 and 2
Only 1 and 4
Only 2 and 3
Only 3 and 4
+ All answers are correct

DISEASES OF THE OPERATED STOMACH

The purpose of the lecture : Acquaintance of students with the diseases of the operated stomach, the reasons for their development, the peculiarities of the clinical course, the course of complicated forms, differential diagnosis, optimal methods of treatment, management of the postoperative period, rehabilitation of patients.

Educational purposes of the lecture: To instill in students the need for a timely adequate operation before the development of formidable complications , and with their development - acquaintance with the most informative and modern methods of diagnostics, surgical treatment of patients, acquaintance with possible complications outside the operation and the operating period, their prevention. Development of students' clinical thinking. Development

of a modern view of the problem from the perspective of world medicine and general practitioner.

Lecture objectives:

29. To give an understanding of the diseases of the operated stomach.
30. Explain the causes and mechanisms of complications.
31. Give a clinical description and possible variants of the course of the disease.
32. Conduct differential diagnosis with other diseases.
33. To acquaint students with modern and most informative methods, examination of patients
34. Demonstration of examples of their surgical practice: patients, slides, radiographs.
35. To prepare and present all the material of the lecture to the students, in the amount necessary for the high-quality training of a general practitioner.

PLAN OF THE LECTURE.

25. The urgency of the problem - 5 min
26. Etiopathogenesis of diseases of the operated stomach.
27. Clinical picture - 10 min
    a) Etiopathogenesis;
    b) Clinic and diagnostics.
    c) Differential diagnosis.
    d) Treatment.
28. Diagnostics. - 10 min
29. Differential diagnostics. - 10 min
30. Treatment - 15 min

7. Disease prevention - 10 min

Recurrence of peptic ulcers. After surgery for peptic ulcer disease, recurrence of the ulcer is a serious complication . Among patients with recurrent ulcers, the overwhelming majority (95-98%) are patients whose indications for primary surgery had a duodenal ulcer. The causes of recurrent ulcers are varied. They can be associated with multiple endocrine adenomatosis.

Symptoms: Ulcer recurrence appears within 2 years after surgery. The leading symptom is pain in the upper abdomen. The pain is constant, worse soon after eating, not relieved by taking antacids or milk. In connection with the penetration of the ulcer, the pain in intensity significantly exceeds the pain that the patient had before the operation. Bleeding is in the form of hematemesis, melena, latent bleeding. The result is anemia. Possible complication of the ulcer by perforation. In the study of gastric secretion, the preservation of the acid-producing function of the operated stomach is found.

The X-ray symptom of ulcer recurrence is a niche, which, however, can be difficult to identify due to cicatricial deformity of the operated organs. The most informative method for diagnosing ulcer recurrence is endoscopic examination.

Treatment: a course of drug therapy is carried out in order to resolve the issue of indications for surgery, as well as as a preoperative preparation to reduce periulcerous infiltration in the tissues and organs into which the ulcer has penetrated. After distal resection of the stomach by the Billroth-I method, ulcers appear in the area of the gastroduodenal anastomosis. The reason for the recurrence of the ulcer is the preservation of the acid-producing function of the stomach due to its economical resection. After distal resection of the stomach using the Billroth-II method, ulcers develop in the jejunum or in the area of the gastrojejunal anastomosis. The reasons for the recurrence of ulcers can be either an economical resection of the stomach, or leaving a section of the antrum above the duodenal stump , which is the source of gastrin, which stimulates the parietal cells of the fundic mucosa.

Peptic ulcer of the jejunum. Symptoms: pain in the upper abdomen, radiating to the left half of the lumbar region, to the left half of the chest . Palpation of the abdomen in the left upper quadrant causes muscle tension and pain in the area of projection onto the abdominal wall of the gastrojejunal anastomosis. Sometimes an inflammatory infiltrate can be felt.

Treatment is conservative: as a rule, it is ineffective. The purpose of the operation is to reduce the acid-producing function of the resected stomach, resection of the jejunum section bearing the ulcer, restore the continuity of the gastrointestinal tract using a method that ensures the passage of food through the duodenum 12 (reconstruction of the gastrojejunal anastomosis into the gastroduodenal anastomosis or reconstructive gastrojejunoduodenal anastomosis).

A gastrointestinal fistula results from the penetration of a postoperative peptic ulcer of the jejunum into the transverse colon.

Symptoms: reduction or disappearance of previously observed pain in the upper abdomen due to peptic ulcer of the jejunum, diarrhea after each meal, excretion of undigested recently eaten food with feces , unpleasant smelling belching, vomiting with an admixture of colonic contents (fecal vomiting), progressive weight loss ... The main factor in the rapid deterioration of the patient's condition is the exclusion of the passage of food through the small intestine, diarrhea. The patient is emaciated, anemic, edema on the legs. In the left upper quadrant of the abdomen, the tension of the muscles of the abdominal wall, pain on palpation. The study reveals anemia. An admixture of colonic contents is found in the gastric contents. Pieces of recently eaten food are visible in the stool.

Diagnosis: The main method for clarifying the diagnosis is X-ray examination. The accepted aqueous suspension of barium sulfate enters the colon through the fistula. With irrigoscopy, the contrast suspension penetrates through the fistula into the stomach. The introduction of air into the rectum causes it to move through the fistula into the stomach, resulting in an increase in the size of the gas bubble of the stomach. To detect a fistula, tests with dyes can be used. After taking methylene blue solution, the solution can be found in the feces. With enemas with methylene blue solution, it can be found in the contents of

the stomach. Endoscopic examination is combined with the introduction of a dye through the rectum, which facilitates the detection of a fistula.

Treatment: surgical only. The purpose of the operation is to separate the organs that form the fistula, to perform an operation on the stomach, aimed at reducing the acid-producing function. For this, a vagotomy can be applied. Some surgeons prefer gastric resection, although this operation is more traumatic. It is advisable to complete the operation with gastroduodenostomy or gastrojejunoduodenoplasty.

Surgical treatment is indicated if conservative treatment is unsuccessful. In case of recurrence of ulcers after vagotomy with drainage surgery due to narrowing of the gastroduodenal fistula or duodenostasis, revagotomy with pyloroantrumectomy is indicated.

Postgastrectomy and postvagotomy syndromes. Pathological syndromes after surgery on the stomach are a special type of complications associated with the restructuring of digestion after surgery. Various operations on the stomach introduce changes inherent to the type of operation in the anatomical and functional relationships between the organs of the digestive tract. As a result of insufficient adaptation and compensation processes after the operation , changes occur in the activity of the digestive system and various disorders of the functions of the digestive and other systems of the human body develop .

The development of post-gastro-resection syndromes is associated with three main reasons. 1. Due to the distal extensive resection of the stomach, the antrum is removed - the main source of gastrin and most of the acid-producing zone of the stomach. 2. In patients who have undergone gastric resection, the food taken due to the loss of the reservoir function of the stomach and the exclusion of gastric digestion quickly enters the small intestine without the necessary preliminary processing. The result can be a dumping reaction and subsequent defects in digestion and absorption. 3. Due to the resection of the stomach by the Billroth method - the II part of the important proximal part of the small intestine - the duodenum 12 remains away from the chyme moving along the intestine. As a result, the duodenum does not take proper part in the neurohumoral regulation of the processes of digestion and absorption. The more extensive the resection of the stomach, the greater the risk of post-gastro-resection disorders. After resections of the stomach, completed by the imposition of a gastrojejunal anastomosis, post-gastro-resection syndromes are observed more often than after resections of the stomach with a gastroduodenal anastomosis.

The syndrome of the "small" stomach is caused by a decrease in the capacity of the stomach cavity after resection. During a meal, the rapid filling of a small cavity of the stomach leads to stretching of its walls, there is a feeling of overflow in the upper abdomen, discomfort, belching, nausea, and vomiting.

Treatment: eating in small portions; with a delay in evacuation associated with stenosis of the gastrointestinal fistula, surgical treatment is indicated - expansion of the gastrointestinal fistula or the imposition of a new gastrointestinal fistula.

Early and late dumping syndrome. In operated patients, food intake can cause reactions of varying severity.

Depending on the time of onset of symptoms after a meal, there are early and late forms of post - alimentary syndromes. Dumping syndrome after gastric resection is observed in the majority of operated patients during recovery, later in 30% of mild and 10% of severe. After vagotomy with gastric-draining operations, dumping syndrome is observed in 12%, rarely severe.

Clinical manifestations: characteristic. Eating food causes a peculiar reaction in patients . During or after eating, after 10-15 minutes, especially after taking sweet, dairy dishes, weakness, dizziness, headache, confusion , sometimes fainting, pain in the heart, palpitations, "hot flashes" throughout the body, abundant sweat. Along with these symptoms, there is a feeling of heaviness and distention in the epigastric region, nausea, scant vomiting, rumbling and colicky cutting pain in the abdomen, diarrhea. Due to severe muscle weakness, patients are forced to take a horizontal position.

Diagnosis: Based on the characteristic symptoms of a dumping response to food intake. For an objective characterization of the severity of the dumping reaction , changes in hemodynamic parameters in comparison with the initial data (pulse rate, blood pressure, volume of circulating plasma) can be used . To provoke a dumping reaction, the patient is given to drink 150 ml of a 50% glucose solution. Changes in heart rate are a constant sign of a dumping response, and the increase in heart rate increases the more severe the dumping response is.

X-ray data are an important adjunct to the clinical assessment of the severity of dumping syndrome. The use of an aqueous suspension of barium sulfate (sulphate) makes it possible to reveal the nature of the operation previously undergone by the patient, anatomical and functional changes in the stomach and small intestine. The most often observed is accelerated emptying of the operated stomach, increased motility of the small intestine, alternating with inertia. The fact of rapid evacuation by the "failure" type of the first portion of gastric contents, when 1/3 or more of it enters the small intestine, is of diagnostic value .

Symptoms: weakness, a sharp feeling of hunger, sucking pain in the epigastric region, tremors, dizziness, palpitations, decreased blood pressure, sometimes bradycardia, pallor of the skin, sweat. These symptoms disappear quickly after eating a small amount of food, especially carbohydrate. To prevent severe manifestations of hypoglycemic syndrome, patients try to eat more often, carry sugar, cookies or bread with them and take them at the first signs of hypoglycemia.

Diagnosis: based on the characteristic complaints of the patient, on the observation data of the patient at the time of the attack, low blood sugar (0.75-0.50 g / l, or 75-50 mg%) at this time. The severity of the symptoms of hypoglycemic syndrome is different: from episodic weakness, quickly passing, to severe manifestations that are observed daily.

The constant combination of hypoglycemic syndrome with dumping syndrome is due to their pathogenetic commonality, while the leading suffering

is dumping syndrome, which should be targeted by therapeutic measures. In patients with dumping syndrome, neuropsychiatric disorders are observed, the main ones being astheno-neurotic syndrome, hysteroform syndrome, depressive syndrome, hypochondriac syndrome.

Treatment: conservative and surgical dumping syndrome. The principles of conservative treatment are as follows.

1. Medical nutrition in order to prevent or reduce the manifestations of the dumping reaction. The diet is physiologically complete, mechanically not sparing. The chemical composition of the diet: high protein content (130-140 g), normal fat content (100-115 g), restriction of complex carbohydrates (310 g), significant restriction or exclusion of simple carbohydrates, normal salt content (15 g). Exclusion of foods and dishes that cause the development of dumping syndrome. Caloric content is about 2800-3200 kcal. All dishes are cooked boiled or steamed. Food is served warm. Avoid hot and cold foods. Separate food intake, dense and liquid, in small portions, 5-6 times a day, in the supine position.

2. To reduce the reaction to the rapid flow of food into the small intestine , novocaine, anestezin, antihistamines (pipolfen, diphenhydramine, suprastin), reserpine, and insulin are prescribed before meals .

3. Substitution therapy: gastric juice, hydrochloric acid with pepsin, pancreatin, panzinorm, abomin, B vitamins, ascorbic acid, folic acid, fat-soluble vitamins, iron, calcium, magnesium, potassium preparations , protein preparations - plasma, protein, protein hydrolysates ...

4. Treatment of psychopathological syndromes in consultation with a psychiatrist.

The principles of surgical treatment are as follows. Surgical treatment of severe and moderate dumping syndrome is indicated in case of ineffectiveness of nutritional therapy and complex drug treatment. The purpose of the operation is to eliminate the anatomical conditions that contributed to the development of dumping syndrome.

In case of dumping syndrome, which has arisen after resection of the stomach with gastrojejunal anastomosis, in order to eliminate the rapid evacuation of food from the stomach stump into the jejunum and improve food digestion, gastrojejunoduodenoplasty is used, which ensures the passage of food through the duodenum. With dumping syndrome that develops after resection of the stomach with gastroduodenal anastomosis, gastrojejunoduodenoplasty can also be performed. In patients who have previously undergone gastric resection for peptic ulcer of the duodenal ulcer, reconstructive gastrojeyunoduodenoplasty can help restore the acid-forming function of the gastric stump mucosa. In this regard, reconstructive gastrojejunoduodenoplasty should be supplemented with vagotomy in order to prevent the occurrence of peptic ulcers in the small bowel graft . The small intestinal transplant provides a portioned evacuation of stomach contents into the duodenum. As a result of gastrojejuno-duodenoplasty, food coming from the stomach into the duodenum is mixed with pancreatic juice and bile, the osmolarity of the contents of the duodenum is aligned with the osmolarity of the plasma, and all food ingredients are hydrolyzed in the lower parts

of the jejunum. Due to this, the processes of digestion and absorption of food hydrolysis products, vitamins, electrolytes are improved in the jejunum . In patients who have undergone reconstructive gastrojejuno-duodenoplasty, the dumping response to food intake becomes less pronounced or does not occur, and body weight increases.

Ejuno- and duodenogastric and alkaline reflux gastritis and reflux esophagitis develop after gastric surgery, accompanied by destruction or dysfunction of the pylorus and lower esophageal sphincter. Such operations include gastric resection and gastrectomy, vagotomy in combination with gastric drainage operations (with pyloroplasty, with gastroduodenostomy, with gastroenteroanastomosis).

Clinical picture and diagnosis: the main symptoms are a burning pain in the epigastric region, which cannot be eliminated by taking antacids, vomiting with an admixture of bile, weight loss. The addition of esophagitis causes heartburn, dysphagia. Examination of the contents of the stomach on an empty stomach can give an alkaline pH value, while stimulating gastric secretion - normo-, hypo- or achlorhydria. In patients who have undergone vagotomy, the test with insulin hypoglycemia may be negative.

X-ray examination of the stomach is not very informative, but it can exclude ulceration. Endoscopic examination and mucosal biopsy are the main methods in the diagnosis of alkaline reflux gastritis and reflux esophagitis.

Treatment: Conservative measures bring minimal success only in mild cases. Cholestyramine is used to bind bile acids, antagonists of H_2-receptors of histamine, carbenoxalone to enhance the synthesis of gastric mucus, but its use is limited due to the aldosterone side effect.

Surgical treatment of alkaline reflux gastritis should be aimed at diverting duodenal contents from the operated stomach. In patients who have previously undergone gastric resection, this is done using a U-shaped gastrojejunal. Reconstructive surgery should be supplemented with a trunk vagotomy, if it was not performed during the previous operation. In patients who have undergone vagotomy with a stomach-draining operation, antrum resection with a U-shaped gastrojejunal anastomosis is performed.

Chronic adductor loop syndrome develops after resection of the stomach with the imposition of a gastrojejunal anastomosis in the case of stenosis of the adductor or discharge loop. The frequency of the adductor loop syndrome reaches 13% on average.

Clinical picture and diagnosis: the main symptom is pain in the epigastric region after eating, especially fatty foods. The origin of pain is different. The pain of a bursting cramping character, radiating to the back, under the right scapula is associated with dyskinesia of the adductor loop, disappears after vomiting. A burning pain in the epigastric region, behind the sternum, radiating to the heart region, is associated with reflux of bile into the stomach and esophagus, caused by damage to the protective barrier of the mucous membrane and an increase in the reverse diffusion of H + ions into the mucous membrane. Taking antacids does not relieve pain . After vomiting, the pain

decreases. Girdle pain is a sign of concomitant pancreatitis. Vomiting with an admixture of bile occurs with a sudden emptying of the leading loop into the stomach stump. An admixture of food in the vomit is observed in cases of food being thrown into the adductor loop. Determined by palpation in the epigastric region, the elastic formation (the adductor loop stretched by the contents) disappears after vomiting. X-ray examination on an empty stomach in the stomach stump is determined by the fluid as a result of intragastric emptying of the adductor loop. Failure to fill the adductor loop with a contrasting mass may be a sign of increased pressure in the adductor loop or its bending in the orifice at the gastrointestinal anastomosis. When the contrast mass is thrown into the adductor loop, there is a long delay in the adductor loop.

Treatment: usually surgical. It is indicated for severe adductor loop syndrome and ineffectiveness of conservative treatment for moderate adductor loop syndrome . It is necessary to eliminate the anatomical conditions that contributed to the stagnation of the contents in the adductor loop due to mechanical reasons. Operations: U-shaped gastrojejunal anastomosis, gastrojejunoduodenoplasty (with a combination of the leading loop syndrome with dumping syndrome), reconstruction of the gastrojejunal anastomosis into a gastroduodenal anastomosis.

Gastrostasis. After stem vagotomy in some patients, especially with inadequacy of the gastric drainage operation, due to impaired gastric motility , its expansion occurs, and evacuation is slowed down. As a result of gastrostasis, nausea, regurgitation, vomiting, and bursting pain in the upper abdomen appear . If the draining operation is adequate, the symptoms of gastrostasis disappear with the treatment of cerucal, benzohexonia as the gastric motility is restored. If the drainage operation is inadequate, a second operation should be performed (correction of the drainage operation or economical gastrectomy using the Billroth-I method). It should be borne in mind that after selective proximal vagotomy, a violation of gastric emptying may occur as a result of scarring of the duodenal ulcer, and then there is a need for a second operation to perform a drainage operation. To avoid such a situation, it is necessary to carry out a preoperative course of antiulcer treatment, and only after that during the operation it will be possible to determine the patency of the duodenum with a healed ulcer.

Duodenostasis. Symptoms: a feeling of heaviness and fullness in the epigastric region, in the right hypochondrium, bitterness in the mouth, nausea, vomiting mixed with bile. X-ray examination reveals a significant expansion of the duodenum and a long delay in it of the contrast suspension. Violation of duodenal motility is observed in some patients with duodenal ulcer, such patients should undergo vagotomy in combination with pyloroanthrumectomy with a Y-shaped Roux-en-Y anastomosis to ensure bile flow from the operated stomach.

Diarrhea - loose, watery stools more than 3 times a day. It is a characteristic consequence of vagotomy, mainly of the stem, in combination with operations on the stomach. It is observed in 10-40% of those operated. The main

factors contributing to the occurrence of diarrhea: decreased production of hydrochloric acid, impaired motility of the gastrointestinal tract, decreased function of the pancreas Imbalance of gastrointestinal hormones, accelerated passage of chyme through the intestine, morphological changes in the intestinal mucosa (jejunit), disorders of bile acid metabolism (holographic diarrhea) , changes in intestinal microflora. Clinical features of postvagotomy diarrhea: sudden onset of diarrhea, pale coloration of feces, sudden cessation of diarrhea. There are three degrees of severity of postvagotomy diarrhea: mild diarrhea - loose stools from 1 time a month to 2 times a week or occasionally after taking certain foods; the average degree of diarrhea loose stools from 2 times a week to 5 times a day; severe diarrhea - watery stools more than 5 times a day, appears suddenly, sometimes immediately after eating any food; lasts 3-5 days or more, accompanied by a progressive deterioration in the patient's condition.

Treatment: complex. When choosing a therapeutic measure, one must bear in mind the connection between diarrhea and the lack of exocrine pancreatic function, intestinal dysbiosis, and dumping syndrome, which can be observed in patients who have undergone vagotomy with pyloroplasty or antrum resection. Cholestyramine is used to neutralize bile acids. Prescribe a diet with the exclusion of milk and other foods that provoke a dumping reaction. Antibiotics are used to influence the bacterial factor. Effective is the use of benzohexonium, which has a regulating effect on the motility of the vagotomy stomach. With hypomotorism, benzohexonium increases the contractile activity of the stomach, with hypermotility, it weakens the motor activity of the stomach, normalizing the rate of its emptying.

Surgical treatment of severe postvagotomy diarrhea that does not respond to conservative treatment consists in inversion of the small intestine segment. In case of impaired absorption of proteins and carbohydrates, a high segment is used at a distance of 120 cm from the duodenojejunal bend; in case of impaired absorption of water and fats , a segment of the ileum is used. Postvagotomy diarrhea occurs predominantly after stem and selective gastric vagotomy in combination with gastric drainage operations.

QUESTIONS

to the audience to establish feedback and clarify the achievement of the lecture goal

1. List the diseases of the operated stomach according to the mechanism of development.
2. The main reason for the development of GOD.
3. Basic diagnostic methods of BOD.
4. Classification of dumping syndrome.
5. Indications for conservative treatment of GOD.
6. Indications for surgical treatment of BOJ.
7. The general principle of surgical treatment for AOD.

TOPIC : MECHANICAL JAUNDICE A . P OSTCHOLECYSTEK TOMIC SYNDROME

The value of studying the topic. A significant place in the structure of surgical and infectious diseases is occupied by diseases occurring with jaundice syndrome. These are primarily cholelithiasis, tumors of the hepatobiliary system, viral hepatitis, the incidence of which in the Republic remains at a high level. Sometimes difficulties arise in their differential diagnosis. Specialists of different profiles are faced with jaundice.

All of the above dictates the need to study the principles of differential diagnosis of jaundice by 5th year students.

The purpose of the lesson:

1. To teach students the principles of differential diagnosis of jaundice, based on clinical and biochemical, immunoserological (ELISA), instrumental (RPHG, ChCHS, CT, MCT, ultrasound) data.

Test questions:

1. Pigment metabolism is normal.
2. Normal indicators of total, (indirect, direct) bilirubin, aminotransferase activity (ALT, AST), alkaline phosphatase, cholesterol level, ether-soluble bilirubin, indicators of protein-sediment samples (thymol and sublimate)
3. Results of RPHG, ChCHS, CT, MCT, ultrasound.
4. ELISA results.
5. The results of a detailed general blood test in order to recognize suprahepatic jaundice.
6. Clinic and peculiarities of the course of diseases in which suprahepatic jaundice is observed. Know the mechanism of development of such jaundice.
7. Clinic and features of the course of viral hepatitis and other hepatitis in which hepatic (parenchymal) jaundice develops. Know the mechanism of development of jaundice with them.
8. Clinic and peculiarities of the course of diseases occurring with obstructive jaundice. Know the mechanism of development of such jaundice.

Practical skills

1. Correctly examine the patient.
2. Registration of medical history.
3. Filling out emergency notifications, statistical coupons, extracts from case histories.
4. Correctly interpret the results of biochemical and serological tests (ELISA), ultrasound.
5. Correctly palpate the liver and spleen.
6. Assess the severity of the disease.
7. Correctly make appointments to the patient, calculate the dose of drugs.
8. Be able to conduct intramuscular, intravenous injections, drip infusions, try to be able to perform the Seldinger catheterization technique.
9. Determine the patient's blood group.
10. Learn how to conduct duodenal intubation.
11. Learn how to administer enemas to the patient.
12. Learn how to conduct paraffin application of the liver.

THEORETICAL PART

JAUNDICE

Jaundice - a distinct clinical symptom of many diseases caused just personal factors, but the report livo manifested Yellow Niemi urine, mucous membranes and eye sclera. The yellow stain all tissues, ekssu date and transudates. Only saliva, tears and gastric juice do not change their color. Yellow okra Shivani associated with the accumulation in the skin, mucous membranes and other tissues pigment biliru bin due to its high content in the blood.

The exchange of bilirubin in the body is a complex and continuous process. In the interest of clinical practice ki he can be represented schematically as follows. Source bilirubin hemoglobin (protein + 4 mole kuls iron subject). The decay of erythrocytes and the distance ther conversion of hemoglobin occurs in the reticuloendothelial system (spleen, liver, bone marrow). In some cases (15%) bilirubin formed at neef ciency erythropoiesis and from hemin cells (15%). During the day, 1% of the erythrocytes circulating in the body breaks down and 100-250 mg of bilirubin is formed. The life span of erythrocytes is 100-130 days. The redox reaction involving different enzyme transformations forms a hemoglobin holeglobin, verdoglobin, biliverdin and bilirubin. Free, unbound bilirubin appears. This bilirubin is transported in the blood by the protein albumin.

Free (indirect) bilirubin is transported to the liver, where it is released from albumin and binds to glucuronic acid. This is how the bound, or as it is also called, direct bilirubin is obtained.

Related (direct) bilirubin becomes vodorastvo -soluble, which facilitates its excretion from the liver glue current in the bile. In bile, bilirubin is a part of macromolecular aggregates (micelles) consisting of cholesterol, phospholipids, bile acids, and protein salts. Ass gives to the intestines, bile bilirubin part vosstanavli INDICATES exposed bacterial dehydrogenases in demon colored urobilinogenovye body (chromogens). A significant portion of the transformed bilirubin (85-90%) returning etsya in liver and other tissues retikulogistiotsitarnoy B Stem, part excreted in the feces, coloring it in korich nevy color (stercobilin). Another part enters the kidneys and excreted in the form of urobilin, giving urine-yellow oran zhevoe staining. With jaundice, the level of bilirubin in the blood invariably rises (hyperbilirubinemia). The holes IU bilirubin level up to 20 mmol / l, and in one hundred ryh units - 1 mg%. There are three types of jaundice: mechanical, parenchyma toznaya and hemolytic.

Jaundice classification

Hemolytic (suprarenal) jaundice itensivnogo results from decay of erythrocytes and excessive generation indirect direct bilirubin. These phenomena occur with hyperfunction of the cells of the reticuloendothelial system (primarily the spleen), with primary and secondary hypersplenism. Various hemolytic anemias can serve as a typical example. In this Obra mations indirect bilirubin is so great that the liver does not have time to turn it into a linked (direct) bilirubin. Causes hemolytic jaundice can also be various facto ry, leading to hemolysis: hemolytic poisons, sucking the blood of the decay products of extensive bruising.

Classification of jaundice and its causes.

Parenchymal (hepatic) jaundice develops as a result of damage to hepatocytes, the ability of which to bind free bilirubin in the blood and convert it to bilirubin glucuronide (direct bilirubin) decreases. In this case, the formed direct bilirubin only partially enters the bile capillaries, and most of it returns back to the bloodstream. The most common causes of parenchymal jaundice are viral hepatitis, leptospirosis (Vasiliev-Weil disease), liver cirrhosis, poisoning with certain types of poisons (carbon tetrachloride, tetrachloroethane, arsenic, phosphorus compounds.

Mechanical (surgical) jaundice caused naru sheniem drainage of bile from the liver into the duodenum. All the way to the outflow of bile can occur prep Corollary due to blockage or compression of the projectile inside Ms.

The most common cause of obstructive jaundice are stones in the bile ducts (50%) and neoplasms (40%) in the ducts, large duodenal papilla, pancreas, gallbladder. Obstructive

jaundice can also be caused by other reasons (10%) - stenosis of the OBD, stricture of the ducts, atresia of the biliary tract, cholangitis, pancreatitis, liver tumors (Fig. 6.2.).

Diagnosis of jaundice

Of great importance in establishing the diagnosis of jaundice have carefully collected history, clinical and laboratory examination and bio chemistry and CBC. Optionally walk examination of feces, which should include all chat occult blood test. When examining urine, an increase in the content of bilirubin and urobilinogen should be excluded. Additional IU tody studies - ultrasound studies of the (ultrasound), liver biopsy and cholangiography (endoscopic or percutaneous) - used for indications according to the type of jaundice.

Anamnesis

Find out the *profession of the* patient; particularly important to determine whether the work is not related to the patient's con tact with the rats, which are perenoschi kami Leptospira (Weil's disease), as well as to require alcohol leniem.

The importance of *a national Supplies Nosta* patient. For example, people from the Mediterranean, Africa or the Far East may be suspected of carriage of HBV and HCV .

In the study of *family history* into account an indication of jaundice, hepatitis, anemia and splenectomy and cholecystectomy in close race stvennikov. Family history of facilitates the diagnosis of hemolytic jaundice, on the investigation of hyperbilirubinemia, hepatitis and cholelithiasis.

Find out if there was any *contact* with yellowness E patients, especially in nurseries, camps, hospitals and schools, the sick branches gemodia Lisa and drug addicts. Diagnostic value can be specified for *injection* during pos Lednov 6 months, such as blood or plasma transfusion, blood samples for analysis, drug administration ticks formulation tuberculin test, tattoos Rovkov and dental intervention. It is also important indications of eating *shellfish,* as well as *a trip* to the regions End Michna on hepatitis. Find out if the patient does not take medicines that can cause devel term jaundice.

A history of dyspepsia, zholchnoy if ki and intolerance of fat allows suspect Vat choledocholithiasis.

The development of jaundice after operations on the biliary tract is possible with abandoned stones, trauma cal stricture bile duct, as well as hepatitis. The cause of jaundice after the removal of the evil quality of neoplasms may be meta stasis in the liver.

Jaundice in alcoholism usually accompanied etsya such symptoms as anorexia, nausea in the mornings, diarrhea and a slight increase in pace ture body. It is also possible soreness Uwe lichenie liver.

The steady deterioration of the general condition and body weight reduction are characteristic zlokache stvennoj tumor.

The nature of the onset of the disease is extremely important. Beginning with nausea, anorexia, aversion to cigarettes (in smokers), as well as the development of jaundice within a few hours and its rapid progression make one suspect viral hepatitis or medicinal jaundice. Cholestatic jaundice develops more slowly and is often accompanied by persistent itching. Fever with chills is characteristic of cholangitis associated with stones or biliary stricture.

A few days before the development pechonochnokletochnoy cholestatic jaundice or darker urine is and feces become bright. When gemoliticheskoy jaundice stool color is not changed.

With hepatocellular jaundice, the general condition of the patient suffers significantly; at cholesta cal jaundice only complaint may itch or jaundice, and symptoms due to the primary disease cause obstruction.

Mild persistent jaundice of varying intensity suggests hemolysis. In cirrhosis usually mild jaundice varying an intensity and is not accompanied by changes Niemi fecal color, but when attaching the stop cerned alcoholic hepatitis jaundice can be intense bleaching feces.

Zholchnoy pain with colic can last a few hours, they rarely wears interspersed camping character. Back pain or epigastric may be due to cancer podzhel zling gland

Objective signs (Figure 6.3).

Age and gender . Gallstones are more likely to form in obese middle-aged women who have given birth. The prevalence of viral hepatitis and the ability creases with age, but with viral hepatitis are B and C, this pattern is not observed. With age, the likelihood of obstruction of the biliary tract by a malignant tumor increases. DOCTOR governmental jaundice develops in children is very rare.

Inspection . Anemia may indicate n Molise, tumors or cirrhosis. In marked decrease in body mass should be suspected OPU Chol. Hemolytic jaundice Skin you pale yellow color, with pechonochnokletochnoy jaundice - with an orange tint, and at The duration Noah biliary obstruction become green. People with pancreatic cancer often slouch. Patients with alcoholism are on observable cirrhosis stigma. Particular Atte of draw on organs, which can locale Call source of liver metastases (breast, thyroid, stomach, colon and rectum, lung) as well as the state of regi -stationary lymph nodes.

Mental status . A slight decrease Institute tellekta with minimal changes personality favors pechonochnokletochnoy des Toohey. The appearance and odor hepatic "slam present" tremor indicates possible to develop ment hepatic coma.

Skin changes . Bruising may witness update themselves on violations of blood coagulation. Devel vayuschayasya in cirrhosis thrombocytopenia purpura may appear on the forearms, in substitution antiplaque or depressions on the shins. Other changes Nia skin with cirrhosis include vascular zvoz daughter, palmar erythema, white nails and Dropped denie hair in places of secondary hirsutism.

In chronic cholestasis can identify follows rows raschosov, pigmentation caused by the excess nym deposition of melanin change fingers as drumsticks, xanthoma on the eyelids (xanthelasma) extensor surfaces and warehouse Kah palms and hyperkeratosis.

Objective signs with jaundice

Pigmentation and ulcers on the legs appear with some forms of congenital hemolytic anemia.

Care should be taken to sites on the skin, which may be malignant swelling pour. When multiple vein thrombosis excluding the removed body pancreatic cancer. Swelling Loda rods may indicate cirrhosis, as well as obstruction of the inferior vena cava, liver or pancreas tumor.

Examination of the abdomen . Expansion okolopupoch GOVERNMENTAL veins - flag enhanced collateral circulation in the portal vein (the usual but due to cirrhosis). Ascites can develop as a result of liver cirrhosis or malignant tumor. At much increased, bugris of the liver is likely that organ cancer. The small size of the liver suggests cha zholom hepatitis or cirrhosis and allow excluded chit extrahepatic cholestasis, whereby the liver is increased and has a smooth surface. In pain GOVERNMENTAL alcoholic fatty liver and cirrhosis can cause an increase in her uniform. Edge of the liver is painful for hepatitis, congestive Ser dechnoy failure, alcoholism, of bacterial Mr. cholangitis and sometimes in tumors. Arterial ny noise over the liver indicates acute alco golny hepatitis or primary liver cancer.

With choledocholithiasis, pain in the gallbladder and Murphy's symptom are possible. Palpable enlarged gallbladder, sometimes seen in the right upper quadrant, cancer requires an exception for gastric cancer.

Abdominal cavity should be thoroughly explores Vat for the exclusion of the primary tumor. Binding tion rectal examination.

Urine and feces . Bilirubinuria is an early sign of viral hepatitis and drug-induced jaundice. From the absence of urobilinogen in the urine allows pref lag complete obstruction of the common bile about the current. Prolonged urobilinogenuria, in which there is no bilirubin in the urine, indicates hemolytic jaundice.

Aholichny chair, existing within length Tel'nykh time, it confirms the diagnosis biliar-hydrochloric obstruction. With a positive sample to rule out occult blood cancer liver-pancreas ampoules, pancreas, kishech nickname and portal hypertension.

Serum biochemical parameters

An increase in the level of bilirubin in serum confirms the presence of jaundice, allows one to judge its intensity and observe its dynamics. If the alkaline phosphatase activity is more than 3 times higher than normal, the GGTP activity is increased and there are no signs of bone damage, the likelihood of cholestasis is very high; high activity of alkaline phosphatase observed also given

Levels of albumin and globulin serum in short-term jaundice changed insignificantly tion. When longer pechonochnokletochnoy jaundice albumin level is reduced, and glo Bulina - increases. In cholestatic des tuhe (electrophoresis) is detected raising and $_2$- and globulins, and at pechonochnokletochnoy jaundice - y-globulin.

In hepatitis serum transaminase activity increased to a greater extent than ho lestaticheskoy jaundice. Significant transient on Vyshen transaminase activity sometimes the Supervisory etsya in acute obstructive biliary stone.

Puncture liver biopsy

In acute jaundice, liver biopsy is rarely necessary; it is carried out in the foundations of Mr. patient with unclear diagnosis and underneath view on the On the presence of jaundice increases the risk of biopsy. Naib Leia considered safe biopsy needle Menghini. Expressed jaundice is not contraindicated Niemi liver biopsy.

When violations of blood coagulation by a conventional percutaneous biopsy is dangerous in such SLN teas transyugulyarnoy resort to biopsy or biopsy under CT or ultrasound seals Rovkov puncture channel (see chap. 3).

Diagnosis of acute viral hepatitis usual but is not difficult. The most difficult diagnosis of jaundice in cholestasis. Nevertheless, in most cases, an experienced histologist can distinguish intrahepatic cholestasis of painting, such as drug or defeat lane between primary biliary cirrhosis, the changes, the Call bathrooms obstruction of the common bile duct. Aude Naco can only be set with much less certainty the very cause of cholestasis.

Laparoscopy

Dark green color and increased liver zholch ny bubble testify in favor of extrahepatic biliary obstruction. Laparoscopy allowing a tumor to identify as nodes and perform their biopsy under visual control. With hepatitis, the liver is yellow-green; cirrhotic measurable nennaya liver has a characteristic appearance. Laparosko Pius not differentiate extrahepatic biliary obstruction, particularly due to the large bile duct cancer, and vnutripecho the parking cholestasis, drug-induced.

Table

Differential diagnosis of jaundice by clinical signs

Clinical sign	**Type of jaundice**		
	Mechanical	**Parenchymal**	**Hemolytic**
Color of the skin	Green yellow with bronze or gray from-tenkom	Red yellow with orange shade	Lemon yellow th
Itchy skin	Pronounced	Expressed	Absent

Feces color	Bleached, gray-white, clayey	At the height of the levania provide colored	Intensely painted
Urine color	Dark	Dark	Usual
Appetite	Violated at long-term tukhe	Decreased or absent	Not broken
Dyspeptic phenomena	Yes, with a long noisy jaundice	Yes	Absent
Stomach ache	Can be	No or not strong nye	No
Weakness, via-lost, weakness	With prolonged jaundice	Yes	No
Gall duct gaze	Increased at low blockage	Not increased	Not increased
Heart rate	Bradycardia	Bradycardia	Normal or tachycardia
Liver	Increased at long-term tukhe	Increased	Moderately increase lychen
Spleen	Not increased	Sometimes they increased chena	Often increase on
Increased bleeding	Yes	Yes	Yes

PRACTICAL PART
Thematic tests

1

1. A patient admitted to the clinic with phlegmonous cholecystitis in the next three days developed chills, jaundice, fever up to 380C. There were no symptoms of peritonitis. What complication of the underlying disease did the patient develop?
Stenosis of the greater duodenal papilla
Empyema of the gallbladder
Pylephlebitis
Subhepatic abscess
+ Purulent cholangitis

2. The main methods for diagnosing the nature and causes of obstructive jaundice include:
1. Plain radiography of the liver and subhepatic space
2. Infusion cholecystocholangiography

3. Percutaneous transhepatic cholangiography
4. Endoscopic retrograde cholangiopancreatography
5. Ultrasonography
1 and 5
2 and 4
1, 2, 4
2, 3, 5
+ 3, 4, 5

3. For the clinic of obstructive purulent cholangitis, the following symptoms are characteristic:
1. Jaundice
2. Chills
3. High level of alkaline phosphatase activity
4. High leukocytosis in the blood test with a shift to the left
5. Possible increase in the size of the liver
Choose the correct combination of answers:
1, 2, 3
1, 2, 4, 5
2, 3, 5
+ All answers are correct
All answers are wrong

4. Laboratory data for obstructive jaundice due to obstruction of the common bile duct with stone are characterized by:
1. Bilirubinemia
2. Bilirubinuria
3. Decrease in alkaline phosphatase in the blood
4. Lack of stercobilin in feces
5. A sharp increase in the activity of serum transaminases
Choose the correct combination of answers:
1, 2, 3
2, 3, 4
+ 1, 2, 4
3, 4, 5
2, 3, 5

5. For jaundice on the basis of choledocholithiasis is not typical:
+ Urobilirubin
Increased alkaline phosphatase
Normal or low blood protein
Increased blood bilirubin
Normal to moderately elevated transaminases

6. Obstructive jaundice caused by choledocholithiasis is not characterized by:
Hyperthermia
Increased direct blood bilirubin
Increased alkaline phosphatase
+ A sharp increase in the level of transaminases in plasma
Lack of stercobilin in feces

7. To clarify the nature of jaundice and its cause, it is not used:
CT scan

+ Intravenous cholecystocholangiography
Percutaneous transhepatic cholangiography
ERPHG
Ultrasound

8. Intermittent jaundice is caused by:
A wedged stone of the terminal section of the common bile duct
Tumor of the common bile duct
Cystic duct stone
+ Valve stone of common bile duct
The structure of the common bile duct

9. The reason for the development of obstructive jaundice in a patient can be all of the above, except:
+ Calculus in the gallbladder neck
Enlargement of the head of the pancreas
Concrement in the proximal part of the common bile duct
Papillitis
Stenosis of the duodenal papilla

SITUATION TASKS

1. A 55-year-old patient suffering from chronic calculous cholecystitis, against the background of an exacerbation, developed sharp pains in the right hypochondrium, nausea, vomiting, a few hours later, yellowness of the sclera appeared, the level of blood amylase was 256 units) What complication should be considered ?

2. A patient who was admitted to the clinic with phlegmonous cholecystitis in the next three days developed chills, jaundice, fever up to 380C. There were no symptoms of peritonitis. What complication of the underlying disease did the patient develop?

3. A 76-year-old patient was admitted to the clinic with a picture of obstructive jaundice, was ill for a month. The examination revealed cancer of the head of the pancreas. Suffers from diabetes mellitus and hypertension. What type of treatment is preferred:

4. At retrograde cholangiopancreatography in a patient with obstructive jaundice, extended stenosis of the mouth of the common bile duct was revealed. Which intervention should be preferred:

5. A 62-year-old patient was transferred from an infectious diseases hospital with a diagnosis of mecha-nic jaundice. A complex of laboratory and instrumental studies revealed that the cause of jaundice is volumetric changes in the pancreas, the nature of which is not entirely clear. Which of the laboratory methods is most informative for the differential diagnosis of chronic pancreatitis and pancreatic cancer?

POSTCHOLECYSTEKTOMIC SYNDROME

The purpose of the lecture : Relationship students with biliary tract disease operated, causes a mi their development, clinical features, complications over n GOVERNMENTAL forms, differential diagnosis, optimal methods leche e Nia, postoperative care, rehabilitation patients.

Educational purposes lectures: Suggestion students need for timely adequate operation to the development of serious complications , and in their development - meeting the most informative and modern methods of diagnosis and surgical treatment of patients, familiarity with in s possible complications is the operation and the operational period of Prof. and galaxies, ... Development of students' clinical thinking. Development of present-day e alternating view of the problem the issue from the perspective of world medicine and general practitioners.

Lecture objectives:

36. To give an idea of the diseases of the operated biliary tract.
37. Explain the causes and mechanisms of complications.
38. Give a clinical description and possible variants of the course of the disease.
39. Make a differential diagnosis with other diseases I mi.
40. To acquaint students with modern and most informative s methods, which examination of patients
41. Demonstration of examples of their surgical practice: patients, slides, cholangiograms.
42. All the lecture material for students to prepare and present, in an b Birmingham, necessary for quality training of general practitioners.

PLAN OF THE LECTURE.

31. The urgency of the problem - 5 min
32. Clinical picture - 10 min
    a) Clinic and diagnostics.
    b) Differential diagnosis.
    c) Treatment.
33. Diagnostics. - 10 min
34. Differential diagnostics. - 10 min
35. Treatment - 15 min

7. Disease prevention - 10 min

In acute destructive inflammation of the gallbladder and complicated forms of chronic cholecystitis, the most rational method of treatment is surgery. Timely removal of the altered gallbladder and restoration of the patency of the bile ducts prevent the patient from working. Currently, thanks to the introduction of new diagnostic / cholangiography methods holangiomanomet - Rhee, holedoskopii) significantly improved the immediate and long-term results of the surgery.

Nevertheless, in a number of patients in 5-30% of cases, the operation does not bring the expected result. Patients continue to complain of constant or periodic attacks of various pains in the hypochondrium and in the epigastric region, accompanied by a short-term increase, sometimes with chills, a more or less pronounced feeling of bitterness in the mouth, belching, etc. In this case, patients lose their ability to work for a long time ...

The appearance of a similar condition, known in the literature as postcholecystectomy syndrome, is mainly due to the following reasons. First of all, it is associated with organic and functional changes in the liver, pancreas and gastrointestinal tract, which developed as a result of prolonged disease of the gallbladder. Such patients require long-term therapeutic treatment, surgical intervention is not indicated for them.

The second reason for PCES is not a radical surgical intervention on the gallbladder, which is usually associated with a serious condition of the patient, which did not allow an adequate operation to be performed. Yet it depends on the insufficient qualifications of the surgeon.

Finally, p / cholecystectomy syndrome occurs as a result of technical errors made during the operation. The latter There are gross violations of the topographic and anatomical relationships in the area of the gallbladder and ducts, which is often associated with the presence of significant inflammatory changes. Technical errors are also possible with anomalies in the development of the biliary tract.

As a result, it is necessary to observe patients in whom the stone was not removed from the common g / duct, a long stump of the cystic duct or even a part of the g / bladder was left, the damage to the g / duct, which the surgeon made during the intervention, was not recognized in a timely manner, etc.

Thus, PCES is a collective term. Under this name, patients with various types of pathology after surgery on the biliary tract are united, some of which require repeated surgical treatment. In this lecture, patients with gland pathology will be considered.

Long stump of the cystic duct

A long stump of the n / duct can cause excruciating persistent or paroxysmal pain in the right hypochondrium. The occurrence of pain is associated with the presence of inflammation or entrapment of the stone in the stump of the duct, as well as neuroma of the stump. Often, the end of the stump expands in a bulbous manner, imitating a small w / bladder, in which acute inflammatory changes can be observed.

Leaving a long stump of the p / duct can cause persistent pain, and in some cases is accompanied by the phenomenon of cholangitis. Diagnosis is by intravenous cholangiography. Treatment is operative and consists in resection of

the excess stump of the p / duct. During the operation, a thorough revision of the extrahepatic ducts and adjacent organs must be performed.

Abandoned bile duct stones

Stones in f / streams can be left during emergency operations in the presence of pronounced inflammatory tissues. This is facilitated by the features of the topography of the distal parts of the general w / p. making it difficult to palpate, where stones are often located. In addition, the lack of conditions for the use of interaoperative cholangiography also contributes to this kind of diagnostic and tactical errors. The differential diagnosis between "forgotten" and recurrent stones (newly formed) is very difficult to carry out, especially in cases when they appear several years after the first operation.

The diagnosis is made on the basis of clinical data and laboratory studies of bilirubinemia, an increase in alkaline fasfatase. In the absence of jaundice, internal cholangiography helps to recognize the stage of the disease, in which in most cases it is possible to identify calculi in the g / ducts. In unclear cases - in the presence of jaundice or suspicion of a malignant lesion of the extrahepatic g / tract, Vater's papilla, pancreas - it is possible to use it through hepatic cholangiography. It should be borne in mind that after this The investigations - dovaniya are bleeding from the bile leakage into the free peritoneal cavity, which may require urgent surgical intervention. Therefore, in some cases, it is more expedient to perform an operation during which a thorough revision of the extrahepatic ducts, liver, p / stomach gland and other nearby organs. Often, cholangiography performed on the operating table allows you to clarify the diagnosis.

To remove calculi from the common g / duct, a supraduodenal choledochotomy is usually performed. The stones are removed with special spoons, forceps or forceps. In case of infringement of a stone in the Vater nipple, duodenotamia and papillotamia are used. In the absence of the phenomena of cholangitis, the section of the duct can be protected tightly with an atraumatic needle, otherwise it is necessary to drain by one of the accepted methods. When combined supraduodenal choledochotomy with duadenopapillotomy, it is necessary to drain the bile ducts. To do this, from the ends of the drainage tube, they pass through the incision of the common bile duct and the dissected Vater papilla and duodenum, and the second is taken out through the operating wound.

Narrowing of the distal common bile duct .

The occurrence of this pathology is played by calculi, causing constant irritation of the mucous membrane of the distal common bile duct, the head of the pancreas is often involved in the process. Inflammatory changes in some patients are so pronounced that, despite the elimination of the underlying cause, the disease progresses and ultimately lead to complete obstruction of the common bile duct. In this case, cicatricial changes can be diffuse, affecting the entire distal common bile duct. The clinical picture is dominated by pain, which is constant or paroxysmal,

and intermittent jaundice with fever and chills. In the terminal stage of the disease, jaundice is constant and severe. Blood tests show leukocytosis, accelerated ESR, increased bilirubin levels and blood alkaline phosphatase. Intravenous cholangiography in the absence of jaundice shows characteristic narrowing of the common bile duct. Diagnosis is difficult with persistent jaundice, when it becomes necessary to Differentiate this complication with obstructive jaundice caused by malignant lesions of the extrahepatic tract or the head of the n / stomach gland. In these cases, duodenography and percutaneous transhepatic cholangiography may help to some extent. With intraoperative cholangiography, choledochoduodenoanastamosis is the operation of choice for better bile outflow.

Biliary fistulas.

Of decisive importance for the diagnosis is an indication of a previously transferred cholecystostomy. However, to clarify the diagnosis, fistulocholangiography should be performed. Its technique is simple and is as follows. In the course of the fistula, withdraw a rubber tube with an inflatable cuff to prevent the flow of bile between the tube and the walls of the fistula. After the introduction of a contrast agent, an x-ray is taken. Treatment consists in the removal of the w / bladder. In the presence of calculi of the bile ducts, they are removed in these cases, they end with the drainage of the common bile duct.

In some cases, fistulas are the result of damage to segmental f / ducts located superficially in the area of the g / bladder bed. In these cases, after cholecystectomy, moderate bile secretion is noted for two months. The diagnosis is made using fistulocholangiography, in which only the fistulous passage and the small ducts communicating with it are filled. Such biliary fistulas usually close on their own and do not require surgical treatment. Traumatic damage to large g / ducts is often observed with atypical formation of the common hepatic and common g / duct, as well as with a pronounced inflammatory process in the hepatic-12 p / ligament, which sharply changes the topographic anatomical relationships. In this regard, during cholecystectomy, care must be taken when highlighting the posterior surface of the w / bladder neck in order to avoid damage to the proximal choledochus or the common hepatic duct. When resecting the stomach, damage to the distal common bile duct is possible. Through the resulting external bile fistula, up to 1 liter of bile can be released per day. To eliminate the formed fistula, several surgical interventions have been proposed, each of which has its own indication and contraindication. Among them, the most accessible is fistuloenterostomy. The essence of the operation is to connect the bile fistula isolated over a length of 2 - 3 cm with the Roux-off loop of the small intestine, held to the site of the anastomosis in the subcutaneous tissue. This operation refers to a non-radical surgical intervention and is used in three cases when the fistula is supported by an irreparable parasitic tumor or there is a contraindication, because the use of complex intra-abdominal anastomoses (the presence of a pronounced adhesive process, the general grave condition of the ball, etc.). The operation of choice when crossing the common bile duct is the

imposition of an anastomosis between the damaged segments end-to-end on the so-called lost drainage, the distal end of which is brought out through the Vater papilla to the end of the 12 p / intestine. The drainage goes away on its own after 3-4 weeks.

This operation is possible only if there is no tension between the mobilized ends of the crossed duct, as well as cholangitis. The disadvantages of this method include the possibility of early withdrawal of "lost drainage (after 2-3 days), which sharply worsens the conditions for the healing of the anastomosis. At the same time, with a prolonged delay in drainage in the duct, a repeated operation is often required to remove it, since due to the incrustation of salts, drainage can cause cholangitis, blockage with the development of obstructive jaundice and biliary cirrhosis.

Bile diversion with traumatic damage to the duct can be carried out by other methods, by imposing an anastomosis between its proximal segment and the loop of the small intestine or 12p / intestine. The anastomosis forms on the drainage the distal end of which is brought out through a separate incision of the intestine and abdominal wall. After healing, the drainage is removed. With a high obstruction of the common hepatic duct, the width of the anastomosis can be increased by the left duct.

The effectiveness of such interventions is evidenced by the rapid healing of the superimposed anastomosis.

Traumatic injury in the area of the confluence of the lobar ducts and the proximal part of the common hepatic duct is the most severe in its consequences. In this case, only intrahepatic ducts can be used to drain bile, and finding them is very difficult. The solution of the issue is helped by the patterns established by the last time, the formation and location of the latter, corresponding to these data are small, merging with each other. form large segmental ducts that drain certain areas of the liver. There are 8 segmental liver ducts in total. Their exposure can be performed from the lower surface in the area of the anterior and posterior corners of the liver to the area of the anterior-right edge of the gallbladder. The isolated duct is anastomosed with the supplied loop of the small intestine, preferably a tank to the side.

In conclusion, it should be noted that for the prevention of complications during surgery, X-ray contrast studies should be widely used, even in the absence of jaundice in the anamnesis. The use of intraoperative cholangiography avoids leaving stones in the ducts. For this purpose, it is advisable to use choledochoscopy.

In order to avoid damage to the common v / duct during cholecystectomy, one should not seek to remove the v / bladder from the neck, especially if there is an inflammatory infiltrate or scars in this area that prevent the release and separate ligation of the cystic artery and duct. In these cases, it is safer to remove the w /

bladder alone, then, after evacuating its contents by puncture, open the lumen and examine it from the inside. This technique makes it easy to isolate the cystic duct.

It is also important to emphasize the desirability of a serious study of highly complex and common liver diseases. gallbladder and w / ways. It is necessary to concentrate these patients in specialized departments, where there is experience in their surgical treatment.

QUESTIONS

to the audience to establish feedback and clarify the achievement of the lecture goal

1. Give a definition to the concept of postcholecystectomy syndrome.
2. Name the most common cause of PCES.
3. What is a residual stone?
4. The most informative diagnostic methods for PCES
5. GP tactics in patients with PCES
6. Prevention of PCES.
7. Decipher RHPG and EPST, CHCHS, HDA

QUESTION N Prom HA T full-time CONTROL IN HOSPITAL SURGERY

1-space questions

Ticket

1. Varicose veins, clinic, specially s e view s of examination and treatment. Klipela-Trennone Syndrome. Etiopathogenesis. Clinic.
1. Renovascular hypertension. Pathogenesis, diagnostics, operative intervention and treatment.

Ticket

1. General symptomatology of esophageal disease, examination methods.
2. The syndrome of vasorenal hypertension. Etiopathogenesis, clinical picture, surgical treatment.

Ticket

1. First aid for esophageal burns. Early and later with a burn of the esophagus.
2. Liver abscess. Etiopathogenesis, classification, developmental pathways and clinical picture.

Ticket

1. Diverticulum of the esophagus. Etiology, classification and treatment.
2. I.B.S. Etiology, clinic, invasive and non-invasive examination methods.

Ticket

1. Takayasu's syndrome. Clinical forms, clinic, diagnosis and treatment.
2. PTFS. Etiology, pathogenesis, clinical picture and diagnostics.

Ticket

1. The concept of cardiospasm. Classification, treatment (cardiodilation technique, conservative and surgical treatment).
2. Concept of diabetic angiopathy clinic and treatment.

Ticket

1. First aid for esophageal burns. Early and later with a burn of the esophagus.
3. Leriche syndrome. Stages of circulatory disorders, diagnostics.

Ticket

1. Occlusive diseases of the lower extremities. Special examination methods. Surgical treatment.
2. Thrombophlebitis of the superficial veins of the lower extremities. Etiology, clinic and treatment.

Ticket

1. Raynaud's disease. Raynaud's syndrome. Etiology, clinic and treatment.
2. Intrahepatic form of portal hypertension. Etiology and surgical treatment.

Ticket

1. Occlusive disease of arterial vessels. Etiology, classification, diagnosis, examination methods.
2. Concept of diabetic angiopathy clinic and treatment.

Ticket

1. Echinococcosis of the liver. Clinic and complications. Immunological and serological diagnostic methods.
2. I.B.S. Diagnostics, indications for surgery.

Ticket

1. Varicose veins. Etiology, classification. Parks Weber-Rubashov syndrome.
2. Obliterating atherosclerosis of the lower limb arteries.

Ticket

1. Methods of surgical treatment of portal hypertension.
2. Takayasu's syndrome. Classification, clinic, diagnosis and surgical treatment.

Ticket

1. Cicatricial narrowing of the esophagus. Clinical manifestation. Diagnostics and treatment.
2. Alveococcosis of the liver. Etiology, clinic. Complication of alveococcosis surgical treatment.

Ticket

1. Concept of portal hypertension, etiology, classification. REH and application.
2. Acute deep vein thrombophlebitis of the lower extremities. Etiology, diagn Ostik , complication and lech tim e.

Ticket

1. Echinococcus of the liver. Etiology, classification, routes of infection, treatment.
2. PTFS. Causes, forms, pathogenesis, diagnosis and treatment.

Ticket

1. Achalasia of the esophagus. Etiology, classification, clinic, diagnosis.

2. Syndrome of Pagett-Schrötter and Klippel-Trenonne. Etiology, clinic, treatment

Ticket

1. Takayasu's syndrome. Clinical manifestations in subclavian, vertebral and carotid arterial forms. Examination and treatment methods.
2. Burns of the esophagus. Etiology, clinic, classification. Late bougie techniques.

Ticket

1. Syndrome of Pagett-Schrötter and Klippel-Trenonne. Etiology, clinic, treatment
2. Causes of nospecific aorto-arteritis, clinical picture and diagnosis.

Ticket

1. Methods of haemorrhage with profuse bleeding of the esophagus.
2. Acute phlebothrombosis of deep veins of the lower extremities. Etiology. White and blue phlegmas, clinic and treatment.

Ticket

1. Functional tests that determine the patency of veins and special research methods. Factors contributing to the outflow of venous blood.
2. Obliterating endoarteritis. Etiology, classification, clinic. Burger's disease. Etiology and clinic.

Ticket

1. Burns of the esophagus with chemicals. Clinic and degree of burn. Indication for both early and late bougienage.
2. Vasorenal hypertension. Signs, clinic treatment.

Ticket

1. Diverticulum of the esophagus. Classification, clinic, diagnostics. Complication of diverticulum.
2.Leriche's syndrome. Obliterating endoarthritis, obliterating atherosclerosis, differential diagnosis.

Ticket

1. Thrombophlebitis of the superficial veins of the lower extremities. Etiology, clinical picture and complications.
2. Takayasu's syndrome. Clinical signs of nameless artery syndrome, methods of examination and treatment.

Ticket

1. Thrombophlebitis of the deep veins of the lower extremities. Thrombophlebitis formation factors.
The concept of phlebothrombosis and its complications.

2. Occlusive arterial vessels, their stenosis, etiopathogenesis, methods of examination and treatment.

Ticket

1. Classification of symptomatic hypertension. Pheochromastoma. Connes syndrome. Itsengo-Cushing's syndrome. Causes, diagnosis and treatment.
2. Echinococcus of the lung. Causes, stages of development, complications. Clinical and radiological signs.

Ticket

1. Classification of symptomatic hypertension. Pheochromastoma. Conn's syndrome. Itsengo-Cushing's syndrome. Causes, diagnosis and treatment.
2. Syndrome of abdominal ischemia, causes. Mesenteric form, clinic and types.

Intermediate 2- ton full-time issues

Ticket

1. Benign lung tumors. Classification. Clinic of a central benign tumor. Jackson stenosis.
2. Chronic lung abscess. Etiology, clinic and treatment.

Ticket

1. Recurrent peptic ulcers. Etiology, clinic, diagnostics and treatment.
2. Acute thrombosis and embolism. Etiology, pathogenesis, clinical picture and treatment.

Ticket

1. Mitral stenosis, etiology, hemodynamic disturbances in stepenes of stenosis. Diagnostics.
2. Bronchiectasis. Etiology. Classification. Clinic. Diagnostics.

Ticket

1. Panyate about pneumathorax and pyopneumathorax Types, clinic, tactics and treatment.
2. Acute and chronic loop adductor syndrome. Etiology, diagnosis. Surgical treatment methods.

Ticket

1. Chronic empyema. Pathological changes. Clinic, diagnosis and treatment.
2. Gangrene of the lungs and gangrenous abscesses. Etiology, clinic and diagnostics.

Ticket

1. Cysts of the lungs. Classification. Etiology, clinic and diagnostics.
2. Classification of acute and chronic arterial thrombosis. Causes of thrombosis and embolism. Objective and subjective signs of acute embolism.

Ticket

1. The concept of heart disease. Classification. Lyutenbache syndrome. Hemodynamic disturbance treatment.

2. Peptic ulcers of the stomach. Etiology, types of Zollinger-Ellison syndrome. Diagnostics.

Ticket

1. Acute and chronic adductor loop syndrome. Etiology, clinic.
2. Acute thrombosis of the lower and upper extremities. Pathogenesis. Tactics, conservative treatment for thrombosis and embolism.

Ticket

1. Coarctation of Alora. Clinic options.
2. Post-resection syndrome. Etiology, pathogenesis and classification.

Ticket

1. Etiology of aortic stenosis. Clinic. Hemodynamic disorders.
2. Mesenteric thrombosis etiology, clinical picture and treatment.

Ticket

1. Acute thromboembolism of arterial vessels. Clinic and diagnostics.
2. Spontaneous pneumothorax. Etiology, clinic and treatment tactics.

Ticket

1. Peripheral lung tumors. Stages and clinical features. Treatment methods.
2. Dumping Syndrome. Early and late Dumping syndrome. Etiology, clinic and diagnostics.

Ticket

1. Mesenteric thrombosis. Clinic, diagnostic methods.
2. Embologic and thrombogenic causes of acute thrombosis and embolism.

Ticket

1. Causes of pneumothorax. Classification, clinic treatment.
2. Opening the ductus arteriosus. Etiology, hemodynamic disorders. Eisenmenger's syndrome.

Ticket

1. Echinococcus of the lungs. Etiology, clinic, diagnosis, complication and treatment.
2. Benign tumors of the mediastinum. Classification, clinic diagnostics.

Ticket

1. Benign lung tumors. Classification, tactics of surgical treatment.
2. Hernia of the esophageal opening. Treatments.

Ticket

1. Acute pleural empyema. Etiology, classification, diagnosis.
2. Complication of pulmonary echinococcus. Clinic, X-ray diagnostics.

Ticket

1. Relaxation of the diaphragm. Etiology, classification, clinic and diagnosis.
2. Gastrointestinal (small intestinal and colonic) fistulas. Etiology, clinic, diagnosis and treatment.

Ticket

1. Paraesophageal and sliding hernia of the esophagus. Clinic, clinical and radiological signs, surgical methods.
2. Tetrad of Fallot. Etiology, clinic and surgical methods of treatment.

Ticket

1. Post-resection syndrome. Classification, diagnostics.
2. Mediastinal cysts. Classification. Diagnostics and treatment.

Ticket

1. Diaphragmatic hernia. Classification, clinic. Etiology. Clinic and treatment.
2. Chronic empyema. Etiology. Clinical and morphological stages. Clinic and surgical treatment.

Ticket

1. Opening the ductus arteriosus (Botall). Etiology. Hemodynamic violation. Diangnostic research.
4. Chronic lung abscess. Etiology. Clinic, complication and treatment.

Ticket

1. Zollinger-Ellison syndrome. Kinds. Clinic and diagnostics.
2. Mitral stenosis. Etiology. The degree of stenosis. Hemodynamics, clinic and treatment.

Ticket

1. Mediastinal cysts. Clinic, diagnosis and treatment.
2. Stenosis of the aortic valve. Etiology. The degree of stenosis. Clinic, treatment.

T E S T ’S ON THE HOSPITAL SURGERY

1. Where is the physiological narrowing of the esophagus. Please enter a wrong answer.
On the back of the heart
Pharyngeal-esophageal part of the esophagus
To the levels of tracheal bifurcation
Above the esophageal opening of the diaphragm

2. Direction of venous outflow from the lower 1/3 of the esophagus. Please enter a wrong answer.
Joins the abdominal artery
Joins the portal vein system
Joins the inferior vena cava system
Joins the superior vena cava system

3. Special methods for examining the esophagus. Please enter a wrong answer.
Thoracoscopy
Fluoroscopy
Fibroesophagoscopy
Esophagotonokymography

4. Common symptoms of esophageal diseases. Please enter a wrong answer.
Constipation
Chest pain
Regurgitation
Salivation

5. Causes of bleeding from the esophagus. Please enter a wrong answer.
Cicatricial narrowing of the esophagus
Portal hypertension
Esophageal carcinoma
Paraesophageal strangulated hernia

6. What substance leads to chemical burns of the esophagus. Please enter a wrong answer.
Hot water
Acetic acid
Ammonia
Caustic soda

7. What organs are most often affected by chemical burns of the esophagus. Please enter a wrong answer.
Small intestine
Physiological narrowing of the esophagus
Pharyngoesophageal narrowing
Stomach

8. Pathalagoanatomical changes with a burn of the esophagus. Please enter a wrong answer.
Esophageal spasm
Esophageal mucosa necrosis
Necrosis of the entire wall of the esophagus
Damage to the epithelium of the esophagus

9. Clinical signs of the acute stage of esophageal burn. Please enter a wrong answer.
Sepsis
Signs of shock
Increased body temperature, decreased urine
Dysphagia

10. Clinical signs of a burn of the esophagus in the recovery stage. Please enter a wrong answer.
Cardiospasm
Cicatricial narrowing of the esophagus
Esophagitis
An esophageal-bronchial fistula is formed

11. What is not used for profuse bleeding of the esophagus from varicose veins?

Endoscopic diathermocoagulation
Blackmore Probe
Pituitrin
Embolization of varicose veins of the esophagus

12. Clinical signs with a burn of the esophagus in the stage of incomplete recovery. Please enter a wrong answer.

Subcutaneous emphysema
Perforation of the esophagus and the development of mediastinitis
Sepsis
Formation of bronchial fistulas in the esophagus

13. Signs manifested in the stage of stricture formation, after a burn of the esophagus.

Gradually developing dysphagia
Sepsis
Mediastinitis
Lung abscess

14. First aid for burns of the esophagus with acetic acid. Please enter a wrong answer.

Overlay tracheostomy and gastrostomy
Gastric lavage with 2% soda solution
Narcotic analgesics
Antihistamines

15. What days are early bougienage performed for burns of the esophagus

7-15 days
2-3 days
4-6 days
13-16 days

16. Methods for the prevention of post-burn strictures. Please enter a wrong answer.

Stimulation of diuresis
Prescribing medicine and food
Corticosteroids
Early bougie

17. Clinical signs of post-burn esophageal strictures. Please enter a wrong answer.

Bleeding
Dysphagia
Belching
Chest pain

18. Complications of post-burn narrowing of the esophagus. Please enter a wrong answer.

Hernia of the esophageal opening of the diaphragm
Diverticulum formation
Cachexia
Chronic esophagitis

19. X-ray signs of esophageal narrowing. Please enter a wrong answer.

Symptom Beggar
Tubular constriction
Inverted candle symptom
Suprastenotic expansion

20. Basic principles of surgical treatment for cicatricial esophageal narrowing. Please enter a wrong answer.

Geller's operation
Plastic surgery of the esophagus with a segment of the colon
Plastic surgery of the esophagus with a segment of the small intestine
Plastic esophagus stomach

21. Complications of bougienage. Please enter a wrong answer.

Esophageal carcinoma
Damage to the esophageal mucosa
Bleeding
Esophageal perforation

22. How is bougienage performed in the presence of a gastrostomy.

Retrograde bougie on "Nita"
Under the control of an esophagoscope
Bougie through the mouth to the "blind"
Dilator Stark

23. A type of bougienage with suprastenotic expansion of the esophagus.

Under the control of an esophagoscope

Bougie through the mouth to the "blind"
Retrograde bougienage
"Bougie without end"

24. The main symptoms of cardiospasm. Please enter a wrong answer.

Gastroesophageal Reflux Esophagitis
Dysphagia
Regurgitation
Pain, belching

25. Pathological changes in cardiospasm. Please enter a wrong answer.

Shortening of the esophagus
Expansion of the esophagus
Narrowing of the distal esophagus
Cicatricial changes in the cardiac sphincter

26. The main signs of achalisia of the cardia.

Dysphagia, regurgitation, pain
Dysphagia, neck bulge, pain
Pain, dysphagia, diarrhea
Constipation. bleeding, pain

27. X-ray signs of cardiospasm.

Expansion of the esophagus, fluid level, mouse tail symptom
Dilation of the esophagus, cascading stomach
Liquid level, free gas above the diaphragm, dilated stomach
Dilation of the esophagus, multiple Kloyber's bowls, “barium spots” symptom

28. Indicate the main methods of cardiospasm treatment.

Cardiodilation
Antihistamines
Antacid therapy
Thermoelectrocoagulation of the mucous membrane

29. What kind of dilator is cardiodilation done?

Plummer pneumatic cardiodilator
Fogarty balloon catheter
With a metal bougie
Using a gastroesophagoscope

30. Indications for surgical treatment of cardiospasm. Please enter a wrong answer.

Positive nitroglycerin test
Ineffectiveness of cardiodilation
S shaped deformity of the esophagus
Inability to perform cardiodilation

31. Types of operations used for cardia achalasia. Please enter a wrong answer.

Judah pyloroplasty
Geller Cardioplasty
Geller's operation with Nissen fundoplication
Operation Shalimov-Godstein

32. Is it a limited protrusion of the esophageal wall?

Diverticulum of the esophagus
Mallory-Weiss syndrome
Takayasu syndrome
Barrett's syndrome

33. By localization, the diverticula of the esophagus are subdivided. Please enter a wrong answer.

Abdominal
Zenker's
Bifurcation
Epiphrenal

34. The reasons for the formation of a pulsatile diverticulum of the esophagus.

High intraesophageal pressure
Inflammatory process in the surrounding tissues
Insufficiency of the Baugen valve
Insufficiency of the pyloric sphincter

35. What is the cause of the formation of traction esophageal diverticulum?

Adhesive inflammatory process in the surrounding tissues of the esophagus
High intraesophageal pressure
Compression by diaphragmatic hernia

Insufficiency of the cardiac sphincter

36. Where do Zenker's diverticula form?

In the wall of the pharynx above the entrance to the esophagus
In the area of the Lestgaft triangle
In the middle third of the esophagus
In the lower third of the esophagus

37. Complications of Zenker's diverticulum?

Phlegmon of the neck, hoarseness
Acute heart failure
Cardiospasm
Reflux esophagitis

38. Where is the epiphrenal diverticulum formed?

Lower third of the esophagus, above the diaphragm
Cardiac part of the stomach
Pharyngeal esophagus
Middle third of the esophagus

39. Clinical signs of Zenker's diverticulum. Please enter a wrong answer.

Chest pain
Bad breath
Regurgitation
Blockade phenomenon

40. Clinical signs of a bifurcation diverticulum. Please enter a wrong answer.

Bulging on the side of the neck
Asymptomatic course
Hiccups
Belching

41. With epiphrenal diverticula observed?

Lower sternum pain, dysphagia
flatulence
Cardiospasm
Headaches

42. Specify the instrumental methods for examining the diverticulum of the esophagus.

X-ray examination of the esophagus
Ultrasound
Thoracoscopy
Irrigography

43. Indications for surgical treatment for esophageal diverticula. Please enter a wrong answer.

All cases of definition of diverticula
For complications of diverticulum (perforation, bleeding, cancer, fistula, ulcer)
Large diverticulum
Esophageal diverticulum ulcer

44. The most common types of operations used for diverticula of the esophagus.

Diverticulectomy
Plastic surgery of the esophagus with a segment of the small intestine
Electrocoagulation
Filling

45. Direction of conservative treatment for diverticula

To prevent food from entering the diverticulum
To prevent esophageal stricture
Prevention of pyloric stenosis
Prevention of stricture of the esophagus of the diaphragmatic foramen

46. Methods of surgical treatment for esophageal burn strictures. Please enter a wrong answer.

Tanner's operation
Esophagoplasty of the small intestine
Colon esophagoplasty
Esophagoplasty of the stomach

47. The average length of the esophagus in men and women.

25cm
30-36cm
26-32cm
29- 35 cm

48. Do coagulative necrosis cause?

Acids

Caustic soda
Potassium permanganate
76% alcohol

49. Is colliquation necrosis caused?

Alkalis
Potassium permanganate
76% alcohol
Acids

50. What physiological constrictions does the esophagus have?

Pharyngoesophageal, bifurcation, epiphrenal
Pharyngoesophageal, epibronchial
Epiphrenal, epibronchial, esophageal
Pharyngoesophageal, cardiac, epibronchial

51. The diameter of the esophagus is normal.

2.7-3 cm
4 cm
4 - 5 cm
1- 1.5 cm

52. What is not included in the function of the esophagus?

Regurgitation
Swallowing
Belching
Vomiting

53. Symptoms not common to esophageal disease?

Mallory-Weiss syndrome
Dysphagia, pain, feeling of pressure
Weight loss, regurgitation
Bleeding, vomiting, belching

54. For what stage of achalasia the cardia corresponds according to the classification of B.V. Petrovsky expansion of the esophagus with persistent spasm?

2 stages
Stage 1
3 stages
4 stages

55. For what stage of achalasia the cardia corresponds according to the classification of B. Petrovsky scar changes in the muscle layers of the lower esophageal sphincter with a pronounced expansion of the esophagus?

3 stages
Stage 1
2 stages
4 stages

56. For what stage of achalasia the cardia corresponds according to the classification of B.V. Petrovsky S- shaped deformity of the esophagus?

4 stages
Stage 1
2 stages
3 stages

57. For what stage of achalasia the cardia corresponds according to the classification of B.V. Petrovsky functional spasm without expansion of the esophagus?

Stage 1
2 stages
3 stages
4 stages

58. What are the most common symptoms of cardiospasm? Please enter a wrong answer.

Bleeding
Dysphagia
Regurgitation
Chest pain

59. Early complications of esophageal burns. Please enter a wrong answer.

Achalasia cardia
Mediastinitis
Perforation
Bleeding

60. The degree of stricture of the esophagus with a narrowing diameter of 1 - 2.5 cm ?
1st degree
2nd degree
Grade 3
4 degree
61. Indicate the diameter of the esophagus is normal.
2.7-3 cm
1- 1.5 cm
4cm
4-5cm
62. Total plastic of the esophagus. Please enter a wrong answer.
Retrocostal
Antisternal
Retrosternal
Transpleural
63. Esophagoplasty with the help of the greater curvature of the stomach - is it?
Operation ZysnoHalpern
Operation Jadu Tonaki
Finisterer's operation
Operation Billroth
64. An operation used to treat cardiospasm. Please enter a wrong answer.
Organoanostomosis
Geller operation
Operation Petrovsky
Operation Gotstein-Shalimov
65. What is the method used for stump plasty after diverticulectomy? Please enter a wrong answer.
According to Kolesov
According to Vishnevsky
According to Petrovsky
According to Depek
66. What material is used for plastic surgery according to the Depek method for esophageal diverticulectomy?
Parietal pleura
Lung tissue
Synthetic material
Diaphragm
67. Indications for surgical treatment for cicatricial narrowing of the esophagus. Please enter a wrong answer.
Taper diameter up to 3 cm
Bougie inefficiency
Perforation of the esophageal wall during bougienage
Narrowing of the esophagus 4-5 degrees according to Belokonov-Ratner
68. What are the complications of tsenker diverticulum? What is the wrong answer?
perforation of a diverticulum into the aorta
The appearance of an ulcer in the diverticulum
Aspiration pneumonia
Phlegmon of the neck
69. Complications of bifurcation diverticulum. Indicate the wrong answer?
superior vena cava perforation
diverticulitis
Perforation and mediastinitis
Perforation, pleural empyema
70. Complications of the epiphrenal diverticulum. Indicate the wrong answer?
phlegmon of the neck

diverticulitis

Ulceration in the area of the diverticulum
Perforation, pleural empyema

71. Who described the first time esophageal diverticula?

Rakketan
Iodlov
Nikoledoin
Billroth

72. Is it true diverticula of the esophagus?

bulging of all layers of the esophagus
mucosa and submucosa of the esophagus
muscle layer
mucous membrane only

73. Who was the first to describe Traumatic hernia of the diaphragm?

Ambruz Paré 1594g
Jean Pitti 1774
Larey 1842
Pirogov 1844

74. Where is bilirubin formed?

in the reticuloendothelial system
digestive system
circulatory system
muscular system

75. Who first described the relaxation of the diaphragm?

Jean Pitti 1774
Ambruz Paré 1594g
Larey 1842
Pirogov 1844

76. What is not included in the bilirubin system?

the effects of glucoronic acid on diglucronoid
hemolysis of erythrocytes
hemoglobin formation
compound of biliverdin with hydrogen

77. What is not included in the education system of Urobilogen?

synthesis of bilirubin in the kidneys
the effects of glucuric acid on bilialbumic acid
recovery of bilirubin glucuronide when exposed to enzymes
secretion of bile

78. The level of direct bilirubin in the blood according to the SI system.

from 0.9 to 4.3 μmol / l
from 0.1 to 0.2 μmol / l
from 0.2 to 0.3 μmol / l
from 1.0 to 0.8 μmol / l

79. The level of total bilirubin in the blood according to the SI system?

from 1.1 to 18.8 μmol / L
from 0.1 to 1.0 μmol / l
from 1.0 to 0.2 μmol / l
from 2.0 to 4.0 μmol / l

80. The level of indirect bilirubin in the blood according to the SI system?

from 1.7 to 13.0 μmol / l
from 0.1 to 0.2 μmol / l
from 0.2 to 0.3 μmol / l
from 0.3 to 0.6 μmol / l

81. What is not the cause of parenchymal jaundice.

congenital spherocytosis
viral hepatitis
cirrhosis of the liver
toxic hepatitis

82. Echinococcus is it?

parasitic disease
infections
allergic diseases
endocrine diseases

83. Ways of infection with echinococcosis?

through the mouth
through blood
sexual
with insect bites

84. Method of surgery for subcompensated stage of portal hypertension

distal splenorenal anastomosis
treatment with antihypertensive drugs
diuretic treatment
Closure of bleeding veins in the esophagus

85. What is used to improve liver regeneration. Please enter a wrong answer.

Operation porticoal anastomosis
partial liver resection
Electrodothermocoagulation of the liver surface
arterialization blood outflow

86. What kind of surgery is used to treat hypersplenism.

splenectomy
splenorenal anastomosis
splenic artery bypass surgery
splenic artery embolization

87. Types of anastomoses in portal hypertension. Please enter a wrong answer.

Splenokalny anastomosis
Portal anastomosis
splenorenal anastomosis
Mesentericokal anastomosis

88. When is a splenectomy done for portal hypertension?

with hypersplenism
in all splenomegaly
bleeding from the esophagus
with atrophic cirrhosis of the liver

89. What does not apply for profuse bleeding of the esophagus

splenectomy
operation Tanner
sclerotherapy of esophageal veins
embolization of esophageal varicose veins

90. It is used for diuretic-resistant resistant ascites. Please enter the wrong answer.

porticoal anastamosis
lymphovenous anastamosis
peritoneo venous anastomosis
Reinfusion of ascitic fluid

91. Complications of porticoal shunting in portal hypertension.

encephalopathy
digestive tract ulcers
liver failure
hemolytic anemia

92. Echinococcus is it?

Parasitic disease
infection
allergic disease
endocrine disease

93. What causes echinococcosis?

helments
microbes
fungal infections
Amoeba

94. Which organ is most often affected by echinococcosis?

liver
kidneys
Spleen
Lungs

95. Early signs of liver echonococcosis

pain in the right hypochondrium
increased body temperature

bleeding from the digestive tract
Yellowness

96. What changes occur in blood echinococcosis?

eosinophilia
leukocytosis
Lymphopenia
thrombocytopenia

97. Signs of echinococcosis when examining the abdomen.

palpable volume formation at its marginal location
flatulence
no answer
tension of the anterior abdominal muscle

98. The most reliable way to diagnose echinococcosis

Ultrasound
Laparoscopy
Fluoroscopy
FGDS

99. With a radioisotope scan of the liver, signs of echinococcosis are ...

Determination of a cold focus in the liver
organ hypertrophy
Hot focus of the liver
violation of the excretory function of the liver

100. What is observed when the echinococcus ruptures in the abdominal cavity.

anaphylactic shock
yellowness of the skin
increase in blood pressure
vomit from coffee grounds

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1

14. GLOSSARY

AND

Atherosclerosis - accumulation of lipids under intimal layer of the artery wall **ANEURYSM** - extension wall and p ter and
ATROPHY - a decrease in the volume of an organ and tissue
AEROCALY - to the exit in a large amount of air with feces
ABTSESS- limited accumulation of pus
A C CIT - fluid accumulation in the abdominal cavity
Anorexia - and loss of appetite
COARTATION OF THE AORTA - congenital segmental narrowing of the aorta
B
BRONHOEKTAZ- extension wall b ronh s
IN
VAZORENAL HYPERTENSION - vaso-vessel, renal kidney
D
HYPOKINESIA - lack of mobility
HYPERTROPHY - an increase in organs and tissues in volume
HEMATOMA - accumulation of blood in some kind of tissues and organs
HYPERTENSION - pressure increase
HYPEREMIA - redness of the skin and mucous membranes
HYPOALBUMINEMIA - a decrease in albumin in plasma
HYPERSPLENISM SYNDROME - increased spleen function - anemia, leukopenia, thrombocytopenia
HYPOVOLEMIA - about hydration about organism and
D
Dysphagia - dis - violation, phagos - swallowing
DIVERTICULE - o bounded expansion of the walls of hollow organs
DISHYDROS - violation of water metabolism
DOLOR- pain
AND
ISCHEMIA - decreased blood supply
INTRACRANIAL - inside the skull
TO
COMPRESSION - with pressure
KARDIOSPAZM- from the absence of an extension Kardial nogo sphincter and esophagus
L
LUTENBACHER SYNDROME A - b Ventricular septal defect and congenital mitral stenosis
M
METEORISM - accumulation of air in the intestines
H
NEPHROPTOSIS - pubescence of the kidneys

NISTAGM - movement of the zirochka in the horizontal and vertical direction
ABOUT
OBLITERATION - blockage of the lumen of blood vessels as a result of thickening of the intima
P
PORTAL HYPERTENSION - increased pressure of the portal vein
PARESTHESIA - numbness, tingling
PROKSIMAL te a ENTEROPATIYA- violation of motor Noah , secretor Noah and obsorbtsion nye function and small intestine
R
RELAXATION - thinning of the diaphragm and its elevated position as a result of disturbance of innervation
RUBOR - redness
FROM
SPLENOMEGALIA- enlargement of the spleen in volume
COLOR- local temperature rise
SMEAL MEAL SINDROME meal in small portions
STILL SINDROME- " robbed and e " syndrome of blood
T
TE RMINAL KOLOPATIYA- second violation evakuotornye function of the terminal part.
THROMBOZ- kon tomir bushliғida kon ivindilaring khosil bўlishi bilan kechadigan pathologik zharayon
TUMOR- edema
E
EXTRACRANIAL - outside the cranial
ENDOVAZAL - inside the vessel
EXTRAVAZAL - outside the vessel

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