Оглавление

JOINT AND SPINE INJURIES

1. ARTHRITIS SYNDROME

Definition.

Arthritis is inflammation of the joint.

The reasons.

Arthritis is a characteristic syndrome of rheumatoid arthritis, rheumatism, ankylosing spondylitis, infectious arthritis, reactive arthritis (postenterocolitis and urogenital), Reiter's disease, Crohn's disease, microcrystalline arthritis (gout, chondrocalcinosis), diffuse connective tissue diseases (systemic lupus erythematosus, systemic scleroderma), some blood diseases (leukemia, multiple myeloma), endocrine pathology (diabetes mellitus, hypothyroidism, etc.).

Arthritis can be caused by immune disorders, infectious agents (bacteria, mycoplasmas, viruses), deposition of endogenous substances in the joint tissue (urate, calcium crystals). A certain role in the development of some forms of arthritis has a genetic factor.

Under the influence of the above reasons, the pathological process develops mainly in the synovial membrane.

The early stage of inflammation is characterized by mucoid swelling of the synovial tissue, sweating of fibrin. Then there is the growth of loose granulation tissue (pannus), creeping onto the cartilage. The cartilage is destroyed, the articular surfaces are covered with fibrous tissue, the joint space narrows.

Symptomatology.

Local complaints.

Joint pain, most intense during second half of the night and morning. During the day the pain becomes weaker. Morning stiffness of movements in the joints, swelling of the joints, limitation of their mobility.

General complaints.

Increased body temperature, chills.

Examination. Swelling of joints, periarticular tissues, color
skin over the joints is normal or there is hyperemia, muscle atrophy
muscles, rheumatoid nodules near the affected joints, tophi in the area of the auricles, near the joints, skin rashes, trophic changes in the nails

Palpation.

Pain at rest and on motion, fluctuation in the joints, soreness of the periarticular tissues, increased skin temperature over the joint, muscle atrophy, rheumatoid, arthritic (tophi) nodes in the subcutaneous tissue, tendons, occipital aponeurosis, decreased muscle strength. The increase in the circumference of the inflamed joint when measuring it with a centimeter tape. Increased articular Richie index.

Study of the function of the joints.

Limitation of active and passive mobility of the joints, up to complete immobility of the joints of the joint (ankylosis). Changes to the Lee functional test.

X-ray examination of the joints.

There are no changes nn reactive arthritis (rheumatic, postenterocolitic and urogenital. In rheumatoid arthritis and other chronic arthritis, osteoporosis and cystic

enlightenment in the area of the epiphyses of bones, the presence of erosion of the articular surfaces, narrowing of the joint space, and fusion of the articular surfaces (ankylosis) are observed.
Radioisotope study with labeled pyrophosphate or technetium: greater incorporation of the isotope into tissues in the presence of active inflammation.
Thermal imaging.
Foci of hyperthermia are revealed in the places of inflammation on the thermograms of the joints,
Arthroscopy - a picture of synovitis.
Biopsy of the synovium. Villus hyperplasia, lymphoid infiltration, fibrin deposits, foci of necrosis.
The study of synovial fluid. Cloudiness, reduction viscosity, leukocytosis and neutrophilia, an increase of more than twice the protein, the presence of ragocytes (neutrophils with the inclusion of immunoglobulins), rheumatoid factor (Ig M), LE (Lupus eritematoidus) - cells, decreased complement levels. LE cells are mature neutrophils, in the cytoplasm of which hematoxylin bodies (nucleoproteins) are found.
Laboratory research. In the general analysis of blood - leukocytosis, a shift to the left, an increase in Erythrocyte sedimentation reaction
Biochemical data - dysproteinemia, increased levels of α1
and α2 - fractions of γ-globulins, fibrinogen, sialic acids, indicators of the diphenylamine test, a significant increase in the content of glycosaminoglycans in the blood serum and an increase in their excretion in the urine. Immunological study - an increase in the titer of anti-streptococcal antibodies, immunoglobulins, CEC, titer of rheumatoid factor, the number of LE-cells, detection of histocompatibility antigens of the HLA system.

2. SYNDROME OF SPONDYLOARTHRITIS

Definition:
Spondyloarthritis is a chronic inflammatory disease of the spine.
Causes:
Spondyloarthritis can be an independent disease - ankylosing spondylitis (Bekhterev's disease).
As a syndrome occurs in psoriatic arthritis
Reiter, ulcerative colitis, Crohn's disease, Whipple's disease, juvenile chronic arthritis, reactive arthritis (yersinia, shigellosis, salmonella), Behcet's disease, acute anterior uveitis, tuberculosis, brucellosis.

Reiter's disease (synonym: urethrooculosynovial syndrome) associated with exposure to infectious factors (chlamydia) on a macroorganism genetically predisposed to a disease.
Crohn's disease - granulomatous, regional or terminal ileitis - is a chronic inflammatory disease that affects any part of the gastrointestinal tract, but more often the terminal part of the small intestine.

Whipple's disease is characterized by diarrhea, steatorrhea, falling
body weight, fever, polyserositis. In the biopsy of the small intestine wall and lymph nodes, the remains of phagocytosed bacteria in macrophages are found.

Behcet's disease is characterized by a clinical triad - recurrent aphthous stomatitis, necrotic-ulcerative
changes in the mucous membrane of the genital organs and inflammatory lesions of the eyes.

Genetic factor, the presence of intestinal and genitourinary infections (yersinia, shigella, salmonella, campylobacter, klebsiella, mycoplasma, ureaplasma, chlamydia). Joints are affected "cartilaginous" type - intervertebral, costovertebral. Features of the inflammatory process - infiltration by lymphocytes and macrophages, indicating an immune nature inflammation, rapid development of fibrous scar tissue with a tendency to calcification and ossification. The ligamentous capsular apparatus, intervertebral discs are affected.

Symptomatology
Complaints: Persistent pain in the spine, especially in the second half of the night and morning hours, decreasing after the start of active movements, physical activity. The pains are combined with morning stiffness in the spine and tension of the rectus muscles of the back. Girdle pains in the chest, aggravated by deep breathing, sneezing, restriction spinal mobility.
Examination. Change in posture - cervical hyperlordosis, increase
thoracic kyphosis, smoothness of the lumbar lordosis ("pose of the petitioner"), smoothness of all physiological curves is possible
spine (straight plank-shaped back). Atrophy of the straight lines
back muscles.
Palpation. Pain on palpation, tapping of the spinous processes of the spine, atrophy of the rectus muscles of the back.
With lateral compression of the chest, sharp pains occur;

to quantify the degree of restriction of mobility of the spine, a number of standard techniques are used: cervical region - symptoms of the ear-shoulder, chin-chest, nape (Forestier symptom), head turns to the sides; thoracic region - chest excursion is reduced, Ott's symptom is positive, torso tilts forward and backward are limited,
to the sides; lumbar region - positive Schober's sign. Thomayer's symptom (fingers-floor) reveals rigidity in the thoracic and lumbar spine.

Radiography of the spine. Anterior spondylitis - destructive changes in the upper and lower sections of the anterior surface of the vertebrae (square, "planed" vertebrae, ossification of the ligaments of the spine, bone bridges (syndesmophytes) between the vertebrae (symptom of "bamboo stick"). Intervertebral joints: destruction of the bone tissue of the articular processes vertebrae, bone ankylosis.

Bone scintigraphy with technetium pyrophosphate. Increased accumulation of the isotope in the affected parts of the spine.

Laboratory research. The general blood test shows increased ESR, leukocytosis with a shift to the left; increased levels of diphenylamine test, sialic acids, α1 α2, γ-fraction of globulins, increased levels of immunoglobulins, Circulating Immune Complex (CIC), HLA-B-27 histocompatibility antigen is determined in the blood

3. SACROILITIS SYNDROME

Definition. Sacroiliitis - inflammation of the sacroiliac
joints.

Causes: Sacroiliitis occurs in Bechterew's disease (ankylosing spondylitis), psoriatic arthritis, Reiter’s disease, rheumatoid arthritis, tuberculosis, brucellosis, some enteropathies

Genetic predisposition, genitourinary and intestinal infection (chlamydia, ureaplasma, mycoplasma, Klebsiella, Campylobacter, Yersinia, Shigella, Salmonella). The sacroiliac joint is a "cartilaginous" joint. Developing progressive destruction of articular cartilage with ankylosing of ileosacral joints

Symptomatology.

Complaints. Pain in the lumbar and sacral regions, buttocks with irradiation to the thigh (lumbalgia, sciatica), increasing when moving.

Examination reveals no pathognomonic signs.

Palpation. Pain in the sacroiliac region
joints. The symptoms of Kushelevsky 1, 2, 3 and tripod allow to reveal pain in the ileosacral joints.

X-ray examination. Osteoporosis, subchondral osteosclerosis along the joint space, then narrowing, blurring
joint space up to ankylosis, usuration of the articular surfaces of the ileosacral joints.

Bone scintigraphy with technetium pyrophosphate. Unilateral or bilateral increased radiotracer accumulation in the ileosacral joints.
Laboratory research. In the general analysis of blood - an increase in Erythrocyte sedimentation rate, leukocytosis with a shift to the left; increase in the level of sialic acids, the level of diphenylamine test, α1, α2, γ-fractions
globulins, increases the level of immunoglobulins, CIC, in blood is determined by the histocompatibility antigen HLA - B27.

4. OSTEOARTHROSIS SYNDROME

Definition. Osteoarthritis (OA) is a degenerative disease
joints, which is based on the primary degeneration of the articular cartilage with subsequent changes in the bone articular surfaces, the development of marginal osteophytes, which leads to deformity of the joints.
Causes: Identify primary and secondary OA. By prevalence OA is distinguished between mono-, oligo- or polyarticular OA.

Primary OA develops on healthy cartilage under the influence of excessive mechanical and functional load (professional, household, sports, overweight).

In secondary OA, degeneration affects already preliminarily altered articular cartilage under the influence of normal physical activity. Changes in the structure and trophism of cartilage are caused by: dysplasia and disturbance of statics; joint injury - intra-articular fracture, dislocation, contusion; arthritis, hemarthrosis hemophilia; osteodystrophy; metabolic disorders - gout, hemochromatosis, chondrocalcinosis; endocrine disorders - diabetes mellitus, acromegaly, diseases of the gonads; on the investigative factor

Under the influence of the above reasons, there is a rapid and early "aging" of cartilage. There is a loss from the main substances of proteoglycans, death of chondrocytes with loss of elasticity, elasticity of cartilage, cartilage tissue disintegration, its ulceration with exposure of the underlying bone. Thereby subchondral osteosclerosis develops with compensatory marginal growths of cartilage and the formation of osteophytes.
Reactive (secondary) synovitis may occur.

Symptomatology
Complaints. Pain of a mechanical nature in the affected joint, appearing or aggravated when walking, subsiding at rest. Renewed pain at the beginning of the movement (starting). Most intense pain in the evening. Possible sudden sharp pain

and complete impossibility of movement in the joint (blockade of the joint), due to the infringement of the “articular mouse” (a piece of cartilage) between articular surfaces. Intermittent swelling joint, limitation of movement, morning stiffness of the joints less than 30 minutes.

Examination. Joint deformity, Heberden's nodes, Bouchard's nodes in areas of distal and proximal interphalangeal joints of hands; swelling of periarticular tissues, muscle atrophy in areas of the affected joint.

Palpation. Soreness, crepitus, increase in the volume of the affected joint.

Study of the function of the joints. Restriction of movements - active and passive.

X-ray examinations of the joints. Articular narrowing gaps, osteophytes, cysts in the epiphyses, subchondral osteosclerosis, “articular mice” (pieces of cartilage) in the joint cavity.

Thermal imaging. On the thermograms of the affected joint, in the presence of synovitis, clearly defined foci of hyperthermia are fixed.

Arthroscopy. A picture of a degenerative cartilage lesion.

Biopsy of the synovium. Villous atrophy, small number of vessels, fields of fibrosis or fatty degeneration.

Joint scintigraphy. On the scintigram, in the presence of synovitis, the focal intensity of isotope accumulation increases.

Synovial fluid. Normal or slightly enlarged cytosis, predominance of lymphocytes, fragments of cartilage, high viscosity.

Laboratory research. Slight increase in Erythrocyte sedimentation reaction in the general blood test (20-25 mm / h), the appearance of C-reactive protein, an increase in the amount of glycosaminoglycans in the blood serum and an increase in their excretion in the urine

SYNDROMES OF THE BRONCHOPULMONARY SYSTEM DEFEAT

1. LUNG TISSUE COMPACTION SYNDROME

Definition. Compaction of the lung tissue - a symptom complex, uniting many diseases, which are characterized by compaction and a decrease in the airiness of the lung tissue. According to the size, there can be focal and lobar compaction.

The reasons.

1. Inflammatory genesis: pneumonia, heart attack-pneumonia (thromboembolism of large or small branches of the pulmonary artery), tuberculosis.

2. Non-inflammatory genesis: cirrhosis of the lung (pneumocirrhosis or carnification) - proliferation of connective tissue in the lung as the outcome of a serious illness; compression atelectasis, tumor.

The clinical picture depends on the cause that caused the compaction of the lung tissue.

Symptomatology of inflammatory compaction of the lung fabrics.

Complaints: Cough (at the beginning, usually dry, then with wet yellow-gray, gray-green, rusty). Dyspnea of inspiratory or mixed nature. Chest pain associated with respiration (when the pleura is involved in the process), increases on inspiration.

Nonspecific complaints: fever, chills, general weakness, decreased performance, decreased or loss of appetite, headache, etc.

Examination: Cyanosis (diffuse), often a blush of the cheeks at elevated temperature, an increase in the number of breaths (inspiratory

shortness of breath), lag of the affected half in the act of breathing, sometimes the patient lies on the affected side. Excursion chest cells are reduced.

Palpation: Increased voice trembling over the affected lobe.

Percussion: Dull or blunted percussion sound. Limitation of the mobility of the lower edge of the lung on inspiration.

Auscultation: Decreased vesicular or bronchial breathing, sonorous fine bubbling rales or crepitus; bronchophony is increased.

Chest x-ray: Uniform (homogeneous) or inhomogeneous (inhomogeneous) shading of irregular shape without clear lines.

Function of external respiration: Restrictive type of violation ventilation - a decrease in VCL, IRV, an increased RR, MVB

General analysis of sputum: Mucopurulent, purulent, "rusty"; in a large number of leukocytes, desquamated epithelium, can be erythrocytes, fibrin, bacteria are determined.

Complete blood count: Leukocytosis with neutrophilic shift to the left, an increase in ESR; with viral damage - leukopenia.

There may be the appearance of CRB, an increase in sialic acids, fibrinogen; dysproteinemia.

Symptomatology of non-inflammatory compaction of the lung fabrics.

Complaints: There may be a cough with sputum, shortness of breath.

Examination: Sometimes some retraction of the chest on the side lesions, the lag of the affected half in the act of breathing.

Excursion of the chest is reduced.

Percussion: Dullness of percussion sound. Limitation of the mobility of the lower lung border on inspiration.

Auscultation: Weakened vesicular breathing.

Chest x-ray: Focal shading, enhanced bronchovascular pattern.

Function of external respiration: Restrictive type of violation lung ventilation - reduced VCL, IRV.

Complete blood count: No changes, erythrocytes may be observed due to hypoxia.

2. BRONCHITIS SYNDROME

Definition: Bronchitis syndrome - acute diffuse inflammation of the bronchial mucosa.

The reasons:

1. inflammatory lesions of the bronchi of bacterial and viral etiology (bronchitis, bronchiectasis, bronchopneumonia, pulmonary tuberculosis);

2. toxic damage to the bronchi (tobacco smoke, industrial dust and irritating gases); smoker's bronchitis, toxic bronchitis;

3. heart failure as a result of stagnation of blood in the lungs ("congestive" bronchitis);

4. endogenous intoxication ("uremic" bronchitis);

5. tumor lesions of the bronchus (benign and malignant tumors of the bronchus);

6. allergic lesion of the bronchi ("allergic" bronchitis).

The main causes of the development of bronchitis syndrome: in the development of bronchitis syndrome, an important role is played by a decrease of the effectiveness of physical protective factors, primarily the ability of the upper respiratory tract to filter the inhaled air and release it from coarse mechanical particles and change in bronchial secretions.

The epithelium of the bronchi is affected, disrupting metabolic processes in cells, leading to their death.

Symptoms of bronchitis syndrome:

Complaints: cough (initially dry, then with mucous or mucopurulent sputum), shortness of breath. General symptoms of intoxication as a manifestation of an infectious process or purulent infection.

Examination: little diagnostic information.

Palpation: voice trembling, as a rule, does not change;

Percussion: usually percussion sound over the lung fields is not changing.

Auscultation: no change or hard breathing with significant bronchial compaction, dry, less often inaudible wet, wheezing Bronchophony is not changed.

X-ray of the lungs: changes are not revealed.

Complete blood count: moderate neutrophilic leukocytosis, slight increase in ESR.

Sputum: mucous or mucopurulent character, cylindrical epithelium, leukocytes.

Bronchoscopy with difficulty in diagnosis, for therapeutic purposes

3. SYNDROME OF BRONCHIAL OBSTRUCTION

The syndrome of bronchial obstruction (or broncho-obstructive syndrome) is divided into three variants: reversible, slightly reversible and progressive.

Definition. Bronchial obstruction syndrome is a clinical symptom complex, including expiratory dyspnea, suffocation, cough, which is based on a generalized violation of bronchial patency.

Development mechanisms:

1) bronchospasm;

2) swelling of the bronchial mucosa with narrowing of the lumen;

3) hyperproduction of bronchial mucus;

4) thickening of the bronchial wall due to inflammation and development fibrosis (loss of elasticity).

The main causes of the development of the syndrome: household (home dust, pets, insects), plant pollen, spores fungi, flour, dyes, foodstuffs, medicines, occupational hazards, microbes, their metabolic products, etc.

Stages of development:

1 - immunological reaction of AG with specific AT (IgE), fixation of this complex on mast,

plasma cells;

2- pathochemical - histamine is released, serotonin, kinins are released from these cells due to alteration, degranulation

3 art. - pathophysiological - swelling of the bronchial mucosa, their exudation, blockage of the bronchi, bronchospasm, bronchial sclerosis.

Complaints: expiratory shortness of breath (difficulty exhaling), unproductive, painful cough (sputum does not go away).

Examination: the forced position of the patient (the patient sits, leaning his hands on his knees, the shoulder girdle is raised), his mouth open, nostrils flaring, swollen neck veins, breathing loud, pained expression. diffuse cyanosis.

The chest is emphysematous (barrel-shaped), in the act of breathing accessory muscles are actively involved. Limited chest excursion

Palpation: the chest is rigid, voice trembling is weakened.

Percussion: boxed percussion sound, increased pitch standing apices of the lungs, the lower borders of the lungs are lowered down, reduced mobility of the lower pulmonary edge on exhalation. Disappearance of absolute dullness of the heart.

Auscultation: breathing is sharply weakened, a lot of dry wheezing and buzzing rales in all lung fields. A sharp weakening of the heart sounds, tachycardia.

Chest X-ray: increased transparency of lung fields, limitation of diaphragm mobility.

Function of external respiration: decrease in FVC, VC, power expiration, expiratory FEV in 1 sec, Tiffno, Gansler indices.

Complete blood count: eosinophilia.

Sputum: light, glassy, viscous; Kurshman spirals, Charcot-Leyden crystals, many eosinophils.

Immunological studies: increased IgE, the presence of antibodies to various allergens.

4. EMPHYSEMA SYNDROME

Definition: Emphysema is an anatomical alteration lungs, characterized by a pathological expansion of the air spaces located distally to the terminal bronchioles, and accompanied by destructive changes walls of the alveoli.

The reasons:

a) primary emphysema;

b) secondary obstructive pulmonary emphysema in patients with chronic obstructive bronchitis, bronchial asthma;

c) compensatory vicarious emphysema of a healthy lung (with removal of the lung or part of it, inflammatory or other nature of its lesions);

d) senile involutive emphysema;

e) acute emphysema (with aspiration of a foreign body, drowning, acute attack of bronchial asthma)

The reasons for the development of primary emphysema remain unclear; much attention is paid to the role of genetic factors (deficiency of α1-antitrypsin), impaired mucopolysaccharide metabolism, the presence of a genetic defect in elastin, birth defects structural glycoproteins.

Forms of secondary emphysema, occurring with destruction of the respiratory section of the lungs, occur as a result of irreversible changes caused by other diseases of the bronchopulmonary system. The main mechanism for the development of emphysema is bronchial obstruction associated with bronchospasm, swelling of the mucosa, blockage of the gaps with viscous mucus, which causes a phenomenon called "air trap". On exhalation, intrathoracic pressure rises, in the alveoli there is hypertension, which leads to overstretching and then destruction of the alveoli.

Symptomatology of emphysema

Complaints: expiratory dyspnea (difficulty exhaling), decreased exercise tolerance.

Physical examination: Diffuse cyanosis, emphysematous (barrel-shaped) chest, expiratory dyspnea. Excursion chest is limited. Swelling of the neck veins. Nails in the form of watch glasses, fingers in the form of "drumsticks".

Palpation: Voice trembling is weakened. Rigid chest cell.

Percussion: Boxed percussion sound. Lower bounds the lungs are lowered down, the mobility of the lower pulmonary edge is limited; the height of standing of the tops of the lungs is increased, the fields of Krenig are increased. Reducing the boundaries of cardiac dullness.

Auscultation: Weakened vesicular breathing, weakened bronchophony. Decreased heart sounds.

Chest X-ray. Increase in total area and transparency of the lung fields, weakening of the pulmonary vascular pattern, the diaphragm is low, little mobile. Heart becomes on a teardrop-shaped and looks reduced. The function of external respiration. Decreased VC, MEVR (maximum expiratory volume rate), PVEFR (peak volume expiratory flow rate), expiratory pneumotachometry, Tiffno indices, Gansler, FVC, FEV1, increase in general and functional residual lung capacity (FRLC)1, residual air.

Complete blood count: erythrocytosis

ECG: signs of hypertrophy of the right ventricle and right atrium.

5. SYNDROME OF ACCUMULATION OF AIR IN THE PLEURAL CAVITY (PNEUMOTHORAX)

Definition. This is a condition in which air accumulates between the parietal and visceral layers pleura when the pleural cavity communicates with the bronchi (closed pneumothorax) or with the environment (open pneumothorax).

The reasons.

1. Subpleural tuberculous cavities, abscesses, bullae (spontaneous pneumothorax).

2. Wounds of the chest (traumatic pneumothorax).

3. The introduction of air into the pleural cavity for therapeutic purposes (artificial pneumothorax).

Symptomatology.

Complaints - intense stabbing pain in the corresponding half of the chest, severe inspiratory dyspnea, often dry paroxysmal cough.

Examination - asymmetry of the chest due to an increase in the affected half, lagging behind in the act of breathing.

Palpation - weakening of voice trembling over the area accumulation of air up to its complete absence.

Percussion - tympanic percussion sound over the affected half of the chest.

Auscultation - breathing over the affected half is sharply weakened or not carried out, there is no bronchophony.

Chest x-ray - clearing is detected without pulmonary pattern in the area of pneumothorax, closer to the root - the shadow of a collapsed lung; shift of the median shadow to the healthy side

6. SYNDROME ACCUMULATION OF FLUID IN THE PLEURAL CAVITY

Definition. This is a condition in which there is an accumulation of inflammatory (exudate) and non-inflammatory (transudate) fluid between the parietal and visceral pleura.

The reasons.

1. Exudative pleurisy of infectious etiology (tuberculosis, complications of pneumonia, abscess and gangrene of the lung).

2. Aseptic pleurisy associated with trauma and intrapleural hemorrhages.

3. Damage to the pleura by a malignant neoplasm (carcinomatosis).

4. Pleurisy in systemic connective tissue diseases (rheumatism, collagenosis).

5. Accumulation of non-inflammatory (transudate) fluid in the pleura in case of heart failure, kidney and liver diseases. With all these processes, fluid accumulates between the layers of the pleura, compression of the area lung on the side of the lesion, which leads to the development of respiratory failure.

Symptomatology.

Complaints: feeling of heaviness, overflow in the affected side chest, dry cough, with significant accumulation fluid appears shortness of breath.

Examination: cyanosis, asymmetry of the chest due to bulging intercostal space in the area of exudate accumulation, lagging of the affected half of the chest in the act of breathing.

Palpation: voice trembling in the area of fluid accumulation not carried out.

Percussion: over the area where the liquid is located – dull percussion sound with an oblique upper border (Damuazo lines). On the painful side between the spine and the line of Damuazo there is a triangle of obtuse-tympanic sound (Garland's triangle) - corresponds to a compressed lung.

The second triangle (Rauhfus-Grocko) is located on the healthy side (legs - diaphragm and spine, hypotenuse - Damuazo line) and is, as it were, a continuation

of the dullness determined on the affected side. It is formed by the mediastinum displaced to the healthy side. There is no mobility of the lower pulmonary edge on the side of the lesion

Auscultation: breathing in the area of fluid accumulation is not felt or is sharply weakened. Above the border of the exudate of the respiratory bronchial tract, which is compression of the lung and the flow of air from it (compression atelectasis). Bronchophony over the solution is not observed.

Chest x-ray: homogeneous shading with oblique located upper border and mediastinal displacement in healthy side.

Instrumental diagnostics. Pleural puncture followed by examination of the effusion. At the points of selection, the amount in the protein, the relative density (for exudate character) relative density more than 1018 and the amount of protein more than 3%). The Rivalta test (a drop of dots in a weak solution of acetic acid with an inflammatory nature) has a certain value for judging the nature of the pleural fluid effusion gives "cloudy precipitation of serolysin"). Sediment items contain cytologically to determine the etiology of pleurisy.

Thoracoscopy with biopsy of the pleura to determine the etiology pleurisy.

7. SYNDROME OF FIBRINOUS PLEURITIS

Definition. This is a pathological condition associated with the deposition of fibrin on the pleura due to various diseases.

The reasons:

1. Complication of pneumonia, lung abscess.

2. Carcinomatosis of the pleura.

3. Pleurisy in systemic connective tissue diseases.

4. Tuberculous pleurisy.

5. Pleurisy in chronic renal failure.

Symptomatology.

Complaints - acute pain in the chest during breathing, aggravated with a deep breath and tilt in the opposite direction.

Cough is usually dry, general malaise, subfebrile temperature.

Examination - breathing is rapid, superficial, sometimes noted forced position on the affected side

Palpation - no changes, there may be a weakening of the voice trembling with massive pleural overlays.

Percussion - restriction of mobility of the lower pulmonary edge on the affected side.

Auscultation. On the side of the lesion, a pleural friction rub is heard; with massive pleural overlays - weakening of vesicular respiration.

Chest X-ray - high standing of the dome of the diaphragm, limited mobility of the lower lung edge, slight clouding of the affected part of the lung edge.

Complete blood count: sometimes there is a moderate leukocytosis.

Thoracoscopy with biopsy of the pleura to determine the etiology pleurisy.

8. FIBROTHORAX SYNDROME

Definition. Obliteration of the pleural cavity with a massive layer of fibrinous tissue.

Causes: This syndrome is observed as an outcome of exudative tuberculous pleurisy, pleural empyema, after operations on lung, traumatic injuries of the chest (after hemothorax). There is an organization of fibrinous masses on the surface of the pleura, the formation of fibrous tissue, which eventually undergoes calcification and even ossification.

The lung is surrounded by a fibrous "shell", which sharply limits its ventilation. Sometimes fibrosis from the pleura passes to the lung tissue (the so-called pleurogenic cirrhosis of the lung).

Complaints: shortness of breath, sometimes "dull" pain on the side of the lesion.

Examination: a decrease in the volume of the chest, the shoulder is lowered by affected side, decreased chest excursion.

Palpation: weakening of voice trembling.

Percussion: dullness throughout the affected lung. The mobility of the lower pulmonary edge is sharply limited. On the affected side, the protrusion of the apexes decreases, and the lower border of the lung rises.

Auscultation: weakening of vesicular breathing, a rough pleural rub may be heard.

Chest x-ray: shading of one lung fields, high standing of the diaphragm; fusion of the costophrenic sinus, mediastinal displacement towards the lesion.

Function of external respiration: restrictive type of violation ventilation. Decreased VCL, IRV, increased frequency of respiratory rate. Indicators the state of

bronchial patency is not disturbed (in the absence of concomitant damage to the bronchi).

9. LUNG CAVITY SYNDROME

Definition. Cavity in the lung - the formation of a cavity in the lung tissue as a result of its necrosis, destruction, purulent fusion

The reasons.

1) lung abscess after opening,

2) tuberculous cavity,

3) tumor decay,

4) Wegener's granulomatosis (hyperergic systemic vasculitis, manifested by the development of necrotizing granulomas),

5) syphilitic gum,

6) fungal infection of the lungs (aspergillosis),

7) lung cysts (polycystic).

The main reasons for the development of a cavity in the lung: the collapse of the lung tissues as a result of exposure to infectious pathogens, tumors, autoimmune disorders.

Symptomatology of the cavity in the lung.

Complaints depend on the underlying disease.

Examination: lag in the act of breathing of the “sick” half of the chest. Restriction of chest excursion.

Palpation: increased voice trembling (the cavity should be large, located no deeper than 2 cm from the surface chest, should communicate with a large bronchus, which we pass).

Percussion: blunt-tympanic or tympanic percussion sound, sometimes with a metallic tint.

Auscultation: bronchial, less often amphoric breathing, resonant coarse and medium bubbling wet rales. Bronchophony

reinforced.

Chest x-ray: the cavity is usually round in shape with a fairly even internal contour, a horizontal level of fluid (with a lung abscess after opening).

Function of external respiration: restrictive type of violation ventilation function of the lungs (decreased VCL and MVL).

Complete blood count: leukocytosis, stab shift to the left, increase in ESR.

Sputum analysis: two-layer purulent, elastic fibers, erythrocytes, leukocytes, microflora, in case of tuberculosis - mycobacterium tuberculosis (BC+), in case of a tumor - atypical cells.

10 ATELEKTASIS SYNDROME

Definition. Atelectasis is the collapse of the lung as a result of blockage of a large bronchus, followed by the gradual resorption of air from the lung below the closure of the lumen. Atelectasis is not primary disease, its cause must be established.

The reasons:

1) tumor of the bronchus,

2) pneumonia,

3) bronchiectasis,

4) cystic fibrosis,

5) foreign body,

6) fungal diseases of the lungs,

7) primary bronchopulmonary amyloidosis,

8) compression of the bronchus by enlarged lymph nodes (tuberculosis, Hodgkin's disease, leukemia).

The cause of atelectasis development is is a blockage of the bronchus, which leads to to impaired ventilation in the lung below the site of blockage, the air is gradually absorbed, the lung tissue becomes dense, and gas exchange is disturbed.

Symptomatology atelectasis.

Complaints depend on the underlying disease.

Local complaints: shortness of breath.

Examination: diffuse cyanosis, shortness of breath, the affected side lags behind in the act of breathing. Excursion of the chest is limited.

Reduction in the volume of one half of the chest (retraction, lowered shoulder).

Palpation: the chest is rigid on the side of the lesion, the voice trembling is weakened.

Percussion: dull or dull percussion sound over the lesion, limitation of lower lung mobility edges, displacement of the lower lung cavity upwards (with complete obstruction of the lower lobe oronch) with lower lobar atelectasis.

Omission of the upper border of the lung with upper lobar atelectasis.

Auscultation: vesicular breathing is sharply weakened, possible crepitus, bronchophony is weakened.

Chest x-ray: homogeneous segment shading lobe or the whole lung, the diaphragm is pulled up, the mediastinum, the trachea are displaced towards shading.

11. ALVEOLITIS SYNDROME

Definition. Alveolitis is diffuse, usually bilateral inflammation of the lung tissue allergic, autoimmune or toxic nature without damage to the bronchial tree, accompanied by the development of respiratory failure.

Causes: There are 3 types of alveolitis: exogenous allergic alveolitis (EAA), toxic alveolitis, idiopathic fibrosing alveolitis (IFA).

The main causes of the development of alveolitis. EAA is based on allergic inflammation of the alveolar and interstitial lung tissue resulting from repeated and prolonged inhalation exposure to organic dust containing actinomycetes, mold, fungal antigens, bird antigens (litter, feathers), as well as wood, flour, wool dust. It leads to the formation of a large amount of CIC (Ag +• At), deposited in the lung tissue with the development of immune inflammation. Toxic alveolitis develops in response to the action drugs (antibacterial, antitumor and other drugs), irritating gases (chlorine, ammonia, hydrogen sulfide), metal vapors, herbicides, plastic vapors. There is toxic edema and necrosis of the lung tissue. EIA - occurs as a result of the action of unknown agents (presumably viruses, bacteria, toxic factors, medicinal drugs) that cause autoimmune inflammation of the alveoli with disorganization of their structures and uncontrolled collagen formation, leading to severe, progressive pneumofibrosis. Toxic and allergic alveolitis ends in recovery. With timely termination the action of an antigen or toxic agent, recovery occurs. Otherwise, they may transform into fibrosing alveolitis.

Local complaints: shortness of breath of an inspiratory nature (character of not being able to take a deep breath). Other pulmonary complaints: chest pain, cough.

Common complaints: fever, chills, weight loss.

Examination: diffuse cyanosis, inspiratory dyspnea (shortening of inhalation and exhalation, "drum fingers" and hourly glass).

Palpation: possible increased voice trembling in the lower chest on both sides.

Percussion: shortening of percussion sound in the lower sections with both sides.

Auscultation: Initially gentle crepitus or finely bubbling 2-sided moist rales, then typical sonorous crepitant wheezing ("crack of cellophane").

Chest x-ray: 2-sided changes first in the lower sections, then diffuse. Initially, increased pulmonary pattern, many foci of infiltration, in the late stage - "cellular" lung (in the form of small enlightenments).

Function of external respiration: Restrictive type of violation ventilation: increased frequency of respiratory rate, decrease in TV, VCL, IRV. Decreased diffusion through the alveolar-capillary membrane, arterial hypoxemia. Lung biopsy: the leading diagnostic method (thickening and infiltration of the alveolar septa, cystic cavities, fibrosis is detected).

Complete blood count: leukocytosis, increased ESR, eosinophilia. Immunological study: an increase in the CEC, immunoglobulins, antipulmonary antibodies (APA). Appear antibodies to the putative antigen (EAA). With toxic alveolitis immunogram is not changed.

12. RESPIRATORY INSUFFICIENCY SYNDROME

Definition.

Respiratory failure (RF) is a condition in which the external (pulmonary) respiration system does not provide normal blood gas composition or this requires excessive stress on the respiratory system.

The main causes

1. Damage to the bronchi with impaired bronchial patency (bronchospasm, bronchorrhea). Occurs in bronchitis

bronchial asthma.

2. Damage to the lung tissue (infiltration, destruction of the lungs, inneumosclerosis, emphysema).

3. Reduction of the functioning lung parenchyma (atelectasis, removal of the lung).

4. Damage to the pleura (pleural effusion, pleural adhesions).

5. Damage to the musculoskeletal skeleton of the chest (kyphoscoliosis, limited mobility of the ribs and diaphragm).

6. Damage to the respiratory muscles (fatigue of the respiratory muscles with prolonged RF, diseases of the spinal cord, myasthenia gravis).

7. Violation of blood circulation in the vessels of the small circle (edema lung, pulmonary embolism).

8. Damage to the respiratory center (drug poisoning, sleeping pills, trauma to the skull).

Complaints: Shortness of breath with significant physical exertion, exceeding the daily (mild), with the usual daily exertion (moderate), at rest (severe CRF).

Inspection. At the beginning of the development of RF, there are no changes, a decrease in the functional tests of Stange and Gench makes it possible to suspect latent respiratory failure. In severe cases of RF, an increase in the frequency and depth of breathing, cyanosis is detected. Accessory respiratory muscles are involved in respiration. The retraction between the costal muscles during breathing is clearly visible. It can be forced position of the patient: sitting with fixation of the shoulder girdle.

Examination of the function of external respiration: Increase in minute volume of respiration (MVR), decrease in lung capacity (VCL), impaired bronchial patency: decrease in forced (FVCL), Tiffno index, maximum expiratory flow rate (EFRC). Decreased diffusion capacity of the lungs. With a restrictive (restrictive) type of ventilation disorders: Decrease in the reserve volume (RV) of inhalation, VCL. With an obstructive type of ventilation disorder, you show a violation of bronchial patency

Blood gas testing: Reduced partial pressure oxygen in arterial blood less than 70 millimeters of mercury. Increase partial pressure of carbon dioxide more than 50 millimeters of mercury.

SYNDROMES IN CARDIOVASCULAR SYSTEM DAMAGE

1. Mitral valve insufficiency syndrome

Definition. Mitral insufficiency is characterized by incomplete closure of the valves during left ventricular systole as a result of damage to the valvular apparatus.

Hemodynamics: blood regurgitation causes volume overload of the left ventricle and left atrium, resulting in hyperfunction, and then hypertrophy of the left heart. An increase in left atrial pressure leads to pulmonary hypertension and right ventricular hypertrophy.

Causes: rheumatism, atherosclerosis, infective endocarditis, trauma, systemic connective tissue diseases. With myocarditis, myocardial dystrophy, relative mitral valve insufficiency develops. Mitral valve dysfunction in CL due to damage to the

papillary muscles or focal damage to the myocardium of the left ventricle. Congenital mitral insufficiency. Mitral valve prolapse.

Symptomatology.

There are no complaints in the compensation stage. In the decompensated phase -

shortness of breath, palpitations, with the development of stagnation in the lungs - cough, attacks of cardiac asthma, hemoptysis. In far gone cases of complaints associated with right ventricular failure (swelling in the legs, pain in the right hypochondrium as a result of an enlarged liver).

Inspection: In the stage of compensation, no any changes. In case of violation of compensation of cardiac activity, "mitral" blush, acrocyanosis. "Heart hump" (if the defect is from childhood).

Palpation: Displacement of the apex beat to the left and down. The push is spilled, reinforced. Systolic trembling at the apex of the heart.

Percussion: Increased relative dullness of the heart left and up. With right ventricular hypertrophy hearts move to the right. mitral configuration of the heart

(waist flattened).

Auscultation - weakening of 1 tone at the top. In the phase of decompensation - accent and splitting of 2 tones over the pulmonary artery.

A systolic murmur is heard at the apex. In terms of timbre, it is blowing, sawing, it is conducted to the armpit. In the position on the left side in the exhalation phase, the noise increases.

Instrumental research.

ECG: signs of myocardial hypertrophy of the left atrium and left ventricle.

FKG. decrease in amplitude of the 1st tone, systolic murmur associated with the 1st tone, decreasing or constant, accent of the 2nd tone pulmonary artery.

Radiography: hypertrophy of the left atrium and left ventricle, mitral configuration, displacement of contrasted the esophagus along an arc of a large radius (more than 6 cm).

EchoCG: expansion of the cavity of the left atrium and left ventricle, a noticeable absence of systolic closure (indirect signs).

Doppler echocardiography reveals turbulent blood flow into the left atrium

2. Mitral stenosis syndrome

Mitral stenosis is a narrowing of the left venous orifice in as a result of fusion of the mitral valve leaflets, their compaction and thickening.

Causes: Rheumatism, very rarely congenital or resulting from infective endocarditis.

Hemodynamics. The "critical area" at which noticeable hemodynamic disorders - 1-1.5 cm2 (normally 4-6 cm2). An increase in pressure in the left atrium leads to an increase in pressure in the pulmonary veins, and the latter causes reflex narrowing of the arterioles of the small circle (Kitaev's reflex). The load on the right ventricle increases, which hypertrophies over time.

Symptomatology.

There are no complaints during the compensation period. In the period of decompensation cough with blood in the sputum, shortness of breath, palpitations, interruptions and pain in the heart, swelling in the legs, pain in the right hypochondrium, an increase in the abdomen.

On examination - cyanotic flush of the cheeks, acrocyanosis; in children

infantilism, "heart hump" (due to hypertrophy and dilatation of the right ventricle), pulsation in the epigastrium due to the right ventricle.

On palpation in the region of the apex (in the position on the left side

on expiration) diastolic trembling is determined - “cat purr".

On percussion, the relative dullness of the heart expands upward and to the right. The configuration of the heart is mitral.

Auscultation - flapping the 1^{st} tone at the top, a click of the mitral valve opening, the “quail” rhythm (clapping 1 tone, normal 2nd tone, mitral valve opening click), accent and bifurcation of 2nd tone on the pulmonary artery, protodiastolic (mesodiastolic) and presystolic murmurs at the apex of the heart. The "quail" rhythm, as well as the increasing presystolic and decreasing protodiastolic murmurs, are best heard on the left side during expiration.

Presystolic murmur disappears with atrial fibrillation.

The pulse is small, often on the radial artery on the left is less than on the right (due to compression of the arteries by the enlarged left atrium), arrhythmia is possible. BP tends to decrease.

Instrumental research.

ECG: left atrial hypertrophy and right atrial hypertrophy ventricle

PhCG: at the apex of the heart - a large amplitude of the 1st tone, a click

opening in 0.08-0.12 sec after the 2nd tone, lengthening the interval Q-1 tone up to 0.08-0.12 sec, decreasing protodiastolic and increasing presystolic murmurs; increase in amplitude and splitting of the 2nd tone on the pulmonary artery.

Cardiac x-ray: flattening of the waist of the heart, bulging the second and third arcs along the left contour due to the pulmonary artery and left atrium, deviation of the contrasted esophagus along a small radius arc (less than 6 cm).

Echocardiography: unidirectional movement of the anterior and posterior leaflets mitral valve (“ski” symptom), thickening of the valve leaflets, expansion of the cavity of the right ventricle and left atrium.

3. AORTIC VALVES INSUFFICIENCY

Aortic valve insufficiency is a defect in which the semilunar valves do not completely close the aortic opening and during diastole blood flows back from the aorta into the left ventricle.

Etiology: rheumatism, infective endocarditis, syphilis, diffuse connective tissue diseases, trauma, atherosclerosis.

Congenital - bicuspid valve, leaflet fenestration, ectasia (expansion) of the aorta due to atherosclerosis, arterial hypertension with the development of relative aortic insufficiency.

Hemodynamics. The reverse flow of blood into the left ventricle leads to its overflow and stretching. Increased work of the left ventricle leads to its hypertrophy and dilatation.

Symptomatology.

There may be no complaints in the compensation stage. Over time, there are dizziness, fainting, pain in the region of the heart of the type of angina pectoris, shortness of breath.

Examination: pallor of the skin, pronounced pulsation of the vessels of the neck

("dance of the carotid"), rhythmic shaking of the head (symptom Musset), capillary pulse. Facies aortalis - pronounced pallor of the face.

Palpation of the heart area - spilled, reinforced, lifting and domed, displaced in the sixth or seventh intercostal space apex beat. Diastolic trembling at the Botkin-Erb point.

Percussion - an increase in the boundaries of relative dullness to the left, aortic configuration of the heart (the waist of the heart is underlined), increased vascular dullness in the second intercostal space due to aortic expansion.

Auscultation – the 2nd tone on the aorta with rheumatic disease is weakened, with syphilitic and atherosclerotic - sonorous, sometimes enhanced. At the Botkin point and in the second intercostal space to the right of the sternum (preferably in the exhalation phase in the patient's standing or sitting position with the torso tilted forward and arms raised up - a symptom of Sirotinin-Kukoverov or lying on the back and on the right side), a soft, blowing, protodiastolic murmur is determined . When listening to large arteries (femoral, carotid), it is possible to identify a double Durozier-Vinogradov murmur and double tone Traube.

The pulse is fast and high (pulsus celer et altus). Arterial increased systolic and pulse pressure, diastolic reduced, in severe cases to 0 - the phenomenon of infinite tone.

Instrumental research.

ECG: signs of left ventricular myocardial hypertrophy.

PhCG: weakening of the 2nd tone on the aorta, immediately after the 2nd tone a decreasing protodiastolic murmur follows.

X-ray: left ventricular hypertrophy, aortic heart configuration, aortic dilatation.

Echocardiography: Valvular vegetations can be seen in infective endocarditis. Indirect signs of defect are dilatation of the left ventricle and hyperkinesis of its walls. diastolic trembling anterior leaflet of the mitral valve.

Doppler echocardiography reveals turbulent blood flow from the aorta to the left ventricle.

On the sphygmogram of the carotid artery, a rapid rise, for the pointed apex of the anacrota, and a rapid fall of the catacrot with a small dicrotic wave are determined

4. SYNDROME OF STENOZIS OF THE OSTIA OF THE AORT

Definition. Aortic stenosis is a narrowing of the aortic opening.

Causes: atherosclerosis, rheumatism, infective endocarditis, congenital aortic stenosis.

Hemodynamics: a significant obstacle to the flow of blood from of the left ventricle is observed with a decrease in the area of the aortic orifice by at least 50%. The pressure in the left ventricle increases in proportion to the narrowing of the aortic orifice. This causes the development of severe left ventricular

hypertrophy. With the weakening of its contractile function, the cavity of the left ventricle increases.

Symptomatology.

Complaints: there are no subjective manifestations in the stage of compensation.

With decompensation - dizziness, fainting, squeezing chest pain. With a decrease in contractility left ventricle (terminal stage) - shortness of breath and cardiac asthma seizures.

Inspection: pallor of the skin, visible mucous membranes; with the development of a defect in childhood - a heart hump

Palpation: systolic trembling in the second intercostal space

on the right at the sternum (especially on exhalation). Intensified apical impulse, an increase in its area, its displacement down and to the left, but not as pronounced as in aortic insufficiency.

Percussion: increase in the left border of relative dullness heart, an increase in the width of the vascular bundle (poststenotic expansion of the ascending aorta).

Auscultation. The 2^{nd} tone on the aorta is weakened, rough, loud systolic murmur with an epicenter in the second intercostal space on the right or at the Botkin point. Noise is conducted to the carotid arteries, it is best heard on exhalation in a horizontal position or in a vertical position, leaning forward slightly. Noise scraping, sawing, cutting.

The pulse is small, slow (pulsus parvus et tardus).

Systolic blood pressure decreases, diastolic blood pressure is normal or increases (arteriolar spasm). Pulse blood pressure decreases.

Instrumental research.

ECG: signs of left ventricular myocardial hypertrophy. In severe cases, blockade of the left bundle of His bundle.

PhCG: weakening of the 2^{nd} sounds on the aorta, systolic rhomboid

murmur over the aorta.

Radiography: left ventricular enlargement, aortic dilatation, aortic configuration of the heart.

Sphygmogram of the carotid artery: slowing down the rise and fall of the pulse wave (slow pulse), low amplitude pulse waves.

Echocardiography: thickening of the valves, a decrease in their divergence, an increase in the thickness of the left ventricle.

Doppler echocardiography - turbulent blood flow in systole at the aortic valve.

Definition. Tricuspid insufficiency is a defect characterized by incomplete closure of the valves of the right atrioventricular orifice.

Causes: organic insufficiency of the tricuspid valve is observed in rheumatism, infective endocarditis

Tricuspid insufficiency may be congenital (Ebstein's anomaly). Relative insufficiency of the three leaflet valves occurs with severe dilatation of the right ventricle in patients with mitral defects, pulmonary hypertension, and diffuse myocardial damage syndrome.

Hemodynamics: during right ventricular contraction, the part of the blood returns to the right atrium, which is simultaneously infused with the usual amount of blood from the vena cava. The process of defect compensation by the right atrium is very limited, therefore venous congestion develops early in the systemic circulation.

Symptomatology.

Complaints: pain in the right hypochondrium, palpitations, edema, abdominal enlargement (ascites).

Inspection: acrocyanosis, swelling and systolic pulsation of the jugular veins, ascites, edema, pulsation in the epigastrium (due to an increase in the right ventricle), systolic swelling of the liver.

Palpation: the apex beat is usually not pronounced. The cardiac impulse, epigastric pulsation is determined.

Percussion: expansion of the right border of relative dullness heart, expansion of absolute dullness.

Auscultation at the base of the xiphoid process reveals weakening of the 1^{st} tone, sonority of the 2^{nd} tone above the pulmonary trunk is weakened, systolic murmur at the base of the xiphoid process, increasing at the height of inspiration (Rivero-Corvallo symptom).

On palpation of the abdomen, an enlarged congestive liver is determined (often pulsating in systole).

Instrumental research.

ECG: signs of myocardial hypertrophy of the right ventricle, right atrium.

PhCG: at the base of the xiphoid process, a decrease in amplitude of the 1st tone, here is a pronounced constant systolic murmur,

whose amplitude increases with inspiration.

X-ray: enlargement of the right heart.

EchoCG: direct signs are absent. Indirect signs can be identified: an increase in the cavities of the right atrium and the right ventricle, an increase in the wall thickness of the right ventricle.

Doppler echocardiography: the reverse flow of blood from right ventricle to the right atrium.

6. HYPERTENSION SYNDROME

Definition. Increase in systolic pressure more than 140 mmHg and diastolic more than 90 mm Hg. called arterial hypertension.

Causes: Long-term increase in blood pressure is observed in: hypertension, kidney disease (damage to the vessels of the kidneys and parenchyma), endocrine diseases (with damage to the adrenal glands, thyroid gland, pituitary gland), hemodynamic arterial hypertension (coarctation of the aorta, aortic defects), brain damage (concussion, tumors).

Symptomatology.

Complaints: headaches, dizziness, decreased severity vision, flies before the eyes, pain and interruptions in the region of the heart, shortness of breath when walking.

Palpation: enhanced, shifted to the left, lifting top push.

Percussion: shift of cardiac dullness to the left.

Auscultation: emphasis of the 2nd tone over the aorta. The pulse becomes hard, tense.

Laboratory and instrumental research methods.

ECG: left ventricular hypertrophy, with the addition of atherosclerosis of the coronary arteries - signs of chronic coronary insufficiency

FGK: accent of the 2nd tone over the aorta. The pulse becomes hard, tense

Echocardiography reveals left ventricular hypertrophy

On x-ray, aortic heart configuration. The aorta is elongated, thickened, expanded.

Ophthalmoscopy reveals narrowing of the arteries, dilated veins, retinal hemorrhages.

7. ANGINA SYNDROME

Definition. Angina pectoris is a clinical syndrome characterized by acute paroxysmal pain in the chest, in the most typical cases behind the sternum. Pain in angina pectoris is caused by transient myocardial ischemia due to insufficiency of the coronary circulation

The reasons. The most common cause of angina pectoris is stenosing atherosclerosis of the coronary arteries or their spasm. Less common as the clinical syndrome of angina pectoris occurs with inflammatory lesions of the coronary arteries, syphilitic mesoaortitis, anomalies in the development of the coronary arteries, aortic defects (with stenosis of the aortic orifice, aortic insufficiency).

The development of angina pectoris syndrome is based on coronary insufficiency - the result of an imbalance between the myocardial oxygen demand and the possibility of its delivery with blood. With insufficient access of oxygen to the myocardium, its ischemia occurs.

Symptomatology.

Complaints: paroxysmal, pressing, squeezing burning pain

behind the breastbone or to the left of the breastbone. Pain radiates to the left

arm, under the left shoulder blade, in the left half of the neck, lower jaw. The pain lasts from several minutes to 20 minutes.

Often an attack of angina pectoris is perceived by patients not as pain, but as an inexpressible discomfort: heaviness, compression, tightness, chest pressure, heartburn. The symptom of a “clenched fist” is characteristic of angina pectoris - to describe his sensations, the patient puts a fist or palm on the sternum. Depending on the circumstances, the time at which pain occurs, there are angina pectoris and rest angina (spontaneous).

Patients with stable exertional angina (stereotypical the nature of pain attacks) are heterogeneous depending on the ability to endure physical activity.

The 1st functional class - ordinary physical activity is not causes angina pectoris, an attack occurs with a large and quickly performed load.

The 2nd functional class - a slight limitation of the physical activity. Typical pain on normal walking distance of more than 500 meters, when walking after eating, in frosty weather, cold wind, rough weather.

The 3^{d} functional class - a pronounced limitation of the physical activity. Angina pectoris occurs during normal walking at a distance of 100-200 meters, rare attacks of rest angina are possible

The 4th functional class - the inability to perform any physical work without discomfort. Rest angina often occurs. In addition to physical activity, the onset of an angina attack provokes cold (spasm of the coronary vessels), food intake, stress (an increase in the number of heartbeats and an increase in myocardial oxygen demand). Electrocardiography is the leading method of instrumental diagnostics.

At the time of an angina attack, a horizontal decrease in the ST segment by at least 1 mm, the appearance of a negative coronary T wave in one or more leads. In spontaneous angina at the time of pain, the ECG sometimes shows a significant rise in the ST segment, which indicates the development of transmural damage. Since during an angina attack it is rarely possible to register ECG, use many hours (Holter) monitoring by recording the ECG on magnetic tape.

If outside of an attack, and sometimes even during an attack, the ECG is not changed, provocative tests are performed to detect myocardial ischemia

• transesophageal atrial pacing;

• tests with physical activity: bicycle ergometry;

• the dipyridamole test causes a "steal" effect and thereby revealing the focus of ischemia

• test with ergometrine and cold - to provoke a spasm coronary vessel

• pharmacological tests: with propranolol, nitroglycerine, potassium chloride test to distinguish between changes caused by myocardial ischemia and other causes.

Coronary angiography reveals the location, extent of the lesion and the degree of narrowing of the coronary arteries, as well as anomalies in the development of the coronary arteries

Echocardiography with angina pectoris reveals a local decrease in myocardial contractility (a sign of ischemia in this zone) during the exercise test.

Myocardial scintigraphy - done more often with c^{201} TL injected intravenously. With coronary insufficiency (at rest or during physical activity) accumulation of the radionuclide in the focus ischemia is reduced.

8. CARDIAC MUSCLE NECROSIS SYNDROME (INFARCTION MYOCARDIA)

Definition. Myocardial infarction (MI) - the formation of a necrotic focus in the heart muscle as a result of an acute violation of the coronary circulation.

Reasons for the development of MI.

• the main cause of MI is atherosclerosis of the coronary arteries, complicated by thrombosis. Spasm of the coronary arteries (often all pathologically altered).

Rare causes of MI are:

• embolism of the coronary arteries, their thrombosis in inflammatory lesions (thrombangiitis, rheumatic coronaritis, periarteritis nodosa, etc.);

• thickening of the arterial wall due to metabolic diseases or intimal proliferation (mucopolysaccharidosis, amyloid, taking contraceptives);

• compression of the mouth of the coronary arteries by a dissecting aortic aneurysm;

• embolism of the coronary arteries (infective endocarditis, mitral valve prolapse, thromboembolism from the left chambers heart disease, myxoma (tumor of the heart), thrombus formation on implanted valves or on coronary angiography, and surgical interventions on the coronary arteries);

• congenital defects of the coronary arteries;

• a sharp discrepancy between myocardial oxygen demand and its intake (aortic heart disease, carbon dioxide poisoning);

• coagulation disorders - thrombosis (polycythemia, thrombocytosis,

disseminated intravascular coagulation).

Symptomatology.

A typical anginal variant: the pain is very intense behind the sternum, in the precordial region. The pain is pressing, squeezing, bursting, tearing, burning with irradiation to the left arm, both arms, shoulder, under the left shoulder blade, neck, lower jaw, interscapular space. The pain lasts for several hours, is accompanied by cold sticky sweat, a feeling of fear (typical anginal, painful form of MI) - status anginosus.

Atypical options:

1. Shortness of breath, shortness of breath with a sharp difficulty in breathing, lack of air, dry cough or with copious pink frothy sputum (asthmatic variant of myocardial infarction);

2. Pain in the epigastric region, nausea, vomiting (gastralgic variant).

3. The heartbeat is constant or paroxysmal, interruptions in work of the heart, feeling of sinking, cardiac arrest, rare pulse (rare heart beats), short-term loss of consciousness (arrhythmic variant of myocardial infarction).

4. Sudden dizziness, darkening of the eyes, nausea, vomiting, severe general weakness, blanching, cold extremities, cold sweat, loss of consciousness (cerebral variant of myocardial infarction; and with a sharp drop in blood pressure - complication of acute myocardial infarction with cardiogenic shock; in the absence of pain - collaptoid variant of MI).

5. Pain only in the arm, shoulder, jaw (left or right), in the spine, etc. (peripheral pain variant).

Atypical variants are most often observed against the background of repeated MI, circulatory failure, in elderly people with severe cardiosclerosis.

Inspection. Paleness of the skin, acrocyanosis. With acute left ventricular failure (cardiac asthma, pulmonary edema) severe shortness of breath, suffocation, cough dry or with pink frothy sputum, bubbling breath. With cardiogenic shock, the face of Hippocrates: the skin is gray-pale in color, covered with cold sticky sweat, cold, marble-cyanotic limbs.

Palpation. With an uncomplicated variant - without features.

With the development of an aneurysm of the heart in the region of the apex, anterior the wall is determined by pathological pulsation in the precordial region. With a rupture (separation) of the papillary muscle, perforation of the interventricular septum, systolic tremor is determined at the apex of the heart.

Percussion. The borders of the heart are expanded to the left in case of complications (aneurysm, heart failure)

Auscultation. 1 tone is weakened or deafness of tones, gallop rhythm - with weakness of the left ventricle. With internal ruptures of the heart, a coarse systolic murmur appears: with a rupture of the interventricular septum, the epicenter of the murmur is above the apex and to the left of the sternum, the murmur is conducted over the entire region of the heart, into the interscapular space; rupture of the papillary muscle leads to mitral insufficiency with the appearance of a rough blowing systolic murmur over the apex with irradiation to the left axillary region. Tones can be arrhythmic due to various rhythm disturbances.

The pulse can be arrhythmic, sharply accelerated, rare, small, thready. Blood pressure during a pain attack may increase, with shock - a sharp decrease in blood pressure.

Resorption-necrotic syndrome.

Biochemical research.

1. The study of the content of enzymes in the blood serum: an increase in CK (and its CKF - fractions from the first hours, A sat and AlAT from the end of the first

day, normalize within a week; an increase in the activity of LDG (total) and its first and second isoenzymes occurs during the first 2-3 days and gradually decreases over 2 weeks.

2. Myoglobin (a protein found only in the myocardium and skeletal muscles) increases in the blood 2 hours after the appearance of pain and normalizes after 28-32 hours.

Complete blood count: leukocytosis from the first hours of the disease, stab shift to the left, increase in ESR from 3-5 days.

Electrocardiography makes it possible to clarify the diagnosis of MI, its localization, the depth of the lesion, the extent, the phase of the course (stage), and to identify rhythm and conduction disturbances. There are five stages:

1. Ischemic stage - within 10-30 minutes after a strong an attack of retrosternal pain, typical of myocardial infarction, In most cases, a pattern is observed that is characteristic of subendocardial ischemia. A high positive "coronary", symmetrical T wave is recorded above the infarction zone. However, in such an early period of myocardial infarction, ECG is rarely recorded, therefore it is almost impossible to detect high positive "coronary" T waves on the ECG

2. Stage of damage - lasts 2 hours, the process is reversible. On the ECG with transmural (subepicardial) damage, the ST segment rises above the isoline, the ST segment is elevated above the isoline in the form of a monophasic curve (the ST segment merges with a positive T wave). In the leads, the positive electrode of which is located above the wall of the left ventricle opposite to the infarction, there is a decrease in the ST segment below the isoline (reciprocal changes). With subendocardial ischemic injury above the infarction zone, the ST segment is located below the isoline.

3. Acute stage (stage of necrosis) - usually lasts 3-5 days. Part of the muscle fibers that were in a state of damage dies at this stage, which leads to a decrease in the damage zone. On the ECG, the ST segment approaches the isoline somewhat, due to a decrease in the damage zone. However, the ST segment is located above the isoline, since around of a heart attack while the zone of transmural damage remains. The zone of necrosis leads to the appearance of an abnormal Q wave:

QS - with transmural infarction or QR - with non-transmural. Due to the fact that at this stage a zone of transmural ischemia is formed on the periphery of the damage zone, a negative symmetrical "coronary" wave is recorded on the ECG

T. In leads located above the wall opposite to the infarction, on the contrary, there is an increase in the amplitude of the positive T wave (reciprocal changes). In this way all 3 zones are simultaneously present on the ECG (necrosis, damage, ischemia).

4. Subacute stage (there is no damage zone, but there is an ischemia zone

and necrosis, where there is a process of resorption, repair) - the ST interval on the isoline, the T wave is negative symmetrical.

5. Cicatricial stage - ischemia disappears, the sign of cicatricial changes in the form of a pathological Q wave persists.

IM localization:

1. MI of the anterior wall, including the apex - changes in I II,

avl, V 3, V 4 leads.

2. Infarction of the lower (diaphragmatic) region - II, III, avf leads.

3. MI of the side wall - I, avl, V 5, V 6 leads.

4. Defeat of a partition V 2, V 3 leads.

Radioisotope diagnosis of myocardial infarction with radioactive pyrophosphate: due to the accumulation of a radioactive substance in the focus necrosis of MI is characterized by a focus of bright "glow".

Echocardiography. There are signs of focal lesions myocardium: paradoxical movement of the interventricular septum, a decrease in its systolic excursion of less than 0.3 cm; a decrease in the amplitude of movement of the posterior wall and akinesia of one of the walls of the left ventricle.

The main complications of MI are cardiogenic shock (decrease in blood pressure below 80/60 mm Hg. Art., oliguria, spasm of peripheral vessels); acute left ventricular failure (pulmonary edema); myocardial rupture (external with the development of cardiac tamponade and internal with the development of a ventricular septal defect); a variety of rhythm disturbances (up to fibrillation ventricles and clinical death) and cardiac conduction (up to a complete blockade and asystole); (up to complete atrioventricular blockade and asystole); fibrinous pericarditis, acute aneurysm.

9. PERICARDIAL SYNDROME

Among the various diseases of the pericardium, the main place belongs to pericarditis, other forms of damage (cysts, neoplasms) are rare. In this development, pericarditis syndromes are considered.

Definition. Pericarditis is an inflammation of the pericardial sac damage to both sheets (epicardium and pericardium), with the deposition of fibrin threads on the sheets or the accumulation of a large amount of exudate in the pericardial cavity. The outcome of pericarditis is not infrequently a thickening of the pericardium

(often up to 1 cm) with the deposition of lime, a rigid membrane around the heart ("shell heart") is formed, which prevents diastolic stretching of the ventricles.

Reasons for the development of pericarditis.

1. Infectious:

1. viral infection (influenza A and B, Coxsackie A and B, ECHO);

2. bacterial infection (pneumococci, streptococci, meningococci, Escherichia coli and other flora);

3. tuberculosis;

4. protozoa;

5. parasitic invasion;

6. fungal diseases.

2. Aseptic pericarditis:

1. allergic diseases (serum sickness, drug allergy

2. diffuse connective tissue diseases (systemic lupus erythematosus, rheumatoid arthritis);

3. with malignant tumors;

4. traumatic;

5. radiation injury, massive radiotherapy; 6. with blood diseases and hemorrhagic diathesis;

7. autoimmune (postinfarction, after surgery on the heart and pericardium);

8. with metabolic disorders (uremia, gout);

9. hypothyroidism;

10. with hypovitaminosis C

The pathogenesis of pericarditis depends on their etiology. Infectious pericarditis is associated with the penetration of microorganisms into the pericardial cavity by the hematogenous, lymphogenous route or when a neighboring purulent focus breaks through. The development of infectious-allergic pericarditis is associated with an allergic reaction in response to an acute or exacerbated chronic infection.

In myocardial infarction, pericarditis occurs as a reactive inflammation of the pericardium in response to necrosis penetrating to the outer surface of the myocardium or due to autoimmune reactions (in Dressler's syndrome). With uremia, pericarditis develops due to the release of urea crystals by the pericardium

and irritation of the pericardial sheets by them. Autoimmune mechanisms play the main role in systemic connective tissue diseases.

Thus, there are two main ways of damage to the pericardium - the direct impact of a pathogenic agent and the development of inflammation on the immune basis.

Symptomatology.

Pericarditis is characterized by two main syndromes:

1. Syndrome of fibrinous pericarditis with deposition on sheets

pericardial fibrin strands (dry or fibrinous pericarditis).

2. syndrome of fluid accumulation in the pericardial cavity (hydropericardium) with signs of compression (tamponade) of the heart; a similar picture is also given by constrictive (adhesive) pericarditis;

a) syndrome of fibrinous pericarditis:

Complaints. A characteristic complaint is pain in the region of the heart.

This complaint is often the only one. Pain is usually intense and prolonged. It is associated with irritation. pain receptors, which are found in large numbers in the pericardium. Pain is more often felt in your anterior chest cage, but can be localized in isolation behind the sternum, in precordial region, in the region of the apex of the heart. The pain intensifies on inspiration, when turning the torso, raising the arms, which is due to additional irritation of the receptors. The pain is not associated with exercise and is not relieved by nitroglycerin. It sometimes decreases in a sitting position with the torso tilted forward. With the inflammatory genesis of pericarditis, an increase in body temperature is possible.

Inspection and percussion. Usually do not reveal any pathological signs.

Auscultation. If the pericardium is inflamed in the anterior region walls and apex, then a pericardial friction rub is heard.

The pericardial friction rub can be quiet or loud, but it is most often rough (scraping, scratching), auscultated best in the zone of absolute cardiac dullness. When pressing with a stethoscope or leaning forward, the noise increases. Sometimes in systole or more often in protodiastole, an additional tone (pericardial tone) is heard.

ECG. The rise of the ST segment is recorded according to the type of monophasic curve in many leads. There are no reciprocal changes present in acute myocardial infarction. Subsequently, biphasic or negative T waves appear without the formation of pathological Q waves.

X-ray and ultrasound examination of the heart. They don't reveal any change.

Biopsy of the pericardium. It is carried out only with an unclear diagnosis, with differential diagnostic purpose.

Laboratory research. With the inflammatory genesis of fibrinous pericarditis, corresponding changes are detected. In other cases, deviations are associated with the underlying disease.

b) syndrome of fluid accumulation in the pericardial cavity (hydropericardium).

Complaints. With a small amount of liquid (50-100 ml), there may be no complaints. However, further accumulation of fluid leads to the appearance of symptoms of total heart failure: severe persistent shortness of breath, which decreases in a sitting position, while leaning forward or standing on kneeling, pressing his face against the pillow ("pose of a praying Mohammedan"). Often the patient is worried about coughing, hemoptysis (stagnation of blood in the lungs). There are pains in the right hypochondrium (enlargement of the liver), swelling in the legs, lower back, in the genital area, the abdomen (ascites) is enlarged.

Inspection. Forced position (sitting or knee-elbow). Swelling of the veins of the neck, cyanosis and swelling of the face, neck (Stokes' collar) due to compression of the veins by fluid and obstruction of blood flow to the heart. Edema on the legs, ascites.

Percussion. The borders of the heart are displaced in all directions. The coincidence of the boundaries of relative and absolute cardiac dullness.

The configuration of the heart resembles a triangle or a trapezoid.

Auscultation. Deafness of heart sounds, tachycardia, sometimes - rhythm gallop, arrhythmia

Pulse, BP. The pulse is paradoxical (when inhaling, the filling of the pulse decreases), small. Blood pressure is reduced, especially systolic.

Liver. Enlarged, dense ("pseudocirrhosis")

ECG. A sharp decrease in the voltage of all teeth. Sometimes - ST changes.

Echocardiography. The main method for confirming the presence of hydropericardium. An "echo-free space" appears between the immobile pericardium and the oscillating one during contractions epicardial heart.

X-ray examination. The shadow of the heart is significant enlarged, triangular. Decrease in cardiac pulsation ("quiet heart").

Central venous pressure (CVP). sharply increased at hydropericardium.

Pericardial paracentesis (pericardial puncture). It is carried out for diagnostic and / or therapeutic purposes in the presence of symptoms of cardiac tamponade.

10. DIFFUSE MYOCARDIAL SYNDROME

Definition - inflammatory or dystrophic lesion of myocardium due to exposure to various pathological factors.

The reasons.

1. Infectious agents (viruses - Coxsackie A, B, influenza, para influenza, measles, herpes, cytomegaloviruses, etc.; bacteria, fungi, protozoa, HIV infection). When exposed to them inflammation develops and the process is called myocarditis.

2. Toxic effects (alcohol, pesticides, professional poisons).

3. Radiation impact on cardiomyocytes.

4. Myocardial damage due to endocrine disorders (thyrotoxicosis, hypothyroidism, hyperproduction of the cortical and medulla of the adrenal glands, etc.), anemia, physical overstrain (sports heart), etc.

Under the action of infectious factors, toxins cause damage to the cell membrane, resulting in the release of cardiac antigens. The immune system causes the production of antimyocardial antibodies that are involved in the formation of immune complexes. This leads to further damage to the myocardium.

When exposed to the above mentioned physical, chemical, endogenous factors is their direct influence on cardiomyocytes with the development of foci of dystrophy, necrosis, which later forms connective tissue occurs. These processes lead to a decrease in contractile and elastic properties of the myocardium, expansion of the cavities of the heart and development in thrombosis due to a decrease in blood flow velocity.

Often it is not possible to clearly establish the cause of myocardial damage, and the expansion of the heart cavity heart with the development of relevant clinical signs is morphologically determined the pathological process is commonly referred to as dilated cardiomyopathy.

Symptomatology of diffuse myocardial injury syndrome consists of signs of total (left and right ventricular) heart failure, a variety of rhythm and conduction disorders of the heart, as well as thromboembolic complications.

As a result, patients complain of shortness of breath during exercise and then at rest. There is a cough, hemoptysis, asthma attacks in a horizontal position (cardiac asthma), bubbling breath with foamy pink fluid from the nasal passages and mouth

(pulmonary edema). All the above complaints are signs of stagnation of blood in the lungs (left ventricular failure). With the development of right ventricular failure, patients are concerned about swelling in the legs, pain in the right hypochondrium and epigastrium (enlarged liver), an increase in the abdomen (ascites).

Heart rhythm disturbances are manifested by a feeling of palpitations, interruptions in the work of the heart, "fading" of the heart. Signs of thromboembolism in a large circle (from the left heart) are loss of consciousness, development of hemiplegia and hemiparesis, speech disorders (embolism in the vessels of the brain); acute back pain, hematuria (renal embolism); pain in any of the limbs, its coldness, lack of pulsation (embolism of peripheral arteries); abdominal pain, vomiting, bloating, signs of intestinal obstruction (mesenteric vessels embolism).

Inspection. Forced position (orthopnea), shortness of breath, yellowness of the sclera and skin, pronounced acrocyanosis, swollen neck veins, diffuse apical impulse, pronounced cardiac impulse, the abdomen is enlarged, the navel is flattened or protruded, swelling in the legs, sacrum, in the lumbar, areas.

Palpation. Spilled apex beat, spilled cardiac push (pulsation to the left of the sternum, spreading in epigastric region).

The liver is enlarged, dense, painful.

Percussion. Above the lungs in the lower sections, dullness of the percussion sound. A sharp expansion of the boundaries of relative and absolute dullness of the heart in all directions. Increased size of the liver.

Auscultation. Above the lungs, it is heard at the site of dullness of percussion sound (in the lower sections along the posterior-lateral surfaces) against the background of weakened vesicular or hard breathing, crepitus, fine bubbling, non-sound (stagnant) wet rales. With cardiac asthma, pulmonary edema, a large number of wet (sometimes dry) rales, bubbling breath.

Heart sounds are muffled or muffled, often the gallop rhythm due to 3 and 4 tones, various rhythm disturbances (extrasystole, atrial fibrillation, paroxysmal tachycardia), murmurs of relative insufficiency of the bicuspid and tricuspid valves.

Pulse. The pulse is frequent, weak filling, arrhythmic; at atrial fibrillation pulse deficit. BP is reduced.

Laboratory data. With myocarditis in the general blood test, leukocytosis, a shift of the leukocyte formula to the left, an increase in ESR. With viral myocarditis, leukopenia is possible (however, a shift to the left in the leukocyte formula remains).

Biochemical study of blood: an increase in the total and MB fractions of CKF, LDG, AST, dysproteinemia (increase in a 2 and y globulins), an increase in fibrinogen, sialic acids, seromucoid. Hyperfermentemia in myocarditis persists for a long time compared with myocardial infarction.

Immunological blood tests: a positive inhibition of leukocyte migration in the presence of myocardial antigen, a decrease in the number of T-lymphocytes and T-suppressors, an increase in the blood levels of Ig A and Ig G; detection in the blood of CIC, antimyocardial antibodies, high titers of antibodies to Coxsackie, ECHO, influenza or other infectious agents. With streptococcal infection (rheumatism) - an increase in titers of antistreptolysin-0 and antistreptohyaluronidase. With dystrophic lesions of the myocardium, dilated cardiomyopathy, the general blood test is not changed.

Instrumental research.

ECG: always significantly changed: reduced T wave and interval S-T in many, sometimes in all leads, a negative T wave is possible, often atrioventricular blocks of varying degrees, blockade of the bundle legs, extrasystoles, paroxysmal tachycardia, atrial fibrillation and flutter.

Often the ECG is similar to macrofocal (even with transmural) myocardial infarction (abnormal Q wave or even QS, ST segment displacement, T wave change). However, with myocardial infarction, the ECG shows a certain dynamics. Signs of atrial and ventricular myocardial hypertrophy are characteristic.

PhCG: in addition to a sharp weakening of heart tones and murmurs, the appearance of additional heart sounds due to severe myocardial damage is characteristic. More often recorded pathologically enhanced third tone (protodiastolic gallop rhythm), and with slowing atrioventricular conduction and a sharp heart weakness - the fourth tone (presystolic gallop rhythm), which, merging with the third tone, forms a (summation) mesodiastolic gallop rhythm.

Echocardiography: cardiomegaly, dilatation of various chambers heart, signs of total myocardial hypokinesia, decreased ejection fractions, often - intracardiac thrombi.

Radiography of the heart: cardiomegaly ("bull's heart"). Lifetime myocardial biopsy: a picture of inflammation (with myocarditis), small- or large-focal cardiosclerosis); dystrophy (often necrosis) of cardiomyocytes.

11. FOCAL MYOCARDIAL SYNDROME

The causes are the same, but more often infections. In most cases, focal myocarditis does not manifest itself. If it manifests itself, then in the form of

rhythm disturbances by the type of extrasystole, atrial fibrillation, conduction disturbances.

On physical examination, there are no changes, with the exception of rhythm disturbance, more often transient.

Conventional laboratory methods (see above) are not very informative.

Of the instrumental methods of research, changes are possible only on the ECG and EchoCG

ECG: various rhythm and conduction disturbances, depression

ST segment, negative T waves (often detected only after exercise).

Echocardiography: the size of the cavities of the heart is normal. In the myocardium, foci (small) of a brighter glow (cicatricial) are sometimes determined.

12. SYNDROME OF INJURED ENDOCARDIA (ENDOCARDITIS)

Definition. Endocarditis is an inflammatory infectious disease of the endocardium with the localization of the pathogen (with infective endocarditis), immune complexes (with rheumatic or other endocarditis) on the * valves of the heart, less often on the parietal endocardium.

The reasons. In the occurrence, the role of infection is clearly traced. Most often, the causative agent of infective endocarditis (IE) are microbes - saprophytes. Most often staphylococci, streptococci are sown, less often - gram-negative bacteria (E. coli, Pseudomonas aeruginosa, Klebsiella, Proteus), fungi, rickettsiae.

There are only a few reports of viral endocarditis.

In rheumatic endocarditis, the disease is preceded by angina, exacerbation of chronic tonsillitis (i.e. beta-hemolytic streptococcus group A).

Predisposing factors for the development of IE:

Upper respiratory infections, oral surgery

(tooth extraction, etc.), operations, instrumental examinations of the genitourinary system. Urinary tract infections. Operations on the heart and blood vessels. Prolonged stay of the catheter in the vein. "Small" skin infections. Congenital and acquired heart defects. Mitral valve prolapse, drug addiction.

If there is a focus of infection in the body (in a patient with an altered reactivity of the body, impaired immune status), bacteremia develops. Microorganisms settle on the valves of the heart and create a "secondary" focus of infection. The fixation and

reproduction of microorganisms on the endocardium with the formation of a polyposis-ulcerative lesion of the heart valves, in the wall endocardium, is promoted by a previous change in the tissue and surface of the valve. The growth of bacteria is accompanied by the formation of vegetations with the destruction of the valves. Tearing off, pieces of valvular microbial vegetations enter various organs with the blood flow and thereby exacerbate septic manifestations, cause the development of thromboembolic complications (infarction of the kidneys, myocardium, spleen, etc.).

Microorganisms fixed on the valves cause prolonged autosensitization and hyperergic damage to organs and tissues (immune generalization of the process). Immune and autoimmune disorders cause the development of vasculitis and visceritis (immunocomplex nephritis, myocarditis, hepatitis, capillaritis, etc.).

In the future, dystrophic changes develop in the affected organs.

The modern theory of the pathogenesis of immune (for example, rheumatic) endocarditis is toxicoimmunological. β-hemolytic streptococcus group A produces substances that have a pronounced cardiotoxic effect, and are capable of suppressing phagocytosis, damaging lysoslysosomal membranes, the main substance of connective tissue.

Palpation. At the stage of formation of valvular heart disease in immune and infective endocarditis, the data correspond to the detected heart disease. In IE, an enlarged spleen (splenomegaly) is palpated.

Auscultation. The most important sign of developing endocarditis is a protodiastolic murmur, which is best heard in Botkin's point, in the vertical position of the patient and on the left side. Noise at first soft short, changeable, later longer, it acquires a sawing character (indicates perforation or detachment of the aortic valve leaflet).

Often, a systolic murmur appears first at the Botkin point due to polypous vegetations on the aortic valve.

With the development of IE against the background of a formed rheumatic defect, new noises appear (systolic, diastolic). A reliable sign of past endocarditis is a further formed heart disease.

Laboratory data for IE:

Complete blood count - leukocytosis with a shift to the left, monocytosis, thrombocytopenia, increased ESR. Urinalysis: microhematuria, proteinuria, cylindruria. An increase in the level of sialic acid globulins, fibrinogen, positive and formol test, C-reactive protein (seromucoid). Bacteremia is detected (take blood for culture before antibiotic treatment, at the height of a sharp rise in body

temperature.) It is better to culture arterial blood. An increase in the titer of antistreptococcal antibodies (antihyaluronidase and antistreptokinase more than 1:30, antistreptolysin more than 1:250).

Instrumental research.

Echocardiography. Thickening, vegetations on the valves of the heart are revealed. In the future - signs of a particular defect hearts.

PhCG - in case of formation of aortic valve insufficiency there is a proto-diastolic murmur associated with the second tone, in Botkin's point and above the aorta.

ECG ~ diffuse changes in the myocardium, possible violations atrioventricular conduction., other rhythm disturbances.

13. HEART FAILURE SYNDROME

Circulatory failure is a pathological condition, which consists in the inability of the circulatory system to deliver to organs and tissues such an amount of blood that is necessary for their normal functioning.

The underlying cause of heart failure is a decrease in myocardial contractility, developed as a result of overload heart pressure, blood volume, or direct myocardial injury.

Pressure overload develops with stenosis of the aortic orifice, pulmonary artery, left and right atrioventricular orifices. Volume overload occurs with regurgitation of the blood, observed in valvular insufficiency of the heart.

Heart failure caused by primary myocardial damage is observed with myocarditis, cardiomyopathies, diffuse cardiosclerosis, large-focal myocardial infarction.

The biochemical basis for the development of heart failure is a violation of the energy supply of the contractile function of the myocardium, ion transport, adrenergic regulation of the myocardium.

Syndrome of acute left ventricular failure occurs in diseases where the left ventricle predominantly suffers (hypertension, aortic disease, myocardial infarction). A manifestation of acute left ventricular failure is cardiac asthma and pulmonary edema.

Symptomatology: asthma attack, severe weakness, cold sweat, position orthopnea, cough with mucous, difficult to separate sputum. A lot of wet and dry rales are heard over the lungs. Heart sounds are weakened, above the pulmonary trunk the 2^{d} tone is strengthened; tachycardia, pulse is frequent, small. With an increase in congestion in a small circle, pulmonary edema develops; the sensation of

suffocation and coughing intensify even more, breathing becomes bubbling, and abundant foamy sputum appears.

A mass of moist rales is heard above the lungs. The pulse is sharply accelerated, filiform.

The syndrome of acute right ventricular failure develops with embolism of the pulmonary artery trunk or its branches.

Symptomatology: sudden increase in breathing, cyanosis, cold sweat, feeling of pressure or pain in the heart area, pulse becomes small and frequent, blood pressure drops, jugular veins swell, liver enlarges, edema later joins.

Syndrome of chronic heart failure.

The severity of clinical symptoms depends on the stage of heart failure.

I stage. Shortness of breath, increased heart rate when performing normal physical activity. At rest, these phenomena disappear. Borders lungs without changes, vesicular breathing with a hard shade.

II A stage. With the defeat of the left section, there is a lack of blood circulation in a small circle. The main complaints of patients are shortness of breath during physical exertion, sometimes attacks of suffocation (cardiac asthma), palpitations. Dry cough appears, with stenosis of the left venous opening - hemoptysis. On examination, there is a cyanotic flush, acrocyanosis.

The borders of the lungs are normal. On auscultation, breathing is rough, moist fine bubbling rales. The heart is enlarged to the left due the ventricle or to the right and up with narrowing of the left venous orifice. The auscultatory picture reflects the nature of the lesion of the heart. Atrial fibrillation, extrasystole, sinus tachycardia are often detected. The liver is not enlarged. There are no edema.

ECHO-KG: decrease in ejection fraction, areas of hypokinesia.

Patients with right heart failure have congestive insufficiency in a large circle. In these patients there are complaints of heaviness in the right hypochondrium, a decrease in diuresis.

Examination reveals acrocyanosis, swollen jugular veins, - swelling on the legs. On auscultation of the lungs, breathing is vesicular. The heart is dilated to the right due to the right atrium, absolute dullness is significantly increased. Above the base of the sternum, you can determine an independent systolic murmur, a positive symptom of Rivero-Corvallo. The liver is enlarged, its surface is smooth, the edge is rounded. Sometimes you can define the presence of free fluid in the abdominal cavity.

II B stage. Shortness of breath at the slightest physical exertion, palpitations, heaviness in the right hypochondrium, reduced diuresis, edema. On examination - the position of orthopnea, anasarca, acrocyanosis.

On auscultation of the lungs, breathing is hard, wheezing is muffled, wet. Often there is a significant expansion of the heart in all directions. Auscultation data correspond to the nature of the heart lesion. Many patients have atrial fibrillation and other rhythm disturbances. The liver is enlarged, painless. Determined free fluid in the abdominal cavity, hydrothorax, hydropericardium

III stage. In patients, disorders are at the forefront water-salt metabolism, dystrophic changes in internal organs. Clinically and radiographically revealed pneumosclerosis is revealed. Patients have hydrothorax, percussion and X-ray revealed dilatation of both ventricles, ascites, anasarca, enlarged liver, marked weight loss.

14. EXTRACRANIAL ARTERY SYNDROME BRAIN (BRACHIOCEOPHALIC ARTERY SYNDROME)

Definition. Syndrome of damage to the extracranial arteries brain consists of symptoms of ischemia of the brain, eyes and upper limbs.

This syndrome is characterized by damage to the common carotid artery, the initial segment of the internal carotid artery, bifurcation of the common carotid artery, vertebral and subclavian arteries.

The reasons:

Intravasal causes: atherosclerosis, nonspecific aortoarteritis, aneurysm of the internal carotid artery against the background of IHD or HBP, arterial microembolism with pieces of blood clots. Extravasal causes: displacement, kink or compression of the vertebral artery by the anterior scalene muscle; cervical osteochondrosis, causing compression of this artery by osteophytes.

Congenital pathology: elongation, sharp tortuosity of brachiocephalic vessels with the formation of kinks.

Symptomatology.

Complaints. Transient disorders of cerebral circulation ("intermittent claudication of the brain" - ischemia in the vertebrobasilar basin): headache in the occipital region, systemic and mixed dizziness, sensation of ringing and noise in the ears, unsteadiness when walking, double vision, bouts of loss of consciousness. Chronic vascular insufficiency of the brain is characterized by headaches, dizziness, memory impairment, decreased intelligence, and performance.

With damage to the carotid and vertebral arteries, patients complain of visual impairment - from a slight decrease to complete blindness.

Inspection. Does not allow to obtain pathognomonic signs.

Palpation. Detects changes in temporal, carotid,

brachial and radial arteries. The pulsation of the arteries may be asymmetric, while it is reduced or absent. At occlusion of the internal carotid artery and its stenosis in the region bifurcation, the pulsation of the temporal artery is preserved and can be reinforced.

Auscultation. A systolic murmur is heard on sleepy brachial arteries, above the brachiocephalic trunk (in the right supraclavicular fossa behind the sternoclavicular joint).

Definition of blood pressure. Asymmetry of blood pressure in the upper limbs with subclavian artery injury. There is a decrease in systolic blood pressure to 80-90 mm Hg pulse pressure up to 15-20 mm Hg

Sphygmography of the carotid and temporal arteries. There is asymmetry in height and deformation of the waves of the curve.

Rheoencephalography. Interhemispheric asymmetry of the blood supply to the brain.

Contrast aortography. Stenosis of the affected segments of the arteries.

Dopplerography of vessels. The turbulent current is registered blood in front of the site of stenosis, a decrease in blood flow velocity below the site of stenosis.

Laboratory data. In the presence of intravascular causes arterial lesions, leukocytosis is possible, an increase in ESR in general blood test. An increase in cholesterol triglycerides, lipoproteins, an increase in the titer of rheumatoid factor, a change in the rheological properties of blood (increase in viscosity, hematocrit, aggregation activity of platelets and erythrocytes), the presence of immunological changes (an increase in circulating immune complexes, immunoglobulins).

15. CHRONIC ABDOMINAL ISCHEMIA SYNDROME

Definition. The syndrome of chronic abdominal ischemia is characterized by ischemic circulatory disorders in the abdominal organs due to impaired patency of the visceral arteries. The syndrome is characterized by a triad of symptoms: paroxysmal abdominal pain, progressive weight loss, and bowel dysfunction.

According to the localization and nature of clinical manifestations, there are forms: 1) celiac, 2) mesenteric small intestine, 3) mesenteric large intestine

The reasons. Violation of the patency of the visceral arteries can be due to intravasal, extravasal causes, congenital pathology.

Intravascular causes: atherosclerosis, systemic vasculitis (nonspecific aortoarteritis, periarteritis nodosa).

Extravasal causes: compression of the celiac trunk by the falciform ligament of the diaphragm; compression associated with high discharge of the celiac trunk from the aorta, kink of the celiac artery. Congenital diseases: fibromuscular dysplasia, hypoplasia and anomalies in the development of visceral arteries.

Symptomatology.

Complaints: Pain in the abdomen after taking a rich and fatty food, 20-25 minutes after eating, lasting 2-2.5 hours (the period of passage of food through the intestines). When defeated the celiac trunk is characterized by severe convulsive pain in the epigastrium; for the superior mesenteric artery - dull, aching pain in the mesogastrium, for the inferior mesenteric artery - aching pain in the left iliac region. Pain decreases with self-restriction in food - "syndrome of small food." Instability of the stool (diarrhea is replaced by constipation), bloating. Unformed feces with poorly digested food, contains mucus. progressive weight loss.

Examination reveals no characteristic features.

Palpation: pain in certain topographic areas of the abdomen, corresponding to the localization of the lesion of the visceral arteries.

Auscultation: vascular systolic murmur over the affected visceral arteries.

Aortography. Intravasal causes of the lesion: narrowing of the artery, ventro-mesenteric anastomosis. Extravasal causes: depression of the vessel wall (defect) followed by post-stenotic expansion. Congenital pathology: clear-cut multiple constrictions.

Ultrasonic dopplerography of vessels. Decreasing linear blood flow speed.

Radiography of the gastrointestinal tract: nonspecific changes - slow passage of barium in the stomach and intestines, a lot of gas in the intestines.

Laboratory data. Caused by disease causing this syndrome

16. SYNDROME OF BIFURCATION OF THE ABDOMINAL AORTA, ILIAC AND LOWER LIMB ARTERIES

Definition. The defeat of the bifurcation of the abdominal aorta, iliac arteries and arteries of the lower extremities is characterized by the development of symptoms of ischemia of the pelvic organs, gluteal muscles and lower extremities. The syndrome develops with lesions of the bifurcation of the abdominal aorta with unilateral lesions of the iliac arteries and with lesions of the bifurcation of the

abdominal aorta and both iliac arteries (Lerish syndrome), with lesions of the distal vascular bed (femoral, popliteal arteries).

The reasons:

Intravasal: atherosclerosis, nonspecific aortoarteritis, obliterating endarteritis, post-emolic occlusion and traumatic thrombosis.

Congenital pathology: hypoplasia or aplasia of the abdominal aorta, fibromuscular dysplasia of the iliac arteries.

Symptomatology.

Complaints. Pain in the calf muscles when walking (intermittent claudication), pain during exercise in the muscles of the buttocks, in the lower back, in the muscles of the thigh (high intermittent claudication), numbness, coldness of the lower extremities, impotence.

Inspection. Hypotrophy of the muscles of the lower extremities, cyanosis of the skin in feet, with pronounced ischemia, edema and hyperemia, trophic changes in the soft tissues of the foot, gangrene.

Palpation. Decreased or absent pulsation in the femurs, popliteal arteries and arteries of the foot.

Auscultation. Systolic murmur over the aorta, iliac and femoral arteries.

Definition of blood pressure. When the abdominal aorta is affected, blood pressure in the lower extremities is reduced or not detected.

Rheovasography, plethysmography, volumetric sphygmography.

Decrease in the main blood flow in the lower extremities, delay and a sharp decrease in amplitude, change in shape pulse wave. Contrast aortography. Erosion of the contours of the arteries, areas of stenosis, sometimes looking like a rosary.

With congenital hypoplasia of the abdominal aorta, iliac arteries, their lumen is evenly narrowed.

Radioisotope angiography. Violation of blood flow in the abdomen

aorta and arteries.

Ultrasonic dopplerography of vessels. Decreasing linear blood flow velocity in both iliac, femoral and popliteal arteries.

Laboratory data. Depending on the cause of development of this syndrome, the following changes are possible: in general blood analysis (leukocytosis, increased ESR), increased cholesterol, triglycerides, lipoproteins, changes in the rheological properties of blood according to coagulography (increased viscosity, hematocrit,

platelet and erythrocyte aggregation, hypercoagulation), immunological changes (increase in circulating immune complexes, immunoglobulins)

SYNDROMES IN GASTROINTESTINAL TRACT AND HEPATO-BILIARY SYSTEM

1. ESOPHAGUS SYNDROME

Definition. This is a set of symptoms that characterize damage to the esophagus in the presence of tumor, inflammatory,cicatricial or other changes in the body.

The reasons. The symptomatology of the esophagus is observed with atresia of the esophagus, congenital stenosis of the esophagus, hernia of the esophageal opening of the diaphragm; with reflux esophagitis, diverticula (saccular bulging of the wall), ulcers of the esophagus, polyps and tumors of the esophagus, achalasia (expansion) of the esophagus. The latter condition is also called idiopathic esophageal dilatation or cardiospasm. The most common cause of inflammatory lesions of the esophagus (esophagitis) is reflux (reflux) of active gastric juice into the esophagus due to cardia insufficiency - the so-called reflux esophagitis, which is most often observed with hiatal hernia. In the formation of hernias, the following factors are important:

1) a sharp increase in intra-abdominal pressure with excessive physical exertion;

2) congenital underdevelopment of the connective structures that strengthen the esophagus in the aperture of the diaphragm or their age-related dystrophic changes in the elderly;

3) traction of the cardial part of the stomach into the chest cavity longitudinal spastic contractions of the esophagus (with esophagospasm) or due to its cicatricial-inflammatory shortening (for example, with scleroderma). With peptic ulcer, cholelithiasis and other diseases, there may be a relative, i.e., functional insufficiency of the cardia due to spastic contraction of the pylorus, an increase in intragastric pressure.

Symptomatology

1) Dysphagia - violation of swallowing, choking, regurgitation,

feeling of holding food (lump) behind the sternum when swallowing solid or liquid food.

2) Esophageal vomiting. The difference from gastric vomiting is frequent absence of nausea and absence of characteristic odor vomit; spitting up undigested food shortly after food. Hypersalivation.

3) Pain behind the sternum of a pressing or burning character, which

often require differentiation from coronary pain; heartburn - a burning sensation behind the sternum (along the esophagus).

4) Bleeding with ulcerative, tumor lesions of the esophagus in the form of vomiting of scarlet blood, regurgitation of food with blood.

X-ray of the esophagus allows you to identify a hernia of the esophageal opening of the diaphragm (with reflux esophagitis) and observe gastroesophageal reflux. This study should be carried out both in a vertical and horizontal position. A peptic ulcer of esophagud is found as a niche at the end or as a relief of the mucous membrane, but it is difficult to identify it.

Esophagoscopy allows you to assess the severity and prevalence of inflammation (esophagitis); the presence of an ulcer of the esophagus, which in most cases appears round or oblong, often with a yellow-gray or bloody coating, under which a purple-red, uneven bottom is found.

Biopsy from the edges and bottom of the ulcer, from areas of mucosal metaplasia allows to differentiate cancerous, tuberculous, syphilitic peptic lesions of the esophagus.

Cytological examination of washing waters. Allows detection of tumor cells, pathogens

2. SYNDROME OF GASTRITIS WITH INCREASED HYDROGEN FUNCTION

Definition. This is a set of symptoms that characterizes inflammatory-dystrophic process in the gastric mucosa, leading to an increase in the secretory function of the stomach.

This condition may be caused by:

1. prolonged uncontrolled intake of certain drugs characterized by local irritant action: foxglove, salicylates, butadione, prednisolone, antibiotics, sulfonamides, etc.

2. rare receptions of coarse, spicy, spicy food;

3. hereditary predisposition;

4. diseases of the endocrine system (thyrotoxicosis, etc.);

5. diseases of organs leading to tissue hypoxia (chronic circulatory failure, etc.);

6. other diseases of the digestive system (cholecystitis, etc.);

7. conditions accompanied by the release of urea, uric acid, indole, skatole through the gastric mucosa (with gout, renal failure).

These factors first cause functional secretory and motor disturbances in the activity of the stomach (hypersecretion and hypermotility), and then - dystrophic changes in the mucosa. Participation in the pathogenesis of the bacterial factor - Helicobacter pylori (Campilobacter.)

Symptomatology.

Pain of a pressing and cutting nature in the epigastric region, without irradiation, increase 1-1.5 hours after eating, often are in the nature of "hungry", "night", "late". The occurrence of pain is associated with hypersecretion of hydrochloric acid in the interdigestive period and the ingress of acidic contents into departments that have an alkaline environment (pylorus, 12 p. K.) due to hypermotility. Unlike pain in peptic ulcer disease, the pain is less intense, spilled.

Dyspepsia.

1) gastric: heartburn on an empty stomach, often accompanies "late" pain; heartburn after eating spicy, rough food. Less often can be vomiting acidic contents; hypersalivation, nausea;

2) intestinal: constipation.

Palpation of the abdomen: diffuse diffuse pain in the epigastric region.

Examination of the tongue: the tongue is moist, the papillae are well expressed, during an exacerbation of gastritis, there may be a white coating on the tongue.

Gastroscopy with biopsy. Gastroscopy reveals hyperemia mucosa (diffuse or in the area of the pylorus) with symptoms hyperplasia. Biopsy reveals Helicobacter pylori X-ray examination reveals hypermotility, hypersecretion on an empty stomach.

Complete blood count without abnormalities.

The analysis of gastric juice reveals an increase in the hourly amount of secretion and an increase in acid production (often both on an empty stomach and basal and stimulated).

pH meter shows low pH values (< 2.0) in the basal and stimulated secretion, shortening of the main time

3. GASTRITIS SYNDROME WITH LOW SECRETORY FUNCTION.

Definition. This is a set of symptoms that characterizes a state of inflammatory-dystrophic changes in the gastric mucosa, leading to a decrease in secretory

function and indigestion. The cause of this condition may be qualitatively inadequate nutrition (deficiency of protein, iron, vitamins); repeated and prolonged malnutrition (addiction to hot, spicy foods, rare meals), alcohol abuse.

Diabetes mellitus, thyrotoxicosis, Addison's disease and other diseases of the endocrine system are accompanied by gastritis syndrome. In renal failure, gout, metabolic products (urea, uric acid, indole, skatol) are released through the gastric mucosa, which cause the development of the so-called elimination chronic gastritis. In 75% of cases, gastritis syndrome is combined with manifestations of the pathology of other digestive organs (cholecystitis, pancreatitis, etc.). Gastritis syndrome develops in conditions that cause tissue hypoxia (chronic circulatory failure, chronic lung disease, anemia).

Under the influence of these factors, first develop functional secretory and motor disturbances in the activity of the stomach, and further dystrophic changes and disturbances in the processes of mucosal regeneration. Structural changes are developing primarily in the epithelium of the surface layers of the mucous membrane. In the future, the glands of the stomach are involved in the pathological process, which are constantly atrophied or rebuilt according to the type of crypts. There is information about the autoimmune nature of mucosal lesions, but the issue has not been fully studied.

Symptomatology

Pain: localized in the epigastric region, occur shortly after eating, characterized by a feeling of pressure, fullness. Patients limit themselves to food due to fear of the occurrence pain.

Dyspepsia:

1) gastric; may be heartburn due to gaping of the esophagus openings of the diaphragm due to a decrease in acidic products in the stomach. More typical nausea, belching food and air, bad breath;

2) intestinal: bloating (flatulence), diarrhea. Occurs in due to a decrease in the production of enzymes below the underlying sections of the gastrointestinal tract due to a reduced amount of hydrochloric acid in the gastric contents. Smaller amounts of proteins, fats, carbohydrates, vitamins, and iron are absorbed in the intestine. Anemia develops, body weight decreases, i.e., a manifestation of impaired absorption (malabsorption) occurs.

Abdominal palpation data: diffuse diffuse pain in the epigastrium.

Examination of the tongue: the tongue is moist, covered with white or yellowish plaque, there may be partial or complete atrophy of the papillae.

Gastroscopy allows you to observe external (superficial) changes in the gastric mucosa, the mucosa is pale, in some places there are areas of hyperemia. The study of biopsy specimens makes it possible to study the histological structure of the deep layers.

X-ray examination allows you to study the dimensions, the location of the stomach, the relief of the mucosa, to identify special forms of gastritis: rigid, polypous, giant hypertrophic.

The analysis of gastric juice by the method of continuous vacuum aspiration allows us to identify different levels of reduction in the hourly secretion tension, debit-hour of hydrochloric acid. If, during parenteral stimulation with histamine (0.025 mg/kg of body weight) in the stimulated secretion, the debit-hour of hydrochloric acid is zero, then we need to talk about true achlorhydria, pH-metry both in the basal phase of secretion and in the stimulated secretion will indicate alkaline and slightly acidic pH (about 6.0).

Complete blood count - iron deficiency often develops anemia

4. SYNDROME OF UNCOMPLICATED GASTRODUODENAL ULCER

Definition.

This is a set of symptoms that characterize a condition accompanied by the formation of an ulcerative defect in the wall of the stomach or duodenum.

The reasons.

1) as the main manifestation of peptic ulcer (peptic ulcer) of the stomach and duodenum;

2) with medicinal ulcers (with prolonged and massive uncontrolled intake of acetylsalicylic acid and its derivatives, derivatives of pyrazolone, indole, histamine, glucocorticosteroids, etc.);

3) with "ischemic ulcers" associated with atherosclerosis, hypertension, congestive heart failure, emphysema;

4) with "hormonal" ulcers, manifested in hyperparathyroidism, ulcerogenic tumors of the pancreas, etc.;

5) in acute stress ulcers, in acute myocardial infarction, burn disease, stroke, etc.;

6) with ulcers, with hepatitis and cirrhosis of the liver, etc.

There are local and general factors that contribute to the formation of an ulcer. Local factors include the high proteolytic activity of gastric juice, the resistance of the so-called mucous barrier of the inner wall of the stomach to the digestive action

of gastric juice, a fairly high level of regeneration of the epithelium of the gastric mucosa and duodenal ulcer. Among the general factors of great importance is the violation of the complex neurohumoral regulation of the function of the stomach, which involves the central nervous system, subcortical centers, some endocrine glands, as well as many gastrointestinal hormones. However, many external factors (disorderly eating, smoking, drinking alcohol, etc.) are starting, violating the mechanism of regulation functions of the stomach and duodenum. In the mechanism of development ulcers in the duodenal region, a certain role is currently assigned to the bacterial factor (Helicobacter Pylori)

In the genesis of gastric ulcer, local factors are of great importance, and duodenal ulcer. - neurohumoral factors.

For both gastric and duodenal ulcers seasonal symptoms are characteristic (exacerbations occur more often spring and autumn), and periodicity (i.e., alternation of exacerbations and remissions of the disease).

Symptoms of gastric ulcer.

Pain - localized in the epigastrium; by nature they can be cutting, pressing, burning. The severity of pain is due to the depth of the ulcer crater, the degree of violation of the motor function of the stomach. With a stomach ulcer, especially a highly located one, the pain often radiates up and to the left; in the left side of the chest. With ulcers of the upper part of the stomach, pain is localized under the xiphoid process behind the sternum, in the region of the heart. During pain, patients tend to take a forced position (they lie on their stomach, sometimes they bring their bent legs to their stomach, etc.). A characteristic feature of pain is their occurrence shortly after eating, and the closer the ulcer is localized from the entrance to the stomach, the faster the pain occurs after eating. For example, with an ulcer of the cardiac region, pain can occur 15-20 minutes after eating, with an ulcer in the antrum - 60-80 minutes after eating.

Dyspepsia:

1) vomiting brings relief to patients, as it often occurs at the height of pain in the epigastrium; patients sometimes on their own cause vomiting;

2) nausea, belching (air, food);

3) heartburn - a burning sensation in the lower third of the stomach, associated

with the throwing of gastric contents into the esophagus;

4) constipation or diarrhea associated with a change in the tone of the colon intestines of neuroreflex genesis;

5) with gastric ulcer, especially in combination with gastritis. Often

appetite is reduced, and the appearance of early pain after taking food causes the sick to refrain from eating.

Examination of the patient.

Autonomic disorders are often found nervous system with a predominance of increased tone of the parasympathetic nervous system: cyanosis of the hands, wetness of the palms, increased sweating, pronounced red, less often white dermographism, temperature asymmetries.

Tongue examination.

The tongue is moist and may be covered with a white coating. On the mucous membrane of the root of the tongue, defects up to 1 cm in size can be detected, which indicates trophic disorders in the mucous membrane of the tongue.

Inspection. Pigmentation is often found on the skin of the abdomen from prolonged use of heating pads.

Palpation of the abdomen. For superficial and deep palpation local pain in the epigastric region is determined. A number of patients have pain during percussion in the epigastric region - Mendel's symptom.

Analysis of gastric juice. With ulcers of the body and cardia of the stomach, in most cases, the indicators of gastric secretion are either lowered or do not differ from the norm.

Analysis of feces for occult blood - is carried out after 3-4 days during the period of exacerbation. Detection of bleeding indicates an exacerbation of the ulcer.

X-ray examination: direct X-ray sign - the presence of a niche (defect) of the stomach wall, filled with a contrast agent and protruding beyond the line of the contour of the stomach. With this method, an ulcer is detected in 80-85% of cases.

Gastroscopy allows you to identify an ulcer, determine its size, the degree of scarring, to differentiate a peptic ulcer from a cancerous one (biopsy).

Symptoms of duodenal ulcer

Pain occurs in the epigastrium (often to the right of the midline),

pain radiates to the back, to the right hypochondrium, under the right shoulder blade. Pain of pressing, burning character. They are often very intense (cutting, penetrating), sometimes the pains are felt slightly (dull pains). During a painful attack, patients tend to take a forced position that alleviates inflammation (lay on their stomach or side, legs are pulled to the stomach). Depending on the food intake with a duodenal ulcer (as opposed to a stomach ulcer), pains are late (1.5-4

hours after eating), nocturnal, and also hungry. Typically, the pain subsides after taking sodium bicarbonate (baking soda) and other antacids.

Dyspepsia:

1) heartburn - the most common manifestation of dyspepsia in duodenal ulcers. Like pain, heartburn occurs rhythmically during the day: on an empty stomach, in a certain connection with food intake, in night hours;

2) vomiting occurs at the height of pain, i.e. on an empty stomach, does not contain

food impurities, brings relief. There is often a period of increased salivation before vomiting;

3) belching with sour or air, nausea;

4) constipation;

5) appetite is often not reduced, sometimes patients note "wolf hunger", which is associated with a tendency to develop hypoglycemia.

Examination of the patient. There may be signs of a disorder of the autonomic nervous system - cyanotic, wet palms, red or white dermographism, increased sweating, etc. Examination of the oral cavity: excessive salivation is often noted, the tongue is wet, sometimes covered with white bloom.

Inspection of the abdomen: Pigmentation may be found on the skin of the abdomen from prolonged use of a heating pad.

Palpation of the abdomen. For superficial and deep palpation local pain in the epigastrium is determined (to the right of middle line). Often there is local pain on percussion in the epigastrium (Mendel's symptom).

Analysis of gastric juice. Characteristics of duodenal ulcers high levels of secretion. The separation of gastric juice only abundant, but also continuous during the day. secretion in the interdigestive period (basal, nocturnal) often reaches a significant value.

Acid and pepsinogen excretion is also increased. A significant release of hydrochloric acid in the interdigestive period indicates a duodenal ulcer.

X-ray examination. The ulcerative niche (defect) of the wall of the duodenum is revealed. Mucosal folds usually converge to a scarring ulcer (a symptom of fold convergence).

Indirect (functional) features include the presence of a large amount of fluid in the stomach on an empty stomach (fasting hypersecretion) and various manifestations of duodenal dyskinesia.

Gastroscopy with biopsy reveals an ulcer, with a biopsy detect Helicobacter pylori, determine the size of the ulcer, the degree of scarring

5. Pyloric stenosis syndrome

Definition. This is a set of symptoms that characterizes narrowing of the pylorus, caused by various reasons.

The most common pyloric stenosis occurs in the complicated course of the ulcer of the pyloroduodenal zone; narrowing of the pylorus may be caused by the localization of the tumor in the wall of the stomach in the corresponding section; congenital narrowing of the pylorus is sometimes diagnosed.

Recurrent exacerbations of pyloric ulcer lead to a cicatricial (fibrous) change in the wall of the outlet section of the stomach, the walls become denser, shortened, deformed, the lumen narrows - the emptying of the stomach is disturbed. In the development of the clinical picture, the stages of compensated (1st stage), subcompensated (2nd) and decompensated (3rd) stenosis are distinguished.

Symptomatology.

Pain - characterized by pressing, dull pain. Often patients characterize these sensations as a feeling of fullness in the epigastrium, fullness. Pain appears after eating (in stage 1), in the stage of decompensation, the pain becomes permanent and disappears only after gastric lavage or vomiting.

Dyspepsia:

1) in the stage of compensation, burping with food and air is most characteristic;

2) to the stage of subcompensation and decompensation after each eating appears vomiting of food eaten (subcompensation) or eaten the day before (decompensation); 3) an unpleasant putrid smell from the mouth due to incomplete emptying of the stomach.

Examination of the patient. Even in the stage of compensation, the general state of health quickly deteriorates, especially in the evening - patients quickly lose weight, become lethargic, convulsions (decompensation) appear due to the loss of chlorides with vomit. The skin is pale due to the addition of anemia, dry (avitaminosis), brittle nails, sparse hair.

Examination of the abdomen. Peristaltic wave-like contractions of the stomach are determined; the contours of the distended stomach sometimes clearly show through the thinned anterior abdominal wall.

Percussion and palpation of the abdomen. Diffuse soreness in the epigastrium is determined, a constant symptom of "splashing noise" in the stomach on an empty stomach. Urinalysis - the amount of urine decreases to 500 ml per day (oliguria).

Blood tests. There is a thickening of the blood: in such an indicator as hematocrit (H t), the numerator increases, indicating an increase in the dense part of the blood (normally Ht 32-68); the number of blood cells in the general blood test increases. Blood chlorides rise.

X-ray study. There is a decrease in tone and expansion of the stomach, strengthening (compensation) or weakening (decompensation) of peristalsis. The stomach on an empty stomach contains a large amount of liquid and food debris, emptying is slow (more than 12 hours).

Gastroscopy. The distended stomach is determined, the contents in stomach on an empty stomach, the impossibility of advancing the gastroscope through the narrowed pylorus into the duodenum

6. ENTERITIS SYNDROME

Definition. This is a set of symptoms that characterize inflammatory or dystrophic lesion of the wall of the thin intestines.

The reason may be:

1) nutritional disorders: irregular meals, alcoholism,

2) food allergy;

3) giardiasis, helminthic colonization of the intestine;

4) chronic poisoning with some toxic chemicals - compounds of lead, mercury, phosphorus, arsenic, etc.;

5) radiation injury;

6) congenital enzymopathies.

Possible 4 mechanisms for the development of enteritis syndrome:

1) direct chronic damaging effect on the wall of the small intestine (toxic, irritating, etc.);

2) immunological mechanisms - the occurrence of hypersensitivity to the products of hydrolysis of nutrients or to the decay products of bacterial cells;

3) violation of the protective mechanisms of the intestinal mucosa;

4) dysbacteriosis - that is, the small intestine is populated by various microorganisms that are not typical for it. As a result aggravated indigestion, and some toxic substances secreted by microorganisms and formed as a result of the breakdown of food products by microbial enzymes have a damaging effect on the intestinal wall.

Symptomatology

Pain - does not always occur, if there are, then they are dull or spastic in nature, localized in the umbilical region.

Enteral dyspepsia is the main manifestation of enteritis: a race of wounds, bloating, rumbling, transfusion in the abdomen.

Enteritic scatological manifestations: frequent stools (up to 15-20 once a day), mushy, with undigested residues food, but no mucus, often offensive, copious, vesiculating gas. The amount of feces per day reaches 1.5-2 kg (polyfecal matter). Sometimes there is a sharp urge to defecate after eating, then there is a sharp weakness, accompanied by cold sweat, trembling hands.

Coprogram: microscopic examination of feces reveals the remains of undigested food (lenteria, drops of neutral fat, crystals of fatty acids and insoluble soaps (steatorrhea), muscle fibers (creatorrhoea), free extracellular starch (amilorrhea), a large amount of mucus evenly mixed with feces.

Manifestations of insufficiency of absorption: with mild manifestations of enteritis, the general condition does not suffer, the weight is stable. In severe cases, there is a decrease in body weight up to cachexia, general weakness, malaise, decreased performance, development of iron deficiency or B 12 deficiency anemia, brittle nails, hair loss, angular stomatitis (jamming), convulsions as a result of calcium absorption disorders. Due to insufficiency of absorption, dysfunction of the endocrine glands develops: pituitary insufficiency with the development of diabetes insipidus (polydipsia, polyuria); adrenal insufficiency with the phenomena of addisonism (skin hyperpigmentation, arterial and muscular hypotension); violation of the function of the gonads, manifested in men by impotence, in women - amenorrhea.

Abdominal palpation data. Soreness is determined in the umbilical region, to the left and above the navel - a symptom of Porges.

Sometimes there is a strong rumbling and splashing on palpation of the caecum due to the rapid passage of chyme through the small intestine and the entry of undigested, unabsorbed contents and intestinal gas into the caecum (Obraztsov's symptom)

Complete blood count - iron deficiency or non-deficiency anemia or anemia of mixed origin, inflammatory changes are detected. Biochemical studies - a decrease in the amount of plasma protein (hypoproteinemia), a decrease in the amount of calcium, plasma iron.

X-ray data: accelerated passage suspension of barium sulfate in the small intestine. Can be determined thickened edematous folds of the mucous membrane, in severe cases - their smoothing due to the process of atrophy. Endoscopic research methods - duodenoscopy with biopsy, jejunoscopy with biopsy, examination of the terminal part of the small intestine during colonoscopy. A relatively rare peculiar form of enteritis is regional enteritis (Crohn's disease), which occurs most often with a predominant progressive lesion of the ileum, fever, hyper α and γ-globulinemia.

7. Colitis Syndrome

Definition. This is a set of symptoms that characterizes inflammatory lesion of the colon wall.

Damage to the wall of the colon can be with:

1) penetration into the intestine of pathogenic bacterial flora (Shigella, Salmonella, Mycobacterium tuberculosis, Gonococcus, Treponema pallidum, etc.)

2) maintenance of inflammation in the wall by opportunistic and saprophytic flora;

3) contamination of the intestine with pathogenic fungi, amoebas, balantidia, helminths;

4) gross malnutrition;

5) concomitant damage to the large intestine in gastritis with reduced secretory function, pancreatitis;

6) toxic damage to the colon

7) intestines (mercury, lead);

8) allergic lesions;

9) prolonged irritation of the colon during coprostasis and abuse of enemas, laxatives;

10) long-term use of antibiotics for the treatment of various

diseases, which leads to the development of intestinal dysbacteriosis.

The main causes of colitis syndrome:

1) direct long-term irritation and damaging the effect of various mechanical and toxic factors on the wall of the large intestine;

2) a decrease in immunogenesis, which gives the possibility of pathogenic

infections persist in the intestine;

3) it is possible that autoantibodies are produced and the epithelium of the intestinal wall modified under the influence of various factors ("autoaggression").

Symptomatology.

Stool disorder: diarrhea from 2-3 times to 10-15 times or more per day or constipation. Often there is an alternation of constipation and diarrhea. A symptom of insufficient bowel emptying is characteristic: the release of a small amount of mushy or fatty feces with mucus and a feeling of incomplete emptying of the intestine. There are false urges to defecate (tenesmus), accompanied by the passage of gas and individual lumps of mucus, feces. Sometimes the feces have a fragmented appearance - "sheep" feces (with spastic colitis of the distal colon), with atony of the large intestine, the feces are enlarged in diameter, dense.

Pain - usually dull, localized in the lateral and lower abdomen, worse after eating and before defecation. Sometimes the pains are spastic in nature (increasingly decreasing), the attack of such pains is accompanied by the discharge of flatus, the urge to defecate. Dyspepsia. The most characteristic swelling hiccups (flatulence). Its cause is a violation of the digestion of food in the small intestine and dysbacteriosis. Sometimes nausea, anorexia, rumbling in the stomach are disturbing.

The general condition usually suffers little if there are no phenomena dehydration due to severe diarrhea and colitis is not accompanied by enteritis. In severe colitis, the absorption of proteins, vitamins, iron is disturbed due to the addition of damage to the small intestine, a violation of absorption develops (see enteritis syndrome).

Abdominal palpation data. For superficial and deep palpation reveals local pain along the way of the large intestine, painful, rumbling, indurated sections of the intestine are palpated, there may be an alternation of spastic contracted and dilated sections of the intestine. Coprological examination - a large number of inflammatory elements, mucus, leukocytes are determined; often a large amount of iodophilic flora, digestible fiber and intracellular starch, as well as erythrocytes (with erosive and ulcerative forms) are determined.

Endoscopic research methods (sigmoidoscopy, colonoscopy) - allows you to assess the condition of the mucous membrane (hyperemia, atrophy), identify erosions, ulcers, polyps. During endoscopy, it is important to biopsy the mucosa for morphological and bacteriological examination.

X-ray examination (irrigoscopy). Often only functional changes are found - acceleration or deceleration of intestinal motility, increased haustration, atony of the wall, etc. In severe lesions, an altered mucosal relief is determined due to inflammatory edema of the mucosa; there may be areas of cicatricial-inflammatory narrowing of the intestinal lumen.

General blood analysis. There may be manifestations of anemia. With exacerbation, there is leukocytosis, an increase in ESR.

A special form of colitis stands out - nonspecific (i.e., not having a single etiological factor) ulcerative colitis.

The most characteristic clinical sign is diarrhea, tenesmus, significant admixtures of mucus and blood in the feces. At endoscopy revealed ulcers in the wall of the colon with signs of bleeding. Between ulcers, polypous and pseudopolypous mucosal overgrowth.

8. PARENCHYMATOUS JAUNDICE SYNDROME

Definition. Jaundice is a yellow discoloration of the skin, sclera, mucous membranes and other tissues of the body as a result of excessive accumulation of bile pigments in the blood. Parenchymal (hepatic) jaundice develops as a result of damage to the cells of the liver parenchyma (hepatocytes).

The reasons. Viral, bacterial, toxic liver damage (hepatitis), liver cirrhosis, liver tumors, acute liver dystrophy when exposed to mushroom poisons, drugs, phosphorus compounds, arsenic, etc.

As a result of various reasons, damage develops liver parenchyma cells (hepatocytes), the ability of which to capture bilirubin from the blood, bind with glucuronic acid and release it into the bile ducts in the form of bilirubin glucuronide (bound - direct bilirubin) is impaired. In the blood serum, the content of direct (bound) and indirect (unbound) bilirubin increases; direct - due to the reverse diffusion of bilirubinglucuronide from the bile into the blood capillaries during degeneration of liver cells; the second - due to insufficiently active function of hepatocytes. Bilirubin appears in the urine (bilirubin glucuronide is water-soluble and passes through capillary membranes) and bile acids, the amount of which gradually increases. Fecal excretion of stercobilinogen is reduced because less bilirubin is excreted by the liver into the intestines, but complete discoloration is rare.

Symptomatology.

Pain - not often noted; if there is, then blunt, bursting, not related to eating. They are associated with rapid increase in the size of the liver.

Dyspepsia. Decreased appetite, may be nausea, vomiting due to disturbances in the emulsification of fats by bile.

General inspection. The skin has a typical saffron-yellow color with a reddish tint. Sometimes on the skin you can find traces of scratching due to the presence of pruritus, but it is much less common than with obstructive jaundice.

With severe damage to the liver parenchyma, there may be a hemorrhagic rash on the skin, palmar erythema, bright red color of the mucous membranes, lips, blush on the cheeks.

Palpation of the abdomen. Painful, indurated edge of the liver. The size of the liver is enlarged. There may be an enlargement of the spleen. The gallbladder is painless, not palpable.

Biochemical indicators. The content of the direct (related) and indirect (unbound bilirubin) is significantly increased.

Significantly changed liver function tests (thymol, bromsulfaleic, elevated levels of cytolytic enzymes (ALAT, AST). The alkaline phosphatase level be slightly elevated. The feces are light because the amount of stercobilin in the feces decreases. The color of the urine is dark due to bilirubinglucuronide (urobilinuria) that has entered the urine. The amount of a 2- and y-glooulins in the blood increases, which indicates an inflammatory process in the body. Acute viral hepatitis is characterized by an increase in the level of the enzyme aldolase

9. SYNDROME OF MECHANICAL JAUNDICE

Definition. Subhepatic (mechanical) jaundice is jaundice, which appears when there are current obstructions bile from the bile ducts into the duodenum.

Causes: with blockage of the common bile duct (stone, cancer, parasites) in the presence of stones, tumors of the pancreas, tumors of the major duodenal papilla, with cicatricial narrowing of the common bile duct, sphincter of Oddi.

The main causes of obstructive jaundice syndrome.

As a result of the presence of an obstruction to the flow of bile from the bile ducts

in them, the interlobular bile capillaries are stretched and bile diffuses into the liver cells, in which dystrophic processes develop. Bile enters the lymphatic spaces and into the blood. In addition, on the periphery of the lobules, as a result of an increase in pressure inside the small bile capillaries, a message appears between them and the lymphatic clefts, through which bile enters the general blood stream.

Symptomatology.

Pain in the right hypochondrium radiating to the back, shoulder blade, neck, arising after ingestion of fatty foods are characteristic of blockage of the bile ducts with a stone in cholelithiasis.

Dyspepsia - jaundice may be preceded by a period of nausea, repeated vomiting with an admixture of bile (with blockage of the ducts by a stone, tumor, etc.).

General inspection. The skin and mucous membranes turn yellow, and then, due to the oxidation of bilirubin to biliverdin, a green and dark olive color is noted. Traces scratching on the skin due to intense itching, which associated with irritation of skin receptors by bile acids that circulate in the blood (cholamia). Patients are lethargic, drowsy, inhibited, which is also associated with cholemia.

Palpation of the abdomen. Pain in the projection of the gallbladder, an enlarged gallbladder may be palpated (with cancer

pancreas, bile duct stone).

The cardiovascular system. Characterized by bradycardia due to

cholemia, which reflexively causes an increase in the tone of the vagus nerve.

Biochemical indicators. Content linked (direct) bilirubin increases more than 20.5 μmol/l. With prolonged jaundice due to impaired liver function, the content of indirect (free) bilirubin also slightly increases.

Bound bilirubin appears in the urine (bile pigments appear in the general urine test), giving it a brown color with a bright yellow yen. Feces become discolored periodically (with incomplete blockage by a stone) or for a long time (with compression of the duct by a tumor); stercobilin is absent in feces.

For long-term jaundice, can be changed liver function tests. Significantly increased levels alkaline phosphatase and y-glutamyl transpeptidase.

Ultrasound examination of the hepato-pancreatic region (ultrasound) A calculus (stone) is determined in a yellow bladder, enlargement of the gallbladder (dropsy, empyema) due to violation of the outflow of bile. An ultrasound can reveal a choledochal stone, induration and enlargement of the head of the pancreas.

Fibrogastroduodenoscopy: special attention should be paid on the area of the large duodenal papilla in the duodenum 12, where it is possible to detect inflammation with edema, which makes it difficult to outflow bile with symptoms of obstructive jaundice; can be identified calculus in the area of the sphincter of Oddi, tumor of the major duodenal papilla. X-ray examination of the gallbladder and biliary tract (cholecystocholangiography) reveals calculi, expansion of the diameter of the bile ducts above the site of obstruction by the stone.

10. PORTAL HYPERTENSION SYNDROME

Definition. This is a set of symptoms that characterize a condition in which there is a persistent increase in blood pressure in the portal vein.

According to the level of compression of the portal vein, portal hypertension is divided into suprahepatic (thrombosis of the hepatic veins - Budd-Chiari syndrome); hepatic (cirrhosis of the liver, rarely - hepatitis); subhepatic (compression at the gates of the liver by a tumor, enlarged lymph nodes). In liver cirrhosis, proliferation and subsequent scarring of the connective tissue at the site of dead liver cells lead to narrowing or complete obliteration of part of the hepatic sinusoids and intrahepatic vessels. As a result, an obstruction to blood flow is created, portal pressure rises, and the outflow of blood from the abdominal organs is disturbed. Under these conditions, the extravasation of fluid from the vascular bed into the abdominal cavity increases and ascites is formed. In the development of ascites in liver cirrhosis, a decrease in plasma oncotic pressure as a result of a decrease in albumin synthesis in the liver also plays a role. Specified factors create conditions for the transition of fluid from vessels to fabrics. This leads to irritation of the baroreceptors of blood vessels and active impulses to the regulatory centers of the brain.

Increased production of antidiuretic hormone exposure to increased water reabsorption in the kidneys. The adrenal glands produce aldosterone. Aldosterone and antidiuretic hormone are not sufficiently inactivated in the liver. All this leads to sodium and water retention in the body.

Edema develops and fluid accumulates in the abdominal cavity (ascites). For a long time, portal circulatory disorders can be compensated by the fact that blood from the portal vein can flow through the existing and normal anastomoses into the superior and inferior vena cava. In portal hypertension, these anastomoses are highly developed.

Symptomatology:

1) disclosure of porto-caval anastomoses in the region of the lower

thirds of the esophagus, stomach, in the area of hemorrhoidal veins, in

system of paraumbilical veins (caput meausae). Extended

Esophageal veins subjectively usually do not give sensations. At

traumatization with rough food, straining may develop

profuse bleeding, manifested by vomiting with an admixture

blood or tarry stools. Extended

veins in the rectal area are manifested by the clinic of hemorrhoids.

2) enlargement of the spleen (splenomegaly) does not cause pain

sensations, but in the enlarged spleen, antibodies to the cells of the hematopoietic system are intensively produced. In the general blood test, anemia, thrombocytopenia, leukopenia, the so-called hypersplenism, are detected.

3) there may be an increase in the liver (occurs not always due to portal hypertension syndrome),

4) an increase in the size of the abdomen due to ascites. Ascites is detected by percussion, a symptom of balloting is detected; when examining the abdomen, the form of a “frog abdomen” is revealed (with unstressed ascites); stretched out

umbilical ring and an umbilical hernia may form

with tense ascites. Often appear at the same time

swelling in the legs, sometimes a little later,

5) data of ultrasound examination of the abdominal organs: an enlarged spleen is detected, often an enlarged liver, an expansion of the portal vein diameter of more than 12 mm, fluid in the abdominal cavity is determined.

6) Dopplerogram of the portal vein: allows you to detect an increase in pressure in the portal vein

parts of the stomach.

8) sigmoidoscopy data: dilated, tortuous veins of the hemorrhoidal plexus are revealed.

9) complete blood count - a tendency to develop normochromic anemia, thrombocytopenia, leukopenia

11. LIVER INSUFFICIENCY SYNDROME

Definition. Liver failure (hepatargia) is a syndrome manifested by signs of liver dysfunction.

The reasons. Liver failure may occur withsevere forms of viral hepatitis, due to poisoning with hepatotropic poisons, with cirrhosis of the liver, tumors, etc.

The genesis of liver failure is based on two processes due to various reasons: severe dystrophy and widespread necrobiosis of hepatocytes, leading to a significant decrease in liver function. In the development of liver failure, the presence of significant collaterals between the systems of the portal and caval veins plays a role, as a result of which the participation of the liver in metabolism is sharply reduced and a significant part of the toxic products are absorbed in the

intestine, entering the systemic circulation bypassing the liver. A variety of manifestations of liver failure are combined by complex disorders of a large number of metabolic processes, a decrease in the antitoxic function of the liver, a disorder of bile formation and bile excretion.

The pathogenesis of hepatic coma is reduced to severe self-poisoning of the body due to the almost complete cessation of liver activity, especially the violation of its antitoxic function. Products of intestinal (bacterial) breakdown of protein, products of violation of protein metabolism, especially ammonia and phenols, which are not neutralized by the liver, lead to poisoning. Normally, most of the ammonia is captured by hepatocytes and, by being included in the ornithine cycle, is converted into urea, which is then excreted by the kidneys. Toxic phenols are inactivated in the liver by combining with glucuronic acid and sulfuric acid. Accumulate in the blood and other toxic substances, electrolyte metabolism is disturbed; occurs in severe cases hypokalemia, alkalosis. The accumulation of all these products leads to general intoxication, toxic damage to the brain and neuropsychiatric disorders.

12. PANCREATIC SYNDROME

Definition. This is a set of symptoms that characterize inflammatory (pancreatitis), tumor, cystic, and other lesions of the pancreas.

Causes of damage to the pancreas can be:

1) irregular diet, abuse of spicy and fatty food

2) chronic alcoholism, especially in combination with a deficiency

protein and vitamins in the diet;

3) when combined with pathology of the biliary system and

duodenum due to increased bile pressure in the common bile duct and bile reflux into the pancreatic ducts;

4) with severe manifestations of atherosclerosis, leading to

violation of the blood supply to the pancreas;

5) many infectious diseases - parotitis, abdominal and

typhus, viral hepatitis;

6) intoxication with mercury, lead, phosphorus, arsenic;

7) allergic conditions;

8) toxic effects of many drugs;

9) stones in the pancreatic ducts;

10) pancreatic cysts;

I) tumors.

The main pathogenetic factors:

1) delayed release and intraorganic activation of pancreatic enzymes, primarily trypsin and lipase, which gradually autolyze the gland parenchyma;

2) proliferation (reactive) of connective tissue, which then cicatricially shrinks, leading to sclerosis of the organ;

3) the processes of producing antibodies to the parenchyma of the pancreas, glands (autoaggression) are of great importance;

4) direct exposure to pathogens penetrating

through the blood or from the duodenum.

Symptomatology.

Pain is localized in the epigastric region on the right (with damage to the head of the gland), with damage to the body, pain is localized in the epigastrium to the left of the midline, with damage to the tail - in the left hypochondrium. Often, the pain radiates to the back and has a girdle character, spreading from the epigastric region to the left along the costal margin to the spine. Pain can radiate to the region of the heart, to the left shoulder blade, shoulder, to the left iliac region. Pain may be dull, constant, may appear soon after eating (especially fatty), may be paroxysmal.

Palpation. With the interest of the head, soreness with palpation of Desjardin's point (6 cm from the navel along the imaginary line from the navel to the right axillary fossa), can be palpated rounded formation in the Chauffard zone. On the left, the Mayo-Robson point is determined - in the left costovertebral angle.

Dyspepsia is expressed almost constantly. There is an increased salivation, nausea, belching, vomiting, flatulence, rumbling in stomach, tendency to diarrhea. In severe cases the so-called "pancreatogenic diarrhea" develops - copious stools, shiny, grayish, poorly washed off the toilet due to a large admixture of fat. Coprological research. Polyfecalia is characteristic, feces are shiny ("fatty"), ointment-like consistency. Microscopy reveals creatorrhea, amylorrhea, steatorrhea.

General inspection. In severe cases, rapidly increasing emaciation of patients, pale skin, manifestations of dehydration and vitamin deficiency (decrease in skin turgor, brittle nails, hair loss, angular cheilitis, polyneuritis, etc.) are characteristic.

Complete blood count: hypochromic anemia is often observed, leukocytosis during an exacerbation.

Biochemical study: an increase in the level of a-amylase in blood and diastase in the urine; in severe lesions of the pancreas due to the development of diabetes mellitus, the level of glucose in the plasma rises, glucose appears in the urine. With swelling of the head of the gland, a violation of the outflow of bile may occur, the content of direct bilirubin in the plasma will increase.

Duodenal sounding. It is carried out with the help of double-channel probe after stimulation of the pancreas glands with secretin and pancreozymin to determine the total amount of juice, its "bicarbonate" alkalinity, the content of trypsin, lipase and amylase.

X-ray of the duodenum. It is carried out in conditions of hypotension. Allows you to identify the deformation of the internal contour of its loop, due to an increase in the head of the pancreas.

Ultrasound examination (sonography). It allows you to determine the size of the gland, density, the presence of cysts and other signs of damage to the pancreas.

Radioisotope scanning of the pancreas. It allows you to evaluate the location, size, density, homogeneity of the body

SYNDROMES WITH KIDNEY DAMAGE

1 Nephritic syndrome

Definition:

A symptom complex that develops as a result of an immunoinflammatory lesion of both kidneys, mainly their glomerular apparatus.

Causes: Acute and chronic glomerulonephritis, diffuse connective tissue diseases, rheumatoid arthritis, systemic vasculitis, infective endocarditis, etc.

Mechanism of development: The glomeruli of the kidneys are gradually damaged

deferred immune complexes formed either in serum or directly in renal tissue. Thus, a chain of cellular and humoral inflammatory reactions is launched, affecting and disrupting the functions of predominantly glomeruli, as well as tubules and interstitium, which is accompanied by the development of edematous, urinary and hypertensive syndromes.

Complaints: swelling, headache, discoloration of urine.

Examination: Paleness and swelling of the face (facies nephritica). Edema - from pastosity of the face and eyelids to anasarca.

Increased blood pressure, mostly diastolic.

Pulse of good filling, tense.

Percussion: Left border of relative cardiac dullness shifted outwards.
Auscultation: emphasis 2 tones on the aorta.

Laboratory data.

In the blood - leukocytosis with a shift to the left, an increase in ESR.

In blood plasma: hypoalbuminemia, hypergammaglobulinemia.

Reberg-Tareev test: decrease in glomerular filtration. At decompensation of chronic glomerulonephritis - hyperazotemia (see renal failure).

Urine: in acute glomerulonephritis - oliguria, hyperstenuria

or relative density of urine at the upper limit of normal.

Macro- or microhematuria, proteinuria

In chronic glomerulonephritis - hypostenuria, hematuria, proteinuria. In the stage of compensation - polyuria, in the stage of de compensation - oliguria up to anuria.

Instrumental research:

With ultrasound and radioisotope scanning of the kidneys - symmetrical diffuse changes. Radioisotope renography: symmetrical decrease in secretory-excretory function.

ECG and echocardiography: signs of left ventricular hypertrophy.

The fundus of the eye: narrowing of the arterioles, the phenomenon of "cross", sometimes - swelling of the nipple of the optic nerve, hemorrhage.

In unclear cases, to identify the cause of nephritic syndrome and determining its variant, a kidney biopsy is performed.

Complications: cerebral hemorrhage, pulmonary edema with high

blood pressure, renal eclampsia (convulsions, loss of consciousness and blindness due to edema of the brain and retina, uremia (with oligo - and anuria).

2. NEPHROTIC SYNDROME

Definition: Clinical and laboratory symptom complex, including massive proteinuria, disorders of protein, lipid and water-salt metabolism, as well as edema that develops with various kidney lesions.

Causes: Glomerulonephritis, diabetic glomerulosclerosis, nephropathy of pregnant women, diffuse diseases of the connective tissues, systemic vasculitis, infective

endocarditis, renal amyloidosis, chronic suppurative diseases of various localization.

Mechanism: Damage to the glomeruli of the kidneys of an immunoinflammatory, metabolic, autoimmune nature (depending on the cause) contributes to the disruption of the electrostatic properties of the capillary loop.

The result is an increase in glomerular permeability to proteins - massive proteinuria, hypoalbuminemia, movement of fluid into tissues (edema), increased formation of lipoproteins.

Complaints: swelling, general weakness, dry mouth.

Examination: The skin is pale. Nails and hair brittle, dull. swelling, expressed in the face, feet, legs, further up to anasarca (ascites, hydrothorax, hydropericardium)

Palpation: The skin is dry. After pressure in the area of edema holes remain.

Laboratory data: In blood plasma: hypoproteinemia, dysproteinemia (hypoalbuminemia, increased α2-, β-globulins), hypercholesterolemia. Dyslipoproteinemia.

In the urine: Massive proteinuria (more than 3.5 g per day).

The relative density of urine is high, but may decrease with progression of renal failure. Cholesterol crystals. Hyaline, granular, waxy casts.

3. ACUTE RENAL INSUFFICIENCY SYNDROME

Definition: A symptom complex that develops as a result of acute violation of the main renal functions and characterized by azotemia, disturbances in the water-electrolyte and acid-base state.

Causes: Any variant of shock, poisoning with exogenous poisons, acute infection, impaired renal blood flow (vascular obstruction), a violation of the outflow of urine from the kidneys (urological obstruction).

Mechanism: Depending on the cause of renal injury blood flow, glomerular filtration, tubular secretion and tubular reabsorption - the corresponding violation kidney function - accumulation of nitrogenous waste – intoxication central nervous system.

Complaints: general weakness, drowsiness, nausea, vomiting, headache pain; a sharp decrease in urine output, which later, with a favorable outcome, is replaced by polyuria.

Inspection: Tongue dry, lined with brown coating. The patient is retarded.

Shortness of breath up to Kussmaul breathing.

Palpation: The skin is dry, flaky.

In blood plasma: Increased content of creatinine and urea (hyperazotemia). Hyperkalemia, hyponatremia, hypocalcemia, hyperphosphatemia. Decreased pH (acidosis). Reberg Tareev's test - glomerular filtration and reabsorption are sharply reduced.

In the urine: Decreased relative density of urine, proteinuria,

hematuria, cylindruria

Radioisotope renography - a sharp violation of all indicators up to the nonfunctional curve

4. CHRONIC RENAL INSUFFICIENCY SYNDROME

Definition: Symptom complex due to a sharp

a decrease in the number and function of nephrons, which leads to a violation of the excretory and endocrine function of the kidneys, a disorder of all types of metabolism, organ activity, and an acid-base state.

Causes: Glomerulonephritis, pyelonephritis, urolithiasis, kidney tumors, arterial hypertension, diffuse connective tissue diseases, diabetes mellitus, amyloidosis, polycystic kidney disease, etc.

Mechanism: A significant decrease in the mass of active nephrons; a progressive inability of the kidney to maintain water-electrolyte and osmotic homeostasis;

Complaints: General weakness, fatigue, polyuria. Subsequently, drowsiness, apathy increases, skin itching, shortness of breath, palpitations, bleeding of various localization appear. The terminal phase is joined by nausea, vomiting, diarrhea (uremic gastroenterocolitis).

Examination: The skin is pale, traces of scratching, subcutaneous

hemorrhages. Tongue dry, brown coating. Shortness of breath

to Kussmaul's breath.

Palpation: The skin is dry, flaky. Skin turgor is reduced.

The pulse is tense, quickened, in the terminal phase slowdown

pulse, arrhythmia.

Increase in blood pressure.

Percussion: In the terminal phase, expansion of the boundaries is possible

relative cardiac dullness (effusion pericarditis) or

isolated left border of relative dullness of the heart (arterial hypertension), dullness over the lower lobes

lungs (uremic pleurisy, pneumonia).

Auscultation: Muffled heart sounds, tachycardia; in the terminal phase of bradycardia, possible pericardial rub

(fibrinous pericarditis).

Laboratory data:

In the general blood test: normochromic anemia, thrombocytopenia, increased ESR.

In blood plasma: an increase in the content of creatinine and urea,

potassium, phosphorus, lowering pH, calcium. In the Reberg-Tareev test: a decrease in glomerular filtration and tubular reabsorption.

Urinalysis according to Zimnitsky: Polyuria, nocturia, hypoisostenuria. In the terminal phase of oliguria and anuria.

Radioisotope renography ~ decrease in excretory secretory function of the kidneys.

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Radioisotope renography - decrease in excretory secretory function of the kidneys.

SYNDROMES IN THE DAMAGE OF THE HEAT-MAITING SYSTEM

1. ANEMIA SYNDROME (GENERAL ANEMIC)

Definition: A symptom complex caused by a decrease in hemoglobin and erythrocytes per unit volume of blood with a normal or reduced volume of circulating blood.

Causes: Blood loss (acute and chronic). Violation of blood formation (deficiency or inability to use iron, vitamins (B12 and folic acid), hereditary or acquired (chemical, radiation, immune, tumor) damage to the bone marrow.Increased blood destruction (hemolysis).

Mechanism: Reduction of hemoglobin functioning in the body - hypoxia - compensatory activation of the sympathoadrenal, respiratory and circulatory systems.

Complaints: General weakness, dizziness, shortness of breath, palpitations, tinnitus.

Inspection. Paleness of the skin and mucous membranes. Dyspnea.

Palpation, pulse of weak filling, quickened, filiform.

Decreased blood pressure.

Percussion: Expansion of relative cardiac dullness to the left

(anemic myocardial dystrophy).

Auscultation. Heart sounds are muffled, quickened. Systolic murmur at the apex of the heart and large arteries.

Laboratory data:

In the general blood test: a decrease in the content of erythrocytes and hemoglobin, increased ESR. Depending on the etiology taking into account the color index, anemia can be hypochromic, normochromic, hyperchromic

2. SYNDROME OF TISSUE DEFICIENCY OF THE GLANDS

Definition: Combines the symptoms caused by a deficiency iron in tissues, excluding hematopoietic tissue.

Causes: Chronic blood loss, increased iron breakdown (pregnancy, lactation, growth period, chronic infections, tumors), iron absorption disorders (gastric resection, enteritis), iron transport.

Mechanism: Iron deficiency is a violation of the activity of numerous tissue iron-containing enzymes.

Complaints: Decreased appetite, difficulty in swallowing, taste perversion - addiction to chalk, lime, coal, etc.

Inspection: Smoothness of the papillae of the tongue. Dryness of mucous membranes. Dryness, brittle hair. striation, brittleness and change nail shapes. Cracks in the corners of the mouth.

Palpation: Dry skin, peeling.

Percussion: Expansion of relative cardiac dullness to the left.

Auscultation: Heart sounds are muffled, quickened.

Laboratory data: In the blood: Decreased serum iron levels, increased total serum iron-binding capacity.

In the general blood test: hypochromic anemia, microcytosis, anisocytosis, poikilocytosis.

Instrumental research.

Esophagogastrofibroscopy: atrophic gastritis.

Study of gastric juice: decrease in gastric secretion (basal and stimulated)

3. HEMOLYSIS SYNDROME

Definition: Symptom complex due to increased breakdown of erythrocytes.

Causes: Congenital diseases with a change in the shape of erythrocytes (microspherocytosis, thalassemia, sickle cell anemia); paroxysmal nocturnal hemoglobinuria, marching hemoglobinuria, poisoning with hemolytic poisons, heavy metals, organic acids; malaria; immune hemolytic anemia.

Mechanism:

a) increased breakdown of erythrocytes in spleen cells - an increase in the formation of indirect bilirubin,

b) the breakdown of erythrocytes inside the vessels - entry into the plasma

blood free hemoglobin and iron.

Complaints: Darkening of urine (permanent or paroxysmal),

pain in the left hypochondrium, chills, vomiting, fever, intense coloring of feces are possible.

Examination: Icteric staining of the skin and mucous membranes.

Palpation: Enlargement mainly of the spleen, to a lesser extent degree - liver.

Laboratory data:

In blood plasma: increased content of indirect bilirubin or free hemoglobin and iron.

In the blood: an increase in reticulocytes, pathological forms erythrocytes, decrease in osmotic stability of erythrocytes; normal color index.

In urine: increased content of stercobilin or hemosiderin.

To exclude the immune etiology of hemolysis are used

Coombs test and aggregate hemagglutination test (detection erythrocyte antibodies).

4. HEMORRHAGIC SYNDROME

Definition: Symptom complex, which is based on increased bleeding.

Causes: Thrombocytopenic purpura (immune origin,

or symptomatic thrombocytopenia with inhibition of bone marrow cell proliferation (aplastic anemia), with bone marrow replacement with tumor tissue (hemoblastosis, tumor metastases in the bone marrow), with increased consumption of platelets (DVC), with a lack of vitamin B 12 or folic acid); thrombocytopathy (more often hereditary dysfunction of platelets); hemophilia (hereditary) deficiency of 8, or 9, or I plasma clotting factors), acquired coagulopathy (deficiency of plasma factors coagulation in many infections, severe enteropathy, liver damage, malignant neoplasms); hemorrhagic vasculitis (immunoinflammatory vascular disease);

hereditary violation of the vascular wall of a separate localization (Randu-Osler telangiectasia), hemangiomas (vascular tumors).

Mechanism:

I. Reduction in the number of platelets or their functional inferiority;

I. Deficiency of coagulation factors in plasma (coagulopathy);

III. Damage to the vascular wall of an immune or infectious-toxic nature (vasopathy).

These 3 mechanisms correspond to 3 variants of hemorrhagic syndrome

	Thrombocytopenia and thrombocytopathy	Coagulopathy	Vasopathy
Complaints	Gingival, nasal, abdominal and uterine bleeding. Bleeding into the skin rubbing the skin arm, blood pressure measurement	profuse, spontaneous, post-traumatic and postoperative bleeding. Massive painful hemorrhages in joints, muscles, cells	Spontaneous hemorrhagic rashes on the skin, more often symmetrical. Possibly hematuria. Or persistent bleeding of 1-2 localizations (gastrointestinal, nasal, pulmonary)
Inspection and palpation	painless, relaxed superficial hemorrhages in	The affected joint is deformed, painful on	Skin rash in in the form of small seals,

	skin and mucous membranes, bruises, petechiae.	palpation. Contractures, muscle atrophy. Hematomas	symmetrical, then acquire purple appearance due to blood soaking. After the disappearance hemorrhage brown pigmentation persists for a long time
Laboratory data			
Bleeding time	Time Extended	norm	norm
Clotting time	norm	Time Extended	norm
Symptom of "twist", "pinch"	+	-	inconstant
Platelet count	lowered	norm	norm
Retraction of a blood clot	Weakened or missing	norm	norm
Thromboelastogram	hypocoagulation	hypocoagulation	norm
Activated (standardized) partial thromboplastin time	norm	enlarged	norm
Prothrombin level plasma	norm	decrease is possible	norm
activated recalcification time	enlarged	enlarged	norm
General analysis blood	Possible normochromic (acute posthemorrhagic) or hypochromic (chronic iron deficiency) anemia	Possible normochromic (acute posthemorrhagic) or hypochromic (chronic iron deficiency) anemia	Possible normochromic (acute to hemorrhagic) or hypochromic (chronic jelly-deficient) anemia. Possible leukocytosis, increased ESR.
General analysis urine: hematuria	Possible	Possible	Possible

SYNDROMES WITH DAMAGE TO THE ENDOCRINE SYSTEM

1. CHRONIC HYPERGLYCEMIA SYNDROME

Definition: Symptom complex due to excessive accumulation of glucose in the blood due to absolute or relative deficiency of insulin.

Causes: Diabetes mellitus type 1 and 2, diseases of the pancreas (pancreatitis, resection of the pancreas), damage to the islets of Langerhans with iron accumulation (hemochromatosis), increased hormonal activity of the thyroid gland, adrenal glands, anterior pituitary gland, long-term administration of certain drugs (corticosteroids) and chemicals (nitrosamines, cyclophosphamide).

Mechanism: Deficiency of insulin Cell starvation of carbohydrates Hyperglycemia Appearance of glucose in the urine due to excess of the renal threshold Increased osmotic diuresis Polyuria Dehydration of tissues Hypovolemia.

Complaints: Thirst, dry mouth, frequent copious urination, general weakness, skin itching, weight loss with increased appetite.

Inspection: Dry skin, there may be traces of scratching. Weight loss.

Often furunculosis. Dryness of visible mucous membranes.

Palpation: Decreased skin turgor. Reduced thickness of subcutaneous fat. Decreased muscle tone. The pulse is rapid, weak filling, thready.

Auscultation. Heart sounds are weakened, quickened.

Laboratory data: In blood plasma: elevated glucose levels and osmolarity.

In urine: increased daily diuresis with increased relative density of urine. Glucosuria.

2. KETOACIDOSIS SYNDROME

Definition: Symptom complex due to accumulation in the blood products of incomplete oxidation of fats - ketone bodies

Causes: Uncompensated diabetes mellitus, prolonged carbon-water starvation, indomitable vomiting, prolonged feverish state.

Mechanism: Carbohydrate deficiency compensatory use fats as energy substrates, the accumulation of ketone bodies in the blood, resulting in intoxication of the central nervous system.

Complaints: nausea, vomiting, general weakness.

Examination: The patient is lethargic, lethargic up to coma. Rubeosis of the cheeks.

Eyeballs are soft. Shortness of breath up to Kussmaul breathing.

The smell of acetone. Tachycardia. Decreased blood pressure.

Laboratory data: decreased pH, increased levels of ketone bodies, decreased levels of potassium.

In urine: positive acetone.

3. HYPOGLYCEMIA SYNDROME

Definition: A symptom complex caused by an insufficient level of glucose in the blood, leading, first of all, to a violation of higher nervous activity.

Causes: an overdose of hypoglycemic drugs in the treatment of diabetes mellitus, insulinoma, severe liver damage, panhypopituitarism, severe hypocorticism and hypothyroidism, prolonged carbohydrate starvation, prolonged diarrhea, fever, heavy physical work, alcohol intoxication, neuroses.

Mechanism: Imbalance of plasma insulin and glucose levels Hypoglycemia Carbohydrate starvation and cerebral hypoxia Compensatory sympathetic activation and release of catecholamines Cerebral edema.

Complaints: Feeling of hunger, headache, irritability, sweating, palpitations, dizziness, trembling.

Inspection. The patient is agitated, aggressive, as hypoglycemia increases, tonic convulsions appear, the extreme degree is hypoglycemic coma. Tremor. Integuments of the increased humidity, visible mucous membranes are wet.

Palpation: Skin turgor is preserved. Muscle tone is increased. Pulse good filling, speedy.

Laboratory data. The level of glucose in the blood plasma is reduced.

4. THYROTOXICOSIS SYNDROME

Definition: A symptom complex caused by hypersecretion of thyroid hormones by the thyroid gland and the development of toxicosis with damage to various organs and systems, primarily the cardiovascular and nervous.

Causes: Diffuse toxic goiter, toxic adenoma thyroid gland, autoimmune thyroiditis, thyroid cancer glands. Mechanism: Increased levels of thyroid hormones in blood - increased peripheral tissue effects of thyroid hormones.

Complaints: Weight loss with increased appetite, permanent palpitations and sweating, trembling, irritability, tearfulness, weakness, a feeling of heat. Possible subfebrile condition, diarrhea.

Review: weight loss. Exophthalmos. positive symptoms Graefe, Möbius, Stelwag, Marie. In severe cases, typical facies based.

Palpation: The skin is moist, elastic, velvety. The subcutaneous tissue is thinned. The thyroid gland is enlarged from 1 to 5 degrees, rarely not palpable. Possible enlargement of the liver (toxic hepatitis), pulse of good filling, frequent. Increase in systolic and pulse blood pressure.

Auscultation: Heart sounds are sonorous, tachycardia, possible systolic murmur at the apex, atrial fibrillation.

Laboratory and instrumental data:

In blood plasma: decreased cholesterol, increased content protein-bound iodine, triiodothyronine and thyroxine,

Increased uptake of radioactive I131 by the thyroid gland.

To identify the specific disease that caused thyrotoxicosis, use ultrasound and radioisotope scanning, puncture of the thyroid gland, determination of the titer

thyroid antibodies.

5. HYPOTHYROISIS SYNDROME

Definition: Symptom complex, which is based on insufficient secretion of thyroid hormones.

Causes: Congenital hypoplasia of the thyroid gland, autoimmune and subacute thyroiditis, thyroidectomy, endemic goiter, tumor or inflammation of the hypothalamic-pituitary region

Mechanism: Decrease in the level of thyroid hormones in the blood - peripheral tissue effects of thyroid hormone deficiency.

Complaints: Weakness, drowsiness, lethargy, constipation, chilliness, weight gain, hair loss, coarsening of the voice. Speech is slow.

Examination: The skin is scaly, pale. Overdevelopment subcutaneous fat. Dense (mucous) edema evenly throughout the body. Tongue enlargement. Movement is slow.

Palpation: Skin turgor is reduced. After pressure in the area edema fossa does not remain. The skin is cold to the touch, dry. The thyroid gland is not palpable or enlarged. Pulse rare, weak filling and tension.

Auscultation: Heart sounds are weakened, bradycardia. Trend to a decrease in blood pressure.

Instrumental and laboratory data:

• in the blood plasma the level of cholesterol is increased, the content of protein-bound iodine, triiodothyronine and thyroxine is reduced,

• reduced absorption of radioactive I131 by the thyroid gland.

To clarify the cause of hypothyroidism, the titer of plasma antibodies to the thyroid gland is examined, a puncture of the thyroid gland, ultrasound and radiography are performed.