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Faculty of surgery

DIFFERENTIAL DIAGNOSIS OF CRITICAL

SURGICAL DISEASES Part (I).

Tutorial for medical students, Pediatric,

medical-preventive and dental faculties

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Abstract:

In the educational-methodical manual for students represented the most important differential diagnosis of surgical diseases. It is the section differential diagnosis raises students ' difficulties in the preparation for practical classes in the Faculty of surgery. Scholastic-methodical allowance provides basic clinical data needed 4 course students of medical universities to prepare for practical classes in the Faculty of surgery and especially while working with clients sick. the topic is given theoretical reference, briefly describes the pathology and allowing to perform differential diagnosis with similar diseases clinic. Such methodical building tutorial will allow students to present more clearly the algorithm of thinking required for diagnosis. We believe that the information will be useful for students of medical universities. The bibliography contains the necessary sources, which will improve the quality of knowledge and enough to fully prepare for classes.

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**DISEASES OF THE THYROID GLAND**

**Theoretical reference**.

**Classification of diseases:**

1. Congenital (ectopia nezareatmene work-flow shhitovidnogo).

2. injuries (open, closed).

3. Vospalitnelnye disease (autoimmunity, strumity, XPonicheskie Autoimmunity).

4. hypothyroidism (cretinism, congenital purchased).

5. tumors (Carcinoma, Sarcoma).

6. Goiter (endemic, sporadic, epidemic, diffuse toxic).

**Goiter** this limited or diffuse thyroid enlargement.

**On the degree of increase is determined by:**

0 art. -Iron not palpated

(I) Calendar-Iron clearly palpated. Swallowing is not visible.

(II) Calendar-Iron is visible when swallowing, well palpable by palpation, but the shape of the neck is not changed.

(III) Church. -Iron increased due to both shares and the isthmus, is clearly visible, there has been a change in the contours of the neck (thick neck).

(IV) Church. -Craw clearly pronounced neck configuration significantly changed.

(V) Church. -Iron reaches huge dimensions (hanging a goiter).

**On the morphological form of increase are divided into:** diffuse, nodular mixed (diffusely-junction).

**On the function of secrete** : eutireoidnyi (with the normal function of the thyroid gland), gipotireoidnyj (with decreased function), gipertireoidnyj (with high function) and gipotireoidnyj with evidence of cretinism.

**On the severity of thyrotoxicosis allocate:** mild, moderate and severe forms.

**By stage of disease (s. Milk) allocate:** nevroticheskuju, nejrogormonalnuju, visceral and distroficheskuju.

**Endemic goiter-** strong increase in thyroid gland as a result of iodine deficiency. Occurs in people living in geographical areas, biosphere which is poor in iodine, while the incidence of adult population of goiter is more than 10%.

Hyperplasia of the thyroid tissue occurs as a result of increased TSH formulation in the pituitary gland in response to thyroid hormone deficiency.

The clinical picture is defined by morphological form, degree of increase in Gland, as well as its functional State. Often increase gland 3-4 degree, often hypothyroidism. The clinical picture eutireoidnyi goitre is very poor: complaints about the sense of awkwardness in the neck, difficulty in breathing, rarely change the voice. Respiratory failure is the most frequent symptom of zagrudinnogo goiter. When inspecting these patients can be noted the expansion of the veins of the neck. Breathing leads to development changes characterized as "zobnoe heart". Impaction of the sympathetic trunk is Horner's syndrome (PTOSIS, miosis, enophthalmos), modified half of the body sweating on the side of the compression. When thyroid palpation mild, often unevenly, movable. Ultrasound and radioisotope scan determine the increase in the thyroid gland.

X-ray study allows you to set a delay on the level of barium aberrant goiter, esophageal offset lateral or anterior direction.

The number of hormone gland in the blood of normal, elevated TSH level. There is also increasing levels of thyroglobulin levels in the blood. Thyrotoxicosis is developing relatively infrequently.

**Sporadic goiter** (struma sporadica), sporadic strumous disease non goiter is a disease characterized by an increase in the thyroid gland, usually without expressed functional organ function. Occurs in people living in areas where there is no the incidence of endemic goitre.

Etiology of sporadic goiter is associated with factors that could inhibit the synthesis or the use of thyroid hormones in the body, with the presence of congenital defects in iodine metabolism, with transport and utilization of thyroid hormones gland or the influence of medicinal and other strumogennyh factors. Additional etiological factors-gender, age, malnutrition, poor sanitary conditions, infections, helminthiasis, prolonged treatment with drugs of iodine and some congenital enzyme defects in the synthesis of thyroid hormones and adrenal glands.

The clinical picture of sporadic goiter does not differ from the clinical picture of endemic goiter.

**Epidemic goiter** occurs in people in poor social conditions (insufficient and poor-quality food, great population density, lack of sanitation, etc.) is characterized by the frequent development of thyrotoxicosis

**Diffuse toxic goiter (graves ' disease)** -autoimmune disease harkterizujushheesja diffuse increase in thyroid gland and hyperthyroidism. Develops due to develop autoantibodies, communicating with TSH receptors on the follicular cells of the thyroid gland, which leads to the development tireotoksicheskogo goiter.

The disease develops severe, often starting torque serves as a trauma. The clinical picture is determined by the hyperthyroidism.

Clinical stages of hyperthyroidism:

stage 1-early functional changes (tireotoksicheskaja neurasthenia); irritability, tearfulness, petulance, shine eye, jekzalftalm.

stage 2-later options. changes (light thyrotoxicosis) fever, agitation, sweating, rapid heartbeat, tremor of the hands, weight loss, diminution in the afternoon. The Exchange raised to 15% -30%.

3 stage-easily reversible organic changes (cf. the severity of hyperthyroidism); agitation, fever, tachycardia, permanent, persistent expansion of the heart, CH-1, weight reduction, reduced ability to work all day. Basal metabolic rate increased by 30% -60%.

4 stage later, slowly reversible organic changes (tireotoksicheskaja dystrophy); myocardiodystrophy with SN-2, degeneration of the liver, gepatomegalia, muscle weakness, loss of earning capacity.

5 degree (Terminal)-later irreversible organic changes (sclerosis): tireotoksicski myocardium sclerosis, ch-3, atrial fibrillation, cirrhosis, ascites, anasarca, hydrothorax.

The main symptoms are: diffuse thyroid enlargement, tachycardia, exophthalmos. Patients complain of general weakness, irritability, tearfulness, fast fatigability. Objectively stated voltage, smaller tremor of the fingers. Thyroid moderate density, moveable, painless. Define positive eye symptoms: Gräfe, Kocher, Delrimplja, Mobius, Shtelvaga. The uptake of radioactive iodine on 4, 6, and 12:00 am increased. When ULTRASOUND iron diffusely increased. The concentration of total T4, free T3 improved almost all patients.

Low serum TSH level. Is determined by the increase in titer of autoantibody citostimulirujushhih (80% of patients).

**Differential diagnosis.** Since leading the clinic toksichekogo goiter is an overactive thyroid (hyperthyroidism), it is necessary to differentiate from other forms of neurosis tireotoksicski neuroses, diseases such as tuberculosis, revmocardit and stomach cancer.

1) Thyrotoxicosis or tuberculosis?

The following symptoms are common to early stages of both zabolvanij: general weakness, eye gloss, sweating, rapid heartbeat, subfebrilnaya temperature, weight loss. About tireotoxicose say: irritability, tearfulness, light tremor of the fingers, tachycardia with little load, increasing basic metabolism, normal ERYTHROCYTE SEDIMENTATION RATE, absence of light fresh foci and infiltrative changes, negative tuberkulinovyh crop samples results of sputum.

2) Thyrotoxicosis or rheumatism?

Weakness, shortness of breath, pain in the heart, sweating, rapid heartbeat, subfebrilnaya temperature is observed in both diseases. However, for the more typical of hyperthyroidism: irritability, tearfulness, glitter eye and expansion of palpebral fissure, hand tremor, hypertrophy of the thyroid gland. All samples on active rheumatic process with tireotoxicose negative.

3) Thyrotoxicosis or cancer?

The similarity of symptoms-rapid weight loss, increasing weakness, gastrointestinal disorders often occur when stomach cancer, as well as with thyrotoxicosis. To avoid diagnostic errors, require careful evaluation of gastriticheskih complaints in patients with thyrotoxicosis and proper survey of patients.

**THYROID CANCER**

Thyroid cancercan occur in childhood and young adulthood, but more often after 40 years. Women have in 4 times more often than men. The most common form of differentiated thyroid cancer, which include papillary (62%) and follicular (18%) cancer. Undifferentiated forms (gigantokletochnyj, solid, small and large-cell carcinoma) are seen less frequently.

Histological classification of thyroid cancer:

-Follicular carcinoma: differentiated, accumulates iodine;

-papillary carcinoma: differentiated, accumulates iodine;

-Squamous Cell Carcinoma;

-undifferentiated (anaplastic) Carcinoma: fusiform, giant cell, melkokletochnaja;

-medullary carcinoma: from the calcitonin-producing p-cells;

-fibrosarcoma, other types of sarcomas;

-karcinosarkoma, malignant lymphoma.

The most benign period has papillary thyroid cancer. It usually manifests itself in the form of a single dense node in the thyroid gland, rarely watch multiple nodes; metastasises in cervical lymph nodes, less celebrated metastasized thyroid share second, extremely rare in the bones and lungs. Tumor growth is very slow. In the surrounding tissue grows late, in the presence of metastases in the lymph nodes of the neck, the last for a long time remained mobile. Jeutireodinoe is typically stored state.

Follicular cancer-dense, rounded shape node metastasises in the bones, lungs, rarely in the brain. Incremental cancer (aberrant) thyroid glands along the sterno-kljuchichno-soscevidnyh muscles, there is usually in young age, develops slowly. Has papilljarnoe or papilljarno-follicular structure.

**Classification** . There are four clinical stage, malignant tumors of the thyroid gland:

stage 1-a small tumor in one of the lobes of the gland without deformation or sprouting capsules gland, smeshhaema, without metastases;

2A phase-multiple tumors of the same size, there are no regional and distant metastases;

stage 2B-single or multiple tumors in the thyroid gland without sprouting capsule and without limitation smeshhaemosti if there smeshhaemyh regional metastasis on the affected side of the neck and in the absence of distant metastases.

3A-stage tumor spread outside the gland capsules and is linked to surrounding tissue or squeezes adjacent organs, malopodvizhna, but in the absence of distant metastases.

3B-tumor stage 1, 2, 3A, but that bilateral smeshhaemyh metastases in neck but in the absence of distant metastases.

stage 4-fixed or flexible swelling, germinating in the surrounding tissue with distant metastasis.

**International Classification:**

  **T0** — the primary tumor tissue in thyroid operation not found

  **T1** -tumor 2 cm or less in greatest dimension within the thyroid gland

  Can sometimes be used supplement:   
T1a-tumor 1 cm or less   
T1b-tumor more than 1 cm, but not more than 2 cm

  **T2** -tumor more than 2 cm but less than 4 cm in greatest dimension within the thyroid gland (i.e. not germinating in the capsule of the gland)

  **T3** -tumor more than 4 cm in greatest dimension within the thyroid or any tumor with minimal spread beyond the thyroid capsule (e.g., germination in short muscle or fatty tissue around.) so, even small tumors of the thyroid gland, sprouting in its capsule, stadirujutsja as T3

  **T4** — tumor in this stage can be divided into two podstadii:

  **T4a** -tumor of any size, germinating capsule of thyroid gland with invasion in subcutaneous soft tissue, larynx, trachea, esophagus or laryngeal nerve

  **T4b** is swelling, germinating predpozvonochnuju fascia, carotid artery or zagrudinne vessels.

  It is important to note that all nedifferncirovannye Carcinoma of the thyroid gland are classified as stage T4, irrespective of their size. For these carcinoma staging is slightly different:

  **T4a** — nedifferenirovannaja Carcinoma, located within the thyroid gland is surgically rezektabelnaja (i.e. completely removed during surgery)

  **T4b** -undifferentiated carcinoma spreading beyond the thyroid gland is surgically nerezektabelnaja (i.e. completely surgically neudalimaja)

    
**The presence of metastases in regional lymph nodes of the neck**

  **NX** — regional metastasis cannot be assessed

  **N0** -lack of regional metastases

  **N1** -regional metastases

  **N1a** -metastases in lymph drainage zone VI (paratracheal, pretracheal and prelaringealnye lymph nodes)

  **N1b** -metastases in lateral cervical lymph nodes on one or both sides, from the opposite side, or in lymph nodes zagrudinne

  **Distant metastases**

  **MX** is the presence of distant metastasis cannot be assessed

  **M0** -no distant metastasis

  **M1** — presence of distant metastases

Method of diagnosis of thyroid cancer is its radioisotope research. Radioactive iodine does not accumulate or weakly accumulates in the tissues of the tumor. ULTRASOUND has diagnostic value in the study of education in the thyroid gland, exceeding the value of 0.5 cm. Puncture site for cytological study increases the accuracy of diagnosis.

Undifferentiated cancer forms when radioimmunologicheskim method is detected change in level of calcitonin. Computed tomography. Definitive diagnosis is based on histological examination.

**Differential diagnosis.**

Thyroid cancer should differentiate with various benign entities;

1) Nodular goiter, adenoma-for them is characterized by clear, rounded shape, smooth contours, elastic consistency. However, these signs may be in intrakapsuljarnom cancer.

1) Chronic thyroiditis Hashimoto-short history, increasingly sick women menopausal, diffuse lesion of the prostate gland in the form of a butterfly, hypothyroidism, tight elasticized Struma without peritireoidita phenomena, moderate Lymphocytosis and a high titer of antibodies to thyroglobulin.

2) Chronic thyroiditis Riedel-1/3 sick men, defeated the unilateral, node in the gland tight, rapidly peritereoidit and Myositis of the cervical muscles, restricted mobility, sprouting with trachea-all this simulates cancer the thyroid gland.

The most reliable diagnostic methods are: ultrasound, CT scan, biopsy.

**AUTOIMMUNITY**

**Autoimmune Thyroiditis (Hashimoto's Thyroiditis)**. At the heart of the disease lies education autoantibodies to thyroglobulin and thyroid tissues.

Slow growth, more or less dense goiter, weakness, gradually develops hypothyroidism symptoms associated with the squeezing of the surrounding tissues. Goiter with surrounding tissue does not heat, its surface is smooth, but sometimes rough. Lymph nodes of the neck is not increased. It is not uncommon to find abnormal clinical forms of the disease: there is light it is possible in the future, thyrotoxicosis, a unilateral process of type hub netoksicski goitre.

In the diagnosis of autoimmune goiter discovery play a role of increasing levels of gamma-globulin, tesselation Xerography, a positive reaction on prednizolonovuju trial-a decrease in the size of goiter.

**Thyroiditis chronic fibrous (goiter Riedel).** In the thyroid gland develops chronic productive process with the growth of connective tissue lymphoid infiltrates, often with a dash of eosinophils.

Clinically detected a gradual increase and compaction thyroid gland rarely one of its share of the surface of the gland is uneven, goiter is very tight-"iron", in advanced cases, it welds with the surrounding tissue, the skin over the movable neck lymph nodes, goiter is not increased. Palpation is painless, but sometimes Notes irradiirujushhaja pain in the ear, hoarseness of the voice, violation of swallowing and dry cough that is associated with involvement in process of recurrent laryngeal nerve, the esophagus and the trachea.

**Acute purulent Thyroiditis** develops when hit by infection in the thyroid gland by hematogenic osteomyelitis.

The disease begins acutely. Concerned about the spontaneous, sharp pain in the neck, increasing tenderness, speaking and swallowing. Chills, fever, painful at first dense, and then fluctuating a tumor on his neck. Weakness, sweating, tachycardia. Leukocytosis, elevated ESR. Accumulation of iodine-131 in the normal or reduced.

Tireodit subacute. Refers to viral diseases. Disease often precede flu, measles, flu.

In the area of the anterior surface of the neck pains, spreading to the lower jaw, ears, back of the neck. Notes: general weakness, sweating, fever and shivering. Iron heavy, intense and sharply painful. In the analysis of blood is determined by increased SEDIMENTATION RATE.

**Differential diagnosis**.

Different types of Autoimmunity should be differentiated with diffuse toxic goiter. A common symptom is diffuse thyroid enlargement. In contrast, if diffuse thyroiditis toxic goiter thyroid by palpation is painless. Identifies signs of thyrotoxicosis, positive symptoms, Delrimplja Graefe, Shtelvaga, Mobius. In the analysis of blood missing signs of inflammatory response. Radioactive iodine uptake is promoted, and the level of T4 and T3 upgraded almost all patients.

The key forms of endemic and sporadic goiter should be differentiated thyroid cancer. Thyroid cancer is determined by a dense, bugristoe education, relatively fast growing. In the later stages it malopodvizhnoe, spajano with the surrounding tissue. Popping pain spreading to shoulders, ear or back of the neck, difficulty in swallowing, voice alteration, expansion of subcutaneous veins of the neck, face, chest, Horner's syndrome. Metastases occur in the lymph nodes of the neck, lungs, mediastinum and bones. When scanning identifies cold sites. ULTRASOUND detects signs of malignancy in the amount of more than 5 mm. B. puncture biopsy allows a cytological study.

Unlike the thyroid cancer endemic and sporadic Zoba administration are not accompanied by the defeat of the regional lymph nodes. The key forms of these diseases when scanning accumulate radioactive iodine-hot pockets.

**Test questions**:

1. Causes leading to development of endemic goiter.

2. Causes leading to the development of chronic thyroiditis.

3. The clinical picture of diffuse toxic goiter.

4. The clinical picture of thyroid cancer.

5. Instrumental and laboratory diagnostic methods applied in thyroid diseases.

6. Instrumental Diagnostics Methods used in thyroid cancer.

**Tests for self-control**:

In a patient with endemic goiter visible increased right share of the thyroid gland. Specify the magnification of the thyroid gland, which corresponds to this clinical picture: 3

1. the level 1

2. degree 2

3. degree 3

4. degree 4

5. degree of 5

For endemic goiter when scanning characteristic identifying pockets of increased accumulation of isotope iodine-131: 2

1. Yes

2. No

Positive eye symptoms of Delrimplja, Shtelvaga, Graefe, Mobius is characteristic for: 3

1. hypothyroidism

2. goiter (II) degree

3. hyperthyroidism

4. cancer of the thyroid

Basic laboratory characteristics of diffuse toxic goiter are: 3

1.reduction of calcium in the blood

2.increase of TSH and T3

3.increase of T4 and T3

4.decrease in T4 and T3

The appearance of osiplostand vote in zobe Riedel explains: 4

1. involvement of the facial nerve

2. involvement of the trachea and esophagus

3. the presence of metastases

4. involvement of the recurrent laryngeal nerves

What are the symptoms of chronic fibrous Thyroiditis: 1, 2, 4

1. by palpation iron tight

2. the skin over the goiter mobile

3. lymph nodes of the neck enlarged, sedentary

4. notes the hoarseness of voice

**MASTITIS**

**Theoretical reference.**

Mastitis is a acute inflammation of the breast tissue.

**Classification of mastitis:**

**On clinical flow:** acute chronic.

**On the functional State of the breast;** laktacionnyj, nelaktacionnyj.

**Upon seeing the targeted structures gland** : parenchymal, interstitial, galaktoforit, areolit.

**Localization:** Antemammarnyj (premammarnyj) subareoljarnyj; intramammarnyj; retromammarnyj; total defeat.

**By the nature of inflammation:**

Serous (starting). Infiltrative.

Purulent: infiltrative-purulent: diffuse, nodular;

abscedirujushhij: Furunculosis areola areola, abscess, abscess inside the gland abscess behind the gland (retromammarnyj); flegmonoznyj, Pyo-necrotic; gangrenous.

by the nature of inflammation are distinguished: negnanye (serous and infiltrative) and fatigue (abscedirujushhij, infiltrative-abscedirujushhij, flegmonoznyj and gangrenous) forms of acute laktacionnogo mastitis.

Depending on the location of the epicenter inflammation mastitis happens: subcutaneous, subareoljarnym, intramammarnym, retromammarnym and total, when affects all divisions of the breast.

**The clinical picture** . For a typical clinical presentation of acute serous mastitis is characterized by acute onset, usually on 2-4 week post-natal period. Body temperature quickly rises to 38-39° c, often accompanied by chills.

Develops general weakness, weakness, headache. There is pain in the breast, but there may be variants of clinical evolution of mastitis, in which common phenomena precede local. When inadequate therapy beginning on mastitis during the 2-3 days goes into infiltrativnuju form. In the mammary gland palpated pretty tight, painful infiltration. The skin over the infiltration is always swollen. Transition in the form of a purulent mastitis occurs within 2-4 days. The temperature rises up to 39° c; appear shivers; growing signs of intoxication: lethargy, weakness, poor appetite, headache. Picking up local signs of inflammation: the swelling and soreness in the defeat, softening in the area of infiltration with infiltrative-purulent form of mastitis.

20% of patients have purulent mastitis manifests itself in the form of abscedirujushhej form. While the predominant options are furunculosis and abscess areola, rarely meet intramammarnyj and retromammarnyj abscesses, constituting a cavity, limited a connective tissue capsule.

10-15% of patients have purulent mastitis is leaking like a flegmonoznaja form. The process captures a large portion of the gland with the fusion of its fabric and the surrounding tissue and skin. Overall status of new mothers in such cases is critical. The temperature reaches 40° c, watch stunning shivers expressed intoxication. Mammary gland dramatically increases in volume, swollen skin, flushed with sinjushnym shade, palpation gland dramatically painful. Flegmonoznyj mastitis can be followed by septic shock.

A rare form of gangrenoznaya mastitis is extremely severe with sharply expressed intoxication and necrosis of the breast. Outcome of gangrenous mastitis is unfavourable.

When any form of mastitis in the best interests of the child, breastfeeding should be discontinued. Indication for suppressing lactation when mastitis include:

-rapidly progressive process, despite the intensive therapy;

-infiltrative mandochagovy-purulent and abscedirujushhij mastit;

-flegmonoznaja and gangrenoznaya forms of mastitis;

-any form of mastitis with recurring throughout;

-latent mastitis, not amenable to combination therapy, including surgical dissection.

**Differential diagnosis**.

Differentiate acute purulent mastitis should be breast cancer, lactostasis, breast fibroadenomatozom, galaktocele, parasitic lesions of the breast, specific breast infection (tuberculosis, syphilis).

Breast cancer has some commonality with acute suppurative mastitis in the following situations: the accession of purulent complications during the disintegration of the tumor in the later stages of the process flow and when the so-called mastitopodobnoj form of breast cancer. General upon accession of purulent complications during the disintegration of the tumor is the presence of sharp pain, redness and infiltration of the affected mammary gland and, in some cases, when an abscedirovanija-voltage fluctuations. Distinguish these conditions allows the lengthy course of disease in breast cancer, the presence of several months surround education in iron, sometimes with the formation of ulceration, the presence of enlarged regional lymph nodes, signs of distant metastasis in flat and spongy bones of the skeleton. Typically, these patients were treated at this disease, received radiation and chemotherapy. Often, it is the patient preklimaktericheskogo and menopause, having abortions in history, in the postpartum period is limited kormivshie breast-feeding. However, when later cancer treatment is sometimes difficult to differentiate the vulgar laktacionnyj not menopausal mastitis and septic complications of tumor in the breast. Histological study of tissue can help, suspicious at the tumor, as well as the instrumental studies (ultrasound, x-ray of the spine, pelvis and thorax, CT) to diagnose indirect signs of tumor growth.

Mastitopodobnaja form of breast cancer is vysokozlokachestvennyh nizkodifferencirovannyh cancers occur, usually at a young age, most often during hormonal adjustment in puberty. Disease in this form of cancer is developing extremely fast, growing as cancerous intoxication, and local changes in the mammary gland, early signs of distant metastasis. Common with purulent mastitis is some external similarity of local symptoms: the presence of diffuse edema, mild redness of the breast, signs of lymphatic flow disorders from the organ (the skin of lemon peel type "); However, against the background of changes resemblingflegmonoznuju form of mastitis, signs of purulent intoxication; worries over the prolonged process (a few weeks in the absence of local speakers from the breast), signs of cancerous intoxication, weak pain syndrome, the young age of the patients (11-15 years) and the appearance of this clinic outside pregnancy and lactation.

Fibroadenomatoz of mammary glands is an extremely common pathology as a climacteric, preklimaktericheskogo, as well as child-bearing age. Some doubts in the differential diagnosis with fibroadenomatoze may occur when its diffuse melkouzlovoj drobinchataja breast form» expressed sore syndrome, especially increasing ovulation phase of the menstrual cycle. Distinctive is the absence of background pain giperemia, infiltration and fluctuations, regularity of occurrence of such complaints and increased them in the middle of the menstrual cycle, the absence of signs of purulent intoxication, usually age patients over 45 years, the absence at the moment of pregnancy and lactation. In doubtful cases, breast ultrasound are useful and thermography. When mastite on breast thermograms visible region of very high heat up in the projection of the hearth of inflammation. Temperature gradient may reach 3° c. normally, according to the nature of the disease, we see hyperemia in axillary projection, reflecting the reactions of the lymphatic system to inflammation, and accordingly is not present when fibroadenomatoze.

Galaktocele (retencionnaja cyst breast slices) occurs relatively rarely and develops several months after cessation of lactation. Patient notes the roundish, with clear contours, elastic, mobile education in the mammary gland. Unlike mastitis skin over him not missing perifokalnaja hyperemic infiltration and swelling of the lymph nodes are not enlarged, regional, mobile and painless. Sometimes positive symptom Kenig. Secondary development of an infected galaktocele, clinic transformed into typical manifestations of breast abscess and almost indistinguishable from itself in acute mastite. Incline toward the diagnosis of nagnoivshegosja galaktocele can only be familiar with the data previously found palpiruemom education.

Parasitic lesion of the breast (jehinokokkovye cysts) are fairly rare: manifest the presence of circular, moveable, flexible education in the mammary gland. From purulent mastitis are also distinguished by the lack of signs of local and General purulent infection. Help diagnose can ultrasound, CT, as well as jepidanamneza, history of hydatidosis other bodies, positive reaction Latex-Agglutination with Antigen cyst. Quite rare when the giant size of parasitic cysts may be clearly defined symptom "gidatid" shake with palpation undulirujushhej gland.

In the last decade owing to the deteriorating socio-economic conditions once again became topical question on the possibility of specific infections (tuberculosis, syphilis), defeat its various bodies, and in particular, breast.

Tuberculosis of the breast lesion can be observed in secondary disseminirovannyh forms of the disease. It is manifested by the presence of dense "cold" infiltrations, an increase in regional lymph nodes, the formation of multiple "lattice fistula with meager kazeoznym otdelemam, lack of acute onset. Distinctive features of nonspecific mastitis is the lack of local hyperemia, sickliness and fluctuations, common symptoms of purulent-septic process, the presence of signs of pulmonary tuberculosis, relevant data, jepidanamneza positive data of serological reactions.

Syphilis breast cancer can be a manifestation of the tertiary, gummoznogo period of disease, when in iron has a tight limited infiltration-Gunma. Unlike purulent mastitis when it lacks hyperemia, oedema and pain by palpation and there are positive serological reaction to antigens pale Treponema, a long period of illness. Also affect mammary gland can and in primary syphilis, with the formation of primary sifilomy (solid shankra) in place of the primary affect pathogen (usually in the area the areola). This classic picture small sores with thick edges and pink rounded bottom, painless by palpation. Serological reaction with another negative characteristic of the kind of sores to distinguish it from nonspecific nagnoitelnogo process (numerous abscess) paraareoljarnoj area

**Test questions**:

1. give a classification of mastitis

2. name a disease with which you must differentiate mastitis.

3. Instrumental methods of diagnosing septic mastitis

4. Prevention of laktacionnyh mastitis.

**Tests for self-control**:

List the forms of purulent mastitis: 1, 2, 3, 4

1. abscedirujushhij

2. infiltrative-abscedirujushhij

3. flegmonoznyj

4. gangrenous

5. serous

List the forms of negnojnogo mastitis: 1.2

1. serous

2. infiltrative

3. abscedirujushhij

4. flegmonoznyj

5. gangrenous

Depending on the location of the epicenter inflammation mastitis happens, select correct answers: 1, 2, 3, 4

1. subcutaneous

2. subareoljarnym

3. intramammarnym

4. retromammarnym

5. mezhmyshechnym

6. total

Transition of serous mastitis in infiltrative and later in purulent occurs: 1

1. for 2-3 days

2. for 4-5 days

3. for 5-6 days

4. for 7-8 days

5. for 10-12 days

Phase of acute mastitis: 1, 3, 4

1. serous

2. fibrinoznaja

3. infiltrative

4. abscedirujushhaja

**DISHORMONAL MASTOPATHY**

**Theoretical reference.**

The largest value among the dyshormonal breast disease is disease of the breast, or fibroadenomatoz, which often lies pathological proliferation of mammary epithelium, collagen stroma transformation on the background of ferrous the body and its subsequent gialinoza with the formation of cysts. When the disease preventive care visits is found more than a quarter of all women, but most often between the ages of 30 to 50 years as fibrocystic form.

Leading etiological factors conducive to the emergence of mastitis, are violations of the regulatory activity of the hypothalamic-gipofizo-adrenal system. A small number of genera, short and inadequate lactation, abortion, inflammatory diseases of the uterus and ovarian follicular ovarian cysts become causes, predraspolagatmi to mastitis. These women appear different menstrual irregularities, violation of genetic function, anovuljarnye cycles.

Pathogenesis of mastitis is largely driven by the persistirujushhim action of prolactin, a breach of the ratio of estrogen and progesterone, the increased levels of follikulinstimulirujushhego hormone and estrogen outside natural mammogeneza.

Morphological picture of mastitis is represented by the combination of proliferative, Dystrophic and atrophic changes in the epithelium of duct, idiosyncratic changes miojepitelija connective tissue stroma and glandular breast body.

Classification.

Distinguish between diffuse and anchor forms of mastitis.

Fibrocystic mastopathy form:

-diffuse fibrocystic breast disease: with predominance of ferrous components (adenosis), with a predominance of fibrous component, with a predominance of cystic component mixed form.

-Nodular fibrocystic breast disease.

**The clinical picture**. Clinical manifestations of mastopathy are: pain, discharge from the nipple, mammary gland seals. In the first phase of development of the disease patients celebrate pain in one or both breasts appearing in predmenstrualnom period which combined with swelling breasts. Pain may be spontaneous or occur only when pressure is applied. You may experience discharge from the nipple-molozivnye, serous, bleeding sometimes. One of the symptoms is breast-seal-determined by palpation in the position of the patient standing, when dolchatost can be detected, stippling ("drobinchataja chest), tjazhistost breast tissue. By palpation in the position of the patient lying on his back, with iron prizhatii the disc to the chest seal disappears (negative symptom Koenig). In the second phase of mastitis frequency of pain becomes less distinct, and little changes in mammary glands depends on the menstrual cycle. There are two clinical forms of mastitis: diffuse, described above, and an anchor. Nodular form of mastitis is a defeat not just iron body and its individual segments and shares. It is characterized by the definition during the feelings of one or (more often) multiple painless seals, not changing throughout the menstrual cycle. Preemptive cystic degeneration of the duct allows to select third-kistoznuju form of mastitis.

Diagnosis of mastitis is based on clinical, radiological and morphological studies. Beskontrastnoe x-ray examination of the breast-a mammogram-allows you to confirm the presence of mastitis, clarify the predominance of Adenomatosis, cysts or fibrous changes observed Dynamics influenced by treatment, identify tumors emerging against the backdrop of mastitis. When it detects a large cystic formations use pnevmokistografiju which gives the opportunity to clarify the completeness of emptying the cyst and identify vnutrikistoznye tumor formation. Morphologic characteristics of mastitis can be obtained by cytological study discharge from the nipple, needle biopsy hotbeds of seals.

**Differential diagnosis**.

Dishormonal mastitis should be differentiated from fibroadenomoj, Gynecomastia, breast cancer, galaktocele.

Fibroadenoma of the breast-occur at younger ages than mastitis, often up to 30 years, sometimes very young girls. Fibroadenoma may be single or multiple, often combined with mastopathy.

By palpation fibroadenoma is determined by clearly defined, dense, rounded knot with a smooth surface, not soldered with the skin and the surrounding tissue. Less frequently, have fibroadenoma krupnobugristuju surface. Their consistency varies, often dense. The skin over the nodes does not change. Sizes vary from fibroadenoma values nut to Mandarin and more (1-2 cm and 5-7 cm). Fibroadenoma malignizirujut less mastopathy. Does not disappear when node palpation lying (symptom Kenig positive). The mammogram is visible shadow of rounded shape with clear contours.

Dishormonal mastitis should also differentiate from breast cancer. Clinical manifestations of fibrous or fibrocystic mastopathy, unlike breast cancer, there are a few days before the onset of menstruation. The pain can be intense and irradiirovat in the arm and shoulder. After menstruation pain disappears, the intensity of the pain decreases. By palpation of the gland indicated mild soreness and identifies sites seal without clear boundaries in the form of a heavyweight fine grit. From the selection of various nature occur nipple.

**Gynecomastia-** disgormonalnoe disease breast cancer in males, which is increased to the size of a woman's breasts through glandular hyperplasia and connective tissue. The disease can develop at any age, usually after the age of 40.

Among important etiological factors reducing androgenic activity in testicular atrophy, cryptorchidism, testicular tumors, orhite, tumors of the adrenal gland and improvement of estrogenic activity.

The basis of the pathogenesis of Gynecomastia lie violations physiological ratios of male sex hormones and estrogen.

Distinguish between diffuse and anchor shape of gynecomastia. To diffuse the characteristic increase in breast cancer, the appearance of painful seals, which is located behind the areola. Seal is in relation to the subject fabrics, skin of the breast and areola are not changed.

When anchor characteristic form: definition by palpation of dense, smooth, rolling aytsevidnyi education with a smooth surface. Accurate diagnosis of true Gynecomastia provides education punctate or remote study drug.

It is also necessary to carry out a differential diagnosis of dyshormonal mastopathy with retentive area breast Cyst (galaktocele).

Galaktocele is formed by blockage of one or more of the milk ducts after lactation or after suffering mastitis. Galaktocele develops gradually and is a retention cyst with growths of polipoznymi epithelium. By palpation in breast cancer is determined by the elastic, painless, still education. From nipple stands out the secret of the grey-green or bloody color. Cytological study identifies epithelial cells, erythrocytes, leukocytes. Under controlled mammography seen cystic formation in the major dairy operations.

**Test questions**:

1. What are the factors which contribute to the development of dyshormonal mastopathy.

2. Name the form dyshormonal mastopathy.

3. What are the methods of diagnosis used in mastitis.

4. List the diseases with which it is necessary to carry out the differential diagnosis of mastitis.

**Tests for self-control**:

Diffuse mastopathy does not apply: 5

1. estrogenic drugs

2. physiotherapy

3. long-term prescription potassium iodide

4. sectoral resection of breast cancer

5. radiation therapy

Listovidnaja fibroadenoma is more common in the age: 2

1. from 20 to 30 years

2. from 30 to 40 years

3. from 40 to 50 years

4. after 50 years

The main manifestations of mastopathy are: 1, 2, 3

1. pain

2. discharge from the nipple

3. compaction mammary glands

4. increase of axillary lymph nodes

Gynecomastia often develops at the age: 3

1. before 30 years old

2. up to 40 years

3. after 40 years

**TUMOR AND CYST OF THE MEDIASTINUM**

**Theoretical reference.**

**Classification** . New growths of the mediastinum include: true tumor, cyst and tumour formation. Clinically they are divided into 4 groups: 1. tumors (benign and malignant) and embryonic true cysts; 2. zagrudinnyj and vnutrigrudnoj goiters (benign and malignant); 3. metastasis of cancer of other organs; 4. psevdoopuholevye lesions of lymph node tuberculosis, jehinokokkoze, etc.

The most common primary tumor and cyst-more than 90% of tumors of the mediastinum.

**The clinical picture**. A characteristic complaint of chest pain patients, the intensity of which depends on the degree of compression of the reflex zones. Often pain syndrome is preceded by a feeling of discomfort, external education in the chest. Another frequent symptom is shortness of breath, which is the result of compression of the airway. Asymmetry can occur during an inspection of the chest, and a bulging deformation of certain areas. Cardiac dullness typically extended heart-felt muted tones. When far before the process in the mediastinum is typical compression syndrome-cyanosis of the face, neck, upper half of the body, swelling of the jugular veins (superior vena cava syndrome "). The basic diagnostic method is x-ray. The study starts with the multi-axle fluoroscopy, then to refine the data obtained using special methods: tomography, pnevmomediastinografiju, rentgenkimografiju, bronhografiju, jezofagografiju, pnevmoperiotoneum, computer tomography. ULTRASOUND to distinguish tumor from cysts. In cases where the nature of the process, is expected to achieve vascular as well as compression or germination of tumors in the heart and large vessels used angiography by the method Seldingera. Upon detection of the pathological process in the anterolateral upper mediastinum-mediastinoscopy with biopsy is performed. Laboratory studies are subsidiary. So Wasserman's reaction will set the sifiliticheskuju nature of the disease, the reaction of the Latex-Agglutination jehinokokkovoe confirm defeat. With nevrogennyh tumors useful iodine-starch Minor trial in which violation is detected by sweating in the skin areas with broken innervaciej.

**Differential diagnosis.**

Differential diagnosis between nevrogennymi and mesenchymal tumors, teratomami, limfosarkomoj and limfogranulomatozom, bronchial cysts, cysts, jenterokokkovymi aspects, zagrudinnym goiter.

Teratomas are composed of various tissue elements and organopodobnyh structures. Allocate mature and immature teratomas. All mature teratomas usually are well encapsulated, rounded or oval in shape. Immature teratomas have kind of solitary sites, sometimes with small cavities. Contents of teratomas-ropy mass, including plots of glands, hair, teeth, and bones.

Clinically distinguish: asymptomatic leaking, clinically manifested and cause teratomas. Located most commonly in the anterior mediastinum teratoma are accompanied by the development of cardiovascular syndrome (tachycardia, feeling the pain in the heart), tracheal and bronchial compression leads to pristupoobraznomu cough, dyspnea, krovoharkanyu. Most patognomonichnyj symptom identification in the sputum of hair and other tissues. When x-ray study is determined by the rounded or oval formation with clear boundaries, not smeshhajushheesja when swallowing and coughing.

Neurogenic tumors more often localized in the posterior mediastinum. From sympathetic nervous trunk occur: ganglionevroma, neuroblastoma, pheochromocytoma, simpatikoblastoma; and the resulting tumors of the peripheral nerves include: Neuroma, Neuroma neurofibroma. Clinical symptoms in nevrogennyh tumors: pain, weakness, shortness of breath, sweating, gipostezii, palpitation, hoarseness of voice, Horner's syndrome, numbness in the hands.

Of crucial importance in the diagnosis of x-ray belongs to exploration, as well as diagnostic sample Minor. To clarify the relationship of the tumor with mediastinal organs used computed tomography.

To mezenhimalnym tumors of the mediastinum include: fibromu (tumor of fibrous connective tissue), lipomu (a tumour of adipose tissue), and osteohondromu to chondro (tumor of cartilage and bone tissue), lejomiomu and rabdomiomu (tumors of muscle tissue), differentiating Hemangioma and limfangiomu (originating from tumor vessels). Tumors of the small size of leak for a long time asymptomatic, and therefore can reach considerable size before the appearance of the first clinical signs of compression of the mediastinum. Malignant neoplasms are manifested earlier that due to the rapid infiltrativnym tumor growth, metastasis and increasing intoxication.

Mediastinal lymphosarcoma and chlamydia. Clinic limfosarkom of Mediastinal Hodgkin's disease and is caused by the disease first intoxication, while large sizes-tumor compression of adjacent mediastinum. With the development of the process occurs remittirujushhaja temperature, hot sweat, weakness, itching, weight loss. Appear shortness of breath, chest pain. Mediastinal lymph node lesion confirmed by limfografiej, mediastinoskopiej with biopsy. The most characteristic changes in the pattern of blood-leucocytosis or lakopenia, increased ERYTHROCYTE SEDIMENTATION RATE.

Bronchial cysts are characterized by commonality of structure with walls of the respiratory tract. Bronchial cysts have a ovoidnuju form, more often they single-Chamber. Neighborhood with tracheal bifurkaciej cysts cause vexed cough, shortness of breath. For localization parajezofagealnoj cysts are characterized by dysphagia. When bronhografii and Bronchoscopy is sometimes detected a cyst, connecting with traheobronhialnym wood. X-ray studies reveal rounded or jellipsovidnoe dimming in the medial areas upper sections of the chest that has the clear outer contour. More often, the shadows of cysts to adjacent densely trachea, displace its and main bronchus.

Jehinokokkovye cyst of the mediastinum are fairly rare, and the clinical picture of Mediastinal hydatidosis depends on the size, location, presence or absence of complications of cysts. When small amounts of parasite cysts during asymptomatic. Large size cyst can cause pain, shortness of breath, bring on dysphagia, compression of the superior vena cava syndrome. In rare cases possible breakthrough cysts in the bronchus or trachea. Festering cysts cause purulent mediastinitis. Diagnosing promotes Eosinophilic-allergic reaction-test Latex-Agglutination. Radiographically detected circular or oval shade with clear contours, shadows of hitinovoj shell and calcifications. Mediastinum Hydatid disease frequently observed, while the defeat of the lungs.

Timomy-tumors of the thymus gland. They are divided into three groups: epithelial, lymphoid, teratoidnye. Clinical manifestations of Tim: weakness, fatigue, sweating, increased body temperature, hoarseness of the voice, a sense of compression in the sternum, weight loss, cough. Growing thymoma is combined with the development of myasthenia. Radiographically detectable shade with clear outlines in the upper and middle mediastinum in the immediate vicinity of the sternum. Confirmation of diagnosis contributed to pnevmomediastinografija and mediastinoscopy with biopsy.

Jenterokistomy-congenital cyst of mediastinum, building the walls of which resembles the structure of the walls of the digestive tract. Depending on the degree of similarity of their structures secrete: my intestine, stomach and intestinal cyst. For jenterokistom characteristic of localization in the back mediastinum, radiographically right from the midline. Clinically apparent chest pains, difficulty breathing. If nagnoenia, perforation of jenterogennyh cysts fast progressing clinic purulent mediastinitis-raspirajushhie fever, pain in the chest and neck, the total heavy septic pulmonary injury status.

Zagrudinnyj goiter manifested symptoms of compression of neighbouring bodies of the neck and mediastinum is cyanosis, edema of the face and the neck, swelling of the veins of the neck. Compression of the trachea, causing shortness of breath at rest, breathing stridoroznoe. Compression of thoracic sympathetic trunk marked tachycardia, heart rhythm, Horner Syndrome (PTOSIS, miosis, enophthalmos). The topical diagnosis set using pnevmomediastinografii, mediastinoscopy, scintigraphy, ultrasonic echolocation.

**HYDATID DISEASE OF LUNG**

**Theoretical reference.**

Hydatid disease of lung-glistnaja invasion, flowing with the development of jehinokokkovoj in lung tissue cysts and specific clinical picture of the disease process.

Infection occurs when non-compliance with the rules of personal hygiene onkosferami (eggs), suffer from eating parasite by helminthes by inhalation or in contact with sick animals. The main hosts of the parasite are carnivorous animals (wolves, dogs, foxes, etc.). Prmezhutochnymi hosts-large and small cattle, as well as people.

**Classification**.

Hydatid disease of lung happens to be primary (lung) and secondary (metastatic). Distinguish one-Chamber (gidatidoz-s ') and Multicam (alveolar) Hydatid disease of lung (alveolokokkoz), uncomplicated and complicated (suppuration, atelectasis, piopnevmotoraks, etc.). In size jehinokokkovye cysts are small (up to 2 cm), medium (2-4 cm), large (4-6 cm) and gigantic (> 6 cm); by quantity is single and multiple; on the configuration-globular and distorted; structure-solid and liquid level.

**Clinical classification of hydatidosis light includes three stages:**

1. Latent (asymptomatic)

2. Clinical manifestations

3. Complications.

**The clinical picture**. In the first stage of the patients have no complaints. At this time, the examination of patients, systems of any signs of pulmonary cyst does not identify.

Clinical phase includes two periods: the period of the development of closed-bladder without complications and period autopsy jehinokokkovoj cysts in the bronchus-outdoor period. In the first period of complaints is scant. The patient's condition is satisfactory, there is a slight pain in my chest, without explicit localization, dry cough, body temperature normal, shortness of breath, no, there are no signs of intoxication. When a physical study only at larger sizes for cysts detected shortening perkutornogo sound, relaxed breathing. In the study of blood indicated moderate Eosinophilia, Latex-Agglutination reaction is positive.

X-ray light is determined by the rounded, homogeneous, clearly limited shade, with no horizontal surface of the liquid and thick capsule around. You can sometimes detect serpovidnuju shadow detached and spavshejsja shell hitinovoj.

With the breakthrough of cysts in bronchial cough appears with departed clear liquid and odourless, with scraps of hitinovoj shells, coughing up blood, weakness, fever, dyspnoea and urticaria, can lead to suffocation. If nagnoenia phlegm abscessed cyst, odourless and does not sloitsja. Temperature, but there are no signs of intoxication. No shortness of breath, cyanosis and no signs of chronic intoxication (fingertip and nail plates). In the lungs moist rales heard melkopuzyrchatye. X-ray light is determined by the cavity with fluid level, does not have a dense capsule. You can sometimes detect serpovidnuju shadow detached and spavshejsja shell hitinovoj. Gap jehinokokkovoj cysts may be accompanied by heavy bleeding.

**Differential diagnosis**.

The differential diagnosis should be performed with lung cancer by lung, bronchoectatic disease, tuberculosis of the lung.

Lung cancer. In the early stage of the disease for peripheral lung cancer, as well as for hydatidosis is characterized by x-ray light round shadow. Unlike cancer, when jehinokokkoze you can install epidemiological history (contact with pets, large and small cattle) and for a long time there is no clinic. Manifestation in lung cancer process begins with subfebriliteta, weakness and dry cough that is the vexed nature with meager amounts of mucous sputum containing blood veins that atypical for cyst during a private bubble. In the subsequent condition of the patient deteriorates progressively increased until pneumorrhagia appears pulmonary hemorrhage, increasing phenomenon of cachexia, anaemia; During germination of a tumour intercostal nerves or with metastatic vertebral body with their further destruction and development radicular compression technology, developing atypical for cyst pain syndrome. When jendobronhialnom tumor growth with time comes the obturation bronchus and development equity atelectasis. There is a sunken chest wall, her breath, vtjanutost and convergence of the intercostal spaces. For hydatid lung cyst is characterized by slow expansive growth and development atelektaticheskogo syndrome, which occurs only rarely.

In the blood of a patient with lung cancer marks the acceleration to the ESR 50-70 mm/h, which can also be observed in patients with jehinokokkom and light. In the analysis of sputum in patients with lung cancer in 83% of cases of microscopically define abnormal cells. Shadow rentgenograficheski cancer tumors of dense, irregular, rough, «landshaftoobraznymi» outlines, has short shadows-"tendrils", dating back to the root of the lung cancer, the so-called "tracks"-a sign of sprouting along the way tumor lymph collectors and bronchi. During the disintegration of the tumor in the center of shadows appears cavity with polycyclic paths containing no liquid level. For jezinokokka, the radiograph of the lungs, is characterized by a clear circular homogeneous shadow with smooth edges. Is sometimes a symptom of "detachment" and signs of defeat lymph collectors never defined.

Lung abscessis characterized by acute circulatory flow process. Flows in the form of two distinct periods: the phase formation and breakthrough phase infiltration and drainage in the bronchus. In the first phase of patients complain of constant chest pain, weakness, fever, non-productive cough, high temperature gekticheskuju, shortness of breath. There has been a sense perkutornogo sound, relaxed entering the breathing. In the blood high Leukocytosis, elevated ESR. If nagnoenia jehinokokkovyh cysts, there is also a high Leukocytosis and elevated ERYTHROCYTE SEDIMENTATION RATE, in this case, however, is not characterized by the emergence of cough; spontaneous same breakthrough nagnoivshejsja cysts in the bronchus or pleural space occurs much later than abscesse. Nagnaivajutsja usually the dead parasites after long term after its invasion. With breakthrough jehinokokkovoj cysts, in case of a festering, phlegm is not characteristic of trehslojnosti abscess. In otharkivaemoj sputum can be found scraps of hitinovoj shell and skoleksy. When a Breakthough in the bronchus jehinokokkovoj cyst of the liver in the sputum, a mixture of bile.

Bronchiectasisis manifested from childhood, is either congenital or caused by diseases such as whooping cough, influenza, Primary Tuberculous Complex. Patients constantly in the morning indicated releases purulent sputum, shortness of breath, subfebrilitet, characterized by phasing of currents, with increasing duration of illness, increasing signs of chronic purulent intoxication (deformation of nail phalanxes, often joins amyloidosis, chronic anemia and hypoproteinemia). Unlike hydatidosis never celebrated eozinofilia, more typical of leucocytosis with a shift of the formula to the left, high ESR. Varies and radiological picture-when bronchiectasis has never indicated a clear circular homogeneous shadow, as in jehinokokke; and when you can always detect bronhografii also missing at jehinokokke, meshkovidnye or chetkoobrazno advanced bronchi.

Tuberculosis of lung, as with complicated shape of the hydatid lung cyst is determined subfebrilnaya temperature, shortness of breath with exertion, coughing. Unlike hydatidosis, lung tuberculosis taped their epidemic anamneses: scanty sputum and until the formation of Caverns is missing hemoptysis. Microscopic examination of sputum determines the presence of m. tuberculosis. Radiographically, tuberculosis identified miliarnye and endemic infiltrative "tricks", and during the disintegration of the cavern with the so-called "track" to the root of the lung. When tuberkulome detects other radiographic signs of tuberculosis are traces of dissiminacii. Positive serological reaction of Mantoux test.

**Test questions**:

1. Specify the path of cyst lesions in the human body.

2. Classification of hydatidosis.

3. The clinical picture of hydatidosis.

4. Clinic breakout jehinokokkovoj cysts in the bronchus.

5. What are the methods of instrumental diagnosis of hydatidosis.

**Tests for self-control**:

What are the most frequent cause of spontaneous pneumothorax: 4

1. lung abscess

2. lung cancer

3. bronchiectasis lung

4. Bullous lung cysts

5. Hydatid disease of lung

For x-ray pictures hydatidosis light characteristic: 2

1. the rounded shadow without clear contours

2. round shade with smooth clear contours

3. round shade with a track to the root of the lung

Hemoptysis is an early symptom for: 5

1. lung abscess

2. lung cancer

3. empiema

4. bulleznah lung cysts

5. all wrong

Select all of the symptoms characteristic of the triad: 2, Horner 3, 5

1. exophthalmos

2. PTOSIS

3. miosis

4. swelling of the face

5. enophthalmos

When identifying a round shadow on Lung radiograph not shown: 5

1. tomography

2. dynamic observation

3. sample von Pirquet and Mantu

4. fibrobronchoscopy with biopsy

5. pnevmomediastinografija

**NONSPECIFIC NAGNOITELNYE LUNG DISEASE**

**Theoretical reference.**

The main reasons for the development of non-nagnoitelnyh lung disease are;

1- highly virulent microbes penetrating in the light fabric;

2-reduction of protective forces of the organism of man;

3-violation of the drainage function of the bronchi;

4-blood circulation disturbance in the lung.

To the chronicity of lung disease nagnoitelnogo results, and inadequate treatment of the acute process.

**ABSCESS AND LUNG GANGRENE**

Abscess and lung gangrene as separate Nosological forms identified in year 1819 3. He also made the first description of gangrene of the lung as the most severe pulmonary pathology. Sauerbruch suggested merging these diseases under the common name of "pulmonary sepsis. The first information about the pathogenesis of lung gangrene published in 1871 g. l. Traube.

Lung abscess-purulent or rot decay plots of lung tissue, often within the segment with the presence of one or more cavities of destruction, filled with dense or liquid pus surrounded by perifocal infiltration of lung tissue. Gangrene of the lung is a suppurative necrosis putrid significant plot of lung tissue, often 2 lobes, the lobes or the entire lung, without clear demarcation signs tending to further spread and reflected dire general condition patient. Unlike the abscess cavity when gangrene of the lung contains sequesters lung tissue.

**Classification**. Distinguish:

1. According to etiology:

(a)) primary and secondary abscesses.

b) aerobic and anaerobic abscesses.

2. Pathogenesis:

a) ajerogenno-aspiration

b) haematogenously-Embolic

traumatic)

g) septic

3. Downstream:

a) sharp

b) chronic

4. Localization:

and parietal)

b) median

in) deep

5. By the nature of the flow:

and uncomplicated)

b) complicated (empyema, lung hemorrhage, sepsis, etc.).

6. Single and multiple

The predisposing factors of pulmonary suppurations include: unconsciousness, alcoholism, drug addiction, epilepsy, brain injury, cerebrovascular disorders, coma, overdosed on sedatives, general anesthesia. In addition, stenozirujushhie esophageal disease, immunodeficiency States.

Fairly high incidence of abscess of lung with pneumonia (from 2 to 5%), closed chest injuries (1.5%), -2 (1.5%) gunshot wounds.

Acute infectious lung destruction are usually nonspecific, but sometimes there are mixed when nonspecific and specific infectious processes develop simultaneously.

Currently, the most commonly found in infectious lung destrukcijah are grammotricatelnye and anaerobic, and also staph. But for the occurrence of infectious lung destruction not enough simple hit of pathogenic microorganisms in lung parenchyma, as with well-preserved cleaning function intraoperative tracheo-bronchial tree in the normal functioning of local and general protective mechanisms, microbial invasion has no clinical manifestations.

For the development of an abscess and lung gangrene should impact of pathogenic factors distorting these mechanisms. These include: high virulence of pathogenic organisms; violation of patency and drainage function of the bronchi; disorders of Microcirculation in the zone of an infectious or traumatic inflammation of lung tissue.

**The clinical picture** of infectious destrukcij of the lung is very diverse and depend on the individual (including immunological) characteristics of the organism, stage of the process, the presence of predisposing and Comorbidities.

There are two periods in the clinical picture: the period of formation of purulent cavities before her breakthrough in the bronchus (closed or blocked stage) and the period after break the abscess in entering the tree (open or deblokirovannaja stage).

During the formation of the infiltrate in patients clinic dominates intoxication-weakness, headache, dry cough, a high fever with a hectic panache, vomiting, tachypnea (ChDD amounts to 40 mins.). Locally observed: a dull pain in the chest on the side of the lesion, dulling the sound perkutornogo, relaxed entering breath over an area of infiltration. In deblokirovannuju the stage suddenly appears a large number of three-layer with gnilostnym sputum odor mouth full. More often than not deblokacija abscess is not sudden nature, and for 2-3 days sick notes a gradual increase of the number of phlegm, reducing, at the same time, the degree of intoxication, improving health, reducing the temperature.

Radiographically in the field infiltration starts determined cavity with fluid level, which, with favourable throughout the process for 5-7 days gradually decreases. In the peripheral blood leucocytosis disappear and palochkojadernyj shift formula.

In some cases with acute abscesse process does not end with the spadeniem cavity and safely the abscess becomes chronic.

The reasons for this are the following:

1. violation of the patency of confluent bronchus, and because of this lack of outflow of the pus out of the cavity;

2. cavity abscess dense dead fragments pulmonary parenchyma, necrotic sequesters;

3. increased pressure in the cavity when coughing and wide lumen draining bronchi;

4. education for the stretch of pleural impeding briefly cavity;

5. when its inadequate drainage.

When gangrene (gangrenoznom abscesse) of the lung disease clinical picture is characterized, in contrast, described a heavy condition of the patient, in which the first place stands the expressed intoxication until the development of septic shock respiratory distress, and often, signs of multiple organ failure. Usually occurs in subjects, weakened, or somatic diseases with psychic disorders (alcoholism, drug addicts, drug addicts), characterized by a suction mechanism of penetration of microorganisms in lung parenchyma.

The condition of patients with septic shock reminds with circulation, akrocianozom, tachycardia and lower blood pressure, disordered consciousness which is almost not found in abscesse. Characteristic selection of a small number of serous mucus and edgy putrid breath. The affected side chest behind when breathing.

Physical data depend on the amount of lung tissue necrosis and severity of decay-dulling perkutornogo sound box shade it over the cavity located destruction subkortikalno; If Auscultation is a significant weakening of the (lack of) respiratory noise, amforicheskij shade over drenirujushhejsja through bronchus cavity are wet wheezing.

Radiographically determined total infiltrativnoe blackout light without limit. Abscess and gangrene, in fact, represent different kinds of destructive inflammation in the lung parenchyma, but abscesse dominates purulent melting, while gangrene-necrosis of lung tissue.

Diagnosis of infectious lung destruction is determined on the basis of anamnesis, assessing clinical manifestations, laboratory data and rentegenologicheskih studies.

The main way of verification of diagnosis are radiological investigations-x-ray, radiography, tomography.

The past decades have been characterized by active introduction of digital medical images for diagnosis of pulmonary suppurations. Leading role in this, no doubt, took the computed tomography (CT). It is, on the one hand, provides invaluable help in the differential diagnosis of abdominal Neoplasms of the lung. On the other hand, it is possible to spend under CT control biopsy of solid light formations, draining abscesses in vnutrilegochnom location and "difficult" trajectory of access to education.

Without systematic repeated x-ray examinations (through the 1-2 days) it is difficult to assess the effectiveness of treatment and exercise correction treatment.

In the list of mandatory instrumental research Bronchoscopy should be noted to enable deletion of tumor nature of the process, hold the fence material for bacteriological and cytological study.

Fibrobronhoskopii in combination with chrezbronhialnoj biopsy of the wall of the Cavitary education, preference should be given to such invasive research, both Transthoracic biopsy chrezlegochnaja wall abscess.

Much later, in diagnostic arsenal Lung Surgery took place ultrasonography. Valuable diagnostic method is bronchial Arteriography.

Catheterization of bronchial artery and other branches of the aorta is performed by the method of access chrezbedrennym Seldingera.

In acute lung abscesse develops gipervaskuljarizacija lung tissue with a significant increase in peripheral branches and intensive parenhimatoznoj phase contrast. Extension, crimp bronhialno-pulmonary messages are typical for a chronic abscess. For easy gipovaskuljarnyj is typical of gangrene variant blood supply to the pathological zones.

Among the laboratory research methods, you must allocate the bacteriological, because they have an influence on the choice of the optimum amount of antiviral therapy.

**Differential diagnosis.**

Differential diagnosis of infectious destrukcij of the lung is very complicated due to the diverse clinical manifestations of disease in different periods.

Quite often the abscess must differentiate with lung cancer. Unlike an abscess, lung cancer is characterized by: a long period subfebriliteta period, long-term buildup "small signs" syndrome, with a meager amount of muco-haemorrhagic sputum, hemoptysis, lack of adherence three-layer rate, as in abscesse.

During lung cancer, not typical for abscess faznosti currents. When the obturation tumour the bronchus and the development of atelectasis share, notes lag chest wall, sinking her in the Act of breathing, umbilicus and resulted in the intercostal spaces. In the sputum and bronchial lavage in 83% of cases detected atypical cells. In the blood is characterized by increased SEDIMENTATION RATE up to 60-70 mm/h, lakopenia, anaemia anaemia. When x-ray study tumor dense, with uneven contours, does not contain the level of the liquid, there are phenomena cancer Lymphangitis (symptom moustache).

In some cases, you must differentiate lung abscesses of hydatidosis. Typical jepidanamneza specific data-accommodation in endemic jehinokokkozu areas. In the first, a period characterized by preclinical complaints sporadic manifestations of hives, vague aches and pains in the chest, a rare dry cough. In the study of blood, usually celebrated eozinofilia (20-25%), there is a positive reaction Latex Agglutination antibody jehinokokku. x-ray study cavity is determined when the liquid formation, with thin walls sometimes visible Crescent shadow when detached hitinovoj shell.

Must be differentiated from bronchiectasis lung abscess. The latter, often flowing, long, long, from childhood. When bronchiectasis are experiencing intermittent exacerbation with febrile fever, cough with a small amount of purulent sputum. Patients have symptoms of chronic purulent intoxication-puffiness of the face, ishudanie, nail plate in the form of a watch glass. Often the disease complicates amiloidnaja disease in which the most often noted kidney transition in chronic renal failure.

The retraction notes Fizikalno healthy side thorax, dulling the sound perkutornogo, multiple small and medium bubble wheezing. Radiographically defined: diffuse pulmonary picture enhancement, increased lung root structure.

More accurate diagnosis is possible if Tomo or bronhografii, allowing to identify the type of localization and bronchiectasis.

Often also requires a differential diagnosis of acute lung abscess with different types of pleural empyemas limitedand so-called pleural lung cavities in which one of the walls of the cavity is pulmonary raspadajushhajasja fabric, another parietal pleura (pleural empyema with destruction of the lung), poddiafragmalnymi abscess. The most informative in such cases, ultrasound and computed tomography.

**BRONCHIECTASIS**

Bronchiectasis-acquired disease characterized by chronic purulent inflammation affecting the entire thickness of the bronchial walls with irreversible change in its structure and function that occurs typically in the lower divisions of the lungs. It is the pathology mostly childhood.

Bronchiectasis (from Greek jektaz-stretching)-morphological concept that refers to a persistent abnormal expansion and deformation of the bronchi.

Congenital bronchiectasis are the result of abnormal development of not only wood but also respiratory departments. They are often combined with malformations of other systems and organs. They should therefore be seen as an integral part of the characteristic set of pathological changes, developing as a result of violations of embryogenesis: cystic lung hypoplasia, Sv-Kartagenera syndrome (bronhoektaza, pansinuit, mirror easy ") syndrome Turpin (bronhoektaza, expansion of oesophagus, congenital abnormalities of the vertebrae and ribs).

Sometimes there is a combination of congenital bronchiectasis with Polycystic pancreas, splitting the upper lip, gluhonemotoj, congenital heart diseases.

Persistent expansion of the small peripheral bronchi can develop as a consequence of pathological processes in the surrounding tissue (a chronic abscess, Fibro-cavernous tuberculosis, chronic pneumonia). Such bronchiectasis commonly referred to as secondary, stressing that this pathological process is a consequence and an integral part of the underlying disease.

The leading role in the development of bronchiectasis is a violation of the patency of the bronchi (medium and small), leading to the formation of obturacionnogo atelectasis. Some patients have an innate predisposition to spadeniju the walls bronchus (maturation of cartilage rings and smooth muscle fibers), increased viscosity rate (at mukoviscedoze), with the formation of dense mucous plugs, obturirujushhih clearance the bronchi. Children malleable bronchus wall often is squeezed by increased lymph nodes with bronhoadenite, often tuberculosis, pneumonia, particularly measles, other respiratory infections that cause hyperplasia of the lymphoid tissue. Below the seat obturation begins to accumulate bronhialny secret.

**Classification bronchiectasis**

1. Origin:

1. Congenital (including combined with other malformations-Sv-Kartagenera syndrome)

2. Acquired (with bronchiectasis)

    (II) On the defeat of the male structures. Lung:

1. With the primary defeat of pulmonary parenchyma

2. Favouring defeat bronchi

   (III) Bronchiectasis. the form:

1. Cylindrical

2. Saccular

3. Fusiform

4. Mixed

  IY .: Flow for clinical

1. Remission

2. Aggravation

3. Constantly recurring within

 Y Complications: availability.

1. Not complicated for

2. Complicated:

-                                             pulmonary hemorrhage

-                                             blood-tinged sputum

-                                             piopnevmotoraksom

-                                             abscedirovaniem

 YI The external respiration function:

1. Without respiratory failure

2. Respiratory failure (I) , (II) and (III) Church.

3. Legern-serdecnaya insufficient.

**Clinical classification of bronhojektaza**

is divided into the form:

  Light form bronhojektaza *x*arakterizuetsja one or two exacerbations during the year, long-lasting remissions. During these periods, the patient feels almost healthy and workable.

  For the expressed form of bronhojektazais characterized by longer worsening every season, with 50-200 ml. purulent sputum for a day. In the period of remission remains cough with phlegm, there is moderate [dyspnoea](https://www.microsofttranslator.com/bv.aspx?from=ru&to=en&a=http%3A%2F%2Fwww.lor-astma.ru%2Fodyshka.htm), reduced ability to work.

  Severe form of bronhojektazanoted frequent, lengthy exacerbations with fever and short-lived remissions. Number of separable phlegm is raised to 200 ml. per day, the smell of phlegm often has a gnilostnym smell. Ability to work during the response is saved.

  Oslozhnjonnaja form bronhojektazacharacterized as signs of severe forms with the addition of secondary complications: cardiopulmonary insufficiency, renal amyloidosis, pulmonary heart, liver amyloidosis, jade.

A detailed diagnosis of bronchiectasis must take into account all listed in the classification of signs marking the localization and the prevalence of the disease process.

**The clinical picture of** bronchiectasis is characterized by duration and circulatory flow, intermittent exacerbations with febrilitetom, copious purulent sputum Office, ever-present and growing during illness chronic purulent intoxication (a symptom of "drumsticks" and "hour glass"), as well as specific complication of bronchiectasis, Amyloidosis of the kidney.

In the first stage of development of the disease indicated a moderate increase small bronchi lumen nakaplivajushhimsja detachable from a bronchial mucous membranes without modifying the epithelium of the walls bronchus.

The transition to the second stage of the disease process means the emergence of purulent inflammation in okkljuzirovannom Division bronchus tree. This is due to a decrease in the efficiency of protective mechanisms of respiratory mucosa lung divisions (alveolar macrophages, immunoglobulin a) under the influence of virus infection, beriberi and diet. Another cause of purulent process is his inhibiting bronchus obturation full evacuation. Purulent inflammation of the bronchial walls applies where there is squamous metaplasia of cylindrical epithelium and ulcerated mucous. In further inflammatory changes are propagated to the bronchial wall layers glubzhelezhashhie, like smooth muscle degeneration occurs and podslizistogo layer. It is at this stage of the disease the pathological process becomes irreversible loss muscle of bronchial wall elements, and hence contractility, and "purifying" function of the bronchi. Short circuit occurs the pathological vicious circle-violation of patency of bronchi-Pyo-inflammatory process is a violation of-progression-purulent inflammation of the deepening of the drainage function of the violations, etc.

In the third stage of development of bronchiectasis pathologic changes expressed in all layers of the bronchial wall and transcend bronchus tree. The bronchi become sharply enhanced in proswete purulent or suppurative exudate ihoroznyj unpleasant spicy scent gnilostnym. Cartilaginous skeleton bronchi is undergoing a rebirth, soedinitelnotkannoe and mucosa ulceration are formed in places whole fields of granulation tissue. This structure leads to lower their bronchial resistance and to the effects of the so-called "bronhodiljatacionnyh" force-enhancing vnutribronhialnogo pressure when coughing, phlegm accumulating tension that ultimately determines the emergence of meshkoobraznyh extensions plots bronchus tree. In the pathological process inevitably involves peribronhialnye tissue (sclerosis peribronhialnoj fiber hypertrophy of bronchial arteries, bronhoadenit) and pulmonary parenchyma (pockets of pneumonia, pnevmofibros). When angiography: increased clearance of bronchial artery in 4-5 times, increases the number of arterio-arterial anastomoses and width clearance. This results in a significant dumping of large arterial blood circulation system of the pulmonary artery, pulmonary hypertension, occurs and subsequently formed "pulmonary" heart. The prolonged existence of purulent process in the lung leads to and progression system lesions: the formation of chronic cardiopulmonary diseases, diffuse chronic purulent bronchitis, emphysema lungs, dystrophy parenchymatous organs and anaemia.

In the diagnosis of bronhojektazij a leading place is occupied by the x-ray examination, and in particular, total and selective bronhografia and tomography. Frequent x-ray symptom of bronchiectasis is to decrease the amount of the affected proportion or all light, compensatory emphysema "intact" the share shift of mediastinum and mezhdolevyh crevices in connection with change of architectonics bronchus tree.

For a full definition of function of the affected Division Lung (pulmonary blood flow) used angiopulmonografija, perfusion scan. Very significant diagnostic importance Bronchoscopy. Bronchoscopic manifestations of jendobronhita allow more detail to judge the quality of sanitation of the lesion and suspect the innate nature of bronchial diseases (bronchial deformation rings, traheobronhomegalija, etc.).

**Differential diagnosis**.

The differential diagnosis should be bronchiectasis with chronic empyema. Similar to bronchoectatic disease is prolonged, chronic disease, the presence of recurrent exacerbations of chronic purulent intoxication. In times of increasing complaints are also available on subfebrilitet, cough with purulent sputum Office coming from the chronic empiema through draining bronchi (with chronic empieme usually has a bronhoplevralnyj fistula). Detected in the blood leucocytosis, shift formula white blood, increased ERYTHROCYTE SEDIMENTATION RATE. Different appearance of the patient: usually chronic pleural empyema occurs in middle and old age, there has been a narrowing of the intercostal spaces, their umbilicus on cavity empiema, dulling perkutornogo sound and relaxed entering or amforicheskoe breath.

Accurately verify diagnosis helps lung x-ray, allowing to visualize chronic cavity empiema having parakostalnoe arrangement and dense wall. In cases of doubt, use polipozicionnuju x-rays of lungs, superjeksponirovannye pictures, bronhografiju and plevrografiju, even rarer, with suspected pleural-mezeteliomu (plevroskopiju) thoracoscopy with biopsy, computed tomography.

In recent years, there is evidence of diagnostic value of ultrasound scanning.

Chronic lung abscess also has similarities with the disease in the form of a long bronchoectatic currents, phase of exacerbation and remission, presence of chronic purulent intoxication. Unlike bronchiectasis with x-ray study identifies located in thicker, rounded shape, cavity formation fluid level.

In chronic abscesse lung bronchi, drainage, in main areas of the lung due to chronic purulent panbronhita may occur secondary bronchiectasic are, however, purely regional, a secondary character. In doubtful cases the differential diagnosis helps bronhografia, which finds out if the disease bronhjektaticheskoj generalized nature of the lesion and the absence of cavity abscess.

Lung cancer may resemble nagnoitelnymi nonspecific lung diseases if jendotrahealno growing tumour the bronchus lumen overlaps, causing atelectasis group segments or share with the development of it abscedirovanija, or dissolution of the tumors with formation of perifokalnyh abscesses. However, when this cancer occurs as a subacute State, preceded by a long period of disease with blood-tinged sputum, increasing intoxication, usually elderly patients are heavy smokers, while sick bronhojektazijami Typically, young age.

In doubtful cases required FBS is used that enables you to visualize the tumor. Also, if you can identify the study x-ray shadow of tumors with polycyclic paths and plots in thicker it "moustache" symptom. In cases of doubt, applied research rinsate on atypical cells, selective bronhografia.

The differential diagnosis should be vnutrisindromno (bronchiectasis, bronhoektaza as a manifestation of other pathological processes-chronic bronchitis, tuberculosis; bronchiectasis with congenital pathology-cystic gipoplazvii, traheobronhomegalii, Sv-Kartagenera syndrome, etc.).

**Test questions**:

1. Specify the mechanisms of penetration of microorganisms in lung parenchyma during occurrence of abscesses.

2. What are the factors that contribute to chronicity acute abscess.

3. List the methods of x-ray studies, applied in a survey of patients with pathology of the lungs.

4. Give classification of bronchiectasis.

5. The goals and objectives of the method bronhografii.

**Tests for self-control**:

With the development of piopnevmotoraksa in abscesse light is primarily shown: 2

1. jendobronhialnoe introduction proteolytic enzymes

2. drainage of pleural cavity

3. antibiotics

4. radiation therapy

5. administering cytostatics

Complication of acute lung abscess may not be: 5

1. breakthrough in the cavity plevralnuu abscess

2. bleeding

3. aspiration of pus in the healthy lung

4. sepsis

5. education dry cavity in the lung

For gangrene of the lung characterized by: 5

1. the development of the disease when areaktivnosti body

2. lack of granuljacionnogo shaft on the border of the lesion

3. widespread necrosis of lung tissue

4. the expressed intoxication

5. all of the above is true

List the main periods in the clinical picture of acute lung abscess: 2.5

1. the period of the alleged well-being

2. the period until the autopsy in bronchus

3. period.

4. the period of decompensation

5. the period after opening in bronchus

List the additional methods of examination of the patient with lung by: 2.4

1. EXAMINATION

2. Lung roentgenoscopy

3. sigmoidoscopy

4. radiography

**ACUTE AND CHRONIC EMPIEMA PLEURA**

**Theoretical reference.**

Empiema pleura is a purulent or gnilostnoe inflammation, developing in the pleural cavity with involvement in the pathological process of parietal and visceral pleura. The term "empiema (in Greek-ulcer) was in use at the time of Hippocrates to designate removes pus in anatomically predugotovlennoj cavity (empyema of the gallbladder). The term "empiema pleura" does not differ in meaning from the term "purulent pleurisy, but more often used in surgical practice.

Pleural empyema is often not an independent disease, and is a pathological process, complicating lung disease, abdominal (pancreatitis, poddiafragmalnye abscesses) and systemic blood diseases, connective fabric. Most often, empiema pleura complicates for acute pneumonia (5-8%), Lung abscess (9-11%), Lung gangrene (80-95%). In closed chest injuries empiema pleura occurs in 3-5% of cases, while penetrating injuries-10-15%.

**Classification empiema pleura:**

A) according to the nature of the occurrence:

a) meta-or parapnevmanicheskie;

b) postoperative;

in) post-traumatic (open, closed);

g) after pneumothorax (spontaneous or therapeutic).

B) on clinical flow:

a) sharp

b) Subacute,

in the) chronic.

Exudate in nature):   
-pus -putrefactive-specific-blended.

G) by pathogenic:

-nonspecific infection (Staphylococcal, streptococcal, pneumococcal)

-anaerobic infection

-specific (TB)

-mixed infection.

D) prevalence and localization:

-unilateral, bilateral;

-total, subtotal,

-separate (apical-top, parietal-parakostalnye, basal-above the diaphragm, mezhdolevye, paramediastinalnye).

The most important classification criterion determining the tactics of treatment and prognosis is the lack of communication of the pleural cavity with external Wednesday (closed pleural empyema) or its availability (open empiema pleura).

On volume of pathological content pleural cavity emit: total empiema pleura, subtotal and separate. On localization of separate empiema pleura are divided into: apical, parietal, basal, mezhdolevye, paramediastinalnye.

Allocate parapnevmonicheskie empiema pleura (a combination of pneumonia, empiema pleura) and empiema pleura with destruction of lung tissue (lung abscess, gangrene of the lung).

The so-called metapnevmonicheskie empiema pleura represent suppuration abacterial parapnevmonicheskogo Pleurisy or hydrothorax, recognized in a timely manner. Emit a special kind of open empiema pleura with destruction of lung tissue is piopnevmotoraks.

By the nature of the changes in the walls of the pleural cavity and clinical flow empiema to 8 weeks is regarded as urgent, and after 8 weeks after emergence-as chronic.

**The clinical picture of** acute empiema fold from syndrome of Pyo-resorptive fever with hectic panache temperature curve, weakness, oznobami, headache, and if there is no adequate treatment is the development of syndrome coherent multiple organ failure, with decompensation of the functions of the vital organs.

When inspecting the chest-lag affected parties in respiratory movements, bulging mezhreberij. Voice trembling in the zone of maximum accumulation of exudate. Percussion is determined by the blunt sound on accumulation of fluid, it is located in the slanting direction (Demuazo line), in the presence of air in the pleural cavity is defined by a horizontal border blunting, and above it is timpanit with metal shade. If auscultation-absence of respiratory noises on the side of the lesion.

The reason for the transition of acute to chronic empiema, frequently late or inadequately used treatment vsledstie which manages, as a result of purulent pulmonary parenchyma destruction, form bronhoplevralnyj fistula, in the consequence and supports the existence of chronic empiema pleura.

Pyo-destructive process in the pleura, raises a number of severe changes in the body by the heart, liver, kidneys, characteristic of long festering intoxication.

When the shell piogenna bacterial enzymes pus may grow beyond the pleural cavity to form ulcers between the muscles of the chest, under the skin or opened outwards. Can also occur spontaneously in purulent breakthrough pericardial cavity, esophagus, through the diaphragm into the abdominal cavity.

Of instrumental Diagnostics is a leading x-ray study. The easiest and most informative method of roentgenologic examination is Plantar fluoroscopy. It allows you to precisely localize the pathological process, determine the degree of separating exudate and also quite accurately determine its volume. To determine the exact size of jempiemnoj cavity, its configuration, condition of the walls, verification and localization of broncho-pleural message is Plantar plevrografija.

Endoscopic techniques (bronchoscopy, thoracoscopy), as well as ultrasound scanning allow you to get a more detailed picture of the nature of morphological changes in pleural sheets and pleural cavity.

The final method of verification of diagnosis is pleural puncture: getting purulent contents from the pleural space suggests the diagnosis empiema pleura is absolutely reliable.

**Differential diagnosis**.

Jempiemu pleura must be differentiated from specific (mikoticheskim, TB) pleural lesion when the primary process precedes development empiema pleura. For correct diagnosis must be focused study of exudate (Mycobacteria, fungi), puncture biopsy of pleura, serological samples as well as thoracoscopy with biopsy. In addition, jempiemu pleura, one has to differentiate the following pathological conditions: gidrotoraksom, haemorrhagic pleuritis subplevralnymi jehinokokkovymi cysts, chronic by light.

Gidrotoraks(fluid in the pleural cavity)-occurs when levojeludockova congestive heart failure, and quite often the subject of differential diagnosis with acute empyema, as tends to become infected and Navigate in acute jempiemu pleura. A number of studies have proven fairly substantial decline in immunity in patients with heart failure, which makes them susceptible to microbial factors.

The hallmark of with some commonality of x-ray pattern is the absence of phenomena gidrotorakse Pyo-resorptive fever and purulent intoxication. However, if questionable clinical and radiological picture helps pleural puncture with the study of the resulting content. For transsudata typical relative density 1001-1015, exudate, 1016-1025 May 30-50 protein content in g/l, transsudate-1-2 g/l assay Livolta on serozomucinov content in may always positive, cell composition in may dominated by leukocytes, transsudate 10-20 of them in sight, dominated by lymphocytes.

Nagnoivshijsja hemothorax is also predstadiej acute empiema pleura, since without adequate discharge his punkcijami he likely can go in acute jempiemu.

Radiographically painting them is very similar. In the study received during puncture of blood samples run Petrova and Efendiyev (Petrova-identification of erythrocyte Lysis "lacquered blood-saline 1:5 punctate dilution. tube pour in some amount of blood from the pleural cavity and dilute with 5 x the amount of distilled water. In neinficirovannoj blood through 5 minutes there is complete hemolysis and the liquid becomes transparent varnish. If blood is pus fluid becomes cloudy with sediment hlopevidnym fibrin; Efendiyeva-change in the ratio of plasma and uniform. Punktat in vitro defend or centrifuged. When two layers-upper (plasma), the lower layer-shaped elements. If the blood is not infected, the correspondence between plasma and formennami will be 1:1. In infected blood, this ratio is changing in the direction of fluid buildup and reduce draught-murk (fibrin).

Bloody pleural effusion-is a classic example of lung cancer. In the development processes of disintegration of lung tumor, clinical signs of intoxication, purulent x-ray picture, very similar to acute empyema (which can also complicate the course of lung cancer, but actually bloody pleural effusion is not requires punkcionnogo or drainage treatment, they even on the contrary can cause infection of the pleural space and development empiema), hence the importance for determining therapeutic tactics of differentiation of these States. The most reliable way is a diagnostic paracentesis. When the effusion Pleurisy hemorrhagic corresponds to the physical parameters transsudatu. On cell composition: dominated by red blood cells and neutrophils are found only isolated, you can also identify and atypical tumor cells. When bacteriological examination of the transudate is sterile.

Subplevralnye jehinokokkovye cyst-especially when inflammatory conditions are very similar to chronic empyema. However, they are characterized by discharge with phlegm scrappy shell hitinovoj and child skoleksov, Latex-Agglutination positive for antigens cyst that corresponds to jepidanamnez. Nagnoivshajasja jehinokokkovaja cyst even when full-fledged conservative treatment Unlike nonspecific chronic empiema does not go into remission phase, because it cannot get rid of scrappy hitinovoj parasite capsules that support nagnoitelnyj process. There is expressed in peripheral blood Eosinophilia (20-25%), which is atypical for chronic empiema pleura.

Chronic lung abscess is similar to chronic empyema: Clinic of chronic purulent infection, duration, circulatory flow with recurrent exacerbations and remissions. To differentiate these conditions can only be achieved with the help of x-ray issledovaniij parakostalnoj localization, and drainage pattern can be quite identical. What sets them apart with chronic empieme cavity is stretched in the vertical direction, abscesse seeks to rounded contour, with chronic empieme wall cavity with increasing disease dates, with thin abscesse are thicker . Helps in the differential diagnosis of polipozicionnoe x-ray examination and Imaging.

**Test questions**:

1. What are the causes of acute chronic empiema transition.

2. Prechislite complications of lung cancer.

3. Give classification of acute and chronic pleural empyemas.

4. List the major clinical signs of acute empiema pleura.

**Tests for self-control**:

The reason for the transition of acute empiema chronic cannot be: 4

1. a failed attempt to obliterate the cavity in the acute period

2. premature removal of drainage

3. most primary cavity

4. tuberculosis and another specific flora

5. bronhoplevralnyj fistula

Chronic pyothorax is considered: 4

1. from the second week

2. the fourth week

3. in six weeks

4. with eight weeks

5. three months

Treatment outcome empiema pleura without bronhoplevralnogo fistula depends on the nature of: 1

1. changes of visceral pleura

2. the number of pus in the pleural cavity

3. change of parietal pleura

4. change of lung tissue

5. microflora

In order to avoid hemorrhage does not recommend in one step pleural punctures to delete more: 3

1. 1000 ml liquid

2. 500 ml of liquid.

3. 3500 ml liquid.

**PNEUMOTHORAX**

**Theoretical reference.**

Pneumothorax is called air congestion between parietal and visceral pleura sheets. Most often have to deal with so-called spontaneous pneumothorax, which occurs as a result of a breach of Hermeticism in the pleural cavity and her communications with air. As a consequence, the visceral pleura exfoliation occurs on parietal.

**Classification of pneumothorax**

|  |
| --- |
|  |

Spontaneous pneumothorax

  Primary (against the backdrop of a healthy lung)

  Secondary (in the face of pathologically changed lung)   
-Against the backdrop of chronic obstructive lung disease (COPD)   
-Against the background of the infectious process   
-Against the backdrop of a tumor process

Traumatic pneumothorax

  Blunt trauma of thorax

  Penetrating wound of thorax

Iatrogenic pneumothorax (due to medical procedures)

  When handling the subclavian vein

  In diagnostic puncture

  When therapeutic puncture

According to the degree of spadenija of lung tissue.

By the presence of voltage in the pleural cavity (stress, stressful).

The availability of the communication with the atmosphere (closedgate, outdoor)

Spontaneous pneumothorax can be unilateral (more often) and bilateral. The emergence of spontaneous pneumothorax usually favors sharp physical tension. Causes include: single and multiple cysts in the lungs, tuberculosis, abscess, gangrene, pneumonia, bronhoektaza, neoplasms, pnevmokistoz, etc.

Pneumothorax can be artificially imposed for therapeutic purposes (e.g., tuberculosis), as well as post traumatic. Depending on the nature of the injury there are various conditions for communication between the pleural cavity and air. In this regard, distinguish between open and closed valve (busy) pneumothorax.

**The clinical picture**. Spontaneous pneumothorax usually occurs suddenly. In patients appears sharp pain on the corresponding side of the thorax, which increases with breathing and is accompanied by a strong and painful cough. Joins and shortness, breathing becomes difficult. In some cases, severe, prolonged pain can cause a State of shock. So heavy and acute onset of spontaneous pneumothorax due to the rapid spadeniem of the lung, mediastinum offset and large blood vessels simply the opposite of easy.

During the examination of a patient with spontaneous pneumothorax forced attention (more often than not sitting) position corresponding to the defeat of the party chest behind when breathing, there is shortness of breath.

When a physical study is pronounced timpanit, absence or lack of breathing and weakened sharply voice jitter on the side of the lesion.

Great value in correct diagnosis of spontaneous pneumothorax is an x-ray study in which is found the air in the pleural cavity, briefly.

Closed pneumothorax occurs when trauma, injuries, accompanied by minor injury of the chest wall and the lung tissue, which quickly sealed in the offset from the tissue of traumatic occlusion edema, fibrin or blood clot . Respiratory and circulatory disorders are usually mild and will soon be compensated. Closed pnevmotorakse the air in the pleural space may also flow through the damaged bronchial tubes. The growing accumulation of air in a closed cavity can cause serious functional disorder due to spadenija lung: shortness of breath, cough, subcutaneous emphysema, dizziness, reduced ad. The skin becomes pale, face and lips sinjushnymi, pulse frequently weak.

When you open the pnevmotorakse that occurs with penetrating injuries of the thorax, the pleural cavity freely communicates with surrounding Wednesday through wall defect or bronchus, develop severe breathing disorders, circulatory disorders, mild spadaetsja and essentially turns off from the Act of breathing. There comes a flotation of the mediastinum is moving it every time you inhale and exhale.

The condition of patients with severe, they are excited, afraid, suffer from severe shortness of breath, sharp pains and painful coughing. Inspection is defined by cyanosis, small and frequent pulse, drop ad, when breathing is audible characteristic flapping or vacuum sound. Often at the time of exit wounds is allocated foaming blood. X-ray study confirms pneumatic or hemothorax.

Outdoor first aid pnevmotorakse comes to imposition occluzionna bandages, hermetically closing the wound, leading into the pleural space. Necessary medical undertaking is wound closure of the chest wall with subsequent aspiracijami air and accumulating in the pleural cavity exudates. While damage lung volume of operation is determined by the nature of the damage (wound closure lung resection).

Particularly severe violations of respiratory and circulatory notes in pnevmotorakse valve, which occurs when the chest wall defect seamed or lung tissue, impermeable to air only in the direction of the pleural cavity. When the pressure in the pleural space progressively increases, Mediastinal move in a healthy direction, develops extensive subcutaneous emphysema. Air from the pleural space is also distributed on kletchatochnym spaces of the chest wall and mediastinum, causing picture intermuscular and Mediastinal emphysema. This condition is called tense pneumothorax.

Depending on the valve is formed in the chest wall or the lung tissue, distinguish between external and internal valve pnevmotoraksy. When injuries accompanied by pneumothorax, the victim status of valve is always heavy, expressed jekspiratornaja shortness of breath, cyanosis, a sharp worsening of emphysema. Notes concern the situation of forced-breathing uchashheno sharply intervention semisentados, superficial, tachycardia, a decline ad.

Radiographically detected by pneumothorax on the side of the lesion, a dramatic shift of mediastinum in the opposite direction. Dome aperture is thickened inferiorly, intercostal gaps widened in intermuscular tissue and mediastinum.

In the absence of timely assistance to victims with valve pneumothorax quickly perish.

First aid is emergency puncture the pleural cavity to reduce stress in it. Subsequent treatment depends on the nature of the existing damage and should ensure the Elimination of valve mechanism, the aspiration of air from the pleural cavity and smoothening out the light.

**Differential diagnosis**.

Pneumothorax must be differentiated from gemotoraksom, empyema.

Hemothorax is the accumulation of blood in the pleural cavity. The source of bleeding in the pleural cavity are pulmonary vessels, less chest wall receptacles, intercostal.

Blood in the pleural cavity izlivshajasja, later 5:00 loses the ability to coagulate. Based on this fact the sample Ruvilua-Grégoire, whether stalled vyjasnjajushhaja bleeding in the cavity plevralnuu: collapse reflects ongoing punctate bleeding, nesvertyvanie-on its termination. If the liquid blood gemotoraksa received 5:00 later after puncture wounds, not folded, bleeding can be considered to be priostanovivshimsja (i.e., blood is not fresh). Gemotoraksa symptoms are signs of internal bleeding: a dull sound with percussion, moving hearts in healthy aside from displacement of the mediastinum, the expansion of the bottom of the aperture and smoothing the intercostal spaces corresponding to half of the chest, weakening or disappearance of breathing noises in auscultation, lack of voice jitter. Gemotoraksa recognition is possible if the puncture and fluoroscopy.

The presence of pus in the pleural cavity is called empyema. The clinical picture of her is folded under the influence of two factors: the number of vypotnoj fluid that can cause misalignment of the mediastinum, respiratory and cardiovascular insufficiency; the nature of the liquid. Being enclosed in the pleural cavity, the fluid sucked and gives symptoms of intoxication (high temperature, weakening and increased heart rate, etc.).

All forms of acute empiema pleura have in common: chest pain, cough, phlegm, shortness of breath, fever, pulse 110-120 beats/min. Perkutorno defines the sense, when piopnevmotorakse-dullness with the upper horizontal border and timapanicheskij sound above it, respectively, the accumulation of air. Auscultation taped weakening or almost complete absence of breathing noises in the crowd places exudate.

Change of blood empieme pleura are typical for acute purulent process: the increase in the number of leukocytes, leukocyte formula has seen a dramatic shift to the left, increased ERYTHROCYTE SEDIMENTATION RATE.

When x-ray study of purulent pleurisy gives continuous dimming or obliquely running lines. Border line corresponds to line Demuazo. It also notes the low standing of the diaphragm.

Diagnosis is completed test protocol in YII or VIII intercostals space. Extracted pus is subjected to microscopic and bacteriological research.

**Test questions**:

1. Give the definition of a pneumothorax.

2. What are the main causes of pneumothorax.

3. List the types of pneumothorax.

4. Mechanism of tense pneumothorax.

5. What are the methods of examination of patients with pneumothorax.

**Tests for self-control**:

The most common cause of spontaneous pneumothorax serves: 4

1. Lung abscess

2. lung cancer

3. bronchiectasis

4. Bullous lung cysts

5. Lung atelectasis

For a tense pneumothorax is not characterized by: 3

1. squeeze the affected lung

2. reduction of venous inflow to heart

3. offset the mediastinum in the affected side

4. increased pressure on affected side

5. sonorous heartbeat

For acute pneumothorax is not typical: 3

1. shortness of breath at rest

2. pain up to shock

3. the horizontal level of the fluid in the pleural cavity

4. tachycardia

5. change the sound perkutornogo

First aid for the valve pnevmotorakse: 1

1. urgent pleural puncture

2. the overlay occluzionna bandages

Outdoor first aid pnevmotorakse: 2

1. urgent pleural puncture

2. the overlay occluzionna bandages

**OBLITERATING DISEASES OF ARTERIES OF EXTREMITIES**

**Theoretical reference.**

**Classification**. To obliterating diseases of arteries of extremities, most commonly found include:

1. obliterating atherosclerosis,

2. obliteriruty endarteriit

3. Raynaud's disease (angiotrofonevroz)

4. Buerger's disease (thromboangitis migrating).

According to the degree of circulatory disorders in limb entails the following stage of the disease (Pokrovsky A.v.):

(I) phase-compensation for circulation;

(II) and stage-subcompensation circulation;

(II) b-stage primary circulatory decompensation;

(III) stage-circulatory decompensation;

(IV) stage-destructive changes in the tissues of the limb.

**The clinical picture of**clinical picture of disease depends on the nature of the vascular lesions of the limbs and the degree of circulatory disorders in the region, krovosnabzhaemym affected vessels. (I) disease stage-functional compensation (spastical form)-the patient it may take more than 1000 m before the advent of peremejateisa hromota; 2A-stage or subcompensation stage, the intensity increases and peremejateisa hromota occurs when passing 200 meters; stage 2B-peremejateisa hromota occurs when passing from 50 to 200 meters; 3 phase-phase decompensation. Pain in the extremities appear alone, intermittent claudication occurs when passing 25-50 meters. 4 stage-stage of the destructive changes. Pain in the limbs become permanent, intolerable.

**Differential diagnosis**.

Case endarteriitom more often males under the age of 40 years. Important etiological factors are: chronic intoxication, round limbs, smoking, stress. Sick obliterating endarteriitom have a youthful appearance. Defeat begins with small vessels of the extremities, usually after a nervous overstress and fatigue. At the beginning of the disease indicated fatigue, cold extremities, paresthesia. The disease evolves to become permanent, pain appear trophic disorders.

For obliterating endarteritis is characterized by the following symptoms: symptom and trial Oppelja-Burger, a symptom of stiffness of fingers-Krakow, symptom, Burdenko, Samujelsa Goldflama, panchenko.

Instrumental methods of the diagnosis in the early stages of the disease define changes ripple and the volume of blood vessels, the reovazogramme decline in blood and reduce collateral blood flow. Doppler determines the sharp decrease in peripheral blood in flotation. The dilatation noted obliteration distal arteries. The walls of arteries are smooth, and they gradually narrow lumen. Collateral blood flow is not expressed.

In anamnesis in patients often marked transferred infectious diseases. Characteristic obliterating endarteritis is the appearance of necrosis in preserved ripple on the femoral and popliteal arteries affected limb.

**Obliterative atherosclerosis.** Develops after the age of 40 years. patients look older than their age. The disease develops slowly, as connected with the violation of carbohydrate and lipid metabolism disorders. contribute to trauma, cooling the body often. violations of the patients of coronary and cerebral circulation, they suffer from hypertension, diabetes. Marks constant paleness of limbs. Circulation distal limbs for a long time is compensated. Often taped Leriche syndrome, i.e. blockade at the level of the bifurcation of the aorta and common iliac arteries. Also as with obliteriruuschem endarteritis expressed symptoms of ischemic Samujelsa, Goldflama, panchenko, Burdenko and others. For obliterating atherosclerosis characteristic symptom "empty veins", manifested in the desolation of venous trunks after the lifting of the lower limbs. In the blood of patients have hypercholesterolemia.

On reovazogramme detected a decrease in blood flow on main arteries, with collateral blood flow is maintained. According to doplerografii, there was a reduction in the flotation index and vascular lesion on segmentarnomu type. The angiogrammah identifies the dense, izedennost aorta deformed outlines main arteries, often contains plots of vascular calcifications. Often missing pulse on femoral vessels. In the absence of pulse on femoral arteries do not always have plots necrosis on foot.

**Raynaud's disease (angiotrofonevroz).** Suffer, usually young women (aged 20 to 30 years) psihostenicheskoj Constitution or suffering from neurosis. Small-calibre arteriol spasm occurs, particularly in the area of the phalanges and toes, nose, ears. Pulse on peripheral arteries preserved large trunks are not affected. Pain, pallor and cold toes appear suddenly, are often intense conditions of low ambient temperature Wednesday. In the later stages of the Blanching of the skin is replaced by cyanosis and then develop nutritional disorders in the form of dry zones of necrosis in the area of the surface layers of nail phalanxes of fingers. In a period of pronounced symptom with cold stress and symptom of Krakow.

**Buerger's Disease** (migrating obliterans). The disease occurs in young men after suffering superficial thrombophlebitis. In the course of superficial veins appear plots in the area of seals trombirovannyh inflamed veins with pain by palpation. After weakening of acute inflammation of the same zone thrombophlebitis appear in other parts of the superficial venous network. After some time (2-6 months) sick note cold extremities, paleness of skin, and then expressed signs of arterial blood supply is intermittent claudication, positive ischemic symptoms, trophic disorders. The cause of the disease is autoimmune process with epithelial lesions of the peripheral vessels, followed by tromboobrazovaniem in their lumen. The clinic and the stage of the disease are similar to manifestations in obliterans endarteritis, however, when the migrated trombangiite marked periods of remission and exacerbation of the process. The disease is progressive and often ends with the development of irreversible trophic violations tissues and gangrene of limbs.

**Acute circulatory extremities.** Develops suddenly. Precede: atherosclerosis, myocardial infarction, abnormal heart valves, the active phase of rheumatism, slowing blood flow, giperkoaguljacionnyj syndrome, trauma, inflammatory changes receptacles. Cause of acute circulatory disorders are thrombosis and embolism.

When emboliah major arterial trunks intensive pain spreading occurs distal to the place of destruction. Pulse below the obstacles is absent. Skin pale colouring, cold to the touch. Broken motor function of limbs and all kinds of sensitivities, hyporeflexia. For progressive, stormy.

When tromboobrazovanii, the vessel lumen, there has been a prodrome: cramps, paresthesia, numbness in the extremities.

When the instrumental study noted a sharp decline in circulation below the level of the blockade. The arteriogrammah visible level of the blockade as a sharp Cliff kontrastirovannogo trunk receptacle. Collateral blood track is not defined.

**Test questions.**

1. Causes leading to the development of obliterating endarteritis obliterans and atherosclerosis.

2. Features of vascular lesions characteristic obliterating endarteritis, Raynaud's disease, arteriosclerosis, Buerger's disease.

3. List the angiographic signs characteristic obliterating endarteritis obliterans and atherosclerosis.

4. Specify the clinical and evolutive particularities characteristic obliterating endarteritis, obliterating atherosclerosis, Buerger's disease, Raynaud's disease.

**Tests for self-control**:

30 years of patient complains of severe pain in her appearance in fingers, toes, mouth feeling of paresthesia. On examination: skin fingers pale, sinjushnaja, cyanosis of the nail. Ripple on the radial artery. Positive cold tryout.

Your diagnosis is as follows: 2

1. Obliteriruty endarteriit

2. Raynaud's Disease

3. Obliterating atherosclerosis

4. Buerger's Disease

Patient 41 year. Sick 3 years. Complains of pain in right foot. Does not sleep because of the pain. Intermittent claudication through 50 m, parestesia, freezing On the right foot. (I) toe of the right foot trophic ulcer. Ripple on the arteries of the foot is not determined. The femur and popliteal arteries pulsing saved.

Your diagnosis: 4

1. Obliterating atherosclerosis

2. Postthrombophlebitic disease

3. Buerger's Disease

4. Obliteriruty endarteriit

5. Raynaud's Disease

Sick of 62 years. Ill 15 years. Complaints of pain in the calf muscles during walking. Intermittent claudication through 100 meters. At night there is no pain. Trophic disorders on the limbs moderately pronounced. Pulse on femoral arteries is determined by good. Pulse on hamstring and arteries stop is not defined.

What are the stage of atherosclerosis obliterans: 3

1. 1 stage

2. 2 stage

3. stage 2B

4. 3 stage

5. 4 stage

Patient 36 years. Sick 5 years. complaints of pain in the left lower limb lameness peremezhajushhujusja. Pain is persistent t , periodically appears swelling of the foot and lower leg; notes hyperemia, pain and compaction on the go, povyshenietemperatury the body. During the examination of the foot and lower leg, otechny cyanotic skin-pink colour, subcutaneous veins dense formations. Pulsation of the arteries of the foot is missing.

Specify the diagnosis: 4

1. Obliterating atherosclerosis

2. Obliteriruty endarteriit

3. Raynaud's Disease

4. Buerger's Disease

5. Acute superficial thrombophlebitis

**THROMBOSIS AND EMBOLISM OF PERIPHERAL ARTERIES OF THE EXTREMITIES**

**Theoretical reference.**

Thrombosis and embolism lead to the development of acute arterial obstruction and ischemia tissues in the region blocked circulation. Embolism and thrombosis could not be regarded as separate diseases, they are always the consequence of jembolo-and trombogennyh diseases.

Thrombosis of arteries are characterized by prizhiznennym education tromboticheskim masses in the lumen of the vessel, as a consequence, the integrity of the vascular wall, slowing blood flow or changes in hemostasis system (Virchow triad).

High frequency of thrombosis in people suffering from obliterating diseases of the arteries, diabetes, and the traumatic injuries of the extremities, the compression of blood vessels. Thrombosis often develop after diagnostic and therapeutic manipulation and operations on the receptacles. Thrombosis can occur against the backdrop of some infectious and hematological diseases.

Embolism is a blockage in the arteries of the jembolom (often organic), brought in from other regions of the artery blood. In the vast majority, causes embolisms are heart disease (myocardial infarction, aortic aneurysm, heart disease, etc.). Sources of emboli may be abdominal aortic aneurysm, aortic ateromatoz. Almost all patients with jembologennymi disease notes rhythm (atrial fibrillation). Factors influencing embolism are meteorological tolerance patients, magnetic disturbance in nature.

Most often, the embolism occurs in the lower extremities and the aorta. More likely to clog up the femoral artery, then-hip, shoulder, Combined embolism occur in 10-12% of cases. Repeated embolism develops in -8 7.5% of cases.

The place of fixation of emboli in arteries limbs depends on many factors, among which are: value dimensions of arteries and circulatory features in the region of defeat.

Tromboticheskaja occlusion or embolization of the lumen of arteries limbs may eventually fail or severe acute ischemia and gangrene limbs respectively, partial or total trade round-trip capability of acute circulatory disorders. With acute jembolicheskaja arterial blockages not only fixing jembola in arterial trunk, but also other factors: arterial spasm, secondary progressive thrombosis.

**Classification of** acute limb ischemia (V.s. Savelyeva) 1. voltage ischemia (MI)-signs of ischemia during exercise; 2. I a-a sense of numbness, cooling, parastezii; 3. I b-persistent pain in the limbs; 4. II and the emergence of paresis; 5. (II) b-quadriplegia; 6. III and the subfascialnyj swelling; 7. III b-partial collective Contracture occurs; 8. (III) in-total CONTRACTURE.

**The clinical picture**. Clinical manifestations of acute circulatory disorders in thrombosis and embolism are pain in the affected limbs, impaired sensitivity to varying degrees of manifestation, movement disorders.

In a survey of patients detected: blanching and cooling of the skin of the leg, absence of vascular pulsation distal to the level of destruction, the retraction of the superficial veins. With the progression of ischemia noted the emergence of contractures and subfascialnogo development of edema. Also suffers and the general condition of the patient-it is progressively getting worse.

For acute Embolic arterial occlusion is characterized by sharp beginning the process on the background of jembologennogo disease. For thrombosis, developing on the background of chronic disease, characterized by latent period of ischemia with possible progression or, if favourable, devolution of ischemia.

Acute occlusion tromboticheskaja more likely to occur in people over 50 years old, with chronic Obliterative diseases of arteries of extremities. Modern diagnostic methods are most informative: Doppler, duplex angioscanning with color coding blood flow to evaluate the extent and nature of impaired blood flow, as well as radiology angiographic study in the region the defeat. With a view to the diagnosis of jembolo-and trombogennyh diseases used ECG, phonocardiography, radiography, investigation of blood coagulation, biochemical blood analysis, etc.

Scope and methods of conservative and surgical treatment of a particular patient is determined depending on the extent of the violation, the circulation and character background and concomitant diseases.

**Differential diagnosis**.

Often acute thrombosis and embolism of lower extremities arteries have to differentiate from acute Ilio-femoral venous thrombosis.

The beginning of the disease severity similar to picture of acute arterial circulation, the sudden emergence of pain in limbs with consequent numbness of limbs, skin becomes pale, cianotichnoj. Painting can develop flaccid paralysis loss of sensation and movement.

The main differential-diagnostic sign of this illness serves limb swelling that comes to the inguinal folds, that never occurs with thrombosis and embolism. The consistency of swelling soft. When cerebral swelling is always tight and subfascialnyj.

When hyperthermia notes Phlebothrombosis of the skin of the leg. Pulsation of the arteries is defined throughout the leg. The most informative method of differential diagnosis is Doppler vascular bed and angiography.

Along with vascular disease acute arterial occlusion must be differentiated from the pathology of the Central and peripheral nervous system. In particular, the lateral mielitom. The disease is characterized by the development within 1-2 hours of flaccid paralysis of the lower limbs, accompanied by loss of sensitivity. Correct diagnosis allows you to set the history and study of pulsation of the arteries.

**Test questions**:

1. What are trombogennye diseases causing acute peripheral arterial thrombosis.

2. List jembologennye diseases causing peripheral arterial embolism of extremities.

3. What are the clinical signs of acute arterial circulatory disorders of extremities.

4. The main methods of diagnosis of acute arterial limb circulation.

**Tests for self-control**:

Ill 35 years suffers from rheumatism 20 years, atrial fibrillation appears periodically. Among the relative prosperity ill suddenly arose a severe pain in his right hand, feeling numb her loss of sensitivity. The patient could not independently raise their hand. When inspecting hand Deathly pale, no edema. Kubitalnoj pulsation and peripheral arteries is not defined. Introduction effect of antispasmodic agents.

Your diagnosis: 2

1. acute coronary arteries the right upper limb

2. right upper extremity artery embolism

3. sharp left plexitis

4. Rheumatoid Polyarthritis, acute exacerbations.

Sick 60 years was treated in the cardiology department at the hypertensive disease, ischemic heart disease. Suddenly she developed severe pain in the left lower limb. Movement in the ball of the foot and toes of the foot became difficult. The pain intensified. Skin pale with cianotichnym shade. Ripple on the popliteal artery and tibial arteries were not determined.

Your diagnosis: 2

1. acute deep Phlebothrombosis

2. acute embolism artery left lower limb

3. acute thrombosis of arteries of the left lower extremity

4. acute violation of cerebral circulation-hemiparesis. A patient 58 years earlier did not ask for medical help. After performing heavy physical work in the garden felt severe pain in his right leg, numbness, loss of its supporting function. When inspecting the skin pale. Movement in the joints is limited. Pulse in the arteries of the extremities is not defined.

Do you think that the patient has: 2

1. acute thrombosis of arteries of extremities

2. acute artery embolism of limbs

3. acute deep Phlebothrombosis limb

4. acute sciatica on the right

Sick of 49 years. Sick obliterating atherosclerosis of lower extremities arteries 7 years. Regularly treated in hospital. After drinking spirits appeared a growing pain in my left leg, cold extremities, paresthesia. When inspecting the skin pale's left leg. Movement in the foot limited, broken skin, pain sensitivity to the lower third of the thigh. Pulse on femoral artery weakened systolic murmur. Pulse on peripheral arteries limbs is not defined.

Your diagnosis: 1

1. acute thrombosis of arteries of extremities

2. acute artery embolism of limbs

3. acute deep Phlebothrombosis

**VARICOSE DISEASE**

**Theoretical reference.**

Under the varicosity refers to abnormal peripheral veins of extremities.

Varicosity in Russia suffers nearly 30 million. people, of whom 15 per cent have trophic disorders. High prevalence of the disease, a large number of recurrences require timely diagnosis and adequate treatment of varicosity.

Varicosity of the lower extremities is polijetiologichnoe disease which in the genesis of importance are: heredity, obesity, violation of neuro-hormonal status, lifestyle features, weakness, congenital venous wall angiodisplazii, excalation veins valve apparatus, etc.

**Classification**.

Clinical classification (CEAR):

Class-0-no symptoms of the disease during inspection and palpation;

Class-1-Telangiectasia and reticular veins;

Class-2-Advanced esophageal veins;

Class-3-presence of edema;

Class-4-skin changes (pigmentation, venous eczema, lipodermatoskleroz);

5 class-skin changes, zazhivshie ulcers;

class-6-skin changes and an active ulcer.

In varicose veins include: form of the disease, the severity of chronic venous insufficiency and complications arising from the disease. The following forms of varicose veins:

1. vnutrikozhnyj and subcutaneous varicose veins without pathological venous reset;

2. segmental varicose veins with reflux of superficial or incompetent perforate veins;

3. common varicose veins on surface and incompetent perforate veins;

4. varicose veins with reflux in the deep veins.

According to the degree of chronic venous insufficiency allocate:

0 art. -missing venous insufficiency;

1 art. -syndrome of "heavy legs"-transient edema;

2 tbsp. -persistent edema, hyper or hypopigmentation, lipodermatoskleroz, eczema;

3 tbsp. -venous trophic ulcer.

Complications-varicosity hemorrhage, thrombophlebitis, trophic ulcers.

**The clinical picture**. In the early stages of varicose veins usually appear gemangiojektazii or advanced esophageal subcutaneous veins. Only a few years or even decades may appear varicose in the basins of small or large subcutaneous veins.

When varicose disease begins with the emergence of common venous nodes, you can select two options for development of the disease: 1. the appearance of varicose veins in the lower leg indicates a preference to defeat the perforant veins; 2. the appearance of varicose veins initially on the thigh and an anteromedial surface of the tibia, the popliteal fossa indicates a vedujushhuju role in the development of disease high Weno-venous reset. Regardless, with a predominance of any dumping of leaking the disease, joining the symptoms of chronic venous insufficiency occurs in the same way.

In the majority of patients through 3-5 years after the appearance of varicose veins are marked functional disorders (complaints about the feeling of heaviness in the legs, pain in the leg, foot and lower leg sponginess) that occur at the end of the day. As the illness progresses, the rising phenomenon of venous insufficiency-swellings become more pronounced and persistent, increasing heaviness in the legs, show signs of violations as trophic zones hyperpigmentation, trophic ulcers, located mainly on the medial surface of the tibia.

Often complicated varicose disease in later stages, thrombophlebitis, Phlebothrombosis, varicose bleeding sites.

Very important in clinical practice is the differential diagnosis of various diseases, involving the development of varicose syndrome.

As a result of the commonality of many clinical manifestations of various pathological conditions, especially, varicose and posttromboflebiticheskoj diseases, congenital venous Dysplasias, lymph outflow any major violations diagnostic difficulties.

When diagnosing varicose veins should solve the following tasks:

1. confirm the presence of pathology of the venous system of the limbs;

2. identify reflux on subcutaneous and incompetent perforate veins;

3. assess the condition of the venous drainage in the deep veins;

4. differentiate the nature of pathological changes in the veins (depending on the disease).

To meet these challenges, it is necessary to carefully collect medical history and patient complaints. The most characteristic manifestation of the defeat of the venous system of the lower limbs is fatigue in the legs after a long stay in ortostaze against. On clinical examination, you must examine both lower limbs, as well as the inguinal area and the front wall of the abdomen. Palpation to detect defects in the fascia of the relevant output insufficient perforant veins. Percussion sample allows you to diagnose valve insufficiency trunk subcutaneous veins. You should always determine the pulsating arteries of the extremities.

When performing various physical samples (Delbe Troyanova-Trendelenburg,-Pertesa, Pratt, Gahenbruha, etc.) the frequency of false-positive or false negative results reaches 60%. In this regard, modern Diagnostics varicose veins should be based on the data of the special methods of instrumental studies.

Ultrasonic dopplerography is a priority, which allows you to reliably estimate the permeability of veins, as well as to identify the most pathological Weno-venous refluxes. The most informative survey is duplex angioscanning with color mapping subcutaneous and perforant veins. Application of ultrasonic methods to assess the condition of the venous system of the lower limbs, allows you to dispense with radiopaque venography.

**Differential diagnosis**.

In clinical practice, most often have to the differential diagnosis of varicosity with posttromboflebiticheskoj Dysplasia, congenital disease, impaired lymph outflow, femoral hernia.

**Postthrombophlebitic disease** -varicose form, it occurs mainly in the age of 40-60 years, usually after suffering previously deep Phlebothrombosis. Distinguish: occlusal, rekanalizacionnuju and mixed forms of the disease, depending on the violation of the patency of the deep veins of the leg. Most often, secondary varicose superficial veins, appears a few months from the beginning of the sharp period of deep Phlebothrombosis and is localized on the tibia, hip, pubic hair and the anterior abdominal wall, depending on the level of okkljuzionnogo violations of venous outflow in the deep veins.

With the progression of chronic venous insufficiency in the clinical picture of the disease appears otechny syndrome, then signs of tissue trophism (hyperpigmentation or hypopigmentation, lipodermatoskleroz, eczema, trophic ulcers) .

Emit: oedematous, ulcer, pain, varicose and mixed forms of the disease.

Unlike varicose veins the first sign of the disease is a swelling, which after a night's sleep is significantly reduced. Feeling of heaviness in the legs, there is pain in the first few weeks of illness, and trophic disorders develop through 3-5 years are often circular nature, quickly progressing.

A significant role in clarifying the nature of the venous outflow has ultrasound venous drainage system.

**Congenital arterial dysplasia** arise from violations of the Embryological Development of vessels most often in clinical practice are found sick infants and young children with low-flow-arterio-venous fistulae (shunts). Under the influence of blood pressure of the walls of veins thin, veins increase significantly in diameter and often on the lateral surface of the femur and tibia, appears varicose countrywide subcutaneous and skin veins.

    Varicose veins are often observed on the anterior abdominal wall and pubis. To touch the skin over enlarged veins hot. Swelling often noted in the distal limbs divisions and is ongoing. Patients constantly bothered by heaviness in the affected limb. Trophic disorders appear in 25-30 years of age and are located on the lateral surface of the tibia. The affected extremity, long healthy at 3-5 cm, characterized by: acromegaly, hypertrichosis.

Unlike varicosity patients notes: Vascular blemishes on the skin in the form of Hemangiomas, venous pulse is defined and often bugged systolic and diastolic Rumble in the ground arterio-venous fistulae.

The most informative methods of research are angiography and ultrasound duplex angioscanning with color coding.

**Violation of lymphatic drainage** (elephantiasis, limfjedema) develops due to congenital hypoplasia of the lymphatic system of the limbs, or after repeated skin Erysipelas limbs, with limfangoitom and lifadenitom. Often, the violation of the lymphatic drainage, associated with damage to the lymphatic vessels and nodes for injuries. Violation of the lymph, lymphatic vessels leads to increased distal to the occlusion, occurs lymph.

The disease develops slowly, swelling gradually growing, becoming dense and do not disappear after horizontal rest. Limb dramatically increased in volume, the skin tight folds. Sometimes there is lymph propotevanie on the skin.

In later cases, the swelling becomes dense, due to the complete replacement of subcutaneous adipose tissue connective tissue. Disturbed trophism of tissues appear eczematous patches, ulcerations. In contrast, Chronic Venous Insufficiency, edema with elephantiasis, continuous and dense. No grid varikozno expanded veins. Venous pressure is not promoted.

According to the ultrasound deep veins intact. Superficial veins are not extended. Limfografija gives you the ability to determine the level of occlusion and the presence of dilated lymph vessels.

**Femoral hernia.** disease is characterized by the appearance of singling out the round shape, located below the pupartovoj ligament. For femoral hernia may be adopted advanced venous site, the confluence of great saphenous vein in femoral. Hernias ' setting allows you to define a hernial gate. Femoral hernia is characterized by symptoms of kashlevogo Jolt and size increases during natuzhivanii. Unlike a hernia, if press Vienna below site a finger or raise the lower extremity varicose spadaetsja node.

**Test questions**:

1. What are the causes of development of primary varicose veins of the extremities.

2. List the functional tests to determine the soundness of limb veins valve apparatus and deep venous patency.

3. Instrumental Diagnostics methods applied in patients with chronic venous insufficiency.

4. What are the ways to prevent varicose veins.

**Tests for self-control**:

What research you can determine the status of the kommunikantnyh veins valve apparatus (choose correct answers):

2, 5.6

1. the remaining sample-Trendelenburg

2. Delbe trial-Pertesa

3. reoflebografija

4. phlebography

5. sample Shejnisa

6. duplex scanning of the veins

With the help of the following studies you can define insolvency valves superficial veins (choose correct answers): 3.5

1. sample Shejnisa

2. Delbe trial-Pertesa

3. the remaining sample-Trendelenburg

4. Pratt-trial (II)

5. sample Gahenbruha

Complications include the varicosity: 3, 4, 6

1. Lymphedema

2. lymphorrhagia

3. bleeding

4. thrombophlebitis

5. subcutaneous tissue induration

6. trophic ulcer

For congenital venous dysplasia characterized by: 2, 3, 5

1. temporary swelling in the affected limb

2. increase limb along the length of the

3. increased skin temperature in the area of dilatation

4. the presence of dense Lymphoedema

5. trophic ulcer

For elephantiasis (Lymphedema) is characterized by clinical symptoms: 4

1. varicose veins, subcutaneous

2. trophic ulcer

3. transient swelling of limbs

4. dense permanent swelling of limbs

Patient 37 years admitted with complaints of left leg swelling, varicose veins on the hip and lower abdomen, often recurrent ulcer prey on the inner surface of the left tibia. The above complaint appeared 3 years ago after surgery, appendectomy, when developed swelling and cyanosis of the left lower extremity. According to the ULTRASOUND found that deep vein of tibia and femoral vein rekanalizovany, has left iliac vein occlusion and perforantnyj reset deep vein blood to surface in the lower third of the tibia. Select the correct diagnosis: 3

1. varicosity of left lower extremity in the stage of decompensation

2. varicose disease of lower limbs left the stage.

3. postthrombophlebitic disease of the left lower extremity trophic disorders under

4. acute ascending throm great saphenous vein of the thigh

5. acute deep vein thrombosis of left lower limb

Sick 22 years came into the clinic with a diagnosis of both lower limbs varicosity. The disease appeared in 16-years of age. When inspecting noted that there are sharp varicose in the basins of large and small subcutaneous veins, varikoznoizmeneny their branches. In the area of ankles a sponginess tissues. Find out from this sick cause varicose veins will allow the following diagnostic tests:

1. Ultrasonic Doppler

2. rising distal functional phlebography

3. radioindikacija with mechennym nine fibrinoguenom

4. thermography limbs

5. sfigmografija

Select the correct combination of answers: 1.2

a) 1.2

b) 1, 2, 4

in) 2, 3, 4

g) 3, 4, 5

d) all true

**ACUTE SUPERFICIAL THROMBOPHLEBITIS**

**Theoretical reference.**

Under the acute thrombophlebitis understand inflammation of the walls of the veins, with formation of a blood clot in her skylight.

Often pungent superficial thrombophlebitis of lower extremities varicose and posttromboflebiticheskoj is a complication of diseases. Therefore, at present, the term is widely received acute varikotromboflebit. Disease is relentless and often recurring for a long time.

Development of thrombophlebitis: contribute to significant changes in the walls of the veins increase clearance, slowing blood flow, changing the adhesive-aggregacionnyh properties of loose blood.

**Classification**. Emit:

1. acute superficial thrombophlebitis in the pool a great saphenous vein;

2. acute superficial thrombophlebitis in the pool of small saphenous vein;

3. acute superficial thrombophlebitis in the pool of large and small subcutaneous veins.

Most common are distinguished: a) local; b) progressing upward.

Distinguished: uncomplicated and complicated (PULMONARY ARTERY THROMBOEMBOLISM, deep vein, periflebit and paravazalnaja Phlegmon).

**The clinical picture**. As a result of the surface location of subcutaneous veins acute superficial thrombophlebitis is bright the clinical picture. While prevail local symptoms: hyperemia skin, infiltration over the affected varicose Vienna, pain, lack of overall limb edema. The main symptom is pain in the course of trombirovannoj of Vienna, increasing tenderness, physical activity. When inspecting a patient in both vertical and horizontal positions, palpiruemyj painful tension bar does not change size and configuration. Most patients general condition remains satisfactory.

Particular attention should be paid to the localization of thrombophlebitis. The presence of an inflammatory infiltrate in the projection of the main stem large or small subcutaneous veins extending proximally to the mouth, defined as acute ascending thrombophlebitis. This condition is very dangerous due to increasing risk of thromboembolitic complications (TAL).

The presence or absence of the ascending nature of thrombophlebitis and determines the tactics of treatment of each patient.

Laboratory Diagnostics assign ancillary: analyses of the blood there is Leukocytosis, increased ERYTHROCYTE SEDIMENTATION RATE, hemostasis system is investigated. Crucial in confirming the diagnosis is given ultrasound duplex scanning veins (veins or ultrasound), which allows to determine with absolute accuracy the extent of thrombosis, its boundaries, set fixed flotiruet or thrombus (free floats). Later after determining treatment tactics prescribed ULTRASOUND EXAMINATION of abdominal cavity organs (often cause thrombophlebitis may be Cancer Pathology), x-rays of the chest.

**Differential diagnosis**.

Acute superficial thrombophlebitis should be differentiated from rozhistym inflammation, lymphostasis, primary varicose superficial veins, Phlebothrombosis.

**Rojistoe inflammation** is acute serum, progressive inflammation of the skin, less mucous membranes caused by Streptococcus mutans. Emit: jeritjematoznuju, bulleznuu, bullezno-necrotic forms of Erysipelas. Arise: pronounced swelling, hyperemia skin, sharp temperature rise of local and General, rezchajshie pain at the slightest touching the surface of the skin. Subsequently formed "bubbles" (Bulla) with izgyazwleniem and skin necrosis. Unlike the superficial thrombophlebitis, Rózsa had no clear connection with the localization and the venous system. When necessary, the ULTRASOUND of the veins, precluding acute thrombophlebitis.

**Lymphangitis is** an acute inflammation of the lymph vessels, which takes place in the form of a mesh or stvolovogo lesions, is the primary complication of Pyo-inflammatory process of the limbs. There is congestion in the form of longitudinal stripes, patients are experiencing itching, burning sensation. It is very important to identify the primary Pyo-inflammatory hearth, which can be localized on the fingers, foot or lower leg. Extension no superficial veins, unlike acute thrombophlebitis.

**Lymphedema** is a chronic disease due to a violation of lymph drainage in the skin, subcutaneous tissue, fascia. Disease develops slowly throughout lymphostasis distinguish two stages: stage 1, limfjedemy (II) stage fibrjedemy. Lymphedema is characterized by: a gradual thickening of the lower limbs, the character of edema skin is dry, tight folds, no network varikozno expanded veins trophic violations tissues lead to the development of maceracij and jekzematoznyh plots.

**Varicose superficial veins-** disease of the lower limbs, accompanied by the emergence of crimping subcutaneous veins, increases, meshkovidnym expansion, gradual development of trophic disorders of the skin in the form of indurative induration, changes its color. Varicose superficial vein without inflammation drips as slowly progressive disease, with the development of chronic venous insufficiency. Inspection and palpation detects softly elastic, spadajushhiesja in a horizontal position, dilated veins, no infiltration and hyperemia, which is typical for superficial thrombophlebitis-vein ULTRASOUND method taped valve insufficiency perforans and ostialnyh, the absence of thrombosis.

**Phlebothrombosis** is thrombosis of deep veins, manifesting raspirajushhim swelling of limbs, pain. The skin becomes pale cianotichnuju color. "Inch" is determined by the difference of the circumference of the tibia and femur compared with a healthy foot. Missing hyperemia and ripple in the projection surface veins. Verified diagnosis ultrasound duplex scanning.

**Test questions**:

1.What are the causes that lead to the development of acute superficial thrombophlebitis.

2.Specify major clinical differences of acute varikotromboflebita of lower limb Erysipelas.

3.What are the main clinical symptoms of thrombophlebitis and diagnostic methods.

4.spend the differential diagnosis of acute thrombophlebitis with acute Phlebothrombosis.

**Tests for self-control**:

Sharp varikotromboflebit is: 1

1. inflammation and thrombosis of large or small subcutaneous veins

2. Portal vein thrombosis

3. flebit brachial veins

4. Parkes-Weber disease-Rubashova

5. aneurysm of the common femoral vein

In the diagnosis of acute varikotromboflebita the most informative is: 3

1. palpation

2. phlebography

3. UZI veins

4. rheovasography

5. all methods

Sharp varikotromboflebit differentiate with all diseases except: 4

1. lymphostasis

2. Erysipelas

3. Lymphangitis

4. obliterating endarteritis

5. varicosity

For acute varikotromboflebita characterized by all except: 1, 2, 5

1. the existence of the syndrome peremejateisa hromota "»

2. express an entire limb edema

3. availability of esophageal varicose veins

4. pain along the course of the trombirovannoj Vienna

**5.** lower abdominal pain

**ACUTE DEEP PHLEBOTHROMBOSIS**

**VEINS OF LOWER EXTREMITIES**

**Theoretical reference.**

Acute Phlebothrombosis is the removal of blood clots in the veins of the lumen in the absence of inflammation of the vessel wall. In the basis of thrombosis in the vessels rested three factors (Virchow triad); slowing blood flow, vascular wall damage (the endothelium), increased blood coagulability

Thrombosis of the lower limbs deep veins are almost always lead to serious consequences. Significant diameter large veins contributes to the formation of jemboloopasnyh and tromboobrazovanij in them often leads to pulmonary artery thromboembolism. In the long run, patients develops postthrombophlebitic the disease, with varying degrees of development of chronic venous insufficiency resulting in disability of patients.

**Classification** is based on topical localization and extent of thrombosis in deep veins:

1. deep vein thrombosis tibia;

2. thrombosis of superficial femoral vein;

3. common femoral vein thrombosis;

4. segmental thrombosis of iliac vein;

 5. common thrombosis of Ilio-femoral segment;

6. systems of internal iliac vein thrombosis;

 7. thrombosis of inferior vena cava: a) infrarenalnyj segment b) renal segment) hepatic segment.

**The clinical picture** of deep vein thrombosis (DVT) consists of a complex of symptoms, characterized by suddenly encountered a violation of venous outflow, while maintaining the flow of arterial blood.

Edema, cyanosis of the skin of the affected limb, raspirajushhego nature of pain, local increase skin temperature, overflow subcutaneous veins, pain in the course of vascular bundle is characterized by varying degrees of acute thrombosis of any localization. Movement joints are limited slightly. Common symptoms of aseptic phlebitis and periflebita-subfebrilitet, weak, adinamia, leucocytosis differ from large numbers of patients.

Clinical diagnosis in General and topical, in particular, is based on an analysis of symptoms caused by circulatory and largely depends on the localization of lesions-Shin, femoral vein or veins of the pelvis.

When a physical study of patients detected positive symptoms, Moses Homansa, Lovenberga. Notes: increase in extremity; on the skin, already on the first day of the disease, detected network extended subcutaneous veins. Symptoms of deep vein thrombosis depends largely upon the degree of narrowing of vessel by a blood clot. The most striking clinical manifestations are observed in full occlusion of veins.

To establish the topical diagnosis and determining the extent and nature of the defeat of thrombosis, most informative studies a duplex ultrasound is a method angioscanning.

Stage pronounced clinical manifestations of acute venous thrombosis is characterized by: swelling of the raspirajushhego nature, pain and a change in coloration of the skin of the leg. Since the swelling of a limb is the main sign of deep Phlebothrombosis, so increasingly differentiate deep veins defeat from other pathological conditions involving this symptom.

**Differential diagnosis**.

Acute deep vein thrombosis of extremities should be differentiated: the insufficiency of blood circulation, lymphostasis, anaerobic flegmonoj, acute arterial insufficiency syndrome long-term strength.

**Circulatory insufficiency**: swelling of the lower limbs develop severe heart pathology, slowly on both legs, accompanied by palpitations, shortness of breath, increased liver aszitom, oliguria. Swelling of the limbs, loose pastoznyj. Pain syndrome is not expressed, no cyanosis and symptoms Homansa and Moses. Application of heart medications, diuretics in congestive heart failure gives a quick positive effect.

**Limb Lymphedema** develops slowly, starting with the distal. Usually it is preceded by diseases such as Erysipelas, recurrent limfangoit, inguinal lymphadenitis, soft tissue tumors, surgical procedures and injuries within the zone of lymphatic drainage. Skin with lymphostasis pale, cool. Edema resistant, dense, reaches a considerable size. Permeability of veins with lymphostasis is not broken, no pain, no advanced subcutaneous veins.

**Anaerobic Phlegmon** occurs when the penetration of anaerobic microorganisms, due to penetrating wounds of soft tissues. For anaerobic Phlegmon is characterized by a significant and rapidly progressing swelling of tissues, the skin is brownish-yellow, sinjushnoj. Suggest the presence of anaerobic Phlegmon enables rapid onset, severe ripping pain in the limbs. The general condition of the patient deteriorates sharply due to intoxication. Sick excited, anxious, later become apathetic, fall into oblivion. Quickly develops toxic hemolytic anemia. Diagnosis is based on the rapidly progressive deterioration, severe intoxication, the presence of gas in the tissues, quick growing swelling of limbs and ULTRASOUND results and bacteriological research.

**Acute arterial occlusion**. Swelling of the limb when ischemia occurs in later stages. Unlike DVT swelling in ischemia subfascialnyj. Acute ischemia begins with severe pain, accompanied by loss of sensitivity, cold limbs. Subcutaneous veins spavshiesja. Swelling of limb development precedes deep paresis. No pulsation of the arteries of the affected limb. Rapidly developing muscular Contracture and gangrene of limbs.

**Syndrome long crushed tissue** ischemic muscle necrosis represents, with subsequent development of acute renal and liver failure. The appearance of edema preceded by prolonged compression of soft tissues limbs. Excepted limb initially pale, cold. Only Sinjushny fingers stop. Dramatically reduced sensitivity. Pulse on peripheral arteries is not defined. The next day the condition of patients with progressive worsening due to self-poisonings, noted weakness, drowsiness, alternating with excitement, there is vomiting, thirst, pain, jaundice, delirium. The characteristic symptom is oliguria urine red, it defines a Myoglobin. Fabric oedematous limb, tight, tense, active movements are absent, a deep sensitivity.

**Test questions**:

1. List the causes leading to the development of acute deep Phlebothrombosis.

2. What are the main clinical manifestations of acute deep Phlebothrombosis.

3. Instrumental research methods used in the diagnosis of deep Phlebothrombosis.

4. Specify the disease, you must carry out the differential diagnosis of acute deep Phlebothrombosis.

**Tests for self-control**:

In acute deep Phlebothrombosis of lower leg level positive symptoms are: 2, 4, 5

1. Samujelsa

2. Moses

3. Goldflama

4. Homansa

5. Lovenberga

The most informative method of instrumental Diagnostics of acute deep Phlebothrombosis is: 3

1. rheovasography 2. phlebography

3. duplex angioscanning

4. radiography

The differential diagnosis of acute deep Phlebothrombosis limbs should be: 2, 3, 4

1. rozhistym skin inflammation

2. lymphostasis

3. acute violation of blood circulation of limbs

4. long-term tissue crushing syndrome

5. acute sciatica

For ileofemoralnogo Phlebothrombosis characteristic: 1

1. pronounced swelling of entire limb

2. violation of movements in joints of limbs

3. raise the temperature of the skin of the limb

4. cold limbs

For deep Phlebothrombosis characteristic: 3

1. decrease skin sensitivity

2. increase skin sensitivity

3. the persistence of skin sensitivity

**POSTTHROMBOPHLEBITIC DISEASE**

**Theoretical reference.**

The term disease postthrombophlebitic (PTFB) understand the symptom occurs when the obstruction of the venous drainage of the affected limb that occurs after suffering deep vein thrombosis and chronic symptoms venous insufficiency (CVI).

Active in the main blood clots deep veins occurs during 3-4 weeks and replaced by active evolution of thrombus, involving macrophages and fibroblasts. The proliferation of fibroblasts in thicker thrombotic mass is accompanied by formation of canals and crevices that are covered by endothelium. In some cases, may develop calcification and ossification of the venous wall. Complete vein recanalization of trombirovannoj leads to the formation of one channel, the destruction of the valves of the veins and to transform the Vienna rigidnuju besklapannuju tube. This is a vertical deep reflux, which further defines the process of formation of the CVI at both mikrocirkuljatornom and makrogemodinamicheskom level. In most cases, patients record PTFB combination of occlusion and recanalization in different venous segments. «Net» occlusion of veins observed in 15% of patients, complete all the veins have taped recanalization 20% of patients. In the greater part of the observations noted a mixed form of diseases.

**Classification** Currently classify PTFB counted 3 main features:

1. the degree of severity of the manifestations of the CVI

2. localization and the prevalence of posttromboflebiticheskogo lesions of deep vein

3. the nature of the evolution of thrombotic occlusion or masses-recanalization.

Localization: bercovyj, popliteal, Femoral, iliac segments lower vena cava.

On the form of defeat: occlusion, recanalization.

According to the degree of CVI: 0-no clinical manifestations, (I) -symptom "heavy legs"-transient edema; (II) -persistent edema-change of pigmentation, lipodermatoskleroz, eczema; (III) -venous trophic Ulcer (zazhivshaja or open).

**The clinical picture**. Postthrombophlebitic disease clinically characterized by expressed varying degrees of chronic venous insufficiency. In patients with limb swelling appears, raspirajushhego nature of pain in the legs, undue fatigability. In the later stages of the disease, swelling of the feet do not disappear even after a night's rest. Appears on the thighs and feet skin hyperpigmentation, signs of skleroticheskogo changes in subcutaneous tissue, often against this background develop microbial eczema, trophic ulcers, difficult to conservative treatment.

In the diagnosis of special importance PTFB ill indicate transferred Phlebothrombosis. They may serve as episodes of sudden swelling of the lower limbs in the long stay on bedrest after injuries, during pregnancy or after childbirth. Swelling in PTFB precedes the appearance of varicose syndrome and other signs of CVI.

You must pay attention to symptoms such as swelling of the thigh, the presence of varicose veins in the suprapubic region and the anterior abdominal wall, development of trophic disorders in the absence of varicose veins hamstring transformation.

Contribute to the formulation of correct diagnosis clinical functional tests (Delbe samples-Pertesa, Pratt-(I), Pratt-II, Shejnisa). an objective assessment of the State of the venous hemodynamics of limbs is determined when the ultrasonic duplex angioskanirovanii, radiopaque venography, reoflebografii.

**Differential diagnosis.**

Differential diagnosis is based on good knowledge of PTFB clinics competing diseases: Varicose disease, elephantiasis, angiodisplazii vessels, diabetic angiopathy.

**With varicose veins**, in contrast, there are no indications women PTFB experiencing thrombophlebitis, Phlebothrombosis or slow development characteristic of varicose veins, subcutaneous transformation the appearance of venous disorders more than 10-15 years from the onset of the disease. When conducting functional tests in patients with varicosity deep veins, usually passable valves of their wealthy and often detected failure ostialnyh valves, and in later stages of insolvency of perforans veins valves. With varicose veins, trophic ulcers appear when CVI III degree, and localized in the "typical" places on the medial surface of the tibia in the lower third.

In the study of veins in patients with PTFB detected: recanalization deep vein valve insufficiency with deep veins reflux of blood flow, multiple valve insufficiency of perforans veins. Trophic disorders, ulcers are formed in patients with 2-3 later years after PTFB onset, have a different localization on the Shin and foot and difficult to conservative treatment often relapse.

**Lymphedema** (elephantiasis) characterized by dense white swelling in the leg, Hyperkeratosis of the skin, increased hair growth, deformation of the limbs. no medical indication migrated venous thrombosis. Skin taken in the fold. The disease can be the result of suffering a Erysipelas. In the diagnosis and differential diagnosis of posttromboticheskoj disease help functional tests: voldyrnaja (MC-Kljura-Aldrich), jodkrahmalnaja (Minor). When USDG veins taped their permeability, viability at lymphostasis, unlike posttromboticheskoj disease. To confirm the defeat of the lymphatic system of the limbs is done limfoangiografija.

**Congenital angiodisplazija** (Parkes-Weber Syndrome and Klippel-Trenaunay Rubashova) develops in early childhood and gradually progresses.

Parkes-Weber Syndrome-Rubashova evident congenital arterio-venous fistulae, often accompanied by increased limb and its size, the appearance of varicose veins and pigment skin changes. There is no indication of the pigment of the skin changes, experiencing deep thrombosis. When instrumental examination (ULTRASOUND, angiography) establishes the existence of arterio-venous fistula. When angiography determined reset contrast agent of artery in advanced venous network.

Klippel-Trenaunay syndrome-congenital Aplasia or hypoplasia of the lower limbs deep veins is uncommon. Characterized by early childhood development, venous insufficiency, compensatory expansion of superficial venous system of the affected limb. There is no indication of a dropped venous thrombosis. Ultrasound duplex scanning detects segmental narrowing or complete absence of the femoral, iliac vein or the presence of Weno-venous reset through surface system.

In **acute and chronic arterial insufficiency**, unlike posttromboticheskoj disease, characterized by symptoms of ischemia-pallor, numbness, cold extremities, muscle wasting, change of skin appendages (hair loss limb phenomenon Onychomycosis stop). By palpation of the vessel is determined by the reduction or absence of pulsation of the femoral, popliteal arteries and stop. Nutritional disorders are expressed also in the emergence of pockets of dry necrosis on the fingers. When the lower limbs of the EGR passage is decreasing limb segments. Unlike the posttromboticheskoj disease with USDG reveal permeability of trunk veins and viability of their valves, but study of the arteries determines the picture of their segmental or diffuse painful defeat.

When **diabetic angiopathy** of lower limbs in contrast to posttromboticheskoj disease, characterized by swelling and expansion not subcutaneous veins. History-long-existing diabetes. The State of the lower limbs is characterized by their pobledneniem, pain in the foot and toes (nejrotroficheskaja form), on your toes foot and heel weeping deep ulceration to the Plantar Aponeurosis, with the progression of the process and decompensation diabetes degenerating into moist gangrene of the lower limb. Appears on the foot and the lower third tibia swelling during illness due to severe dismetabolicheskimi disorder. Analyses of blood in the urine, Hyperglycemia, glycosuria, ketoacidosis.

**Test questions**:

1. Call classification of posttromboticheskoj diseases.

2. List the clinical symptoms characteristic of the disease posttromboticheskoj.

3. Call it functional and instrumental methods sample the venous study confirming and posttromboticheskoj form of the disease.

4. Hold differential diagnosis posttromboticheskoj with varicosity disease and other diseases with similar clinical picture.

**Tests for self-control**:

For posttromboflebiticheskoj disease is characterized by the following symptoms: 1, 2, 3

1. transferred earlier acute venous thrombosis

2. swelling of a limb, not disappearing after a night's rest

3. varicose veins on pubic hair

4. trophic ulcer located on the plantar surface of the foot

5. expressed pain in the knee or ankle joints when walking

6. hyperthermia to 38-39° c.

Specify the symptoms characteristic of the CVI: 6

1. feeling of heaviness in the legs by the end of the working day

2. numb and cold limbs

3. night cramps

4. sharp pain in the calf muscles that appear when walking

5. swelling of distal limbs at the end of the day

6. all of the listed symptoms

What are the main methods of diagnosing CVI: 7

1. functional tests

2. ultrasound examination

3. rentgenokontrastnaja venography

4. radionuclide venography

5. determination of prothrombin index

6. flebotonometrija

7. all the methods listed

Specify the disease, you must carry out diffdiagnostiku posttromboflebiticheskoj diseases: 3.4

1. obliterating atherosclerosis of the arteries of the lower limbs

2. acute deep Phlebothrombosis of lower extremities

3. varicosity

4. limfjedema of the lower extremity

5. Buerger's disease

6. superficial varikotromboflebit.

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