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Харківський національний медичний університет

Кафедра Внутрішньої медицини №3
Факультет VI по підготовці іноземних студентів

ЗАТВЕРДЖЕНО
на засіданні кафедри внутрішньої медицини №3
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Зав. кафедри _____д.мед.н., професор Л.В. Журавльова

МЕТОДИЧНІ ВКАЗІВКИ
для студентів

з дисципліни «Внутрішня медицина (в тому числі з ендокринологією)
студенти 4 курсу I, II, III медичних факультетів, V та VI факультетів по підготовці іноземних студентів

Тиреотоксикоз. Клінічні форми, діагностика, лікування. Пухлини щитоподібної залоз та патологія при щитоподібних залоз

Харків 2016

Topic – «Thyrotoxicosis. Clinical forms, diagnostic, treatment. Tumors of thyreoid gland and pathology of parathyroid glands»

1.The number of hours - 5

Actuality: Thyroid gland disease is one of the most popular in Ukraine affect patients of working age, degrade the quality of life and reduce its duration.

Aim:

1. To learn the method of determining the etiologic factors and pathogenesis of diffuse toxic goiter. Work out techniques of palpation of the thyroid gland.
2. To familiarize students with the classifications of goiter by OV.Nikolaєv (1955 г.) And WHO (1992 г.).
3. To distinguish a typical clinical picture of diffuse toxic goiter (DTG).
4. To acquainte with the atypical clinical variants of diffuse toxic goiter.
5. To acquaint students with the possible complications of DTG.
6. To determine the basic diagnostic criteria for Graves' disease
7. To make a plan to examinate patients with Graves' disease.
8. Analysis of the results of laboratory and instrumental studies, which are used for the diagnosis of DTG.
9. Differential diagnosis between DTG and goiter
10. Technology of formulation of the diagnosis of DTG and goiter.
11. Making a treatment plan for patients with DTG and goiter.
12. Deontological and psychological characteristics of patients with DTG and goiter.
13. To distinguish a typical clinical picture of thyriod cancer.
14. To distinguish a typical clinical picture of thyriod cancer.
15. Making a treatment plan for patients with thyriod cancer.
16. To distinguish a typical clinical picture of parathyroid glands disease.
17. To distinguish a typical clinical picture of parathyroid glands disease.
18. Making a treatment plan for patients with parathyroid glands disease.

What the student should know?

1. Definition of DTG, goiter, hyperthyroidism.
2. Epidemiology of DTG and goiter in Ukraine.
3. Risk factors for DTG and goiter.
4. The mechanism of hormonal and metabolic disorders of DTG and goiter.
5. Etiology and pathogenesis of DTG and goiter.
6. Classification of degree of thyroid enlargement.
7. The clinical picture of DTG.
8. The typical clinical picture of thyrotoxicosis.
9. Multiple organ complications of thyrotoxicosis.
10. Diagnostic criterias for DTG and goiter.
11. Analysis of results of hormonal studies.
12. Diagnostic value of ultrasound examination of the thyroid gland. Radioisotope examination of thyroid gland (radiometry, scanning).
13. Diagnosis of eye`s lesions. Selection of methods of treatment of eye complications.
14. Selection of treatment for DTG and goiter.
15. Thyriod cancer – classification, symptoms, diagnosis and treatment.
16. Hypo and hyperparathyroidism - classification, symptoms, diagnosis and treatment.

What the student should be able to do?

1. To determine a risk factors for DTG and goiter.
2. To diagnose DTG and goiter.
3. To perform palpation of thyroid gland.
4. To determine the degree of thyroid enlargement.
5. To diagnose the syndrome of thyrotoxicosis.
6. To determine the severity of syndrome of thyrotoxicosis.
7. To detect the presence and nature of ocular complications of DTG.
8. To determine the nature of multiple organ complications of DTG and goiter.
9. To analyze the results of hormonal examination and functional tests.
10. To evaluate the results of ultrasound and radioisotope examination of thyroid gland.
11. To make the differentsional diagnosis between syndrome of thyrotoxicosis and goiter.
12. To estimate the dynamics of thyreoid status of patients during thyreostatic treatment.
13. To be able to correct the dose of thyreostatics and accompanying medicine as patients reach euthyroid state.
14. To Draw up a long-term treatment of DTG and its complications, the technology to attract patients to complicity in the treatment process.
15. To interacte with neighboring specialists (surgeon, ophthalmologist, cardiologist) at the stage of establishing a complete diagnosis, method of treatment and long-term observation of the patient with DTG or goiter.
16. To determine a risk factors for thyroid cancer.
17. To diagnose thyroid cancer.
18. To analyze the results of hormonal examination and functional tests in thyroid cancer.
19. To diagnose the diseases of thyroid glands.
20. To analyze the results of hormonal examination and functional tests in thyroid cancer.
21. To diagnose and analize the results of tests in hypo and hyperparathyroidism.

Diseases that are accompanied by thyrotoxicosis.

Thyrotoxicosis (hyperthyroidism) – is a syndrome, whose presence is associated with an increased content of thyroid hormones in the blood, which occur with various diseases or excessive exogenous entry of thyroid hormones.

Thyrotoxicosis is observed during diffuse toxic goiter, multinodular toxic goiter, thyrotoxic adenoma, subacute thyroiditis (first 1-2 weeks), postpartum (silent) thyroiditis, autoimmune thyroiditis, thyroiditis that developed after exposure of ionizing radiation, tireotropinome, syndrome of not adjustable TSH secretion, follicular thyroid cancer and its metastases, ectopic goiter (ovarian tissue), excessive intake of iodine (iodine-hyperthyroidism), trophoblastic tumors, which secrete human chorionic gonadotropin, iatrogenic and "artificial or conditional" thyrotoxicosis.

The etiology, pathogenesis, clinical manifestations of diffuse toxic goiter.

Diffuse toxic goiter - thyroid disease, which manifests with diffuse thyroid enlargement and thyrotoxicosis. The disease has many names - the names of authors who had described it - Basedow's disease (in Germany), Grave's disease (in Britain), and Flajani`s disease (in Italy), "hyperthyroidism with diffuse thyroid enlargement", "" autoimmune thyrotoxicosis".

The disease is more common in women than in men (correlation is 5:1, 7:1), and affects mainly those aged 30-50 years.

Diffuse toxic goiter - autoimmune disease that develops in patients with heredity. Debated the nature of inheritance – is it autosomal recessive or autosomal dominant. The most probable is multifactorial (polygenic) inheritance type.

Established that during diffuse toxic goiter significantly reduced suppressor activity of mononuclear cells in peripheral blood, this immune defect persists even after these patients achieved euthyroid state as a result of thyreostatics treatment. Reduced activity of T suppressor - is congenital specific disturbance in individuals predisposed to the development of diffuse toxic goiter.

Clinical manifestations

In most cases, the development of diffuse toxic goiter occurs slowly, symptoms increase gradually. The disease progresses, however, there are some cases of acute disease development.

1. The defeat of the thyroid gland

Goiter – is enlarged thyroid gland of various degrees - is a characteristic feature of diffuse toxic goiter.

The thyroid gland is uniformly, diffusely enlarged, palpated on the anterior and lateral surfaces of the neck. In most cases the gland is increased to II-III degree. The degree of increased thyroid gland often does not correspond to the severity of the disease. The size of goitre can vary: it increases after excitement, after initiation of treatment it is gradually reduced.

As a rule, men with severe clinical form of diffuse toxic goiter had the thyroid gland increased slightly, palpated with difficulty, since gland is increased mainly by lateral lobes, which lies close to the trachea.

Gland is not palpable when it is located in atypical position - the ring-like and retrosternal goiter (gland is located in a ring around the trachea).

Symptom of Valsalva is defined in the case of ring- like or retrosternal goiter, the upper pole is slightly above the handle of the sternum only during coughing – it is a semi-submersible or "diving" goiter.

The thyroid gland is often dense, had elastic consistency. The presence of one or more nodes in the tissue of diffusely enlarged gland talk about mixed toxic goiter.

The thyroid gland is movable during swallowing, not adherent to surrounding tissues.

Cervical lymph nodes and tonsils may increase during diffuse toxic goiter.

Blood supply of thyroid gland increased, blowing systolic murmur auscultate by phonendoscope when it pressed to thyroid gland, palpation accompanied by a feeling of tremor.

Symptom of Gutmann - auscultated subtle hum under the thyroid gland.

There are several classifications of the degrees of thyroid enlargement. In clinical practice, until now used the classification proposed by O.V. Nikolaev in 1955

0 degree - the thyroid gland is not palpable;

I degree - by palpation determined the increase of isthmus of thyroid gland;

II degree - by palpation determined the increase of the lateral lobes of thyroid gland;

III degree - visually determined the increase of thyroid gland ("bull neck");

IV degree - a significant increase of thyroid gland (goiter clearly visible);

V degree - huge goiter.

I and II degrees attributed to the increase of thyroid gland, and the III-V degrees of increased thyroid gland is actually goiter.

Also WHO classification is used (1992)

0 degree - goiter is not visible and not palpable;

I degree - the formation is palpated on the neck corresponding the enlargement of the thyroid gland, is shifted during swallowing, but not visible in normal neck position, in the thyroid gland one or more nodes can be palpated;

II degree – thyroid gland is palpable and clearly visible in the normal position of the head.

2. The defeat of the cardiovascular system

Dominate place in the clinical picture of thyrotoxicosis occupy cardiovascular disorders.

Cardiovascular disorders in diffuse toxic goiter determine, on the one hand, the pathological sensitivity of the cardiovascular system to catecholamines, on the other - the direct impact of the excess of thyroxine on the myocardium. Notes the sum of the excess secretion of thyroid hormones and the effect of increased sympathetic activity on heart and peripheral circulation. The disorder of hemodynamics, the disparity between the level of delivery, consumption and utilization of oxygen to the heart muscle leads to severe metabolic-dystrophic damage and the development of thyrotoxic cardiomyopathy.

A. Stage of functional disorders.

At the stage of functional disorders symptoms caused by hyperkateholemia, hypersensitivity of receptors, hypersympathicotonia. The hyperkinetic type of hemodynamics with increasing of the cardiac performance, increasing blood flow is formed.

Characterized by palpitations growing during exercise.

Tachycardia is a constant, does not change with a change of body position from vertical to horizontal, and does not disappear during sleep. Frequency of heart rate ranges from 90 to 150 beats per minute, extrasystole is possible.

Specific for thyrotoxicosis is the opposite changes in systolic blood pressure (increase) and diastolic blood pressure (reduction). Systolic blood pressure is high caused by the hyperkinetic type of circulation with increased heart stroke volume and cardiac output.

Diastolic pressure decreases because of reduction in the total peripheral vascular resistance caused by the expansion of peripheral arterioles. Paresis of peripheral vessels is an adaptation factor – the increase of blood supply of skin vessels contributes to heat loss, prevents "overheating" of the internal organs (provides anti-aging dermatological effect of short thyrotoxicosis). The skin is warm to the touch, moist due to compensatory expansion of peripheral vessels.

The difference between systolic and diastolic blood pressure, pulse pressure increased sharply, and causes a subjective sensation of pulsation of the neck vessels, feeling of the pulse in the neck, head and abdomen.

Pulse accelerated, increased content and stress.

There is nagging pain in the heart (cardialgia) or angina - usually in patients with concomitant coronary artery disease.

Heart sounds are loud, auscultated a functional systolic murmur with a peak at Botkin`s point, at the basis of the heart, blood vessels in the neck. The origin of the noise associated with the acceleration of blood flow, increase of pulmonary artery, swelling of the papillary muscles. In the early stages of development of toxic goiter borders of the heart are not changed.

On the electrocardiogram - tachycardia, high voltage of P and T waves.

The dyspnea, palpitations, intermissions are caused by the addition of circulatory deficiency.

Severe cardiac arrhythmias are characteristic - extrasystole, tachysistole form of ciliary arrhythmia, paroxysmal or permanent form.

The ciliary arrhythmia has a paroxysmal character at the beginning, and then goes into the persistent form. The ciliary arrhythmia may be associated with extrasystole. Paroxysms of the ciliary arrhythmia sometimes can be the only manifestation of diffuse toxic goiter. Abnormal heart rate often occurs in patients older than 40 years.

Permanent form of the ciliary arrhythmia leads to the progression of circulatory failure by right ventricular type with an increase of the liver, peripheral edema, extravasation into the pleural cavity, ascites.

Circulatory failure and arrhythmias during thyrotoxic cardiomyopathy are resistant to cardiac glycosides.

In the elderly age thyrotoxicosis may manifest itself by attack of the ciliary arrhythmia, which represents a difficulty for the diagnosis of the disease. In the interictal period these patients had general condition that remains satisfactory, and heart rate may be within normal limits.

The expansion of the heart is caused by the overload of myocardium (the symptom of Grokko).

The ECG: sinus tachycardia, sinus arrhythmia, atrial, atrial flutter, atrial or ventricular premature beats, ciliary arrhythmia, acceleration or deceleration of atrioventricular conduction.

Signs of hypoxia and focal ischemia of the left ventricle: the ultimate violation of the ventricular complex (depression of S- T segment, negative or biphasic T wave). Signs of "fatigue" myocardium - the amplitude and duration of P and T waves are reduced.

3. Psycho-emotional disorders

Disorders of the nervous-psychic activity - the most important manifestation of diffuse toxic goiter.

Diffuse toxic goiter is characterized by irritability, excitability, and lability of mood. These patients are excessively active, restless, fidgety, conflict, mistrust, intolerant, unable to concentrate. Working ability and memory are reduced. During the interview and examination of the patient we can note that patients had euphoria and at the same time they are crying, wordiness, fussiness. Patients are constantly shifting from one thought to another, active, carry out a lot of unreasonable movements.

Severe forms of thyrotoxicosis may be complicated by psychosis, depression, psychotic reactions.

Sleep disturbances are characteristic – difficulty to fall asleep, restless sleep.

The heat generation is increased because of the activation of metabolism under the influence of thyroid hormones. Disorders of thermoregulation are manifested with almost constant feeling of heat, intolerance of high ambient temperature, thermal procedures. During severe forms of thyrotoxicosis constant low-grade fever is observed.

Symptom of sheets – patients at night sleep under a sheet.

Symptom of Lukatello - the temperature in the armpits is above the temperature in the mouth 0,2-0,3 C.

4. Thyrotoxic encephalopathy

The lesion of central nervous system can develop by the type of thyrotoxic encephalopathy. In this case, patients had such complains as headache, dizziness, photophobia, disturbance of sleep.

Sometimes autoimmune encephalitis is revealed. The damage of brain in the region of stem and hypothalamus is possible: leukocyte infiltration, degeneration of cells in the brain stem, in the nuclei of the hypothalamus, medulla oblongata.

The patients also had clinical signs of pyramidal tract lesions: hyperreflexia, anizorefleksiya, the absence of cutaneous reflexes, the reflexes of oral automatism, central paresis of mimic muscles, disorders of coordination.

The central paresis of facial muscles, pyramidal signs (hyperreflexia, anizorefleksiya), sometimes paresis and muscle atrophy are determined.

Symptom of Dalmedi - "frozen face", "angry look".

Symptom of Geoffroy – delay of formation of folds on the forehead when looking up. Vegetovascular symptom is caused by vasomotor lability.

Symptom of Maranon - enhanced vasomotor response during stimulation of neck skin, tachysystole, sense of heat, associated with increased of tone sympathoadrenal system.

Typical is fine tremor of hands and eyelids. Tremor is continuous, not reduced by diverting attention, during rest or sleep, the level of tremor corresponds the severity of the disease.

Symptom of a telegraph pole - shaking of head, lips, tongue, feet, and whole body.

Symptom of Marie (Charcot) – trembling of fingers on outstretched hands.

Evident tremor leads to disruption of the handwriting, inability to perform precise movements - patients struggling to button his buttons, changing handwriting.

Symptom of "saucer" - if the patient is holding an empty cup and saucer, a tinkle sound is published by a fine tremor of the hands.

Deep tendon reflexes were increased.

Hyperkinesia and fussiness are typical.

5. Thyrotoxic myopathy

One of the early and persistent symptoms of the disease is muscular weakness. It varies from a sense of express fatigability to severe muscle weakness and atrophy of muscles of the proximal extremities. The most expressed is defeat of muscles of the pelvic girdle and thighs, rare the shoulder girdle and hands.

Patients can not stand up without outside assistance, walk up the stairs.

6. Thyrotoxic myelopathy

Thyrotoxic myelopathy – is the involvement of the anterior horns of the spinal cord, manifested by muscle atrophy and paresis of the proximal extremities, mainly the pelvic girdle. Thyrotoxic myelopathy combined with thyrotoxic myopathy in severe cases.

7. Dysfunction of the gastrointestinal tract

The most frequent symptom of diffuse toxic goiter - a progressive weight loss with increased appetite. In the elderly age appetite may be reduced.

"Fat Bazedov" is possible - growth of weight during thyrotoxicosis, especially in men.

Evident thyrotoxicosis is accompanied by an increase of intestinal peristalsis –The frequentcy of stool is up to 3-4 times a day, with a formed or semi-liquid feces. During severe thyrotoxicosis patients can have frequent diarrhea, exhausting the patient. Sometimes spastic constipation is developed.

8. Changes in the skin

Skin becomes soft, thin, and velvety, with a less amount of wrinkles; these patients look younger. A positive cosmetic effect of thyrotoxicosis caused by increased blood supply of the dermis, but this effect is very short, after several months skin aging processes are accelerated.

General sweating is typical, skin is moist and warm.

Hyperpigmentation of eyelids, pigmentation in areas of friction of clothing, itchy skin is possible.

Symptom of Vigouroux - reduced electrical skin resistance.

Sometimes observed pigmentary form of diffuse toxic goiter: hyperpigmentation of skin folds, especially in areas of friction (neck, back, elbows, etc.) hyperpigmentation of the whole body can develop, that look like melasma during Addison's disease. The reason of hyper pigmentation is the excess of intermedin, in severe cases the failure of the adrenal cortex can join.

Often patients notice itching, traces of scratching appear on skin. The urticarial may develop.

The vitiligo may develop in some patients - an autoimmune skin lesion with the appearance of areas of depigmentation.

Hair is dry, thin, brittle, easily fall out, early turn grey. Alopecia is possible – the local loss of a hair on head.

Nails become brittle, thin and transparent.

Local myxedema - an independent autoimmune disease that occurs during diffuse toxic goiter in 4% of patients, almost always combined with ophthalmopathy. Sometimes an autoimmune dermopathy, as well as autoimmune ophthalmopathy occurs through 4-20 month after treatment of diffuse toxic goiter with radioactive iodine. The term "local myxedema" does not correspond to modern views of pathogenesis and development of this pathology; a more appropriate term is "autoimmune dermopathy."

The skin becomes edematous, thickened, with prominent hair follicles, purplish-red or brown color, it looks like the skin of an orange, often accompanied by considerable erythema, burning and itching. Localization of autoimmune dermopathy: the front surface of the tibia, dorsal surface of the fingers and wrists. Histologically in the peripheral layer of the skin revealed edema, increased infiltration of mucopolysaccharides and mucin, "splitting" of collagen bundles into individual collagen fibers.

9. Defeat of bones

During severe and prolonged thyrotoxicosis observed widespread, moderate osteoporosis caused by the catabolic action of excess of thyroid hormones and the destruction of the protein matrix, that holds the calcium in the cancellous tissue, disorders of calcium-phosphorus balance, resorption of bone tissue. Pain in the bones and in the back have "osteoporotic" origin.

The acceleration of growth is observed among children.

Thyroid acropathy is possible - thickening of the phalanges of fingers caused by swelling and periosteal neoplasms of bone tissue. The swelling of soft tissues and underlying bone in the hands and wrists is possible. Radiologicaly: subperiosteal bone formation (soap bubbles). Scanning with 99mTc-pyrophosphate of the affected area: involved in the pathological process of soft (area of local myxedema) and bone tissues selectively absorb these isotopes.

Thyrotoxic and endocrine ophthalmopathy.

A. Ocular symptoms of thyrotoxicosis

Most of patients have protrusion of the eyeballs - exophthalmus.

Thyrotoxic exophthalmus is a functional disorder caused by hyperkateholemia and disturbance of the vegetative innervation of the eye. The only one manifestation is the symptoms associated with the retraction of the upper eyelid.

Thyrotoxic exophthalmus usually develops with the manifestation of the disease, usually develops gradually, but sometimes within a few days or even hours. In some cases it is the first symptom of thyrotoxicosis.

As a rule, exophthalmos is bilateral, symmetric, and rarely unilateral.

There is no disturbance of eye function; there is no double vision.

Between the severity of diffuse toxic goiter and exophthalmus degree there is no parallelism. Thyrotoxic exophthalmus completely disappears after compensation of thyrotoxicosis.

Ocular symptoms observed during thyrotoxic exophthalmus

Dalrymple - wide-open eye slits, which gives an amused, frightened look.

Jellinek (Teller) - pigmentation of skin of eyelids.

Shtelvaga - a rare and incomplete blinking.

Krause - reinforced shine of eyes.

Singer - swelling and bag- like eyelids.

Brahma - during laughing the palpebral fissure is not narrowing, the eyes are wide open.

Rozenbaha - fine and quick tremor of droped eyelids.

Beysmana - quiet hum, listened over closed eyes of the patient.

Gifford - the difficulty of reversing eyelids caused by myogenic retraction.

Knisa - anisocoria.

Levy - pupil dilation under the influence of adrenaline.

Becker - reinforced pulsation of retinal arteries.

Symptoms related to abnormality of oculomotor responses:

Wilder - a twitch of the eyeball during alternate reduction and abduction.

Mobius - during a fixed view on a close subject eyes can not be in a position of convergence for long period of time, and then one of them soon moves outwards.

Cowan – during checking friendly reaction of pupils observed pupillar vibration.

Development of the following symptoms associated with an increased tone of smooth muscle fibers, which are involved in the lifting of the upper eyelid, which are innervated by the sympathetic nervous system.

During fixation in front of patient`s eyes there are a moving object, that moves downward observed such symptoms:

Graefe - upper eyelid first lag behind a bit and then catches up with moving iris of an eyeball - this involves a white strip of the sclera between the upper eyelid and the iris.

Popov - the upper eyelid of the patient pull down unevenly.

Sentona - the upper eyelid of the patient lifted because of spastic contraction of forehead muscles.

During fixation in front of patient`s eyes there are a moving object, that moves upward observed such symptoms:

Kocher - upper eyelid moves up faster than the eyeball, with the formation of white strips of sclera between the upper eyelid and the iris.

Eye symptoms described above are the part of the syndrome of thyrotoxicosis should be distinguished from autoimmune ophthalmopathy.

B. Autoimmune ophthalmopathy

Autoimmune ophthalmopathy is an independent autoimmune disease. The immunocomplex of lesion tissues of the orbit is accompanied by infiltration, edema and proliferation of postbulbar muscle fibers and connective tissue. Synonyms of autoimmune ophthalmopathy, previously used in clinical practice: edematous exophthalmus, malignant exophthalmus, exophthalmus neurodystrophic, orbitopathy, endocrine exophthalmus.

Autoimmune ophthalmopathy can occur as a separate, independent from thyrotoxicosis disease or combined with diffuse toxic goiter or local myxedema.

Characteristic symptoms of autoimmune ophthalmopathy: exophthalmus, conjunctivitis and keratitis.

Exophthalmus during autoimmune ophthalmopathy is asymmetric, may be unilateral, combined with edema and infiltration of the eyelids.

Constant symptom is double vision (diplopia) caused by changes in the oculomotor muscles.

Normally, the protrusion of the eyeball is 16-19 mm. There are three degrees of ophthalmopathy during which protrusion of the eyeball increases till 3-4 mm, 5-7 mm and above 8 mm.

Conjunctivitis that is complicating ophthalmopathy, manifested by lacrimation, photophobia, burning of eyes, feeling of foreign body, "sand" in the eyes, feeling of pressure on eyes, pain in eyeballs. It is determined by hyperemia, edema of the conjunctiva, eyelid edema, injection of scleral vessels, restricted movements of the eyeball, reduced corneal reflexes. Neoplasm of vessels is a bad prognostic sign.

Keratitis develops during a significant ophthalmopathy (III degree) - eyeballs protrude from the orbit, eyelids and conjunctiva are swollen, inflamed; because of permanent drying of the cornea, inflammation and ulceration are formed, that can lead to corneal leukoma and the reduction of vision up to complete blindness.

Ocular symptoms of autoimmune ophthalmopathy

Mann - the eyes seem to be at different levels.

Endrashika - the paresis of the periorbital muscles.

Swecker - lateral fixation of the eyes is impossible.

Abad i – spasm of erector of upper eyelid.

Balle - the complete disappearance of voluntary movements of the eyeballs with preservation of the pupil reaction and automatic eye movements.

Complications of an autoimmune ophthalmopathy:

Worsening of color perception, especially the red;

Constriction and defects of visual field;

Mechanical compression of optic nerve by edematic, increased in volume retrobulbar fiber;

Neuropathy of optic nerve with decreased visual acuity;

Swelling of the optic nerve papilla;

Occlusion of central retinal vein,

Lagophthalmos - incomplete closure of eyelids, accompanied by erosions, ulcers of the cornea.

Age-related features of the flow of toxic goiter among children and people of elderly age.

Atypical variants of thyrotoxicosis

1. Triiodothyronine thyrotoxicosis.

T4 is normal, T3 increased. Clinical manifestations are classic. There are 5% of patients with diffuse toxic goiter and 50% of patients with toxic adenoma.

2. "Apathetic thyrotoxicosis."

Usually is met among people of the elderly age: inhibition of the nervous system with apathy, depression, weight loss, muscular weakness. Exophthalmus develops very rarely. Often there is no enlargement of the thyroid gland. - subclinical thyrotoxicosis.

Signs of myocardial damage are dominated such, as severe arrhythmias, ciliary arrhythmia, rapidly developing heart failure, refractory to medical therapy. The level of thyroid hormones may be normal - expressed hypersensitivity of tissues to thyroid hormone.

3. Juvenile thyrotoxicosis.

Diffuse toxic goiter among children and adolescents characterized by the absence of classical signs of the disease and ophthalmopathy. The thyroid gland does not reach the size observed during diffuse toxic goiter among adults.

Diffuse toxic goiter among children of preschool age are found rarely, the frequency of DTG increases in adolescence, among girls toxic goiter occurs 5-7 times more often than among boys. Such patients complain of increased fatigability and general weakness, decreased ability to concentrate. Teenagers begin study badly in school, skip lessons, there are changes in behavior. The girls later than usual had menarche and the menstrual cycle is established.

Substantiation of diagnosis of thyrotoxicosis.

Investigation of hormonal background

Preferred methods of direct determination of level of thyroid hormones in the blood, because they are more accurate, they can be used for diagnosis in patients who took thyreostatics and iodine preparations. Radioimmunological methods are used.

Typically is increase basal level of thyroid hormones: T3 (N 1,2-2,7 nmol / L), T4 (N 70-120 nmol / L). The total content of thyroxine in blood serum increased from 130-140 nmol / L till 400 or more. Increased content of free thyroxine in blood serum - more than 70-120 nmol / L, and the percentage of free thyroxine - more than 0,05%. The absolute content of the triiodothyronine is more than 2.7 nmol / L, the percentage of free triiodothyronine - 4,5%, the coefficient of triiodothyronine binding - 0,05 and over. The last three indicators can be used to assess the severity of diffuse toxic goiter and to control the dynamics of the disease. T3 is more active and increased to a greater extent than his predecessor, T4. Sometimes the level of T4 is normal.

Decrease of TSH is characteristic - compensatory reduction of thyroid-stimulating activity of the hypothalamic-pituitary system to increase the level of thyroid hormones. Basal level of TSH is increased only in the case of TSH-producing adenoma of pituitary and as a result secondary thyrotoxicosis.

Test with TRH (rifatiroini) is carried out in cases where the basal values of T3 and T4 are normal, but there are symptoms of thyrotoxicosis. During diffuse toxic goiter test is negative - the introduction of TRH do not increase TSH level: there is a profound inhibition of TSH-producing function of pituitary, there is no reaction of the pituitary gland to hypothalamic stimuls.

Ther are signs of adrenal dysfunction. At the beginning of the disease - hyperfunction of adrenal cortex: the level of free and total cortisol in blood serum is increased, excretion of 17-OCS with urine is increased. During prolonged and severe course of thyrotoxicosis functional reserves of adrenal are depleted, a relative adrenal insufficiency is formed.

Drug treatment, surgical treatment of toxic goiter, the use of 131-iodine for therapeutic purposes.

Indications for surgical treatment:

1) The inefficiency of drug therapy for 4-6 months from the beginning of treatment – the exacerbation of the disease at maintaining dose of thyreostatics
2) Rapid enlargement of the thyroid gland till 4-5 degrees;
3) High density of the thyroid gland with symptoms of compression of the neck;
4) The severity of thyrotoxicosis;
5) Abolish of mercasolil caused by allergies, granulocytopenia;
6) Recurrent toxic goiter;
7) Complication of thyrotoxicosis by encephalopathy or ophthalmopathy;
8) The development of complications of diffuse toxic goiter during associated diabetes mellitus;
9) The nodal, mixed, atypical, and aberrant forms of goiter;
10) Pregnancy and lactation;
11) Severe thyrotoxicosis complicated by ciliary arrhythmia.

Complications of surgery treatment:

1) Postoperative thyrotoxic crises,
2) The injury or paresis of recurrent nerve;
3) The hypoparathyroidism and tetany;
4) Early hypothyroidism (up to 6 months);
5) Late hypothyroidism (more than 6 months after surgery);
6) Recurrence of the disease;
7) Postthyrotoxic encephalopathy

Treatment with radioactive iodine

Therapeutic use of radioactive iodine. Treatment method is based on the property of thyroid gland to absorb radioactive iodine selectively. Beta-rays formed by the decay of radioactive iodine, destroy the cellular elements of the thyroid parenchyma, but they do not destroy surrounding tissue because of a short-range (2 mm) beams. Iodine-131 is used for the treatment. Patients must be treated at least in age 40 years.

Indications for therapy with radioactive iodine:

1) Patients with severe heart failure surgical treatment of which is risky;

2) A combination of diffuse toxic goiter with tuberculosis, severe hypertension, cardiac infarction, neuropsychiatric disorders, hemorrhagic syndrome;

3) Relapse of thyrotoxicosis after subtotal thyroidectomy;

4) Patient's categorical refusal from the surgery treatment of the gland.

Contraindication to the treatment with radioactive iodine: pregnancy, lactation, young age, a major degree of thyroid enlargement or retrosternal location of goiter, blood diseases, kidney diseases, ulcerous disease, nodular forms of goiter.

Complications of treatment of goiter.

Side effects of thyreostatics:

1) Itching, hives, dermatitis;

2) Toxic-allergic granulocytopenia, agranulocytosis, leukopenia, pharyngitis;

3) Arthralgia, myalgia;

4) Nausea, vomiting, diarrhea, fever;

5) Medical hypothyroidism;

6) Strumagennic effect – caused by excessive blockade of the synthesis of thyroid hormone, the synthesis of TSH is increases, that cause increase of hyperplasia, hypertrophy of the thyroid gland.

To prevent the strumagennic effect merkazolil combined with L-thyroxine 50-100 mg / day or triiodothyronine 10-20 mg / day. At the same time the dose of mercasolil is increases slightly.

With a decrease of white blood cells less than 4x10 9 / L, segmented less than 40% of merkazolil is canceled for a few days, leucogen, leykopoetin, and nukleinat sodium are prescribed. Lithium carbonate is used as thyreostatics three times a day 0.3 g. If after 7 days the level of white blood cell is returned to normal, mercasolil is used again, reducing the dose. When you save changes - carbonate, Lugol's solution, prednisolone 15 mg / day is prescribe lithium. Control of clinical blood must be carried out every 7-10 days among all patients receiving merkazolil.

«Cancer of thyroid gland and the diseases of parathyroid glands».

Pathological classification of tumours of a thyroid gland. A substantiation of the diagnosis of a cancer of a thyroid gland.

To malignant tumours of a thyroid gland belong a cancer. Frequency of a cancer of a thyroid gland amounts 0, 4-1 %. Women ill in 5-7 times more often than men. The greatest number of patients is fitted on age of 40-50 years.

It is not found out etiology of cancer of a thyroid gland. It is noticed, that the cancer develops on a background to a struma in 80-90 % of cases and in 10 times more often on a struma in endemic areas. The central and mixed struma in connection with its frequent regeneration is considered as premalignant condition. Chronic inflammatory processes in gland, tonsils, x-ray irradiation, residing at areas, adverse on radiological conditions, also can provoke development of a cancer of a thyroid gland. It is impossible to exclude thyrotropine influence on progressing growth of a tumour due to stimulation of hyperplastic processes in a gland.

Classification of a cancer of a thyroid gland. There are following clinical forms of malignant new growths of a thyroid gland:

1) a primary malignant tumour;

2) a malignant tumour which has developed on a background before existing disease of a thyroid gland;

3) metastasing adenoma;

4) malignant tumours of additional thyroid glands.

The international classification of a cancer of a thyroid gland (1966) considers prevalence of a tumour (T), involved regional lymph nodes (N) and presence of the remote metastasises (M).

T-0 - the tumour is not palpated;

T-1 - the tumour is in tissue of gland, deformation or displacement of organ not be proved;

T-2 - numerous tumours or one big, that serves as the reason of deformation of gland;

T-3 - the tumour falls outside the limits of gland, its personal property rises or occurs infiltration of surrounding structures.

N-0 - cervical lymph nodes are not palpated;

N-1 - the increased mobile lymph nodes on the involved side;

N-2 - the increased mobile lymph nodes bilateral or on the opposite side;

M-0 - the remote metastasis are not present;

M-1 - the remote metastasis.

In a clinical course of a cancer of a thyroid gland allocates four stages:

I - the tumour is localized in one lobe of a thyroid gland, does not change the form of organ, does not give metastasises;

II - the configuration of gland changes, there are metastasis on the sick side;

III - the tumour sprouts through a capsule of gland in surrounding tissues, there is a squeezing the next organs. Mobility of gland is limited, bilateral metastasises in the regional lymph nodes are determined;

IV - the tumour sprouts in the next organs and tissues, immovable, there are close and remote metastasises.

Clinic of a cancer of a thyroid gland.

Clinic of a cancer of a thyroid gland is scanty in an initial stage. Only local condensation of a thyroid gland, more often in one lobe is determined.

If the cancer develops on a background of a struma, a fast growth of tumour is determined, except the "struma" anamnesis, we can determined his condensation, restriction of mobility, germination in the next organs - a trachea, a throat, a gullet, muscles, a neurovascular bunch of a neck. It leads to change of a voice, dysphagia, venous haemostasia.

Clinical diagnostics of a cancer of a thyroid gland разновески. Vigilance should be in any condensation of a thyroid gland, presence of node, his growth, restriction of mobility, increase regional lymph nodes. Thus, taking into account residing at endemic area on a struma or radiologically polluted.

Substantiation of the diagnosis of a cancer of a thyroid gland.

In some cases changes of peripheral blood such as leukocytosis, increase to ESR are observed.

Radioisotope scanning of a thyroid gland by radioactive iodine enables to show asymmetry scanned pictures or "cold" units with the low accumulation of an isotope. The same method detects the remote metastasises of a cancer which absorb I^{131}. At scanning from Se 75-methionine the opportunity of revealing of a cancer as "cold" zones of a tumour intensively absorb an isotope considerably much increases.

Unequal contours, the contrast substance collects under a gland capsule are defined qt thyrolymphography. Radiologically there are metastasises in a lung, bones, displacement of a trachea or a gullet at late stages.

The size of a thyroid gland, his connection with surrounding tissue, presence of calcifications are defined at pneumothyroidography.

Tomography specifies localization of metastasises, an ectopic location of a tumour. The computer tomography allows to define the form, the sizes, the contours, structure of node, presence and prevalence of metastasises, and a degree of attraction in pathological process of vessels of a neck and the next tissue.

It appears chaotic neovascularisation at angiography.

By means of one-dimensional and bidimensional echography (ultrasonic scanning) it is possible to receive the information on character and prevalence of pathological process. At a local arrangement of a tumour or her metastasis which can not differ from dense nodes or an adenoma (benign tumour). Involving in process of adjacent tissues in them there are cells of condensation and braces. Echography in a combination with radionuclide scanning allows to establish in most cases the sizes and structure of a tumour which has great value at a choice of a method and volume of operative intervention.

Treatment of a cancer of a thyroid gland

Treatments do into two stages: radical operation, then radiotherapy (X-rays therapy, gamma-ray telethepary, iodine or cobalt radiotheraphy). We shall admit only to operation at initial stages with absence of metastasises in regional lymph nodes. Enucleation of tumours at early stages do with a resection of a gland lobe. A tumour with germination of surrounding tissues delete within the limits of healthy tissues. In cases metastasis tumours in regional lymph nodes carry out total thyroidectomy with removal of regional lymph nodes.

During the postoperative period appoint X-ray therapy in a total doze 6000-8000 R. Recently prefer gamma-rays teletherapy with three-four fields in a total doze 3000-5200 rad (10000 - 12 000 R).

Methods of beam therapy are the base at an inoperable cancer of a thyroid gland. Radioactive iodine prescribes at hormone-active tumours and its metastasises. A single doze is 165-370 MBq, total is 1850-7400 MBq.

Separate forms of a cancer of a thyroid gland

More often there are differentiated forms of a cancer of a thyroid gland, to which concern papillary (62 %) and follicular (18 %) cancer. Not differentiated forms are observed less often.

Papillary cancer of a thyroid gland has the most benign clinical course, affects persons of all age classes, but more often in the age of 30-50 years. The disease at women meets in 2,5 times more often, than at men.

Clinic. It appears usually in the form of single dense node in a thyroid gland, plural nodes are seldom observed. Metastasing in cervical lymph nodes, there are metastasises in the friend to a lobe of a thyroid gland less often, the extremely rare in bones and in a lung. Growth of a tumour is very slow, in surrounding tissues sprouts late, at presence of metastasises in lymph nodes of neck, the last ones are also long remain mobile. It is usually kept an euthyroid status.

Diagnostics is based on revealing of single node in a tissue of a thyroid gland which does not absorb some radioactive iodine, and increased lymph nodes of a neck on the sick side. The final diagnosis concerns on the basis of histologic research.

Treatment. Treatment is surgical intervention with the further irradiation of a thyroid gland and regional lymph nodes. Reception of thyroidine 0, 025-0,05 g to a doze which causes thyrotoxicosis in the light form, assists braking thyrotropic function of a hypophysis.

Life expectancy at absence of metastasises or with metastasises in neck lymph nodes is from 5 till 15 years or more. At presence of the remote metastasises the prognosis worsens.

The follicular cancer meets more often at persons in the age of 40-60 years.

Clinic. Nodes of dense, rounded forms metastasizes at a bone, lungs, less often - in brain. It is quite often distinguished only at revealing metastasis at a bone. The tumoral tissue is more active, than at papillary cancer, radioactive iodine collects, but in much smaller measure, than in not replaced tissue of a thyroid gland.

Diagnostics is based on presence of node and metastasises in a thyroid gland which poorly absorbs an isotope. The final diagnosis finds on the basis of histologic research.

Treatment. Total thyroidectomy. At presence of metastasises which grasp radioactive iodine, in 6 months after operation beam therapy is appointed, in some months is given repeatedly I^{131} to full oppression of involve by its metastasises.

Thyroidine is prescribeed after treatment by radioactive iodine(from 0, 025-0,05 г per day to a doze which causes light manifastations thyreotoxicosis). It assists oppression to thyrotropic function of a hypophysis which stimulates growth of metastasises. Life expectancy is smaller, than at papillary cancer.

Giant-cell cancer concerns to not differentiated tumours of a thyroid gland, it is characterized by fast growth. The tumour functionally inactive, does not grasp radioactive iodine. It meets mainly at persons of advanced age. Treatment operative.

Solid small-celled and large cell cancer differs a malignant course, fast growth with lesion of surrounding tissues, early occurrence of the remote metastasises and dissemination in lymph nodes of a neck. It meets more often in advanced age. Treatment is operative with the further beam therapy. Life expectancy is about 10 years after operation.

Lymphosarcoma, fibrosarcoma, epidermoid carcinoma are seldom tumours of a thyroid gland.

At present, conventional echographic signs are not exist which are characteristic only for carcinomas. Nevertheless association of such signs as indistinct contours, the solid structure low echogenicity, presence of microcalcifications (dot hyperechogenic inclusions without an acoustic shadow or with a thin acoustic shadow), allows to suspect malignant character of such formation with the big percent of probability. According to the literary data, microcalcifications find at morphological research in 37 % of thyroid carcinomas.

Treatment operative is with the further beam therapy. Life expectancy is 1-3 years.

Metastasing adenoma is a tumour of a thyroid gland, which metastasizes in a bone, lungs, liver and brain. Metastasises keep structure of a thyroid gland, accumulate radioactive iodine, they can be shown by means of radiological research receptions and scanning. Metastasing at a bone quite often conducts to pathological fractures.

Duration of disease is 20-30 years. Now, consider that a source of metastasises is the malignant new growth of a thyroid gland of the smallest sizes which appears only at purposeful histologic researches.

Different morphological variants of adenomas echographically do not differ. Adenomas can be both single, and plural. Their form is oval or rounded. Contours, as a rule, is precise. Echogenicity is low, average or increased, in most cases is low. Adenomas of average and increased echogenicity, as a rule, have thin hypoechogenic rim on periphery - “ halo sign ”. Representation about given echographic sign as criterion of high quality is denied. In an adenoma there can be zones which contain a liquid and calcifications.

At color Doppler scanning by the most frequent variant expressed vascularisation on periphery of formation. Characteristic hypervascularisation is both on periphery, and in the central part for independent adenomas.

Anatomico-physiological data of parathyroid glands.

Parathyroid glands (superior andd inferior) settle down on a back surface of a thyroid gland and consist of ferruterous cells such as parathyrocytes, which located in the form of braces-trabecules.

Parathyroid glands product parathyroid hormone (PTH) - parathormone, parathyrin which in a combination to a hormone of a thyroid gland called calcitonin regulate an metabolism of calcium and phosphorus.

Parathormone. The mechanism of action.

Parathyrin causes increase of concentration of calcium in blood serum. Parathyrin activates increase resorption of calcium in bones, stimulates function of osteoblasts, reduces

calcium excretion and increases phosphorus excretion by kidneys, stimulates absorption of calcium in digestive tract, increases contents of calcium and phosphorus in blood serum.
Calcitonin has opposite action – it decreases in a level of calcium in blood sputum. Calcitonin improves fixing calcium in bones.

In blood sputum contains on the average, calcium from 2,1 up to 3 mmole/l and phosphorus from 0,65 up to 1,6 mmole/l. Increase of a level of calcium in blood is accompanied by reduction in a level of phosphorus and on the contrary. In the form of phosphoric-calcium connections they contain in chondral and bone tissues and in dents. About 99 % of calcium and 66 % of phosphorus are in bones. During a day the adult person with food stuffs should receive about 1000 mg of calcium.

Functional condition parathyroid glands and a level of calcium in blood are regulated by nervous and hormonal ways. Reduction of concentration of calcium in blood stimulates secretion of parathyrin with following increase of a level of calcium in blood.

Regulation of an exchange of calcium and phosphorus from parathyrin is carried out by calciferol and vitamin D which will stimulate absorption calcium and phosphorus, will mobilize calcium from bones, assists reabsorption of calcium and phosphorus in renal tubules.

Hyperparathyreosis. Etiology. Pathogenesis. Classification.

Hyperparathyreosis is a disease parathyroid glands which it is characterized by increase of secretion of parathormone and development of hypercalcaemia.

Etiology, classification. Primary hyperpathyreosis is more often caused by an adenoma or hyperplasia and less often by a carcinoma of parathyroid glands. Adenomas of parathyroid glands single or plural, appear in 80-85 % of patients. Hyperplasia is observed in 15-20 % of patients. The cancer of parathyroid glands meets in 0, 5-3 % of cases.

Hyperparathyreosis caused by hyperplasia or new growths of parathyroid glands, happens sporadic or family to autosome-dominant type of inheritance. Hereditary primary hyperparathyreosis is one of components of syndromes plural endocrine adenomatosis (MEA) or neoplasia (MEN) more often. MEN type I meets in 90 % of patients and MEN type of II type - in 10-50 % of patients. In both cases hyperparathyreosis caused by hyperplasia of all parathyroid glands, less often – of adenomas.

Pathogenesis. Development of an adenoma parathyroid glands connected with two types of mutations. I type is a mutation in mitosis control; II type is a mutation of the mechanism of the final control of secretion parathormonea calcium. It is considered, that the mutation concerns one of genes which code fibers which take part in transport of calcium at a cell parathyroid glands.

The mutant cell has increased secretory activity and gives a new clone of cells which amount uncontrollably increases with formation of an adenoma which sometimes carries out independent secretion of a hormone. In some cases under influence calcitriole or a low level of calcium there is a population quickly proliferated cells of parathyroid glands which leads primary either secondary hyperplasia or a hyperplastic adenoma, and also to development polyclonal adenomas of parathyroid glands.

Clinic, clinical forms of hyperparathyreosis.

Primary hyperparathyreosis, caused by hypercalciaemia owing to hypersecretion parathormone, it is shown by great variety of symptoms. Distinguish some clinical forms: bone, renal, gastrointestinal (a ulcer to a stomach, a pancreatitis, a cholecystitis), cardiovascular (an arterial hypertension) and others.

In 50 % of cases of illness runs asymptomatic and only revealed hypercalciaemia allows to suspect hyperparathyreosis.

Clinical displays primary hyperparathyreosis are characterized the central nervous system with presence of fatigue, weakness, a headache, depression, infringement of appetite, psychoses and comas.

Lesion of muscles and joints appears myopathy, a gout, pseudopodagra, chondrocalcinoses, an erosive arthritis.

Lesion of eyes with development of a cataract, and also adjournment of calcium in superficial layers of a cornea and forward to a boundary plate is observed.

Development of an arterial hypertensia and arrhythmia, calcifications of hearts and vessels are observed from cardiovascular system.

Development of a gastroesophagal reflux, a stomach and duodenal, a cholelithiasis, a pancreatitis, a lock is quite often defined because of lesion of digestive organs.

Characteristic is the lesions of kidneys with presence of polyuria and polydypsia, reduction in concentrating abilities of kidneys, development nephrolithiasis, nephrocalcinose and renal tubule acidosis.

The clinic becomes complicated presence of a fever and development of an anemia.

It is necessary to note, that atrophic processes of big groups of muscles with decrease in amplitude of potentials their contractions are involved.

Sometimes there are difficult lesions of bones presence to an fibrotic-cystic ostitis and replacements hematopoietic tissues of a bone brain a connective tissue.

Diagnostics of hyperparathyreosis.

For confirmation of the diagnosis shown biopsy a bone tisuue. Irrespective of weight of disease, histologically it is defined a thining of compact substance of tubular bones, a cell of replacement of a bone tissue by fibrous tissue with a plenty of osteoclasts and macrophages, loaded hemosiderin (bones and brown tumours).

At densitography of bones there is a significant decrease in density of a bone tissue, diffuse demineralisation of a bone tissue, diffuse osteolysis, subperiostal resorption of a bone tissue in phalanxes of fingers of a hand, subperiostal erosions in long tubular bones and bones of a skull.

At percussion above bones of a skull there is a sound of ''a ripe water-melon''. In bones of a backbone there are changes with a different degree to a bony rarefication. The increased risk of spontaneous crises of bones of a forearm, femurs and a backbone is characteristic. Quite often patients specify reduction of growth during disease, there are changes of proportions of a body. In position standing wrists of arms can reach a level of a knee joint.

Nephrolithiasis is characterized by presence of one stone or plural calculi in both kidneys. Surgical removal of a stone does not lead to recover. Stones of kidneys consist from oxalate or phosphate of calcium. Nephrocalcinosis spreads less often, runs across from calcifications of renal tubules (epithelial layer, base membranes and interstitial layer), with decrease glomerule filtrations and functions proximal departments renal tubules (aminoaciuria, glycosuria, decrease in concentration ability of kidneys).

The stomach ulcer is characterized by the frequent aggravations, the expressed painful syndrome and quite often becomes complicated perforation. The dairy diet and alkaline salts as medical means are not recommended, as lead to development a hypercalcimic crisis (hyperparathyreosis and alkalic-lactic syndrome). The peptic ulcer can be display of syndrome MEN I or MEN II, and also be united with Zollinger-Ellison syndrome.

Lesion of cardiovascular system is characterized by adjournment of salts of calcium in a myocardium, calcification of coronary arteries and valves of heart (aortic, mitral), a hypertrophy of left ventricle and presence of a hypertensia.

Laboratory diagnostics includes a number of exams.

1. Definition of concentration of free calcium in blood serum. Sometimes with increase of parathormone concentration of free calcium remains normal (normocalcimic hyperparathyreosis), that can be caused by disturbance of tubule reabsorption of calcium, calcium absorption in intestines, presence of avitaminosis D (at accompanying treatment by vitamin D arises hypercalciaemia, at isolated - restoration normal calciaemia); at early stages of development

primary hypercalciaemia. Reliability of the diagnosis proves to be true provocative teste with thiazide-type diuretics: it is normalized up to the end of the first week with hydrocotisone at absence hyperparathyreosis.

1. Definition of level of PTH in blood septum with simultaneous measurement of a level of the common or free calcium.
2. Definition of a level of the common or nephrogenic cAMP in urine.
3. Definition of a level of calcium in urine (happens normal or increased)
4. Definition of phosphates in blood sputum (meets hypophospataemia at reduction to threshold tubule reabsorption of phosophate and reduction of the relation in maximal tubule phosphate reabsortion by the speed tubule filtration).
5. Definition of the relation in chloride/phosphates in blood sputum (in norm which is less 32; increases) is more often.
6. Definition alkaline phosphotase, fibers, creatinine, residual nitrogen of blood sputum.
7. The clinical analysis of blood (normochromic anemia, moderate leucocutosis and increases ESR).

Tool diagnostics includes a number of exams.

1. Ultrasound exam in 50-60 % of cases allows to show increase parathyroid glands.
2. CT with enhance and MRI allow to show lesion parathyroid glands in 90 % of cases.
3. Subtractional scintigraphy with thallium (Tl^{201}) and technetium (Tc^{99m}).
4. Selective phlebography with catetherisation of a unpaired thyroid plexus from definition of concentration parathormone.
5. Electrocardiography.
6. X-rays bones of a skeleton and kidneys.
7. Densitometry of bones.
8. The analysis bone biopsy by means of quantitative histometry.

The diagnosis is based on data of the anamnesis, complaints, a clinical picture and results of additional exams. Constant attributes of hyperparathyreosis are hypercalciaemia, hypophosphatemia, increase in contents alkaline phosphatase in blood sputum. Sometimes, hypomagnesaemia, increase of chlorides (above 102 mmole/l), decrease in bicarbonates (hyperchloric acidose) is defined. The relation of concentration of chlorides in blood and phosphates makes 33:1. There is an increase calcium excretion, phosphorus and hydroxyproline with urine, and also contents interleukin-6 and the tumour factor necrosis in blood sputum.

Differential diagnostics hyperparathyreosis.

Conditions and diseases which are accompanied by development of hypercalciaemia are considered. Allocate 5 groups of diseases with hypercalciaemia.

1. Hypercalciaemia owing to excessive secretion parathormone:

a) primary hyperparathyrosis;
b) secondary hyperparathyrosis;
c) tertiary hyperparathyrosis;
d) plural endocrine adenomatosis (MEA) I-st and II-th type;
f) hyperparathyreosis with ectopic production of parathormone (pseudoparathyperparathyreosis).

2. Endocrinopathic hypercalciamia:

a) thyrotoxicosis;
b) adrenal chronic insufficiency;
c) pheochromocytoma;
d) adenoma which to secret vasoactive intestinal peptide.

3. Malignant new growths:

a)osteolythic metastasis of malignant tumours at a bone;
b) diseases of system of blood (leukaemia, lymphoma, lymphogranulomatosis, multiple myeloma).

4. Medicamentous hypercalciaemia:
a) alkalic-lactic syndrome;
b) treatment thyazide-type diuretics;
c) overdose of vitamins A and D;
d) treatment by preparations of lithium;
f) treatment of a cancer of a mammary gland with oestrogen, antioestrogen and testosterone.

5. Hypercalciaemia:
a) fractures of bones;
b) somatic diseases which arrest the patient to a bed for long term.

Secondary hyperparathyreosis is compensatory reaction on long hypocalciaemia that develops owing to disturbances of processes absorption in intestines (syndrome malabsorption) or rickets, Fanconi's syndrome and chronic renal insufficiency. Contents of calcium in blood sputum in normal or low (there is no time does not happen increased), and concentration of inorganic phosphorus is increased (at renal form secondary hyperparathyreosis) or low (at the intestinal form secondary hyperparathyreosis).

Clinically secondary hyperparathyreosis appears with symptoms and attributes of the basic disease. Paraesthesias are observed different localization with characteristic spasms of muscles of hands or feet during hypocalciaemia. In proximal parts of extremities there is a weakness of muscles. Changes of a bone tisuue are shown by a bony rarefication, an osteosclerosis or an fibrotic-cystic osteitis.

Chronic renal insufficiency is accompanied by disturbance of activity of enzyme I-hydroxylase and insufficient formation of metabolite of vitamin D($1,25(OH)_2$-D_3), that is designated on processes of absorption of calcium in intestines. There is hypocalciaemiae, hypophosphateamia, an increase of a level of alkaline phosphatise in blood sputum, presence hyperchloremic acidose owing to reduction reabsorption of bicarbonates in renal tubules, and also surplus parathormone.

At carrying out of a hemodialysis on chronic renal insufficiency develops in patients also hypocalciaemia, and also a bony rarefication, osteodystrophy. The increased secretion parathormone strengthens changes in a bone tissue. Secondary hyperparathyreosis at a chronic hemodialysis quickly passes in tertiary hyperparathyreosis when hyperplasia of parathyroid glands transformed to an adenoma which to excessively secrets parathormone.

Pseudohyperparathyreosis or ectopic hyperparathyreosis meets at malignant tumours of different localization, more often bronchogenic cancer, less often at a cancer of a mammary gland. Sometimes tumours produce parathormone or parathormone like substance which is accompanied hypercalciaemia. Hypercalciaemia connects with ability of these tumours to form osteoclast-active factor which stimulates osteoclast osteolysis which action depress steroids at multiple myeloma and lymphome. Metastasing malignant tumours in a bone tissue is accompanied by increase of a level prostaglandin E2 which strengthens inflammatory reaction and destruction of a bone tissue.

Hyperparathyreosis meets at three hereditary syndromes which are transferred on autosome-dominant type: plural endocrine adenomatosis I type ((I) and II type (II)), family hypocalciuric hypercalciaemia. MEA I (Wermer's syndrome) – adenomatosis of hypophysis, pancreas and parathyroid glands. Chromophobic adenoma of a hypophysis can run across with a clinical picture acromegaly or Itsenko-Cushing illnesses. Third of patients has a stomach ulcer at Wermer's syndrome.

Sipple's syndrome (MEA II) runs across in the form of two variants.

MEA IIA - hyperparathyreosis, medullary cancer of a thyroid gland and pheochrocytoma, are accompanied by the increased secretion of calcitonin.

MEA II B – medullary cancer of a thyroid gland, pheochrocytoma, plural neuromatosis of mucous membranes, deformation of a skeleton which reminds Morphane's syndrome. Plural neurinomas are localized on conjuctive, mucous membranes of oral cavity, of digestive tract.

Family hypocalciuric hypercalciaemia meets at young age, has a benign course, despite of hypercalciaemia and hypermagnesiaemia, reduction calcium and magnesium excretion with the urine, the expressed signs of hyperparathyreosis are absent.
Hypercalciaemia is accompanied by decrease calcium absorption in intestines and excessive it excretion with urine and faeces at thyreotoxicosis.
Hypercalciaemic crisis is observed at primary and tertiary hyperparathyreosis, intoxications by vitamin D and hypercalciaemia, that is united with malignant tumours. The increase in contained calcium above 3,49 mmole/l leads to development of signs to an calcium intoxication. The crisis develops at the raised level of calcium of blood from above 3,99 mmole/l.
Hypercalciaemic crisis is accompanied anorexia, faintness, continuous vomiting, pains in epigastric areas, locks, polydypsia, polyuria. Then develops oligouria and anuria, dehydration of an organism, the hypotonia of muscles and acute muscular weakness, pains in bones. At the first o'clock there can be an arterial hypertension. A skin is dry, with on traces of scratch through a strong itch. Ligament reflexes are low. There are psycho-neurologic affections in the form of depression, obscure in consciousnesses, coma, psychoses or psychomotor excitation. Anuria develops, cardiovascular insufficiency, oppression of activity of CNS, oppression of the respiratory and vascular centers and develops irreversible shock. Hypercalciaemic crisis can be accompanied difficult by gastrointestinal bleedings, development of intravascular thromboses, ‘’DIC-syndrome’’.

Treatment of hyperparathyreosis.
The optimum method of treatment primary hyperparathyreosis is surgical intervention. Medicamental treatment is appointed patients in the age of 50 years with abstinent hypercalciaemia, slightly low weight of bones and the mentioned function of kidneys is low.

The indications to surgical treatment.
The parameter for surgical treatment is clinical displays of hypercalciaemia, increase of calcium in of blood sputum which exceeds norm on 0, 25-0,4 mmole/l, presence hypercalciaemic crisises in the anamnesis, decrease in speed glomerule filtration is more than on 30 % in comparison with norm, presence nephrolithiasis, decrease in weight of bones is more than on 2 standard deviation; daily excretion is more than calcium 10 mmole; the age is more than 50 years; impossibility of long supervision over patients.
Radical treatment in first two day is accompanied hypocalciaemia, develops complication: a syndrome of hungry bones (variations of fleeting hypocalciaemia), a syndrome hypoparathyreosis, damage of recurrent nerve.
The most complex difficult is palliative operation. Among invasive methods of treatment apply injections of plenty of X-rays contrast agent means in artery which feed parathyroid glands and transcutaneous introduction ethanole in adenoma parathyroid glands under the control of ultrasoins.

Medicamental therapy of hyperparathyreosis.

Struggle with hypercalciaemia by means of forced diuresis: into the first 2 hours enter intravenously 3 l a isotonic solution of chloride of sodium, then prescribe 100 mg furosemide each 2 hours or 40 mg ethacrynic acids.

Phosphates enter in the form of 0,1 M of the phosphatic buffer (500 ml) intravenously slowly or in tablets, for 3-4 receptions. Intravenous infusion phosphates carry out under the control of a level of urea, phosphorus and others electrolytes of blood sputum.

Antiresorptive therapy with application biphospates: etidronat on 10-20 mg/kg on times or klodronat on 1,0-3,0 g/day. Pamidronat apply in a doze of 15-60 mg intravenously 1 time on times or orally on 1200 mg on times during 2 weeks. Residronat apply intravenously on 10 mg of

1 times on day. Sodic salt ethylendiamintetraacetum acids apply with count of 50 mg on 1 kg of weight of a body intravenously during 4-6 hours.

Expedient it is considered also application of an antibiotic mitramycin in a doze of 25 mkg/kg intravenously at hypercalciaemic crisis, and at a chronic course in a doze of 10-12 mkg/kg 1-2 times for a week. It is necessary to consider his toxicity with development thrombocytopenia, necrosis tissues of a liver and proteinuria.

The increase calcium excretion with urine and reduction of his absorption in intestines is observed also after the use glucocorticoids. Prednisolone prescribes on 40-80 mg per day.

At hypercalciaemic crisises in patients with massive metastasises of malignant tumours prescribe indometacin on 25 mg each 6 hours or acetylsalicilic acid which have properties to block synthesis prostaglandins.

Treatment of hypercalciaemic crisis carries out also by means of a dialysis with special solutions without calcium.

The positive effect at hypercalciaemia finds out also calcitonin at parenteral introduction (intravenously, intramuscularly, hypodermically) or cimetidin (blocker H 2-receptors histamine).

At women from primary hyperparathyreosis in postmenopause hypercalciaemia eliminate by the use oestrogene in association with progesterone.

The choice of therapy depends on a degree quality of hypercalciaemia.

The postoperative period and rehabilitation of patients.

Preventive maintenance postoperative tetany consists in purpose of a dairy diet with high contents of calcium and low - phosphorus, preparations of vitamin.

Hypoparathyreosis. Etiology. Pathogenesis. Classification. Clinical forms.

Hypoparathyreosis is a disease caused by insufficiency of secretion parathormone of parathyroid glands, decrease calcium reabsorption in renal tubules which is accompanied by reduction of absorption of calcium in intestines with development hypocalciaemia.

Allocate some groups of diseases and states which are the reason hypothyreosis.

Deficiency parathormone caused postoperative hypothyrosis, idipathic hypoparathyreosis connected with syndrome De Georges (agenesia of parathyroid glands), metastasises in parathyroid glands, lesion of parathyroid glands at granulamatosis, hemochromatose, Wilson's disease, intoxications of aluminium, after treatment I131.

Oppression of secretion parathormone, increase of a level of magnesium in blood sputum and after treatment amiphostine.

Resistency to parathormone is observed at pseudohypoparathyroesis with type Ia, Ib, Ic, II, at presence autoantibodies to parathormone and deficiency of magnesium.

The strengthened grap of calcium for bones at a syndrome of "hungry" bones, after treatment I131, at treatment osteomalation of vitamin D and owing to disturbance of an exchange of vitamin.

Postoperative hypoparathyreosis more often caused to damages or removal of parathyroid glands at surgical interventions, stress owing to partial or fleeting deficiency parathormone and clinically expressed hypocalciaemia and also after massive transfusion of plasma or cytratic blood.

Idiopathic hypoparathyreosis is observed at any age in the form of sporadic and hereditary cases. It is shown hypocalciaemia, absence or a low level of parathormone in blood sputum, increase of a level of calcium after introduction of parathormone and hyperphosphate, decrease in a level of osteocalcitonin and 1,25 2-D3 in sputum at normal equal 25-D3, decrease excretion calcium in untreated patients.

Hereditary hypoparathyreosis is a component autoimmune multigland syndrome (AMGS type I), that is observed at chronic general candidosis and primary adrenal insufficiency, and also at primary hypothyrosis, primary hypogonadism, a chronic hepatites with the expressed activity,

at a syndrome malabsorption, vitiligo, autoimmune gastritis, alopecia, steatorrhea. Isolated idiopatic hypoparathyreosis meets at any age, concerns to family diseases with autosome-recessive or autosome-dominanant type of the inheritance, caused by deficiency of synthesis or secretion parathormone.

Hypoparathyreosis as Kirns-Seir's syndrome is united with pigmentary retinitis, ophthalmoplegia, ataxia, atrioventricular blockade, myopathy. Hypoparathyreosis as Cann's syndrome is united with a growth inhibition and narrowing bone-marrow cavities of long tubular bones. Sometimes hypoparathreosis is united with deafness, mitral insufficiency, nephritis. Born dysgenesia of parathyroid glands is observed at syndrome De Georges, it appears with aplasia or dysplasia of parathyroid glands, thymus dysplasia owing to disturbance of emryogenesis in the third and fourth pharyngeal pockets.

Hypoparathyreosis with hemochromatosis is accompanied by deposit of iron, destruction and fibrosis in parathyroid glandsax.

Hypoparathyreosis at Wilson's disease caused to deposit of copper in parathyroid glands.

Parathormone in chronic renal insufficiency partial or full deficiency is connected with deposit of aluminium in parathyroid glands.

On a background of the increased level of parathormone at a syndrome of resistency to parathormone, it is observed hypocalciaemia and hypophosphateamia. This condition is defined as pseduohypoparathyreosis.

At type Ia (hereditary Albright's osteodystrophy) symptoms of hypocalciaemia are united with stunting, brachydaclia, menolike face, adiposity, wing folds on a neck, numerous foci of hypodermic calcification or ossification, sometimes intellectual backwardness.

With this condition it allocate pseudohypoparathyreosis type Ib, what caused with defect of a receptor of parathormone; type Ic - caused by defects adenilatcyclase and Gsa; type II - connected with hereditary infringements of a metabolism of vitamin D.

Other forms of resistency to parathormone connect with family resistency of kidneys to parathormoney, resistency to endogenous parathormone, defect of synthesis 1,25-(VOH)2D3.

Clinic of hypoparathyreosis

The disease is characterized symptom association that includes convulsive reductions of skeletal and corpulent muscles owing to insufficiency of parathormone with development hypocalciaemia and increase nervous and muscular excitability.

Tetany distinguishes in attacks, equivalent tetany, and latent tetany.

The tetany attack arises spontaneously or is provoked by mechanical, acoustic irritation or hyperventilation. Begins suddenly or from harbingers (the general weakness, muscular pains, paraesthesia in the field of the face, extremities). With the lapse of time join fibrillar twitching of separate muscles which pass in tonic or clonic convulsions. Convulsions of muscles of the superior extremities appear prevalence of action of muscles which carry out bending, and the extremity accepts a position "an obstetrician's hand". Convulsions of muscles of the inferior extremities are accompanied by prevalence of action of muscles which carry out extension of extremities and plantar bending - "a horse foot". Convulsions of muscles of obverse muscles are accompanied trismus, convulsions of eyelids, a sardonic smile or " the fish mouth ". Muscular convulsions are very painful.

Functions of vegetative nervous system rise. During an attack of tetany the bronchospasm, high perspiration, renal and hepatic colics owing to a spasm of smooth muscles in renal pelvis, ureters and Oddi's sphincter are observed. It is sometimes observed laryngospasm. Involving spasms of muscles of vessels, attacks of a migraine or Raynaud's syndrome are observed. There are attacks which remind attacks of epilepsy.

Latent tetany is a condition which the attack of tetany can be caused only provoking diagnostic procedures.

Trousseau's syndrome is convulsive contractions of a wrist in the form of “an obstetrician’s hand”, that come in 2-3 minutes after imposing a plait.

Chvostek’s symptom is contraction of muscles of the face at strokes of hammer or palpation in the region of an exit of facial nerve (ahead from tragus). At Chvostek-I contraction of muscles of a corner of a mouth is observed only. At Chvostek-II there is a contraction of muscles of a corner of a mouth and wings of a nose. Chvostek-III it is characterized by contraction of all muscles of half of face.

Erb’s symptom is a decrease of a threshold of sensitivity stimulation by a galvanic current, occurs convulsive contraction of muscles.

Witt’s symptom - at percussion on external edge of an eye pole occurs contraction of a circular muscle of an orbit and a muscle of a forehead.

Schlisinger’s symptom - after passive bending an extremity in a coxofemoral joint at a straightened knee joint is observed convulsive contraction of muscles of a hip and supination of foot.

Test is applied to revealing the latent form of tetany with hyperventilation of lungs. Alkalosis which develops at hyperventilation, leads to decrease in calcium and occurrence of positive Trousseau’s and Chvostek’s symptoms.

At latent hypothyreosis is observed frequently a dry and icteric skin, fungoid lesions of wrist, eczema or psoriasis, dryness and fragility of hair, drop out of eyelashes and eyebrowes, fragility of nails, sometimes a cataract, hypodermic calcificates in the region of auricles (with presence of hyperphosphateamia), ossificated myositis. At pseudohypoparathyreosis there is a shortening of metacarpal, metatarsal bones and phalanxes, lesion of a teeth - caries, premature drop out.

The diagnosis proves to be true the clinical displays, the anamnesis data, the indication on treatment by radioactive iodine, at decrease in a doze thyroid hormones, after application of diuretics, at presence of infectious diseases, after operation on a thyroid gland, at diseases of a liver and a dive, presence chronic renal insufficiency, diseases of intestines, a chronic pancreatitis.

Diagnostics hypoparathyreosisy. Additional test maneuvers

Decrease in calcium in blood sputum is defined below 1,87 mmole/l, and the ionized calcium - below 1,07 mmole/l.

Test is applied to revealing latent hypoparathyreosis from EDTA or trilone B.

Diseases proves to be true presence of a triad: hypocalciaemia, hyperphosphateamia, hypocalciaemiauria.

There is a lengthening interval Q-T, isoelectric interval S-T at constant wave T on ECG. X-rays of soft tissues allows to show a kidney calcification, muscles, basal ganglions, and X-rays of skeleton - a osteoporosis, subperiostal resorption of bones, rachitic deformations of a skeleton.
Differentiated diagnostics of hypoparathyreosis

The differential diagnosis is spent with diseases and conditions which lead to development hypocalciaemia. First of all hypocalciaemia is consequence of insufficiency of secretion parathormone in patients with hypoparathyreosis caused autoimmune genesis with multiendocrine autoimmune syndrome, the MEDAS-syndrome, syndrome De Georges, born ugliness, immunological insufficiency, postoperative hypoparathyrosis, hypoparathyreosis after treatment by radioactive iodine, owing to a trauma, a sarcoidosis, a tuberculosis, tumours of a neck with destruction parathyroid glands.

Allocate functional hypoparathyreosis at insufficient secretion of parathormone in reply to hypocalciaemia, that is observed in newborns that were born from mothers sick on hypoparathyreosis, and also at idiopatic neonatal hypocalciaemia, at hypomagnesaemia (a malabsorption, a vomiting, a diarrhea, a steatorrhea, a diabetes, a acute pancreatitis, an alcoholism)), at insufficiency of vitamin D (a alimentary deficiency, lack of a ultra-violet irradiation, a malabsorption, a steatorrhea, a syndrome of short intestines, a sprue, a chronic

pancreatitis). It is necessary to consider hypocalciaemia, that is consequence of peripheral resistency to parathormone, which is observed at pseudohypoparathyreosis (Albright's syndrome), hypomagnesaemia, uraemia, insufficiency of vitamin.

The use of thiazid-type diuretics, laxatives, phenobarbital, anticonvulsant means, after massive transfusion of a citrate blood, overdose calcitonin, the use of biphosphonates is quite often defined, hypocalciaemia is after introduction of phosphates, EDTA, mitramycin, actinomycin, neomycin.

It is necessary to note, that at pseudohypoparathyreosis the level of parathormone in blood is increased, as well as at lack of vitamin D, and also at renal insufficiency. At avitaminosis D hypocalciaemia is united not only with the increased level of parathormone in blood, but also with hypophosphateamia and increase in contents alkaline phosphatase in blood sputum.

Treatment of hypoparathyreosis

The basic place in treatment belongs to vitamin D in a combination to drugs of calcium. Vitamin D2 calciferol or ergocalciferol, vitamin D3 - cholecalciferol, dihydrotachysterolate-10, tachyrol, hydroxylecalciferol-oxidevite, uran-a, a-calcidol, 1-alphacalcidol, 1,25-dihydrooxycholecalciferol - rocaltrol or calcitryol is applied.

Vitamin D2 is prescribeed on 1-2 mg per day, gradually increasing a doze up to 0,25 mg with each 14 days to increase of calcium in blood up to 2 mmole/l.

After achievement nomocalcieamia in patients translates on a diet with the increased contents of calcium - dairy-vegetable diet. For restriction absortion of phosphates prescribe 20-30 mg dihydroxide aluminium.

At tetany intravenous introduction of 10 % of a solution of chloride of calcium or gluconate calcium is recommended. At an inefficiency after the injected doze it is recommended intravenous injection of calcium at the rate of 15-20 mg of calcium on 1 kg of weight of a body (in 1 ml of 10 % of a solution of gluconate calcium 9 milligram of calcium contains).

Solution of calcium injects together with 5 % a solution of glucose during 4-6 hours.

At treatment hypocalciaemia it is necessary to carry out constantly the control over contents of calcium, phosphorus, magnesium in blood sputum, and also behind a functional condition of kidneys. Achievement of last years are applications of transplantation parathyroid glands.

Control of entry-level knowledge:

Task 1

Hyperfunction of the thyroid gland during diffuse toxic goiter associated with:

a) hypersecretion of TSH,

b) hypersecretion tiroliberina;

c) hyperproduction of thyrostimulated immunoglobulins

Task 2

Which of the following studies are used for differential diagnosis between neurosis and diffuse toxic goiter?

a) test with TSH,

b) test with triiodothyronine**;**

c) test with potassium perchlorate;

d) uptake of I-131 by thyroid gland

Task 3

Which of these drugs affect the absorption of thyroid I131 mostly?

a) bromine preparations

b) vitamin B**;**

c) enteroseptol;

d) asparkam

Task 4

The treatment of endemic diffuse goiter:

a) conservative;

b) surgical,

c) combined

Task 5

What is a purpose to scanne the thyroid gland?

a) determination of thyroid size,

b) defines the functional state,

c) determination of tissue structure,

d) determination the presence of nodes units and determination of their functional state

Task 6

What is a disease that causes raise of the control of thyroid function by the hypothalamic-pituitary system?

a) endemic goitre;

b) primary hypothyroidism,

c) diffuse toxic goiter

Task 7

What is age of patients who need prolonged drug therapy of diffuse toxic goiter and surgery treatment is carried out only by indication?

a) the elderly people with cardiac arrhythmias, and IBS;

b) y middle-aged persons,

c) children and adolescents;

d) age does not influence the choice of treatment

Task 8

What therapeutic dose of thyroxine is prescribed to patients with a normal level of TSH in the blood:

a) 2 mg / kg,

b) I mg / kg**,**

c) 0,5 mg / kg

Task 9

How long can be carried out antithyroid drug therapy of diffuse toxic goiter under the condition to achieve and maintain euthyroid state?

a) 3 months

b) 6 months,

c) 1 - 1,5 years

Task 10

What complications can be observed during treatment of thyreostatics from imidazole group?

a) thrombocytopenia,

b) dyspeptic disorders,

c) leukopenia,

d) strumogennic effect,
e) all of the above

Task 11

What symptoms can help you to suspect the presence of hypocorticism among patients with DTG?

a) hypotension,
b) weight loss,
c) flat-sugar curve,
d) all of the above

Task 12

Which of these types of arrhythmias is most typical for diffuse toxic goiter?

a) paroxysmal tachycardia;
b) atrium extrasystole;
c) ventricular extrasystole,
d) ciliary arrhythmia,
e) sinus tachycardia

1	2	3	4	5	6	7	8	9	10	11	12
C	B	A	A	D	C	A	B	C	E	D	E

Control of leaf-level knowledge

Task 1

What changes are common to blood test during diffuse toxic goiter?

a) leukopenia,
b) accelerated erythrocyte sedimentation rate,
c) lymphocytosis,
d) all of the above

Task 2

The most optimal time for surgical treatment of diffuse toxic goiter during pregnancy:

a) first 2 months,
b) end of the first and beginning of second trimester,
c) second trimester,
d)3 trimester;
e) surgical treatment is not recommended

Task 3

Apathetic form of thyrotoxicosis is characterized by:

a) apathy, depression,
b) significant weight loss,
c) proximal myopathy, a shimmering
d) 80-arrhythmias;
e) all of the above

Task 4
What examination can help to differentiate sporadic goiter from endemic?
a) level of T4 in blood; 0-the level of TSH in blood;
b) ultrasonic scanning of thyroid gland,
c) dayly urinary iodine excretion

Task 5
Which drugs are most appropriate to assign for treatment of endemic goiter with 3 degree of patients of elderly age?
a) antistrumine;
b) thyroxine;
c) triiodothyronine;
d) thyreocomb

Task 6
Local myxedema is characterized by edema at the following locations of the patient body, except:
a) front surface of the tibia;
b) waist;
c) orbits of the eyes;
d) hip

Task 7
What severity of thyrotoxicosis is determines?
a) enlargemed of goiter,
b) degree of tachycardia,
c) the dynamics of body mass

Task 8
The most informative examination of nodular goiter is:
a) ultrasonic scanning of thyroid gland,
b) scan thyroid gland,
c) puncture biopsy,
d) thermography;
e) uptake of I131 by thyroid gland

Task 9
A patient with progression of thyrotoxic ophthalmopathy with compensated hyperthyroidism is receiving mercasolil, first of all necessary:
a) to increase the dose of mercasolil;
b) to reduce the dose of mercasolil;
c) to designate lithium carbonate;
d) to designate glucocorticoids

Task 10
What is the optimal period for surgical treatment of diffuse toxic goiter in the presence of severe liver and heart damages?
a) after achieving eutiroid state;
b) 1 month later after the compensation of thyrotoxicosis,
c) 2-3 months later after the compensation of thyrotoxicosis

Task 11

Sympathadrenalic reactions during diffuse toxic goiter, caused by:
a) increased production of catecholamines,
b) hypersensitivity of β-receptors of catecholamines;
c)direct action of thyroid hormones

Task 12
The most effective method of treatment of endocrine ophthalmopathy is:
a) subtotal resection of the thyroid gland;
b) paraorbital introduction of glucocorticoids,
c) irradiation of orbits;
d) receiving of glucocorticoids with further irradiation of the orbits

1	2	3	4	5	6	7	8	9	10	11	12
D	B	E	C	D	B	B	C	D	C	B	D

Tests

Task 1
What drug should be prescribed to a pregnant with gestation of 12 weeks after removal of the nodular goiter?
a) antistrumin;
b) thyroxine;
c) iodine preparations

Task 2
The main mechanism of antithyroid effect of glucocorticoids is characterized by:
a) changed sensitivity of tireotrofs against thyrotropin-releasing hormone;
b) reduction of transition of T4 to T3;
c) oppressioned formation of thyreostimulated antibodies;
d) reduction of synthesis of steroid hormones;
e) blocked absorption of iodine thyroid

Task 3
What is a reason of increased level of secretion of thyroid hormones?
A) mental and physical stress;
b) acute change in the temperature
c(eating disorders

Task 4
Which of tumors often combined with nodular goiter?
a) liver tumors; 0lung tumors;
b**)** uterine fibromyoma;
c) ovarian tumors;
d) mammary tumors

Task 5
The classical picture of congenital unrecognized hypothyroidism among children manifests itself at age of 5 -6 months:
a) delayed mental and physical development;

b) dysfunction of internal organs;
c) trophic disorders of skin;
d) all of the above

Task 6
What could cause the development of iatrogenic thyrotoxicosis?
a) overdose of thyroid hormones during treatment of hypothyroidism;
b) hypersensitivity of receptors to endogenous thyroid hormones;
c) all of the above

Task 7
The negative effect of B- adrenergic blockers on fetus among patients with diffuse toxic goiter caused by:
a) teratogenic effects; 0-inhibition of cardiac excitation of the fetus;
b) inhibition of excitation of the respiratory center of the fetus;
c) damaging effect on the thyroid gland;
d) risk of developing hypothyroidism

Task 8
Triiodothyronine thyrotoxicosis is characterized by:
a) increased level of T3 in the blood;
b) normal levels of T4 in the blood;
c) presence of both signs

Task 9
What blood pressure is characteristic for uncomplicated form of diffuse toxic goiter?
a) elevated systolic and diastolic pressure;
b) increased systolic and decreased diastolic pressure,
c) diastolic pressure increased with normal systolic;
d) diastolic pressure increased with reduced systolic;
e) reduced systolic and diastolic pressure

Task 10
What method is most informative for the diagnosis of ectopic goiter?
a) thermography;
b) survey radiography of the chest,
c) roentgenoscopy of retrosternal space,
d) ultrasonography;
e) scan with I131.

Task 11
What is the average daily requirement for iodine of the human body?
a) 50 – IOOmkg;
b**)** 50-250mkg;
c) 250-350mkg

Task 12
The most characteristic feature of endemic goiter is:
a) high (50%) uptake of iodine 131 by thyroid gland for 24 hours;
b) low excretion of iodine with urinary (less than 50 mg a day)

c) normal level of total T4 and decreased level of free T4

1	2	3	4	5	6	7	8	9	10	11	12
B	C	B	B	D	A	B	C	B	E	B	B

Test questions

1. The indicators of iodine metabolism
2. Methods for determining the size of thyroid gland.
3. The definition of "goiter".
4. Classification of goiter by Nikolaєva (1955).
5. Classification of goiter by WHO (1992)
6. The definition of «thyrotoxicosis ".
7. The list of diseases that are accompanied by thyrotoxicosis.
8. Determination of diffuse toxic goiter (DTG).
9. The etiology, pathogenesis and clinical manifestations of diffuse toxic goiter.
10. Clinical syndromes that occur among patients with diffuse toxic goiter.
11. The defeat of the thyroid gland during diffuse toxic goiter.
12. Symptoms of Valsalva, Gutman.
13. The defeat of the cardiovascular system during diffuse toxic goiter.
14. ECG changes during diffuse toxic goiter.
15. Psycho-emotional disturbances during diffuse toxic goiter.
16. Thyrotoxic encephalopathy during diffuse toxic goiter.
16. Thyrotoxic myopathy during diffuse toxic goiter.
17. Thyrotoxic myelopathy during diffuse toxic goiter.
18. Dysfunction of the gastrointestinal tract during diffuse toxic goiter.
19. Changes in the skin during diffuse toxic goiter.
20. Local myxedema.
21. Bone diseases during diffuse toxic goiter.
22. Thyrotoxic and endocrine ophthalmopathy.
23. Eye symptoms of thyrotoxicosis.
24. Thyrotoxic exophthalmos and its symptoms Dalrymple, Ellinek; Shtelvag; Krause, Singer; gates of Rozenbaha; Beysman; Gifford; Knis; Lewe; Becker.
25. Symptoms caused by disturbance of oculomotor reaction: Wilder, Mobius; Kouen; Graefe, Popov; Senton; Kocher.
26. Autoimmune ophthalmopathy.
27. Ocular symptoms of autoimmune ophthalmopathy: Mann; Endrashik; Swecker; Abadi; Ballee.
27. Complication of autoimmune ophthalmopathy:
28. Features of toxic goiter among children and elderly people.
29. Atypical variants of thyrotoxicosis: "apathetic" thyrotoxicosis and juvenile thyrotoxicosis.
30. Substantiation of diagnosis of thyrotoxicosis.
31. Investigation of hormonal levels.
32. Test of TRH.
33. Drug treatment during diffuse toxic goiter.
34. Surgical treatment of toxic goiter.

35. Therapeutic use of 131-iodine.
36. The indication for treatment with radioactive iodine.
37. Complication after treatment of goiter.
38. The indication for surgical treatment during diffuse toxic goiter.
39. Complication after surgical treatment of diffuse toxic goiter.
40. Side effects of thyreostatics.

Practical tests

1. To determine by palpation the degree of thyroid enlargement.
2. To identify the risk factors for diseases of thyroid gland, possible etiological factors; the initial signs of the disease; to assess the adequacy of diagnostic measures; to establish pharmacological anamnesis; to determine the main stages of the disease.
3. To substantiate the diagnosis of thyrotoxicosis.
4. To determine the nature of complications of thyrotoxicosis.
5. To determine the presence and nature of ocular symptoms.
6. To assess the results of clinical laboratory and instrumental examinations.
7. To determine the severity of thyrotoxicosis.
8. To make a differential diagnosis of goiter.

Protocol of the clinical examination of the patient

Name, surname of the patient_____
Age_____ **Profession**_____

Complaints of the patient_____
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_____
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_____

Anamnesis morbi
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_____

Last exacerbation_____
_____
_____

Anamnesis morbi
_____
_____
_____
_____

Results of the physical examination:

Preliminary diagnosis:

Plan of investigation:

Results of the additional methods of investigations:

Rationale of the clinical diagnosis:

Clinical diagnosis:
Main disease

Accompanying disease

Complications_____
_____
_____
_____

Treatment:
1. Regime_____
2. Diet_____
3._____
4._____
5…….

Literature:

1. Balabolkin M.I. Endocrinology. -M.: Universe Publishing, 1998. -352 p.
2. Balabolkin M.I., Klebanov, E.M., Kreminskaya V.M. Differential diagnosis and treatment of endocrine diseases. Leadership. - Moscow: Medicine. 2002. – 752 p.
3. Braverman E.M. Thyroid Diseases. Trans. from English.. - M.: Medicine, 2000. - 417 p.
4. Dedov I.I., Melnichenko G.A., Andreeva E.N., Arapova S.D. Rational pharmacotherapy of diseases of endocrine system and metabolic disorders: a guide for physician. / under the general editorship by I.I. Dedov. G.A. Melnichenko. - M.: Litera, 2006. – 1080 p.
5. Dedov I.I., Balabolkin M.I. Marova E.I., Diseases of the endocrine system: a guide for physician./ under the general editorship by I.I. Dedov. - M.: Medicine, 2000. – 568 p.
6. Clinical Endocrinology. Guide for physicians. 3rd ed. corr. and add. under the general editorship by N.T. Starkova. - St. Petersburg: Medicine, 2002 - 576 p.
7. U.V. Polyachenko, V.G. Perederiy, O.P. Volosovets, V.F. Moskalenko, etc. Medical education in the world and in Ukraine. Textbook. - K.: Book plus. 2005.-384 p.
8. Chernobrov A.D. and other. Standards of diagnosis and treatment of endocrine diseases. / under the general editorship by M.D. Tronko. - K.: Health of Ukraine. 2005. – 312 p.
9. Tronko M.D., Bodnar P. M., Komisarenko U.I. The history of endocrinology in the Ukraine. - K.: Health, 2004. – 68 p.
10. Epstein, E.V., Matyaschuk S.I. Ultrasound of thyroid gland. Atlas / guide. - Kiev: K V I C, 2004. – 382 p.
11. Dwarfism: Medical and Psychosocial Aspects of Profound Short Stature. Betti M, Aelson. 2005.-368 p.
12. Endocrinology and metabolism / Ed. by Pinchera. - London: McGraw Hill Int., 2001. -811p.
13. Handbook of Physiology. Section 7: Endocrine system. Volume III: Endocrine regulation of Water and electrolyte balance. / Ed. by J.C. S. Fray. - Oxford University press, 2000.-seven hundred fifty p.
14. Textbook of endocrine physiology / Ed. by J.E. Griffin, S.R. Ojeda. - 4-th ed. -Oxford University press, 2000. - 490 p.
15. Thyroid Disorders. Mario Skodur, Jesse B. Wilder. - Cleveland Clinic Press, 2006. - 224p.

Інформаційні ресурси

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Методична вказівка переглянута і затверджена на засіданні кафедри: _____

З доповненнями (змінами) _____

Завідувач кафедри Л.В. Журавльова