VOLGOGRAD STATE
MEDICAL UNIVERSITY
Department of Pathological Anatomy

Diseases of endocrine glands. Diabetes mellitus. Thyroid gland disease. Endocrine gland tumors. Diseases of others endocrine glands.

Guidelines for students
III year of medical faculty
for practical exercises in pathological anatomy

Volgograd, 2020

Diseases of the liver and biliary system

1. The purpose of the lesson. To study the etiology, pathogenesis, morphological characteristics, complications and outcomes of diseases of the organs of the endocrine system.

2. Requirements for the level of the student in the development of the discipline - pathological anatomy. The student must know:

1. Definition, etiology, classification, pathological anatomy of pituitary diseases.
2. Definition, etiology, classification, pathological anatomy, thyroid disease.
3. Definition, classification, pathological anatomy of diseases of the parathyroid glands.
4. Definition, classification, pathological anatomy of adrenal gland diseases.
5. Definition, etiology, classification, pathological anatomy of diabetes.
6. Definition, classification, pathological anatomy of tumors of the endocrine glands.

3. Theoretical aspects.

Disorders of the Endocrine System

Pancreatic endocrine diseases

Diabetes. This is a chronic disease caused by absolute or relative insulin deficiency, leading to disruption of all types of metabolism, primarily carbohydrate, vascular damage (angiopathy) and pathological changes in various organs and tissues.

Diabetes mellitus is the most common endocrine disease, which, according to WHO, affects more than 100 million people worldwide. The incidence of diabetes (especially in industrialized countries) is constantly growing, annually increasing by 6-10%. In this regard, diabetes mellitus (along with cardiovascular and oncological diseases) was included in the group of diseases that are the most common causes of death and disability of patients.

In accordance with the WHO classification (1999), there are:

1) type 1 diabetes mellitus, manifested by the destruction of β-cells of pancreatic islets with absolute insulin deficiency (autoimmune and idiopathic);

2) type II diabetes mellitus, which is based on changes in β-cells, leading to relative insulin deficiency and insulin resistance;

3) other specific types of diabetes - genetic defects in β-cell function, genetic defects in the action of insulin, unusual forms of immune-mediated diabetes;

4) gestational diabetes mellitus (diabetes of pregnant women).

Diabetes mellitus type I and II. Etiology. Type I diabetes is usually found in children and adolescents (juvenile diabetes), although in some cases it can also develop in adults. The disease is characterized by acute onset and absolute insulin deficiency. As a rule, the disease occurs after a viral infection (infection with the Koksaki VZ and B4 viruses, mumps, cytomegalovirus infection, rubella, retroviral infections, measles, chicken pox, infectious mononucleosis, hepatitis). Viruses damage the cytoplasmic membrane of клеток cells by the type of cross-immunity and in individuals with a genetic predisposition lead, on the one hand, to inflammation of the islets (insulitis), and on the other hand, they activate the mechanisms of apoptosis of β-cells. Genetic dependence is characteristic. So, the identity of identical twins is up to 50%. Predisposition to type I diabetes is combined with the presence of histocompatibility complex genes HLA DR3, DR4, DR3 / DR4 and certain alleles of the HLA DQ locus, as well as B8, B15, Dw3, Dw4, Drw3, Drw4, the presence of which significantly increases (by 2-10 times) the risk of the disease. At the same time, B7 and Drw2 antigens are considered as protective, since they provide a decrease in the incidence of type 1 diabetes.

Predisposing or resolving factors are also important, among which are drugs, chemicals (pentamidine, alloxan, streptozotocin, etc.), as well as food components (cow albumin, which is ingested by artificial feeding, smoked foods containing N-nitroso compounds). These substances can have a direct toxic effect on β-cells, triggering or contributing to the launch of immune

responses to cell components, increase the sensitivity of cells to damage and the body's need for insulin, while reducing the production of the latter in the pancreas.

Type II diabetes mellitus usually develops in adults over 40 years of age (elderly diabetes), and the likelihood of illness increases with age. The role of genetic predisposition is extremely great. Thus, the concordance of monozygotic twins is 90%, and according to some reports, it reaches 100%. The disease is based on inadequate function of β-cells and insulin resistance of tissues. External resolving factors are numerous: obesity, age, physical inactivity, mental and physical trauma, etc. All of them cause compensatory hyperinsulinemia, which develops in response to the increased need for this hormone of insulin-dependent tissues, which include adipose tissue, as well as myocardium, skeletal muscle , lining of the eyes, liver, pancreas. In addition, hyperinsulinemia occurs due to relative insulin resistance, which over time becomes absolute. A constant load on the β-cells of pancreatic islets (Langerhans islets) leads to their decompensation and, consequently, to relative, and in severe cases, absolute insulin deficiency with the development of hyperglycemia.

Pathogenesis. In type I diabetes mellitus, in most patients who died less than 1 year after the onset of the disease, immune insulitis is noted, which is characterized by the presence of inflammatory T-lymphocyte infiltrate (primarily CD8 and CD4), B-lymphocytes, macrophages, and natural killers (NK cells). T lymphocytes interact with antigen molecules of the HLA system located on the surface of β-cells to form antigen-presenting cells. The complex of the latter with T-lymphocytes causes unregulated activation of the cells of the immune system that produce autoantibodies that damage β-cells. Autoreactive T-lymphocytes can be present in the patient’s body for many years, possessing the ability to destroy target cells even in transplants of the gland transplanted for the treatment of diabetes. In 1-2 years after the manifestation of type I diabetes mellitus, the content of cytoplasmic antibodies to pancreatic islet antigens is significantly reduced and is observed only in individual patients.

Activated macrophages and T-lymphocytes damage β-cells either by direct contact with them, or indirectly through free radicals and cytokines (IL-β, TNF-a, y-INF). Nitric oxide is also involved in the destruction of β-cells, which is formed by macrophages or synthesized directly in the cells of the islets themselves. The expression of Fas receptors in patients with type I diabetes mellitus and in individuals with a high predisposition to it is significantly reduced, which leads to a slowdown in the removal of activated T-lymphocytes and, therefore, stimulates the activity of autoimmune processes, especially since the ratio of helpers and suppressors is disrupted. Insulin leads to a significant decrease in the total number of β-cells, which leads to absolute insulin deficiency. Atrophy of the islets develops in the pancreas and connective tissue grows.

In type II diabetes mellitus, in the blood of overweight people, not only glucose but also free fatty acids that inhibit carbohydrate metabolism are constantly increasing, and therefore obesity (both genetically determined and acquired) is considered a risk factor. In addition, in patients there is a defeat of cellular receptors for insulin, a constant decrease in their concentration and affinity as hyperinsulinemia and body weight increase. The postreceptor mechanism of glucose transport is also affected by the suppression of transporter proteins. Mutations of the insulin receptor gene may also be the cause of insulin resistance.

In patients, the processes of lipolysis, glycogenolysis and gluconeogenesis are enhanced, stimulated by the contrainsular hormone glucagon, the content of which increases. With obesity in fatty tissue and muscle tissue, an increase in the content of the name, inhibitory effect of insulin due to blockade of the β-subunit of the insulin receptor, and a decrease in the synthesis of the protein-transporter GLUT-4 were found. A relative insulin deficiency develops, since the number of cells of pancreatic islets remains within the age norm. However, in the future, the functional ability of β-cells is depleted, which is facilitated by pancreatic lipomatosis, which occurs with general obesity, as well as focal islet amyloidosis, often noted in elderly people. Subsequently (in 15–20% of patients), atrophy of the islets develops, primarily β-cells, which leads to absolute insulin deficiency, characteristic of severe type II diabetes mellitus.

Diabetes mellitus MODY-muna. Significant distribution (2-5% of all cases of diabetes) is

associated with monogenic genetic defects of β-cell function, the so-called MODY-type disease (maturityonset diabetes of the young - late diabetes of the young), characterized by an autosomal dominant type of inheritance and manifestation in age 13-20 years old.

The clinical course of MODY-type diabetes is the same as type II diabetes mellitus, in connection with which it was previously considered as a special variant of the latter, developing in children and adolescents. However, the pathogenetic features of MODY-type diabetes served as the basis for its isolation in a separate group.

Mitochondrial diabetes. It is caused by a point mutation in mitochondrial DNA and transport RNA, leading to the development of slowly progressive type I or II diabetes mellitus in combination with frequent hearing loss and MELAS syndrome (mitochondrial myopathy, lactic acidosis, encephalopathy and stroke-like attacks). It is rare, transmitted through the maternal line.

Gestational diabetes mellitus (pregnancy diabetes). The disease occurs in about 2-4% of pregnant women and, as a rule, passes after childbirth. Despite the usually mild course, the disease leads to frequent pregnancy complications: polyhydramnios, arterial hypertension (due to kidney damage), early and late gestosis, secondary infection (especially the urinary tract), termination of pregnancy, premature birth, various injuries due to the birth of a large fetus. In children, as a result of relative insulin deficiency, the risk of various malformations (diabetic embryopathy) increases by more than 2 times. As a rule, a de-adaptation syndrome develops, in connection with which the postnatal mortality of newborns is quite high. These disorders are caused by a constant lack of glucose in the tissues, energy deficiency of the fetus, chronic hypoxia arising from the defeat of the placental histohematological barrier due to metabolic disorders and diabetic microangiopathy. In addition, hyperglycemia in pregnant women leads to the formation of diabetic macrosomia - a characteristic increase in the fetus for the disease.

Other types of diabetes. Insulin deficiency and hyperglycemia can develop as a manifestation of a disease that leads to secondary lesions of the islet apparatus of the pancreas. This is most often noted in chronic pancreatitis, idiopathic hemochromatosis ("bronze diabetes"), benign and malignant tumors of the pancreas, various dishormonal pathologies (Itsenko-Cushing's disease and syndrome, acromegaly, carcinoid syndrome, thyrotoxicosis, etc.), prolonged use and drugs, for example glucocorticoids (steroid diabetes), analgesics, heroin, chloroform, ether, etc., leading to atrophy of β-cells. In some cases, diabetes is a manifestation of various genetic diseases. So, hyperglycemic syndrome is noted with chromosomal diseases of Down, Klinefelter, Shereshevsky-Turner, Moriak, as well as with gene pathologies such as Louis-Bar syndrome (diabetes, cerebellar ataxia, conjunctival telangiectasia, humoral immunodeficiency, primarily IgA, delayed, Werner (diabetes, hypogonadism, alopecia, bilateral cataract, decreased intelligence, stunting), Ore (diabetes, hypogonadism, gigantism, congenital dementia, epilepsy), Truell-Junet (diabetes, acromegaly, colloid goiter, diffuse hyperost cranial vault), etc.

Changes in organs and tissues in diabetes. An increase in blood glucose initially has a compensatory character, since under conditions of insulinopenia, which leads to a decrease in the permeability of the histohematic (primarily blood-brain-brain) barrier, the required amount of glucose enters the tissues only at an increased sugar concentration. However, with an increase in glucose levels of 3.75 times, total glucose utilization increases only 2.25 times, and recycling only 2 times. In addition, prolonged hyperglycemia in itself contributes to the development of insulin resistance and damages cells (the phenomenon of glucose toxicity), leads to a decrease in the number of glucose transporter proteins, and reduces the secretory activity of β-cells. All this reduces the utilization of carbohydrates by tissues and causes a violation of other types of metabolism. Energy starvation of tissues develops, which is also facilitated by the fact that the use of lipids and proteins for the energy replacement of glucose increases oxygen consumption by 15-20%.

Hyperglycemia leads to the development of hyperlipidemia, stimulates the processes of lipid peroxidation, causing non-enzymatic glycosylation. An intracellular polyol glucose oxidation pathway is activated that does not require the participation of insulin. In this case, non-phosphorylated glucose under the action of aldose reductase is converted to sorbitol cyclic alcohol,

part of which forms fructose with the participation of sorbitol dehydrogenase. Under normal conditions, up to 1% glucose is exchanged along the sorbitol path, while under conditions of insulin deficiency this amount increases to 10%. Accumulation in the eyes, neurolemma (Schwann membranes) of peripheral nerves, kidneys and blood vessels of sorbitol and fructose, which have hydrophilic properties, causes severe swelling and tissue damage, thickening of the basement membranes of blood vessels, changes the adhesive properties of blood cells.

The reaction of sorbitol formation, unlike phosphorylation, proceeds with the breakdown of ATP in cells, which exacerbates energy deficiency. Significant metabolic disturbances are accompanied by hemodynamic disturbances of the type of DIC.

As a result, with diabetes mellitus, a progressive lesion of various organs and tissues occurs. Patients develop severe changes not only in the pancreas, but also in the liver, blood vessels, retina, kidneys, and nervous system (diabetic angiopathies, retinopathy, nephropathy, neuropathy).

Pancreas. In patients who have died from diabetes mellitus, this organ is usually reduced, with type I diabetes mellitus it has a dense consistency due to fibrosis, combined with pronounced atrophic changes in the lobules. Microscopically, rare small pancreatic islets with a reduced number of degranulated β-cells are observed.

In type II diabetes mellitus, the pancreas can be increased in size due to lipomatosis, but small lobules are visible in the section. Microscopic examination of β-cells is small, their number may decrease (especially with a long course of the disease). Pronounced layers of adipose tissue dividing lobules are detected. Sometimes deposits of amylin, a focal amyloid, are found.

Liver. The organ has the appearance of fatty hepatosis (up to the development of "goose liver").

To a large extent, the current course of both types of diabetes is caused by diabetic angiopathies, so diabetes is even called an exchange-vascular disease. It is due to vascular damage to diabetes! ranks first among the causes of blindness, in patients 17 times more likely to develop kidney damage, 2-3 times more often - myocardial infarction and strokes, 5 times more often - gangrene of the lower extremities than in people of the same age and gender with normoglycemic indices.

Diabetic macroangiopathy. It is characterized by damage to arteries of medium and large caliber and is found, as a rule, in people of mature and old age, and therefore is most pronounced in type II diabetes mellitus. Its manifestations are atherosclerosis, which is usually more pronounced and more common than in non-diabetic patients (diabetes mellitus is a risk factor for atherosclerosis), and Menkeberg's mediacalcinosis and diffuse intimal fibrosis are much less common. As a result of the defeat of large arteries, numerous vascular necrosis and gangrene of the lower extremities arise.

Diabetic microangiopathy is generalized in nature, develops at any age, while there is a direct dependence on the duration of diabetes. Arterioles and capillaries of various organs and tissues are affected (especially often the kidneys, retina, skin, skeletal muscle). Along with nonspecific changes (plasma impregnation, vascular wall hyalinosis, dystrophy, cell proliferation and atrophy), a thickening of the basement membranes of the endothelial lining due to the accumulation of PAS-positive substances (primarily type IV collagen) is observed. The pathogenesis of diabetic angiopathy involves metabolic, hormonal, hemorheological, immunological, genetic and other factors leading to impaired vascular permeability and damage to the vascular wall.

Diabetic retinopathy. It affects almost 100% of patients with diabetes for more than 15 years. In addition to the morphological changes characteristic of the diabetic microangiopathy underlying this eye pathology, with retinopathy, along with hyalinosis and obliteration in the retinal capillaries and venules, microaneurysms develop, and perivascular - edema, hemorrhages, dystrophic and atrophic changes in the optic nerve. The following forms of diabetic retinopathy are distinguished:

- non-proliferative, or simple, diabetic retinopathy, characterized by individual microaneurysms, pinpoint hemorrhages, as a rule, only in the paralosa. Vision is not changed or

slightly reduced;

- proliferative retinopathy, in which neoplasm of the capillaries, extensive hemorrhages and sclerosis of the retina and optic nerve papilla occur, which can lead to the development of glaucoma, retinal detachment and loss of vision.

In addition, in patients with diabetes, sudden vitreous hemorrhages are possible with the development of blindness in one eye.

Diabetic nephropathy. In patients with diabetes mellitus in the kidneys, diabetic intracapillary glomerulosclerosis occurs, leading to severe nephrotic syndrome, named after the authors who first described it, Kimmelstil-Wilson syndrome. The kidneys are symmetrically reduced in size, with a fine-grained surface, a dense texture due to the growth of connective tissue (diabetically wrinkled kidneys). Microscopic examination distinguishes the following varieties of glomerular changes characteristic of the disease:

- nodular (nodular) glomerulosclerosis, observed in 15–35% of patients and which is specific for diabetes mellitus. It is characterized by the proliferation of mesangial cells and their production of a membrane-like substance with the formation of homogeneous eosinophilic and PAS-positive rounded formations;

- diffuse glomerulosclerosis, which most often develops in patients and manifests itself in diffuse thickening of the basement membranes of capillaries, is accompanied by proliferation of glomerular mesangium;

- mixed diabetic glomerulosclerosis.

An electron microscopic study in the glomeruli of the kidneys shows an increase in mesangium and proliferation of mesangial cells (intercapillary glomerulosclerosis), accompanied by diffuse thickening of the basement membranes of the capillaries.

In addition, patients exhibit hyalinosis not only bringing, but (unlike hypertension) and efferent glomerular arterioles along with hyalinization and sclerosis of larger arterial vessels. With decompensation of diabetes mellitus, the development of exudative manifestations of diabetic glomerulopathy in the form of “fibrin caps” and “capsule drops” arising due to a significant increase in the permeability of the glomerular filter is possible. In the tubules, protein (up to vacuole) and fatty (in the presence of nephrotic syndrome) epithelial dystrophy are noted. In the proximal tubules, glycogenous epithelial infiltration is detected due to the polymerization of glucose reabsorbed from primary urine.

In addition to these complications, resulting in the development of chronic renal failure in diabetes mellitus, non-specific changes often occur in the kidneys - acute and chronic pyelonephritis, sometimes leading to papillonecrosis (necrotic papillitis, medullary necrosis of the kidneys) and acute renal failure. Violation of the passage of urine up to the development of hydronephrosis is facilitated by atony of the bladder due to diabetic neuropathy.

Diabetic neuropathy. Its frequency correlates with the duration and severity of diabetes, reaching 90% or more with a disease duration of over 25 years. Symmetric damage to the sensitivity of peripheral nerves (paresthesia, violation of contact, temperature, vibration, pain sensitivity), most pronounced in the distal extremities, especially the lower ones, is most often noted. Motor nerves are somewhat less likely to be affected. In this regard, painless forms of myocardial infarction, disorders of the motility of internal organs are often noted in patients. In the pathogenesis of neuropathy, in addition to metabolic factors, diabetic microangiopathy of neural vessels (vasa nervorum), which aggravates metabolic disorders and hypoxia, is of great importance. Segmental demyelination, edema and dystrophy of the axial cylinders develop, leading to a decrease in the speed of impulses along nerve fibers.

In patients with diabetes, vitiligo, xanthomatosis, and lipoid necrosis of the skin often occur. Significantly increases the risk of gallstone disease due to metabolic disorders and atony of the gallbladder. Due to secondary immunodeficiency, purulent complications (pyoderma, furunculosis, bronchopneumonia, sepsis) often join, pyelonephritis and tuberculosis are possible.

Modern treatment has led to a significant increase in the life expectancy of patients with diabetes. In this regard, death in diabetes is associated with complications of the disease

(myocardial infarction, cerebrovascular accident, gangrene of the lower extremities, renal failure, secondary infection). From hyperglycemic coma, no more than 2-5% of patients are currently dying.

FEEDBACK: General _ Endocrine Gland Hormone Release Response Target Organ + Hypothalamus GHRH CRH TRH GnRH ACTH TSH LH/FSH + Anterior Pituitary GH + Target Organ All cells + Adrenal glands + Thyroid + + Ovaries Testes ↑Growth ↑Cortisol All cells ↑Estrogen ↑Protein ↑Progesterone synthesis ↑Testosterone ↑Metabolism Hypopituitarism ↓ Secretion of anterior pituitary hormones

Causes Infection or inflammation Autoimmune disease Tumor (adenoma) Hypoxic necrosis of pituitary or hypothalamus Hyperpituitarism ↑ Secretion of anterior pituitary hormones Causes: Malfunction of pituitary or hypothalamus Primary adenoma of hormone-secreting cell tumor: whether its under or over functional organ depends on the cells comprising the cell mass. Lack of feedback from target gland no neg feedback to reg release (lack of inhibitory influences) -> rev and release things uncontrollably

Thyroid Diseases secretes TH (metabolic activator for most cells - incr things) Anatomy of the Thyroid Gland paired four dots - parathyroid glands (Calcium reg) PTH profile hard to regulate (assoc with thyroid gland removal - problem for parathyroid) Hypothyroidism Causes of Hypothyroidism Malfunction of thyroid gland Low TH with high TSH & TRH Lack of negative feedback by TH Malfunction of pituitary gland Low TSH causes low levels of TH TRH from hypothalamus is high Malfunction of hypothalamus Low TH, TSH, TRH trying to get thyroid to release shit b/c low TH -> incr TSH and TRH trying to stimulate the pituitary gland! Diseases of Hypothyroidism Hashimoto's disease Autoimmune destruction of the thryroid Endemic Goiter Dietary deficiency of iodide

Thyroid carcinoma Diseases of Hypothyroidism Hashimoto's disease Endemic Goiter Dietary deficiency of iodide Thyroid carcinoma adenoma (below) hypertrophy - low levels of TH, not enough being produced hypothalamus doesnt understand its due to lack of Iodine, so it stimulates the entire system and in response to stimulus it tries to accommodate by becoming bigger -> aka make more except it cant b/c no iodine Normal Thyroiditis non functional, cant build and secrete TH Clinical Manifestations Sluggishness, slow thinking, clumsy ↓ HR, enlarged heart & ↓ CO Bogginess & edema of skin Intolerance to cold ↓ MR, ↓ caloric requirement, ↓ appetite Constipation Dry, flaky skin & thin hair not moving fluid around as efficienctly, less removal of wastes? if person undergoes someting that needs incr metabolism, body incapable of rising to the ocasion to facilitate that. Treatment Synthetic thyroxine giving up thyroid b/c HRT is successful (thyrozine pill in morning, thats it) Iodide replacement for goiter not usually a first world problem (Iodine supplementation in foods) dlvping country -> can arise as a problem radioactive iodine - ppl exposed -> it accumulates in thyroid (destroys it) - had to have thyroid taken out and got thyroxine therapy Special Considerations Pediatric Congenital hypothyroidism (cretinism) Low TH with high TSH & TRH type of dwarfism the problem is in the gland (low TH can't fed bk, not making any, so being stimulated!) Geriatric Myxedema usually seen in elderly cant mount stress response - lead to fatal cascade of events Hyperthyroidism Causes of Hyperthyroidism Dysfunction of thyroid gland ↑ TH, ↓ TSH & TRH Negative feedback on release by TH Dysfunction of pituitary ↑ TH & TSH, ↓ TRH Negative feedback from TH & TSH Dysfunction of hypothalamus ↑ TH, TSH, TRH neg fbk of TH -> decr TSH and TRH Diseases of Hyperthyroidism Grave's disease Autoimmune disorder Autoantibodies mimic action of TSH ↑ TH, ↓ TSH & TRH stimulating the thyroid to make more TH ! Rc's can't distinguish b/w endogenous TH and autoantibodies, so as if being perpetually stimulated due to neg fdbk Nodular goiter ↑ size of thyroid due to ↑ demand for TH Clinical Manifestations ↑ HR ↑ Muscle tone, tremors, irritability ↑ BMR, ↑ heat production Intolerance to heat, excess sweating Weight loss, ↑ hunger Staring appearance Exophthalmos Goiter Clinical Manifestations ↑ HR ↑ Muscle tone, tremors, irritability ↑ BMR, ↑ heat production Intolerance to heat, excess sweating Weight loss, ↑ hunger Staring appearance Exophthalmos Goiter eye bulging out reversed after treatment

Diagnostic Tools History & Physical exam Blood tests measuring TH (T3 & T4) TSH, TRH ↓ Serum lipids ↓ Sensitivity to insulin (hyperglycemia) burning all the fuel in the body Complications Dysrhythmias Thyrotoxic crisis (thyroid storm) Tachycardia Agitation Tremors Hyperthermia Death chronic elevation in HR elevated body temp! snow ball into irreversible fatal problem - brain damage? Treatment Drugs that block TH production Beta blockers takes heart problems out of it (reg heart, decr HR) Radioactive iodine pretty much exclusively goes to thyroid -> can destroy some tissue to reduce size/production of TH Partial or total thyroidectomy enlarged goiter thyroid being removed

Adrenal Insufficiency The Adrenal Glands secrete mineralcorticoids and glucocorticoids & norepi and epi dysfunction of specific region can cause hyper/hypo production of specific hormone which specific zone is affected!?!??! Adrenal Gland Kidney Adrenal Artery Adrenal Vein Abdominal Aorta Inferior Vena Cava Causes of Adrenal Insufficiency Dysfunction of adrenal gland (primary) ↓ glucocorticoids (especially cortisol) ↑ ACTH & CRH Adrenal androgens & aldosterone normal,↓ or ↑ Dysfunction of pituitary (secondary) ↓ glucocorticoids, ACTH, ↑ CRH ↓ Adrenal androgens If zero ACTH, ↓ aldosterone Dysfunction of hypothalamus (secondary) ↓ glucocorticoids, ACTH, CRH Diseases of Adrenal Insufficiency Addison's disease ↓ glucocorticoids &↑ ACTH & CRH Aldosterone deficiency primary dysfunction of the adrenal glands b/c other areas of adrenal glands are affected Hyponatremia, b/c of incr output dehydration & ↓ BP Secondary adrenal insufficiency Cortisol used therapeutically Inhibits pituitary release of ACTH as a HRT to maintain body's ability to maintain the stress response reg both acute and chronic stress Primary Adrenal Insufficiency Disorder of the adrenal glands themselves usually autoimmune Most cases of Addison's disease are caused by the gradual destruction of the adrenal cortex, by the body's own immune system. About 70 percent of reported cases of Addison's disease are due to autoimmune disorders, in which the immune system makes antibodies that attack the body's own tissues or organs and slowly destroy them. Clinical signs don't show up until approximately 90% of the adrenal tissue is destroyed. Often both glucocorticoid and mineralcorticoid hormones are lacking disadvantage of effective therapy due to lack of residual tissue left Primary Adrenal Insufficiency Hypotrophic Adrenals small, reduction in tissue mass, loss most functional cellular mass within the glands - cant support endocrine metab fn Normal Adrenals Secondary Adrenal Insufficiency in this form, there is a lack of ACTH, which causes a drop in the adrenal glands' production of cortisol but not aldosterone. b/c its secretion is not linked to/responsive to ACTH (ACTH doesnt reg aldosterone) hydration profile and circ pls pr- values are similar b/c no changes in adolsterone. so if see reduced lvls of cortisol, with low ACTH and normal aldosterone = pituitary problem May occur after treatment with glucocorticoids Stressor Hypothalamus CRH (-) inhibit releasing hormone, neg fdbk to reduce cortisol response/stress response hyperactivation or incr stress -> lots of inflammation where need to mount stress response/cortisol response. if endogenous cortisol is insufficient to mount response, get on steroids and exogenous cortisol Tx Pituitary ACTH Glucocorticoids: Rheumatoid Arthritis Asthma Colitis Adrenal Cortisol Stressor (-) Hypothalamus CRH Pituitary ACTH Glucocorticoids: Rheumatoid Arthritis Asthma Colitis Adrenal (-) Cortisol Stressor (-) Hypothalamus CRH (-) X Pituitary ACTH Glucocorticoids: Rheumatoid Arthritis Asthma Colitis Adrenal Cortisol Stressor (-) inhibit endogenous cortisol, reduces stress response Hypothalamus CRH (-) X (-) Glucocorticoids: Rheumatoid Arthritis Asthma Colitis (prevalent in chronic stress) acute - want cortisol chronic - dont want cortisol lvls to be high -> cause long term problems Pituitary ACTH Adrenal Cortisol Stressor (-) Hypothalamus CRH (-) X (-) Glucocorticoids: Rheumatoid Arthritis Asthma Colitis Pituitary ACTH X Adrenal Cortisol Stressor (-) Hypothalamus CRH (-) X (-) Glucocorticoids: Rheumatoid Arthritis Asthma Colitis Pituitary ACTH Cortisol X Adrenal does not rebound quickly after chronically suppressing cortisol, can mess up acute cortisol response/stress repsonse where u need acute high cortisol release Clinical Manifestations Depression Fatigue Anorexia, vomiting, diarrhea, nausea Hyperpigmentation if ACTH is high Sparse body hair in women Lack of response to stress can be life threatning bonzing of skin through activation of melanin Complications Adrenal crisis after physical or emotional stress Life threatening Volume

depletion Hypotension Vascular collapse can be fatal Treatment Cortisol replacement Aldosterone can be administered, for short period of time, to have stress response Glucocorticoids during periods of stress M&M high without treatment morbidity and mortality high w/o Tx Glucocorticoid Excess Causes of Glucocorticoid Excess Adrenal tumor Adenoma of the pituitary Nonpituitary tumor (ectopic source) Diseases of Glucocorticoid Excess Cushing's syndrome Any condition with ↑ glucocorticoids Cushing's disease ↑ glucocorticoids due to malfunction of anterior pituitary Excess ACTH specific due to ant pit dysfunction! Clinical Manifestations Altered fat metabolism & Muscle weakness Hypertension Weight gain Reversible form of DM ↓ Immune & inflammatory reactions mental dysfn assoc with high cortisol lvls Extreme mood changes, psychosis, suicide Masculinization of women & children Bronzing of skin Patient with Cushings Patient with Cushings Same patient after Treatment " Buffalo Hump " " Moon Face " Central Obesity if on high glucocorticoids therapy, can get some of these symptoms. Complications M&M high without treatment 50% die within 5 years Cause of death Suicide Overwhelming infections CAD from severe hypertension Treatment Surgery for tumors Radiation therapy Drugs that block steroid synthesis Discontinue corticosteroid therapy Growth Hormone Deficiency Cause of GH Deficiency Pituitary adenoma Hypoxic necrosis Inflammation of pituitary If at hypothalamic level: Malnutrition Sleep deprivation Prolonged exercise or emotional stress Low estrogen levels Diseases of GH Deficiency Dwarfism Reduction of growth potential Alteration in metabolic functioning in adults Clinical Manifestations Children Short stature ↓ Muscle mass & ↑ SQ fat stores Bright mentally Delayed onset of puberty Adult-onset Alterations in physical & mental well-being Treatment SQ recombinant GH Pediatric Consideration May be normal genetic predisposition Accompany genetic abnormalities Illnesses treated with chronic corticosteroids Growth Hormone Excess Causes of GH Excess Usually caused by GH-secreting tumor of anterior pituitary Growth Hormone Insulin-Like Growth Factor -I Increase in bone/soft tissue growth • increase in bone length before epi. plate closure • increase in bone width after epiphyseal plate closure Diseases of GH Excess Gigantism Excess longitudinal growth of bones Result of GH excess before puberty Acromegaly Connective tissue proliferation in adults Growth of cartilage in hands & feet,nose, jaw, chin & facial bones Proliferation of connective tissue in internal organs Clinical Manifestations Gigantism Tall stature Acromegaly Thickening of Fingers Jaw Forehead Hands & feet Complications Acromegaly Cardiac hypertrophy Hypertension Diabetes Mellitus ↑ Glucose & ↓ cellular insulin sensitivity Treatment Surgical excision of GH-secreting tumor Radiation Bromocriptine Transsphenoidal Surgery Polycystic Ovary Syndrome What is PCOS ?? Complex, heterogeneous endocrine disorder Key Features: biochemical and clinical hyperandrogenism reproductive morbidity: - menstrual dysfunction - infertility Prevalence: Approximately 4-8% of women of reproductive age exhibit polycystic ovaries. Polycystic Ovarian Syndrome (PCOS) Overview Ø PCOS is a complex endocrine disorder affecting women of childbearing age characterized by increased androgen production and ovulatory dysfunction Ø PCOS is the leading cause of anovulatory infertility and hirsutism Ø Women with PCOS have an increased risk of miscarriage, insulin resistance, hyperlipidemia, type 2 diabetes, cardiovascular disease, and endometrial cancer. Clinical Features of PCOS Obesity 50% of patients; associated with impaired glucose tolerance and insulin receptors. Amenorrhea/ Oligomenorrhea May result from high testosterone plasma levels Infertility 75% of cases; ovulatory failure Hirsutism; Acne Increased androgen activity Pathogenesis of PCOS FSH LH Pathogenesis of PCOS FSH LH Follicular Maturation Stromal and thecal Androgen production Ovarian overgrowth Anovulation Pathogenesis of PCOS FSH LH Follicular Maturation Excess Androgens Stromal and thecal Androgen production Ovarian overgrowth Adrenal Androgens Anovulation Pathogenesis of PCOS FSH LH Follicular Maturation Excess Androgens Stromal and thecal Androgen production Ovarian overgrowth Adrenal Androgens Anovulation Pathogenesis of PCOS FSH LH Follicular Maturation Estrone Production Excess Androgens Stromal and thecal Androgen production Ovarian overgrowth Adrenal Androgens Anovulation Pathogenesis of PCOS FSH LH Excess Estrone Follicular Maturation Estrone Production Excess Androgens Stromal and thecal Androgen production Ovarian overgrowth Adrenal Androgens Anovulation Pathogenesis of PCOS FSH LH

Excess Estrone Follicular Maturation Estrone Production Excess Androgens Stromal and thecal Androgen production Ovarian overgrowth Adrenal Androgens Anovulation FSH FSH LH FSH Normal Ovulatory Cycle LH FSH FSH FSH LH FSH Normal Ovulatory Cycle LH FSH LH FSH PCOS Ovary LH Serious Health Consequences of PCOS Women with PCOS have 7x greater risk of heart disease and heart attack Myocardial infarction Unstable angina Congestive heart failure Hypertension 40% of PCOS patients develop Type II diabetes by the age of 40. Uterine malignancies Management of patients with PCOS Infertility Increase rate of ovulation through the controlled of insulin reduction by diet Suppress elevated LH levels Hyperandrogenism Reduce insulin drive Anti androgen medication Long term complications Correction of metabolic and cardiac risk factors Diseases Associated with the Parathyroid Glands Hyperparathyroidism Hypoparathyroidism Location of the Parathyroid Glands Effects of Parathyroid Hormone Release of PTH: Increase the concentration of plasma Ca++ Three main ways: It activates osteoclasts - these resorb (degrade) bone, thus releasing some of the calcium stored in bone in times of hypocalcaemia (low blood calcium) for other functions. 2. It promotes the reabsorption of calcium from the urine - PTH stimulates cells of the kidney to reabsorb calcium (and excrete phosphate) from the urine, reducing the loss of calcium in hypocalcaemia. 3. It promotes the activation of vitamin D precursors in the kidney - active vitamin D (1.25-cholecalciferol) promotes the uptake of calcium from digested food in the small intestine. This raises calcium levels when they are low. Hyperparathyroidism PTH Plasma Calcium (hypercalcemia) Hypercalcemia Mineral deposits: Kidney stones Cortical bone density: Arthralgia Altered cardiac function: hypertension, blockade Hyperparathyroidism Causes 3 Main Causes: 1. Benign adenoma of parathyroids: 80% 2. Hyperplasia of parathyroids: 10% 3. Parathyroid carcinoma: 1% Hypoparathyroidism Causes: Damage to the parathyroids incurred during operations on abnormal parathyroid tissue or the thyroid gland. Very rarely autoimmune processes where the glands are attacked by the body's own immune system. Hypoparathyroidism Results in Hypocalcemia Neuro: Paresthesias, muscle spasm, tetany, SEIZURE Visual: Cataracts, optic neuritis Pulmonary: Bronchospasm CV: Prolonged QT, CHF, Hypotension GI: Dysphagia, abdominal pain, biliary colic GU: Preterm labor.

4. Lesson plan

1) To study the following macropreparations, to describe them according to the scheme for the description of macropreparations.

Macropreparations.

1. To study aortic atherosclerosis according to a macroscopic picture. Describe the aortic atherosclerosis macropreparation. Pay attention to the integrity, thickness and color of intimacy, localization, quantity, texture, shape and color of fibrous plaques, fat spots and streaks, complicated lesions.

2. To study the gangrene of the foot on a macroscopic picture. Describe the gangrene foot macropreparation. Pay attention to the volume and area, color, texture and integrity of the affected tissue, the clarity of the demarcation line.

3. To study secondarily shriveled kidneys in diabetes mellitus (diabetic glomerulosclerosis) according to a macroscopic picture. Describe macropreparation "Secondarily shriveled kidney (diabetic glomerulosclerosis)." Pay attention to the size, texture, surface, thickness of the cortical and medulla in the section.

4. To study the nodular colloid goiter on a macroscopic picture. Describe the nodal colloid goiter macropreparation. Pay attention to the size, surface, consistency of the gland, the safety of the capsule, structural features and color in the section.

5. Examine the adrenal gland by a macroscopic picture. Describe the adrenal gland adenomas. Indicate to the localization, size, color, shape, type of tissue in the section and the boundaries of the tumor.

2) Examine the following micropreparations, sketch them, indicate and mark the pathological changes with arrows, using the atlas of micropreparations.

Micropreparations.

1. To study the pancreas in diabetes mellitus by a microscopic picture. Describe the micropreparation "Pancreas in diabetes mellitus" (hematoxylin and eosin staining). Pay attention to the size and number of pancreatic islets (islets of Langerhans), the state of β-cells and stroma of the gland; cellular composition, localization and severity of infiltrate.

2. To study the vascular hyalinosis of the skin in diabetes mellitus by a microscopic picture. Describe the micropreparation “Hyalinosis of skin vessels in diabetes mellitus” (hematoxylin and eosin staining, PAS reaction). Pay attention to the vessels of the dermis (the thickness and structure of their walls, the width of the lumen, the number of endothelial cells and pericytes, the thickness of the basement membranes); the state of the epidermis and the internal matrix of the dermis. Mark the color of the vessel wall during the PAS reaction.

3. To study diabetic glomerulosclerosis according to the microscopic picture. Describe the micropreparation "Diabetic glomerulosclerosis" (hematoxylin and eosin staining, PAS reaction). Pay attention to the size of the glomeruli; the prevalence, structure, and color of changes in the mesangium and basement membranes of capillaries; walls and lumens of glomerular arterioles. Mark the color of focal deposits in the mesangium, the basement membranes of the capillaries and the walls of the glomerular arterioles during the PAS reaction.

4. Examine the nodular colloid goiter on a microscopic picture. Describe micropreparation "Nodal colloid goiter" (hematoxylin and eosin staining, PAS reaction). Pay attention to the size, shape of the follicles and the epithelium lining them, the state of the colloid. Note the color and color intensity of the colloid during the PAS reaction.

5. To study diffuse toxic goiter by microscopic picture. Describe micropreparation "Diffuse toxic goiter" (hematoxylin and eosin staining). Pay attention to the size and shape of the follicles and the epithelium lining them, the state of the colloid; cellular composition, localization and severity of infiltrate.

6. To study papillary thyroid cancer according to the microscopic picture. Describe micropreparation "Papillary thyroid cancer" (stained with hematoxylin and eosin). Pay attention to the size and structure of the papillae of the tumor (size, shape and mitotic activity of parenchyma cells; size, shape, inclusion, vacuolization of nuclei; vacuolization and amount of stroma); features of the location of the tumor in relation to the preserved tissues of the gland; localization and cellular composition of the infiltrate; secondary changes in tumor tissue**.**

3) Examine the following electron diffraction patterns using an atlas of micropreparations.

Electron micrograph.

1.Diabetic glomerulosclerosis. Describe the electron diffraction pattern "Diabetic glomerulosclerosis". Pay attention to the localization and structure of the newly formed substance, the width of the mesangium, the thickness of the basement membranes of the capillaries, the state of the endothelium and podocytes.

4) Solve the following situational problems (case study) using the tutorial.

Situational tasks (case study).

Case 1

Patient K., 35 years old, was admitted to the hospital with complaints of increased irritability, periods of palpitations, increased tearfulness, over the past few months, noted swelling in the region of the front surface of the neck. The patient underwent an operation to remove the thyroid gland. Operational material: 1) macroscopic picture: thyroid gland with multiple nodes with a diameter of 0.5-2 cm; 2) microscopic picture: the gland tissue is represented by follicles of various sizes, the follicular epithelium is high, proliferation of the follicular epithelium is noted.

Questions for Case 1:

1) What pathology of the thyroid gland did the patient have?

2) What is the type of disease described?

3) What is the reason for the described clinical symptoms?

Case 2

Patient S., 32 years old, died of uremia, history of a periodic increase in blood sugar to 10-12 mmol / l for 16 years, was treated with insulin. At autopsy: a sharp smell of urine comes from all organs, on the epicardium - multiple films and filaments of fibrin, on the mucous membrane of the esophagus and stomach - multiple erosions and acute ulcers, in the lungs - the phenomena of edema and pneumonia (in the lumens of the alveoli - edematous fluid, fibrin, neutrophils), in the brain - the phenomena of edema; in the pancreas - sclerosis, lipomatosis and atrophy of the gland tissue (microscopically - sclerosis of a significant number of islets of Langerhans, preserved hypertrophy of the islets, the phenomena of sclerosis and lipomatosis); the kidneys are sharply reduced, wrinkled, in sections of gray-pink color, the cortical substance is sharply thinned, the boundary between the layers is fuzzy (microscopically - glomerulosclerosis).

Questions for Case 2:

1) What pancreatic disease has occurred?

2) What pathological process has occurred in the kidneys?

3) What is the relationship of the pathological process in the kidneys with pancreatic disease?

4) What complication developed?

Case 3

Patient M., 35 years old, previously living in central Russia, moved to Altai Territory. A few years later, the thyroid gland increased. There were no other clinical symptoms. He was offered surgical removal of an enlarged part of the thyroid gland. Microscopic examination revealed: large and small follicles filled with a dense, homogeneous colloid.

Questions for Case 3:

1. What kind of goiter arose in this patient?

2. How can it be characterized clinically?

3. Indicate the type of goiter, depending on its microscopic structure.

Case 4

Patient K., 28 years old, over the past 3 years noted an increase in the thyroid gland. On palpation, the iron is dense, somewhat soldered to the surrounding tissues. In the anamnesis - an infection suffered several years ago. The enlarged thyroid gland is removed promptly. Microscopic examination revealed: wide fields of connective tissue, among which are small islands of follicles.

Questions for Case 4:

1. What thyroid disease has occurred?
2. What are the main symptoms of this disease?
3. What is the outcome of this disease?

Case 5

Patient S., 42 years old, turned to an endocrinologist about an enlargement of the thyroid gland, as well as with complaints of fatigue, weakness, pain in the joints, irritability. Objectively: enlarged thyroid gland, enlarged thyroid gland removed promptly. Microscopic examination: the growth of lymphoid tissue in the stroma of the gland with the presence of lymphoid nodules, atrophy and focal sclerosis of the parenchyma.

Questions for Case 5:

1. What thyroid disease has occurred?
2. What is the basis of the pathogenesis of this disease?
3. Indicate the possible outcome of this disease?

5) Answer the following questions of the current test control.

QUESTIONS OF THE CURRENT TEST CONTROL:

Select all correct answers.

1. Morphological changes in the pancreas in type I diabetes mellitus:
a) insulin
b) a decrease in the number and size of pancreatic islets,
c) fibrosis of the islets,
g) deposition of amyloid (amylin) in the islets,
e) β-cell degranulation.

Select all correct answers.

2. Characteristic of type I diabetes mellitus:
a) absolute insulin deficiency,
b) insulin resistance,
C) the connection with the antigens of the HLA-D system,
g) autoimmune disease,
e) the development of the disease in people younger than 30 years old.

Choose one correct answer

3. The following prevail in insulin infiltrate in patients with type I diabetes mellitus:
a) T lymphocytes,
b) polymorphic nuclear leukocytes,
c) red blood cells,
g) epithelioid cells,
e) multinucleated giant cells.

Select all correct answers.

4. Risk factors for developing type I diabetes mellitus:
a) viral infection,
b) certain histocompatibility antigens,
c) obesity,
g) streptococcal infection,
e) toxic damage to β-cells.

Select all correct answers.

5. Characteristics of type II diabetes mellitus:
a) circulating antibodies to islet cells,
b) genetic predisposition
c) insulin resistance,
g) focal atrophy, amyloidosis of pancreatic islets,
e) relative (rarely absolute) insulin deficiency.

Select all correct answers.

6. The development of diabetic macroangiopathy contribute to:
a) hyperlipidemia,
b) arterial hypertension,
c) nonenzymatic glycosylation of lipoproteins,
d) activation of thrombus formation processes,
e) frequent secondary infections.

Select all correct answers.

7. Clinical and morphological characteristics of diabetic glomerulosclerosis:
a) proteinuria,
b) exudative glomerular changes,
c) necrotic papillitis,
g) nodular glomerulosclerosis,
e) diffuse glomerulosclerosis.

Choose one correct answer
8. The most characteristic sign of diabetic microangiopathy, relevant for its diagnosis:
a) vascular hyalinosis (hyaline arteriolosclerosis),
b) generalized and severe atherosclerosis,
c) Menkeberg media calcinosis,
g) proliferation of endotheliocytes and pericytes,
e) diffuse thickening of the basal membranes of the vascular intima.
Choose one correct answer
9. In a 41-year-old man who came from East Transbaikalia, an examination showed an increase in the thyroid gland, which causes difficulty in swallowing, weight gain, puffiness. Face, lethargy, edematous, pasty thickening of the skin. It is known that many residents of this region have a similar condition. Conclusion:
a) endemic goiter,
b) sporadic goiter.
Choose one correct answer
10. A 32-year-old woman has increased irritability, insomnia, weight loss, exophthalmos, tachycardia, an enlarged thyroid gland. Conclusion:
a) endemic goiter,
b) sporadic goiter,
c) Graves disease
g) thyroiditis Hashimoto,
d) Riedel thyroiditis.
Choose one correct answer
11. During a histological examination of a uniformly enlarged fleshy brown thyroid gland of a 64-year-old woman who died of chronic cardiovascular failure, a pronounced diffuse lymphoplasmocytic stromal infiltration with the formation of lymphoid follicles was found, displacing the atrophic parenchyma and not affecting the organ capsule. Conclusion:
a) Graves disease,
b) Hashimoto's thyroiditis,
c) Riedel thyroiditis,
d) thyroiditis de Curven. Select all correct answers.
12. Characteristics of diabetic macroangiopathy:
a) atherosclerosis,
b) Menkeberg media calcinosis,
c) diffuse fibrosis of intimal arteries,
d) bulbous sclerosis of arteries,
e) hyaline arteriolosclerosis.
Select all correct answers.
13. Morphological manifestations of diabetic nephropathy:
a) nodular glomerulosclerosis,
b) diffuse glomerulosclerosis,
c) hyalinosis of glomerular arterioles,
g) heart attacks of the kidneys,
e) exudative glomerular changes.
Choose one correct answer
14. A 42-year-old man was admitted with complaints of lethargy, episodes of confused consciousness, and mental disorders, especially those that often occur after sleep or exercise and quickly pass after eating or intravenous glucose. When examined in the body of the pancreas, a tumor with a diameter of 1.5 cm, having the structure of an adenoma, was detected and then removed. Conclusion:
a) insulinoma
b) gastrinoma,

c) somatostatinoma
g) carcinoid,
e) vipoma.
Choose one correct answer
15. In a 34-year-old man, two "kissing" callous ulcers of the pyloric stomach were found, accompanied by hyperacidity of the gastric juice, hypergastrinemia. A tumor with a diameter of 2 cm was detected in the tail of the pancreas. Conclusion:
a) gastric ulcer,
b) Zollinger – Ellison syndrome,
c) Menetrie disease
g) insulinoma
e) vipoma.
Choose one correct answer
16. A 49-year-old woman revealed multiple hormonally active tumors of the thyroid and pancreas, the pituitary gland (with the development of hypercalcemia, Zollinger-Ellison syndrome, Cushing's syndrome), and widespread subcutaneous fat lipomatosis. Conclusion:
a) multiple endocrine neoplasia type I,
b) multiple endocrine neoplasia type II,
c) multiple endocrine neoplasia of type III (or 116),
g) carcinoid syndrome,
e) Werner-Morrison syndrome.
Choose one correct answer
17. A 27-year-old woman noted a painful symmetrical enlargement and tightening of the thyroid gland, accompanied by minor symptoms of hyperthyroidism, low-grade fever, moderate leukocytosis. Histological examination in the gland revealed macrophage granulomas with giant cells and areas of proliferation of connective tissue. Conclusion:
a) Hashimoto's thyroiditis,
b) Riedel thyroiditis,
c) thyroiditis de Curven,
g) diffuse toxic goiter,
e) sporadic goiter.
Choose one correct answer
18. Zollinger-Ellison syndrome develops with:
a) diabetes
b) α-cell insuloma,
c) β-cell insuloma,
g) an insuloma of G cells,
e) chronic pancreatitis.
Choose one correct answer
19. A common complication of diabetes due to macroangiopathy is:
a) blindness,
b) myocardial infarction,
c) diabetic glomerulosclerosis,
g) pyoderma,
e) diabetic polyneuropathy.
Choose one correct answer
20. In a 64-year-old patient, histological examination of an enlarged thyroid gland revealed large rounded follicles filled with a dense PAS-positive colloid and lined with monomorphic flattened epithelium. Conclusion:
a) Graves disease,
b) Hashimoto's thyroiditis,
c) Riedel thyroiditis,

g) follicular adenoma of the thyroid gland,
e) colloid goiter.
Choose one correct answer
21. A 68-year-old man underwent a biopsy about the diffuse enlargement and densification of the thyroid gland, accompanied by hypothyroidism. Small atrophic follicles were found, surrounded by extensive fields of fibrous tissue. Conclusion:
a) follicular adenoma of the thyroid gland,
b) Hashimoto's thyroiditis,
c) Riedel thyroiditis,
d) Graves disease,
e) diffuse colloid goiter.
Choose one correct answer
22. Diabetes insipidus develops with a lesion:
a) adenohypophysis,
b) neurohypophysis,
c) pancreatic islets,
g) adrenal cortex,
d) the adrenal medulla.
SITUATIONAL OBJECTIVE
In the study of biopsy material of the thyroid gland in a 50-year-old woman with thyrotoxicosis, a whitish-gray dense encapsulated node with a diameter of 2 cm was constructed from papillary growths of the cubic epithelium surrounding the psammal corpuscles. Hypochromic, nucleolar-free uneven cell nuclei contain eosinophilic inclusions.
Choose one correct answer
23. Thyroid disease:
a) follicular adenoma,
b) papillary cancer
c) medullary cancer
d) anaplastic carcinoma.
Select all correct answers.
24. An immunohistochemical study in tumor cells reveals:
a) thyroid stimulating hormone,
b) thyroglobulin,
c) T3,
g) prolactin,
d) T4,
e) keratin.
Choose one correct answer
25. Metastases are usually localized in:
a) the mediastinum,
b) liver
c) axillary lymph nodes,
d) cervical lymph nodes,
e) lungs.

6. List of recommended literature:

Basic literature:

1. “Basic pathology” Vinay Kumar, Ramzi S. Cotran, Stanley L. Robbins, 1997.

Additional literature:

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3. “General and Systemic Pathology” Joseph Hunter, 2002.
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6. “Histopathology. A Color Atlas and Textbook” Damjanov I., McCue P.A. – Baltimore, Philadelphia, London, Paris etc.: Williams and Wilkins, A Waverly Co., 1996.
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8. “Pathology” Eds. Rubin, J.L. Farber – Philadelphia: Lippincott Raven Publ, 1998 (3th).
9. “Pathology Illustrated” Govan A.D.T., Macfarlane P.S., Callander R. – Edinburgh: Churchill Livingstone, 1995 (4th).
10. “Robbins Pathologic Basic of Disease” Eds. R.S.Cotran, V.Kumar, T.Collins – Philadelphia, London, Toronto, Montreal, Sydney, Tokyo: W.B.Saunders Co., 1998 (6th).
11. “Wheater’s Basic Histopathology. A Color Atlas and Text” Burkitt H.G., Stevens A.J.S.L., Young B. – Edinburgh: Churchill Livingstone, 1996 (3th).
12. “Color Atlas of Anatomical Pathology” Cooke R.A., Steward B. – Edinburgh: Churchill Livingstone, 1995 (10th).
13. “General Pathology ” Walter J.B., Talbot I.C. – Edinburgh: Churchill Livingstone, 1996 (7th).
14. “Concise Pathology” Parakrama Chandrasoma, Glive R. Taylor.
15. “Pathology” Virginia A. LiVolsi, Maria J. Merino, John S. J. Brooks, Scott H. Saul, John E. Tomaszewski, 1994.
16. “Short lectures on pathology” Zagoroulko A., 2002
17. “Robbins pathologic basis of diseases” Cotran R., Kumar V., Collins T.
18. “General pathology” Dr. Fatma Hafez, 1979.
19. “Anderson’s Pathology ” Damjanov I., Linder J. – St. Louis: Mosby Inc., 1995 (10th).

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