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EMERGENCY CONDITIONS IN ENDOCRINOLOGY

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INTRODUCTION

Endocrinology is one of the youngest and most rapidly developing branches of medicine. In terms of general biological and general medical significance, it occupies an important place among other medical disciplines.

The separation of clinical endocrinology as a separate discipline in a medical university is associated with the deepening of our knowledge about the role of endocrine glands in health and disease, with the introduction of hormonal research in various fields of medicine, with the successful use of hormonal drugs for endocrine and non-endocrine diseases, with the growth of some forms of endocrine pathology.

Clinical endocrinology reflects modern advances in the study of endocrine glands, their regulation, tissue metabolism, progress in the synthesis of new hormonal drugs.

This work is devoted to the presentation of the causes of development, clinical picture, modern laboratory diagnostics and treatment of the most common acute complications of endocrine diseases.

The importance of clinical signs of complications of endocrine diseases and the principles of their treatment is necessary for doctors of all specialties, given the prevalence and growth of endocrine pathology diseases, for their timely and early diagnosis and treatment in order to prevent life-threatening conditions.

1. EMERGENCY CONDITIONS IN DIABETOLOGY.

Diabetes mellitus (DM) emergencies are represented by diabetic ketoacidosis (DKA), hyperosmolar non-ketoacidotic state (GONS), lactic acidosis (lactic acidosis) and hypoglycemia. In this publicationprovides the main recommendations for the diagnosis and treatment of these conditions, which came to international practice, confirmed by research and included in the methodological recommendations, within the framework of the federal program "Diabetes mellitus".

1.1. Diabetic ketoacidosis (DKA).

DKA - This is a severe, acute complication of diabetes mellitus caused by insulin deficiency with subsequent poisoning of the body, and primarily the central nervous system, ketone bodies, dehydration, a violation of the acid-base balance towards acidosis and severe tissue hypoxia. DKA is an acute, diabetic metabolic decompensation requiring emergency hospitalization, manifested by a sharp increase in the level of glucose and ketone bodies in the blood, the appearance of ketone bodies in the urine and metabolic acidosis, with varying degrees of impairment of consciousness to coma or without it. The frequency of DKA in European countries is about 0.0046 cases / patient / year (without division into DM types 1 and 2). According to our data, the frequency of DKA in type I diabetes reaches 0.2-026, with type 2 diabetes - 0.07 cases / patients / year. In trained patients regularly determining glycemia by glucometer and test strips and

independently correcting therapy, depending on the results obtained, the frequency of DKA can be reduced to almost zero. Thus, it is by no means an inevitable complication of diabetes mellitus. Mortality in DKA is 5-14% according to domestic data, and 0.65-33% according to foreign data.

The reasons the development of DKA are absolute (type I diabetes) or pronounced relative (type 2 diabetes) insulin deficiency in the body. These include conditions with a sharp increase in the level of counterinsular ("stress") hormones, concomitantintercurrent diseases (especially infections - 20-30% of cases of DKA, other acute diseases and injuries - 10-20%), pregnancy, long-term decompensation of diabetes of both types, pancreatectomy .. However, the main factor in the development of DKA is incorrect behavior of patients: skipping or unauthorized cancellation of insulin injections (about a third of DKA cases), insufficient self-monitoring of metabolism, ignorance of the rules for self-selection of the insulin dose in intercurrent diseases or the consumption of more carbohydrates from food, the introduction of expired or improperly stored insulin, incorrect injection technique or an unnoticed malfunction of a syringe-pen. Medical errors are also frequent: late diagnosis of newly diagnosed type 1 diabetes (5-39% of DKA cases), delay in prescribing insulin for prolonged type 2 diabetes,

The lack of insulin in the body is accompanied by an increase in the serum concentration of glucagon and other insulin antagonist hormones, followed by a decrease in glucose production by the liver, a sharp decrease in glucose absorption by muscle and adipose tissue, resulting in severe hyperglycemia. Insulin deficiency leads to the breakdown of proteins and lipids in the body. The resulting amino acids and free fatty acids (FFA) are also included in the synthesis of glucose in the liver, exacerbating hyperglycemia. A sharp increase in the concentration of FFA in insulin deficiency leads to the accumulation of by-products of their decay - ketone bodies (acetone, acetoacetic and beta-hydroxybutyric acids). The latter are dissociated with the formation of H ions. Thus, metabolic acidosis develops.

Thus, ketoacidosis in DKA is the result of processes occurring mainly in the liver, and the main reason for the formation of ketone bodies is a lack of insulin, which led to an increased breakdown of fat in its own fat depots. Food fats are not involved in enhancing ketoacidosis.

Excess blood glucose causes osmotic diuresis and dehydration. The loss of water reaches 5-12 liters. Intra- and extracellular dehydration may be accompanied byhypovolemic circulatory failure and hypoxia of the central nervous system (CNS) and peripheral tissues .. Compensatory increase in the secretion of catecholamines and aldosterone leads to Na retention and K excretion in the urine, but hypernatremia in DKA develops rarely, since Na is excreted in the form of salts of keto acids. An important component of metabolic disorders in DKA is hypokalemia, which, in addition to the loss of K with urine, is caused by acidosis, in which K ions are exchanged for Na. In the event of a fall in renal perfusion, urinary K excretion is insignificant and normo- or hyperkalemia may occur.

However, even with sufficient renal perfusion, the initial K level can be increased due to the release of K from cells in conditions of insulin deficiency. Violation of the acid-base balance occurs only with a strong decrease in blood pH,

Clinic.DKA develops, as a rule, within a few days (less often within a day), when symptoms of insulin deficiency and dehydration develop (polyuria, dry skin and mucous membranes, thirst, weight loss, weakness). Then the symptoms of ketoacidosis join (the smell of acetone from the mouth, nausea, vomiting, often of the "coffee grounds" type due to hemorrhagic gastritis, noisy deep breathing of the Kussmaul type). On physical examination, signs of dehydration are expressed, including dry skin and mucous membranes, a decrease in the turgor of the skin, muscles and eyeballs, a decrease in blood pressure (BP), and an acceleration of the pulse. Often there is a so-called diabetic pseudoperitonitis, simulating the symptoms of an "acute" abdomen: abdominal pain, tension and soreness of the abdominal wall, decreased peristalsis. These symptoms are caused by the irritating effect of ketone bodies on the gastrointestinal tract, dehydration of the peritoneum. Impaired consciousness develops in parallel with hyperglycemia,dehydration, hypoxia and acidosis of the central nervous system. Depression of the central nervous system is initially manifested by irritability and headache, then lethargy, lethargy, and drowsiness occur. The state of stunnedness gradually increases to the degree of a stopper and coma (10-15% of patients).

DKA is an indication for emergency hospitalization. At the preclinical stage or in the admission department, an express analysis of glycemia is carried out and the determination of ketone bodies in the urine using test strips (with anuria, ketosis is diagnosed by analyzing serum from the same test strips). At the prehospital stage, a 0.9% NaCl solution should be started intravenously at a rate of about 1 L / hour and, if possible, 20 units should be injected intramuscularly. short-acting insulin (ICD).

Laboratory diagnostics... The following laboratory control is carried out in the hospital:

1) rapid blood glucose analysis - once an hour until glycemia drops to 13-14 mmol / l, then once every 3 hours;
2) urine analysis for acetone, if possible - ketone bodies in serum - 2 times in the first 2 days, then once a day;
3) general analysis of blood and urine - initially, then once every 2-3 days;
4) Na and K blood - 2 times a day;
5) serum creatinine - initially, then I every 3 days.
6) hematocrit, gas exchange and blood pH - 1-2 times a day until the normalization of acid-base balance (ACB).

Mandatory hourly control of urine output through the catheter until dehydration is eliminated or until consciousness and urination are restored. Control of central venous pressure (CVP) is highly desirable. Blood pressure, heart rate and body temperature are measured every 2 hours, ECG monitoring and ECG registration are performed at least 2 times a24 hours, if an infection is suspected as a cause of DKA - X-ray of the lungs, culture of blood and urine.

Differential diagnosis of DKA is carried out with GONS, "hungry" ketosis, alcoholic ketoacidosis. as well as other types of metabolic acidosis with a large anionic difference (lactate acidosis, poisoning with salicylates, methanol, paraldehyde).

DKA therapyconsists of five main components - insulin therapy, rehydration, correction of electrolyte disturbances, elimination of acidosis and treatment of the disease. which contributed to the development of DKA.

Insulin replacement therapy is the only type of etiological treatment for DKA. A "low dose regimen" is used - 4-10 units of short-acting insulin (CDI) in 1 hour (on average 0.1 units per 1 kg of body weight per hour), which allows maintaining an optimal level of insulin in serum (50-100 μU / ml), eliminating the main links in the pathogenesis of DKA. DKA insulin therapy should be carried out by intravenous continuous infusion. First, 10-14 units of short-acting insulin (ICD) are injected intravenously, then they switch to continuous administration using a perfuser at a rate of 0.1 units / kg per hour. To prevent the sorption of insulin on the plastic, human serum albumin is added (50 U ICD + 2 ml of 20% albumin + 0.9% NaCl solution up to 50 ml). If there is no perfuser, the ICD will inject once an hour with a syringe into the "rubber band" of the infusion system. It is more convenient to dial ICD (for example, 6 U) into a 2 ml syringe and add 0.9% NaCl solution to 2 ml, thereby increasing the volume of the injected mixture and insulin can be injected very slowly - in 2-3 minutes. Some authors recommend injecting 10 IU of ICD directly into the infusion bottle for every 100 ml of 0.9% NaCl (without albumin) and dripping at a rate of 60 ml / h. In this case, it is impossible to accurately take into account and adjust the dose of the administered CDI, even with its excess in the mixture, since 8-50% of the dose will be absorbed on the materials of the infusion system. Even if at first a certain amount of the mixture is jetted through the system, further sorption of insulin does not stop and accurate metering of the dose is impossible. If, for some reason, it is impossible to immediately establish intravenous administration of ICD, its first injection is given intramuscularly. Sometimes intramuscular injection of ICD at a dose of 6 units. given hourly as an alternative to intravenous administration. However, you should not rely on good absorption of insulin injected intramuscularly (especially subcutaneously) in case of precoma and coma, since a violation of microcirculation impairs the absorption of insulin into the blood.

ICD is dosed according to actual blood glucose levels. If in the first 2-3 hours, despite an adequate rate of rehydration, glycemia does not decrease, then the next dose of ICD is doubled. Glycemia should not be reduced faster than 5.5 mmol / L per hour and no more than 13 mmol / L on the first day. A sharper decline creates a reverseosmotic gradient between intra- and extracellular space; and osmotic imbalance syndrome with a risk of cerebral edema. At a glucose level of 14 mmol / L and below, the infusion of a 5% glucose solution is started, continuing to inject an ICD of 4-8 U. at one o'clock. It should be emphasized that glucose is administered not for the treatment of DKA as such, but for the prevention of hypoglycemia and maintenance of osmolarity, if insulin deficiency is practically

eliminated, but the patient is still unable to eat. After the restoration of consciousness, subject to stabilization of blood pressure, a glycemic level of 11-12 mmol / l and below and pH> 7.3, they switch to subcutaneous administration of ICD - at the beginning, fractionally, 10-14 units. every 4 hours Adjusting the dose depending on the glycemia. On the first day of subcutaneous therapy, you can simultaneously prescribe extended-acting insulin of 10-12 units. 2 times a day.

At an initially normal serum Na level (<145 mEq / L), a 0.9% NaCl solution is used for rehydration, and for hypernatremia (> 150 mEq / L), a 0.45% NaCl solution (hypotonic). The rehydration rate is 500 mm per hour in the first 4 hours and 250 ml per hour thereafter (up to 6-8 liters in total); in case of arterial hypotension, the rate of rehydration increases. Such rehydration more quickly restores the level of bicarbonate, eliminates the anion difference and increases the concentration of Na and C1 in blood plasma less. The rate of rehydration is optimally adjusted for CVP: without this, fluid overload can cause pulmonary edema. The volume of fluid injected per hour with the initial pronounced dehydration can exceed the hourly urine output by no more than 500-1000 ml. With systolic blood pressure below 80 mm Hg. Art. or CVP less than 4 cm of water column. plasma substitutes are shown.

In children and adolescents who have an increased risk of cerebral edema during DKA treatment, rehydration is recommended at a rate of 10-20 ml / kg in the first hour and not more than 50 ml / kg in the first 4 hours of therapy. If rehydration is started with a hypotonic solution, it is administered in smaller volumes; about 4-14 ml / kr per hour.

Correction of electrolyte disturbances is extremely important. Against the background of insulin administration and rehydration, K will enter the cell massively, and also continue to be excreted in the urine. Even with an initially normal and elevated K level (for example, more than 6.5 meq / l), during adequate rehydration and insulin therapy, it can be expected to decrease rapidly, usually 3-4 hours after the start of pH normalization. Therefore, if there is no anuria, then from the very beginning of insulin therapy, even with normokalemia, a continuous infusion of K is started (the target level is 4-5 meq / l). Table 1 shows the rate of administration of potassium preparations in DKA.

The rate of administration of potassium preparations in DKA is presented in Table 1.

Table 1. The rate of administration of potassium preparations in DKA.

K plasma (meq / l)	At pH less than 7.1	At pH more than 7.1	Without pH, rounded
Less than 3	3	2.5	3
3 - 3.9	2.5	2.0	2
4 - 4.9	2.0	1,2	1.5
5 - 5.9	1.5	0.8	1.0
More than 6	Do not administer potassium preparations		

If the K level is unknown, intravenous infusion of K preparations is started no later than 2 hours after the start of insulin therapy, under the control of ECG and diuresis. Do not enter more than 15-20 g of potassium per day. After elimination from DKA on days 5-7, oral administration of K drugs is prescribed. The introduction of phosphates does not provide proven benefits in the treatment of DKA.

The etiological method for correcting metabolic acidosis in DKA is insulin therapy (see above). It should be remembered that, despite the pronounced acidosis of the blood, the pH in the central nervous system remains close to normal for a long time. Sodium bicarbonate has a negative inotropic effect on the myocardium and reduces the sensitivity of the vessels to catecholamines, which can increase hypotension. The administration of sodium bicarbonate is associated with a very high risk of other complications: hypokalemia, intracellular acidosis (although blood pH may rise), "paradoxical" acidosis of the lictor, which contributes to cerebral edema. That is why the indications for the use of sodium bicarbonate are significantly narrowed and its routine use is strongly discouraged. Soda can be administered only when blood pH is less than 6.9-7.0 and only under the control of blood gases, K and Na levels. Bicarbonate is administered in a dose of 2.5 ml of a 4% solution per 1 kg of body weight, intravenously slowly (no more than 4 g per hour). If it is not possible to determine the acid base balance, then the risk of "blind" introduction of alkalis far outweighs the potential benefit. There is no need to prescribe to patients a solution of baking soda per os or per rectum, to drink alkaline mineral water, which was widely practiced earlier.

Hspecific intensive activities with DKA, the provision of respiratory function is included, with pO2 below 80 mm Hg. Art. - oxygen; if necessary - installation of a central venous catheter; in case of loss of consciousness - the installation of a gastric tube for constant aspiration and a catheter into the bladder for an hourly assessment of the water balance: if necessary, antibiotics, heart medications.

After the restoration of consciousness, swallowing, in the absence of nausea and vomiting, you can start a fractional sparing diet with a sufficient amount of carbohydrates and a moderate amount of protein (porridge, mashed potatoes, bread, broth, omelet), with additional subcutaneous injection of ICD at a dose of 4-8 U per reception write. 1-2 days after the start of a meal, if there is no exacerbation of diseases of the gastrointestinal tract (GIT), the patient can be transferred to a regular diet. Early initiation of food intake reduces the risk of hypoglycemia in direct DKA treatment Limit fat intake drastically and it is not advisable to prescribe a hypocaloric diet.

1.2 Hyperosmolar non-ketoacidotic state (GONS).

GONS occurs about 6-10 times less frequently than DKA. It is characterizedlack of ketosis and acidosis and severe hyperglycemia. The majority of patients with GONS are older persons, and more often with type 2 diabetes;

female sex is also a risk factor. Mortality with HONS is higher than with DKA (12-58%).

The reasons development of GONS are conditions that contribute to insulin deficiency: infections (40-60% of cases of GONS), other acute diseases, surgical interventions and injuries (10-15%), newly diagnosed diabetes (33%), failure to comply with the doctor's recommendations for treatment (10- 15%), incorrect medical recommendations (prohibition of sufficient fluid intake when thirsty). Among intercurrent diseases, those that increase dehydration, i.e. accompanied by vomiting, diarrhea. GONS connection with the use of drugs that reduce the volume of circulating fluid or increase glycemia (diuretics, corticosteroidsand others), has not been unequivocally proven.

Pathogenesis... Main links of GONS- hypoglycemia, hypernatremia and dehydration with subsequent impairment of consciousness. Patients of old age often do not notice the gradually salting out dehydration: their thirst is reduced, there is no sufficient intake of fluid in the body, and the increasing osmotic diuresis leads to severe dehydration. It is not completely clear why, with GONS, insulin deficiency leads to high glycemia, but without the formation of ketone bodies. It is likely that hyperosmolarity and dehydration in themselves suppress lipolysis and ketogenesis, so insulin deficiency only causes hyperglycemia, but notketoacidosis. Plasma hyperosmolarity is due to the presence of highly osmotic compounds, primarily glucose and Na. They diffuse weakly inside the cells, which leads to pronounced intracellular dehydration. Hypernatremia in GONS develops due to compensatory overproduction of aldosterone in response to dehydration, but unlike DKA, Na is not excreted along with ketone bodies. The level of Na also increases in the cerebrospinal fluid, which must maintain osmotic equilibrium with plasma under conditions of hyperglycemia; there is an increase in the flow of Na from the brain cells into the cerebrospinal fluid and reverse flow K. The arising violation of the membrane potential of neurons is accompanied by the development of neurological symptoms.

Clinic GONS develops more slowly than DKA, over several days, sometimes weeks or even months. Symptoms of an extreme degree of dehydration dominate, often - circulatory failure up to hypovolemic shock, with oligo - and anuriaafter a period of massive polyuria. Since ketone bodies are not formed, there are no classic clinical symptoms of DKA: nausea and vomiting are less characteristic (they are not caused by GONS itself, but rather a concomitant disease leading to GONS), there is no Kussmaul breathing and the smell of acetone. A feature of GONS is the presence of polymorphic neuropsychiatric symptoms. Most often these are convulsions, speech disorders, paresis and paralysis, pathological reflexes, stiff neck muscles, psychomotor agitation, less often - hallucinatory-delusional symptoms, etc. These symptoms do not fit into any clear syndrome, are variable and disappear after excretion from the GONS. Impairment of consciousness is more pronounced than with DKA (lethargy and confusion - 40-50%, stupor and coma - 27-54% of patients), and directly depends on the value of

plasma osmolarity. If the latter is clearly not increased, then the presence of a stupor or coma should be alerted in terms of an intracranial catastrophe. For GONS, more than for DKA, coagulation disorders with the development of disseminated intravascular coagulation, thrombosis and thromboembolism are characteristic.

The normal value of blood osmolarity is 285-295 mosmol / l, a clear increase is considered to be a level above 320 mosmol / l.

To diagnose GONS, it can be calculated as follows; osmolarity (mosmol / l) = 2 | Na + K (meq / l)] + glucose (mmol / l) + urea (mmol / l) + 0.03 x total protein (g / l). Since the indicators of urea, protein and K have a very weak effect on the final indicator, they can be ignored. Thus, the "effective osmolarity" (EO) is calculated by the formula: EO = 2 x Na, meq / l + glucose, mol / l.

Since the combined development of DKA and GONS is possible, laboratory control for both types of decompensation is the same, and osmolarity should always be calculated. Both DKA and GONS, especially with severe hypotension and hypoxia, can be complicated by lactic acidosis, which sharply worsens the prognosis, therefore, it would be desirable to determine the level of lactate in the blood in all cases of acute diabetic decompensation.

Table 2 presents the laboratory and clinical characteristics of DKA and GONS.

Table 2. Diagnostic criteria for DKA and GONS...

Indicator	DKA			GONS
	easy	moderate	heavy	
Glycemia mmol / l	More than 13.0	More than 13.0	More than 13.0	Usually, more than 30.0
arterial pH noisy blood	7.25 - 7.30	7.0 - 7.24	Less than 7.0	More than 7.3
Serum bicarbonate, meq / l	15 - 18	10 - 15	Less than 10	More than 15
Ketone bodies in urine	+	++	+++	Norm weak +
Osmolarity Serum mosmol / l	Variable	Variable	Variable	More 320
Impaired consciousness and level of wakefulness	Absent	Absent or drowsiness	Stupor / coma	Stupor / coma

GONS treatment is carried out according to the same general principles as DKA, with some peculiarities. Saline solutions are contraindicated at Na levels> 165 mEq / L; rehydration begins with 2% glucose solution (5% solution is diluted

twice with sterile water for injection). At a Na level of 145-165 meq / l, rehydration is carried out with a 0.45% (hypotonic) NaCl solution (a 0.9% NaCl solution is diluted twice with sterile water). With a decrease in the Na level to less than 145 meq / l, they switch to 0.9% NaCl: this solution is used from the very beginning of rehydration ifthe patient has an initial arterial hypotension. The initial volume of rehydration with GONS is greater than with DKA: in the first 2 hours I l per hour, then 250-500 ml under the control of CVP.

In no case should glycemia be reduced faster than 5.5 mmol / L per hour, and osmolarity should not be reduced by more than 10 mosmol / L per hour. A more drastic decrease is fraught with pulmonary and cerebral edema. Adequate rehydration with GONS in itself leads to a decrease in glycemia, therefore, in the first hours of infusion therapy, insulin is not injected at all, then it is administered in very small doses (2 ICD units per hour). If, after 4-5 hours from the beginning of rehydration, after a decrease in the level of Na, severe hyperglycemia persists, then they switch to the insulin dosing regimen recommended for DKA.

Infusion of K with GONS is usually required in a larger volume than with DKA. The introduction of bicarbonate is practically not indicated, since the pH at GONS, as a rule, exceeds 7.3. The rest of the events are held similarly DKA treatment.

1.3. Lactic acidosis (lactic acidosis).

Lactic acidosis- an emergency with a high mortality rate (30-90%), which is the most difficult to diagnose and treat. More often, with diabetes, lactic acidosis of mixed etiology (type A + type B) occurs.

In pathogenesis which involves the decompensation of diabetes, hypoxia, a decrease in renal excretion of lactate in diabetic nephropathy, inextremely rare cases - taking biguanides (metformin). However, more often lactic acidosis develops without taking biguanides. against the background of a rather pronounced insulin deficiency and severe concomitant pathology - acute and chronic renal, hepatic and heart failure, severe infections, intoxication, including alcoholic, senile, etc.

Diagnostics. A clear diagnostic criterion for lactic acidosis is serum lactic acid levels above 5.0 meq / l. The diagnosis is also possible when the lactate level is 2.25 meq / l and the arterial blood pH is less than 7.25. Thus, for diagnosis, it is necessary to determine the concentration of lactate, which is still extremely rare. Help can below bicarbonate level (<18 mEq / L) combined with an anion difference of 15 mEq / L or more.

Clinic... Diagnosis is complicated by the fact that the clinical picture of lactic acidosis is nonspecific and at first resembles the decompensation of diabetes mellitus or increased side effects of biguanides: nausea, vomiting, diarrhea, abdominal pain, weakness. The only specific symptom is muscle pain caused by the accumulation of lactic acid. Severe acidosis with thirst, hyperventilation, confusion and coma can develop within hours. Hyperlactacidemia and acidosis negatively affect the myocardium, increasing the risk of acute heart failure and

severe arrhythmias, which, together with severeperipheral vasodilation leads to hypotension and collapse.

Lactic acidosis therapy is aimed at eliminating lactate and metformin (if the patient has taken it), combating shock, hypoxia, acidosis, electrolyte disturbances and concomitant diseases. If lactic acidosis is caused by an overdose of metformin. enterosorbents are shown. Artificial hyperventilation helps to eliminate excess CO2 caused by acidosis. In this case, the intracellular pH in hepatocytes and cardiomyocytes can be restored, which contributes to the metabolism of lactate and a decrease in its concentration. To inhibit the formation of lactate, intravenous infusion of ICD is required at a rate of 2-5 units. per hour and intravenous glucose 5-12.5 g per hour. The use of sodium bicarbonate can lead to deterioration due to a paradoxical increase in intracellular acidosis, a decrease in cardiac output and an increase in lactate production. If soda is still used, then with extreme caution: at pH <7.0 in small doses - 100 ml of a 2.4% solution once intravenously drip, very slowly. Hypotension is corrected according to general principles. Intensive therapy aimed at combating shock, hypoxia and acidosis, as well as intravenous glucose and insulin, rarely lead to success, unless the only effective measure to eliminate excess lactate (and metformin) is hemodialysis with lactate-free buffer. It allows to save the life of about 60% of patients with lactic acidosis.

1.4. Hypoglycemic state.

Hypoglycemia, including hypoglycemic coma,one of the most common acute complications of diabetes. In a person without diabetes, hypoglycemia is considered to be a glucose level below 2.8 mmol / L in combination with clinical symptoms, or a level below 2.2 mmol / L regardless of symptoms. With diabetes, this definition is not always applicable, since some patients (for example, those who maintain a close-to-normal glycemic level for a long time or have autonomic neuropathy) do not feel a drop in glycemia even to a level below 2 mmol / L, while others (as a rule, long-term decompensated) feel the symptoms of hypoglycemia when the blood glucose level is above 5 mmol / l. It should be emphasized that for any antihyperglycemic therapy, the lower target glycemic level is 3.3 mmol / L.

Regardless of subjective symptoms, hypoglycemia is considered mild, which the patient independently stopped taking carbohydrates; and severe - hypoglycemia, in which, regardless of the degree of impairment of consciousness, the help of another person was required (parenteral administration of glucose, glucagon or oral administration of carbohydrates to a patient who has not yet lost consciousness). Severe hypoglycemia (TG) is the cause of death in 3-4% of patients with diabetes. According to our data, in type 1 diabetes the frequency of TG is 0.08-0.13, with type 2 diabetes about 0.04 cases per patient per year.

The main cause of hypoglycemia is an excess of insulin in relation to the intake of carbohydrates from the outside (food) or from an endogenous source (liver).

The triggering factors for the development of hypoglycemia are listed in table. 3.

Table 3. Triggering factors for the development of hypoglycemia.

Directly related to drug-hypoglycemic therapy	Overdose of insulin, sulfonylmocevin preparations, or glinides	Errors: * doctor: too low target glycemic level, too high doses of antihyperglycemic therapy * patient (error in dialing a dose, too high doses, lack of self-control and training) * insulin syringe - pens (numbers too high)
		Intentional massive overdose (for suicidal purposes)
	Changes in the pharmacokinetics of insulin or oral medications	* drug change without adequate glycemic control
		* delayed excretion of insulin (renal and hepatic failure, antibodies to insulin)
		* incorrect injection tactics (change in depth or incorrect change of the injection site, massage of the injection site or the action of high temperature)
		* drug interactions of sulfonylurea drugs
	Increased insulin sensitivity	* prolonged physical activity, early postpartum period
Nutrition related	Skipping or insufficient carbohydrate intake (without dose adjustment)	
	Short-term physical activity (without additional carbohydrate intake)	
	Alcohol intake	
	Intentional weight loss or fasting (without a corresponding dose reduction of antidiabetic drugs)	
	Slowing gastric emptying	
	Malabsorption syndrome (eg, with enzymatic pancreatic insufficiency)	
	Pregnancy (first trimester) and breastfeeding	

The most common are the discrepancy between the dose of hypoglycemic drugs and the amount of carbohydrates eaten, physical activity above normal and alcohol intake. The risk of TG increases if the patient does not constantly have easily digestible carbohydrates with him to relieve mild hypoglycemia, and also has a history of repeated TGs, a long duration of diabetes mellitus, loss of feeling of hypoglycemic symptoms, and low social status.

Pathogenesis. Normally, at a blood glucose level of about 3.8 mmol / L, the release begins to increasecounterinsular hormones, sufficient to prevent hypoglycemia. Stimulation of the autonomic nervous system with a decrease in glycemia to about 3.3 mmol / l is manifested by the so-called autonomic symptoms (see below). If the glycemia falls below 2.7 mmol / L, symptoms of insufficient

glucose supply to the brain (neuroglycopenia) occur. With a sharp drop in glycemia, autonomic and neuroglycopenic symptoms occur simultaneously. In patients withwith short-term diabetes, the counter-regulation system functions in the same way as in healthy people, and with prolonged diabetes, its dysfunction may occur; decrease in the secretion of glucagon, later and adrenaline, which increases the risk of triglycerides.

The counter-regulation system does not function even with a high concentration of insulin in the blood. For these reasonspatients with diabetes should never wait for hypoglycemia to end on their own, always take urgent measures for stopping it.

Clinic. Clinical manifestations of hypoglycemia are composed of vegetative and glycopenic syndromes.

Vegetative symptoms ("harbingers") include palpitations, tremors, pallor of the skin, sweating, nausea, hunger, anxiety, aggressiveness, mydriasis

To neuroglycopenic -weakness, impaired concentration., headache, dizziness, paresthesia, fear, disorientation, speech, visual, behavioral disorders, amnesia, impaired coordination, confusion, coma, convulsions. Not all symptoms occur with every hypoglycemia; in the same patient, her picture may be different. The peculiarities of alcoholic hypoglycemia include the difficulty of recognizing it by patients around them (similarity of symptoms of hypoglycemia and intoxication), delayed onset, and the possibility of repeated hypoglycemia. The last two signs are also characteristic of hypoglycemia caused by prolonged physical activity.

In mild hypoglycemia, the consequences of insufficient glucose supply to the cells of the cerebral cortex are completely reversible: in hypoglycemia, a compensatory increase in cerebral blood flow is noted by 2-3 times. However, TGs are accompanied by a high risk for the patient (Table 4).

Table 4. Complications and consequences of severe hypoglycemia (TG).

Cardiovascular	Neuropsychic	Others
Atrial fibrillation	Convulsions	Bone fractures
Paroxysmal ventricular tachycardia	Coma Paresis and paralysis	Dislocated joints Damage to soft tissues
Painless myocardial ischemia	Focal motor and sensory symptoms	Traumatic brain injury
Angina pectoris	Decortication	Burns
Myocardial infarction	Disorders of memory and intelligence	Hypothermia
Sudden death	Conduct and personality disorders	Road traffic accidents and accidents
Stroke	Psychoses	

Since the symptoms of hypoglycemia are nonspecific, variable and can mimic neuropsychiatric diseases, the diagnosis needs laboratory confirmation.

Treatment... A diabetic patient receiving insulin, sulfonylurea or glinide preparations. must have fast-absorbing carbohydrates with him at all times. Mild hypoglycemia is stopped by taking carbohydrates in the amount of 1.5-2 (XE): sugar (3-6pieces, it is better to dissolve in water or tea), (1.5-2 tablespoons), or 200 ml of sweet fruit juice, or 100 ml of lemonade (Pepsi-Cola, Fanta), or 5 large glucose tablets (pack of 10 tablets each 3 g in candy form). After stopping nocturnal hypoglycemia, you additionally need to eat 1 XE of slowly digestible carbohydrates (a piece of bread or 2 tablespoons of porridge, etc.).
With TG, the unconscious patient should be laid on its side and the oral cavity should be freed from food debris; do not pour sweet solutions into the mouth (risk of asphyxia). Intravenous stream injected 20-60 ml (maximum 100 ml) of 40% glucose solution, until consciousness is restored. An alternative, especially at home before the arrival of the medical team, is the subcutaneous or intramuscular injection of 1 ml of glucagon, which is produced in the form of syringe tubes and is intended for administration by the patient's relatives. Consciousness is usually restored within 5-10 minutes after the administration of glucagon; if this does not happen, the injection can be repeated. Glucagon is ineffective for alcoholic hypoglycemia and massive (usually deliberate) overdosehypoglycemic drugs.

If consciousness is not restored after the injection of 60-100 ml of 40% glucose solution, intravenous drip administration is started 5-10% glucose and hospitalize the patient. In such cases, it is necessary to exclude a massive overdose of hypoglycemic drugs and other conditions that could be triggered by hypoglycemia, primarily vascular accidents and traumatic brain injury. Intravenous infusion of 5-10% glucose can be continued as long as necessary until glycemia normalizes. If TG is caused by an overdose of oral drugs, then gastric lavage, enterosorbents are indicated, and if it was caused by an intentional massive overdose of insulin, surgical excision of the injection site is performed.

1.5. Insulin allergy

Allergic reactions to insulin occur in 5-30% of cases. An allergy to insulin can occur due to the action of insulin itself or impurities introduced into it for the purpose of preservation, creating an acidic environment or prolonging its action. Allergic reactions can occur with the use of any insulin, but most often with the introduction of prolonged-release insulin. Insulin allergy is most often local, less often general. Distinguish between immediate and delayed forms of an allergic reaction. With an immediate form of allergy, 10-30 minutes after the administration of insulin, pale pink erythema, urticaria or more pronounced manifestations appear on the skin, which can appear from a week to several months. With a delayed form of an allergic reaction, changes at the insulin injection site occur after 24-29 hours and are characterized by the appearance of infiltrates. General reactions to insulin are manifested by itching, urticaria rash, joint pain, fever, gastrointestinal disturbances, angioedema. In some cases, anaphylactic shock occurs.

In the pathogenesis of the latter, the antigen-antibody reaction plays a major role. Released biologically active substances (serotonin, histamine, bradykinin, etc.)

lead to spasm of smooth muscles, bronchi, increased permeability of the vascular wall with the formation of edema, severe hypotension. Anaphylactic shock occurs in the first minutes after the injection of insulin. Patients complain of itching of the skin, numbness of the lips, tongue, face, chest tightness, irritability, fear, abdominal pain, etc. There is hyperemia or pallor of the skin, urticaria, Quincke's edema, excessive sweating, acrocyanosis. The pulse is fast and threadlike. Severe hypotension. The heart sounds are muffled at the apex, the accent is 2 tones over the pulmonary artery. Breathing is noisy, wheezing, with prolonged exhalation. Dry rales are heard in the lungs. In the future, pulmonary edema may develop. In some cases, gastrointestinal and uterine bleeding is observed. Sometimes a general allergic reaction occurs slowly, gradually, a fever and pulmonary edema develops, which disappear after the withdrawal of insulin.

Treatment.

If a local allergic reaction is not pronounced to the injected insulin, it is replaced with another insulin preparation. Treatment with a new insulin drug begins with the intradermal administration of a test dose, followed by bringing it to the required level.

If the change in the insulin drug is ineffective, then antihistamines, 10% calcium chloride solution are started.

If it is impossible to replace the injected insulin with another type of it, the previous insulin preparation is injected for some time in one syringe with microdoses (less than 1 mg) of hydrocortisone.

If you are allergic to insulin with desensitizing therapy, you can desensitize with small doses of insulin. For this purpose, 4 units of insulin are diluted in 400 ml of isotonic sodium chloride solution. 0.1 ml of this solution (1/1000 U of insulin) is injected intradermally into the forearm. Every 30 minutes, a solution containing 1/500, then 1/250 and 1/125 U of insulin is introduced. On the 2nd day, a solution containing 1/100, 1/50 and 1/12 U of insulin is injected at the same intervals. On the 3rd day, a solution containing 1/4, 1/2 and 2 U of insulin is also introduced every 30 minutes. While maintaining a pronounced sensitivity to insulin, desensitization is stopped.

To accelerate the resorption of infiltrates to the affected areas, calcium chloride electrophoresis is prescribed.

In case of anaphylactic shock, complex therapy is prescribed urgently:

a) 2-3 ml of 3% prednisolone solution or 2-3 ml of 0.4% dexamethasone solution in 10-15 ml of 5% or 40% glucose solution is injected intravenously. If there is no effect within 15-20 minutes, the administration of glucocorticoids is repeated;

b) 0.5 ml of 0.1% adrenaline solution is injected into the area of insulin injection;

c) if, against the background of the use of glucocorticoids, the collapse lasts more than 20-30 minutes, 5 ml of 0.2% norepinephrine solution in 500 ml of 5% glucose solution or isotonic sodium chloride solution is injected intravenously (20-40 drops per minute). Treatment is carried out under the control of blood pressure. To combat collapse, transfusion of rheopolyglucin (400-800 ml) is also used. if necessary, prescribe cardiac glycosides;

d) antihistamines are prescribed. Intravenously and intramuscularly, 2-3 ml of 1% diphenhydramine solution, 2% suprastin solution or 2.5% diprazine solution are injected;

e) to combat bronchospasm, 10 ml of a 2.4% solution of aminophylline in 10-20 ml of a 40% glucose solution is prescribed intravenously. In case of acute laryngeal edema with the threat of asphyxia, an urgent tracheostomy is indicated;

f) when seizures appear, 2-4 ml of 0.5% diazepam solution in 10-20 ml of 40% glucose solution is injected intravenously;

g) to combat increased vascular permeability, 5-8 ml of a 5% solution of ascorbic acid is prescribed intravenously in combination with 10 ml of a 10% solution of calcium chloride or calcium gluconate;

7). Reorganization of all foci of infection is necessary.

1.6. Typical mistakes in the management of patients with acute complications of diabetes mellitus

Gastrointestinal symptoms, which are common with DKA, sometimes with elevated serum amylase and leukocytosis, can lead to hospitalization of a patient with DKA in the surgical or infectious diseases department. To exclude such an error when any patient with diabetes develops at least one of the following symptoms - nausea, vomiting, abdominal pain - it is necessary urgently determine glycemia and acetonuria. However, DKA itself can develop against the background of an acute catastrophe in the abdominal cavity or infection, therefore, appropriate alertness and examination are always necessary. DKA does not cause an increase in temperature, therefore, in the presence of fever, a possible infectious or inflammatory process should be excluded.

It is necessary to determine the glycemia of any patient in an unconscious state, otherwise erroneous diagnoses of "cerebrovascular accident", "coma of unknown etiology" are often possible, while the patient has DKA, GONS or hypoglycemic coma. The problem is that none of the clinicalThe symptoms of all the types of emergencies described above in diabetes are not pathognomonic, and a detailed history collection (rate of coma development, trigger factors, etc.) for an ambulance team (EMS) for obvious reasons is far from always possible. In this regard, the most important measure that could significantly speed up the diagnosis and appropriate therapy is the determination of glycemia at the prehospital stage, for which, in a number of regions, the equipping of ambulance teams with glucometers has already begun.

In patients with ketoacidosis, nausea, vomiting, and loss of appetite are often observed. As a result, the patient usually refuses or cannot eat. Termination of insulin administration in these cases is unacceptable. In patients with a sharply weakened or absent insulin secretion, even in the absence of food intake, it is necessary to maintain a certain level of insulin in the blood, which is normally provided by its basal secretion. This is all the more necessary for patients in a state of ketoacidosis or ketoacidotic coma.

Attempts to prevent or eliminate DKA by dietary intervention are erroneous. The etiopathogenetic treatment for DKA is only insulin, recommendations are honey, lemons, etc. with acetonuria or other signs of ketosis are unreasonable. Similarly, the administration of glucose from the very beginning of DKA therapy, which is often practiced in our country, is not a means of treating DKA, which was discussed above. Carbohydrates, including easily digestible ones, will serve as an independent remedy only for ketosis of fasting, which is combined with low blood glucose values and does not threaten life; insulin deficiency in this case is not. To correct fasting ketosis, it is enough to expand nutrition due to slowly digestible carbohydrates.

A common mistake in the management of patients with diabetes mellitus is the incorrect assessment of the severity of metabolic disorders. At the same time, moderate decompensation of diabetes mellitus, accompanied by ketosis and ketonuria, is regarded as ketoacidosis. It should also be borne in mind that signs of ketosis can persist for several days after the characteristic manifestations of ketoacidosis are eliminated.

Unlike ketoacidosis, in ketosis, the patient's condition is moderate, clear consciousness, there are no clinical signs of severe dehydration (decreased skin turgor and eyeball tone) and hypovolemia (decreased systemic blood pressure, compensatory tachycardia, etc.). Indicators of the acid-base state (pH, bicarbonates, shift of buffer bases, etc.) are within the normal range or borderline values. Carrying out rehydration and insulin therapy in such patients according to the regimen recommended for patients

with diabetic ketoacidosis and ketoacidotic coma, inappropriate.

Patients with ketosis do not have clinically significant hypovolemia and centralization of blood circulation; therefore, in this category of patients it is permissible to administer insulin preparations subcutaneously. To eliminate ketosis, a basic-bolus insulin regimen is usually used: two injections of medium-duration insulin in a previously used or slightly increased dose are supplemented with 4-5 injections of short-acting insulin (before breakfast, lunch and dinner, at 23-24 pm and 5-6 o'clock in the morning). After stopping the phenomena of ketoacidosis, the morning and evening doses of insulin are gradually canceled. Dosages are adjusted based on the dynamics of glycemic indicators. For a rough calculation of the daily insulin requirement in the setting of fever, it is useful to know the empirical rule: "... with an increase in body temperature by one degree above 37.5 ° C, the daily need for insulin increases by 25%."

When choosing the route of insulin administration, one should not go to the other extreme, trying to inject insulin subcutaneously, despite the presence of severe dehydration, hypovolemia and centralization of blood circulation (all these disorders are fully present in patients with diabetic ketoacidosis and hyperosmolar non-ketoacidotic coma). As a result of the centralization of blood circulation, microcirculation in the subcutaneous fat is sharply disturbed. Therefore, insulin, like other drugs, is poorly absorbed and does not have the expected hypoglycemic

effect. In addition, when administered subcutaneously, insulin is deposited in the subcutaneous fat. After elimination of the phenomena of centralization of blood circulation and restoration of microcirculation in peripheral tissues, this creates the risk of developing "rebound" hypoglycemia, the reason for which is the active absorption of previously administered insulin. Given the above, patients with
for diabetic ketoacidosis and hyperosmolar states, insulin preparations should only be administered intravenously. Moreover, only short-acting insulins are used, since insulins of medium duration and long-acting are not intended for intravenous administration. Subcutaneous administration of insulin to such patients is unacceptable!

Carrying out insulin therapy for ketosis, it should be borne in mind that with subcutaneous administration of short-acting insulin, the real duration of its hypoglycemic effect does not exceed 4-5 hours. Therefore, when it is administered 4 times a day (i.e. every 6 hours), it is impossible to maintain the required concentration of insulin in the blood during the day. As a result, the patient experiences a short-term (within 1-2 hours) insulin deficiency 4 times a day, which enhances ketogenesis and contributes to the preservation of ketosis and ketonuria. It is also erroneous to administer short-acting insulin five times during the day in the absence of injections at night (usually from 21-22 pm to 8-9 am).

In this case, at night, insulin deficiency is sharply exacerbated, which
provokes an increase in ketonemia and the maintenance of ketosis. In all these cases, the administration of short-acting insulin must be supplemented by injections of medium-acting insulin.

With the ineffectiveness of basic-bolus insulin therapy as a result of severe decompensation of metabolism and insufficient absorption of insulin from the subcutaneous fat, it is sometimes tried to frequently (every 2-3 hours) inject short-acting insulin subcutaneously for the purpose of intensive insulinization. The use of this approach is methodologically incorrect; in these cases, it is more expedient to switch to intravenous administration of short-acting insulin preparations.

Treatment of ketosis does not require a reduced diet with limited fat in the daily diet and replenishment of the daily calorie intake from carbohydrates. The concept of the antiketogenic effect of carbohydrates was formed in the "pre-insulin era", when there was no single effective method for the prevention and treatment of ketoacidosis - insulin therapy. Recommendations to eat honey, lemons, cereals do not lead to a positive effect in such patients. Only "hungry" ketosis can be eliminated by taking carbohydrates, and in cases where insulin deficiency is the cause of ketosis, the introduction of carbohydrates does not in itself suppress the production of ketone bodies.

For the same reason, there is no need to start rehydration in patients with ketoacidotic coma with the administration of glucose solutions or continue its administration after metabolic acidosis has been eliminated, glycemia normalized, and the patient's ability to eat food containing carbohydrates is restored.

When treating patients with diabetic coma, one should remember about the need for sufficiently intensive rehydration under the control of hourly urine output

and indicators of central venous pressure. Insufficiently active rehydration will not lead to the desired positive effect, and excessive intravenous infusion can lead to overload of the heart with volume and the development of pulmonary edema.

In the treatment of diabetic ketosis and ketoacidosis, loop diuretics (furosemide) are sometimes tried to accelerate the elimination of ketone bodies. The use of forced diuresis in such patients not only does not lead to the desired detoxification effect, but also significantly worsens the condition of patients due to the progression of dehydration and hypovolemia. The use of furosemide in a patient with ketoacidosis is justified only if this condition is aggravated by acute renal failure or cerebral edema.

Serious errors that can lead to death are associated with misinterpretation of the abdominal syndrome,characteristic of ketoacidosis. This symptomatology can be easily confused with signs of an acute surgical pathology of the abdominal cavity, which can develop in any person, including a patient with diabetes mellitus. The development of an "acute abdomen" in such a patient leads to a syndrome of mutual burdening and the rapid onset of clinical symptoms of ketoacidosis. Moreover, often this symptomatology begins to dominate in the clinical picture of the disease, and the manifestations of acute surgical pathology fade into the background and are masked by signs of abdominal syndrome.

Attempts to find out what has arisen earlier - signs of damage to the abdominal organs or decompensation of diabetes mellitus - often turn out to be unsuccessful and give dubious and conflicting data. Laboratory tests are also of little help in diagnosis. Changes in the general blood count (leukocytosis, shift in leukoformula "to the left", increase in ESR), hyperglycemia, ketonemia and azotemia can occur in both cases. At the same time, unjustified laparotomy in a patient in a state of ketoacidosis inevitably leads to the development of a lethal outcome. Therefore, in case of any suspicion of the presence of an "acute abdomen" in a patient with ketoacidosis, it is necessary to use the entire arsenal of laboratory and instrumental examinations to exclude or confirm the presence of acute surgical pathology.

In such cases, laparoscopy plays an important role, which can be performed despite the severe general condition of the patient. In addition, it should be remembered that in a state of ketoacidosis, only emergency operations of a resuscitation nature (tracheotomy, ligation of a large bleeding vessel, etc.) are possible. In all other cases, even if the patient needs urgent surgery, preoperative preparation is carried out, including the entire set of measures to eliminate ketoacidosis (rehydration, insulin therapy in low doses, etc.). Carrying out such activities for several hours plays the role of a trial treatment, leading to a regression of the phenomena of peritonism and other signs of abdominal syndrome.

Symptoms of acute surgical pathology of the abdominal cavity in this case persist and become more typical for a particular surgical disease. In any case, the decision to perform surgery in a patient with severe decompensation of diabetes mellitus should be made by the most experienced surgeon.

When treating patients with diabetic ketoacidosis, one should not use a regimen of high doses of insulin, which provides for the simultaneous administration of

several tens (up to 50 or more) units of insulin. High doses of insulin cause a rapid decrease in plasma glycemia and osmolarity, which leads to an osmotic imbalance between the osmotic pressure of cerebral tissue and blood plasma. As a result, fluid moves from the vascular bed into the cranial cavity and cerebral edema develops. However, it should be noted that the development of cerebral edema is possible with the correct conduct of insulin therapy. Children and elderly patients with cerebral atherosclerosis are most prone to this complication, the mortality rate during its development exceeds 70%.

Typically, cerebral edema develops 4-6 hours after starting treatment. It is usually preceded by a "light period" characterized by a short-term improvement in the patient's condition, a decrease in glycemia and a decrease in the severity of metabolic acidosis. Then the patient's condition begins to progressively deteriorate again: cerebral symptoms appear and rapidly increase: headache, dizziness, nausea, vomiting, body temperature rises, bradycardia develops, convulsions appear. The deterioration of the condition occurs against the background of a continuing decrease in glycemia. The reaction of the pupils to light disappears, ophthalmoplegia is possible. In the absence of adequate treatment, the cerebellum is wedged into the foramen magnum and respiratory arrest occurs, and then cardiac activity.

The diagnosis of cerebral edema in patients with diabetic coma is very important, since the development of this complication requires a radical change in treatment tactics. You should be especially careful in patients with hyperosmolar non-ketoacidotic coma. A feature of these comas is the presence of various neurological symptoms, which are often mistaken for a manifestation of cerebral edema and, instead of carrying out adequate rehydration, high doses of diuretics are administered.

In the treatment of cerebral edema, the main role is assigned to the appointment of osmotic, and then loop diuretics. Mannitol is used as an osmotic diuretic, which is administeredintravenous drip in the form of a 10-20% solution. The solution for infusion is prepared from dry matter immediately before its start; water for injection or 5% glucose solution is used as a solvent. When calculating dosages, they are based on the calculation of 1-2 g per kg of patient weight. It should be borne in mind that crystallization of a 20% mannitol solution is possible at room temperature. Therefore, to dissolve the crystals, it may be necessary to heat the solution in a water bath with periodic shaking. Before starting the infusion, the mannitol solution is cooled to body temperature. As a loop diuretic, furosemide (lasix) is usually used at a dose of 80-120 mg. In order to reduce the increased permeability of cerebral vessels and the blood-brain barrier, corticosteroids are prescribed: intravenously administered prednisolone 60-90 mg 2-3 times a day or dexamethasone at an initial dose of 10-12 mg, then continue the introduction of dexamethasone intramuscularly at 4 mg every 6 hours. You can also use hydrocortisone at a dose of up to 1 g per day. Active hyperventilation of the lungs is carried out, hypothermia of the brain is used.

circulation, absorption of fluid from the gastric tract is impaired. Therefore, if, due to the severity of the condition, the patient cannot drink a sufficient amount of liquid, its additional administration is carried out only intravenously.

The main mistakes in the diagnosis of hypoglycemia are associated with the absence. When carrying out rehydration in patients with hyperosmolarity, they sometimes try to inject distilled water into the stomach through a nasogastric tube. This recommendation arose at a time when hypotonic intravenous fluids were not available. The introduction of fluid into the gastric cavity in such patients is always associated with the risk of its aspiration, in addition, due to the pronounced centralization of laboratory confirmation. This is especially true for nocturnal hypoglycemia; to diagnose it by indirect signs (sweating, disturbed sleep, "food" or nightmares, morning headache) is extremely dangerous. Laboratory confirmation is also important because it is not uncommon for patients with very high glycemia, i.e. having severe insulin deficiency, experience severe hunger (glucose does not enter cells due to insulin deficiency) and may complain of sweating and palpitations (autonomic neuropathy with prolonged diabetes mellitus). Without laboratory determination of glycemia, these clinical signs are mistakenly interpreted as hypoglycemia, which leads to an inadequate reduction in insulin dose. Laboratory confirmation and then electroencephalography are required for the differential diagnosis between TG, accompanied by convulsive syndrome and epilepsy.

The introduction of thiamine in TG has neither a pathogenetic rationale, nor a proven clinical value, therefore, it is not necessary. Administration of epinephrine or corticosteroids for prolonged triglycerides, although recommended by some authors, has never been studied in controlled trials. Its feasibility is questionable for the following reasons. Intravenous administration of 60-100 ml of 40% glucose increases glycemia by. 4-7 mmol / l. therefore, should restore consciousness in any patient with TG. If this does not happen, it remains to assume either a concomitant "non-hypoglycemic" cause of loss of consciousness (then adrenaline or corticosteroids are not shown even theoretically), or that TG is caused by a massive overdose of hypoglycemic drugs that continue to be absorbed into the blood and act. ININ this case, adrenaline and corticosteroids will also not help, since their mechanism of action is to stimulate the production of glucose by the liver, but this process is completely blocked in case of massive overdose.

When providing first aid to patients with diabetic and hypoglycemic coma, they sometimes forget about the need to restore and ensure airway patency. Loss of consciousness leads to a significant decrease in muscle tone, which can cause retraction of the root of the tongue and obstruction of the airways. In addition, the oral cavity may contain vomit, removable dentures, food debris that the patient tried to swallow in order to relieve hypoglycemia, and other foreign bodies. Therefore, the first thing to do when assisting a patient in a coma is to cleanse his oral cavity from foreign bodies and remove removable dentures. Then, if the patient retains spontaneous breathing and cardiac activity, he is given a restorative position and in this position is transported to the intensive care unit.

If a hypoglycemic etiology of a coma is suspected until the moment of recovery of consciousness, in no case should you try to feed or drink the patient. These attempts, at best, will lead to the development of aspiration pneumonia, at worst - to asphyxia.

When relieving lung hypoglycemia, you should not consume a large amount of easily digestible carbohydrates, it is enough to eat 1-2 XE. It is known that 1 XE increases blood sugar by an average of 2.0 mmol / l, so 2 XE is usually quite enough to normalize blood glucose. Consumption of large amounts of carbohydrates leads to the development of "rebound" hyperglycemia. An increase in the amount of carbohydrates to 3-4 XE may be required in cases where the patient has depleted glycogen stores in the liver, for example, when hypoglycemia occurs in the morning on an empty stomach or after prolonged physical exertion.

2. DISEASES OF THE PERIOCYTHOUS GLANDS

2.1. Hyperparathyroid crisis (hypercalcemic crisis, acute hyperparathyroidism, acute hyperparathyroid intoxication).

Hypercalcemic crisis- an emergency condition caused by a sharp increase in the level of calcium in the blood serum and characterized by severe neuromuscular and neuropsychiatric disorders, increased blood clotting up to the occurrence of thrombosis and disseminated intravascular coagulation, as well as the development of acute cardiovascular failure, which in the most severe cases is accompanied by a stop heart in the phase of systole. The patient's life is threatened when the calcium level is above 3.5-4.0 mmol / l. In this case, treatment must be started immediately.

Hypercalcemic crisis is a rare pathology, however, if it develops, mortality can reach 50-60%.

Etiology.The development of a hypercalcemic crisis most often occurs against the background of adenoma or hyperplasia of the parathyroid glands, with vitamin D poisoning, as well as in patients with malignant tumors with massive metastases to the bone tissue. A sharp increase in hypercalcemia with the development of a crisis is usually associated with the effect of one or more provoking factors:

- exacerbation of primary hyperparathyroidism in the absence of adequate treatment;
- rapid dehydration in patients with primary hyperparathyroidism and hypercalcemia;
- treatment with thiazide diuretics;
- hypervitaminosis of vitamin D (an attempt to treat osteoporosis typical of hyperparathyroidism with calcium and vitamin D preparations; poisoning with high doses of an oil solution of this drug, which is widely used in poultry farming as a food additive to feed to increase the egg production of birds, etc.);

- consumption of large amounts of milk, calcium-rich food additives and soluble antacids (sodium bicarbonate, calcium carbonate, etc.) - lactic acid syndrome or Burnett's syndrome.

Pathogenesis. An excess of parathyroid hormone leads to the mobilization of calcium from bone tissue due to the predominance of bone resorption processes over bone formation. This leads to bone demineralization and an increase in calcium. In addition, the synthesis of calcitriol, a highly active form of vitamin D, is activated, which leads to excessive absorption of calcium in the intestine and an increase in its concentration in the blood.

Mobilization of calcium from bone tissue can also be triggered by prolonged immobilization, pregnancy, destruction of bone tissue as a result of multiple myeloma, or massive metastases of malignant tumors in the bone.

Hyperparathyroidism increases gastrin levels and stimulates gastric secretion, which leads to the development of recurrent peptic ulcers, which are often regarded as manifestations of peptic ulcer disease.

Hypercalcemia leads to increased urinary calcium excretion and the development of hypercalciuric tubulopathy, which is clinically manifested by symptoms of peripheral diabetes insipidus, refractory treatment with antidiuretic hormone drugs. In addition, as a result of severe hypercalcemia, calcium salts are deposited in various organs and tissues with the development of symptoms of damage to the corresponding organs.

The non-specific nature of the initial symptoms of hyperparathyroidism objectively complicates its timely diagnosis. Often, these patients are treated for a long time for peptic ulcer disease, menopausal osteoporosis, etc. However, attempts to treat such ulcerative lesions of the stomach and duodenum with a milk diet (contains a lot of calcium and phosphorus) and soluble antacids (enhance calcium reabsorption) are ineffective and can provoke the development of a crisis. Attempts to treat hyperparathyroidism-induced osteoporosis with calcium and vitamin D supplements can also aggravate existing hypercalcemia and cause a hypercalcemic crisis.

Clinic. As a rule, the development of a hypercalcemic crisis occurs against the background of an already existing primary hyperparathyroidism, which is manifested by the following main syndromes.

1. Renal syndrome: polyuria, polydipsia, hypoisostenuria and nephrolithiasis.

2. Syndrome of bone changes: diffuse osteopenia, subperiosteal resorption and osteolysis of the terminal phalanges of the hands and feet, fibrocystic osteitis with the development of skeletal deformities and pathological fractures.

3. Gastrointestinal syndrome: anorexia, nausea, flatulence, constipation, weight loss, the risk of developing peptic ulcers of the stomach and duodenum, pancreatitis, pancreocalculosis, cholelithiasis.

4. Syndrome of cardiovascular disorders: arterial hypertension, left ventricular myocardial hypertrophy, cardiac arrhythmias.

5. Visceropathy syndrome: the deposition of calcium salts in various organs and tissues.

6. Syndrome of mental disorders: depression, drowsiness, memory impairment.

7. Syndrome of neurological disorders: radicular disorders, symptoms of tension, paresthesia, paresis of the muscles of the pelvic girdle and lower extremities.

Under the influence of provoking factors, the patient's condition begins to deteriorate rapidly, appetite completely disappears, nausea, indomitable vomiting, abdominal pain, peritonism, and constipation appear. Sometimes the pain is shingles in nature, which leads to an erroneous diagnosis of acute pancreatitis. The body temperature rises, often up to 39-40 ° C. Severe muscle weakness, decreased skeletal muscle tone, weakening of tendon reflexes, and bone pain are observed. The skin is dry, due to severe itching, traces of scratching are often visible on it. Psychoneurological disorders appear and gradually increase in the form of depression or psychomotor agitation up to psychoses. As hypercalcemia progresses, consciousness becomes confused and coma develops.

Activation of blood coagulation factors against the background of severe hypercalcemia often leads to the development of intravascular thrombosis and even disseminated intravascular coagulation syndrome. Intensive secretion of gastric juice against the background of a hypercalcemic crisis often leads to ulcerative damage to the walls of the stomach and duodenum with the development of severe gastrointestinal bleeding.

In the first hours after the development of a crisis, an increase in blood pressure is possible. However, as dehydration progresses, polyuria characteristic of hyperparathyroidism is replaced by oligo- and anuria, against which the symptoms of cardiovascular failure begin to progress and blood pressure decreases. With very high calcemia (up to 4.99 mmol / l), irreversible depression of the respiratory and vasomotor centers occurs, the clinical picture of shock develops and a lethal outcome occurs.

With a hypercalcemic crisis against the background of clinical symptoms of hyperparathyroidism, the following clinical syndromes appear.

1. Abdominal syndrome (acute epigastric pain, nausea, indomitable vomiting, possible gastric bleeding, perforation of stomach and duodenal ulcers, acute pancreatitis).

2. Syndrome of neuropsychiatric disorders (confusion, stupor or hallucinations, which are replaced by doubt and coma).

3. Dehydration syndrome (dry skin and visible mucous membranes, decreased skin turgor, sharpening of facial features).

4. Syndrome of impaired thermoregulation (febrile body temperature).

5. Syndrome of acute cardiopulmonary failure (severe shortness of breath, cyanosis, tachycardia, gallop rhythm, arrhythmias, arterial hypotension, threadlike pulse, vascular collapse and cardiac arrest in the systole phase).

6. Renal syndrome (anuria, increasing azotemia).

7. Syndrome of neuromuscular disorders (hypo- and areflexia, severe muscular hypotension, due to weakness of the respiratory muscles, it may be necessary to ventilate the lungs).

8. Hypercoagulability syndrome (thromboembolic complications, possibly the development of disseminated intravascular coagulation syndrome).

The diagnosis of a hypercalcemic crisis is confirmed by the presence of a high level of blood calcium, changes in the ECG characteristic of hypercalcemia (shortening of the QT interval, widening of the QRS complex), the presence of a tumor of the parathyroid glands according to ultrasound and computed tomography.

Differential diagnosis... Individuals with a characteristic history (hyperparathyroidism, tumors, vitamin D poisoning) do not have problems with the diagnosis of a hypercalcemic crisis. In the absence of a typical history, there may be a need for differential diagnosis with renal colic, exacerbation of peptic ulcer disease, acute pancreatitis, myasthenic crises, gastrointestinal bleeding. Differential diagnosis is based on the study of the level of calcium, which in hypercalcemic crisis exceeds 3.5 mmol / l.

Treatment of hypercalcemic crisis. The following measures are shown to relieve this emergency.

1. **Emergency hospitalization** in the endocrinology or intensive care unit.

2. **Forced diuresis**. Within 3 hours, 3.0 liters of isotonic sodium chloride solution is injected intravenously, in combination with the introduction of 80-100 mg of furosemide per hour. Due to the threat of hypokalemia during the infusion, it is necessary to control the level of basic electrolytes. Subsequently, under the control of diuresis, arterial and central venous pressure, the volume of the infused fluid can be increased to 8-9 l / day, furosemide is administered every 2 hours at a dose of 100 mg. It should be remembered that ignoring these indicators can lead to overload of the heart with volume and the development of pulmonary edema.

3. **Binding of calcium by complexones**. 5% solution of sodium salt of ethylenediaminetetraacetic acid is dissolved in 300-400 ml of 5% glucose and injected intravenously, based on the calculation of 50 mg / kg of weight. When determining the amount of sodium salt of ethylenediaminetetraacetic acid, it should be remembered that 1 ml of its 5% solution contains 50 mg of the active ingredient (ampoules of 5 and 10 ml are produced). The infusion should be carried out very slowly, at a rate of 8-12 drops per minute, it takes about 4-6 hours to carry

out the infusion. With a faster introduction, there is a threat of glomerulosclerosis, hemorrhages in parenchymal organs, collapse.

4. **Fixation of calcium in the bones**. For this purpose, calcitrin is injected intravenously every 8 hours at the rate of 1-4 U / kg of body weight.

5. **Prescribing corticosteroids**. Hydrocortisone hemisuccinate is used, which is slowly injected intravenously 3 times a day, 50 mg
Corticosteroids are most effective for vitamin D poisoning and tumors.

6. **Extracorporeal methods of treatment.** To accelerate the excretion of calcium, hemodialysis or peritoneal dialysis with calcium-free dialysate is performed.

2.2. Hypoparathyroid crisis.

Hypocalcemic crisis - an emergency that occurs when the blood calcium level drops to 1.9-2.0 mmol / l, ionized calcium - less than 1 mmol / l, manifested by the development of convulsive (tetanic) syndrome**.**

Etiology. The most common cause of hypocalcemia and hypocalcemic tetany is hypoparathyroidism, resulting from damage to the thyroid glands during surgery on the thyroid gland or surgical treatment of hyperparathyroidism.

The reasonsthe development of clinically significant hypocalcemia can be varied. The main ones are listed in the etiopathogenetic classification of hypocalcemic syndrome below.

Classification of hypocalcemic syndrome...

A. Violation of hormonal regulation of calcium-phosphorus-magnesium metabolism

I. Deficiency of parathyroid hormone.

1. Removal or injury of the parathyroid glands during surgery.

2. Radioiodine therapy for diseases of the neck organs.

3. Hemochromatosis.

4. Tumor metastases in the parathyroid glands.

5. Congenital underdevelopment of the parathyroid glands (idiopathic hypoparathyroidism).

6. Autoimmune destruction of the parathyroid glands. Violation of the action of parathyroid hormone.

1. Pseudohypoparathyroidism (Martin-Albright syndrome):
a) type I;
b) type II.
2. Pseudohypohyperparathyroidism (Costello-Dent syndrome).

III. Disruption of the secretion or action of two other hormones that regulate calcium-phosphorus-magnesium metabolism.

one**...** Excessive secretion of calcitonin:

a) medullary thyroid cancer;

b) other apudomas.

2 vitamin deficiency D or D-hormone dysfunction:

a) rickets or osteomalacia in adults;

b) malabsorption syndrome;

c) tissue insensitivity to vitamin D.B. Functional hypocalcemia

1. Alkalosis (respiratory, metabolic, iatrogenic).
2. Hyperproteinemia (hyperalbuminemia).
3. Hungry Bones Syndrome.

four**...** Neonatal hypocalcemia of infants born to mothers with hyperparathyroidism.

5. Increased calcium uptake by osteoblastic tumors.

6. Chronic renal failure.

7. Endocrinopathies (diabetes mellitus, hypogonadism, hypopituitarism).

8. Acute destructive pancreatitis.
9. Rhabdomyolysis.

B. Toxigenic and iatrogenic hypocalcemia

1. Excessive intake of phosphorus into the body.
2. Hypomagnesemia.
3. Administration of EDTA preparations.
4. Treatment with mithramycin, neomycin, cisplatin.
5. The use of phenobarbital, diphenin, glucagon, laxatives, antacids.
6. Massive infusion of citrated blood or extracorporeal hemoperfusion.

Pathogenesis. Lack of parathyroid hormone reduces the absorption of calcium in the intestine, slows down its mobilization from the bones and reduces its reabsorption in the renal tubules.

Since the exchange of calcium and magnesium is closely interrelated, a patient with hypoparathyroidism develops not only hypocalcemia, but also hypomagnesemia. In addition, with a lack of parathyroid hormone, phosphaturia decreases, which leads to an increase in blood phosphate levels.

Disruption of phosphate metabolism disorganizes the work of the buffer systems of the blood and leads to the development of alkalosis, which potentiates an increase in convulsive readiness. For hypocalcemia of another etiology,

hyperphosphatemia is uncharacteristic. The developing electrolyte imbalance changes the permeability of nerve cell membranes, enhances the penetration of sodium into the cell and the release of potassium. As a result, the processes of polarization of neuronal membranes in the area of synapses are disrupted, which significantly increases neuromuscular excitability and general autonomic reactivity. All this leads to the development of convulsive syndrome, the extreme manifestation of which is hypoparathyroid tetany.

It should be borne in mind that clinically significant vitamin D deficiency develops only with the combined influence of two factors:

1) insufficient intake of vitamin into the body with food;

2) lack of action of sunlight on the skin, where it is synthesized under the influence of ultraviolet radiation.

Deficiency of vitamin D leads to a disruption in the synthesis of calcium in the intestine, a binding protein and a slowdown in the absorption of calcium from food in the gastrointestinal tract. In addition, vitamin D deficiency slows down the reabsorption of calcium in the distal renal tubules. This leads to impaired absorption and increased excretion of calcium, the end result of which is hypocalcemia, with an exacerbation of which, the development of a hypocalcemic crisis with attacks of tetany is possible.

With tetany, cramps are possible not only of skeletal muscles, but also of smooth muscles of internal organs, including coronary arteries. This can lead to the development of a typical clinical picture of ischemia and myocardial damage.

Clinical picture... The development of a crisis and the appearance of a convulsive syndrome are usually preceded by precursor symptoms: numbness, a feeling of crawling "goose bumps", burning, tingling, tension, stiffness in the muscles of the extremities. Sometimes, warning signs may be absent.

Paresthesias (numbness, tingling, sensation of coldness, heat) begin with the lips, gradually spread to the hands and feet, accompanied by fibrillar muscle twitching and cramps in certain muscle groups. As hypocalcemia progresses, these symptoms develop into extremely painful, transient tonic seizures, which typically first occur in the skeletal muscles of the upper and then in the lower extremities. As hypocalcemia progresses, seizures become generalized with the involvement of the muscles of the trunk, facial and respiratory muscles, as well as the muscles of the internal organs.

From the side of the upper extremities with tetany, flexor tone prevails, from the side of the lower extremities - the extensors. During an attack, the arms are usually bent at the joints, the patient's hands take the shape of an "obstetrician's hand." The patient's legs are extended, brought to each other, the patient's feet are in a state of sharp plantar flexion (horse foot, pedal spasm). There is a trismus of the masticatory muscles, a sardonic smile, a "fish mouth", cramps of the eyelids. In severe cases, opisthotonus develops - convulsive extension of the trunk backward.

Cramps in the intercostal muscles and diaphragm lead to difficulty breathing. The most dangerous manifestation of tetany is laryngospasm, which is more common in children and manifests itself as inspiratory dyspnea, noisy stenotic

breathing, cyanosis, and foam on the lips. The combination of laryngo- and bronchospasm with tonic convulsions of the diaphragm and respiratory muscles can lead to asphyxia, which is fatal if intubation is not done. Sometimes a tracheostomy may be required. Death can also occur from tetany of the heart.

The act of swallowing is impaired due to spasm of the smooth muscles of the esophagus. As a result of pylorospasm, nausea, vomiting, and epigastric pain are possible. Spasm of the intestinal muscles causes intestinal colic, bladder - leads to anuria.

The duration of tetany attacks can be from several minutes to several hours, their frequency varies widely. Attacks can occur both spontaneously and under the influence of mechanical, thermal, electrical, or painful stimuli. Sometimes cramps can be triggered by muscle tension, nervous shock, hot bath, and straightening of the limbs.

In addition to disorders on the part of the somatic nervous system, concomitant dysvegetosis is observed, manifested by profuse sweating, bronchospasm, renal and (or) hepatic colic, vomiting, and diarrhea.

Hypocalcemic syndrome often occurs under the guise of acute psychopathies (paranoid or hallucinatory syndromes, depressive-catatonic psychosis), as well as severe neurological pathology (dysarthria, dysphagia, cranial nerve paresis, extrapyramidal and brainstem disorders, spastic paresis of the extremities, epileptic seizures).

When examining a patient with a hypocalcemic crisis, the following clinical syndromes can be distinguished.

1. Tetanic (convulsive) syndrome. In contrast to epilepsy, consciousness during tetany is preserved, however, with especially severe attacks, the development of a fainting state is possible.

2. Autonomic dysfunction syndrome. With the predominance of the tone of the sympathetic nervous system due to spasm of the peripheral arteries, the attack proceeds with pallor, tachycardia, and increased blood pressure. If the tone of the parasympathetic department predominates, vomiting, diarrhea, polyuria, bradycardia, arterial hypotension are typical.

3. Syndrome of damage to the central nervous system and brain disorders. During severe attacks of tetany, it is possible to develop cerebral edema with stem and extrapyramidal symptoms, sometimes typical epileptiform seizures occur.

4. Visceropathy syndrome. When X-ray examination, calcifications are often found in organs and tissues, intracranial calcification is possible in the area of the basal ganglia, hypothalamus, cerebellum, with a long history of hypoparathyroidism, cataracts are often detected.

5. Syndrome of lesions of the skin and its derivatives. Characterized by dryness and flaking of the skin, often the presence of eczema, exfoliative dermatitis, the appearance of areas of pigmentation and depigmentation of the skin (vitiligo). Often develops - candidiasis. Blisters with transparent liquid contents (exfoliative dermatitis) may appear on the skin. Hair growth is impaired

throughout the body, early gray hair and baldness appear. Brittle nails are observed, the nail plates are pale, dull, often mycotically altered.

Diagnosis of hypocalcemic crisis can be verified if:

- total calcemia less than 1.9-2.0 mmol / l and the level of ionized calcium less than 1 mmol / l;
- a typical reaction of Sulkovich (hypocalciuria up to 10-50 mg / day at a rate of 200-400 mg / day);
- excretion of phosphorus in urine less than 2.8 g / day;
- low levels of parathyroid hormone in the blood;
- ECG signs of hypocalcemia (lengthening of QT and ST intervals);
- typical X-ray changes (signs of osteoporosis and osteosclerosis, periostosis of long bones, ribbon-like seals of metaphyses, calcification of costal cartilage, calcium deposits in the meninges and synaptic ganglia). When examining a patient suffering from attacks of hypocalcemic tetany in history, but at the time of examination being in the interictal period, it may be useful to know the tests to detect latent tetany. To identify signs of increased convulsive readiness, the presence and severity of the following symptoms are most often assessed.

1. Weiss symptom. In order to check this symptom, a neurological hammer is tapped along the outer edge of the orbit. With increased convulsive readiness as a result of tapping, contraction of the circular muscle of the orbit and the frontal muscle occurs.

2. Trousseau's symptom. To identify the symptom, it is necessary to squeeze the shoulder by applying a tourniquet or inflating the sphygmomanometer cuff. In 1-3 minutes after such compression, a characteristic spasm of the muscles of the hand occurs, giving it the shape of an "obstetrician's hand". Mechanical pressure on the brachial nerve along the medial edge of the biceps brachii results in a similar spasm of the carpal muscles.

3. Khvostek's symptom. In order to check this symptom, a neurological hammer is tapped at the exit site of the facial nerve in front of the external auditory canal (in front of the tragus). With increased neuromuscular excitability, in response to this, there is a reduction in facial muscles in the corresponding half of the face. Depending on the intensity of muscle contraction, there are three degrees of severity of this symptom:

- III degree - only the muscles of the corner of the mouth contract (occurs in 25% of healthy people);
- II degree - contraction of the muscles of the corner of the mouth and wings of the nose;
- I degree - contraction of the entire facial muscles of the corresponding half of the face.

Some authors regard the most intense reaction of the mimic muscles as the III degree of severity of the symptom, and the minimum - as the I degree of the response. However, a significant increase in neuromuscular excitability is indicated precisely by the pronounced spasm of the muscles during the symptom check.

4. Schlesinger's symptom. To test this symptom, the straightened leg is passively quickly bent at the hip joint; during flexion at the hip joint, the leg remains straight at the knee joint. With increased neuromuscular excitability, the patient has an involuntary convulsive contraction of the extensor muscles of the thigh and foot, leading to the supination of the latter.

5. Hoffmann's symptom. When checking this symptom, press on the areas located in the projection of the branching of the peripheral nerves. In favor of increased neuromuscular excitability is evidenced by the appearance of paresthesia in response to pressure in these areas.

If questionable results are obtained during the study of these symptoms, a test with hyperventilation can be carried out, after which the above-mentioned symptoms are re-examined.

Differential diagnosis. Tetany stomach can mimic "acute abdomen" syndrome, and tetany of the heart is difficult to distinguish from an attack of angina pectoris or myocardial infarction. It is also difficult to exclude acute forms of coronary artery disease in these cases because as a result of coronary spasm during hypocalcemic crisis, not only prolongation of the QT interval, but also depression of the ST segment, sharpening or inversion of the T wave is often recorded on the ECG. Calcemia study helps the correct diagnosis. In the course of differential diagnosis of hypocalcemic crisis, the presence of anamnestic data indicating damage to the parathyroid glands should also be taken into account. Most often these are surgical interventions on the thyroid and parathyroid glands, treatment with radioactive iodine, etc.

Hypocalcemic tetany should be distinguished from normocalcemic tetany, which develops as a result of alkalosis. The cause of alkalosis in this case is most often the following reasons:

- hyperventilation (usually in patients with hysteria and neuroses),
- repeated vomiting (in patients with decompensated pyloric stenosis).

The phenomena of tetany caused by hyperventilation have a clear relationship with respiratory failure and disappear a few minutes after its normalization. Patients with repeated vomiting usually have a characteristic history and clinical symptoms of gastrointestinal tract disease.

In addition, hypocalcemic tetany should be differentiated from convulsive syndrome of a different etiology.

Epilepsy. An epileptic seizure is accompanied by the development of a convulsive syndrome. But with a hypocalcemic crisis, in contrast to an epileptic seizure, the patient's consciousness is usually preserved, there are no biting of the tongue and a period of amnesia characteristic of epilepsy. After an epileptic seizure, the patient usually falls asleep, which is uncharacteristic for a hypocalcemic crisis. Achievement of normocalcemia quickly relieves not only the manifestations of convulsive syndrome, but also signs of increased convulsive readiness on the EEG. However, against the background of hypoparathyroidism, the development of typical epilepsy is possible.

Hypoglycemia. Severe hypoglycemia may be accompanied by the development of seizures, which should be differentiated not only from epilepsy, but also from hypocalcemic tetany. However, in case of severe hypoglycemia, the patient quickly falls into a coma, and there are indications of diabetes mellitus in the anamnesis. Laboratory examination reveals a low level of glycemia, after intravenous administration of glucose, a pronounced positive effect is observed.

Tetanus. In patients with tetanus, the development of convulsive syndrome is preceded by a characteristic history: usually puncture wounds of the lower extremities, contaminated with soil particles, as well as the lack of measures to prevent this disease (administration of toxoid, tetanus toxoid, etc.). Sometimes the tetanus pathogen enters the body due to skin damage from burns or frostbite. The incubation period ranges from 3 to 30 days, but usually ranges from 1 to 2 weeks. With tetanus, prodromal symptoms can be observed: pain in the wound, muscle twitching around it. The development of convulsive syndrome begins with the involvement of facial muscles (trismus, sardonic smile), then the process spreads to the muscles of the trunk and limbs. The muscles of the feet and hands are usually free of tension. The body temperature is elevated

Treatment of hypocalcemic crisis. For the relief of acute hypocalcemia, the following measures are shown.

1. **Emergency hospitalization in a specialized department.**
2. **Parenteral administration of calcium salts.** Most preferred is an intravenous infusion of 10-20 ml of a 10% solution of calcium chloride, gluconate or lactate dissolved in 500 ml of 5% glucose. Very slow intravenous administration of these solutions in the same doses is also possible. However, with this route of administration, venous thrombosis sometimes develops at the injection site. In addition, with extravasal ingestion of calcium solutions, massive necrosis of the surrounding tissues develops. If calcium administration does not relieve the attack, and the presence of hypocalcemia is not in doubt, alkalosis or hypomagnesemia should be assumed. In such cases, a trial treatment is shown by intravenous administration of 10 ml of a 5% solution of ascorbic acid and 10 ml of a 25% solution of magnesium sulfate.
3. **Symptomatic treatment of convulsive syndrome.** After the relief of seizures by intravenous administration of calcium, sedatives and antispasmodics are prescribed in usual doses (bromides, barbiturates, papaverine, tranquilizers, etc.).
4. **Fight against asphyxia.** With the development of laryngospasm and asphyxia, intubation and mechanical ventilation are indicated. Sometimes, to restore breathing, you have to resort to tracheostomy.
5. **Prescription of vitamin preparations D or similar.**

With a hypocalcemic crisis, the most active vitamin D preparation is used - calcitriol (rocaltrol), which is administered orally at 0.25-1 μg 1 time per day. The dose is selected individually, depending on the severity of the clinical symptoms of

convulsive readiness, as well as the results of monitoring the levels of calcium, phosphorus and magnesium in the blood. Instead of calcitriol, it is possible to prescribe At-10 (tachystin), which is taken at 1-2 mg (40-80 drops) every 6 hours until the seizure syndrome stops, followed by a dose reduction by 2 mg every 2 days.

6. **Neutralization of the negative effects of phosphates.** A calcium-rich dairy-vegetable diet is high in phosphorus, which enhances urinary calcium excretion and skeletal absorption. To bind phosphorus, the patient should take 20-40 ml of a 4% suspension of aluminum hydroxide during meals.

7. **Correction of acid-base balance.** With a hypoparathyroid etiology of a hypocalcemic crisis, to eliminate the alkalosis characteristic of these cases, the administration inside after eating ammonium chloride is indicated at 3-7 g per day.

3. DISEASES OF THE THYROID GLAND

3.1. Thyrotoxic (thyroid crisis)...

This is a severe complication of toxic goiter, caused by internal or external (infection, intoxication, etc.) stress, accompanied by a pronounced increase in the level of thyroid hormones, a sharp excitement of the sympathoadrenal system and the toxic effect of hormones on metabolism, visceral organs with a possible fatal outcome.

The reasons. Thyrotoxic crisis develops in patients with thyrotoxicosis and only under the influence of some provoking factor. Most often, this is a surgical intervention on the thyroid gland, accompanied by its mechanical irritation and damage, which leads to the entry of thyroid hormones into the blood. More than 90% of crises occur 1-2 days after surgery. Another important cause of the crisis is an intercurrent infection or inflammatory process (influenza, pneumonia, acute tonsillitis, etc.), especially in the thyroid gland itself. Other reasons are also possible, for example: pain factor - rough palpation of the thyroid gland, any, even small, operations, colic (renal, hepatic, etc.), neuropsychic and physical stress, strong excitement, mental trauma, etc. Decompensation of diabetes mellitus can cause a thyrotoxic crisis,

It is logical to assume that a thyrotoxic crisis should most often occur in severe thyrotoxicosis. However, it is also possible with moderate and even mild thyrotoxicosis, if the conditions have developed for this, primarily in the form of a provoking factor. A thyrotoxic crisis does not always have an acute onset; in about a third of patients, it develops gradually, within one or more days.

More often observed in the warm season and develops in 0.02-0.05% of patients with thyrotoxicosis. Women get sick more often than men (9: 1).

Modern antithyroid therapy, good preoperative preparation of patients (achieving a euthyroid state), improvement of surgical techniques and methods of anesthesia, as well as correct management in the postoperative period, the use of glucocorticoid hormones before surgery, on the day of surgery and for several days after it have sharply reduced the frequency postoperative crises. In specialized surgical hospitals, thyrotoxic crises, as a rule, are not observed. A spontaneous thyrotoxic

crisis occurs in patients with undiagnosed or insufficiently treated thyrotoxicosis under the influence of various provoking moments.

Pathogenesis of thyrotoxic crisisnot fully understood. However, the following links of pathophysiological mechanisms can be distinguished with no doubt:

* rapid activation of sympathoadrenal receptors under the influence of provoking factors;

* additional increase in blood levels of free hormones T3 and T4;

* decreased function of the adrenal cortex;

* violation of the detoxification function of the liver;

* violation of vascular permeability, the penetration of this protein into the perivascular space, which leads to tissue hypoxia and a sharp metabolic disorder with the development of acidosis and damage to the nervous tissue.

All this causes hypoxia, hyperactivity of the higher parts of the central nervous system, its hypothalamic-pituitary parts and deep disorganization of the neurohumoral regulation of all systemic functions and, above all, the function of the thyroid gland.

An important factor in the development of a thyrotoxic crisis is adrenal insufficiency. It was found that in the initial stage of thyrotoxicosis, the function of the adrenal cortex slightly increases, then, as thyrotoxicosis becomes more severe, it begins to decrease, first to a normal level, and then adrenal insufficiency develops. The overproduction of thyroid hormones is accompanied not only by increased breakdown and excretion of corticosteroids, but also by a qualitative change in their biosynthesis, i.e. the formation of less active hormones. As a result, at a certain stage of thyrotoxicosis, the resulting adrenal cortex insufficiency is aggravated by a thyrotoxic crisis. This pathogenetic mechanism is confirmed not only by direct studies, but also by the efficacy of glucocorticoids in the treatment of thyrotoxic crisis.

In recent years, the state of the kallikrein-kinin system has been increasingly important in the pathogenesis of thyrotoxic crisis. In a thyrotoxic crisis, the activity of proteolytic enzymes increases, the processes of fibrinolysis and the release of plasmin into the bloodstream, which activates the kallikrein-kinin system, increase. in turn, leading to further activity of the kallikrein-kinin system and an increase in the yield of free kinins. Excessive production of thyroid hormones activates the breakdown of proteins, which in turn leads to azotemia, creatinuria, impaired carbohydrate metabolism (due to increased neoglucogenesis, increased absorption of glucose in the intestine, inhibition of its fixation in the liver in the form of glycogen and an increase in glycogenolysis due to a sharp increase in the effect of catecholamines under the influence of thyroid hormones); deficiency of mineralocorticoids with impaired water-salt metabolism (excretion of water, sodium, chlorides, magnesium from the body increases, the content of potassium, calcium and other electrolytes increases, which leads to dehydration and hypotension). In addition, oxidative phosphorylation, which is one of the causes of muscle weakness and an increase in body temperature, is disrupted, the

monoamoxidase activity of the myocardium is suppressed, which increases its sensitivity to catecholamines, increases tachycardia, dystrophic processes in the myocardium, contributing to the development of heart failure. deficiency of mineralocorticoids with impaired water-salt metabolism (excretion of water, sodium, chlorides, magnesium from the body increases, the content of potassium, calcium and other electrolytes increases, which leads to dehydration and hypotension). In addition, oxidative phosphorylation, which is one of the causes of muscle weakness and an increase in body temperature, is disrupted, the monoamoxidase activity of the myocardium is suppressed, which increases its sensitivity to catecholamines, increases tachycardia, dystrophic processes in the myocardium, contributing to the development of heart failure. deficiency of mineralocorticoids with impaired water-salt metabolism (excretion of water, sodium, chlorides, magnesium from the body increases, the content of potassium, calcium and other electrolytes increases, which leads to dehydration and hypotension). In addition, oxidative phosphorylation, which is one of the causes of muscle weakness and an increase in body temperature, is disrupted, the monoamoxidase activity of the myocardium is suppressed, which increases its sensitivity to catecholamines, increases tachycardia, dystrophic processes in the myocardium, contributing to the development of heart failure.

Hypocorticism in a thyrotoxic crisis leads to adynamia, acute heart failure, gastrointestinal disorders, decreased adaptation of the body, increased vascular permeability, etc.

Thus, from a pathophysiological standpoint, a thyrotoxic crisis is characterized by:

* hypermetabolism caused by hyperactivity of thyroid hormones with severe hyperthermia, dehydration and electrolyte imbalance;

* sympathoadrenal hyperactivity with excessive production of catecholamines, increased sensitivity of peripheral tissues to beta-blockers, to their normal content with cardiac, circulatory and psychoemotional disorders;

* Insufficiency of the adrenal cortex with metabolic, morphological disorders in various organs and systems, with cardiovascular and abdominal disorders;

* activation of the kallikrein-kinin system, which aggravates cardiovascular, abdominal, autonomic and other disorders.

The clinical picture of a thyrotoxic crisis is caused by the action of thyroid hormones, catecholamines, and a deficiency of adrenal cortex hormones.

In most cases, a thyrotoxic crisis has an acute onset. Symptoms of thyrotoxicosis rapidly increase: mental and motor restlessness increases, the skin becomes hot and moist, tachycardia grows, reaching 150-200 beats per minute, atrial fibrillation, severe shortness of breath often occurs, signs of heart failure appear and rapidly increase, the body temperature rises to 40 -41 ° C. Blood pressure first rises with a significant increase in pulse pressure (due to a decrease in diastolic blood pressure to 0), and then falls. An increase in blood pressure at the beginning of a crisis is associated with the excitement of the sympathoadrenal division of the autonomic nervous system, followed by paralysis of peripheral

vessels, leading to a drop in blood pressure and the development of hemodynamic collapse. An important factor in the fall in blood pressure is the insufficiency of the adrenal cortex, as well as damage to the nerve nodes and plexuses of the heart. The rush of blood to the skin causes increased sweating, redness of the face, elbows and knees, followed by dryness and paleness of the skin. Shortness of breath, difficulty in breathing develop, sometimes turning into pulmonary edema due to toxic damage to the cardiovascular system and a decrease in the contractile function of the myocardium.

Clinic... Some authors identify three main clinical variants of thyrotoxic crisis:

*cardiovascular,

*abdominal,

* neuropsychic.

Cardiovascular option manifested by tachycardia, extrasystole, atrial fibrillation, cardiovascular failure, shock.

The abdominal variant is manifested by nausea, vomiting, diffuse abdominal pain, diarrhea, enlarged liver, jaundice;

The neuropsychic variant is characterized by muscle hypertonicity with increased tendon reflexes, tremors, seizures, flaccid paralysis (usually of the legs), mental agitation, manic psychosis, delirium, stupor, coma.

If a patient with thyrotoxicosis had concomitant diseases (peptic ulcer, cholecystopancreatitis, bronchial asthma, diabetes mellitus, etc.), then the thyrotoxic crisis proceeds under the guise of their exacerbation, which greatly complicates the diagnosis of the crisis. Cases of an atypical course of thyrotoxic crisis under the guise of anaphylactic or septic shock, periodic flaccid paralysis, withdrawal symptoms, and surgical abdominal pathology have been described.

Depending on the degree of increase in body temperature, pulse rate and neuropsychiatric disorders, the thyrotoxic crisis is divided into mild, moderate and severe.

With a mild crisis, the body temperature rises to 38 ° C, tachycardia up to 100-120 beats per minute, but patients behave relatively calmly. With a crisis of moderate severity, body temperature reaches 38-39 ° C, tachycardia up to 120-140 beats per minute, patients are agitated. In severe crisis, the body temperature is above 39 ° C, tachycardia - 150-160 beats per minute, the pulse is often arrhythmic. The pronounced motor excitement of the patient can be replaced by adynamia, periodically - by loss of consciousness. With an unfavorable course, after 24-48 hours, and with a violent course after 12-24 hours, a severe crisis is replaced by an acute, comatose phase. The severity of the patient's condition is growing. Hyperthermia reaches 41-43 ° C, tachycardia - 200 beats per minute, atrial fibrillation, severe hypotension, weakness, respiratory failure, severe muscle hypotension occur, weakening and partial extinction of reflexes. Sharp agitation with mental disorders, sometimes similar to hallucinatory disorders, and catatonic syndrome are replaced by progressive lethargy, increasing confusion of consciousness up to coma.

Thyrotoxic coma is most often fatal. In this regard, the change in the patient's psyche acquires prognostic value. The cause of death is most often acute heart failure, insufficient function of the adrenal cortex, acute liver failure. Currently, mortality in thyrotoxic crisis has decreased to 10% as a result of the use of complex therapy (antithyroid drugs, beta-blockers, adrenolytic, corticosteroid drugs, administration of crystalloid and colloidal solutions, cardiac glycosides, antiarrhythmic and other drugs).

Thyrotoxic crisis must be differentiated from cardiovascular failure, hypertensive crisis in patients with thyrotoxicosis, pneumonia, acute gastroenteritis, acute encephalitis, psychosis. With indomitable vomiting, diarrhea, azotemia, acetonuria, hyperbilirubinemia, jaundice often occur, in connection with which the thyrotoxic crisis has to be differentiated from diabetic, uremic, hepatic coma. In this case, it is necessary to be based on the anamnesis, the clinical picture of the thyrotoxic crisis and the indicated diseases, as well as on specific laboratory parameters. Due to the urgency of diagnosis and treatment of thyrotoxic crisis, the history and clinical manifestations become decisive for the diagnosis. Laboratory diagnostics are usually delayed and therefore not critical.

Treatment of a thyrotoxic crisis... *Therapy should be aimed at the main pathogenetic links:*

1. Suppression of synthesis, blockade of secretion and a decrease in the activity of thyroid hormones with the help of mercazolil, tyrosol, propicil and iodine preparations.

1. Reducing the excitability of the central nervous system by the administration of chlorpromazine, droperidol, suprastin, promedol.

2. Decrease in sympathoadrenal activity under the influence of beta-blockers.

1. Restoration or replacement of the function of the adrenal cortex with glucocorticoid hormones.

3. Decrease in the activity of the kallikrein-kinin system by the administration of contrikal, glucose solution, isotonic sodium chloride solution.

4. Overcoming fever, dehydration, intoxication, acidosis, infectious complications, cardiovascular failure.

5. Symptomatic therapy aimed at eliminating individual symptoms (syndromes) eight. Treatment of the underlying disease, against the background of which a thyrotoxic crisis developed.

Emergency care consists in emergency hospitalization of the patient. In the first minutes, it is necessary to measure blood pressure, record an ECG, and determine the blood glucose content by the express method. With a sharp psychomotor agitation, 10 ml of a 20% solution of sodium oxybutyrate is slowly injected intravenously in 20 ml of isotonic sodium chloride solution or 1-2 ml of seduxen solution (Relanium) or intramuscularly 1-3 ml of a 2.5% solution of levopromazine (tizercin) or 1 ml of 0.5% haloperidol solution. Treatment is continued with the introduction of anaprilin or obzidan - 1-4 ml of 0.1% solution in

10 ml of isotonic sodium chloride solution under the control of blood pressure. 50-60 mg of mercazolil or tyrosol is administered through a gastric tube.

Subsequently, during the first hour of intensive care, it is necessary to establish ECG monitoring, perform a general blood and urine test, a biochemical blood test for the content of glucose, proteins, bilirubin, creatinine, urea, electrolytes. Shown are physical cooling (wet wrap, ventilator, ice packs), intramuscular injection of 2-4 ml of 50% solution of analgin, 2 ml of 1% solution of diphenhydramine, 1 ml of pipolphene. Salicylates, acetylsalicylic acid are contraindicated, since, competing with thyroxine and triiodothyronine for the connection with thyroxine-binding blood proteins, they increase the level of free thyroxine and triiodothyronine in the blood. Oxygen therapy is required, the introduction of broad-spectrum antibiotics.

For continuous infusion therapy, a catheter is inserted into the subclavian vein and 500 ml of 5% glucose solution, 1-2 ml of 0.025% strophanthin solution (dropwise), 3 ml of 3% prednisolone solution, 200 mg of cocarboxylase, 10 ml 5 % ascorbic acid solution, insulin depending on glycemia.

During the first day, it is necessary to conduct a blood test for the content of thyroid hormones, coagulability (coagulogram), and re-examine the necessary indicators. An hour after taking mercazolil (tyrosol), 10 ml of Lugol's solution, 500-1000 ml of 5% glucose solution or isotonic chloride solution must be injected intravenously

3.2. Hypothyroid coma.

Hypothyroid coma - an extremely severe manifestation of hypothyroidism, characterized by a rare exacerbation of all symptoms of the disease and loss of consciousness.

The main reasons leading to the development of hypothyroid coma:

* hypothermia;
* infectious and inflammatory processes, in particular, pneumonia (it proceeds without fever, tachycardia, leukocytosis);
* surgical interventions;
* injuries;
* treatment with sedatives, tranquilizers, neuroleptics, hypnotics, especially when these funds are used often and in large doses;
* long-term undiagnosed hypothyroidism and, therefore, long-term absence of treatment;
* non-systematic treatment, taking substitution drugs in insufficient doses, especially in the cold season;
* alcohol abuse, alcohol intoxication;
* gastrointestinal and other bleeding;
* hypoglycemia of any origin;
* hypoxia of any genesis.

At the heart of pathogenesishypothyroid coma is a severe, progressive disorder of all types of metabolism caused by prolonged thyroid insufficiency, which is manifested by a sharp suppression of oxidative enzymatic processes in the brain

tissue. Of great importance is also the toxic effect of carbon dioxide accumulating in the body due to alveolar hypoventilation, as well as hypothermia, severe adrenal insufficiency, cardiovascular insufficiency. For hypothyroid coma, the following symptoms are characteristic:

- gradual development of coma: progressive lethargy, drowsiness, gradually giving way to complete loss of consciousness;
- the patient's skin is dry, flaky, pale icteric, cold, body temperature is significantly reduced ("hypothermic coma");
- the face is pasty, puffy, pale yellowish, characteristic dense swelling of the hands, legs, feet;
- severe bradycardia, deafness of heart sounds;
- arterial hypotension, possible development of left ventricular failure;
- rare breath;
- accumulation in the serous cavities (abdominal, pleural, pericardial cavity) of a mucin-like substance, which is manifested by a shortening of the percussion sound in the lateral parts of the abdomen, the expansion of the borders of the heart, the appearance of a zone of dull percussion sound over the lungs with the disappearance of vesicular breathing in this zone; recognition of this syndrome is facilitated by ultrasound;
- decreased muscle tone;
- a sharp decrease and even disappearance of tendon reflexes;
- oligo / anuria;
- atony of smooth muscles, which is manifested by syndromes of acute urinary retention or rapidly developing dynamic and even mechanical (megacolon) intestinal obstruction.

Clinic... Hypothyroidism can occur at any age, women are more often sick. Patients present characteristic complaints that allow suspecting the disease:

- progressive general and muscle weakness; increased fatigue;
- feeling of constant chilliness;
- drowsiness;
- increase in body weight;
- decreased memory;
- swelling of the face, hands, often the whole body;
- constipation;
- difficulty speaking;
- dry skin;
- hair loss;
- sexual dysfunction;
- changing the timbre of the voice to a low, rude one;
- hearing loss (due to edema of the Eustachian tube and structures of the middle ear).

When examining patients, the following manifestations draw attention to themselves:

- patients are adynamic, apathetic, inhibited, drowsy; slow;
- the skin has a pale yellowish tint (pallor is due to anemia, a yellowish tint, especially in the area of the palms, is associated with a decrease in the formation of vitamin A from provitamin A carotene); cold (especially hands, feet);
- pronounced peeling of the skin and hyperkeratosis, mainly in the area of the elbow, knee joints, heel area;
- the face is puffy with pronounced periorbital edema, the eyelids are swollen, the lips are thickened;
- body temperature is lowered;
- the arms and legs are edematous, and no depression remains after pressure, in contrast to cardiac edema; with severe degrees of hypothyroidism, severe swelling of the whole body is possible;
- hair is dull, brittle, falls out on the head, eyebrows (Hertog's symptom), extremities, grows very slowly; nails are thin, break easily, dull with longitudinal or transverse striation;
- speech is slowed down, patients have difficulty pronouncing words (this is largely due to a significant increase in language, which causes dysarthria), often patients have difficulty remembering the necessary words, which slows down speech; low, rough voice (swelling and thickening of the vocal cords);
- symptoms of ophthalmopathy may appear (as a rule, with an autoimmune form of the disease);
- muscle damage is characteristic, which is based on edema, dystrophy and hypertrophy of muscle fibers. Mostly the proximal muscles of the limbs, shoulder and pelvic girdles are affected. The clinical manifestations of hypothyroid myopathy are muscle weakness, muscle pain and stiffness, and an increase in muscle volume and density.

  Hoffman's syndrome is characteristic, which includes:

  1. hypertrophy of the proximal muscles;
  2. muscle weakness, painful muscle cramps;
  3. muscle stiffness after exercise;
  4. delayed muscle relaxation after exercise;
  5. bradycardia.

Changes in organs and systems

Respiratory system.

Due to the swelling of the nasal mucosa, nasal breathing is difficult. Vasomotor rhinitis is common. Patients are prone to the development of pneumonia and acute respiratory viral diseases.

The cardiovascular system

The clinic of damage to the cardiovascular system is due to the duration and severity of hypothyroidism.

The greatest changes are revealed with pronounced and long-term current forms. Changes in the cardiovascular system are always observed and are caused by a deficiency of thyroid hormones.

Clinical signs of damage to the cardiovascular system:

- cardialgia, typical attacks of angina pectoris are possible;
- cardiac arrhythmias - in the overwhelming majority of patients, bradycardia, extrasystolic arrhythmia, and only in 5-10% of patients, tachycardia is possible (probably due to anemia and severe circulatory failure);
- an increase in the boundaries of the heart (due to the development of myocardial dystrophy and cardiomegaly, as well as hydropericardium). Pericardial effusion is detected by echocardiography in 30-80% of patients in an amount of 15 to 100 ml or more; pericardial fluid is rich in cholesterol and protein;
- deafness of heart sounds, intense systolic murmur in the apex of the heart;
- with severe myocardial damage and severe hydropericardium, circulatory failure develops;
- on the ECG, in addition to bradycardia, there is a low voltage of the teeth, a decrease in the ST segment downward from the isoline;
- blood pressure is increased in 10-50% of patients, in the rest it can be normal or low. The causes of arterial hypertension are an increase in peripheral resistance, a decrease in the production of atrial natriuretic factor, an increase in the sensitivity of the arterial wall to the vasoconstrictor action of catecholamines.

Digestive system

With hypothyroidism, atrophy and edema of the mucous membrane of the stomach and intestines develops, as well as a decrease in the motor function of the gastrointestinal tract. Clinically, this is manifested by chronic gastritis (decreased appetite, feeling of heaviness in the epigastrium after eating, belching with air, decreased acidity and volume of gastric juice), impaired intestinal absorption. Violation of the motor function of the gastrointestinal tract leads to nausea, vomiting, distension of the stomach, intestines, severe constipation, in severe cases - to megacolons and even paralytic intestinal obstruction.

Urinary system

Renal function in hypothyroidism is impaired. The rate of renal blood flow, glomerular filtration decreases, which leads to a decrease in urine output, fluid and sodium retention in the body. In urine tests, proteinuria is noted. Patients are prone to the development of chronic pyelonephritis.

Nervous system

In 80% of patients, damage to the peripheral nervous system in the form of mononeuropathy is observed. This is manifested by paresthesias, numbness of the arms and legs, the development of carpal, cubital tunnel syndromes (due to compression of the nerve trunks in the osteo-ligamentous canals) is possible, a decrease in tendon reflexes is characteristic.

Violation of the functional state of the brain is manifested by a decrease in memory, mental performance, non-criticality towards oneself, others, lack of communication, lethargy, drowsiness. In severe cases, mental disorders are possible: depression, hallucinations, delirium.

Since thyroid hormones are necessary for the maturation and differentiation of the central nervous system, with congenital hypothyroidism, in the absence of timely replacement therapy with thyroid hormones, normal mental development is disturbed, mental underdevelopment (cretinism) is observed.

Endocrine system

In patients with hypothyroidism, signs of damage to many endocrine glands are often observed.

There is a decrease in the functional reserves of the hypothalamic-pituitary-adrenal system. A decrease in the formation of cortisol and a decrease in its metabolism are characteristic. The function of the sex glands in women is impaired: there is amenorrhea, infertility. In primary hypothyroidism, there is often a combination of hyperprolactinemia, galactorrhea, and amenorrhea. Lack of thyroid hormones leads to an increase in the secretion of thyroliberin, which increases the production of prolactin by the adenohypophysis, hyperprolactinemia blocks the action of gonadotropins at the ovarian level, which leads to amenorrhea.

In men, libido sexualis disappears, sexual weakness develops.

Often, hypothyroidism is combined with type 1 diabetes mellitus, while frequent hypoglycemia is possible, since hypothyroidism disrupts the absorption of carbohydrates in the intestine. The combination of hypothyroidism with diabetes is due to the development of autoimmune mechanisms.

Hematopoietic system

Patients, as a rule, develop hypochromic anemia (due to a decrease in iron absorption in the small intestine and the absence of a stimulating effect of thyroid hormones on erythropoiesis), less often - B12-deficiency anemia (due to impaired absorption of vitamin B12 in the intestine).

Laboratory and instrumental data.

1. OAK: hypochromic anemia, tendency to leukopenia, lymphocytosis, increased ESR.
2. OA urine: possible proteinuria, decreased urine output.
3. BAC: an increase in the content of cholesterol, triglycerides, prebeta and betalipoproteins in the blood, a decrease in the content of total protein and albumin, hypergammaglobulinemia is possible, an increase in the level of creatine phosphokinase, lactate dehydrogenase.
4. II blood: changes are observed mainly in hypothyroidism caused by autoimmune thyroiditis.
5. ECG: bradycardia, low voltage of the teeth, a decrease in the ST interval from top to bottom from the isoline.
6. Echocardiography: an increase in the size of the heart, signs of a decrease in myocardial contractility, the presence of hydropericardium.
7. Ultrasound of the thyroid gland: reduction in size in primary hypothyroidism (atrophic form of autoimmune thyroiditis, strumectomy).

8. Computer or magnetic resonance imaging of the brain: detecting an increase in the pituitary gland if secondary hypothyroidism is due to a tumor lesion of the pituitary gland.
9. Reflexometry: lengthening of the Achilles tendon reflex more than 300 ms.
10. Study of the functional state of the thyroid gland: indicators are significantly reduced.

Treatment for hypothyroid coma.

1. Substitution therapy with thyroid drugs.
2. The use of glucocorticoids.
3. Fight against hypoventilation and hypercapnia, oxygen therapy.
4. Elimination of hypoglycemia.
5. Normalization of the activity of the cardiovascular system.
6. Elimination of severe anemia.
7. Elimination of hypothermia.
8. Treatment of concomitant infectious and inflammatory diseases and elimination of other causes that led to the development of coma.

Patients in a hypothyroid coma should be admitted to the intensive care unit and intensive care unit. It is necessary to puncture the cubital vein and immediately take blood to determine the content of thyroxine, triiodothyronine, thyrotropin, cortisol, glucose, sodium, chlorides, blood gas composition, and acid-base balance. However, the removal of the patient from a hypothyroid coma should be started immediately, without waiting for the results of the analysis. It is also necessary to record an ECG, make a catheterization of the bladder, and schedule a consultation with a neurologist.

1. Replacement therapy with thyroid hormones.

Thyroid hormone replacement therapy is the mainstay of treatment for a patient in a hypothyroid coma. However, given the great importance of glucocorticoid insufficiency in the development of hypothyroid coma and the impossibility in some cases to differentiate between primary and secondary hypothyroid insufficiency, treatment of hypothyroid coma should be carried out by the combined administration of thyroid and glucocorticoid drugs.

The drug of choice in thyroid hormone replacement therapy in hypothyroid coma is L-thyroxine due to its less negative effect on the myocardium. This is extremely important for patients with coronary artery disease. The transformation of thyroxine into triiodothyronine, which is more active, occurs gradually in peripheral tissues.

Treatment is recommended to start with intravenous administration of thyroxine 250 mg every 6 hours, which leads to an increase in the level of the hormone in the peripheral tissues until saturation within 24 hours. Then they switch to maintenance doses of thyroxine - 50-100 mcg per day.

Recommends the initial daily dose of thyroxine (400-500 mcg) to be administered slowly intravenously. On the following days, the dose is reduced to 50-100 mcg.

Due to the fact that the action of thyroxine appears later than triiodothyronine, intravenous administration of triiodothyronine at a dose of 25-50 μg was previously recommended. Triiodothyronine exhibits metabolic effects much faster and quickly penetrates the central nervous system through the blood-brain barrier. However, intravenous administration of triiodothyronine can have a negative effect on the cardiovascular system. Therefore, there is a method of treatment with triiodothyronine by introducing it through a gastric tube. Initially, 100 μg of triiodothyronine is administered, then adding 25-100 μg every 12 hours, specifying the dose depending on the dynamics of the increase in body temperature and clinical symptoms. Absorption through the gastrointestinal mucosa of both triiodothyronine and thyroxine in hypothyroid coma is significantly reduced. However, in the absence of thyroxine preparations for intravenous administration,

Treatment with thyroid drugs is carried out under the obligatory control of ECG, blood pressure, heart rate, respiratory rate.

2. The use of glucocorticoids.

Treatment with glucocorticoid drugs should be carried out simultaneously with the use of thyroid drugs, since the latter can aggravate adrenal insufficiency in patients.

It is recommended to inject 10-15 mg of prednisolone or 25 mg of water-soluble hydrocortisone (hydrocortisone hemisuccinate) intravenously or through a gastric tube every 2-3 hours simultaneously with thyroid hormones, and intramuscularly - 50 mg of hydrocortisone 3-4 times a day. After 2-4 days, the dose of glucocorticoids is gradually reduced.

50-100 mg of hydrocortisone hemisuccinate should be administered simultaneously intravenously, and the daily dose of this drug may be 200 mg.

After recovery of consciousness, improvement of general condition, normalization of heart rate and respiratory rate, glucocorticoids are gradually canceled.

3. Fight against hypoventilation and hypercapnia, oxygen therapy.

In patients in hypothyroid coma, there is a gradual decrease in respiration, alveolar hypoventilation, and the development of respiratory acidosis.

To eliminate acidosis, improve pulmonary ventilation, humidified oxygen is inhaled through nasal catheters. However, with the development of severe acidosis, this is not enough. In this case, it is necessary to switch to artificial ventilation.

With a sharp decrease in the rhythm of breathing, 2-4 ml of cordiamine should be injected intravenously, which stimulates the respiratory center. The introduction of cordiamine during the day can be repeated 3-4 times under the control of blood pressure.

4. Elimination of hypoglycemia.

Normalization of blood glucose levels helps to improve the function of the brain, myocardium, and kidneys. To eliminate hypoglycemia, intravenous administration of 20-30 ml of 40% glucose solution and intravenous drip infusion

of 500-1000 ml of 5% glucose solution are recommended, depending on the level of CVP, heart rate, diuresis.

5. Normalization of the cardiovascular system.

Patients in a hypothyroid coma often have severe arterial hypotension, and often collapse and symptoms of left ventricular failure.

However, sympathotonic drugs, especially norepinephrine, are contraindicated. Against the background of treatment with thyroid drugs, they can cause the development of myocardial infarction and aggravate existing heart failure or provoke it.

To combat collapse, intravenous drip administration of rheopolyglucin, polyglucin, 10% albumin solution, 5% glucose solution, isotonic sodium chloride solution is used. The volume of infusion therapy is purely individual and depends on the indicators of CVP, blood pressure, urine output, the severity of symptoms of heart failure and can range from 0.5 to 1 liter of fluid per day. Giving large amounts of fluids can be dangerous due to cardiac overload and worsening heart failure.

To increase blood pressure, you can use intravenous drip of angiotensinamide, it has a strong pressor effect due to an increase in peripheral vascular resistance, especially small-caliber arterioles. The drug has little effect on venous tone. Angiotensinamide does not have a direct effect on the heart and does not cause arrhythmias in therapeutic doses.

Angiotensinamide is available in vials of 0.001 g. The contents of the vial are dissolved in 250 ml of 5% glucose solution (the concentration of the resulting solution is 4 μg of the drug in 1 ml) and injected intravenously at a rate of 20 drops per minute (4 μg / min). If the effect is insufficient, it is necessary to increase the rate of administration to 10-20 μg / min. With an increase in systolic blood pressure up to 100 mm Hg. Art. the infusion rate can be reduced to 1-3 mcg / min. The drug can be administered for a long time (over several hours). When using angiotensinamide, bradycardia may increase, in this case, it is necessary to administer 0.5-1 ml of a 0.1% atropine solution subcutaneously.

With the development of heart failure, cardiac glycosides are traditionally recommended (strophanthin - intravenously drip of 0.3-0.5 ml of 0.05% solution per 300 ml of 5% glucose solution). However, it should be borne in mind that in hypothyroidism (especially in hypothyroid coma), the myocardium is hypersensitive to cardiac glycosides and glycosidic intoxication can easily develop.

In order to improve metabolic processes in the myocardium, intravenous administration of 50-100 mg of cocarboxylase (vitamin B1 coenzyme), 0.02 g of pyridoxal phosphate (vitamin B6 coenzyme), 2 ml of 0.5% lipoic acid solution, 5 ml of 10% mildronate solution is useful.

6. Elimination of severe anemia

With severe anemia, when the hemoglobin content drops to 50 g / l or even lower, it is necessary to transfuse the erythrocyte mass, in the most severe cases - repeatedly.

Relief of anemia reduces hypoxia of organs and tissues, including the brain, and promotes the fastest recovery from coma.

Treatment with thyroid medications also helps to reduce anemia.

7. Elimination of hypothermia

A progressive decrease in body temperature significantly worsens the prognosis. To warm the patient, it is necessary to wrap up with blankets, and also slowly raise the room temperature (by 1 "C per hour, but not higher than 25 ° C). Active warming of the patient with heating pads, lamps, hot water bottles is not recommended, because this is peripheral vasodilation. , which impairs the blood flow in the internal organs and causes the development of collapse.In the future, as the effect of thyroid hormones manifests itself, the body temperature gradually rises.

8. Treatment of infectious and inflammatory diseases and elimination of other causes of hypothyroid coma

To combat infectious and inflammatory diseases, treatment with broad-spectrum antibiotics is carried out. However, the doses of antibiotics before the patient comes out of coma can be reduced, given the slowdown in metabolic processes and the elimination of antibiotics from the body.

In order to avoid the development of hypothyroid coma, hypothyroidism should be diagnosed in a timely manner, a euthyroid state should be achieved, a thorough clinical examination of the patient and lifelong replacement therapy should be carried out, and the causes that can lead to the development of coma should be eliminated in a timely manner.

In the absence of timely and adequate treatment, patients in a hypothyroid coma die mainly from respiratory and heart failure, in some cases from cardiac tamponade due to a large accumulation of effusion in the pericardial cavity. 40% of patients die even with promptly initiated vigorous therapy.

End of form

4. ADRENAL DISEASES

4.1. Addison crisis (acute adrenal insufficiency) is a formidable complication of Addison's disease.

The reasons its developments are as follows:

* acute infections,
* intoxication,
* surgical interventions,
* pregnancy,

insufficient treatment for Addison's disease.

Due to the lack of cortisol and aldosterone in the body, dehydration of the body, collapse, impaired renal function, severe hypoglycemia, etc., develop, which mainly determine the clinical manifestations of this severe complication. An addisonic crisis develops more often gradually, less often - acutely (within a few hours). With the gradual development of the Addison crisis, the symptoms of the

disease increase over several days or even weeks. Gradually, general weakness increases, appetite decreases, pigmentation increases, rapid weight loss is noted, abdominal pain often occurs, sometimes simulating acute diseases of the abdominal organs. Nausea appears, often the smell of acetone from the mouth, there is indomitable vomiting, diarrhea, which increase dehydration. The latter is expressed by a decrease in skin turgor and intraocular pressure, a sharp drop in blood pressure, thickening of blood, etc. The phenomena of acute cardiovascular failure are increasing. In the absence of infection, the body temperature is lowered. Clonic seizures and meningeal syndrome often occur,

Laboratory... Lymphocytosis and eosinophilia are noted in the blood. In some cases, secondary normocytic anemia is observed. ESR is reduced, and in the presence of an active tuberculous process as an etiological factor of chronic adrenal insufficiency, it is increased. Often (with a severe form of the disease) there are hyponatremia, hypochloremia, hyperkalemia. The content of ACTH in the blood is increased by the mechanism of "feedback", and cortisol, fasting blood sugar is reduced. Excretion of 17-OCS in the urine is reduced. Indicators of the glucose tolerance test after glucose load are flat, with a pronounced hypoglycemic phase by the 3rd hour after exercise, hypoalbuminemia, hyperglobulinemia, and a tendency to hypocholesterolemia are often noted. In idiopathic Addison's disease, an increase in the titer of antibodies to the adrenal tissue is detected by an indirect immunofluorescent method. Excretion of potassium in the urine is reduced, sodium and chloride - increased. The content of aldosterone, 17-KS and 17-OCS in the urine is reduced. With a severe form of the disease, a decrease in basal metabolism is noted.

Addison crisis treatment includes primarily: corticosteroid replacement therapy,
activities aimed at combating dehydration,
* electrolyte disturbances,
* collapse,
* hypoglycemia,
* concomitant infection.

During the first 4-6 hours, 100 mg of water-soluble hydrocortisone in 500 ml of 5% glucose solution or isotonic sodium chloride solution is injected intravenously. At the same time, 75-100 mg of hydrocortisone is injected intramuscularly, followed by repeated injections of hydrocortisone intravenously or intramuscularly at 50-100 mg every 4-6 hours, depending on the patient's condition. On the first day and according to indications on the second day, intravenous drip of 100-400 mg of hydrocortisone or 30-90 mg of prednisolone is prescribed. The daily dose of hydrocortisone is usually 300-600 mg, but may be higher if necessary. In the following days, with an improvement in the patient's condition and an increase in systolic blood pressure to 115-120 mm Hg. Art., and diastolic up to 70 - 80 mm Hg. Art. the dose of glucocorticoids is gradually reduced and hydrocortisone is prescribed only intramuscularly at 25 mg 4 times a day. Depending on the patient's condition, they are gradually transferred to oral therapy (prednisone, dexamethasone, etc.). In combination with the indicated treatment for low blood

pressure, a 0.5% oily solution of DOXA is prescribed in a dose of 1-2 ml (5-10 mg per day) intramuscularly. The indication for increasing the dose of glucocorticoids is severe tachycardia in combination with low systolic (below 100 mm Hg) and diastolic pressure (below 60 mm Hg) .To combat dehydration and collapse, intravenous or intramuscular drip injection is prescribed 2-3 l 5% glucose solution in isotonic sodium chloride solution with the addition of 50 ml of 5% ascorbic acid solution and 4-6 ml of cordiamine. With a sharp drop in blood pressure, along with hydrocortisone and DOXA, 1-3 ml of a 0.1% solution of epinephrine, or a 0.2% solution of norepinephrine, or 1-2 ml of a 1% solution of mezaton are added to the dropper. In case of indomitable vomiting, 5-10 ml of 10% sodium chloride solution is injected intravenously to compensate for the electrolyte deficiency. When indicated, antibiotic therapy is performed.

4.2. Catecholamine crisis

Catecholamine crisis Is a pathological condition caused by a massive release of catecholamines by a hormone-active tumor - pheochromocytoma, and accompanied by a paroxysmal increase in blood pressure that can cause life-threatening cardiovascular complications (pulmonary edema, myocardial infarction, acute cerebrovascular accident, etc.). The incidence of such tumors varies from 1 to 3 cases per 10,000 population, 1 case of pheochromocytoma accounts for approximately 1000 patients with arterial hypertension (Dedov I.I., Melnichenko G.A., Fadeev V.V., 2000).

Etiology. The reason for the development of catecholamine crises is the presence in the patient of pheochromocytoma (chromaffinoma) - a tumor consisting of chromaffin cells. These cells are able to secrete catecholamines (adrenaline, norepinephrine and dopamine) and are normally found mainly in the adrenal medulla and sympathetic ganglia.

In most cases, pheochromocytoma is an encapsulated, well-vascularized tumor measuring 1-14 cm and weighing from 1 to 60 g, although much larger neoplasms have been described. It should be noted that there is no clear relationship between the intensity of catecholamine crises and tumor size.

Usually pheochromocytoma is benign and is localized in one of the adrenal glands, more often in the right. Multiple tumors located outside the adrenal medulla are found in no more than 10% of cases. Malignant tumors from chromaffin tissue (pheochromoblastomas) occur in 6-15% of cases, mainly in pediatric patients. From a histological point of view, pheochromocytomas and pheochromoblastomas are very similar, and the conclusion about the malignancy of the tumor is made mainly by the presence of invasive growth and metastases.
With extra-adrenal localization, pheochromocytoma is most often found at the location of the sympathetic ganglia of the abdominal cavity and the aortic ganglion of Zuckerkandl. Even more rare cases of its localization in the organs of the mediastinum, cranial cavity, bladder, etc. are described.

The hormonal activity of pheochromocytoma is unstable, and for a long time (sometimes up to several months) this tumor can be in a state of functional rest. In

this case, the patient may not be bothered by anything. However, periodically, under the influence of provoking factors, and sometimes spontaneously, for no apparent reason, a massive release of catecholamines occurs from the tumor tissue, leading to the development of characteristic symptoms of a catecholamine crisis.

The reasons... Most often, the development of a crisis is triggered by the following factors:

1) physical stress;

2) fear or emotional overexcitement;

3) prolonged fasting;

4) plentiful food intake;

5) rough palpation or abrupt change in body position;

6) hypothermia;

7) alcohol intake;

8) taking certain medications (insulin, cardiac glycosides, sympathomimetics, histamine, morphine, fentanyl, etc.);

9) eating certain foods (citrus fruits, cheese, chocolate, mustard, red wines);

10) urination (if localized in the wall of the bladder).

Pathogenesis... Massive release of catecholamines, activates the adrenergic receptors of the heart and blood vessels. As a result, tachycardia appears, cardiac output and peripheral vascular resistance increase, and blood pressure rises significantly (up to 250/130 mm Hg and more). There is an overload of the heart with pressure, which can cause acute left ventricular failure with the development of pulmonary edema or severe myocardial ischemia, up to the development of its non-coronary necrosis. For obvious reasons, especially severe consequences are observed in patients with concomitant ischemic heart disease and arterial hypertension. A significant increase in blood pressure can lead to the development of acute left ventricular failure, pulmonary edema, acute myocardial infarction, hemorrhagic stroke and other cardiovascular pathology.

The positive batmotropic effect of catecholamines provokes the development of a variety of cardiac arrhythmias, from frequent extrasystoles to paroxysmal tachycardia and ventricular fibrillation, followed by asystole. But as a result of a pronounced overload of the myocardium, any disturbances in rhythm and conduction are possible.

The pronounced activation of the autonomic nervous system leads to the appearance of a shallow tremor, profuse sweating, and pallor of the skin.

With a protracted (more than 2-3 hours) course of the crisis, there is an accumulation of metabolites of catecholamines with the development of a "rebound" adrenergic imbalance in the activity of alpha and beta adrenergic receptors. As a result, the phenomenon of uncontrolled hemodynamics may

develop, manifested by abrupt changes in blood pressure, which are refractory to drug exposure (catecholamine shock).

The appointment of beta-blockers can also lead to adrenergic imbalance. Blockade of beta-adrenergic receptors with preserved sensitivity of alpha-adrenergic receptors leads to increased arterial spasm, an increase in peripheral vascular resistance and a further paradoxical increase in blood pressure.

Catecholamines are rapidly metabolized, so the duration of a catecholamine crisis in typical cases ranges from several minutes to 1 hour. However, a longer course of the crisis is also possible - up to 2-3 days and even one week.

Clinic. The frequency of catecholamine crises varies from one attack within a few months to 10-15 during the day. When analyzing anamnestic data, it is often possible to notice that as the disease progresses, the frequency of crises increases. The development of a crisis can be both with initially normal blood pressure, and against the background of its previous persistent increase.

In typical cases, the catecholamine crisis begins suddenly after exposure to the above provoking factors or for no apparent reason. A pronounced (more than 200/100 mm Hg) increase in blood pressure in combination with a triad of symptoms, the sensitivity and specificity of which exceeds 90% is considered classic:

- headache;
- sweating, up to torrential sweat;
- tachycardia.

In the absence of these symptoms, the diagnosis of a catecholamine crisis seems unlikely. In addition, there may be observed: general weakness, nausea, dizziness, vomiting. Often visual impairment (up to amaurosis), anxiety, fear, irritability. On examination, attention is drawn to the pallor of the skin, small-sweeping tremors of the body and limbs. Possible pain in the abdomen and heart, changes in peripheral blood (leukocytosis, lymphocytes, eosinophilia), hyperglycemia, the appearance of protein in the urine. With the development of cardiovascular complications, typical symptoms of pulmonary edema, acute myocardial infarction, cerebral stroke, cardiogenic shock, etc. appear.

With a protracted course of catecholamine crisis, it is possible to develop catecholamine shock with erratic changes in blood pressure, refractory to traditional vasopressor therapy.

The end of the crisis occurs as quickly as its onset. Blood pressure decreases to the initial level, the pallor of the skin is replaced by its redness, profuse sweating and salivation are possible. After a crisis, there is usually a copious flow of urine with a low specific gravity. For a long time after the crisis, weakness, a feeling of "weakness" persists.

When examining a patient with a catecholamine crisis, the following clinical syndromes can be distinguished.

1. Syndrome of catecholamine arterial hypertension:

* rapid pronounced increase in blood pressure, mainly due to * its systolic values;
*headaches;

• dizziness;
• flashing "flies" before the eyes;
• deterioration of vision, up to amaurosis.

2. Syndrome of catecholamine heart damage:

* cardialgia and (or) anginal pain;
  • tachycardia (sometimes in violation of conduction or sinus node work - bradycardia);
  • cardiac arrhythmias (sinus tachycardia with polytopic extrasystole);
  • diffuse metabolic disorders in the myocardium, signs of subendocardial ischemia, damage and systolic overload (negative T wave, depression of the ST segment, etc.).

3. Syndrome of hyperactivity of the autonomic nervous system:

• severe sweating;
• shallow tremor;
• dilated pupils;
• anxiety, fear, psychomotor agitation.

4. Abdominal syndrome:

• abdominal pain without clear localization and connection with food intake;
• dyspeptic symptoms (nausea, vomiting).

5. Syndrome of secondary disorders of carbohydrate metabolism:

• hyperglycemia;
• glucosuria (with prolonged crisis).

6. Syndrome of hypermetabolism:

• recent history of weight loss despite normal diet and physical activity;
• subfebrile body temperature.

7. Hematological syndrome:

• leukocytosis;
• lymphocytosis;
• eosinophilia.

8. Kidney dysfunction syndrome:

• proteinuria, cylindruria;
• transient polyuria after the relief of the crisis.

9. Syndrome of possible cardiovascular complications:

• pulmonary edema
• cardiogenic shock;
• acute myocardial infarction;
• acute disorders of cerebral circulation, etc.

To confirm the catecholamine etiology of hypertensive crisis, the most informative study is the excretion of adrenaline, norepinephrine and their metabolites (metanephrine, normetanephrine, vanillyl mandelic acid) in a 3-hour portion of urine collected after the crisis. With a catecholamine crisis, these indicators are many times higher than normal values.

Normal excretion of catecholamines and vanilyl mandelic acid in a 3-hour urine sample

Investigated substances	Excretion, ng / min
Norepinephrine	**10-60**
Adrenalin	**2-20**
Vanillyl mandelic acid	**3-23**

Diagnosis can also be verified by determining the daily excretion of catecholamines, as well as the levels of adrenaline, norepinephrine and their metabolites in the blood plasma. An increase in the level of adrenaline in the blood plasma above 300-500 pg / ml (the norm is up to 100 pg / ml), of norepinephrine - above 1500-2000 pg / ml (the norm is up to 500 pg / ml) is of diagnostic value. However, when these studies are carried out outside the catecholamine crisis, false negative results can be obtained.

Existing provocative pharmacological tests (with histamine, tyramine, glucagon, metoclopramide, etc.) are potentially dangerous and impracticable against the background of a catecholamine crisis. A certain diagnostic value in a patient with a catecholamine crisis may have tests with alpha-blockers (phentolamine, tropafen, prazosin), which are both diagnostic and therapeutic measures. Intravenous bolus of 5 mg of phentolamine or 1-2 mg of tropafen is administered and the change in blood pressure is monitored for 5-10 minutes. The test is considered positive with a decrease in systolic and diastolic blood pressure by 40 and 25 mm Hg. Art. respectively.

Topical diagnosis of pheochromocytoma is carried out using computed tomography or magnetic resonance imaging. At the initial stages of diagnosis, useful information can be obtained using ultrasound of the adrenal glands. For extra-adrenal tumor localization or the search for metastases, it is useful to conduct a radioisotope scan with radioactive iodine-labeled metaiod-benzylguanidine.

It should be noted that in the presence of clinical symptoms of a catecholamine crisis, treatment begins even before laboratory and instrumental confirmation of this diagnosis is obtained.

Differential diagnosis. When discussing the differential diagnosis of a catecholamine crisis, it should be borne in mind that pheochromocytoma has a reputation as a "great pretender" for a reason. Catecholamine crises can mimic a wide variety of surgical, urological, obstetric-gynecological and neuropsychiatric pathologies. Against the background of a crisis, it is possible to erroneously assume the presence of a perforated stomach ulcer, acute intestinal obstruction, acute cholecystitis and pancreatitis, toxicosis of pregnant women, etc. sweating, you should always remember about the possibility of a pheochromocytoma.

In therapeutic practice, catecholamine crises most often have to be differentiated from arterial hypertension. At the same time, the most difficult thing is to reasonably suspect the presence of pheochromocytoma as a cause of a periodic

increase in blood pressure in a patient. The idea that we are dealing with a catecholamine crisis is suggested by the following features of the course of arterial hypertension:

- rapid onset of crises and their rapid self-relief;

* thin constitution of a patient with arterial hypertension;

- connection of a hypertensive crisis with the above provoking factors;
- paradoxical increase in blood pressure in response to traditional antihypertensive therapy (beta-blockers, ganglion blockers, dibazol, etc.);
- relatively young age of the patient and small (less than 2 years) duration of the disease.

Purposeful laboratory and instrumental examination (determination of catecholamines and their metabolites in blood and urine, ultrasound, computed and magnetic resonance imaging of the adrenal glands) allows to confirm or reject the diagnosis of pheochromocytoma.

Often, catecholamine crises have to be differentiated from diencephalic disorders - cluster crises that arise as a result of a sharp imbalance in the tone of cerebral vessels and are accompanied by paroxysmal headaches, moderate increases in blood pressure, psychopathological and vegetative symptoms (pallor, sweating, tremors, pain in the abdomen, vomiting , transient visual impairment up to amaurosis). Cluster crises usually occur in men aged 20-30 years, when analyzing anamnestic data, a family-hereditary burden is revealed for this symptomatology. On average, diencephalic crises are characterized by a longer course (several hours or days) than catecholamine crises. The final phase of the crisis is also often significantly delayed (for hours or even days).

Sometimes in patients with catecholamine crises, the presence of thyrotoxicosis is suspected, for which an acceleration of metabolism and hypersympathicotonia are also typical. An ultrasound of the thyroid gland and the study of thyroid hormones - free thyroxine and TSH help to clarify the diagnosis.

Speaking about the differential diagnosis of catecholamine crises, it should be emphasized once again that pheochromocytoma is a rather rare pathology. Therefore, the assumption about the presence of catecholamine crises in a patient is made much more often than this pathology is found in reality.

Treatment.

1. Emergency hospitalization in a specialized department.

2. Mode. The patient is assigned to bed rest, the head end of the bed is raised in order to create conditions for orthostatic lowering of blood pressure.

3. Blockade of alpha-adrenergic receptors. Every 5 minutes, until the crisis stops or blood pressure drops, alpha-blockers are slowly injected intravenously:

- phentolamine (regitin) 2-5 mg of active substance per 10 ml of isotonic solution;
- tropafen 1-2 ml of 2% solution per 10 ml of isotonic sodium chloride solution.

After lowering and stabilizing blood pressure, alpha-blockers are administered intramuscularly in the same doses every 2-4 hours. Dosages and frequency of administration in a particular patient are determined by the dynamics of blood pressure. Parenteral administration of adrenergic blockers continues throughout the first days of therapy, then they switch to oral administration of phentolamine in doses of 25-50 mg (1-2 tablets) every 3-6 hours for the entire period of time until the surgical removal of this tumor.

4. Vasodilators. To lower blood pressure with a catecholamine crisis, sodium nitroprusside may be administered. 100 mg of this drug is dissolved in 500 ml of 5% glucose solution and injected intravenously under the control of blood pressure.

5. Blockade beta-adrenergic receptors. If severe tachycardia persists against the background of treatment with adrenergic blockers (only after an effective blockade of alpha-adrenergic receptors!), Treatment can be supplemented by the appointment of beta-blockers. They start with propranolol preparations for parenteral administration (obzidan, inderal): 1-2 ml of a 0.1% solution is diluted in 10 ml of isotonic sodium chloride solution and injected intravenously slowly every 5-10 minutes under the control of blood pressure and heart rate. The total dose of propranolol should not exceed 5-10 mg. After elimination of tachycardia, they switch to taking propranol preparations orally at 20-40 mg 3-4 times a day.

6. Emergency surgery. It is indicated in the absence within 2-3 hours of the effect of correctly conducted conservative treatment. The need for surgery is justified by the high risk of developing catecholamine shock with a protracted course of the crisis.

7. Control of the volume of circulating blood. After blockade of alpha-adrenergic receptors, a pronounced decrease in blood pressure is possible, which, for obvious reasons, cannot be corrected by the introduction of sympathomimetics. Correction of arterial hypotension is carried out by increasing the volume of circulating blood due to intravenous drip infusion of polyglucin, rheopolyglucin and other plasma-substituting solutions. To avoid volume overload of the heart, transfusion of blood substitutes is carried out under the control of central venous pressure or (if technically possible) the pressure of pulmonary artery wedging.

5. DISEASES OF THE PIPOPHYSIS

5.1. Hypopituitary coma

Hypopituitary (pituitary) coma- exacerbation of chronic insufficiency of the adenohypophysis with panhypopituitarism. It is rare in children.

Etiology. The most common cause of pituitary insufficiency in adults is postpartum necrosis of the anterior pituitary gland due to profuse bleeding and shock. Panhypopituitarism develops as a result of tumors, cysts, trauma, infections, inflammation in the hypothalamic-pituitary region, burns, bleeding, congenital malformation of the pituitary gland and primary damage to the hypothalamus, with insulin shock, X-ray therapy and isotope irradiation of the pituitary gland, hypophysectomy.

The reasons coma development is varied. Coma can be triggered by hypothermia, physical or mental trauma, surgery, anesthesia, infection, careless use of diuretics, acetylsalicylic acid, insulin, barbiturates and other factors.

Pathogenesis... The pathogenesis of panhypopituitarism is based on insufficiency of the adenohypophysis with a sharp decrease in the production of tropic hormones and the function of peripheral endocrine glands. Adaptive responses to stressful situations do not develop. The pathogenesis of hypopituitary coma includes a complex set of metabolic disorders characteristic of hypoglycemic, hypothyroid coma, acute adrenal insufficiency caused by pronounced polyhormonal insufficiency.

Clinic. A hypopituitary coma develops gradually. In patients with panhypopituitarism, weakness, weakness, chilliness increase, nausea, vomiting, headache, dizziness, and constipation appear. There is no appetite, there is a loss of body weight. The patient is indifferent, inhibited, speech is quiet, slow, incoherent. Along with depression, there are bouts of irritability, excitement with hallucinations, followed by drowsiness, deafness. Drowsiness progresses, turning into a stupor and coma. Amimatic, indifferent face, sharp, waxy pallor. The skin is dry, thin, as if transparent. Hair is dry, sparse, brittle, dull. The body temperature is sharply reduced. Heart sounds are very attenuated. Bradycardia, severe, severe hypotension. Breathing is shallow, slow, arrhythmic, rare. Diuresis is reduced. There may be convulsions due to hypoglycemia.

* hypothyroid,
* hypoglycemic,
* hyperthermic,
* option with water-electrolyte shifts.

In hypopituitary coma, a low level of all tropic hormones (corticotropin, thyrotropin, somatotropin, gonadotropins, etc.), T3, T4, cortisol, 17-KS, 17-OCS, leukopenia, lymphocytosis, normochromic anemia, eosinopenia, hypoproteinemia are found in the blood. In addition, the hypothyroid coma is characterized by severe hypercholesterolemia, the predominance of signs of adrenal pathology - hyponatremia, hypochloremia, hyperkalemia, hypoglycemia, an increase in the content of urea and residual nitrogen. Differential diagnostic criteria for hypopituitary coma, in contrast to coma caused by insufficiency of individual endocrine glands, are signs of combined damage to a number of endocrine glands.

Treatment hypopituitary coma should be urgent and consist in the appointment of intravenous glucocorticoids (hydrocortisone - from 100 to 250 mg, prednisolone - 25-50 mg) under the control of blood pressure, glycemia, and the general condition of the patient. With severe thyroid insufficiency, thyroid hormones are indicated, preferably triiodothyronine at an initial dose of 10-20 μg / day intravenously (levo-triiodothyronine) or through a gastric tube under the control of pulse rate, respiration, blood pressure, ECG data, rectal temperature. If necessary, the dose of triiodothyronine can be increased. In parallel with glucocorticoids, DOXA is prescribed (1-2 mg / kg of a 0.5% solution intramuscularly). In case of intractable collapse, it is justified to use a 1% solution of mezaton or 0.2% solution of

norepinephrine (0.1-0.2-0.5 ml) intravenously. For the correction of water-electrolyte disorders, hypoglycemia is administered intravenously drip 5-10% glucose solutions, saline solutions, isotonic sodium chloride solution. Persistent hyponatremia (sodium content 115 mmol / l and below) requires the appointment of 10-20 ml of 10% sodium chloride solution intravenously. Depending on the level of glycemia, additional intravenous administration of 40% glucose (20-40 ml) is possible. Correction of violations of CBS is carried out with a 4% solution of sodium bicarbonate. Transfusion of plasma substitutes is used. In the presence of symptoms of diabetes insipidus, pituitrin -0.6-0.8 ml is used subcutaneously or intramuscularly 1-2 times a day). To improve oxidative processes, cocarboxylase is prescribed intravenously (100-200 μg), ascorbic acid (5% solution - 5-10 ml), pyridoxine (5% solution, 2 ml), cyanocobalamin (200-500 μg), ATP. Apply oxygen therapy with humidified oxygen, antibiotics. With severe hypothermia, the patient is warmed. All the necessary measures are taken to combat cardiovascular and respiratory failure. As the patient's condition improves, consciousness is restored, the temperature rises, they give sweet drinks, fruit drink, and gradually reduce the amount of transfused liquid. When blood pressure is normalized, DOX is canceled, hydrocortisone is injected intramuscularly, the dose is reduced. Starting from the 3rd day, with further improvement of the patient's condition, laboratory parameters, infusion therapy is stopped, the dose of hydrocortisone, troiodothyronine is reduced by half. Gradually they switch to oral glucocorticoids, the dose of triiodothyronine is reduced to 10-20 μg / day. After removing the patient from a hypopituitary coma, an adequate maintenance dose of glucocorticoids is selected individually, thyroid and sex hormones) in the form of replacement therapy, carrying out their correction in stressful conditions. The prognosis for hypopituitary coma is determined by the timeliness of diagnosis and treatment, and its adequacy. Mortality is up to 25%.

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SITUATION TASKS

1) Patient K., 32 years old, while on a business trip, noted the appearance of weakness, thirst, dry mouth, headache, drowsiness. Soon, nausea, vomiting, and abdominal pains, growing in intensity, joined. With suspicion of "acute abdomen", the patient was urgently taken to the surgical department. Loss of consciousness occurred in the emergency room. Breathing is noisy, the skin is dry, cold, muscle hypotonia, the smell of acetone from the mouth.

1. What state should you think about?
2. What are the urgent actions?

2) Patient A., 42 years old, suffers from diabetes mellitus, recently began to drink alcohol and stopped administering insulin.
On examination: the consciousness is confused, the skin is dry, pale, breathing is deep, noisy of the Kuss-Mawl type, with a pungent smell of acetone from the mouth. the eyeballs are soft, the pupils are constricted, muscle hypotonia, soreness of the abdomen.

1. Your diagnosis.

2. Make a differential diagnosis with hyperosmolar coma.

3) A patient who had an elevated fasting blood sugar (15.8 mmol / l) the day before underwent a glucose tolerance test. After 2 hours after that, the patient lost consciousness.

1. What complication did the patient develop and what caused it?
2. Did the doctor make a mistake? If so, which one?

4) Patient K., 17 years old, unconscious. According to the mother, she suffers from a severe form of type 1 diabetes mellitus, does not follow the diet. In the morning, the usual dose of prolonged-release insulin was given.
Objectively: the skin is moist, the pupils are wide, the eyeballs are tense, the muscle tone is increased, the reflexes are revitalized. Breathing is even, without the smell of acetone in the exhaled air. Pulse 92 beats per minute, BP - 130/80 mm Hg.

1. What is your diagnosis?
2. Perform differential diagnosis with hyperglycemic ketoacidotic coma.
3. What research is needed to differentiate these conditions?

5) Patient D., 28 years old, suffers from type 1 diabetes mellitus, a severe form. In the evening after work, he drank a large amount of alcohol, after which he suddenly lost consciousness and was taken to the hospital.
Objectively: the skin is moist, the muscle tone is increased, sometimes there are cramps. Breathing is even, pulse is 104 beats per minute, blood pressure is 90/60 mm Hg.

1. What is your diagnosis?
2. What are the urgent actions?

6). Delivered unconscious patient. According to relatives, he is sick with diabetes mellitus and receives insulin therapy.
Objectively: the skin is moist, the muscle tone is increased, agitated. In blood sugar - 2.5 mmol / l, in urine - sugar and acetone are negative.

1. What is your diagnosis?
2. Treatment of this patient?

7) Patient O., 54 years old, suffering from mild diabetes mellitus and concomitant hypertension, took hypothiazide for a long time without medical supervision. Against this background, sharp girdle pains in the epigastric region, indomitable vomiting suddenly appeared, and the general condition worsened. Thirst intensified, dry mouth, confusion of consciousness, at times epileptiform

convulsions soon joined, breathing became rapid, superficial, without the smell of acetone in the exhaled air.

1. What state should you think about?
2. What are the emergency activities?

8) A 56-year-old patient was delivered to the emergency room in an unconscious state. Despite the fact that the blood sugar reached 60.5 mmol / l, there is little urine and no acetone in it. Periodically, the patient has convulsions and signs of severe dehydration of the body.

1. What disease and its complication should you think about?
2. What is the diagnosis and emergency treatment?

9) Patient M., 62 years old, for diabetes mellitus and obesity took dibotin at a dose of 150 mg per day for a long time. After suffering pneumonia, she began to notice pain in the muscles, behind the breastbone, apathy, anorexia, insomnia. I did not go to doctors. Soon there was nausea, vomiting, delirium with excitement. In this regard, the patient was taken to the clinic.
On admission: no consciousness, dry skin, Kuss-Mawl breathing, no acetone odor. In the blood: sugar - 9.4 mmol / l, ketone bodies - 130 μmol / l, pH - 7.0, traces of sugar in urine, no acetone.

1. What state should you think about?
2. Differentiate with ketoacidotic coma.
3. Assign specific research.

10) Patient M., 52 years old, suffering from diabetes mellitus and concomitant chronic bronchitis for 5 years receives adebit at a dose of 200 mg per day. After physical overstrain, the patient's condition worsened at night. There were nausea, vomiting, pain in the muscles and behind the breastbone. Soon abdominal pains, motor excitement and delirium followed, followed by drowsiness and loss of consciousness.
When examined by an ambulance doctor: breathing like Kuss-Mawl, there is no smell of acetone in the exhaled air, the skin is dry, acrocyanosis.

1. How to assess this condition?
2. What are the urgent actions?

11) Patient B., 22 years old with diffuse toxic goiter without adequate preoperative preparation underwent subtotal thyroidectomy. A few hours later, the temperature increased, hyperemia of the face and trunk appeared, increased sweating, tachycardia (118-126 beats per minute). These changes were not judged correctly by the physician. The next day, hyperthermia reached 40 * C, tachycardia - 160 beats per minute, motor restlessness, shortness of breath, lethargy, delirium appeared.

1. What state should you think about?
2. What are the urgent actions?

12) Patient R., 25 years old, was operated on for diffuse toxic goiter. A month before the operation, she was treated in the endocrinology department, took Mercazolil. She was discharged home in satisfactory condition. I did not take treatment at home. Before the operation, the pulse rate was 98 beats per minute, the blood pressure was 130/60 mm Hg. on the 2nd day after the operation, the condition deteriorated sharply. The patient is agitated, complains of palpitations, sweating, twice had vomiting, diarrhea. Pulse 170 per minute, arrhythmic (atrial fibrillation). BP - 150/60 mm Hg. Art. Heart sounds are loud. Breathing is vesicular. The abdomen is soft, diffuse soreness in the intestines .. the lower edge of the liver protrudes 4 cm from under the costal arch. Temperature 40 * C.

1. What is your diagnosis?
2. The reason that caused a sharp deterioration in the condition.
3. Which of the drugs should be prescribed?

13) Which of the following drugs should be prescribed to a patient with a thyrotoxic crisis?

1.reserpine,
2.Lugol's solution (per os or IV),
3.diiodotyrosine,
4.triiodothyronine,
5. L-thyroxine,
6.prednisolone or hydrocortisone (per os or IV),
7.strophanthin,
8.beta-blockers,
9.Sodium chloride saline and IV glucose,
10. mercazolil,
11.amidopyrine,
12.cocarboxylase,
13.ascorbic acid
14. seduxen,
15.hemodesis.

14) Patient T., 38 years old, underwent extirpation of the thyroid gland for cancer. On the day of the operation, parasthesias in the fingers, muscle twitching, and stiffness of the facial muscles appeared. Soon there were tonic cramps in the muscles of the forearm, hands (obstetrician's hand), trismus of the facial muscles, speech became slurred, breathing noisy with difficult exhalation, the face was hyperemic, hyperhidrosis. Consciousness is preserved.

1. What condition did the patient develop?
2. What are the urgent actions?

15) Patient R., 31 years old, was operated on for diffuse toxic goiter. Two weeks after the operation, the patient developed short-term, lasting 1-2 minutes,

convulsive contractions of the arm muscles, accompanied by facial numbness. Convulsions occurred 1-2 times a day during work.
Objectively: the pulse is 72 beats per minute, the blood pressure is 120/70 mm Hg. From the side of internal organs - without deviation from the norm. Symptom Trousseau, Khvostek 1 - positive.

1. Diagnosis?
2. What laboratory methods of research are necessary to carry out to establish a diagnosis?
3. Which of the drugs should be administered to stop the attack and how much?

16) Patient Z., 42 years old, suffering from Addison's disease, stopped taking prednisolone for 2 months. Complains of a sharp general weakness during the day, a sharp weakness, dizziness, abdominal pain, frequent vomiting, which does not bring relief, a decrease in body weight during this time by 14 kg.
Objectively: a serious condition, confused consciousness. Height 178 cm, weight 47 kg. Intense tanned skin. BP - 70/40 mm Hg, pulse - 120 beats per minute. Heart sounds are muffled at all points. The abdomen is somewhat tense, painful on palpation in the epigastric and umbilical regions.

1. What complication did the patient develop?
2. What is the treatment tactics?

17) Patient R., 33 years old, complains of recurrent attacks of severe headache, accompanied by palpitations, sometimes shortness of breath. More often, an attack occurs during physical exertion, washing clothes, washing floors. The called "ambulance" found an increased blood pressure - 220/110 mm Hg. The patient was admitted to the hospital. During her stay in the hospital, there was twice a similar attack with an increase in blood pressure to 250/120 mm Hg. Has been ill for about a year. At the beginning, the attacks were disturbed occasionally - once every 1-2 weeks, recently they have become more frequent up to 1 time in 1-2 days. During an attack in the blood - leukocytosis, hyperglycemia, outside the attack - the norm.
Objectively: height 162 cm, body weight 56 kg. Pulse 82 beats per minute, rhythmic. The left border of the heart is expanded. BP - 135/80 mm Hg. Art. Other data unchanged. General analysis of urine and blood is normal. Isotope renography - the norm, fundus - the arteries are narrowed.

1. What is the presumptive diagnosis?
2. What hormones have a hypertensive and at the same time hyperglycemic effect?
3. What antihypertensive drugs are indicated for pheochromocytoma?

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TEST PROBLEMS

1) Hyperosmolar coma with diabetes develops:
1.in young people
2.in people over 60 years old
3.in people with type 1 diabetes
4.in people with type 2 diabetes
5.on the background of intercurrent infections and dehydration

2) Hypoglycemic coma is characterized by:
1.inhibition
2.excitation
3.dry skin
4.convulsions
5.the smell of acetone from the mouth

3) The main provoking factor of ketoacidotic coma is:
1.the body's loss of fluid
2.surgical intervention
3.pregnancy
4.inadequate insulin therapy

5.concomitant pathology (myocardial infarction, pneumonia, pyelonephritis)

4) In hyperosmolar coma, the osmolarity of the blood exceeds:
1.150 mlosm / l
2.200 mlosm / l
3.250 mlosm / l
4.300 mlosm / l
5.500 mlosm / l

5) To remove from a hypoglycemic coma, you must:
1.v / v physiological sodium chloride solution
2.5% glucose solution IV drip
3.insulin
4.40% glucose solution IV jet
5.adrenaline
6.prednisolone

6) The clinical manifestations of developing diabetic ketoacidosis are:
1.polyuria, polydipsia
2.anorexia
3.nausea, vomiting
4.decrease in body weight
5.all of the above

7) Hyperosmolar coma is characterized by
1.breathing of Kuss Mawl
2.the moisture of the skin
3.decrease in plasma osmolarity
4.decrease in plasma pH
5.polyuria
6.acetonuria

8) The metabolic signs of hyperosmolar coma are:
1.hypernatremia and ketoacidosis
2.ketoacidosis and hyperglycemia
3.hyperglycemia and hyperosmolarity
4.hyperosmolarity and hypernatremia
9) Hyperlactacidemic coma is characterized by:
1.young age
2.hypoxia
3.hyperglycemia 14 mmol / l -33 mmol / l
4.hyperglycemia 9 mmol / l - 14 mmol / l
5.breathing of Kuss-Mawl

6.acetonuria

10) The etiological factors of hyperglycemic coma cannot be
1.glucagon
2.insulin
3.prolactin
4.adrenocorticotropic hormone (ACTH)
5.somatotropic hormone (STH)

11) Diabetic ketoacidotic coma is not typical
1.increase in the level of ketone bodies in the blood
2.increase in blood pH
3.decrease in blood pH
4.pyruvatemia
5.lactacidemia

12) Hypoglycemic coma is characterized by:
1.inhibition
2.excitation
3.dry skin
4.aggression
5.the smell of acetone from the mouth

13) A ketoacidotic coma differs from a hyperosmolar coma based on the definition of all of the above, with the exception of:
1. the smell of acetone in the exhaled air
2. the presence of neurological symptoms
3. glycemia
4. breathing pattern
5. ketone bodies

14) Does not indicate the presence of a hypoglycemic coma
1.low blood sugar
2.the rapid development of coma
3.normal or high blood pressure
4.Even breathing
5.Rare heart rate

15) Treatment for diabetic coma does not include
1.immediate hospitalization and intensive 2.insulin therapy
3.Elimination of disorders of carbohydrate and lipid metabolism
4. Combat dehydration
5.Previous insulin regimen and dose
6. Combat cardiovascular failure

16) Clinical symptoms of hyperlactacidemic coma are manifested by all of the above, except:

1.nausea, vomiting
2.severe arterial hypotension
3.oliguria up to anuria
4.increase in body temperature
5.Kussmaul breathing, shortness of breath

17) Do not use in the treatment of hyperlactacidemic coma

1. intravenous administration of 2-5% sodium bicarbonate solution under control of blood pH and blood potassium level
2. administration of large doses of insulin
3. intravenous administration of 1% methylene blue solution
4. oxygen therapy
5. the introduction of cardiac and vasodilators

18) The initial symptoms of severe hypoglycemia do not appear

1.excitement, aggressiveness of patients with diabetes
2. confusion
3.increased tendon and periosteal reflexes
4.decreased muscle tone
5.Positive Babinsky symptom

19) In diabetic ketoacidosis, infusion therapy begins with intravenous administration:

1.isotonic sodium chloride solution
2.5% glucose solution
3. hypotonic (0.45%) sodium chloride solution
4. reopolyglyukina
5. hemodesis

20) The clinical symptoms of hyperosmolar coma are manifested by all of the above, except:

1.arterial hypertension
2.signs of dehydration
3.various neurological disorders
4.dysfunction of the cardiovascular system
5. shortness of breath

21) The initial dose of intravenous administration of short-acting insulin per hour for diabetic coma per 1 kg of body weight is:

1.100 units / hour
2.50 units / hour
3.25 units / hour

4.2-4 units / hour
5.1-0.2 units / hour

22) A thyrotoxic crisis can develop in all of the listed cases, except for:
1.Undiagnosed toxic goiter
2.operative intervention on the thyroid gland
3.treatment with radioactive iodine upon reaching euthyroidism
4.A sharp withdrawal of antithyroid drugs
5.infections, intoxication, stressful situations

23) Hypothyroid coma can be triggered by:
1.radiopaque diagnostic studies
2.pneumonia
3.the intake of phenobarbital in the usual dose
4.Cooling
5.all of the above

24) For a hypothyroid coma, everything is characteristic, except:
1.severe hypothermia in the absence of infection
2.increasing inhibition of the central nervous system (stupor, coma)
3.progressive bradycardia
4.progressive arterial hypotension
5.hyperglycemia

25) In acute adrenal insufficiency in patients with pain in the abdomen are most characteristic:
1. flatulence
2.vomiting
3.diarrhea
4.tachycardia
5.the fall in blood pressure

26) The manifestations of acute adrenal insufficiency are not refers to:
1.muscular weakness
2.nausea
3.arterial hypotension
4.peritoneal irritation syndrome
5.hypoglycemia

27) Addison crisis is clinically manifested by all of the above, except:
1.sharp dehydration
2.collapse
3.Acute cardiovascular failure

4. arterial hypertension
5.edema

28) With the gradual development of an Addison crisis, all of the listed symptoms develop, except:
1. Increased pigmentation of the skin and mucous membranes
2.A sharp increase in weakness
3.Fast weight loss
4.constipation
5.nausea, vomiting

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