STATE UNIVERSITY
«UZHHOROD NATIONAL UNIVERSITY»
MEDICAL FACULTY N 2
Department of the Physiology and Pathophysiology

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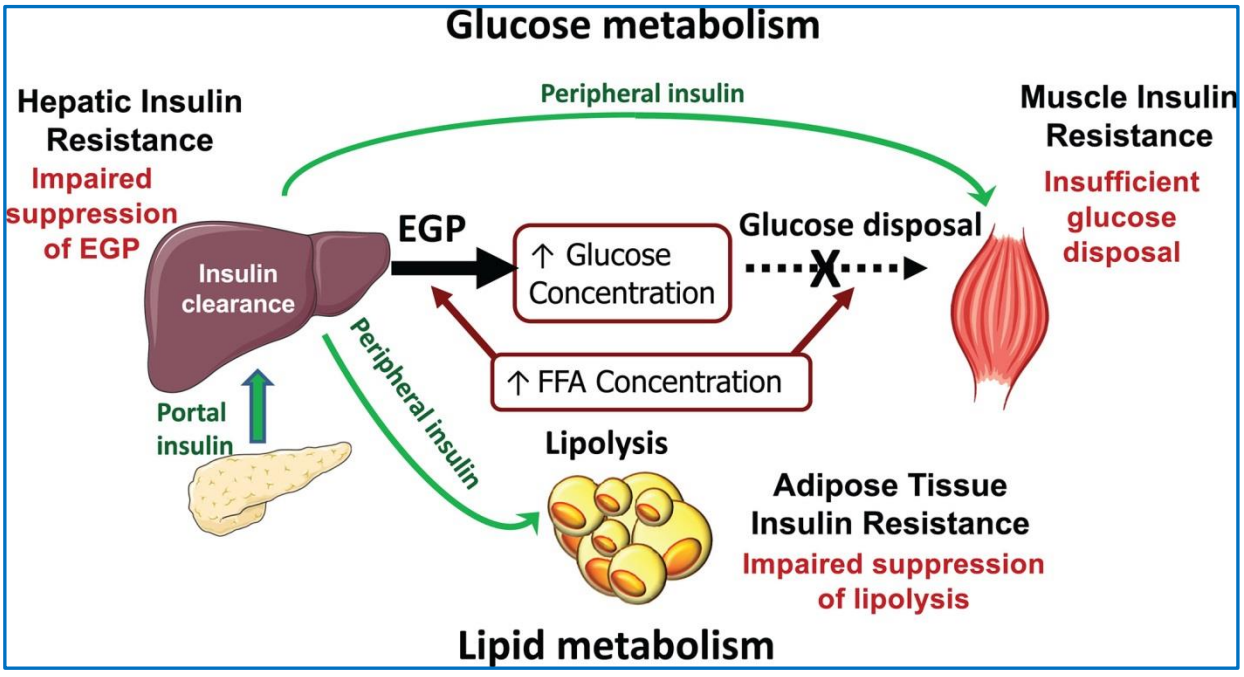
TYPICAL DISORDERS OF METABOLISM

METHODICAL INSTRUCTIONS
for practical classes and self-study on Pathophysiology
for 3rd year students
of medical faculty №2, specialty 222 “Medicine”



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Methodological instructions for practical classes on Pathophysiology for students of the Medical faculty № 2 from the section “Typical Disorders Of Metabolism" have been prepared in accordance with the requirements of the Syllabus on Pathophysiology for students of the medical faculty of higher medical educational institutions of the III-IV levels of accreditation.

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Methodological instruction to practical lesson № 11
Module 1. General pathology

Theme: DISORDERS OF ENERGETIC METABOLISM. CARBOHYDRATE METABOLISM DISORDERS. DIABETES MELLITUS

Student should know:

- Etiologic factors of disorders of energetic metabolism.
- Definition of notion "diabetes mellitus", "insulin resistance".
- Classification of diabetes mellitus.
- Features of Langergan's isles damage. Major symptoms of diabetes mellitus.
- Complications of diabetes mellitus.

Student should be able to:

- Explain mechanisms of disorders of energetic metabolism.
- Explain the etiology of diabetes in the context of general ideas on the etiology of multifactorial disease.
- Analyze metabolism in the pathogenesis of basic types (type 1, type 2) diabetes.
- Analyze the results of glucose tolerance test (practical skill).

LIST OF CONTROL QUESTIONS

1. The energy balance: positive and negative.
2. Basic metabolism. Pathological changes in metabolism: etiology and pathogenesis.
3. Disorders of energy supply cells. Transport of nutrients through cell membranes.
4. Disorders of intracellular catabolic paths.
5. Disorders of cellular respiration. Effect and mechanism of separation oxidation and phosphorylation.
6. Role of energetic disorders in development of cell injury.
7. Malabsorption of carbohydrates, the synthesis, cleavage and deposit of glycogen transport of carbohydrates in the cell. Disorders of the nervous and hormonal regulation of carbohydrate metabolism.
8. The syndrome of hypoglycemia: types, causes, mechanisms. Pathogenesis of hypoglycemic coma.
9. The syndrome of hyperglycemia: types, causes and mechanisms of development.
10. Diabetes. Definition, classification (WHO). Experimental modeling of diabetes.
11. The etiology and pathogenesis of diabetes mellitus type 1. The role of genetic factors and environmental factors in its origin and development. Pathogenesis absolute insulin deficiency, its manifestations and

consequences: disorders of energy, protein, carbohydrate, fat, water and electrolyte metabolism, acid-base status.

12. The etiology and pathogenesis of diabetes mellitus type 2. The role of genetic factors and environmental factors in its origin and development. Variations of relative insulin deficiency in diabetes type 2 (secretory disorders in insulin resistant tissues). Manifestations and consequences of relative insulin deficiency. The concept of the metabolic syndrome.
13. Complications of diabetes. Coma: types, causes and mechanisms of development, manifestations, principles of therapy. Long-term complications (macro, microangiopathy, neuropathy, fetopathiyi etc.), Their general characteristics.
14. Preventing the emergence and development of diabetes. Principles of treatment of diabetes. Prevention of complications.

Metabolism performs the energetic (energy formation) and plastic (synthesis of necessary products) functions.

The dynamics of metabolism is provided by:

• Entry of simple and complex substances (proteins, carbohydrates, lipids, vitamins, electrolytes) with food into the digestive tract.
• Primary splitting of the substances into simpler products with the aid of digestive enzymes.
• Absorption of them into the blood.
• Transport of the substances with the blood to organs and tissues.
• Catabolism (intracellular splitting of substances by intracellular enzymes).
• Formation of intermediate products, which in their turn serve as substrates for various metabolic pathways.
• Anabolism (synthesis of substances for organs).
• Deposition of reserve.
• Final product formation.
• Final product excretion.

Disorders are possible at any stage of metabolic transformation of substances, which is provided with the help of enzymes. The reaction substrate-enzyme is a basis of metabolism. So, metabolism consists in two opposite processes — *anabolism* and *catabolism*, which are connected dynamically. Metabolism disturbances are divided into *acquired* and *hereditary* as well as *increase* and *decrease.*

Basal metabolism is a quantity of energy produced in the organism under standard conditions.

Basal metabolism is increased with hypersecretion of hormones with a catabolic effect — thyroxin, parathyrine, progesterone, somatotropin, vasopressin. Adrenaline stimulates basal metabolism, especially in case of cooling (insulin suppresses trembling). Basal metabolism is increased in tumors of the endocrine glands with hypersecretion of hormones with a catabolic effect.

Basal metabolism reduction accompanies:

• starvation (at the second stage) due to reorganization of metabolism for a more economical use of energy;

• hypoxia at the stage of decompensation.

In patients with endocrine diseases accompanied with hyposecretion of hormones of a catabolic effect the level of basal metabolism is decreased. The examples are the following:

• hypothyroidism;

• hypofunction of the pituitary gland accompanied with a decreased production of thyrotropin and corticotropin (ACTH);

• hypofunction of the sex glands (castration, climax);

• bilateral lesion of the suprarenal glands (more often of tuberculosis genesis as it is in Addison's disease);

• obesity.

Carbohydrates are an important energy source for cells, and for some of them (nervous) carbohydrates are essential. Disorders of carbohydrate metabolism may take place at any point of carbohydrate balance — digestion and absorption, maintenance of the blood sugar level, intermediate metabolism, formation of reserves in the form of glycogen, correlation with other types of metabolism.

Regulation of carbohydrate balance is provided by nervous and hormonal mechanisms as well as by carrier proteins transporting glucose (GLUT) into cells via the cellular membrane.

The following hormones provide the hormonal control of carbohydrate metabolism and elevate the blood glucose level by different mechanisms - adrenaline, glucagon, thyroxin, corticotropin and somatotropin. They stimulate either glycogenosis or glyconeogenesis. *Adrenaline* causes short-term hyperglycemia by stimulating glycogenolysis in the liver. Glucagon activates glycogenolysis, inhibits accumulation of glycogen in the liver, has glyconeogenetic, lipolytic and insulin-stimulating effects. *Thyroxin* stimulates glucose absorption in the intestines, activates liver Phosphorylase and limits organism tolerance to carbohydrates. Hyperfunction of the thyroid glands is characterized by a decreased tolerance to carbohydrates. *Glucocorticoids* (hormones of the zona fasciculata of the adrenal cortex) raise the blood glucose level by glyconeogenesis activation (glucose synthesis from amino acids). They induce the synthesis of matrix RNA, which is responsible for the enzymes of glyconeogenesis formation. They decrease the cellular membrane permeability for glucose and inhibit the rate of hexokinase reaction and hexose-6phosphate formation. *Corticotropin* acts similarly to glucocorticoids because it stimulates their secretion. It activates glyconeogenesis and inhibits hexokinase activity. *Somatotropin* (the hormone of growth produced in the adenopituitary gland) impairs tolerance to carbohydrates, causes hyperglycemia, ensures hyperplasia of the a-cells of the pancreatic islets, stimulates glucagon synthesis, activates liver insulinase, lipolysis and glyconeogenesis (from fatty acids). It is only *insulin* (secreted by pancreatic ß-cells) that decreases the blood glucose level and provides balance.

Insulin insufficiency is metabolic and pathophysiological disorders in the organism caused by insulin amount or function disorders.

Absolute (pancreatic) insulin insufficiency is a result of decreased insulin biosynthesis or secretion. **Relative (extrapancreatic)** insulin insufficiency refers to the situation, when insulin production is normal, but metabolic disturbances and clinical picture are the same as in absolute insulin deficit.

Pathophysiological derangement, which is observed in insulin insufficiency, is a consequence of metabolism disorder. It is necessary to distinguish (a) pathophysiological consequences of insulin insufficiency and (b) pathophysiological consequences of prolonged and incomplete compensation of insulin insufficiency with the aid of drugs that takes place in clinical practice.

Pathophysiological disorders are the following.

• Metabolic acidosis may be decompensated and results in coma development.

• Ketonemia and ketoacidosis cause intoxication and may result in coma development.

• High blood glucose level increases osmotic blood pressure and may cause comatose state.

• Constant loss of glucose in the urine overloads all hormonal mechanisms of blood sugar level support (secretion of glucocorticoids and other contrainsulin hormones). Overloading of hormonal mechanisms leads to their exhaustion.

• Atherosclerosis acquires a generalized form.

• Excessive synthesis of glyco- and mucoproteids leads to the development of vessel hyalinosis, which is accompanied by autoimmune inflammation and aggravates atherosclerotic damage. Vascular pathology (angiopathy) is the main reason for invalidity and death.

• Lipid infiltration of the parenchymatous organs (liver, myocardium) leads to failure of these organs and aggravates pathophysiological disorders with various clinical symptoms.

• Glycosuria promotes osmotic diuresis, which can reach 10-12 1/day. Loss of water leads to dehydration. Although patients compensate it by drinking, electrolytes (Na, K, Mg, P) are lost in the urine. Loss of water results in hyperosmolarity and thirst.

• Glycolization of various proteins (Hb, enzymes, collagen, etc.) has different consequences depending on the type of damaged proteins.

• Formation of immune antibodies is suppressed. It results in decreased resistance of the organism to infection (immunodepression).

• Regenerative and plastic processes are suppressed resulting in decreased wound healing. Any trauma is accompanied by incapability of regeneration and frequently results in formation of the so-called trophic ulcers, whose development is promoted by atherosclerosis.

• Growth retardation is observed in children.

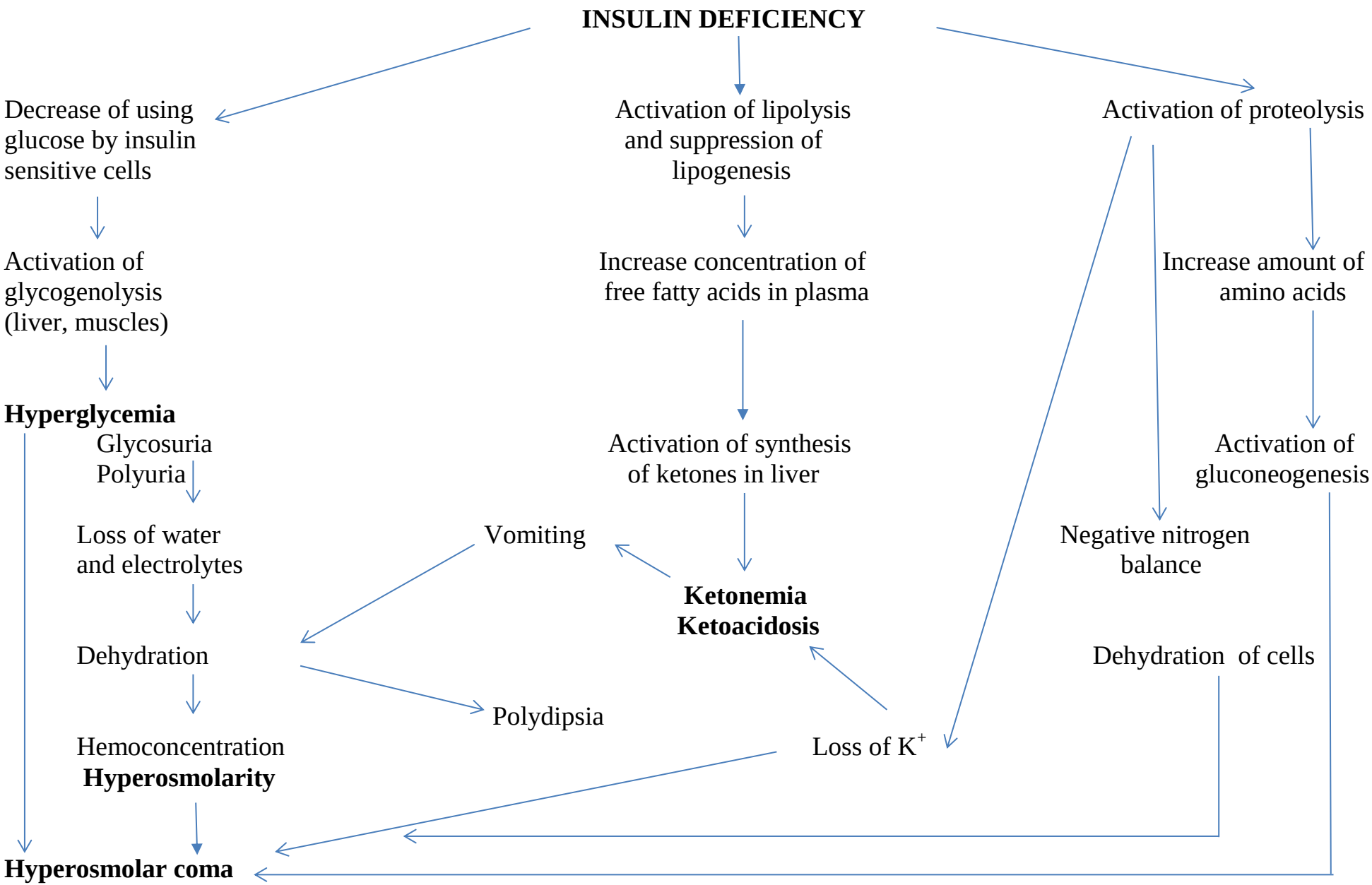


Fig. 2 Pathogenesis of absolute insulin deficiency

Diabetes mellitus is a syndrome of impaired carbohydrate, protein, an d fat metabolism resulting from either lack of insulin secretion or decreased s ensitivity of the tissues to insulin.

According to clinical classification DM is divided into *primary* (idiopathic) and *secondary* (symptomatic as a complication of another disease). In its turn, primary (idiopathic) DM is subdivided into two types depending on the form of insulin insufficiency (pathogenic classification) — *insulin-dependent (type 1, IDDM) and non-insulin-dependent (type 2, NIDDM).* Type 3 diabetes mellitus is connected with pregnancy *(gestational DM).*

Type 1 diabetes mellitus develops as a result of synergetic effects of geneti c, environmental, and immunological factors that ultimately destroy the pancreatic β- cells.

In type 1 DM, the concordance rate is about 50%, indicating that a genetic predisposition is involved. Environmental factors include hypothetically viruses (coxsackie and rubella most prominently) bovine milk proteins, and nitrosourea compounds. It now appears that in individuals who have genetic predisposition indicated by the HLA pattern, a triggering event that is environmental, initiates formation of antibodies against islet cells, and the resulting infiltration of the islets with lymphocytes (“insulitis”) leads over a period of years to loss of β- cells and frank diabetes. The lack of insulin decreases the entry of glucose into various “periph eral” tissues and increases the liberation of glucose into the circulation from the liver that results in hyperglycemia.

The high blood glucose causes more glucose to filter into the renal tubules than can be reabsorbed, and the excess glucose spills into the urine causing glucosuria. The renal threshold for glucose i.e. the plasma level at which glucose first appears in the urine in more than the normal minute amounts, is about 200mg/dl of arterial plasma, which corresponds to a venous level of about 180 mg/dl The very high levels of blood glucose (sometimes as high as 8 to 10 times normal) can cause severe cell dehydratation through the body because of the increases osmotic pressure in the extracellular fluids. In addition to this, the loss of glucose in the urine causes osmotic diuresis resulting in extracellular dehydratation. Thus, polyuria (excessive urine excretion), intracellular and extracellular dehydratation and polydipsia (increased thirsts) are classic symptoms of diabetes.Type1 diabetes is a catabolic disorder in which circulating insulin is virtually absent; glucagon level is elevated, and pancreatic βcells fail to respond to insulin producing stimuli. The shift from carbohydrate to fat metabolism in diabetes increases the release of the keto acids into the plasma more rapidly than they can be taken up and oxidized by the tissue cells. As a result, the patients develop severe metabolic acidosis from the excess keto acids (ketoacidosis).Kussmaul’s respiration (i.e., rapid and deep breathing) that occurs as a compensatory response to the acidosis, and a fruity odor on the patient’s breath cause d by increased acetone are classic sings of the disorder. Ketoacidosis in association with hydratation caused by excessive urine formation can lead rapidly to diabetic coma and death unless s the condition is treated immediately with large

amount of insulin. Failure to use glucose or energy leads to increased utilization and decreased storage of protein, as well as fat. Therefor e, the patients with untreated DM have rapid weight loss and asthenia (lack of energy) in spite of increased appetite and eating large amounts of food (polyphagia). Type 2 diabetes mellitus has a strong genetic, or at least familial, component. Environ mental factors such as nutrition and physical activity modulate phenotypic expression of t he disease. Almost all patients with type 2 diabetes are overweight, and the presence of obesity is important consideration in the development of this form of the disease. Type 2 DM describes a condition of fasting hyperglycemia that occurs despite the availability of insulin. In contrast to type 1, type 2 diabetes mellitus, is associated with increased plasma insulin concentration, i.e., hyperinsulinemia. This occurs as a compensatory response b y pancreatic β-cells for diminished sensitivity of target 8 tissues to the metabolic effects of insulin, a condition referred as to insulin resistance. Insulin resistance impairs the peripheral glucose utilization and storage, raising blood glucose and stimulating a compensatory increase in insulin secretion with resultant hyperinsulinemia. In time, the insulin response by the β-cells declines because of exhaustion. Because people with type 2 diabetes do not have an absolute insulin deficiency, they are less prone to ketoacidosis than the people with type 1 diabetes.

Clinical Manifestations

Renal, vascular, neurological and other long-term effects of DM are the following.

Polyuria is increased diuresis. An increase of osmotic pressure of the urine and a decrease of water reabsorption underlie it. Diuresis may reach 10—12 1 daily.

Polydipsia is thirst accompanied by dryness in the mouth and on the skin. Polyphagia is increased appetite as a result of carbohydrate starvation of tissues.

Muscle and general weakness is a consequence of energy tissue starvation. Atherosclerosis is a common complication of DM.

Atherosclerosis is a big problem of modern medicine and more often is based on DM. Consequences of vessel atherosclerosis cause death.

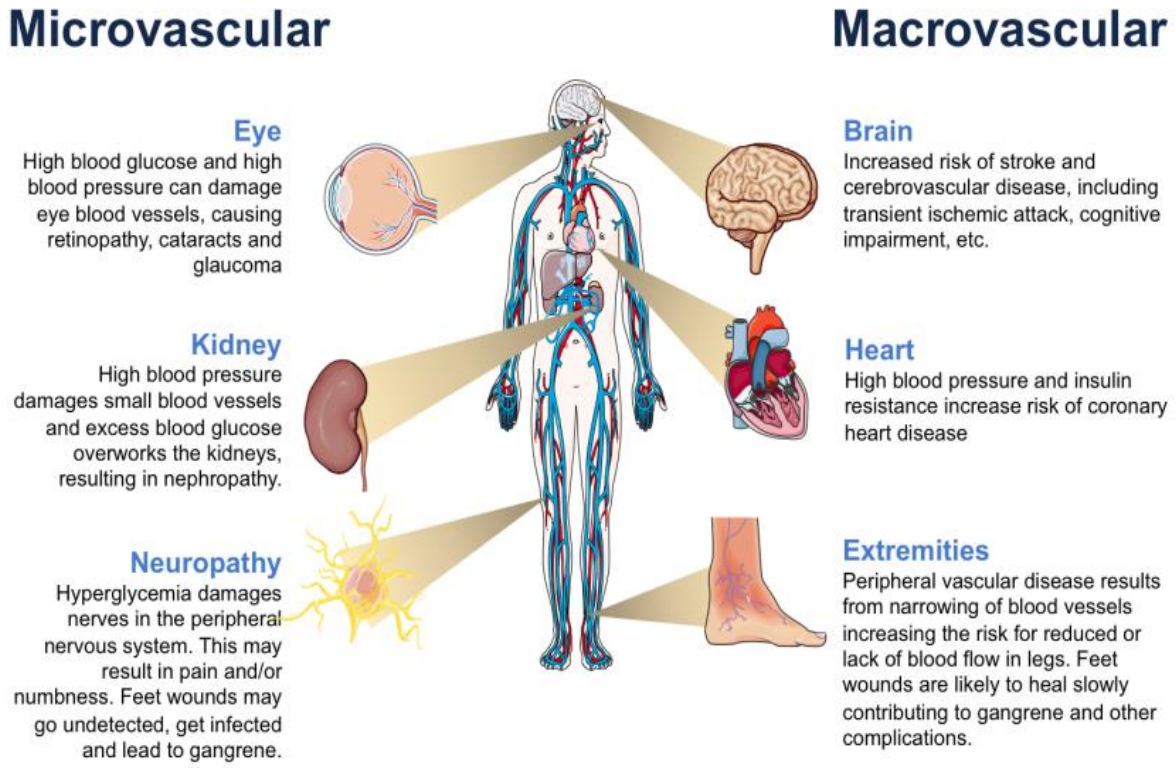
Angiopathy (macro- and microangiopathy) is a pathology of large and small vessels, which develops as a result of atherosclerosis and hyalinosis. Chronic vascular syndrome includes pathology of the cerebral, coronary, renal, retinal and arterial vessels of the lower extremities. It is not difficult to understand the wide range of pathology that may develop — stroke, myocardial infarction, amputation of extremities, blindness. Arterial hypertension is an increase of arterial blood pressure.

Nephropathy is renal dysfunction, which manifests itself through many renal and systemic symptoms including the development of the renal type of arterial hypertension.

Immunodepression manifests itself through increased sensitivity to infection. Almost all patients suffer from furunculosis. Many diabetics are ill with tuberculosis.

Neuropathy is a disorder of the morphology and function of the peripheral nerves (sensitive, motor and vegetative ones).Partial or complete loss of sight is

retinopathy. It is a result of eye vessel impairment and cataract (crystalline sclerosis).
Depression of regenerative processes (as a result of depressed protein synthesis) is manifested by problems with wound reparation and trophic ulcer development.
Body mass alterations are emaciation (a result of lipolysis activation) or obesity as a result of insulin resistance.



Chronic complications of DM

Diabetic Coma

The most violent complication of DM is comatose state. The manifestations of coma are the following: loss of consciousness, arterial hypotension, Kussmaul's disorder of respiration, a smell of acetone from the mouth.
There are several types of diabetic coma. They are:
Acidotic (lactacidemic), which is a result of lactic acid accumulation and blood pH reduction.
Ketonemic, which is a result of the toxic effect of ketone bodies.
Hyperosmolar, which is a result of high hyperglycemia.
Hypoglycemic, which is a common result of insulin overdose.

KROK 1_mcqs (A is correct answer):

1.Baby refuses the breast, he is anxious, presents with arrhythmic respiration. The urine smells of "brewers yeast" or "maple syrup". This pathology was caused by the

inherited defect of the following enzyme:

A. Dehydrogenase of branched-chain alpha-ketoacids
B. Glucose 6-phosphate dehydrogenase
C. Glycerol kinase
D. Aspartate aminotransferase
E. UDP-glucuronil transferase

2. After a sprint an untrained person develops muscle hypoxia. This leads to the accumulation of the following metabolite in muscles:

A. Lactate
B. Ketone bodies
C. Acetyl CoA
D. Glucose 6-phosphate
E. Oxaloacetate

3. Characteristic sign of glycogenosis is muscle pain during physical work. Blood examination reveals usually hypoglycemia. This pathology is caused by congenital deficiency of the following enzyme

A. Glycogen phosphorylase
B. Glucose 6-phosphate dehydrogenase
C. Alpha amylase
D. Gamma amylase
E. Lysosomal glycosidase

4. A patient is ill with diabetes mellitus accompanied by hyperglycemia on an empty stomach (7,2 mmol/l). The hyperglycemia rate can be retrospectively estimated (over the last 4-8 weeks before the examination) on the ground of the rate of the following blood plasma protein:

A. Glycated hemoglobin
B. Albumin
C. Fibrinogen
D. C-reactive protein
E. Ceruloplasmin

5. A patient ill with neurodermatitis has been taking prednisolone for a long time. Examination revealed high rate of sugar in his blood. This complication is caused by the drug influence upon the following link of carbohydrate metabolism:

A. Gluconeogenesis activation
B. Glycogenogenesis activation
C. Intensification of glucose absorption in the bowels
D. Inhibition of glycogen synthesis
E. Activation of insulin decomposition

6. A 62 year old patient who previously worked as stoker was admitted to a hospital with complaints about general weakness, abrupt weight loss, hoarse voice, dyspnea, dry cough. Laryngoscopy revealed a tumor in the pharynx that invaded vocal cords and epiglottis. What is the most probable cause of tumor development?

A. Polycyclic aromatic carbohydrates
B. Nitrosamines
C. Aromatic amines and amides
D. Retroviruses
E. Ionizing radiation

7. Patients who suffer from severe diabetes and don’t receive insulin have metabolic acidosis. This is caused by increased concentration of the following metabolites:

A. Ketone bodies
B. Fatty acids
C. Unsaturated fatty acids
D. Triacylglycerols
E. Cholesterol

8. A 60-year-old male patient has a 9-year history of diabetes and takes insulin Semilente for the correction of hyperglycemia. 10 days ago he

began taking anaprilin for hypertension. One hour after administration of the antihypertensive drug the patient developed hypoglycemic coma. What is the mechanism of hypoglycemia in case of anaprilin use?

A. Inhibition of glycogenolysis
B. Reduction of glucagon half-life
C. Increase of insulin Semilente half-life
D. Increase of bioavailability of insulin Semilente
E. Decrease in glucose absorption

9. Inherited diseases, such as mucopolysaccharidoses, are manifested in metabolic disorders of connective tissue, bone and joint pathologies. The sign of this disease is the excessive urinary excretion of the following substance:

A. Glycosaminoglycans
B. Amino acids
C. Glucose
D. Lipids
E. Urea

10. The genetic defect of pyruvate carboxylase deficiency is the cause of delayed physical and mental development and early death in children. This defect is characterized by lacticemia, lactaciduria, disorder of a number of metabolic pathways. In particular, the following process is inhibited:

A. Citric acid cycle and gluconeogenesis
B. Glycolysis and glycogenolysis
C. Glycogenesis and glycogenolysis
D. Lipolysis and lipogenesis
E. Pentose phosphate pathway and glycolysis

11. A female patient complains of vision impairment. On examination she was found to have obesity, fasting hyperglycemia. What complication of diabetes can cause vision impairment?

A. Microangiopathy
B. Macroangiopathy
C. Atherosclerosis
D. Neuropathy
E. Glomerulopathy

12. In a young man during exercise, the minute oxygen uptake and carbon dioxide emission equalled to 1000 ml. What substrates are oxidized in the cells of his body?

A. Carbohydrates
B. Proteins
C. Fats
D. Carbohydrates and fats
E. Carbohydrates and proteins

13. A 39-year-old female patient with a history of diabetes was hospitalized in a precomatose state for diabetic ketoacidosis. This condition had been caused by an increase in the following metabolite level:

A. Acetoacetate
B. Citrate
C. Alpha-ketoglutarate
D. Malonate
E. Aspartate

14. According to the results of glucose tolerance test, the patient has no disorder of carbohydrate tolerance. Despite that, glucose is detected in the patients's urine (5 mmol/l). The patient has been diagnosed with renal diabetes. What renal changes cause glucosuria in this case?

A. Decreased activity of glucose reabsorption enzymes
B. Increased activity of

glucose reabsorption enzymes
C. Exceeded glucose reabsorption threshold
D. Increased glucose secretion
E. Increased glucose filtration

15. Examination of a 56-year-old female patient with a history of type 1 diabetes revealed a disorder of protein metabolism that is manifested by aminoacidemia in the laboratory blood test values, and clinically by the delayed wound healing and decreased synthesis of antibodies. Which of the following mechanisms causes the development of aminoacidemia?

A. Increased proteolysis
B. Albuminosis
C. Decrease in the concentration of amino acids in blood
D. Increase in the oncotic pressure in the blood plasma
E. Increase in low-density lipoprotein level

16. A 60-year-old male patient has type II diabetes. A doctor has prescribed him synthetic hypoglycemic long acting drug that is sulfonylurea derivative. What drug is it?

A. Glibenclamide
B. Butamide
C. Metformin
D. Actrapid (soluble insulin)
E. Acarbose

17. A child has a history of hepatomegaly, hypoglycemia, seizures, especially on an empty stomach and in stressful situations. The child is diagnosed with Gierke disease. This disease is caused by the genetic defect of the following enzyme:

A. Glucose-6-phosphatase
B. Amyloid-1,6-glycosidase
C. Phosphoglucomutase
D. D Glycogen phosphorylase
E. Glucokinase

18. Pancreas is known as a mixed gland. Endocrine functions include production of insulin by beta cells. This hormone affects the metabolism of carbohydrates. What is its effect upon the activity of glycogen phosphorylase (GP) and glycogen synthase (GS)?

A. It inhibits GP and activates GS
B. It activates both GP and GS
C. It inhibits both GP and GS
D. It activates GP and inhibits GS
E. It does not affect the activity of GP and GS

20. One of the factors that cause obesity is the inhibition of fatty acids oxidation due to:

A. Low level of carnitine
B. Impaired phospholipid synthesis
C. Excessive consumption of fatty foods
D. Choline deficiency
E. Lack of carbohydrates in the diet

31. Patient with diabetes didn't get insulin injection in time that caused hyperglycemic coma (glucose in the blood 50mmol/L). What mechanism is prevalent in the development of the coma?

A. Hyperosmia
B. Hypokaliemia
C. Hypoxia
D. Hyponatremia
E. Acidosis

32. Galactosemia has been revealed in a child. Concentration of glucose in the blood has not considerably

changed. What enzyme deficiency caused this illness?

A. Galactose-1-phosphate uridyl transferase
B. Amylo-1,6-glucosidase
C. Phosphoglucomutase
D. Galactokinase
E. Hexokinase

33. The patient with diabetes mellitus has been delivered in hospital in the state of unconsciousness. Arterial pressure is low. The patient has acidosis. Point substances, which accumulation in the blood results in these manifestations:

A. Ketone bodies
B. Amino acids
C. Monosaccharides
D. High fatty acids
E. Cholesterol esters

34. The B cells of endocrine portion of pancreas are selectively damaged by alloxan poisoning. How will it be reflected in blood plasma?

A. The content of sugar increases
B. The content of fibrinogen decrease
C. The level of sugar decreases
D. The content of globulins decreases
E. The content of albumins decreases

35. A patient with the symptoms of acute alcoholic poisoning was brought to the hospital.
What carbohydrates metabolism changes are typical for this condition?

A. The gluconeogenesis velocity in liver is decreased
B. The gluconeogenesis is increased in liver
C. The breakage of glycogen is increased in liver
D. The anaerobic glucose metabolism predominates in muscles
E. The anaerobic breakage of glucose is increased in muscles

36. Patient with diabetes mellitus experienced loss of consciousness and convulsions after an injection of insulin. What might be the result of biochemical blood analysis for concentration of sugar?

A. 1,5 mmol/L
B. 8,0 mmol/L
C. 10,0 mmol/L
D. 3,3 mmol/L
E. 5,5 mmol/L

37. Diabetes mellitus causes ketosis as a result of activated oxidation of fatty acids. What disorders of acid-base equilibrium may be caused by excessive accumulation of ketone bodies in blood?

A. Metabolic acidosis
B. Metabolic alcalosis
C. Any changes woun't happen
D. Respiratory acidosis
E. Respiratory alcalosis

38. A patient is ill with diabetes mellitus that is accompanied by hyperglycemia of over 7,2 mmol/l on an empty stomach. The level of what blood plasma protein allows to estimate the glycemia rateretrospectively (4-8 weeks before examination)?

A. Glycated hemoglobin
B. Albumin
C. Fibrinogen
D. C-reactive protein
E. Ceruloplasmin

39. A patient was delivered to the hospital by an emergency team. Objectively: grave condition, unconscious, adynamy. Cutaneous

surfaces are dry, eyes are sunken, face is cyanotic. There is tachycardia and smell of acetone from the mouth. Analysis results: blood glucose -20,1 micromole/l (standard is 3,3-5,5 micromole/l), urine glucose - 3,5%(standard is - 0). What is the most probable diagnosis?

A. Hyperglycemic coma
B. Hypoglycemic coma
C. Acute heart failure
D. Acute alcoholic intoxication
E. Anaphylactic shock

40. A child is languid, apathetic. Liver is enlarged and liver biopsy revealed a significant excess of glycogen. Glucose concentration in the blood stream is below normal. What is the cause of low glucoseconcentration?

A. Low (absent) activity of glycogenephosphorylase in liver
B. Low (absent) activity of hexokinase
C. High activity of glycogen synthetase
D. Low (absent) activity of glucose 6-phosphatase
E. Deficit of a gene that is responsiblefor synthesis of glucose 1-phosphaturidinetransferase

41. A child's blood presents high content of galactose, glucose concentration is low. There are such presentations as cataract, mental deficiency, adipose degeneration of liver. What disease is it?

A. Galactosemia
B. Diabetes mellitus
C. Lactosemia
D. Steroid diabetes
E. Fructosemia

42. A 45 y.o. woman suffers from Cushing's syndrome - steroid diabetes. Biochemical examination revealed: hyperglycemia, hypochloremia. Which of the under-mentioned processes is the first to be activated?

A. Gluconeogenesis
B. Glycogenolysis
C. Glucose reabsorption
D. Glucose transport to the cell
E. Glycolysis

43. A patient ill with neurodermatitis has been taking prednisolone for a long time. Examination revealed high rate of sugar in his blood. This complication is caused by the drug influence upon the following link of carbohydrate metabolism:

A. Gluconeogenesis activation
B. Glycogenogenesis activation
C. Intensification of glucose absorption in the bowels
D. Inhibition of glycogen synthesis
E. Activation of insulin decomposition

44. A 12-year-old teenager has significantly put off weight within 3 months; glucose concentration rose up to 50 mmol\l. He fell into a coma. What is the main mechanism of its development?

A. Hyperosmolar
B. Hypoglycemic
C. Ketonemic
D. Lactacidemic
E. Hypoxic

45. A 15-year-old patient has fasting plasma glucose level 4,8 mmol/l, one hour after glucose challenge it becomes 9,0 mmol/l, in 2 hours it is 7,0 mmol/l, in 3 hours it is 4,8 mmol/l. Such parameters are characteristic of:

A. Subclinical diabetes mellitus
B. Diabetes mellitus type 1
C. Diabetes mellitus type 2
D. Healthy person
E. Cushing's disease

46. A patient with insulin-dependent diabetes mellitus has been administered insulin. After a certain period of time the patient developed fatigue, irritability, excessive sweating. What is the main mechanism of such presentations developing:
A. Carbohydrate starvation of the brain
B. Increased glycogenolysis
C. Increased ketogenesis
D. Increased lipogenesis
E. Decreased glyconeogenesis

47. A comatose patient was taken to the hospital. He has a history of diabetes mellitus. Objectively: Kussmaul breathing, low blood pressure, acetone odor of breath. After the emergency treatment the patient's condition improved. What drug had been administered:
A. Insulin
B. Adrenaline
C. Isadrinum
D. Glibenclamide
E. Furosemide

48. A woman complains of visual impairment. Examination revealed obesity in the patient and her fasting plasma glucose level is hyperglycemic. What diabetes complication can cause visual impairment/blindness:
A. Microangiopathy
B. Macroangiopathy
C. Atherosclerosis
D. Neuropathy
E. Glomerulopathy

49.A 30-year-old man with diabetes mellitus type I was hospitalized. The patient is comatose. Laboratory tests revealed hyperglycemia and ketonemia. What metabolic disorder can be detected in this patient?
A. Metabolic acidosis
B. Metabolic alkalosis
C. Respiratory acidosis
D. Respiratory alkalosis
E. Acid-base balance is normal

50.A 56-year-old man complains of thirst and frequent urination. The endocrinologist diagnosed this patient with diabetes mellitus and prescribed him glibenclamide. What mechanism of action does this drug have?
A. Stimulation of β-cells of islets of Langerhans
B. Facilitates glucose uptake by the tissues
C. Facilitates glucose transport through cell membranes
D. Suppression of α-cells of islets of Langerhans
E. Inhibits glucose absorption in the intestine

A patient was hospitalized in a comatose state. The patient has a 5-year long history of diabetes mellitus type 2. Objectively respiration is noisy, deep, with acetone breath odor. Blood glucose is 15.2 mmol/L, ketone bodies - 100 micromol/L. These signs are characteristic of the following diabetes complication:
A. Ketoacidotic coma
B. Hepatic coma
C. Hyperglycemic coma
D. Hypoglycemic coma
E. Hyperosmolar coma

Tests for Self-Control

1. A DM patient was taken to a hospital unconscious. There is observed Kussmaul's respiration, arterial pressure of 80/50 mmHg, a smell of acetone from the mouth. Accumulation of what substances provoked such a state?
A. Ketone bodies.
B. p-Lipoproteins.
C. Lactate.
D. Blood glucose.
E. Cholesterole.

2. A 48-year-old man is ill with diabetes. He was hospitalized in unconscious state after significant physical load. Respiration is superficial, arterial pressure — 80/40 mmHg, glycemia — 1.88 mmol/1. What kind of coma has developed?
A. Lactacidemic.
B. Hyperketonemic.
C. Hypoglycemic.
D. Hyperosmolar.
E. Hypoosmolar.

3. A 12-year-old teenager grew thin. The level of glucose in the blood was 50 mmol/1. Later coma developed. What was the main mechanism of coma?
A. Ketonemic.
B. Hypoglycemic.
C. Hyperosmolar.
D. Lactacidemic.
E. Hypoxic.

4. A patient with type 1 diabetes was injected insulin. Later he suffered from sickness, irritability, sweating. What is the basic mechanism of hypoglycemia, which developed?
A. Intensifying of ketogenesis.
B. Intensifying of glycogenolysis.
C. Carbohydrate starvation of the brain.
D. Intensifying of lipogenesis.
E. Depressing of glyconeogenesis.

5. A patient is ill with diabetes. Regenerative processes are reduced, wounds do not heal for a long time. What is the cause of such changes?
A. Acidosis.
B. Accumulation of ketone bodies.
C. Depressed synthesis of proteins.
D. Decreased content of glucose in cells.
E. Lipid disbolism.

Recommended literature:

Basic

1. Simeonova N.K. Pathophysiology/ N.Simeonova.// Kyiv, Ukraine. – 2010. – 187-222pp.
2. Krishtal N.V. Pathophysiology: textbook/ N.Krishtal et al.// Kyiv: AUS Medicine Publishing, 2017. – 95-104 pp.
3. Victor N. Jelski, Svetlana V. Kolesnikova. Handbook Of Pathophysiology Part 1: General Pathophysiology. - Donetsk, Ukraine. – 2009. – 108-120 pp.

Additional

4. Porth, Carol. Essentials of pathophysiology: concepts of altered health states /Carol Mattson Porth ; consultants, Kathryn J. Gaspard, Kim A. Noble. — 3rd ed. 2011 Wolters Kluwer Health | Lippincott Williams & Wilkins. – 2011. – 1282 p.
5. Robbins Pathology basis of disease / Cotran R.S., Kumar V., Robbins S.L. - 2000.

Methodical instruction to practical lesson № 12
Module 1. General pathology
Theme: LIPID AND PROTEIN METABOLISM DISORDERS

Student should know:

- Main stages of protein and lipid metabolism.
- Causes and types of obesity.

Student should be able to:

- Explain mechanisms of development of atherosclerosis, gout, uremia.

LIST OF CONTROL QUESTIONS

1. The concept of positive and negative nitrogen balance. Disorders of the main stages of protein metabolism. Azotemia, productive and retention. Disorders of the blood protein, hyper-, hypo-, dysproteinemia.
2. Disorders of digestion and absorption of lipids.
3. Disorders of lipid transport in the blood. Hyper-, hypo-, dyslipoproteinemia.
4. Modern classification dyslipoproteinemia (primary and secondary, LP phenotype, with high or low risk of atherosclerosis) criteria hypercholesterolemia, hypertriglyceridemia, low HDL.
5. The etiology and pathogenesis of primary (hereditary, family) and secondary (eating disorders, obesity, diabetes, kidney diseases, hypothyroidism, liver cirrhosis, AIDS, under the influence of drugs) dyslipoproteinemia.
6. The definition of obesity. Types of obesity. Experimental models. The etiology and pathogenesis of obesity. Mechanisms of fatty degeneration of liver.

Lipids are the most important source of energy: their calorific value is higher than that of carbohydrates and proteins. Lipids are light and therefore are the most convenient form of energy accumulation. Besides, they are a source of oxidative water. Lipids and carbohydrates are converted into each other (lipogenesis from carbohydrates with the aid of insulin; glyconeogenesis from fatty acids with the aid of glucocorticoids).

As to lipid absorption in the bowels, there are some causes of disorder:

• lack of bile (liver insufficiency, calculous cholecystitis), which emulsifies lipids by bile acids and exposes them to pancreatic lipase;

• inflammation of the bowel mucosa;

• pancreatic insufficiency (the pancreas is the main source of lipase in the bowels).

If lipids are not absorbed in the bowels, they appear in feces *(steatorrhea*). Absorption of fat-soluble vitamins (A, D, K, E) is connected with lipid absorption. Corresponding clinical manifestations of a- and hypovitaminosis (bleeding, rachitis, vision and endocrine system impairment) develop in case of insufficient absorption of lipids and vitamins.

An excessive content of lipids in the blood is called ***hyperlipemia***, lowered — **hypolipemia**

Lipoproteins – spheric particles transporting non-polar lipids (TAGs, cholesterol esters)by blood

Composition and properties

– inside of sphere - non-polar lipids

– surfice of sphere -polar molecules (phospholipids, non-esterified cholesterol)- are important for transport of particles in plasma

– apo-LPs - are important for LPs metabolism

Different types of LPs differs by their density, by volume of transporting lipids, by size, by amount and kind of apo, by location of their creation, by their metabolism.

Characteristics of main types of LPs

• **Chylomicrons(CM) –** the lowest density, the largest size

• **VLDL –** smaller and more dense than CM – they transport endogenous TAGs synthetised in liver

• **IDL –** particles with properties between VLDL and LDL

• **LDL –** containe cholesterol esters, mainly

• **HDL –** the smallest size and the highest density – they are able to transport cholesterol from peripheral tissues to liver (reversal transport of cholesterol)

Obesity is an excessive accumulation of lipids in the adipose tissue.

As to etiology, obesity is divided into ***acquired and constitutional***. As to pathogenesis, it is divided into ***alimentary, endocrine and cerebral*** (hypothalamic). The pathomorphological classification is based on the size and quantity of adipocytes. According to this classification, two types of obesity are distinguished ***hypertrophic and hyperplastic***. Besides, ***primary (constitutional) and secondary (symptomatic***) obesity is distinguished.

Obesity is a result of imbalance between energy production and use. Three basic pathogenetic mechanisms are important in obesity pathogenesis:

• increased intake of food, which does not correspond to energy expenditure;

• decreased mobilization of fat from the depots as a source of energy;

• excessive fat formation from carbohydrates.

Primary (constitutional) obesity is observed in 55—65 % of all cases. The gene of obesity and its product leptin are being investigated. The structure and function of the systems, which regulate alimentary behavior and lipid metabolism peculiarities can be inherited — adipose tissue peculiarities, adipocyte quantity and size. All these factors must be taken into account in obesity pathogenesis. In general, obesity is inherited polygenetically. In means that it is determined by genetic, environmental factors and acquired diseases.

Primary cerebral (hypothalamic) obesity is observed in 16-20 % of all cases. Excessive consumption of food, which is provoked by increased appetite, can result from increased excitability of the alimentary center, which is situated in the anterolateral nuclei of the posterior hypothalamic region. All the reasons affecting the alimentary center can be a cause of prolonged alimentary excitation and alimentary obesity as a result. Chronic stress, which is often accompanied with overeating, is an example.

Cerebral (hypothalamic) obesity may be modeled under experimental conditions by damaging the ventromedial nuclei of the hypothalamus. Hyperphagia, which is obtained in an experimental animal, results in obesity, which is similar to human hypothalamic obesity. Hypotonia of the sympathetic nervous system prolongs fat mobilization from the adipose tissue. Signals from the alimentary tract receptors are important in the activity of the alimentary center. A definite degree of stomach extension after eating inhibits the activity of the alimentary center. In case of decreased sensitivity of the stomach wall receptors, alimentary center inhibition develops only in excessive stomach extension.

Together with the nervous system the endocrine one accomplishes regulation of fat mobilization and deposition. Adrenaline and insulin have the most potent influence but in the opposite direction. Adrenaline stimulates lipolysis. Insulin stimulates lipogenesis by the synthesis of neutral fats from glucose and fatty acids, inhibits lipolysis, decreases the blood sugar level, stimulates appetite. All conditions, which decrease the blood glucose level, stimulate pancreatic island function and are accompanied by hunger provoking overeating.

The same form of obesity and decreased fat mobilization is observed in hypofunction of the pituitary, thyroid, adrenal and sex glands (*endocrine obesity*). Obesity is more often observed in women older than 50 (climacteric obesity is an example).

Proteins play a central role in the vital activity of the organism. They determine the structure and function of any organ. Each organ has its specific proteins structural proteins, enzymes, receptors, transport proteins, etc. Anabolism and catabolism of proteins are regulated by hormones. The latter serve as a signal for the activating or inhibiting effect on the processes of transcription in the genome. Then, with the aid of enzymes, many functional proteins (the structural and receptor proteins, etc.) are synthesized. So, it is a classical scheme: hormone — gene — enzyme.

Anabolic effect of hormones consists in activation of the processes of protein synthesis in comparison with their disintegration. *Somatotropic hormone* is a hormone of growth. It activates protein synthesis. It activates lipid oxygenation and neutral lipid mobilization and thus leads to sufficient release of energy, which is necessary for protein synthesis. *Insulin* provides transmission of amino acids through the cellular membranes into cells and thus provides protein synthesis and inhibits gluconeogenesis. Lack of insulin leads to protein synthesis decrease. *Sex hormones* (testosterone, progesterone) refer to anabolics and activate protein synthesis.

Catabolic effect of hormones consists in activation of protein disintegration in comparison with protein synthesis. *Thyroxin* increases the amount of active sulfhydric groups in the structure of some enzymes. Tissue cathepsins are activated and their proteolytic effect gets increased. It increases the activity of some aminooxydases — thus desamination of some amino acids is increased. *Glucocorticoids* (Cortisol) activate protein disintegration. Protein expenditure increases gluconeogenesis. Protein synthesis is decelerated.

The causes, pathogenesis and manifestations of protein disbolism are dividedinto large groups:

1. Systemic (protein imbalance in the whole organism) and local (in tissues).
2. Hereditary and acquired.
3. Disorder of anabolism and catabolism {synthesis and disintegration).

Systemic increase of protein content in the organism takes place only under physiological conditions (growth, pregnancy). Under pathological conditions an increase of protein content can be only local as hypertrophy, hyperplasia, tumor. Decrease of protein content in the organism occurs more often. The main reasons are:

- lower entry of proteins into the organism;
- disorder of protein digestion and absorption;
- increased loss of proteins; • increased disintegration of proteins;
- decreased protein synthesis.

The causes may be acquired and genetically determined. Enzymopathy underlies it more often.

The integral patterns of systemic protein balance are:

- Nitrogen balance.
- Blood protein level.
- Protein composition of the blood.

Positive nitrogen balance reflects intensification of protein synthesis (prevalence of protein anabolism over catabolism). Besides physiological processes (growth and pregnancy), intensive nourishment after starvation or wasting diseases can be an example. It is such a state, when less nitrogen is excreted from the organism than obtained with food. It is observed in case of curative injection of anabolic hormones (somatoptropin, androgen). **Negative nitrogen balance** reflects intensification of protein disintegration (catabolism) or loss. Starvation, infection, protein loss in the urine (proteinuria in renal pathology), loss with exudate in burns, with intestine content (diarrhea), thyreotoxicosis, and fever are examples. Excessive secretion or curative injection of catabolic hormones (thyroxin, cortisole) are also examples.

Quantitative and qualitative disorders of blood protein composition reflect various pathologic processes. They are manifested by changes in the organism during infection, neoplasia, allergy, inflammation, etc. *Hyperproteinemia* is always relative, caused by hemoconcentration. Its absolute variety is observed in some types of leukemia with formation of anomalous proteins (paraproteins).

Hypoproteinemia is, as a rule, absolute and occurs during starvation, neoplasia, diseases of the liver (decreased production of proteins) and kidneys (increased protein loss in the urine), disorder of protein absorption in the digestive tract. *Dysproteinemia* is a imbalance between the content of albumins, globulins and other proteins in the blood.

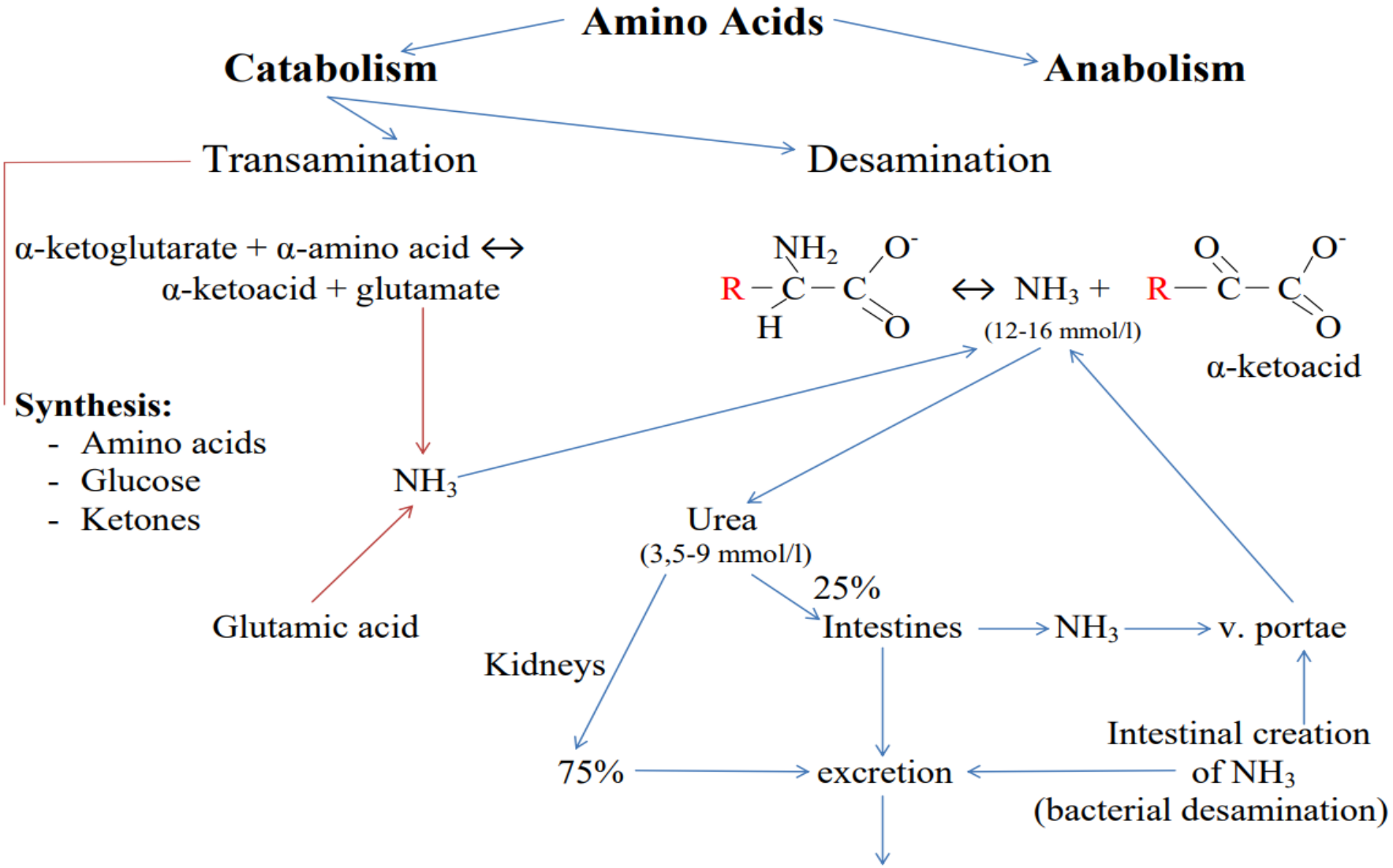


Fig. 3 Metabolism of proteins

KROK 1_mcqs (A is correct answer):

1.A patient with high rate of obesity was advised to use carnitine as a food additive in order to enhance "fat burning". What is the role of carnitine in the process of fat oxidation?

A. Transport of FFA (free fatty acids) from cytosol to the mitochondria
B. It takes part in one of reactions of FFA beta-oxidation
C. Transport of FFA from fat depots to the tissues
D. FFA activation
E. Activation of intracellular lipolysis

2. Examination of a patient revealed II grade obesity. It is known that he consumes a lot of sweets and rich food, has sedentary way of life. That's why anabolic metabolism has the priority in his organism. Which of the following pathways is amphibolic?

A. Cycle of tricarboxylic acids
B. Glyconeogenesis
C. Lipolysis
D. Glycolysis
E. Fatty acids oxidation

3. A 58-year-old patient suffers from the cerebral atherosclerosis. Examination revealed hyperlipidemia. What class of lipoproteins will most probably show increase in concentration in this patient's blood serum?

A. Low-density lipoproteins
B. Cholesterol
C. High-density lipoproteins
D. Chylomicrons
E. Fatty acid complexes with albumins

4. Examination of a man who had been working hard under higher temperature of the environment revealed abnormal quantity of blood plasma proteins. What phenomenon is the case?

A. Relative hyperproteinemia
B. Absolute hyperproteinemia
C. Absolute hypoproteinemia
D. Dysproteinemia
E. Paraproteinemia

5. A patient with obliterating atherosclerosis underwent sympathectomy of femoral artery in the region of femoral trigone. What type of arterial hyperemia was induced by the operation?

A. Neuroparalytic
B. Reactive
C. Metabolic
D. Neurotonic
E. Functional

6. Urine analysis of a 12-year-old boy reveals high concentration of all aliphatic amino acids with the highest excretion of cystine and cysteine. US of kidneys revealed kidney concrements. What is the most likely pathology?

A. Cystinuria
B. Alkaptonuria
C. Cystitis
D. Phenylketonuria
E. Hartnup disease

7. Toxic affection of liver results in dysfunction of protein synthesis. It is usually accompanied by the following kind of dysproteinemia:

A. Absolute hypoproteinemia
B. Relative hypoproteinemia

C. Absolute hyperproteinemia
D. Relative hyperproteinemia
E. Paraproteinemia

8. Electrophoretic study of a blood serum sample, taken from the patient with pneumonia, revealed an increase in one of the protein fractions. Specify this fraction:
A. γ-globulins
B. Albumins
C. α1-globulins
D. α2-globulins
E. β-globulins

9. A 42-year-old male patient with gout has an increased blood uric acid concentration. In order to reduce the level of uric acid the doctor administered him allopurinol. Allopurinol is the competitive inhibitor of the following enzyme:
A. Xanthine oxidase
B. Adenosine deaminase
C. Adenine phosphoribosyltransferase
D. Hypoxanthinephosphoribosyltransferase
E. Guanine deaminase

10. A 53-year-old male patient is diagnosed with Paget's disease. The concentration of oxyproline in daily urine is sharply increased, which primarily means intensified disintegration of:
A. Collagen
B. Keratin
C. Albumin
D. Hemoglobin
E. Fibrinogen

11. A 46-year-old female patient consulted a doctor about pain in the small joints of the upper and lower limbs. The joints are enlarged and shaped like thickened nodes. Serum test revealed an increase in urate concentration. This might be caused by a disorder in metabolism of:
A. Purines
B. Carbohydrates
C. Lipids
D. Pyrimidines
E. Amino acids

A biochemical urine analysis has been performed for a patient with progressive muscular dystrophy. In the given case muscle disease can be confirmed by the high content of the following substance in urine:
A. Creatine
B. Porphyrin
C. Urea
D. Hippuric acid
E. Creatinine

12. A patient with homogentisuria has signs of arthritis, ochronosis. In this case, the pain in the joints is associated with the deposition of:
A. Homogentisates
B. Urates
C. Phosphates
D. Oxalates
E. Carbonates

13. A patient with hereditary hyperammonemia due to a disorder of ornithine cycle has developed secondary orotaciduria. The increased synthesis of orotic acid is caused by an increase in the following metabolite of ornithine cycle:
A. Carbamoyl phosphate
B. Citrulline
C. Ornithine
D. Urea
E. Argininosuccinate

14. In case of alkaptonuria, homogentisic acid is excreted in urine in large amounts. The development of this disease is associated with a disorder of

metabolism of the following amino acid:

A. Tyrosine
B. Phenylalanine
C. Alanine
D. Methionine
E. Asparagine

15. Patients with erythropoietic porphyria (Gunther's disease) have teeth that fluoresce with bright red color when subjected to ultraviolet radiation; their skin is light-sensitive, urine is red- colored. What enzyme can cause this disease, when it is deficient?

A. Uroporphyrinogen III cosynthase
B. Uroporphyrinogen I synthase
C. Delta-aminolevulinate synthase
D. Uroporphyrinogen decarboxylase
E. Ferrochelatase

16. Symptoms of pellagra (vitamin PP deficiency) is particularly pronounced in patients with low protein diet, because nicotinamide precursor in humans is one of the essential amino acids, namely:

A. Tryptophan
B. Threonine
C. Arginine
D. Histidine
E. Lysine

17. A denaturation of proteins can be found in some substances. Specify the substance that is used for the incomplete denaturation of hemoglobin:

A. Urea
B. Toluene
C. Sulfuric acid
D. Nitric acid
E. Sodium hydroxide

18. For a long time a 49-year-old woman had suffered from glomerulonephritis which caused death. The autopsy revealed that the size of her kidneys was 7x3x2,5 sm, weight 65,0 g, they were dense and small-grained. Microscopically: fibrinogenous inflammation of serous and mucous capsules, dystrophic changes of parenchymatous organs, brain edema. What complication can cause such changes of serous capsules and inner organs?

A. Uraemia
B. Anemia
C. Sepsis
D. DIC-syndrome
E. Thrombopenia

19. After a serious viral infection a 3-year-old child has repeated vomiting, loss of consciousness, convulsions. Examination revealed hyperammoniemia. What may have caused changes of biochemical blood indices of this child?

A. Disorder of ammonia neutralization in ornithinic cycle
B. Activated processes of amino acids decarboxylation
C. Disorder of biogenic amines neutralization
D. Increased purtefaction of proteins in intestines
E. Inhibited activity of transamination enzymes

20. Cerebral trauma caused increase of ammonia formation. What amino acid takes part in removal of ammonia from cerebral tissue?

A. Glutamic
B. Tyrosine
C. Valine
D. Tryptophan

E. Lisine

21. Ammonia is a very toxic substance, especially for nervous system. What substance takes the most active part in ammonia detoxication in brain tissues?

A. Glutamic acid
B. Lysine
C. Proline
D. Histidine
E. Alanine

22. Nappies of a newborn have dark spots that witness of formation of homogentisic acid.
Metabolic imbalance of which substance is it connected with?

A. Thyrosine
B. Galactose
C. Methionine
D. Cholesterine
E. Tryptophane

23. A 65 year old man suffering from gout complains of kidney pain. Ultrasound examination revealed renal calculi. The most probable cause of calculi formation is the strengthened concentration of the following substance:

A. Uric acid
B. Cholesterol
C. Bilirubin
D. Urea
E. Cystine

24. Synthesis of phospholipids is disordered under the liver fat infiltration. Indicate which of the following substances can enhance the process of methylation during phospholipids synthesis?

A. Methionine
B. Ascorbic acid
C. Glucose
D. Glycerin
E. Citrate

25. Examination of cell culture got from a patient with lysosomal pathology revealed accumulation of great quantity of lipids in the lysosomes. What of the following diseases is this disturbance typical for?

A. Tay-Sachs disease
B. Gout
C. Phenylketonuria
D. Wilson disease
E. Galactosemia

26. A 70 year old man is ill with vascular atherosclerosis of lower extremities and coronary heart disease. Examination revealed disturbance of lipidic blood composition. The main factor of atherosclerosis pathogenesis is the excess of the following lipoproteins:

A. Low-density lipoproteins
B. Cholesterol
C. High-density lipoproteins
D. Intermediate density lipoproteins
E. Chylomicrons

27. A patient who had been working hard under conditions of elevated temperature of the environment, has now a changed quantity of blood plasma proteins. What penomenon is the case?

A. Relative hyperproteinemia
B. Absolute hyperproteinemia
C. Absolute hypoproteinemia
D. Disproteinemia
E. Paraproteinemia

28. A 58-year-old patient suffers from the cerebral atherosclerosis. Examination revealed hyperlipidemia. What class of lipoproteins will most probably show increase in concentration in this patient’s blood serum?

A. Low-density lipoproteins
B. High-density lipoproteins
C. Fatty acid complexes with albumins
D. Chylomicrons
E. Cholesterol

29. Cholesterol content in blood serum of a 12-year-old boy is 25 mmol/l. Anamnesis states hereditary familial hypercholesterolemia caused by synthesis disruption of receptor-related proteins for:
A. Low-density lipoproteins
B. High-density lipoproteins
C. Chylomicrons
D. Very low-density lipoproteins
E. Middle-density lipoproteins

30. A 49-year-old man complains of pain in his metatarsophalangeal joints and joint deformation. In blood hyperuricemy can be observed. X-ray has revealed metatarsophalangeal joint space narrowing, erosion, periarticular calcification of the both joints, osteoporosis. Microscopyhas revealed inflammatory granulomatous reaction surrounding necrotizing masses in the area of the first metatarsophalangeal joint. Choose the most likely diagnosis:
A. Gout (podagra)
B. Pyrophosphate arthropathy
C. Rheumatoid arthritis
D. Hyperparathyroidism
E. Urolithiasis

31. A therapeutist has an appointment with a 40-year-old patient complaining of recurrent pain attacks in his hallux joints and their swelling. Urine analysis revealed its marked acidity and pink color. What substances can cause such changes in uri ne?
A. Uric acid salt
B. Chlorides
C. Ammonium salts
D. Calcium phosphate
E. Magnesium sulfate

32. A patient with diabetes mellitus suffers from persistently non-healing surgical wound, which is a sign of disrupted tissue trophism. What is the cause of such disorder?
A. Disruption of protein metabolism regulation
B. Hypoglycemia
C. Ketonemia
D. Increased lipid catabolism
E. Anemia

33.During examination of a teenager with xanthomatosis the family history of hypercholesterolemia is revealed. What transportable lipids are increased in concentration in case of such a disease?
A. Low-density lipoproteins
B. Chylomicrons
C. Very low-density lipoproteins
D. High-density lipoproteins
E. Intermediate-density lipoproteins

34.A 52-year-old man presents with fever and pain in the joints. Both of his fi- rst metatarsophalangeal articulations are deformed, swollen, and reddened. Blood urea is high. The patient is diagnosed with gout. What is the main developmental factor in the pathogenesis of this disease?
A. Hyperuricemy
B. Argininosuccinic aciduria
C. Hyperazotemia
D. Hyperaminoacidemia
E. Citrullinuria

35.A sick child presents with high content of phenyl pyruvate in urine (normally it is practically absent). Blood phenylalanine level is 350

mg/L (norm - 15 mg/L). What disease are these symptoms characteristic of?

A. Phenylketonuria
B. Albinism
C. Tyrosinosis
D. Alkaptonuria
E. Gout

36. A 67-year-old male patient consumes eggs, pork fat, butter, milk and meat. Blood test results: cholesterol - 12,3 mmol/l, total lipids - 8,2 g/l, increased low density lipoprotein fraction (LDL). What type of hyperlipoproteinemia is observed in the patient?

A. Hyperlipoproteinemia type IIa
B. Hyperlipoproteinemia type I
C. Hyperlipoproteinemia type IIb
D. Hyperlipoproteinemia type IV
E. Cholesterol, hyperlipoproteinemia

37. A patient underwent a course of treatment for atherosclerosis. Laboratory tests revealed an increase in the antiatherogenic lipoprotein fraction in the blood plasma. The treatment efficacy is confirmed by the increase in:

A. HDL
B. VLDL
C. IDL
D. LDL
E. Chylomicrons

38. Increased HDL levels decrease the risk of atherosclerosis. What is the mechanism of HDL anti-atherogenic action?

A. They remove cholesterol from tissues
B. They supply tissues with cholesterol
C. They are involved in the breakdown of cholesterol
D. They activate the conversion of cholesterol to bile acids
E. They promote absorption of cholesterol in the intestine

39. Steatosis is caused by the accumulation of triacylglycerols in hepatocytes. One of the mechanisms of this disease development is a decrease in the utilization of VLDL neutral fat. What lipotropic prevent the development of steatosis?

A. Methionine, B6, B12
B. Arginine, B2, B3
C. Alanine, B1, PP
D. Valine, B3, B2
E. Isoleucine, B1, B2

40. Disruption of nerve fiber myelinogenesis causes neurological disorders and mental retardation. These symptoms are typical for hereditary and acquired alterations in the metabolism of:

A. Sphingolipids
B. Neutral fats
C. Higher fatty acids
D. Cholesterol
E. Phosphatidic acid

41. A 12-year-old patient was found to have blood serum cholesterol at the rate of 25 mmol/l. The boy has a history of hereditary familial hypercholesterolemia, which is caused by the impaired synthesis of the following protein receptors:

A. Low density lipoproteins
B. High density lipoproteins
C. Chylomicrons
D. Very low density lipoproteins
E. Intermediate density lipoproteins

Tests for Self-Control (give correct answers)

1. A 15-year-old boy suffers from alkaptonuria. His urine becomes black after settling. Metabolism of what substance is genetically disturbed?
A. Uric acid.
B. Cysteine.
C. Alanine.
D. Urea.
E. Tyrosine.

2. In some months after birth a child has a CNS lesion. The skin and hair brightened. Addition of a solution of trichloracetic iron to fresh urine leads to the appearance of olive-green coloring. What kind of hereditary disbolism took place?
A. Tyrosinosis.
B. Alkaptonuria.
C. Fructosuria.
D. Phenylketonuria.
E. Albinism.

3. Examination of a child with oligophrenia showed an increased level of phenylalanine and phenylpyruvate in the blood and neurolymph. The reaction of the urine with trichloracetic iron is positive. With deficiency of what enzyme is the child's disease connected?
A. Tyrosinase.
B. Hexokinase.
C. Phenylalanine hydroxylase.
D. Dopamine hydroxylase.
E. Hydroxyphenylpyruvate oxydase.

Recommended literature:

Basic

1. Simeonova N.K. Pathophysiology/ N.Simeonova.// Kyiv, Ukraine. – 2010. – 223-243pp.
2. Krishtal N.V. Pathophysiology: textbook/ N.Krishtal et al.// Kyiv: AUS Medicine Publishing, 2017. – 105-121 pp.

Additional

3. Porth, Carol. Essentials of pathophysiology: concepts of altered health states /Carol Mattson Porth ; consultants, Kathryn J. Gaspard, Kim A. Noble. — 3rd ed. 2011 Wolters Kluwer Health | Lippincott Williams & Wilkins. – 2011. – 1282 p.
4. Robbins Pathology basis of disease / Cotran R.S., Kumar V., Robbins S.L. - 2000.

Methodological instruction to practical lesson № 13
Module 1. General pathology

Theme: WATER AND ELECTROLYTIC METABOLISM DISORDERS. MICROELEMENTS EXCHANGE DISORDERS.ACID-BASE BALANCE DISORDERS

Student should know:

- Normal acid-base balance, laboratory criteria.
- Classification of water and electrolytic metabolism disorders.
- Causes and pathogenesis of water and electrolytic metabolism disorders.

Student should be able to:

- Explain development of clinical manifestations of disorders of acid-base status.
- Explain main principles of treatment of acid-base balance disorders.
- Explain relationships in pathogenesis of water and electrolytic metabolism disorders.

LIST OF CONTROL QUESTIONS

1. Positive and negative water balance. Role of hypothalamus, hypophysis, adrenal cortex in regulation of water-electrolytic balance. Dehydration: extracellular and intracellular; hypo-, iso-, hyperosmolar. The causes and mechanisms of development. Protective and compensatory mechanisms.
2. Excessive water in the body. Hypo-, iso and hyperosmolar hyperhydria, causes and mechanisms of development, protective, compensatory responses. Extra- and intracellular hyperhydria.
3. The definition of "swelling", types of edema. The causes and mechanisms of edema. Starling theory of the pathogenesis of edema. Swelling caused by changes in oncotic pressure of the blood and tissue fluid. The role of vascular permeability disorders and outflow of lymph in the pathogenesis of edema. Swelling caused by a delay of sodium and / or water in the body. Mixedematous swelling. Principles of treatment of edema.
4. Hyper- and hyponatremia. The causes and mechanisms of development. Disorders caused by changes in the concentration of sodium ions in the extracellular fluid.
5. General characteristics of disorders of acid-base status.
6. Acidosis, definition, classification, basic laboratory criteria.
7. Respiratory acidosis: causes and mechanisms of development, clinical manifestations.

8. Nonrespiratory acidosis (metabolic, secretory, exogenous): causes and mechanisms of development, the relationship between CBS and electrolyte disorders.
9. Acidosis with increased and normal anionic difference.
10. Alkalosis, definition, classification, basic laboratory criteria. Gas alkalosis: causes and mechanisms of development, clinical manifestations.
11. Respiratory alkalosis (secretory, exogenous): causes and mechanisms of development. The role of blood buffer systems, ion exchange systems of respiratory and renal mechanisms of compensation and correction of CBS.
12. Pathological changes in the body in disorders of acid-base status.

Water composes 60 % of body mass (from 45 % in thin aged persons to 70 % in the young). It is one of the most important constants of the organism.

Water of the blood (intravascular) composes about 5 %. It is the circulating blood volume. 93 % of it is pure water. The rest is bound with blood cellular elements.

Intracellular water composes 35—45 %. This volume is constantly regulated and must not be changed. The intracellular fluid is present in three conditions: a) water of the cytoplasm bound with hydrophilic structures; b) water connected with the surface of colloid structures; c) water in the cytoplasmic lacunas, which is the most mobile, relatively free water of cells. In different pathologic conditions the intracellular fluid volume changes at the expense of the mobile water volume.

Extracellular (interstitial) water composes about 15 %. Only the quantity of this water may change significantly. The interstitial fluid is close to the blood plasma (except protein contents) and washes cells with ion and molecular substrates. This fluid is in constant exchange with the blood plasma so that approximately 20 1 of fluid with dissolved substances comes into tissues from vessels daily and the same amount returns into the systemic blood flow. 3 1 of the fluid returns through the lymphatic vessels.

Extracellular (transcellular) water (1—3 %) forms the digestive juice, cerebrospinal fluid, and the kidney tubule fluid.

Local (tissue) mechanisms regulate the water balance between the blood and tissues through the capillary walls. E. Starling and other scientists (Vidal, Fisher) studied the factors, which determine liquid passing from the blood stream into the intercellular space (filtration) and its return into the vessels. This balance is regulated by physicochemical mechanisms:

• *Hydrodynamic pressure difference between the blood and extracellular fluid. The blood moves in the capillaries at a* definite speed and under a definite pressure, which results in the formation of hydrodynamic force, which makes water go out of the capillaries into the interstitial space. The higher blood pressure and the less tissue fluid pressure, the higher effect of hydrodynamic force. Hydrodynamic blood pressure in the arterial section of the capillaries is 35—40

mmHg, and in the venous section — 10—15 mmHg.

• *Oncotic pressure* (of proteins) difference between the blood and interstitial liquid. An increase of vascular permeability for proteins occurs in a number of pathologic processes and essentially influences this parameter.

• *Osmotic pressure* difference between the blood and interstitial liquid (Vidal).

• *Interstitial tissue pH*. The hydrophilic nature of colloids depends on H+ concentration and rises in acid media. Then colloids swell and detain more water (Fisher).

The resultant force is called *filtration pressure*. The ratio of these forces determines the passage of liquid into tissues from the arterial part of the capillaries and its return to the blood in the venous part.

Systemic mechanisms regulate the water balance between the organism and the environment. It is regulated by biological (neurohumoral) mechanisms with the participation of such a high-level organ as the hypothalamus.

• Participation of the *volumoreceptors* of the vascular wall and the left atrium of the heart, which control the circulating blood volume.

• Participation of *osmoreceptors* of the vascular wall (arches of aorta and carotid sinus), which control blood osmotic pressure.

• Participation of *aldosterone* of the adrenal cortex, which regulates (increases) Na+ reabsorption from the primary urine into the blood (antinatriuretic mechanism).

• Participation of the *hypothalamus*, which reacts both to decreased blood volume and increased blood osmolarity in response to volumo- and osmoreceptor signals. It releases vasopressin (antidiuretic hormone, ADH) by the supraoptical and paraventricular hypothalamic nuclei. The point of vasopressin action is the renal tubule epithelium. Vasopressin increases water reabsorption from the primary urine and thus regulates diuresis (the primary urine quantity is 180 1/day; the final urine quantity — 1—2 1).

• Participation of the kidneys with their receptors and renin. Stimulation of the adducting renal arteriole volumoreceptors and of the osmoreceptors of the macula densa of the juxtaglomerular complex intensifies renin synthesis and release. Angiotensin II, which is formed under renin influence, increases aldosterone secretion and stimulates the thirst center located in the lateral part of the hypothalamus.

• Retention of water and Na+ in the organism is opposed by two mechanisms of natriuresis: 1) renomedullar prostaglandins;
2) atrial natriuretic factor (ANF, atriopeptide of 28 amino acids).

Water imbalance is divided into two varieties — ***positive and negative.***

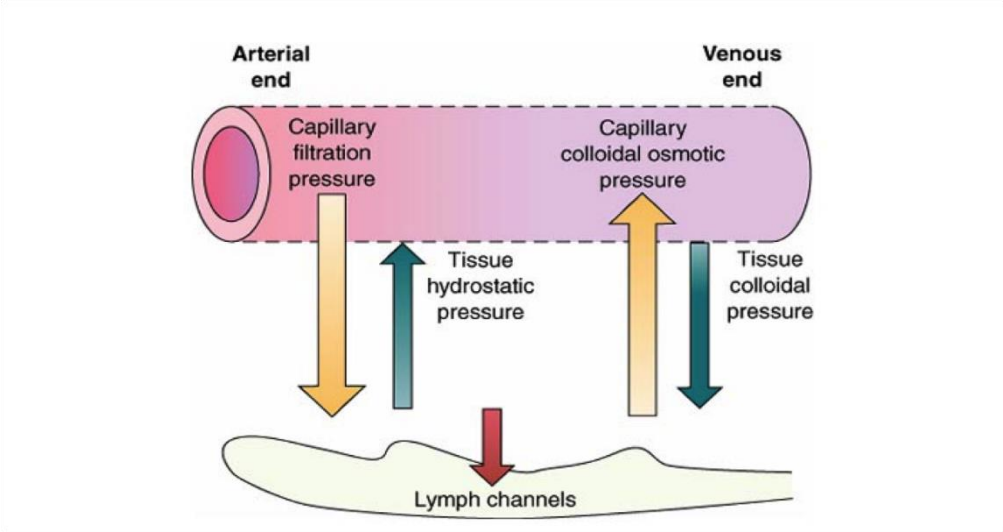
Positive water balance (hyperhydration) is water retention in the organism.

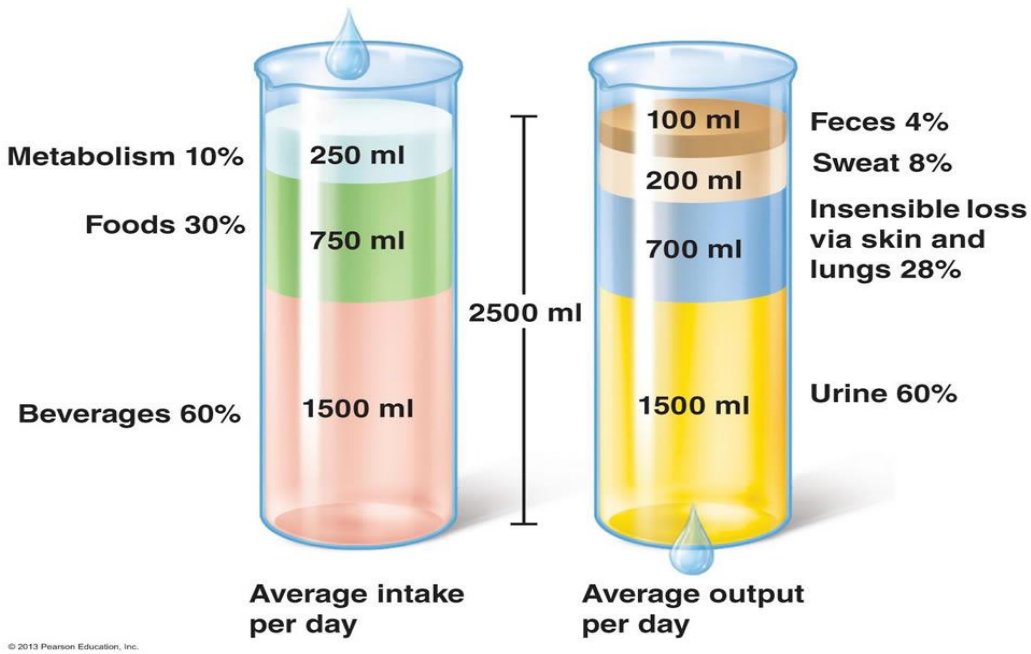
Negative water balance (hypohydration) is water loss by the organism.

Depending on osmotic concentration, hypo- and hyperhydration are subdivided into three types: *isoosmolar, hypoosmolar and hyperosmolar*. (The normal osmotic concentration of the blood and intercellular fluid is about 0.3 osmol/1 = 300

mosmol/1.)

In clinical practice it is manifested in the form of two syndromes — ***hyperhydration (edema) and dehydration.***





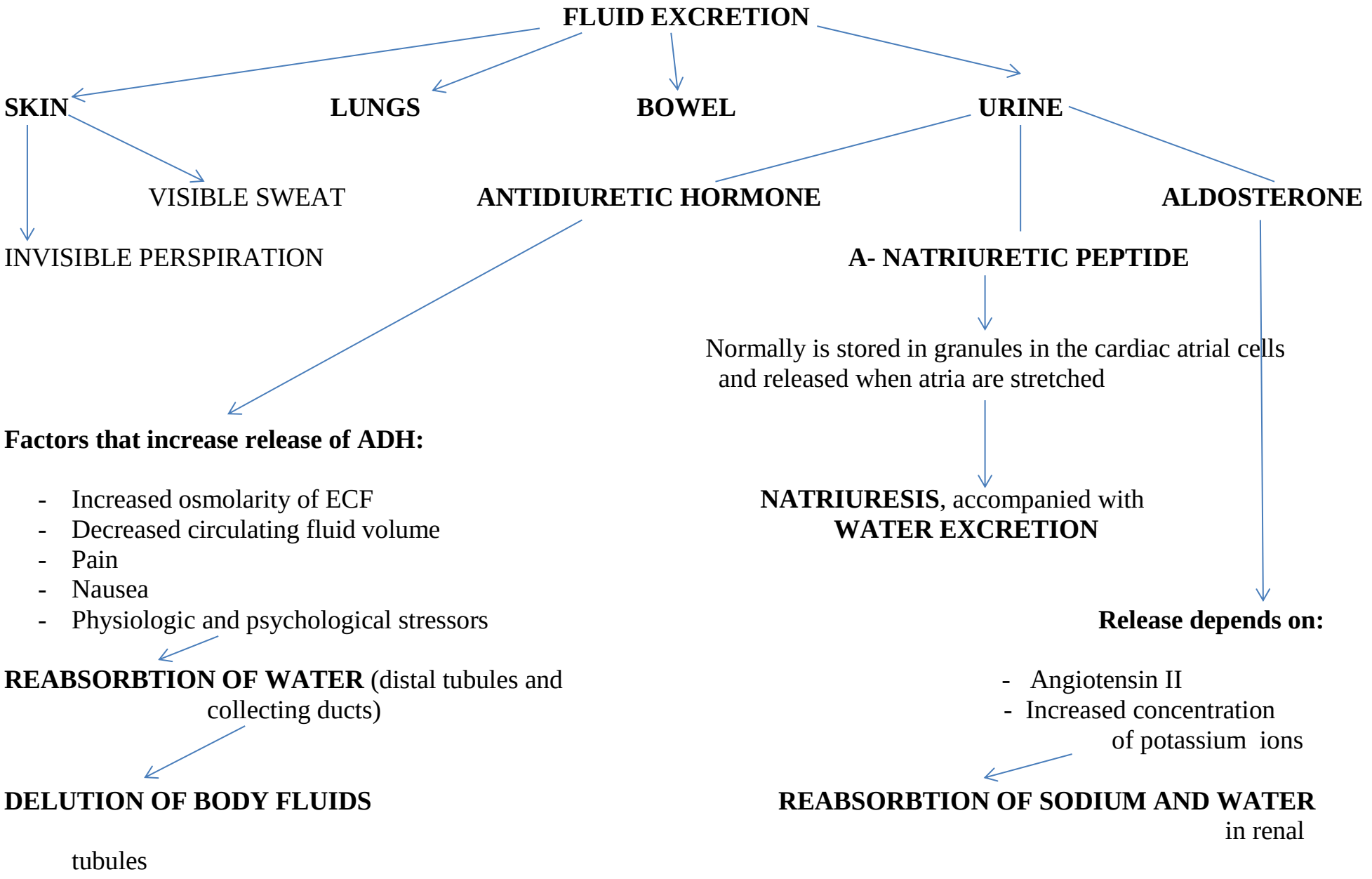


Fig. 4 Fluid Excretion

Edema is a typical pathological process, which is characterized by a positive water balance and accumulation of water in the interstitial space.

The principal pathogenic factors of edema are the following.

• Increased hydrodynamic pressure in the venous part of the vascular flow (local venous hyperemia, inflammation, and cardiac insufficiency).

• Decreased colloid osmotic blood pressure (in case of a decreased concentration of plasma proteins — hypoproteinemia, especially of highly hydrophilic albumins in fasting, nephritic syndrome, hepatic insufficiency, etc.).

• Increased permeability of the capillary vessels, which occurs:

• under the influence of BAS (histamine, serotonin, kinins, prostaglandins, etc.);

• in capillary wall dystrophy (fasting, disturbed neurotrophic supply, etc.).

• Increased colloid-osmotic pressure in tissues due to accumulation of osmotic and oncotic substances: electrolytes, proteins, metabolic products (in inflammation, allergy).

• Lymph outflow disorders (mechanic or dynamic lymphatic insufficiency).

• Disorders of the nervous and humoral regulation of the water-electrolyte balance («wrong switching» of the antidiuretic and antinatriuretic systems, as well as impaired sensitivity of volumo- and osmoreceptors, secondary aldosteronism, hypothyroidism).

Types

Edema is divided into ***local and systemic***.

According to pathogenesis (the main pathogenetic factor), edema is divided into

- ***hydrodynamic,***
- ***oncotic, osmotic,***
- ***membranogenic,***
- ***lymphogenic.***

Clinical practice divides edema according to localization and causes into

- ***inflammatory,***
- ***allergic,***
- ***toxic,***
- ***venous,***
- ***neurogenic,***
- ***lymphogenic (which are local)***
- ***cardiac,***
- ***fasting,***
- ***nephritic,***
- ***hepatic,***
- ***endocrine (which are systemic).***

The etiology and pathogenesis of each type of edema are different.

If left untreated, edema can cause:

1. Increasingly painful swelling
2. Difficulty walking
3. Stiffness
4. Stretched skin, which can become itchy and uncomfortable
5. Increased risk of infection in the swollen area
6. Scarring between layers of tissue
7. Decreased blood circulation
8. Decreased elasticity of arteries, veins, joints and muscles
9. Increased risk of skin ulcers

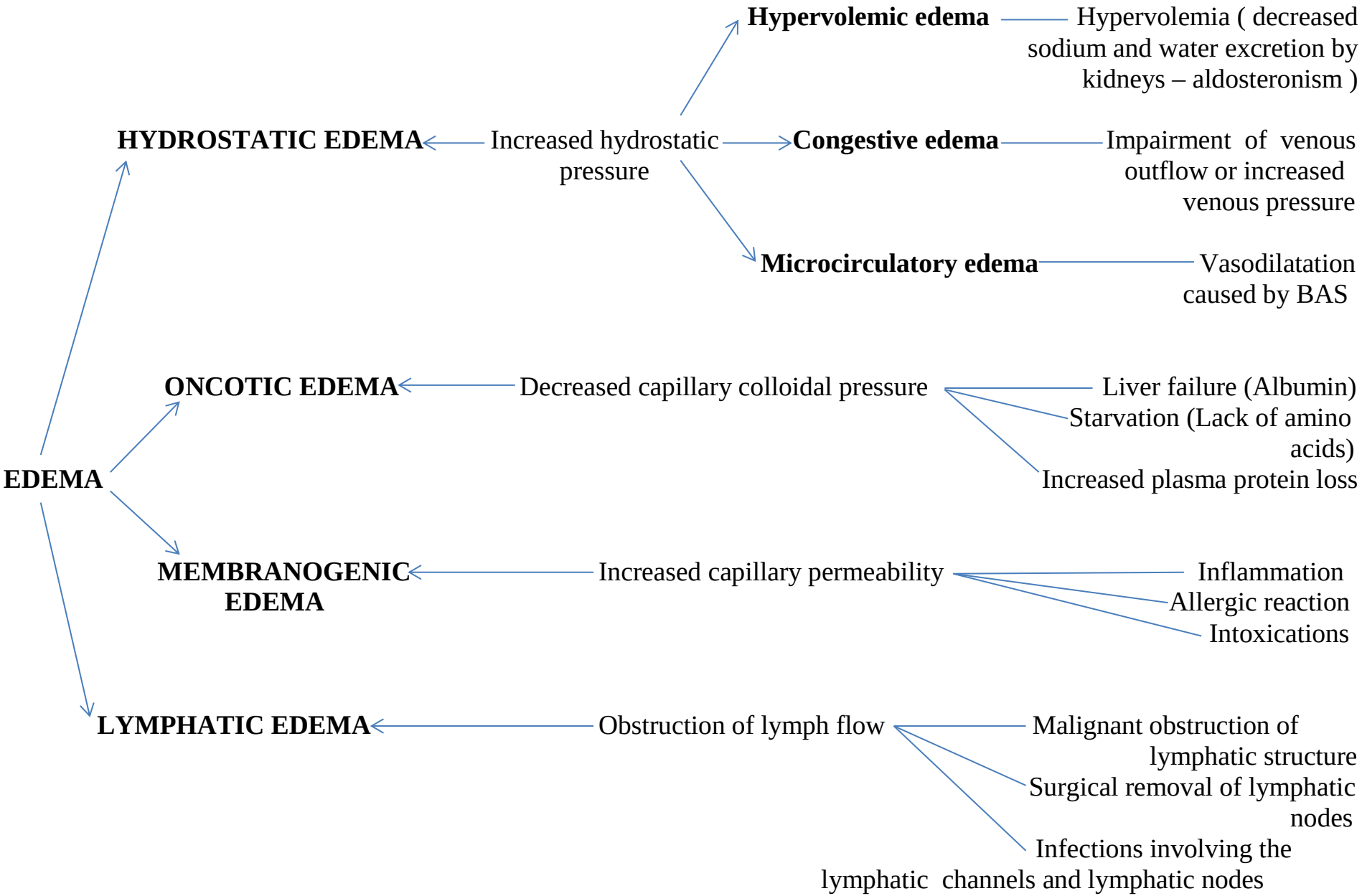

EDEMA
HYDROSTATIC EDEMA
Increased hydrostatic pressure
Hypervolemic edema
Hypervolemia (decreased sodium and water excretion by kidneys – aldosteronism)
Congestive edema
Impairment of venous outflow or increased venous pressure
Microcirculatory edema
Vasodilatation caused by BAS
ONCOTIC EDEMA
Decreased capillary colloidal pressure
Liver failure (Albumin)
Starvation (Lack of amino acids)
Increased plasma protein loss
MEMBRANOGENIC EDEMA
Increased capillary permeability
Inflammation
Allergic reaction
Intoxications
LYMPHATIC EDEMA
Obstruction of lymph flow
Malignant obstruction of lymphatic structure
Surgical removal of lymphatic nodes
Infections involving the lymphatic channels and lymphatic nodes

Fig. 5 Edema

Acid-base balance is maintenance of constant H+ concentration (medium pH). Different media of the organism have their own pH rates. The saliva and intestinal juice have alkaline reaction. The gastric juice contains a large amount of free hydrochloric acid (HC1) and has high acidity reaction (pH approaches 1). Blood pH is 7.35-7.45. The level of blood pH less than 6.8 or higher than 7.8 is not compatible with life. Excessive formation of H+ ions leads to pH reduction. A decrease of H+ ion formation elevates pH.

Mechanisms of Acid-Base Balance Regulation

Since the majority of intermediates (products of intermediate metabolism) are acids, pH regulation is provided continually. Many mechanisms (in the liquid media, blood and cells) are constantly participating in acid-base balance regulation.

1. **Buffer systems** neutralize surplus of acids and alkalis, transferring them into a form convenient for further secretion by the lungs and kidneys.
   - Hydrocarbonate buffer system H2C03/NaHC03 = 1/20 maintains pH in the blood plasma and interstitial fluid. This buffer has a volatile form of acid (CO), which can be easily excreted by the lungs.
   - Phosphate buffer system NaH2P04/Na2H P04 = 1/4 participates in acidbase regulation in the kidneys.
   - Hemoglobin buffer functions in erythrocytes.
   - Protein buffer regulates intracellular pH.

2. **Lungs** role consists in constant discharge of carbon acid in the form of carbon dioxide C02.

3. **Kidneys** role consists in acid-base regulation through three mechanisms:

- Acidogenesis is secretion of H+ ions into the renal tubules.
- Ammoniogenesis is formation and secretion of ammonia ion (NH3) into the renal tubules. Then, NH3 reacts with H+ to form NH4+. Afterwards it is accompanied by anion CI". Neutral ammonium salt NH4C1 is formed and excreted in the urine.
- Reabsorption of bicarbonate (NaHC03) in the renal tubules.

Consequently, urine examination reflects the acid-base state. Two urine indices are of practical use. They are:

- urinary acidity tested by titration;
- ammonium salt content.

4. **Aldosterone** (hormone of the adrenal cortex) supports acidogenesis in the kidneys, participates in H+ and Na+ exchange (H+ ion secretion and Na+ ion reabsorption).

Etiological factors of acid-base balance disorder can be *exogenous or endogenous; physical, chemical and biological.*

Exogenous causes are the following:

- mechanical trauma of the chest that impairs gas exchange in the lungs;
- excessive intake of acid or alkaline products (mineral water, different diets);

• oxygen deficiency in the inhaled air and accumulation of incompletely oxidized metabolites in the organism;

• surplus of C02 in the inhaled air (submarine accidents);

• poisoning that manifests itself through vomiting (loss of acid) and diarrhea (loss of alkalis);

• starvation;

• infection that results in pulmonary or renal insufficiency and in disorder of the main physiological mechanisms of acid-base balance regulation.

Endogenous causes are the following:

• disorders in metabolism regulation and accumulation of acid intermediates (DM);

• pathology of the organs that participate in acid-base balance regulation (inflammation, vascular disorders leading to malfunctioning of the lungs, kidneys, and adrenal glands);

• various diseases that manifest themselves through vomiting, diarrhea, or excessive salivation.

PATHOGENESIS

Stage of compensation manifests itself through normal blood pH in spite of etiological factor influence. It is possible due to mechanisms of compensation.

Stage of decompensation develops if mechanisms of compensation are exhausted. Dysfunction of the organs, which participate in pH regulation, provokes decompensation.

CLASSIFICATION

There are several classifications of acid-base balance disorders depending on the principle, which is assumed as a basis.

Acidosis (decreased pH) and alkalosis (increased pH) are distinguished according to the direction of acid-base disorder.

In their turn, each of them is further subdivided. ***Compensated and decompensated.***

Gaseous and non-gaseous (syn. respiratory and non-respiratory) — connected or not connected with volatile acids (carbon dioxide) or organic acids.

Exogenous and endogenous. This classification is based on the origin of the etiological factor. The endogenous type is subdivided into two types — ***metabolic*** (accumulation of acid metabolites) and ***excretory disorders*** (kidney or lung insufficiency).

Acute and chronic disorders reflect the type of clinical duration.

Local (pH changes in tissues) and ***systemic*** (in the blood).

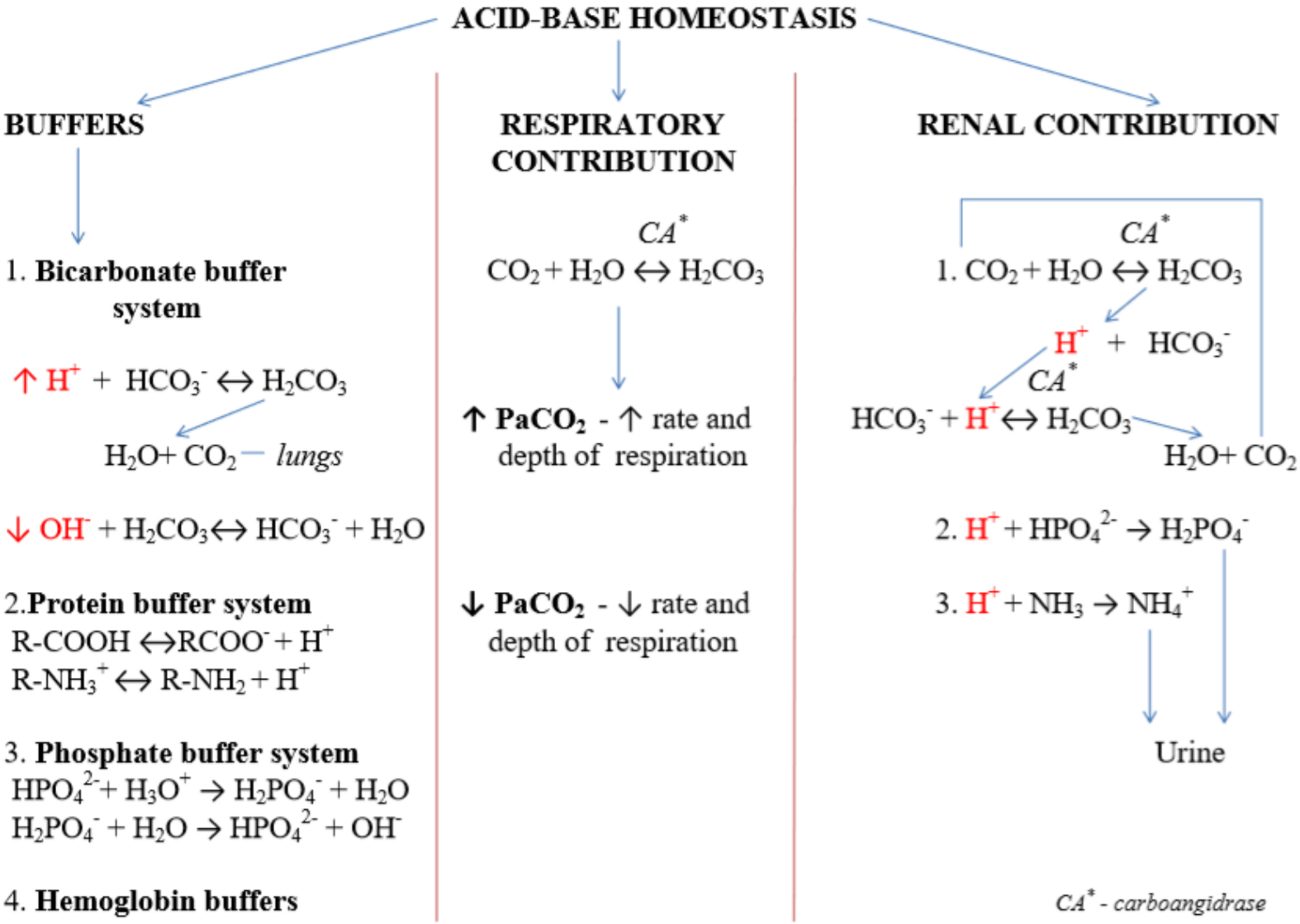


Fig. 6 Acid-base homeostasis

ACID-BASE BALANCE DISORDERS

	METABOLIC ACIDOSIS	***METABOLIC ALCALOSIS***	***RESPIRATORY ACIDOSIS***	***RESPIRATORY ALCALOSIS***
CAUSES	Ketoacidosis; burns; tissue hypoxia; kidney diseases (oliguria); diarrhea; intestinal decompression.	Excessive intake of HCO_3^-; massive transfusion with citrated blood; emesis; gastric suction; hyperaldosteronism; hypokaliemia.	COPD; pneumonia; severe asthma episode; pulmonary edema; kyphoscoliosis; chest injury; Respiratory depressant drugs.	Hypoxemia; acute pain; alcohol withdrawal; stimulation of the brainstem.
SYMPTOMS	Headache; Abdominal pain; Central nervous system depression.	Postural hypotension; Bilateral muscular weakness; tetany; confusion; lethargy; coma	Headache; Tachycardia; Cardiac dysrhythmias; Neurologic abnormalities.	Paresthesias; Carpal and pedal spasms; Cerebral vasoconstriction.
LABORATORY CRITERIA	pH < 7,36; HCO_3^- < 22 mmol/l	pH > 7,44; HCO_3^- > 26 mmol/l	pH < 7,36; $PaCO_2$ > 44 mm Hg	pH > 7,44; $PaCO_2$ < 36 mm Hg
UNCOMPENSATED STAGE	$\frac{\downarrow [HCO_3^-]}{N\,[H_2CO_3]} = \downarrow$ pH	$\frac{\uparrow [HCO_3^-]}{N\,[H_2CO_3]} = \uparrow$ pH	$\frac{N\,[HCO_3^-]}{\uparrow [H_2CO_3]} = \downarrow$ pH	$\frac{N\,[HCO_3^-]}{\downarrow [H_2CO_3]} = \uparrow$ pH
COMPENSATED STAGE	$\frac{\downarrow [HCO_3^-]\ primary}{\downarrow [H_2CO_3]\ compensatory}$	$\frac{\uparrow [HCO_3^-]\ primary}{\uparrow [H_2CO_3]\ compensatory}$	$\frac{\uparrow [HCO_3^-]\ compensatory}{\uparrow [H_2CO_3]\ primary}$	$\frac{\downarrow [HCO_3^-]\ compensatory}{\downarrow [H_2CO_3]\ primary}$
COMPENSATION BY…	Hyperventilation → ↓ $PaCO_2$	Hypoventilation → ↑ $PaCO_2$	Excretion of H^+ by kidneys → ↑ HCO_3^- *(retention of HCO_3^-)*	Excretion of HCO_3^- by kidneys → ↑ H^+ *(retention of H^+)*

Fig. 7 Acide-base balance disodrers

KROK 1 _ mcqs (A is correct answer) :

1.A patient with chronic heart failure with edema has increased level of blood aldosterone.
What diuretic would be most effective in this case?

A. Spironolactone
B. Triamterene
C. Acetazolamide
D. Hydrochlorothiazide
E. Furosemide

2. 12-year-old teenager has significantly put off weight within 3 months; glucose concentration rose up to 50 mmol/l. He fell into a coma. What is the main mechanism of its development?

A. Hyperosmolar
B. Hypoglycemic
C. Lactacidemic
D. Ketonemic
E. Hypoxic

3. Despite the administration of cardiotonics and a thiazide diuretic a patient with chronic heart failure has persistent edemata, there is a risk of ascites. What medication should be administered in order to enhance the diuretic effect of the drugs used?

A. Spironolactone
B. Furosemide
C. Amiloride
D. Clopamide
E. Manithol

4. A patient with a pathology of the cardiovascular system developed edemata of the lower extremities. What is the mechanism of cardiac edema development?

A. Increased hydrostatic pressure at the venous end of the capillary
B. Increased oncotic pressure
C. Increased hydrostatic pressure at the arterial end of the capillary
D. Reduced osmotic pressure
E. Lymph efflux disorder

5. A patient who had been continuously taking drugs blocking the production of angiotensin II developed bradycardia and arrhythmia. A likely cause of these disorders is:

A. Hyperkalemia
B. Hypokalemia
C. Hypernatremia
D. Hypocalcemia
E. Hypercalcemia

6. In course of an experiment there has been an increase in the nerve conduction velocity. This may be caused by an increase in the concentration of the following ions that are present in the solution around the cell:

A. Na+
B. K+ and Cl−
C. K+ and Na+
D. Ca2+ and Cl−
E. Ca2+

7. A blood drop has been put into a test tube with 0,3% solution of NaCl. What will happen to erythrocytes?

A. Osmotic hemolysis
B. Shrinkage
C. Mechanical hemolysis
D. Any changes will be observed
E. Biological hemolysis

8. A patient has an increased pyruvate concentration in blood. A large amount of it is excreted with

the urine. What vitamin is lacking in this patient?

A. B1
B. B2
C. B3
D. B6
E. E

9. A patient presents with twilight vision impairment. Which of the following vitamins should be administered?

A. Retinol acetate
B. Pyridoxine hydrochloride
C. Cyanocobalamin
D. Nicotinic acid
E. Ascorbic acid

10. 50-year-old patient complains about general weakness, appetite loss and cardiac arrhythmia. The patient presents with muscle hypotonia, flaccid paralyses, weakened peristaltic activity of the bowels. Such condition might be caused by:

A. Hypokaliemia
B. Hypophosphatemia
C. Hyperkaliemia
D. Hyponatremia
E. Hypoproteinemia

11. A patient underwent an operation on account of gall bladder excision that resulted in obstruction of Ca absorption through the bowels wall. What vitamin will stimulate this process?

A. D3
B. PP
C. C
D. B12
E. Increased production of glucocorticoids

12. A doctor recommends a patient with duodenal ulcer to drink cabbage and potato juice after the therapy course. Which substances contained in these vegetables help to heal and prevent the ulcers?

A. Vitamin U
B. Pantothenic acid
C. Vitamin C
D. Vitamin B1
E. Vitamin K

13. By massive diarrhea has low water rate in the extracellular space, high water rate inside the cells and low blood osmolarity. What is such disturbance of water-electrolytic metabolism called?

A. Hypoosmolar hypohydration
B. Hyperosmolar hypohydration
C. Osmolar hypohydration
D. Hypoosmolar hyperhydration
E. Hyperosmolar hyperhydration

14. After severe viral hepatitis a 4 year old boy presents with vomiting, occasional loss of consciousness, convulsions. Blood test revealed hyperammoniemia. Such condition is caused by a disorder of the following biochemical hepatic process:

A. Disorder of ammonia neutralization
B. Disorder of biogenic amines neutralization
C. Protein synthesis inhibition
D. Activation of amino acid decarboxylation
E. Inhibition of transamination enzymes

15. A 26 year old pregnant woman is under treatment at an in-patient hospital. After a continuous attack of vomiting she was found to have reduced volume of circulating blood. What kind of change in general blood volume is the case?

A. Polycythemic hypovolemia
B. Simple hypovolemia

C. Oligocythemic hypovolemia
D. Polycythemic hypervolemia
E. Oligocythemic hypervolemia

16. Inflamation is characterised by increasing penetration of vessels of microcirculation stream, increasing of their fluid dynamic blood pressure. Increasing of the osmotic concentration and dispersity of protein structures present in the intercellular fluid. What kind of edema will appear in this case?

A. Mixed
B. Hydrodynamic
C. Colloid-osmotic
D. Lymphogenic
E. Membranogenic

17. Some diseases reveal symptoms of aldosteronism with hypertension and edema due to sodium retention in the organism. What organ of the internal secretion is affected on aldosteronism?

A. Adrenal glands
B. Testicle
C. Ovaries
D. Pancreas
E. Hypophysis

18. Inflammation is characterised by increasing penetration of vessels of microcirculation stream, increasing of their fluid dynamic blood pressure. Increasing of the osmotic concentration and dispersity of protein structures can be found in the intercellular fluid. What kind of edema are to be observed in this case?

A. Mixed
B. Hydrodynamic
C. Colloid-osmotic
D. Lymphogenic
E. Membranogenic

19. The concentration of albumins in human blood sample is lower than normal. This leads to edema of tissues. What blood function is damaged?

A. Maintaining the oncotic blood pressure
B. Maintaining the pH level
C. Maintaining the body temperature
D. Maintaining the blood sedimentation system
E. All answers are correct

20. Chronic glomerulonephritis was diagnosed in a 34-year-old patient 3 years ago. Edema has developed within the last6 months. What caused the edema?

A. Proteinuria
B. Hyperproduction of vasopressin
C. Liver disfunction of protein formation
D. Hyperosmolarity of plasma
E. Hyperaldosteronism

21. A person has reduced diuresis, hypernatremia, hypokalemia. Hypersecretion of what hormone can cause such changes?

A. Aldosterone
B. Vasopressin
C. Auricular sodiumuretic factor
D. Adrenalin
E. Parathormone

22. A patient who suffers from severe disorder of water-salt metabolism experienced cardiac arrest in diastole. What is the most probable mechanism of cardiac arrest in diastole?

A. Hyperkaliemia
B. Hypernatremia
C. Organism dehydratation
D. Hypokaliemia
E. Hyponatremia

23. A patient who suffers from heart failure has enlarged liver, edemata of lower extremities, ascites. What is the leading mechanism in the development of this edema?

A. Hydrodynamic
B. Colloid osmotic
C. Lymphogenous
D. Membranogenic
E. -

24. Osmotic pressure of a man's blood plasma is 350 mosmol/l (standard pressure is 300 mosmol/l). First of all it will result in high secretion of the following hormone:

A. Vasopressin
B. Aldosteron
C. Cortisol
D. Adrenocorticotropin
E. Natriuretic

25. A hypertensive glucose solution was introduced to a patient. It will intensify water movement:

A. From the cells to the intercellular liquid
B. From the intercellular liquid to the capillaries
C. From the intercellular liquid to the cells
D. From the capillaries to the intercellular liquid
E. There will be no changes of water movement

26. A concentrated solution of sodium chloride was intravenously injected to an animal. This caused decreased reabsorption of sodium ions in the renal tubules. It is the result of the following changes of hormonal secretion:

A. Aldosterone reduction
B. Aldosterone increase
C. Vasopressin reduction
D. Vasopressin increase
E. Reduction of atrial natriuretic factor

27. A patient has a decreased vasopressin synthesis that causes polyuria and as a result of it evident organism dehydration. What is the mechanism of polyuria development?

A. Reduced tubular reabsorption of water
B. Reduced tubular reabsorption of *Na* ions
C. Reduced tubular reabsorption of protein
D. Reduced glucose reabsorption
E. Acceleration of glomerular filtration

28. A patient is 44 years old. Laboratory examination of his blood revealed that content of proteins in plasma was 40 g/l. What influence will be exerted on the transcapillary water exchange?

A. Filtration will be increased, reabsorption- decreased
B. Both filtration and reabsorption will be increased
C. Both filtration and reabsorption will be decreased
D. Filtration will be decreased, reabsorption- increased
E. Exchange will stay unchanged

29. Packed cell volume of a man was40% before the trauma. What packed cell volume will be observed 24 hours after blood loss of 750 ml?

A. 30%
B. 40%
C. 55%
D. 45%
E. 50%

30. A patient ill with enteritis accompanied by massive diarrhea has low water rate in the extracellular space, high water rate

inside the cells and low blood osmolarity. What is such disturbance of water-electrolytic metabolism called?

A. Hypoosmolar hypohydration
B. Hyperosmolar hypohydration
C. Osmolar hypohydration
D. Hypoosmolar hyperhydration
E. Hyperosmolar hyperhydration

31. A 45 year old woman is ill with breast cancer. Her left arm has symptoms of lymphatic system insufficiency – limb edema, lymph node enlargement. What form of lymphatic circulation insufficiency is it?

A. Mechanic insufficiency
B. Dynamic insufficiency
C. Resorption insufficiency
D. Combined insufficiency
E. -

32. A patient who suffers from severe disorder of water-salt metabolism experienced cardiac arrest in diastole. What is the most probable mechanism of cardiac arrest in diastole?

A. Hyperkaliemia
B. Hypernatremia
C. Organism dehydratation
D. Hypokaliemia
E. Hyponatremia

33. A patient who suffers from heart failure has enlarged liver, edemata of lower extremities, ascites. What is the leading mechanism in the development of this edema?

A. Hydrodynamic
B. Colloid osmotic
C. Lymphogenous
D. Membranogenic
E. -

34. As a result of increased permeability of the erythrocyte membrane in a patient with microspherocytic anaemia (Minkowsky-Shauffard disease) cells receive sodium ions and water. Erythrocytes take form of spherocytes and can be easily broken down. What is the leading mechanism of erythrocyte damage in this case?

A. Electrolytic osmotic
B. Calcium
C. Acidotic
D. Protein
E. Nucleic

35. A 56 year old patient suffering from cardiac insufficiency has edema of feet and shins, edematous skin is pale and cold. What is the leding mechanism of edema pathogenesis?

A. Rise of hydrostatic pressure in venules
B. Drop of oncotic pessure in capillaries
C. Increase of capillary permeability
D. Disorder of lymph outflow
E. Positive water balance

36. A patient with nephrotic syndrome has massive edemata of his face and limbs. What is the leading pathogenetic mechanism of edemata development?

A. Drop of oncotic blood pressure
B. Increase of vascular permeability
C. Rise of hydrodynamic blood pressure
D. Lymphostasis
E. Increase of lymph outflow

37. A patient ill with enteritis accompanied by massive diarrhea has low water rate in the extracellular space, high water rate inside the cells and low blood osmolarity. What is such

disturbance of water-electrolytic metabolism called?

A. Hypoosmolar hypohydration
B. Hyperosmolar hypohydration
C. Osmolar hypohydration
D. Hypoosmolar hyperhydration
E. Hyperosmolar hyperhydration

38. A patient was admitted to the infectious department. His symptoms: dry skin, decreased skin turgor, rice-water stool. The patient was diagnosed with cholera. What disorder of water- electrolytic balance is most often observed in this disease?

A. Isoosmotic hypohydration
B. Hyperosmotic hyperhydration
C. Hypoosmotic hypohydration
D. Hyperosmotic hypohydration
E. Hypoosmotic hyperhydration

39.A patient with diabetes developed a diabetic coma due to the acid-base imbalance.
Specify the kind of this imbalance:

A. Metabolic acidosis
B. Metabolic alkalosis
C. Respiratory acidosis
D. Gaseous alkalosis
E. Non-gaseous alkalosis

40. A patient with respiratory failure has blood pH of 7,35. pCO2 test revealed hypercapnia. Urine pH test revealed an increase in the urine acidity. What form of acid-base imbalance is the case?

A. Compensated respiratory acidosis
B. Compensated metabolic acidosis
C. Decompensated metabolic acidosis
D. Compensated respiratory alkalosis
E. Decompensated respiratory alkalosis

41. A hypertensive patient had been keeping to a salt-free diet and taking antihypertensive drugs together with hydrochlorothiazide for a long time. This resulted in electrolyte imbalance. What disorder of the internal environment occurred in the patient?

A. Hypochloremic alkalosis
B. Metabolic acidosis
C. Hyperkalemia
D. Hypermagnesemia
E. Increase in circulating blood volume

42. After taking poor-quality food a patient developed repeated episodes of diarrhea. On the next day he presented with decreased arterial pressure, tachycardia, extrasystole. Blood pH is 7,18. These abnormalities were caused by the development of:

A. Nongaseous acidosis
B. Nongaseous alkalosis
C. Gaseous alkalosis
D. Metabolic alkalosis
E. Gaseous acidosis

43. A 12-year-old teenager has significantly put off weight within 3 months; glucose concentration rose up to 50 mmol/ l. He fell into a coma. What is the main mechanism of its development?

A. Hyperosmolar
B. Hypoglycemic
C. Ketonemic
D. Lactacidemic
E. Hypoxic

44. An infant has pylorospasm, weakness, hypodynamia, convulsions as a result of frequent vomiting.What kind of acid-base dysbalance is it?

A. Excretory alkalosis
B. Excretory acidosis

C. Metabolic acidosis
D. Exogenous nongaseous acidosis
E. Gaseous alkalosis

45. A 2 year old child with mental and physical retardation has been delivered to a hospital. He presents with frequent vomiting after having meals. There is phenylpyruvic acid in urine. Which metabolism abnormality is the reason for this pathology?
A. Amino-acid metabolism
B. Lipid metabolism
C. Carbohydrate metabolism
D. Water-salt metabolism
E. Phosphoric calcium metabolism

46. An infant has pylorospasm, weakness, hypodynamia, convulsions as a result of frequent vomiting. What kind of acid-base dysbalance is it?
A. Excretory alkalosis
B. Excretory acidosis
C. Metabolic acidosis
D. Exogenous nongaseous acidosis
E. Gaseous alkalosis

47. After taking poor-quality food a patient developed repeated episodes of diarrhea. On the next day he presented with decreased arterial pressure, tachycardia, extrasystole. Blood pH is 7,18. These abnormalities were caused by the development of:
A. Nongaseous acidosis
B. Gaseous acidosis
C. Nongaseous alkalosis
D. Gaseous alkalosis
E. Metabolic alkalosis

48. Only one factor can influence the charge of amino acid radicals in the active center of enzyme. Name this factor:
A. pH medium
B. Pressure
C. Temperature
D. The presence of a competitive inhibitor
E. The surplus of a product

49. A worker has decreased buffer capacity of blood due to exhausting muscular work The influx of what acid substance in the blood can cause this symptom?
A. Lactate
B. Pyruvate
C. 1,3-bisphosphoglycerate
D. α-ketoglutarate
E. 3-phosphoglycerate

50. A pregnant woman had been having toxicosis with severe repeated vomiting for 24 hours. In the end of the day there appeared tetanic convulsions and fluid loss. What shift of acid-base state caused these changes?
A. Excretory alkalosis
B. Gaseous alkalosis
C. Gaseous acidosis
D. Metabolic acidosis
E. Excretory acidosis

51. As a result of exhausting muscular work a worker has largely reduced buffer capacity of blood. What acidic substance that came to blood caused this phenomenon?
A. Lactate
B. Pyruvate
C. 1,3-bisphosphoglycerate
D. 3-phosphoglycerate
E. -

52. A group of mountain climbers went through the blood analysis at the height of 3000 m. It revealed decrease of*HCO*3 to 15 micromole/l (standard is 22-26 micromole/l). What is the mechanism of *HCO*3 decrease?

A. Hyperventilation
B. Intensification of acidogenesis
C. Hypoventilation
D. Decrease of ammoniogenesis
E. Decrease of bicarbonate reabsorption in kidneys

53. A 35 y.o. patient who often consumes alcohol was treated with diuretics. There appeared serious muscle and heart weakness, vomiting, diarrhea, AP- 100/60mm Hg, depression. This condition is caused by intensified excretion with urine of:
A. Potassium
B. Sodium
C. Chlorine
D. Calcium
E. Phosphates

54. Examination of a patient revealed hyperkaliemia and hyponatremia. Low secretion of which hormone may cause such changes?
A. Aldosteron
B. Vasopressin
C. Cortisol
D. Parathormone
E. Natriuretic

55. A newborn child with pylorostenosis has often repeating vomiting accompanied by apathy, weakness, hypertonicity, sometimes convulsions. What disorder form of acid-base balance is it?
A. Nongaseous alkalosis
B. Gaseous alkalosis
C. Gaseous acidosis
D. Metabolic acidosis
E. Excretory acidosis

56. An infant has apparent diarrhea resulting from improper feeding. One of the main diarrhea effects is plentiful excretion of sodium bicarbonate. What form of acid-base balance disorder is the case?
A. Metabolic acidosis
B. Metabolic alkalosis
C. Respiratory acidosis
D. Respiratory alkalosis
E. No disorders of acid-base balance will be observed

57. Disorder of the airways passage in small and middle bronchi was revealed in the patient.
What disorder of the acid-base equilibrium can be detected in the blood?
A. Respiratory acidosis
B. Metabolic acidosis
C. Respiratory alkalosis
D. Metabolic alkalosis
E. --

58. A newborn child with pylorostenosis has often repeating vomiting accompanied by apathy, weakness, hypertonicity, sometimes convulsions. What disorder form of acid-base balance is it?
A. Nongaseous alkalosis
B. Gaseous alkalosis
C. Gaseous acidosis
D. Metabolic acidosis
E. Excretory acidosis

59. An infant has pylorospasm, weakness, hypodynamia, convulsions as a result of frequent vomiting. What kind of acid-base dysbalance is it?
A. Excretory alkalosis
B. Excretory acidosis
C. Metabolic acidosis
D. Exogenous nongaseous acidosis
E. Gaseous alkalosis

60. After taking poor-quality food a patient developed repeated episodes of diarrhea. On the next day he presented with decreased arterial

pressure, tachycardia, extrasystole. Blood pH is 7,18. These abnormalities were caused by the development of:

A. Nongaseous acidosis
B. Gaseous acidosis
C. Nongaseous alkalosis
D. Gaseous alkalosis
E. Metabolic alkalosis

61. A newborn child with pylorostenosis has often repeating vomiting accompaniedby apathy, weakness, hypertonicity, sometimes convulsions. What disorder form of acid-base balance is it?

A. Nongaseous alkalosis
B. Metabolic acidosis
C. Gaseous alkalosis
D. Gaseous acidosis
E. Excretory acidosis

62. Glomerular filtration of a person, who has been starving for a long time, has increased by 20%. The most likely cause of filtration changes in the given conditions is:

A. Decrease of blood plasma oncotic pressure
B. Increase of systemic blood pressure
C. Increase of renal filter permeability
D. Increase of filtration factor
E. Increase of renal plasma flow

63. Atria of an experimental animal were superdistended with blood, which resulted in decreased reabsorption of *Na+* and water in renal tubules. This can be explained by the influence of the following factor on kidneys:

A. Natriuretic hormone
B. Aldosterone
C. Renin
D. Angiotensin
E. Vasopressin

64. For people adapted to high external temperatures profuse sweating is not accompanied by loss of large volumes of sodium chloride. This is caused by the effect the following hormone has on the perspiratory glands:

A. Aldosterone
B. Vasopressin
C. Cortisol
D. Tgyroxin
E. Natriuretic

65. An infant has pylorospasm, weakness, hypodynamia, convulsions as a result of frequent vomiting. What kind of acid-base disbalance is it?

A. Excretory alkalosis
B. Excretory acidosis
C. Metabolic acidosis
D. Exogenous nongaseous acidosis
E. Gaseous alkalosis

66. A 30-year-old man with diabetes mellitus type I was hospitalised. The patient is comatose. Laboratory tests revealed hyperglycemia and ketonemia. What metabolic disorder can be detected in this patient?

A. Metabolic acidosis
B. Metabolic alkalosis
C. Respiratory acidosis
D. Respiratory alkalosis
E. Normal acid-base balance

67. A patient suffers from disrupted patency of the airways at the level of small and medium-sized bronchial tubes. What changes of acid-base balance can occur in the patient?

A. Respiratory acidosis
B. Respiratory alkalosis
C. Metabolic acidosis
D. Metabolic alkalosis
E. Acid-base balance remains unchanged

68.Ketosis develops in the patients with diabetes mellitus, as the result of activation of fatty acids oxidation processes. What acidbase imbalance can result from accumulation of excessive ketone bodies in the blood?
A. Metabolic acidosis
B. Metabolic alkalosis
C. No imbalance occurs
D. Respiratory acidosis
E. Respiratory alkalosis
69.A woman with hypophyseal diabetes insipidus developed a water-mineral imbalance. What type of water-mineral imbalance develops in such cases?
A. Hyperosmolar dehydration
B. Hypoosmolar dehydration
C. Isoosmolar dehydration
D. Hypoosmolar hyperhydration
E. Hyperosmolar hyperhydration
70.A 40-year-old man with impaired venous patency in the lower limbs developed edemas. What mechanism plays the main role in the development of this disturbance?
A. Elevated filtration pressure
B. Positive fluid balance
C. Decreased gradient of osmotic pressure
between blood and tissue
D. Disturbed humoral regulation of watermineral balance
E. Hypoproteinemia

Tests for Self-Control

1. A 38-year-old patient with a history of hepatitis abused alcohol. Over time symptoms of cirrhosis appeared with ascites and edema of the lower extremities. What type of changes in blood composition caused edema development?
A. Hypocholesterinemia.
B. Hypoglobulinemia.
C. Hypoalbuminemia.
D. Hypokalemia.
E. Hypoglycemia.
2. A girl is 6 years old. After eating an orange she suffered from edemas of the eyelids, lips and tongue mucosa. Earlier after eating oranges rash and itch appeared. What pathogenetic mechanism is the main one in girl edematization?
A. Decreased oncotic blood pressure.
B. Disorder of lymph drainage.
C. Increased oncotic blood pressure.
D. Increased permeability of the capillaries.
E. Increased hydrostatic blood pressure in the capillaries.
3. A patient with third-degree burns had hypoproteinemia and edemas. What is the basic mechanism of such edemas?
A. Increase of oncotic blood pressure.
B. Decrease of oncotic blood pressure.
C. Increase of hydrostatic pressure of the venous blood.
D. Decrease of hydrostatic pressure of the arterial blood.
E. Decrease of circulating blood volume.
4. A person who was stung by bees suffers from edema of the upper extremities and face. What is the basic pathogenetic mechanism of edema development?

A. Increased permeability of the vessel walls.
B. Increased hydrostatic pressure in the capillaries.
C. Decreased hydrostatic blood pressure.
D. Increased oncotic tissue pressure.
E. Decreased oncotic blood pressure.

5. Complete (with water deprivation) nutritional starvation is associated with generalized edemas. What is the main mechanism of edema development in such a case?
A. Decrease of hydrostatic blood pressure.
B. Decrease of oncotic blood pressure.
C. Decrease of osmotic blood pressure.
D. Increased oncotic pressure of the intracellular fluid.
E. Increased osmotic pressure of the intracellular fluid.

6. A patient is ill with pneumosclerosis. Pulmonary hypertension and right ventricular heart failure with ascites and edemas have developed. What is the basic pathogenetic mechanism of edema development?
A. Increased hydrostatic vein pressure.
B. Increased oncotic tissue pressure.
C. Decreased oncotic blood pressure.
D. Decreased osmotic blood pressure.
E. Increased permeability of vessels.

7. A dog who was put in a thermostat at a temperature of 40°C showed respiration rate increase. What kind of water-electrolyte imbalance occurred?
A. Isoosmolar dehydration.
B. Hypoosmolar dehydration.
C. Hyperosmolar dehydration.
D. Positive water balance.
E. Hypoosmolar hyperhydration.

8. A patient was hospitalized to an infectious diseases hospital with complaints of intractable vomiting. What type of water-salt imbalance developed?
A. Hypoosmolar dehydration.
B. Isoosmolar dehydration.
C. Hyperosmolar dehydration.
D. Hypoosmolar hyperhydration.
E. Hyperosmolar hyperhydration.

9. A patient used mineralocorticoids for a long time. As a result muscle weakness developed. What has caused the development of such failure?
A. Hyponatremia.
B. Hyperkalemia.
C. Hypernatremia.
D. Hypokalemia.
E. Hypervolemia.

10. While climbing a mountain a climber feels activation of breathing, dyspnea, headache, and palpitation. What type of acid-base imbalance has developed?
A. Respiratory alkalosis.
B. Metabolic alkalosis.
C. Non-respiratory alkalosis.
D. Respiratory acidosis.
E. Excretory acidosis.

Recommended literature:

Basic

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4. Porth, Carol. Essentials of pathophysiology: concepts of altered health states /Carol Mattson Porth ; consultants, Kathryn J. Gaspard, Kim A. Noble. — 3rd ed. 2011 Wolters Kluwer Health | Lippincott Williams & Wilkins. – 2011. – 1282 p.
5. Robbins Pathology basis of disease / Cotran R.S., Kumar V., Robbins S.L. - 2000.