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Federal State Budgetary Educational Institution of Higher Education "North Ossetian State Medical Academy" of the Ministry of Health of the Russian Federation

Department of Pathological Physiology

APPROVED

Minutes of the meeting
of the Central Coordinating
Educational and Methodological Council
on May 23, 2023, Protocol No. 5

EVALUATION MATERIALS

in the discipline of Pathophysiology, clinical pathophysiology for students of the 3rd year of study in the specialty 31.05.01.Medical business

Reviewed and approved at the meeting of the department
dated May 19, 2023 (Protocol No. 9)

Head of the Department, MD, Professor



I.G. Dzhioev

STRUCTURE OF EVALUATION MATERIALS

1. Title page
2. Structure of evaluation materials
3. Review of evaluation materials
4. Passport of evaluation materials
5. Set of evaluation materials:
 - Questions to the module
 - Exam questions
 - a bank of situational tasks /practical tasks /business games
 - standards of test tasks (with title page and table of contents),
 - examination tickets /test tickets

FEDERAL STATE BUDGETARY EDUCATIONAL INSTITUTION OF HIGHER
EDUCATION "NORTH OSSETIAN STATE MEDICAL ACADEMY" OF THE
MINISTRY OF HEALTH OF THE RUSSIAN FEDERATION

REVIEW

of evaluation materials on the discipline "Pathophysiology-pathophysiology of the head and neck" for students studying in the specialty 31.05.03 "Dentistry".

The evaluation materials were compiled at the Department of Pathological Physiology on the basis of the work program of the discipline "Pathophysiology-pathophysiology of the head and neck" and meet the requirements of the Federal State Educational Standard of the specialty 31.05.03 Dentistry,

Evaluation materials include:

- module questions
- exam questions
- a bank of situational tasks /practical tasks / business games
- standards of test tasks (with title page and table of contents),
- examination tickets / test tickets

The bank of situational tasks/practical tasks/business games includes the tasks themselves and answer templates. All tasks correspond to the work program "Pathophysiology-pathophysiology of the head and neck" and cover all its sections. The number of test tasks is 700. The complexity of the tasks varies. The number of tasks for each section of the discipline / practice is sufficient to carry out knowledge control and eliminates the repeated repetition of the same question in different variants. The bank contains answers to all test tasks and tasks.

The number of examination tickets is sufficient for the examination and excludes the repeated use of the same ticket during the exam in one academic group on the same day. Examination tickets are made on the forms of a single sample in a standard form, on paper of the same color and quality. The exam ticket includes 3 questions. The wording of the questions coincides with the wording of the list of questions submitted for the exam. The questions of one ticket relate to different sections of the program, which allows you to more fully cover the material of the discipline.

In addition to theoretical questions, a bank of situational tasks (analyses, prescriptions, radiographs, electrocardiograms, etc.)/practical and business games is offered. Situational tasks / practical tasks / business games make it possible to objectively assess the student's level of assimilation of theoretical material during the current, control of academic performance and intermediate certification. The complexity of the questions in the examination tickets is evenly distributed.

There are no comments to the reviewed evaluation materials.

In general, assessment materials on pathophysiology - the pathophysiology of the head and neck contribute to a qualitative assessment of the level of students'

proficiency in universal/general/professional competencies. Peer-reviewed evaluation materials can be recommended for use for current and intermediate certification at the Faculty of Dentistry for 2nd year students.

Reviewer:

Chairman of the TSUMK of Natural Science and Mathematical Disciplines with the subcommittee of expertise of evaluation materials,
Associate Professor of the Department of Chemistry and Physics
Botsieva N.I.

Passport of evaluation materials
on the discipline "Pathophysiology - pathophysiology of the head and neck"
for 2nd year students in the specialty 31.05.03 Dentistry

№п/п	Name of the supervised section (topic) of the discipline/module	The code of the competence being formed (stage)
1	2	3
Вид контроля		
	Current/Intermediate	
1	"Nosology", "Hereditary pathology", "Allergy".	ОПК-9
2	"Typical pathological processes: local circulatory disorders, inflammation, fever."	ОПК-9
3	"Typical pathological processes: hypoxia, metabolic disorders".	ОПК-9
4	"Pathophysiology of the blood system".	ОПК-9
5	"Pathophysiology of the cardiovascular and respiratory systems".	ОПК-9
6	"Pathophysiology of the digestive system, liver and kidneys".	ОПК-9
7	"Pathophysiology of the endocrine system", "Pathophysiology of the central nervous system".	ОПК-9

Questions for module No. 1 for 2nd year students of the Faculty of Dentistry.

1. General nosology

1. Subject, tasks, methods and structure of pathophysiology. The values of pathophysiology as the basis of modern clinical medicine.

2. The concept of etiology and pathogenesis. Reactionary views in matters of etiology. Modern scientific-based understanding of the causes of the disease.
3. Structural elements of the disease: pathological reaction, pathological process, pathological condition, vicious circle.
4. The concept of disease. Disease as a dialectical unity of damage and protective and adaptive reactions of the body. The social and biological essence of the disease. Symptoms of the disease (Sidengam, Hippocrates, Pavlov).
5. Stages of the disease, its outcomes: recovery, death.
6. Compensatory, reparative and adaptive mechanisms of recovery. The role of the central nervous system in them. The doctrine of nervous trophism and its significance in pathology. The works of R. Orbeli, A.D. Speransky, N.N. Zaiko, G.P. Kryzhanovsky.
7. Terminal states. Dynamics and mechanisms of death. Methods of resuscitation.
8. The role of the constitution in pathology.
9. Hereditary diseases and predispositions. Their causes and pathogenesis. Mutagens. The importance of ionizing radiation and environmental pollution in the occurrence of mutations.
10. Classification of primary pathogenetic mechanisms of diseases. Chromosomal diseases, their mechanisms, methods of study, types of inheritance, manifestation of major chromosomal diseases and syndromes (46:21,21,1521; 47:21,21,21; 45:X0; 47:XXU; 47:XXX; 47: HUU, etc.)
11. Pathogenic effect of environmental factors on the body. Radiation sickness. The effect of weightlessness on the body. The effect of electric current on the body.

2. Pathophysiology of typical peripheral circulatory disorders.

1. Local anemia. Causes and mechanism of development, the importance of collateral circulation. Heart attacks, their mechanisms.
2. Arterial hyperemia, causes, mechanisms of development. The role of overload in periodontal and changes in regional hemodynamics.
3. Venous hyperemia, causes, mechanism of development, features of microcirculation. Consequences in dental pathology.
4. Thrombosis, embolism. Pathogenesis, consequences.
3. Inflammation.
5. Inflammation, its main signs, forms and phenomena. Classification of inflammation. Features of productive inflammation of the root system of teeth.
6. Inflammatory reaction of dental pulp, periodontal tissues and salivary glands. Features of the stage of proliferation in the tissues of the dental system. Granulomatous inflammation.
7. Vascular phenomena in the focus of inflammation (disorders of microcirculation, permeability).

8. Emigration of leukocytes and exudation (mechanism and significance), the role of neutrophilic leukocytes in severe forms of inflammation in periodontal disease.
9. Metabolism and physico-chemical changes in the focus of inflammation, their causes and mechanism of development. The ratio of local and general manifestations of inflammation.
10. The significance of the works of Mechnikov and Gamalea for the correct understanding of inflammation. Biological significance of inflammation. Relative expediency of protective and adaptive reactions of the body during inflammation.
11. Etiology and pathogenesis of periodontal diseases. Pathogenic classification and the main mechanisms of development of periodontal diseases.
12. Inflammatory processes in periodontium: formation and composition of plaque and its role in the development of periodontal diseases, changes in general and local immunity.
13. Structural disorders in the gum in gingivitis, periodontitis. Osteolytic process in bone tissue during inflammatory processes in periodontal.
14. Functional trauma (periodontal hyperfunction, pathogenesis, stages). Inflammation of the maxillofacial region, etiology of the "entrance gate".
15. The importance of reactivity in the development of inflammatory processes in the b/l region (normoergic, hyperergic and hypoallergenic)
16. The main pathogenesis of inflammatory processes in the maxillofacial region, ways of spreading inflammation from the primary focus to the surrounding tissues
17. Sialoses and sialoadenitis are the main links of pathogenesis, principles of modeling and diagnosis of diseases of the salivary glands.

4. Pathophysiology of thermoregulation changes.

1. Overheating and fever (features of temperature rise in both cases).
2. The mechanism of thermoregulation changes in various stages of fever.
3. Types of temperature curves in fever.
4. Exo- and endogenous pyrogens. The mechanism of action of pyrogens on the body. Pyrotherapy.
5. Changes in metabolism, blood circulation, respiration, urinary system, digestion in fever.
6. Changes in the function of the salivary glands and the condition of the oral cavity in fever.
7. The importance of fever for the body.

Questions for module No. 2 Faculty of Dentistry

Allergy.

1. Allergy, definitions, allergens and their types, brief description
2. Classification of allergic reactions and mechanisms of their development, stages
3. Allergic reactions of immediate type 1 type (examples and the main characteristic of these reactions).
4. Allergic reactions of immediate type 2 type (examples and the main characteristic of these reactions).
5. Allergic reactions of immediate type 3 type (examples and the main characteristic of these reactions).
6. Mediators of allergic reactions of immediate type.
7. Pathophysiological bases of methods for detecting allergies of immediate and delayed types.
8. Delayed allergic reactions (examples and the main characteristic of these reactions).
9. Anaphylactic shock, clinical variants, flow rate, treatment
10. Diagnosis of allergies.
11. Intolerance of plastic dentures, definition, etiology, pathogenesis.
12. Clinical picture of intolerance to acrylic prostheses.
13. Clinical picture of intolerance to dental “restorations” made of metal.

Pathophysiology of typical metabolic disorders.

14. Pathology of basal metabolism and specifically dynamic action of food substances. Separation of oxidative phosphorylation.
15. Types of fasting and changes in metabolism during fasting.
16. Hyper-, hypo- and vitamin deficiency. Hypovitaminoses: primary (exogenous) and secondary (endogenous) fat-soluble vitamins.
17. Hyper-, hypo- and vitamin deficiency. Hypovitaminoses: primary (exogenous) and secondary (endogenous) water-soluble vitamins.
18. Classification of violations of acid-base balance in the organism and its significance in the clinic.
19. Compensation mechanisms in acute and chronic disorders of acid-base balance.
20. Typical disorders of protein metabolism in various pathological processes.
21. Pathophysiology of disorders of carbohydrate metabolism of acquired and congenital nature (galactosemia, glycogenoses).
22. Pathophysiology of water-salt metabolism. Swelling.
23. Pathophysiology of the violation of the alkaline-acid state of the body.
24. The role of CBS in the development of caries and inflammatory periodontal diseases and pathology of the oral mucosa. Principles of regulation of braids in the oral cavity.

25. Pathophysiology of metabolism. The role of metabolic disorders in the development of pathology of the dental and maxillary system.

26. Pathophysiology of phosphorus-calcium metabolism, osteoporosis, osteomalacia.

Hypoxia.

27. Definition of the concept. General characteristics of hypoxia.

28. Conditions determining the resistance of organs and tissues to oxygen starvation.

Hypoxia. The role of hypoxia in the development of dental diseases.

29. Classification of hypoxic states.

30. Indicators of the gas composition of arterial and venous blood in various types of hypoxia.

31. Adaptive reactions in hypoxia (emergency and long-term), their mechanisms.

Questions for module No. 3 for students

2 courses of the dental faculty.

1. Basic properties and functions of blood. Age-related features of the morphological composition of blood.

2. Degenerative and regenerative forms of erythrocytes.

3. General characteristics of anemia and principles of their classification.

4. Acute posthemorrhagic anemia. Pathogenesis and blood pattern.

5. Chronic posthemorrhagic anemia. Pathogenesis and consequences.

6. Signs of erythropoiesis insufficiency.

7. Anemia from lack of antianemic factor. Pathogenesis and blood pattern

8. Congenital and acquired hemolytic anemia in children. Features of pathogenesis, blood pattern.

9. Hereditary hemolytic anemia. Pathogenesis and blood pattern.

10. Anemia from lack of iron. Pathogenesis and blood pattern.

11. Aplastic anemia. Pathogenesis and blood pattern.

12. Degenerative and regenerative forms of leukocytes.

13. Leukocytosis and leukopenia. Agranulocytosis and other leukemoid reactions.

14. Degenerative, regenerative and mixed nuclear shifts and their significance for the clinic.

15. Classification of leukemias. Methods of laboratory differentiation of acute leukemia.

16. Etiology of leukemia. Changes in leukopoiesis: the picture of peripheral blood in leukemia

Questions for module No. 4 for students

2 courses of the dental faculty.

1. Pathophysiology of systemic circulation. General etiology and pathogenesis of disorders of S.S.S. Circulatory insufficiency, its forms, hemodynamic parameters in children.
2. Violations of the central regulation of the heart: reflex changes in the work of the heart in physiological conditions (reflexes of Loven, Bembridge, etc.) and in pathological conditions (Kitaev reflex, inadequate visceral-cardiac reflexes). Age-related features of the organization of extracardial compensation mechanisms in norm and pathology.
3. Mechanisms of urgent and long-term adaptation of the heart to intermittent and constant loads. Hyperfunction and hypertrophy of the myocardium, features of the hypertrophied heart, mechanisms of decompensation in children with heart defects (congenital and acquired).
4. Coronary insufficiency: transient ischemia, myocardial infarction (pathogenesis and consequences). Fibrillation of the heart. Age-related features of myocardial ischemia.
5. Circulatory insufficiency in pericardial pathology. Cardiac tamponade (causes, mechanism, consequences). Functional arrhythmias in children (etiology, pathogenesis).
6. Vascular-type hemodynamic disorders: fainting, collapse, shock. Experimental models of the main forms of SS-insufficiency.

EXAM QUESTIONS

I.GENERAL NOSOLOGY

1. Tasks, methods and structure of pathophysiology The importance of experiment in the development of pathophysiology and clinical medicine.
2. Structural components of the disease: pathological reaction, pathological process, typical pathological process, pathological condition, "vicious circle", clinical nosology.
3. Modern definition of the disease. Views on the disease and its symptoms (Sidengam, I.P.Pavlov).
4. Periods and outcomes of the disease. Mechanisms of recovery and the role of the central nervous system in them.
5. The concept of etiology and pathogenesis. Features of the etiology and pathogenesis of diseases of the dentition and oral mucosa. The main hypotheses of the concept of the theory of etiology and pathogenesis of periodontal diseases.
6. Terminal states. The mechanism of death. Intensive care and intensive care.
7. Hereditary forms of pathology, their differences from congenital and acquired forms. Mutagens, types of mutation, the importance of ecology in the occurrence of

mutations. Mechanisms of genetic hereditary pathology. Molecular hereditary diseases and predisposition.

8. Dental manifestations of hereditary diseases and syndromes.

II. TYPICAL PATHOLOGICAL PROCESSES.

A. Pathophysiology of typical peripheral circulatory disorders.

9. Local anemia. Causes and mechanism of development, the importance of collateral circulation. Heart attacks, their mechanisms.

10. Arterial hyperemia, causes, mechanisms of development. The role of overload in periodontal and changes in regional hemodynamics.

11. Venous hyperemia, causes, mechanism of development, features of microcirculation. Consequences in dental pathology.

12. Thrombosis, embolism. Pathogenesis, consequences.

B. Inflammation.

13. Inflammation, its main signs, forms and phenomena. Classification of inflammation. Features of productive inflammation of the root system of teeth.

14. Inflammatory reaction of dental pulp, periodontal tissues and salivary glands. Features of the stage of proliferation in the tissues of the dental system. Granulomatous inflammation.

15. Vascular phenomena in the focus of inflammation (disorders of microcirculation, permeability)/

16. Emigration of leukocytes and exudation (mechanism and significance), the role of neutrophilic leukocytes in severe forms of inflammation in periodontal disease.

17. Metabolism and physico-chemical changes in the focus of inflammation, their causes and mechanism of development.

18. The ratio of general and local reactions in inflammation.

19. Etiology and pathogenesis of periodontal diseases.

20. Pathogenic classification and the main mechanisms of development of periodontal diseases.

21. Inflammatory processes in periodontium: formation and composition of plaque and its role in the development of periodontal diseases, changes in general and local immunity.

22. Structural disorders in the gum in gingivitis, periodontitis.

23. Osteolytic process in bone tissue during inflammatory processes in periodontal.

24. Functional trauma (periodontal hyperfunction, pathogenesis, stages).
25. Inflammation of the maxillofacial region, etiology of the “entrance gate”
26. The importance of reactivity in the development of inflammatory processes in the b/l region (normoergic, hyperergic and hypoallergenic)
27. The main pathogenesis of inflammatory processes in the maxillofacial region, ways of spreading inflammation from the primary focus to the surrounding tissues

B. Pathophysiology of changes in thermoregulation.

28. Overheating and fever (features of temperature rise in both cases).
29. The mechanism of thermoregulation changes in various stages of fever.
30. Types of temperature curves in fever.
31. Exo- and endogenous pyrogens. The mechanism of action of pyrogens on the body. Pyrotherapy.
32. Changes in metabolism, blood circulation, respiration, urinary system, digestion in fever.
33. Changes in the function of the salivary glands and the condition of the oral cavity in fever.
34. The importance of fever for the body.

G. Pathophysiology of typical metabolic disorders.

35. Pathology of basal metabolism and specific dynamic action of food substances. Separation of oxidative phosphorylation.
36. Types of fasting and changes in metabolism during fasting (the work of the Pashutin school).
37. Hypovitaminosis brief description and dental manifestations of changes in periodontal tissues with vitamin deficiency. C, A, D, etc.

D. Pathophysiology of hypoxia.

38. Definition of the concept. General characteristics of hypoxia.
39. Classification of hypoxia.
40. Indicators of the gas composition of arterial and venous blood in various types of hypoxia.
41. Adaptive reactions in hypoxia are urgent and long-term.

E. Allergy.

42. Allergy, definitions, allergens and their types, brief description

43. Classification of allergic reactions and mechanisms of their development, stages
44. Anaphylactic shock, clinical variants, rate of flow, treatment
45. Intolerance of plastic dentures, definition, etiology, pathogenesis.
46. Clinical picture of intolerance to acrylic prostheses.
47. Diagnosis of intolerance.
48. Prevention and principles of treatment of intolerance phenomena.
49. Clinical picture of intolerance to dental “restorations” made of metal.

III. BLOOD PATHOPHYSIOLOGY

50. Basic properties and functions of blood.
51. Degenerative regenerative forms of erythrocytes.
52. General characteristics of anemia and principles of their classification.
53. Acute and chronic posthemorrhagic anemia. Pathogenesis and blood pattern.
54. Hereditary hemolytic anemia. Thalassemia, pathogenesis, changes in the dental system. Signs of erythropoiesis insufficiency.
55. Anemia from iron deficiency, pathogenesis and blood pattern.
56. Aplastic anemia, pathogenesis and blood pattern.
57. Anemia from lack of antianemic factor Castle or Addison-Birmer disease. Pathogenesis. Clinical picture, changes in the oral mucosa.
58. Leukocytosis and leukopenia.
59. Degenerative and regenerative and mixed nuclear shifts their significance for the clinic.
60. Degenerative and regenerative forms of leukocytes.
61. Leukemias. Definition of the concept, general characteristics. Etiology of leukemia. Change in leukopoiesis: the picture of peripheral blood.
62. Classification of leukemias. Methods of laboratory differentiation of acute leukemia.
63. Leukemia, definition, classification, ulcerative-necrotic lesions of the oral mucosa.
64. Werlhof's disease, the main hemolytic signs, complications after tooth extraction, prevention of these complications.

IV. PATHOPHYSIOLOGY OF BLOOD CIRCULATION AND RESPIRATION.

65. Violation of the central regulation of the heart: reflex changes in the work of the heart in physiological conditions (reflexes of Loven, Beimbridge, etc.) and conditions of pathology (Kitaev reflex), inadequate viscerocardial reflexes.

66. Pathophysiology of systemic circulation. General etiology pathogenesis of disorders of the cardiovascular system. Circulatory insufficiency of its form, hemodynamic parameters.
67. Mechanisms of urgent and long-term adaptation of the heart to intermittent and constant loads. Hyperfunction and hypertrophy of the myocardium, features of the hypertrophied heart decompensation mechanisms.
68. Coronary insufficiency: transient ischemia, myocardial infarction (pathogenesis and consequences). Changes in the main hemodynamic parameters in circulatory disorders of the cardiac type. Disorders in the tissues of the oral cavity in chronic insufficiency of the cardiovascular system.
69. Circulatory insufficiency in pericardial pathology. Cardiac tamponade (causes, mechanisms, consequences).
70. Vascular-type hemodynamic disorders: fainting, collapse, shock.
71. Modern ideas about hypertension. Hereditary predisposition, provoking factors in the pathogenesis of hypertension.
72. Pathogenesis of renal hypertension. Renoprivny and renopressor and mechanisms.
73. Symptomatic hypertension. The role of disorders of the nervous regulation of arterial pressure of the endocrine glands.
74. The concept of respiratory failure, its indicators, general etiology. And pathogenesis. Shortness of breath, stenosis, asphyxia. Pathological forms of breathing.
75. Respiratory disorders in pneumonia, emphysema, bronchial asthma, various types of pneumothorax.
76. Features of respiratory disorders in dental diseases and interventions. Connection of respiratory disorders with pathology of oral tissues.

V. PATHOPHYSIOLOGY OF THE LIVER.

77. Experimental modeling of the main types of liver pathology (Ecca fistula, Ecca-Pavlova, extirpation of the liver. Angiostomy in London).
78. Pathogenetic classification of jaundice and brief description
79. Analysis of the phenomena of liver failure.
80. Hemolytic jaundice and analysis of accompanying changes in the body.
81. Pathogenesis of changes in infectious and toxic jaundice.
82. Mechanical jaundice and analysis of accompanying changes in the body
83. Portal hypertension. Changes in the tissues of the oral cavity in chronic liver failure.

VI. PATHOPHYSIOLOGY OF URINATION AND URINARY EXCRETION.

84. Basic functional tests of the kidneys.

85. Polyuria, oliguria, hypostenuria, hyperstenuria, isostenuria, nocturia, causes and mechanism of occurrence.

86. Qualitative disorders of urine composition: albuminuria, cylindruria, hematuria, hemaglobinuria. Causes and mechanism of development

87. Etiology and pathophysiological mechanisms of development of the main clinical and laboratory syndromes of renal insufficiency in acute nephritic syndrome.

88. Etiology and pathophysiological mechanisms of development of the main clinical and laboratory syndromes of renal insufficiency in chronic nephritic syndromes.

89. Nephrotic syndrome.

90. Changes in the tissues of the dental system in chronic renal failure.

VII. PATHOPHYSIOLOGY OF THE ENDOCRINE SYSTEM.

91. The main pathophysiological mechanisms of hormonal disorders, the role of the hypothalamic-pituitary neurosecretory system in their occurrence.

92. Thyroid diseases and dental examination data.

Federal State Budgetary Educational Institution

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Department of Pathophysiology

Faculty of Dentistry Course 2

Discipline pathophysiology-pathophysiology of the head and neck

Situational task

Task 1

A pregnant woman M. turned to a genetic consultation. She reported that her maternal brother (fathers are different) is ill with phenylketonuria. Her daughter from her first marriage is healthy. She also said that in the family of her second spouse N. there were marriages between close relatives, but no one was ill with phenylketonuria. The examination of the woman M. and her real spouse did not reveal any abnormalities in their state of health.

Questions

1. What is the type of inheritance of phenylketonuria and how is it characterized?

2. What is the probability of the development of phenylketonuria in the sons and daughters of a woman M?
3. What are the manifestations of phenylketonuria and what are they caused by?
4. Which protein (enzyme, structural protein, receptor, membrane transporter) is encoded by an abnormal gene in this form of pathology.
5. How is this disease recognized in newborns?
6. How can we prevent the development of phenylpyruvic oligophrenia in children?

Answers to situational task 1

1. Phenylketonuria is inherited by an autosomal recessive type. This type of inheritance is characterized by the following:

- a sick child is born to healthy heterozygous parents,
- men and women are sick,
 - both men and women can transmit the disease;
 - the probability of inheritance is 25% (if the parents are heterozygous),
- in a homozygous state, as a rule, complete penetrance is observed, the disease proceeds with relatively the same severity in different patients;
 - symptoms of the disease are usually detected in early childhood;
 - new mutations are extremely rare,
- the disease occurs as a result of mutations of genes encoding the synthesis of a number of enzymes (in the vast majority of cases — phenylalanine hydroxylase).

2. If the spouse is not a carrier of the defective gene, then the probability of M.'s descendants getting sick is 0.

3. Clinical manifestations of phenylketonuria: oligophrenia, pathological reflexes, epileptic seizures. Another name for this disease is phenylpyruvic oligophrenia. The causes of the development of oligophrenia are not precisely established; it is assumed that nerve cells are damaged by products of phenylalanine metabolism (possibly phenylpyruvate).

4. With phenylketonuria, the synthesis of phenylalanine hydroxylase is disrupted.

5. There is an approximate test with ferric chloride (urine is tested on diapers). The screening program (including the federal program in Russia) provides for the determination of the level of phenylalanine in blood plasma, phenylpyruvate in urine. Blood from newborns is taken on the 3rd-5th day after birth, i.e. still in the maternity hospital (earlier than 3 days is ineffective due to the large number of false negative conclusions). In case of a positive result, a clarifying biochemical diagnosis is carried out. This is already a more complex procedure, sometimes multi-stage. Firstly, it is necessary to confirm hyperphenylalaninemia and, secondly, to understand its cause. It can be caused by typical (classical) phenylketonuria

(phenylalanine hydroxylase deficiency), variant or atypical forms of this disease, resistant to diet therapy, hereditary hyperphenylalaninemia (benign), other forms of metabolic disorders.

6. The development of the disease can be prevented if the intake of phenylalanine with food is significantly reduced. It is recommended to adhere to such a diet constantly.

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Health of the Russian Federation

Department of Pathophysiology
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Situational task No.

Kostya N., 8 years old, was admitted to the clinic of children's diseases. His parents are alarmed by the frequent development of otitis media, sore throats, rhinitis, conjunctivitis, bronchitis, pneumonia, enterocolitis in the child. This hospitalization is associated with the suspected development of endocarditis and sepsis.

The examination revealed: leukopenia due to a significant decrease in the number of lymphocytes, mainly their T pool and to a lesser extent — B-lymphocytes; a decrease in the content of IgA and IgM in the blood (by 40 and 50% of the norm, respectively), the IgG level is at the lower limit of the norm; the reaction of lymphocytes to phytohemagglutinin is reduced.

Questions

1. How do you designate a pathological condition that has developed in a child? Justify the answer.
2. What are its possible causes?
3. What is the mechanism of development and consequences of this condition, judging by laboratory data?
4. How do you explain the facts of a decrease in the reaction of lymphocytes to phytohemagglutinin and a significant decrease in the content of IgA and IgM in the blood at the IgG norm?
5. What manifestations of the child's painful condition can largely be the result of reducing the level of IgA and IgM?

Answers to the situational problem 7

1. Combined T- and In an immunodeficiency state. This is evidenced by: frequent infections, a decrease in the number of lymphocytes, mainly their T-pool, to a lesser extent in the pool; decreased functional activity of T-lymphocytes, as well as the content of IdA and IdA in the blood.

2. This is a hereditary form of pathology. Autosomal recessive type of inheritance. Louis Bar syndrome. A decrease in the production of IdA and IdE and an increase in the level of fetal proteins (fetoproteins) are characteristic of this syndrome. The presence of fetal proteins is a consequence of thymus aplasia.

3, 4. The condition that has developed in a child is a consequence of a violation of the proliferation and maturation of T* lymphocytes, including T* lymphocytes* helper cells and, as a consequence, disorders of the regulation of the processes of proliferation and differentiation of B* lymphocytes into plasma cells producing IdA and IdA (as evidenced by laboratory data). In this regard, the reaction of blast transformation to the stimulator of this reaction — phytohemagglutinin, as well as the subsequent maturation of T * lymphocytes, is reduced. The reduced titer of IdA and IgE at the IgG norm is due to a significant selective violation of the transport of calcium ions, which affect the proliferation and maturation of T * lymphocytes (in particular, T*helper cells). In this regard, there is a shortage of T * helpers. With Louis Bar syndrome, normal Ig indicators are possible, some patients have hypergammaglobulinemia.

5. The decrease in IgA content can be explained primarily by the absence of plasma cells synthesizing these AT. The formation of anti-IdA AT also increases IgA catabolism. The tendency to respiratory diseases is noted in patients with IgA deficiency, IgE deficiency creates unfavorable conditions for the development of some forms of pneumonia, enterocolitis.

Federal State Budgetary Educational Institution
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Health of the Russian Federation

Department of Pathophysiology
Faculty of Dentistry Course 2
Discipline pathophysiology-pathophysiology of the head and neck

Situational task No. 3

Mining industry worker Ch . 36 years old, he was admitted to the clinic with
suspected silicosis. Complaints of shortness of breath, especially pronounced when

walking and physical exertion, constant cough (dry, sometimes with a small amount of sputum), chest pain.

Data on the gas composition of arterial blood and spirometry:

Gas composition of arterial blood

raO₂ (mmHg) 90

After a test with arbitrary hyperventilation of the lungs 92

raSO₂ (mmHg) 40

Oxygen capacity 19.2 volume %

SaO₂ 94.3%

Spirometry

GEL 4.2 l

FZHEL1 2.6 l

WEL (% of the required value) 92

The Tiffno coefficient ? (calculate)

MOD (% of due value) 124

Additional data

Respiratory rate 19 in 1 minute

Questions

1. Does the patient have signs of a disorder of the gas exchange function of the lungs? If yes, then specify them. Explain the answer.
2. Does the patient have alveolar ventilation disorders? If yes, then determine the type of disorder (obstructive or respiratory).
3. Considering the possibility of developing pneumoconiosis, how do you propose to assess the diffusion capacity of the lungs?
4. What is your general conclusion about the possible nature of violations of the gas exchange function of the external respiratory system?

Answers to the situational task 16

1. Signs of disorders of the gas exchange function of the lungs in this patient are: shortness of breath, increased respiratory rate and MOD, hypoxemia, a decrease in the coefficient Tiffno; no significant change in RAO₂ after the sample with arbitrary hyperventilation.
2. The patient has signs of impaired alveolar ventilation of a predominantly obstructive type. This is evidenced by a decrease in the Tiffno coefficient (less than 62%), an increase in respiratory rate and MOD.
3. The diffusion capacity of the aerogematic barrier is evaluated based on the results of a sample with arbitrary hyperventilation of the lungs. In this case, the raO₂ index (92 mmHg) differs little from that before the sample. Consequently, the diffusion capacity of the lungs for oxygen is reduced.

4. General conclusion: the patient has impaired lung gas exchange function due to a disorder of alveolar ventilation of a predominantly obstructive type (apparently due to obstruction of the airways with sputum), as well as as a result of a decrease in the diffusion capacity of the walls of the alveoli.

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Situational task No. 4
HYPOXIA

When solving tasks 1-10, you should focus on the values of the following indicators.