Патологическая физиология

Пособие в двух частях
для студентов факультета иностранных учащихся

Часть 2
Патофизиология систем

Pathophysiology

Manual in 2 parts for the faculty of foreign students

Part 2
Systemic Pathophysiology

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Grodno State Medical University

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Manual «Pathophysiology. Manual in 2 parts for the faculty of foreign students. Part 2. Systematic Pathophysiology» is intended for the faculty of foreign students (English medium of instruction) for successful studying the subject. It contains the information according to purposes and questions to topics, the description of the laboratory works, tests, tasks, literature, list of test questions.

Responsible for the issue: vice rector, Vorobiev V.V.
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<thead>
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<th>Abbreviation</th>
<th>Description</th>
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<tbody>
<tr>
<td>ACTH</td>
<td>adrenocorticotropic hormone</td>
</tr>
<tr>
<td>CI</td>
<td>color index</td>
</tr>
<tr>
<td>TEG</td>
<td>thrombelastography</td>
</tr>
<tr>
<td>R</td>
<td>reaction time</td>
</tr>
<tr>
<td>K</td>
<td>thrombin time</td>
</tr>
<tr>
<td>MA</td>
<td>maximum amplitude</td>
</tr>
<tr>
<td>T</td>
<td>total time of blood coagulation</td>
</tr>
<tr>
<td>ADP</td>
<td>adenosine diphosphate;</td>
</tr>
<tr>
<td>DIC</td>
<td>disseminated intravascular coagulation</td>
</tr>
<tr>
<td>BW</td>
<td>body weight</td>
</tr>
<tr>
<td>DIC</td>
<td>disseminated intravascular coagulation</td>
</tr>
<tr>
<td>DNA</td>
<td>desoxy ribonuclein acid</td>
</tr>
<tr>
<td>fig</td>
<td>figure</td>
</tr>
<tr>
<td>VIP</td>
<td>vasoactive intestinal peptide</td>
</tr>
<tr>
<td>GABA</td>
<td>gamma-aminobutiric acid</td>
</tr>
<tr>
<td>Hb</td>
<td>hemoglobin</td>
</tr>
<tr>
<td>MCH</td>
<td>mean corpuscular hemoglobin</td>
</tr>
<tr>
<td>RNA</td>
<td>ribonuclein acid</td>
</tr>
<tr>
<td>Rt</td>
<td>reticulocytes</td>
</tr>
<tr>
<td>VC</td>
<td>vital capacity</td>
</tr>
<tr>
<td>LDH</td>
<td>lactate dehydrogenase</td>
</tr>
<tr>
<td>ALT</td>
<td>alanine aminotransferase</td>
</tr>
<tr>
<td>RBC</td>
<td>red blood cells</td>
</tr>
<tr>
<td>HR</td>
<td>heart rate</td>
</tr>
<tr>
<td>RR</td>
<td>respiratory rate</td>
</tr>
<tr>
<td>TV</td>
<td>tidal volume</td>
</tr>
<tr>
<td>IRV</td>
<td>inspiratory reserve volume</td>
</tr>
<tr>
<td>ERV</td>
<td>expiratory reserve volume</td>
</tr>
<tr>
<td>RV</td>
<td>residual volume</td>
</tr>
<tr>
<td>IC</td>
<td>inspiratory capacity</td>
</tr>
<tr>
<td>FRC</td>
<td>functional residual capacity</td>
</tr>
<tr>
<td>VC</td>
<td>vital capacity</td>
</tr>
<tr>
<td>TLC</td>
<td>total lung capacity</td>
</tr>
<tr>
<td>S</td>
<td>body area</td>
</tr>
</tbody>
</table>
LESSON № 19

TOPIC: PATHOLOGY OF CIRCULATING BLOOD VOLUME. BLEEDING.

Aim of the lesson: to study the types of circulating blood volume disorders, their causes and mechanisms of development, to study pathogenesis and compensatory mechanisms after acute bleeding.

QUESTIONS:

2. Categorization of disorders of circulating blood volume (hypervolemia, hypovolemia).
5. Erythrocytosis. Polycythemia or Wakes’s disease.
8. Parameters of severity of bleeding.
9. Factors which affect bleeding outcome.
Fig.1. Changes of the blood volume circulation.

Dark part of strips corresponds to hematocrit, their general length – general blood volume

1-9 – the main typical changes:
1 – simple normovolemia,
2 – oligocythemic normovolemia,
3 – polycythemic normovolemia,
4 – simple hypervolemia,
5 – oligocythemic hypervolemia,
6 – polycythemic hypervolemia,
7 – simple hypovolemia,
8 – oligocythemic hypovolemia,
9 – polycythemic hypovolemia.
Table 1. Parameters of bleeding severity

<table>
<thead>
<tr>
<th>Severity</th>
<th>Volume of blood loss (ml)</th>
<th>Blood density (un.)</th>
<th>[Pt] (g/l)</th>
<th>Hct (%)</th>
<th>АД s. (mm Hg)</th>
<th>Puls (beet/min)</th>
<th>Condition of CNS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mild</td>
<td>350-750</td>
<td>1054-1057</td>
<td>62-65</td>
<td>40-44</td>
<td>N</td>
<td>80-100</td>
<td>Mild exitation</td>
</tr>
<tr>
<td>Moderate</td>
<td>750-1500</td>
<td>1059-1053</td>
<td>54-61</td>
<td>32-36</td>
<td>&lt;100</td>
<td>100-120</td>
<td>exitation</td>
</tr>
<tr>
<td>Severe</td>
<td>1500-2000</td>
<td>1044-1049</td>
<td>53-45</td>
<td>23-30</td>
<td>&lt;80</td>
<td>120-140</td>
<td>inhibition</td>
</tr>
<tr>
<td>Oversevere</td>
<td>&gt; 2000</td>
<td>&lt; 1044</td>
<td>&lt; 45</td>
<td>&lt; 23</td>
<td>&lt;60</td>
<td>&gt;140,</td>
<td>precoma</td>
</tr>
</tbody>
</table>

Reticulocytes (Rt) are premature red blood cells, typically composing about 0,2 – 1,2% (2-12‰) of the red cells in the human body. Rt develop and mature in the red bone marrow and then circulate for about a day in the blood stream before developing into mature red blood cells. Like mature red blood cells, Rt do not have a cell nucleus. They are called Rt because of a reticular (mesh-like) network of ribosomal RNA that becomes visible using a microscope with certain stains such as new methylene blue.

Rt appear slightly bluer than other red cells when looked at with the normal Romanowsky stain.

To accurately measure Rt counts, we can use automated counters or microscope.
Laboratory work 1. Measurement of reticulocyte count in the blood using microscope

**Description of the work 1.** To measure reticulocyte count students use light microscope.

The blood samples with *brilliant creasil blue* paint should be used for investigation. Rt count estimation is performed simultaneously with erythrocytes count (1000) in separated area. Rt count reports the number of reticulocytes as a percentage (%) of the number of red blood cells or promille (1‰ = 0,1%). Reticulocytes are newly-produced red blood cells. They are slightly larger than totally mature red blood cells, and have some residual ribosomal RNA. The presence of RNA is visualized as a blue web-like structure.
Tasks

1.
Patient, 35 years old, male, arrives to the hospital after chest trauma.  
Clinical findings: paleness, blood pressure 70/40 mm Hg, weak puls, frequent breath, X-ray chest examination reveals severe darkness in thoracic cavity.

Blood analysis, after 4 days of hemostatic operation: Hb – 71 g/l, RBC – 3 x 10¹²/l, Rt – 12 %, WBC – 10,2 x 10⁹/l.

Blood sample: a lot of polychromatophills, 2 oxyphilic normocytes.

Make hematological conclusion according classification.

2.
Patient, 42 years old, arrives to the genicological hospital after prolonged uteric bleeding (about 2-3 weeks).

Clinical findings: paleness, rapid puls, uteric myoma (benign tumor).

Blood analysis: Hb – 68 g/l, RBC – 2,8 x 10¹² /l, Rt – 0,05 %, WBC – 4 x 10⁹/l, erythrocyte sedimentation rate (ESR)– 8mm/hr.

Blood sample: hypochromia of erythrocytes, anyzocytosis, microcytosis, poikilocytosis, and single polychromatophills.

Biochemical analysis: serum Fe concentration 6 μM

What pathology has got the patient?
Make hematological conclusion.
Tests:

1. **Hematocrit in oligocythemic normovolemia:**
   a) increases  
   b) decreases  
   c) is normal

2. **Hematocrit in polycythemic normovolemia:**
   a) increases  
   b) decreases  
   c) is normal

3. **Hematocrit in simple normovolemia:**
   a) increases  
   b) decreases  
   c) is normal

4. **What kind of normovolemia leads to the increase of blood viscosity, susceptibility to thrombosis?**
   a) simple  
   b) polycythemic  
   c) oligocythemic

5. **In what normovolemia appears anemia and hypoxia?**
   a) simple  
   b) oligocythemic  
   c) polycythemic

6. **How is hematocrit changed in simple normovolemia?**
   a) it is increased  
   b) it is decreased  
   c) it has no changes

7. **How is hematocrit changed in oligocythemic hypovolemia?**
   a) it is increased  
   a) it is decreased  
   b) it has no changes
8. How is hematocrit changed in simple hypovolemia?  
a) it is increased  
b) it is decreased  
c) it has no changes

9. How is hematocrit changed in polycythemic hypovolemia?  
a) it is increased  
b) it is decreased  
c) it has no changes

10. How is hematocrit changed in olygocytemic hypovolemia?  
a) it is increased  
b) it is decreased  
c) it has no changes

11. Dehydration leads to:  
a) olygocytemic hypovolemia  
b) polycythemic hypovolemia  
c) olygocytemic normovolemia

12. Excess entrance of water leads to  
a) polycythemic normovolemia  
b) olygocytemic hypervolemia  
c) polycythemic hypovolemia

13. Chronic hypoxia results in:  
a) olygocytemic hypervolemia  
b) polycythemic normovolemia  
c) polycythemic hypovolemia

14. Depression of the erythropoiesis leads to:  
a) polycythemic normovolemia  
b) polycythemic hypovolemia  
c) olygocytemic normovolemia

15. Massive haemolysis of erythrocytes results in:  
a) olygocytemic hypervolemia  
b) polycythemic hypovolemia
c) olygocythemic normovolemia

16. Massive hemotransfusion leads to:
   a) simple hypervolemia
   b) olygocythemic hypervolemia
   c) olygocythemic normovolemia

17. The causes of simple hypovolemia are:
   a) dehydration
   b) hemorrhage (immediately after it)
   c) erythremia
   d) hemorrhage (in a few days)
   e) massive introduction of plasma substitutes

18. The causes of polycythemic hypovolemia are:
   a) dehydration
   b) massive haemolysis of erythrocytes
   c) erythremia
   d) anemia
   e) hemorrhage

19. The causes of olygocythemic normovolemia are:
   a) dehydration
   b) tissue hypoxia
   c) renal insufficiency
   d) erythremia
   e) haemolysis of erythrocytes

20. Polycythemic hypervolemia results from:
   a) erythremia
   b) dehydration
   c) anemia
   d) haemorrhage

21. Massive introduction of the isotonic solutions leads to:
   a) simple hypervolemia
   b) polycythemic hypervolemia
   c) olygocythemic hypervolemia
   d) polycythemic hypovolemia
22. The causes of simple hypovolemia are:
   a) acute haemorrhage (in 30-40 minutes)
   b) moderate haemorrhage (in 24 hours)
   c) haemolysis of erythrocytes

23. What conditions lead to polycythemiac hypovolemia?
   a) extensive burns
   b) overheating
   c) water intoxication
   d) erythremia

24. The causes of simple hypervolemia are:
   a) massive blood transfusion
   b) nephrotic disease
   c) erythremia
   d) intravenous introduction of salt solution

25. The causes of olygocythemic normovolemia are:
   a) massive infusion of plasma substitute
   b) moderate hemolysis of RBC
   c) massive hemolysis of RBC

26. Hydremic phase of acute haemorrhage is characterized by:
   a) simple hypovolemia
   b) olygocytemic hypovolemia
   c) olygocytemic normovolemia
   d) simple hypervolemia

27. Reflectory phase of acute haemorrhage is characterized by:
   a) simple hypovolemia
   b) olygocythemic hypovolemia
   c) olygocythemic normovolemia
   d) simple hypervolemia

28. The reasons of polycythemic normovolemia are:
   a) living in the hills
   b) dehydration
c) decreased synthesis of erythropoethine

29. The reasons of simple hypervolemia are:
   a) infusion of plasma substitute
   b) blood transfusion
   c) packed red cell transfusion
   d) reduction of excretory function of the kidneys

30. The reasons of polycytemic hypervolemia are:
   a) haemotransfusion
   b) packed red cell transfusion
   c) erythremia
   d) infusion of polyglucine

31. The causes of olygocythemic hypervolemia are:
   a) packed red cell transfusion
   b) reduction of excretory function of the kidneys
   c) haemotransfusion
   d) excess of vasopressin

32. What phase of acute haemorrhage leads to simple hypovolemia?
   a) reflex
   b) hydremic
   c) bone-marrow

33. The reasons of simple hypovolemia are:
   a) lack of erythropoetin
   b) haemorrhage
   c) erythremia

34. The causes of polycythemic hypovolemia are:
   a) erythremia
   b) lack of vasopressine
   c) packed red cell transfusion

35. The reasons of olygocythemic hypovolemia are:
   a) diarrhea
   b) aplasia of bone marrow
   c) intravenous introduction of salt solution
36. The causes of oligocythemic hypovolemia are:
   a) polyuria
   b) living in the hills
   c) lack of synthesis of erythropoetine

37. The first hours after acute haemorrhage are characterized by:
   a) polycythemic hypovolemia
   b) simple hypovolemia
   c) oligocythemic hypovolemia

38. The second day after acute haemorrhage is characterized by:
   a) polycythemic hypovolemia
   b) oligocytemic (normovolemia) hypovolemia
   c) oligocythemic hypervolemia

39. What is the reason of compensation in reflex phase of haemorrhage?
   a) stimulation of baroceptor reflexogen regione
   b) activation of sympathoadrenal system
   c) decreased stroke volume
   d) decreased peripheral resistance

40. What is the reason of adjustment of blood volume in haemorrhage?
   a) spasm of the peripheral vessels
   b) activation of blood coagulation system
   c) decreased diuresis
   d) redistribution of the water between sectors

41. What is the reason of normalization of blood pressure in reflectory phase of haemorrhage?
   a) output of blood from store
   b) increased sympathetic influence of cor
   c) stimulation of baroceptor reflexogen regione
   d) activation of renin-aldosterone-angiothensin system
42. What is the reason of normalization of blood pressure at hydremic phase of haemorrhage?
   a) activation of renin-aldosterone-angiothensin system
   b) increased output of vasopressine
   c) entrance water from intersticium to the vessels
   d) centralization of blood circulation

43. The main links of pathogenesis of middle degree of haemorrhage are:
   a) cells dehydration
   b) disorder of microcirculation
   c) disorder of oxygen transport function of hemoglobin

44. The reflex phase of acute haemorrhage is characterized by:
   a) hyperventilation
   b) bradycardia
   c) appearance of young red blood cells in blood
   d) tachycardia

45. The reflectory phase of acute haemorrhage is characterized by:
   a) increased of the peripheral resistance
   b) increased synthesis of angiotensine
   c) increased synthesis of prothein

46. In what time after acute haemorrhage reticulocytosis will occur?
   a) in 5-6 hours
   b) in 4-5 days
   c) in 24-48 hours
   d) immediately after haemorrhage

47. Adaptive response in acute haemorrhage in few hours is:
   a) decreased venous return
   b) centralization of blood circulation
   c) tissue hypoperfusion
   d) hyperventilation

48. Factors of the severe consequences of the bleeding are:
   a) female sex
   b) newborn period
c) old age  
d) slow velocity of the bleeding

49. *Normal count of reticulocytes is:*  
a) 0-1 %  
b) 2-12 %  
c) 20-25 %  
d) 25-50 %

50. *Compensated hemorrhagic shock will occur in loss of:*  
a) 20-30 % blood volume  
b) 30-40 % blood volume  
c) > 40 % blood volume

51. *Decompensated reversible hemorrhagic shock will occur in loss of:*  
a) 20-30 % blood volume  
b) 30-40 % blood volume  
c) > 40 % blood volume

52. *The reason of hemorrhagic shock is loss of blood:*  
a) more than 10% blood volume  
b) more than 30% blood volume  
c) more than 20% blood volume

53. *The torpid phase of hemorrhagic shock is characterized by:*  
a) unconscious  
b) decreased blood pressure  
c) excitement  
d) increased cardiac output  
e) multiple organ failure

54. *The pathogenic factors of hemorrhagic shock are:*  
a) decreased blood pressure  
b) decreased coronary circulation  
c) increased venous return  
d) increased blood viscosity
Answers:

1b, 2a, 3c, 4b, 5b, 6c, 7b, 8c, 9a, 10b, 11b, 12b, 13b, 14c, 15c, 16a, 17b, 18a, 19e, 20a, 21c, 22a, 23ab, 24a, 25b, 26bc, 27a, 28a, 29b, 30bc, 31bd, 32a, 33b, 34b, 35b, 36c, 37b, 38b, 39ab, 40cd, 41abc, 42abc, 43b, 44ad, 45ab, 46b, 47bd, 48bc, 49b, 50a, 51b, 52b, 53abe, 54abd.

LITERATURE:

1. Lecture material.
LESSON № 20

TOPIC: ANEMIAS.

Aim of lesson: to study causes and mechanisms of different types of anemias. Analyse changes in blood in patients with different types of anemias.

STUDY QUESTIONS:

2. Categorization of anemia by:
   - etiopathogenesis,
   - color parameter,
   - severity of anemia,
   - regenerative possibility,
   - mechanism of erythropoiesis,
   - erythrocyte’s size.
3. Posthemorrhagic anemia. Description, the picture of blood in acute and chronic posthemorrhagic anemias.
6. Hemolytic anemias. Types (congenital, autoimmune e.g.). The picture of blood. Clinical symptoms.
7. Newborn hemolytic anemia.

Laboratory work 1. Measurment of hemoglobin concentration in the blood by Sali method.

Description of the work. Few drops of 0,1N HCl solution should be added to cylinder of the Sali hemometer until low border.
Then 0,02 ml of investigated blood is added to HCl solution and is mixed for 5 min in hemometer at room temperature. The brown color of hematin chloride will develop. This color should be changed to standard color of hematin chloride, which is presented in hemometer, by water addition. Hemoglobin level is estimated by solution level in cylinder (1g% = 10g/l).

**Laboratory work 2. Measurement of hemoglobin concentration in the blood by spectrophotometer.**

**Description of the work.** Then 0,02 ml of investigated blood should be added to 5 ml of transforming solution. It should stay for 10 min at room temperature. After that the investigated sample is inserted in photometer KFK-3, and is compared to transforming solution as control at optical distance ($\lambda$) of 520 nm. Students estimate hemoglobin concentration using calibration curve.

**Laboratory work 3. Calculation of erythrocyte count in Goryaev camera.**

**Description of the work.** A rabbit blood should be dissolved 200 times by 3% solution of acetic acid. 0,02 ml of investigated blood is added to 4
ml of 3% sodium chloride solution. Mix and flood Goryaev camera by this solution.

Fig. 6. Scheeme Goryaev camera for calculation of the blood cells

Under microscopat small magnification students calculate erythrocyte count in 5 big shade quadrants in diagonal direction.

Fig. 7. Scheeme of quadrants at Goryaev camera for calculation of the blood cells

The sum of calculated RBCs should be put to the formula:

\[ X = \frac{A \times 200 \times 4000}{80} \times 10^6, \text{where} \]

\[ X \quad \text{– RBC count per 1 liter of the blood,} \]
\[ A \quad \text{– sum of RBC in 5 big shade quadrants,} \]
\[ 200 \quad \text{– dissolving degree,} \]
Normal count of RBC is: $4,0 - 5,1 \times 10^{12} / l$ (for male)
$3,9 - 4,7 \times 10^{12} / l$ (for female)

**Laboratory work 4. Calculation of color index.**

**Description of the work 4.**

Color index (CI) = (Hb(g/l) × 3) /first three figures of RBC count
Normal color index is 0,85 – 1,05

*For example:*  
Hb  56 g/l  
RBC  3,5 × 10^{12} /l

\[
CI = \frac{56 \times 3}{350} = 0,48
\]

Conclusion: hypochromic anemia

![Blood film in iron deficiency anemia](image)

**Fig. 8. Blood film in iron deficiency anemia**

1 – normochromic erythrocyte  
2, 3 – erythrocyte hypochromic, microcyte  
4 – polymorphnucleic leucocyte  
5 – lymphocyte  
6 – platelets
### Table 2. Blood parameters in norm

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Value of the index in «manual» calculation method</th>
<th>In automatic calculation on apparatus Hemacomp-10</th>
</tr>
</thead>
<tbody>
<tr>
<td>Erythrocytes (RBC)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>in women</td>
<td></td>
<td></td>
</tr>
<tr>
<td>in men</td>
<td></td>
<td></td>
</tr>
<tr>
<td>(3,9 – 4,7) × 10^{12} /l</td>
<td>(4,2 – 5,4) × 10^{12} /l</td>
<td></td>
</tr>
<tr>
<td>(4,0 – 5,1) × 10^{12} /l</td>
<td>(4,6 – 6,2) × 10^{12} /l</td>
<td></td>
</tr>
<tr>
<td>Hemoglobin (Hb)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>in women</td>
<td>120 – 140 g/l</td>
<td>120 – 160 g/l</td>
</tr>
<tr>
<td>in men</td>
<td>130 – 160 g/l</td>
<td>140 – 180 g/l</td>
</tr>
<tr>
<td>Hematocrit (Hct)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>in women</td>
<td>36 – 42 %</td>
<td>37 – 47 %</td>
</tr>
<tr>
<td>in men</td>
<td>40 – 48 %</td>
<td>41 – 53 %</td>
</tr>
<tr>
<td>mean corpuscular volume of erythrocyte – MCV</td>
<td>80 – 100 μm^3</td>
<td>79 – 95 μm^3</td>
</tr>
<tr>
<td>mean corpuscular hemoglobin in erythrocyte – MCH</td>
<td>25,4 – 34,6 pg</td>
<td>27 – 31 pg</td>
</tr>
<tr>
<td>mean corpuscular hemoglobin concentration in erythrocyte – MCHC</td>
<td>30 – 38 g/dl</td>
<td>32 – 36 g/dl</td>
</tr>
<tr>
<td>Color index</td>
<td>0,85 – 1,05</td>
<td>-</td>
</tr>
<tr>
<td>Reticulocytes</td>
<td>2 – 12‰</td>
<td>-</td>
</tr>
<tr>
<td>White blood cells (WBC)</td>
<td>(4– 9)× 10^9 /l</td>
<td>(4,5 – 10,5) × 10^9 /l</td>
</tr>
<tr>
<td>Neutrophils</td>
<td>1 – 6 %</td>
<td>40 – 70 %</td>
</tr>
<tr>
<td>band forms</td>
<td>47 – 72 %</td>
<td>-</td>
</tr>
<tr>
<td>neutrophils</td>
<td>0,5 – 5 %</td>
<td>0,0 – 7 %</td>
</tr>
<tr>
<td>Eosinophils</td>
<td>0 – 1 %</td>
<td>0,0 – 1,5 %</td>
</tr>
<tr>
<td>Basophils</td>
<td>18 – 40 %</td>
<td>19 – 48 %</td>
</tr>
<tr>
<td>Lymphocytes</td>
<td>2 – 9 %</td>
<td>3,4 – 9 %</td>
</tr>
<tr>
<td>Monocytes</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Thrombocytes (Plt)</td>
<td>180 – 320 × 10^9 /l</td>
<td>150 – 400 × 10^9 /l</td>
</tr>
</tbody>
</table>
Table 3. **Anemias classification** (Litvitsky P.F., 1997 expanded edition)

<table>
<thead>
<tr>
<th>Criteria</th>
<th>Classification</th>
</tr>
</thead>
<tbody>
<tr>
<td>According to reason</td>
<td>• primary (inherited, innate)</td>
</tr>
<tr>
<td></td>
<td>• secondary (acquired)</td>
</tr>
<tr>
<td>According to etiopathogenesis</td>
<td>• posthemorrhagic</td>
</tr>
<tr>
<td></td>
<td>• dyserythropoietic</td>
</tr>
<tr>
<td></td>
<td>• hemolytic</td>
</tr>
<tr>
<td>According to erythropoiesis</td>
<td>• normoblastic</td>
</tr>
<tr>
<td></td>
<td>• megaloblastic</td>
</tr>
<tr>
<td>According to erythrocytes size</td>
<td>• normocytic ($\approx 7,1 - 7,9$ mkm)</td>
</tr>
<tr>
<td></td>
<td>• microcytic ($&lt; 7,1$ mkm)</td>
</tr>
<tr>
<td></td>
<td>• macrocytic ($&gt;7,9$ mkm)</td>
</tr>
<tr>
<td></td>
<td>• megalocytic ($&gt; 12$ mkm)</td>
</tr>
<tr>
<td>According to color index (CI)</td>
<td>• normochromic ($CI = 0,85-1,05$)</td>
</tr>
<tr>
<td></td>
<td>• hypochromic ($CI &lt; 0,85$)</td>
</tr>
<tr>
<td></td>
<td>• hyperchronic ($CI &gt; 1,05$)</td>
</tr>
<tr>
<td>According to bone marrow ability to regeneration (according to reticulocyte number)</td>
<td>• regenerator ($Rt$ to $5 %$)</td>
</tr>
<tr>
<td></td>
<td>• hyperregenerative ($&gt; 5 %$)</td>
</tr>
<tr>
<td></td>
<td>• hypo- and aregeneratory ($&lt; 0,2 %$)</td>
</tr>
<tr>
<td>According to course</td>
<td>• acute</td>
</tr>
<tr>
<td></td>
<td>• subacute</td>
</tr>
<tr>
<td></td>
<td>• chronic</td>
</tr>
<tr>
<td>According to severity</td>
<td>• minor ($Hb$ 120-90g/l, $Er –$ not lower than $3,0\times 10^{12}/l$)</td>
</tr>
<tr>
<td></td>
<td>• moderate ($Hb$ 90-70g/l, $Er –$ not lower than $2,5\times 10^{12}/l$)</td>
</tr>
<tr>
<td></td>
<td>• major ($Hb &lt; 70$ g/l, $Er –$ not lower than $2,5\times 10^{12}/l$)</td>
</tr>
</tbody>
</table>
Tests:

1. Content of reticulocytes in hemolytic anemia is:
   a) 0-1 \%o
   b) 2-12 \%o
   c) 20-25 \%o

2. Sideropenic syndrome is the result of lack of:
   a) copper
   b) iron
   c) vitamin B_{12}
   d) folic acid

3. Sideroachrestic anemia is the result of:
   a) deficiency of copper
   b) deficiency of iron
   c) disturbance utilization of iron by cells
   d) deficiency of folic acid

4. Mean corpuscular hemoglobin (MCH) in erythrocytes is:
   a) 15,2-20,4 pg
   b) 25,4-34,6 pg
   c) 35,5-43,2 pg

5. Mean corpuscular hemoglobin (MCH) in iron deficiency anemia is:
   a) 15,2-20,4 pg
   b) 25,4-34,6 pg
   c) 35,5-43,2 pg

6. Mean corpuscular hemoglobin (MCH) in vitamin B_{12} deficiency anemia is:
   a) 15,2-20,4 pg
   b) 25,4-34,6 pg
   c) 35,5-43,2 pg

7. Destruction of erythrocytes in spleen is called:
   a) erythropoiesis
   b) erythrodiapedesis
   c) erythrodieresis
8. The term «poikilocytosis» means:
   a) RBCs with a normal content of hemoglobin;
   b) abnormally shaped RBCs;
   c) RBCs that are irregular in size.

9. Anisocytosis is the changing of:
   a) shape of RBCs
   b) size of RBCs
   c) content of hemoglobin in RBCs

10. Anisochromia – is:
    a) changing of erythrocyte's shape
    b) changing of erythrocyte's size
    c) different intensity of erythrocyte stain

11. In what anemia the color index is increased?
    a) acute posthemorrhagic anemia
    b) vitamin $B_{12}$ deficiency anemia
    c) chronic posthemorrhagic anemia

12. In what anemia the count of reticulocytes is reduced?
    a) acute posthemorrhagic anemia
    b) hemolytic anemia
    c) aplastic anemia

13. In what anemia the count of reticulocytes is increased?
    a) acute posthemorrhagic anemia
    b) vitamin $B_{12}$ deficiency anemia
    c) aplastic anemia

14. Megaloblastic anemia is:
    a) chronic posthemorrhagic anemia
    b) folic acid deficiency anemia
    c) aplastic anemia
    d) hemolytic anemia

15. In what anemia RBCs contain abnormal hemoglobin:
    a) thalassemia
16. *Syntesis of hemoglobin S is representative for:*
   a) thalassemia
   b) sickle-cell anemia
   c) elliptocytosis

17. *Decreasing activity of what enzyme of RBCs leads to hemolytic anemia due to deficiency of ATP?*
   a) dehydrogenase glucose 6-phosphate
   b) sodium-potassium ATPase
   c) pyruvate kinase

18. *Decreasing activity of what enzyme of RBCs leads to hemolytic anemia due to oxidative stress?*
   a) dehydrogenase glucose 6-phosphate
   b) pyruvate kinase
   c) hexokinase

19. *Abnormality synthesis of what substance lead to microspherocytosis?*
   a) hemoglobin A
   b) 2,3-biphosphoglyceric acid
   c) spectrin

20. *Abnormality synthesis of what substance lead to elliptocytosis?*
   a) hemoglobin A
   b) 2,3-biphosphoglyceric acid
   c) spectrin

21. *What kind of anemia is the result of action of ionizing radiation?*
   a) aplastic anemia
   b) iron deficiency anemia
   c) hemolytic anemia

22. *What kind of anemia is the result of disturbance of synthesis in stomach parietal cells intrinsic Castl's factor?*
   a) hemolytic anemia
   b) iron deficiency anemia
23. What disturbance leads to anemia in deficiency of vitamin B\(_{12}\) and folic acid?
   a) decrease of synthesis of nucleic acids
   b) intensification peroxidation
   c) deranged glycolysis

24. What kind of anemia is characterized by megaloblastic type of hemapoiesis?
   a) hemolytic anemia
   b) chronic posthemorrhagic anemia
   c) vitamin B\(_{12}\)-deficiency anemia

25. What kind of anemia is characterized by decreasing synthesis of heme?
   a) iron deficiency anemia
   b) sickle-cell anemia
   c) thalassemia

Anwers:
1bc, 2b, 3c, 4b, 5a, 6c, 7c, 8a, 9b, 10c, 11b, 12c, 13a, 14b, 15a, 16b, 17c, 18a, 19c, 20c, 21a, 22c, 23a, 24c, 25a

LITERATURE:
1. Lecture material.
**LESSON № 21**

**TOPIC:** QUANTITATIVE DISORDERS OF NEUTROPHILS. LEUKOCYTOSIS AND LEUKOPENIA.

**Aim of the lesson:** to study main causes and mechanisms of leukocytosis and leucopenia development.

**QUESTIONS:**
1. Leukocytes. Stages of leucopoiesis. Leukocyte’s description at different stages of leucopoiesis.
2. Functions of leukocytes (neutrophils, eosinophils, basophils, monocytes, lymphocytes).
7. Nuclear shift in leukocyte formula. Types (to-left, to-right).

**Table 4. Normal leukocyte formula (%)**

<table>
<thead>
<tr>
<th></th>
<th>Common</th>
<th>Basophils</th>
<th>Eosinophils</th>
<th>Myelocytes</th>
<th>Metamyelocytes</th>
<th>Band forms</th>
<th>Segments</th>
<th>Lymphocytes</th>
<th>Monocytes</th>
</tr>
</thead>
<tbody>
<tr>
<td>%</td>
<td>100</td>
<td>0-1</td>
<td>1-5</td>
<td>0</td>
<td>0</td>
<td>1-6</td>
<td>45-70</td>
<td>18-40</td>
<td>2-9</td>
</tr>
<tr>
<td>× 10⁶ /l</td>
<td>4000-9000</td>
<td>0-80</td>
<td>80-400</td>
<td>0</td>
<td>0</td>
<td>70-420</td>
<td>3150-5600</td>
<td>1260-2800</td>
<td>140-740</td>
</tr>
<tr>
<td>Children (5 days-5 years) %</td>
<td>100</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>25-40</td>
<td>40-65</td>
<td>2-9</td>
</tr>
</tbody>
</table>
Laboratory work 1. Calculation of leukocyte formula in the blood.

**Description of the work.** Blood sample painted by Romanovski-Gymza is used for calculation of leukocytemic formula in the blood. Under microscope large magnification students estimate the leukocytes according to their nuclea form and size, color of granuls until to the sum of leucocytes equelled 100. The moving of blood sample for this calculation should be according zigsage line. All meeting leucocytes are classificated according to their type and the result is the leukocytemic formula in percentages.

![Fig. 9. Toxigenic granules, cytoplasm vacuolization in leucocytes](image)

Fig. 10. Neutrophilic leucocytosis
Tests

1. One of the developmental stages of neutrophilic leukocyte is:
   a) myeloblast
   b) prolymphocyte
   c) promonocyte
   d) monoblast

2. One of the developmental stages of lymphocyte is:
   a) myeloblast
   b) lymphoblast
   c) promonocyte
   d) monoblast

3. Stages of development of monocyte are:
   a) myeloblast
   b) prolymphocyte
   c) promonocyte
   d) monoblast

4. Morphologically recognized granulocytic cell is:
   a) lymphoblast
   b) myeloblast
   c) monoblast
   d) erythroblast

5. Granulocytes synthesize in:
   a) spleen
   b) bone marrow
   c) liver
   d) nodi lymphatici

6. What cells are mononuclear leukocytes?
   a) eosinophil
   b) monocyte
   c) neutrophil
   d) basophil
7. The functions of neutrophils are:
   a) synthesis of antibodies
   b) phagocytosis
   c) secretion of enzymes and bactericide agents
   d) secretion of histamine and heparin

8. The calculation of leucocytes count occurs in:
   a) blood smear
   b) Goryave camera
   c) special tube
   d) photocolorimetric determination

9. The calculation of leucocyte formula occurs in:
   a) blood smear
   b) Goryaev camera
   c) special tube
   d) photocolorimetric determination

10. Leukocyte formula is:
    a) percentage of the different types of the leukocytes
    b) absolute leukocytes count
    c) relation of nonmature forms of leucocytes to mature ones
    d) relation of granulocytes and nongranulocytes

11. Children have the following peculiarities of leukocyte formula:
    a) lymphocytes predominate at birth
    b) neutrophils predominate on the 5th year
    c) leukocyte formula is the same as in adults on the 5th day of life
    d) lymphocytes predominate at 2 years old

12. Physiologic leukocytosis occurs:
    a) in administration of glucocorticoids
    b) after food intake
    c) in physical activity
    d) in sleep

13. The causes of relative leukocytosis are:
14. *Acute purulent inflammatory processes lead to:*
   a) eosinophilia
   b) lymphocytosis
   c) neutrophilic leukocytosis

15. *What type of leukocytes increase in allergic reactions more often?*
   a) eosinophils
   b) neutrophils
   c) lymphocytes

16. *What type of leukocytosis appears in chronic inflammatory processes very often?*
   a) eosinophilic
   b) basophilic
   c) neutrophilic
   d) monocytic

17. *Index of the nuclear shift is relation:*
   a) of the count of immature forms of neutrophils to mature ones;
   b) of granulocytes and nongranulocytes;
   c) of the count of granulocytes to band forms;
   d) of myeloblasts to myelocytes.

18. *The types of nuclear shift to the left are:*
   a) myelocytic
   b) degenerative
   c) monocytic
   d) leukemoid

19. *The nuclear shift to the right is the increase of the:*
   a) common count of leukocytes
   b) percent of the mature neutrophils
   c) percent of the lymphocytes
   d) count of granular leukocytes
20. *The nuclear shift to the left is:*  
   a) decrease the mature leucocytes from common count of leucocytes  
   b) increase the count of immature neutrophils  
   c) increase percent of the lymphocytes  
   d) decrease the count of granular leukocytes  

21. *Nuclear shift shows:*  
   a) type of leukocytosis  
   b) severity of inflammation  
   c) type of leukopenia  
   d) stage of inflammation  

22. *What index is used for assessment of degree of the nuclear shift in leukogramm?*  
   a) Bobrov's  
   b) Tiffno  
   c) nucleocytoplasmic  

23. *When does eosinophil leukocytosis occur?*  
   a) in viral diseases  
   b) in autoimmune processes  
   c) in bacterial infection in recovery  

24. *What diseases can lead to eosinophilia?*  
   a) croupous pneumonia  
   b) tuberculosis  
   c) helminthic invasion  
   d) cardiac infarction  
   e) viral hepatitis  

25. *Pathologic leukocytosis can occur:*  
   a) after a hot bath  
   b) at pregnancy  
   c) at administration of glucocorticoids  
   d) at helminthiasis  

26. *What type of leukocytes increase in viral infection more often?*
a) eosinophils  
b) neutrophils  
c) monocytes  
d) lymphocytes

27. Administration of cytostatics leads to:
   a) neutrophilia  
b) monocytosis  
c) thrombocytosis  
d) agranulocytosis

28. Mycosis leads to:
   a) eosinophilia  
b) lymphocytosis  
c) monocytopenia

29. We should differentiate leukemoid reaction of neutrophils type with:
   a) chronic lymphatic leukemia  
b) chronic myeloid leukemia  
c) acute myeloblastic leukemia  
d) acute lymphoblastic leukemia

30. The criteria of agranulocytosis is:
   a) count of leukocytes below $2.5 \times 10^9/\text{l}$  
b) count of leukocytes below $1 \times 10^9/\text{l}$  
c) absence of agranular leukocytes  
d) increasing count of agranular leukocytes

31. The term “agranulocytosis” denotes:
   a) increased count of agranulocytes in blood  
b) abrupt decreased count of granulocytes in blood  
c) disappearance of specific granulosity in cells

Answers:
1a, 2b, 3cd, 4b, 5b, 6b, 7bc, 8b, 9a, 10a, 11d, 12bc, 13ad, 14c, 15a, 16d, 17a, 18bd, 19b, 20b, 21b, 22a, 23bc, 24c, 25cd, 26d, 27d, 28b, 29b, 30b, 31b
LITERATURE:
1. Lecture material.
LESSON № 22

TOPIC: LEUKEMIAS.

Aim of the lesson: to study the picture of perefericial blood of more frequent forms of human leukemia.

QUESTIONS:

1. Leukemia. Definition. Etiology.
3. The particularities of leukemic cells.
8. The difference between leukosis and leukocytosis.

Laboratory work 1. Microscopia of blood samples in patients with acute myeloid leukemia.

Description of the work. Blood sample painted by Romanovski-Gymza is used for diagnostic procedure of acute myeloid leukemia. Under microscope large magnification students estimate the type of leukocytes.
The myeloblasts are marker of myeloid leukemia in peripheral blood. The characteristic sign of acute myeloid leukemia is the big amount of myeloblasts, small level of mature segment neutrophills and absence of metamyelocytes and myelocytes (hiatus leukemicus). Such signs occur during acute myeloid leukemia only.

Students make a drawing of acute myeloid leukemia in copybooks.

**Laboratory work 2. Microscopia of blood samples in patients with chronic myeloid leukemia.**

**Description of the work.** Blood sample painted by Romanovski-Gymza is used for diagnostic procedure of chronic myeloid leukemia. Under microscope large magnification students estimate the leukocytes according to their nuclea form and size, granul’s color. The myeloblasts are marker of myeloid leukemia in peripheral blood.
The characteristic sign of chronic myeloid leukemia is the presence of metamyelocytes, myelocytes and eosinophilic-basophilic association. Eosinophilic-basophilic association is the increasing percent of basophills and eosinophills in leukocytemic formula simultaneously. Such increase generally occurs during chronic myeloid leukemia.

Students make a drawing of chronic myeloid leukemia in copybooks.

**Laboratory work 3. Microscopia of blood samples in patients with chronic lymphoid leukemia.**

**Description of the work.** Blood sample painted by Romanovski-Gymza is used for diagnostic procedure of chronic lymphoid leukemia. At microscope large magnification students estimate the type of leukocytes. The lymphoblasts are markers of lymphoid leukemia in peripheral blood.
The characteristic sign of chronic myeloid leukemia is increased percent of lymphocytes (lymphocytosis) and Botkin-Humprecht bodies. Such signs occur during chronic lymphoid leukemia only. Students make a drawing of chronic lymphoid leukemia in copybooks.

Tests

1. The term "anaplasia" means:
   b) increase of tumor mass
   c) accelerated tumor cells division
   d) differentiation disorders of the tumor cells

2. Intensity of glycolisis in the tumor cells:
   a) rises
   b) diminishes
   c) is unchanged

6. Synthesis of nucleic acids in the malignant cells:
   a) rises
   b) diminishes
   c) has no change

7. The type of regulation of tumor cells growth is:
   a) endocrine
b) autocrine

c) paracrine

8. *Proliferation of the malignant cells is conditioned by:*
   a) activation of the oncogenes
   b) intensification of apoptosis
   c) inactivation of the suppressor genes
   d) derangement of apoptosis

9. *Carbohydrate metabolism in tumor is characterized by:*
   a) increase of the glycogen synthesis
   b) intensification of glycogenesis
   c) activation of glycolysis
   d) intensification of glycogenolysis

10. *Lipid metabolism in tumor is characterized by:*
    a) intensification of lipolysis
    b) intensification of lipogenesis
    c) decrease of the fatty acids synthesis

11. *First of all ren sarcoma metastasize into:*
    a) liver
    b) organs of pelvis minor
    c) intestine
    d) lung

12. *Antigenic complex in malignant cells is characterized by:*
    a) increase of normal gens synthesis
    b) appearance of the carcinoembryonic antigens
    c) appearance of the specific antigens

13. *Malignant tumor is characterized by:*
    a) expansive growth
    b) the absence of metastases
    c) autonomy, self-regulation of growth
    d) cellular atypism

14. *Bening tumor is characterized by:*
    a) expansive growth
b) the absence of metastases
c) metastases
d) cellular atypism

12. Stages of the tumor development are:
a) promotion
b) progression
c) translocation
d) inversion

13. The local effects of the tumor include:
a) bleeding
b) bowel obstruction
c) anemia
d) anorexia
e) endocrine syndromes

14. Common systemic effects of the tumor include:
a) bleeding
b) bowel obstruction
c) anemia
d) anorexia
e) endocrine syndromes

15. Anticancerogenic protection includes:
a) antioxidants
b) lymphocytes
c) glucose
d) ammonia

16. The mechanisms of anticancerogenic protection are:
a) antinocellular
b) antimutative
c) antimicrobial

17. In aleucaemic type of leucaemias:
a) amount of leukocytes increases in blood
b) amount of leukocytes decreases in blood
c) normal count of leukocytes in blood
d) blasts are absent

18. *Chronic myeloleukaemia may be associated with:*
   a) increasing blast count
   b) decreasing blast count
   c) increasing amount of basophils and eosinophils
   d) absent hiatus leucaemicus

19. *Following changes are observed in peripheral blood in acute myeloblastic leukaemia:*
   a) elevated peripheral blood myeloblast count
   b) low peripheral blood myeloblast count
   c) the presence of Botkin-Gumbrecht bodies
   d) the presence of hiatus leukemicus

20. *Following changes are observed in the peripheral blood in chronic lymphoblastic laeucaemia:*
   a) decreased count of lymphoblasts
   b) increased count of myeloblasts
   c) the presence of Botkin-Gumbrecht bodies
   d) increased amount of eosinophils and basophils

21. *Following changes are observed in the peripheral blood in acute lymphoblastic leucaemia:*
   a) small count of lymphoblasts
   b) increased count of megaloblasts
   c) increased count of lymphoblasts
   d) the presence of Botkin-Gumbrecht bodies

22. *Hiatus leukemicus is the absence of:*
   a) immature cells
   b) morphologically don’t differentiation cells
   c) mature cells

23. *Increased count of eosinophils and basophils can be observed in:*
   a) chronic lympholeukosis
   b) chronic myeloleukosis
   c) acute lympholeukosis
   d) acute myelogenous leukemia
24. *Leucaemia is:*  
a) benign tumor of hematopoietic tissue  
b) sign of the inflammation  
c) malignant tumor of hematopoietic tissue  
d) sign of the allergy

25. *Acute leucaemia differs from chronic one by:*  
a) the presence of anemia  
b) the absence of hiatus leukemicus  
c) the presence of hiatus leukemicus  
d) small blast count in peripheral blood

26. *Chronic leucaemia differs from acute leukosis by:*  
a) the absence of hiatus leukemicus  
b) the presence of hiatus leukemicus  
c) small blast count in peripheral blood  
d) increase of blast count in peripheral blood

27. *The main change in a hemogram in acute leucamias is:*  
a) low percent of the blasts  
b) the presence of the cells of 5 class of maturation  
c) the presence of hiatus leucaemicus  
d) high percent of the blast cells

28. *The main change in a hemogram at chronic leucamias is:*  
a) low percent of the blasts  
b) the presence of the cells of 5 class of maturation  
c) the presence of hiatus leukemicus  
d) high percent of the blast cells  
e) the absence of the blasts

29. *In acute lecaemia:*  
a) cellular differentiation fully stops on the 2-4 class level  
b) cellular differentiation don’t fully stops on the 2-4 class level

**Answers:**
1c, 2a, 3a, 4b, 5acd, 6cd, 7a, 8d, 9bc, 10cd, 11ab, 12ab, 13ab, 14cde, 15ab, 16ab, 17cd, 18bcd, 19ad, 20a, 21ac, 22a, 23b, 24c, 25c, 26ac, 27cd, 28ab, 29a.

LITERATURE:
1. Lecture material.
LESSON № 23

TOPIC: DISORDERS OF HEMOSTASIS. THROMBOPHILIC DISORDERS OF HEMOSTASIS. THROMBOSIS AND EMBOLISM.

Aim of the lesson: to study causes and mechanisms of thrombosis and embolism development.

QUESTIONS:

1. Hemostasis, its components (blood vessels, platelets, plasma coagulation factors).
2. Disorders of hemostasis (thrombophylitic, hemorrhagic, thrombohemo-rrhagic hemostasiopathias).
3. Description of vessel-platelet hemostasis.
4. Description of hemocoagulate hemostasis.
5. Thrombosis. Outcomes and consequences.
7. Mechanism of red thrombosis development.
8. Causes and conditions of thrombosis development. Virchow’s triad:
   a. vascular injury
   b. blood coagulant system activation
   c. blood flow decrease
11. Causes of fat, gas and air embolism development.

Table 5. Glossary of Coagulation Proteins
<table>
<thead>
<tr>
<th>Factor</th>
<th>Synonym</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Fibrinogen</td>
</tr>
<tr>
<td>II</td>
<td>Prothrombin</td>
</tr>
<tr>
<td>III</td>
<td>Tissue thromboplastin, tissue factor</td>
</tr>
<tr>
<td>V</td>
<td>Proaccelerin, labile factor</td>
</tr>
<tr>
<td>VII</td>
<td>Proconvertin, stable factor</td>
</tr>
<tr>
<td>VIII</td>
<td>Antithemophilic factor</td>
</tr>
<tr>
<td>IX</td>
<td>Christmas factor</td>
</tr>
<tr>
<td>X</td>
<td>Stuart factor</td>
</tr>
<tr>
<td>XI</td>
<td>Plasma thromboplastin antecedent</td>
</tr>
<tr>
<td>XII</td>
<td>Hageman factor</td>
</tr>
<tr>
<td>XIII</td>
<td>Fibrin-stabilizing factor</td>
</tr>
<tr>
<td>Prekallikrein</td>
<td>Fletcher factor</td>
</tr>
<tr>
<td>HMWK</td>
<td>Fitzgerald factor</td>
</tr>
</tbody>
</table>

Vascular Injury

Exposure of Circulating Blood To:

**Intrinsic**

Foreign Surface
XII
Prekallikrein
HMW-K

- XI $\rightarrow$ $\text{X}_A$
- $\text{IX}_{Ca^{2+}}$ $\rightarrow$ $\text{IX}_A$

- $\left[ \text{VIII} + \text{IX}_A \right] + \text{PL} + \text{Ca}^{2+}$

**Extrinsic**

Tissue Thromboplastin (III)
(Containing Phospholipid)

- $\left[ \text{VII} + \text{Ca}^{2+} \right]$

**Contact Phase**

- Complex

Common

- X $\rightarrow$ $\text{X}_A$

- $\left[ \text{V} + \text{X}_A \right] + \text{PL} + \text{Ca}^{2+}$

**Prothrombin (II)**

- Thrombin

Fibrinogen (I) $\rightarrow$ Fibrin Monomer $\rightarrow$ Fibrinopeptides A & B

- Fibrin Polymer
  - (Insoluble-Unstable)
  - Non-Covalent

Fibrinogen (I) $\rightarrow$ Fibrin Monomer $\rightarrow$ Fibrinopeptides A & B

- Fibrin Polymer
  - (Insoluble-Stable)
  - Covalent
Fig. 14. Scheme of blood coagulation

LABORATORY WORK

Laboratory work 1. White thrombus formation in peritoneum vessels of a frog.

**Work description:** after fixation of the frog and exposure of mesentery students watch the normal blood flow in mesentery vessels under microscope small magnification. The sodium chloride crystal should be put on vein confluence under microscope magnification control. Students investigate the process of white thrombus formation in frog peritoneum during 20-40 min using microscope.

![Fig. 15. White thrombus](image)

Students analyze, draw and make conclusions.

Laboratory work 2. Red thrombus development in peritoneum vessels of a frog.

**Work description:** using the same frog students damage the vein by sharp needle under microscope small magnification. Then students watch the process of red thrombus formation, analyze, draw and make conclusions.
Fig. 16. Thrombus in the veins of peritoneum of frogs
1. veins
2. crystall NaCl
3. white thrombus
4. place of the vessel lesion
5. red (obstructive) thrombus

Students analyze, draw and make conclusions.

Laboratory work 3. Fat embolism of peritoneum vessels in a frog.

Work description: after fixation of the frog and exposure of mesenteric students watch the normal blood flow in mesenteric vessels under microscope small magnification. After middle thoracotomia fat solution should be administrated intracardially and students watch the emboli circulation and embolism of small peritoneum arteries.

Laboratory work 4. Thrombelastography (TEG).
Work description: Thrombelastography (TEG) is a method of testing the efficiency of coagulation in the blood. The method is performed on the Thromboelastometer at constant temperature of 37°C.
A small blood sample (typically 0.36 ml) is placed into a cuvette (cup) which is rotated gently through 4° 45’ (cycle time 6/min) to imitate sluggish venous flow and activate coagulation. When a sensor shaft is inserted into the sample a clot forms between the cup and the sensor. The speed and strength of clot formation is measured in various ways (now usually by computer), and depends on the activity of the plasmatic coagulation system, platelet function, fibrinolysis and other factors which can be affected by illness, environment and medications.

The patterns of changes in strength and elasticity in the clot provide information about how well the blood can perform hemostasis (the halting of blood flow), and how well or poorly different factors are contributing to clot formation.

![Diagram of thrombelastogram](image)

**Fig. 17. Outline of thrombelastogram and its indices**
*the indices are described lower in the text*

Four values that represent clot formation are determined by this test:

R – value (or reaction time). The R value represents the time until the first evidence of a clot is detected.

K – value (thrombin time). The K value is the time from the end of R until the clot reaches 20 mm and this represents the speed of clot formation. The angle is the tangent of the curve made as the K is reached and offers similar information to K.

MA – maximum amplitude. The MA is a reflection of clot strength.

T – total time of blood coagulation (5-10 min normally).
Fig. 18. Thromboelastogramms

a – healthy
b – hypercoagulation phase of the DIC-syndrom
c – connecting phase of DIC-syndrom;
d – e – hypocoagulation phase of the DIC-syndrom

Fill in a table:

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<th>What thrombus localization leads to?</th>
<th>Pulmonary artery emboli</th>
<th>Stroke</th>
<th>Infarctum myocardium</th>
<th>Gangrene of low extremities</th>
<th>Infarctum of intestine</th>
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Fill in a table:

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<th>Left parts of the heart</th>
<th>Aorta</th>
<th>Venous of low extremities</th>
<th>Pelvic veins</th>
<th>Venous sinuses of the brain</th>
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**Tests**

1. *The functions of platelets in hemostasis are:*
   a) angiotrophic
   b) adhesive
   c) coagulative
   d) bactericidic

2. *Platelet precursor is:*
   a) plasmacytoblast
   b) myeloblast
   c) megacytoblast
   d) lymphoblast

3. *Which factor can initiate blood coagulation?*
   a) factor I
   b) factor X
   c) factor XII
   d) prothrombin

4. *What platelet factor takes part in the prothrombinase synthesis?*
   a) 3
   b) 4
   c) actomyosin
   d) thromboxane

5. *Inducer of platelets aggregation is:*

6. Antiaggregant for thrombocytes is:
a) thrombin
b) ADP
c) collagen
d) aspirin

7. Extrinsic coagulation pathway of hemostasis includes activation of:
a) factor VII
b) factor VIII
c) factor IX
d) factor XII

e) factor XIII

8. The factor which converts the prothrombin to thrombin:
a) factor I
b) factor VII
c) factor IXa
d) factor Xa
e) factor XIII

9. The platelet-vascular hemostasis is necessary for:
a) white thrombus formation
b) red thrombus formation
c) the postcagulative changing of the thrombus

10. The anticoagulants are:
a) fibrinogen and fibrin-split products
b) antithrombin III
c) heparin
d) ADP

11. Fibrin-split products stimulate:
a) synthesis of factor III
b) destruction of fibrin
c) activation of factor XII

12. *The clot retracts by:*
   a) fibrin-stabilizing factor
   b) thrombocytes factors
   c) kinins system

13. *The synthesis of thrombin is blocked by:*
   a) calcium ions
   b) collagen
   c) von Willebrand factor
   d) anticoagulants

14. *The endothelial cells of intact vessels prevent blood coagulation by secretion of:*
   a) prostacyclin
   b) thromboxane
   c) factor IX
   d) vitamin K

15. *Antithrombogenic property of the endothelium is caused by production of:*
   a) prostacyclin
   b) protein C
   c) NO
   d) angiotensin II

16. *Procoagulant activity of the endothelium is caused by the production of:*
   a) prostaglandin I\(_2\)
   b) NO
   c) angiotensin II
   d) endothelin

17. *Anticoagulant system is aimed at:*
   a) depression of blood coagulation
   b) thrombolysis

18. *Plasmin system is aimed at:***
a) depression of blood coagulation  
b) thrombolysis

19. What thrombus forms the first phase of blood clot formation?  
a) white  
b) red

20. The distinctive signs of a clot are:  
a) has white head that fixed to the vessels  
b) synthesis intravital  
c) doesn’t fixed to the vessels  
d) synthesis posthumously

21. The major constituent of red thrombus is:  
a) fibrin  
b) RBC  
c) WBC  
d) albumens  
e) platelets

22. The major constituent of white thrombus is:  
a) fibrin  
b) RBC  
c) WBC  
d) albumens  
e) platelets

23. Types of endogenous embolism are:  
a) thromboembolism  
b) gas embolism  
c) air embolism  
d) embolism by foreign bodies  
e) cellular embolism

24. Types of exogenous embolism are:  
a) gas embolism  
b) thromboembolism  
c) air embolism  
f) embolism by foreign bodies
d) embolism by bacteria

25. What is the localization of the thrombus in embolism of the pulmonary vessels?
   a) in the veins of the greater circulation
   b) in the right part of the heart
   c) in the left part of the heart
   d) in arteries of the greater circulation
   e) in portal veins system

28. What is the localization of thrombus in embolism of arterial vessels of greater circulation?
   a) in the veins of the greater circulation
   b) in the right part of the heart
   c) in the left part of the heart
   d) in arteries of the greater circulation
   e) in portal veins system

Answers:
1abc, 2c, 3c, 4a, 5bd, 6d, 7a, 8d, 9ac, 10abc, 11b, 12ab, 13d, 14ab, 15abc, 16cd, 17a, 18b, 19a, 20ab, 21cd, 22a, 23e, 24abe, 25cde, 26ab, 27cd.

LITERATURE:
1. Lecture material.
LESSON № 24

TOPIC: DISORDERS OF HEMOSTASIS. HEMORRHAGIC DISORDERS OF HEMOSTASIS.

Aim of the lesson: to study hemostatic disorders with hemorrhagia, to meet the methods of hemostatic investigation.

QUESTIONS:

5. Disorders of blood vessels. Reasons and mechanisms of their development, clinical manifestations:
   - scurvy,
   - Henoch-Schonlein purpura,
   - Rendu-Osler-Weber syndrome.
   - coagulation disorders, connected with breaking of the 1-st phases of blood coagulation. Hemophilia.
   - coagulation disorders, connected with breaking of the 2-nd phases of blood coagulation.
   - coagulation disorders, connected with breaking of the 3-d phases of blood coagulation.
Laboratory work 1. Calculation of thrombocytes amount in blood.

Work description. The students investigate blood samples painted by Romanovsky-Gymza in separated area. Thrombocytes look like small violet points between erythrocytes. These points should be calculated per 1000 RBC, the level of which has already been detected. The sum of platlets (A) is used for thrombocytes amount calculation according to the formula:

$$\text{Thrombocytes} = \frac{A \times RBC}{1000},$$

where

A – sum of platlets, estimated per 1000 RBCs,
RBC – erythrocytes level per liter.

Fig. 19. Scheme of DIC-syndrom
Tests:

1. *Disturbances of blood coagulation lead to:*
   a) disease of hemostasis
   b) disease of homeostasis
   c) metabolism disorder
   d) inflammation

2. *Hemostasiopathia is the disturbance of:*
   a) the whole haemostasis system
   b) blood coagulation
   c) anticoagulation system
   d) platelets
   e) vessel wall

3. *Hemorrhagic hemostasiopathia is a kind of hemostasis disorder connected with:*
   a) increased coagulability of blood
   b) increased bleeding
   c) increased thrombocytopoiesis
   d) simultaneous thrombosis and bleeding

4. *Trombophilic hemostasiopathias is a kind of hemostasis disorder connected with:*
   a) increased coagulability of blood
   b) increased bleeding
   c) increased thrombocytopoiesis
   d) simultaneous thrombosis and bleeding

5. *Trombohemorrhagic hemostasiopathias is a kind of pathology with:*
   a) increased bleeding
   b) increased trombocytopoiesis
   c) simultaneous thrombosis and bleeding
6. *What concerns to hemorrhagic hemostasiopathias?*
   a) DIC 
   b) thrombosis 
   c) angiopathies 
   d) coagulation disorders 
   e) thrombocytopenia 

7. *The count of platelets in hemorrhagic syndrome is less than:*
   a) $150 \times 10^9 /l$ 
   b) $320 \times 10^9 /l$ 
   c) $50 \times 10^9 /l$ 
   d) $400 \times 10^9 /l$ 

8. *The causes of thrombocytopenia are:*
   a) malignant tumor 
   b) radiation 
   c) acute hemorrhage 
   d) icterus 

9. *Idiopathic thrombocytopenic purpura is a:*
   a) coagulation disorder 
   b) thrombocytopenia 
   c) angiopathy 
   d) thrombophilia 

10. *The causes of angiopathy are:*
    a) infectious disease 
    b) allergy 
    c) diabetes mellitus 
    d) hypovitaminosis P and C 

11. *Scorbutus is a:*
    a) coagulation disorder 
    b) thrombocytopenia 
    c) angiopathy 
    d) trombohemorrhagic hemostasiopathias 

12. *Henoch-Schonlein purpura is:*
    a) coagulation disorder
b) thrombocytopenia  
c) angiopathy  
d) trombohemorrhagic hemostasiopathias

13. The mechanism of increased hemorrhage in immune thrombocytopenic purpura is:  
a) decreased platelet adhesion  
b) decreased platelet aggregation  
c) lack of nutrition function of the platelets  
d) disorder of vasoactive function of platelets

14. Glanzmann thrombasthenia is:  
a) angiopathy  
b) qualitative disorder of platelets  
a) coagulation disorder  
b) disorder of plasmin system

15. Hemorrhagic hemostasiopathies are:  
a) qualitative disorders of platelets  
b) angiopathy  
c) posthemorrhagic anemia  
d) leukemoid response

16. Following tests are used for assessment of aggregation properties of trombocyte hemostasis component:  
a) pinch test  
b) Nesterovs test  
c) tromboelastography  
d) aggregatogram

17. Anticoagulative system is:  
a) antithrombin III  
b) antihemophilic globulin  
c) angiotensin  
d) plasmin

18. Anticoagulants are:  
a) antithrombin III
b) antihemophylic globulin

c) heparin

d) plasmin

19. **Fibrinolytics are:**
   a) antithrombin III
   b) antihemophylic globulin
   c) heparin
   d) plasmin

20. **Anticoagulative effect of heparin is** inhibition of:
   a) prothrombinase synthesis
   b) the synthesis of thrombin
   c) fibrin synthesis
   d) all the phases of blood coagulation

21. **The anticoagulative effect of plasmin is:**
   a) inhibition of prothrombinase formation
   b) inhibition of thrombin formation
   c) inhibition of fibrinogenesis
   d) activation of fibrinolysis

22. **Coagulation disorders are:**
   a) scorbuto
   b) Henoch-Schonlein purpura
   c) idiopathic thrombocytopenic purpura
   d) haemophilia

23. **The coagulation disorders are:**
   a) hemorrhagic newborn disease
   b) Henoch-Schonlein purpura
   c) idiopathic thrombocytopenic purpura
   d) hemophylia

24. **Coagulation disorder of the first phase of blood coagulation is:**
   a) hemorrhagic newborn disease
   b) lack of Fitzgerald factor
   c) idiopathic thrombocytopenic purpura
   d) hemophylia
25. The cause of the coagulation disorders with disturbance of the first phase of blood coagulation is the deficiency of:
   a) vitamin K
   b) Fletcher's factor
   c) Hageman's factor
   d) fibrinogen

26. Deficiency of what factor leads to hemophilia A?
   a) VIII
   b) IX
   c) XI

27. Deficiency of what factor leads to hemophilia B?
   a) VIII
   b) IX
   c) XI

28. Deficiency of fibrinogen leads to disorder of:
   a) synthesis of prothrombinase
   b) thrombinogenesis
   c) fibrinogenesis
   d) retraction and fibrinolysis

29. Lack of plasmin leads to disorder of:
   a) prothrombinogenesis
   b) trombinogenesis
   c) fibrinogenesis
   d) retraction and fibrinolysis

30. Deficiency of prothrombin leads to disorder of:
   a) prothrombinogenesis
   b) trombinogenesis
   c) fibrinogenesis
   d) retraction and fibrinolysis

31. Deficiency of retractozim leads to disturbance of:
   a) synthesis of prothrombinase
   b) synthesis of thrombin
32. Deficit of calcium leads to disturbance of:
   a) synthesis of prothrombinase
   b) synthesis of thrombin
   c) synthesis of fibrin
   d) all phases of secondary hemostasis

33. Lack of calcium leads to:
   a) increasing hemorrhage
   b) Thrombosis
   c) DIC
   d) Excess of thrombus retraction

34. The coagulation disorder with disturbance of the 2\textsuperscript{nd} stage of blood coagulation is:
   a) hemorrhagic newborn disease
   b) lack of fibrinogen
   c) idiopathic thrombocytopenic purpura
   d) excess use of coumarin drugs

35. The coagulation disorder with disturbance of the 3\textsuperscript{rd} stage of blood coagulation are:
   a) hemorrhagic newborn disease
   b) lack of kininogen
   c) idiopathic thrombocytopenic purpura
   d) lack of fibrinogen

36. Lack of vitamin K leads to:
   a) coagulation disorder
   b) DIC
   c) thrombocytopenia
   d) angiopathy

37. Hemophilia is a:
   a) coagulation disorder
   b) thrombocytopenia
   c) angiopathy
d) thrombohemorrhagic hemostasiopathias

38. Von Willebrand disease is:
   a) angiopathy
   b) thrombocytopenia
   c) coagulation disorder
   d) admixed hemorrhagic hemostasiopathias

39. DIC concerns to hemostasiopathias:
   a) thrombophilic
   b) hemorrhagic
   c) thrombohemorrhagic

40. The causes of DIC are:
   a) malignant tumor
   b) acute radiation sickness
   c) acute bleeding
   d) cardiac insufficiency

Answers:
1a, 2a, 3b, 4ac, 5c, 6cde, 7c, 8ab, 9b, 10d, 11c, 12c, 13c, 14b, 15ab, 16d, 17ad, 18ac, 19d, 20d, 21d, 22d, 23ad, 24bd, 25bd, 26a, 27b, 28c, 29d, 30b, 31d, 32d, 33a, 34ad, 35d, 36a, 37a, 38d, 39c, 40ab.

LITERATURE:
1. Lecture material.
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<th>Hemogramm № 2</th>
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### Hemogramm № 5

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### Hemogramm № 6

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### Hemogramm № 8

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<td>-------</td>
</tr>
<tr>
<td>Reticulocytes</td>
<td>0.9 %</td>
</tr>
<tr>
<td>Hemogramm № 9</td>
<td>Hemogramm № 10</td>
</tr>
<tr>
<td>---------------</td>
<td>---------------</td>
</tr>
<tr>
<td>Hemoglobin (Hgb)</td>
<td>119</td>
</tr>
<tr>
<td>g/l</td>
<td></td>
</tr>
<tr>
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<td>4,1 x 10^{12} / l</td>
</tr>
<tr>
<td>10^{12} / l</td>
<td></td>
</tr>
<tr>
<td>Leucocytes (WBC)</td>
<td>57 x 10^9 / l</td>
</tr>
<tr>
<td>10^9 / l</td>
<td>Eosinophils</td>
</tr>
<tr>
<td>Basophils</td>
<td>0 %</td>
</tr>
<tr>
<td>Eosinophils</td>
<td>0 %</td>
</tr>
<tr>
<td>Neutrophiles:</td>
<td></td>
</tr>
<tr>
<td>myelocytes</td>
<td>0 %</td>
</tr>
<tr>
<td>methamyelocytes</td>
<td>0 %</td>
</tr>
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<td>band forms</td>
<td>0 %</td>
</tr>
<tr>
<td>eutrophils</td>
<td>9 %</td>
</tr>
<tr>
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<td>7 %</td>
</tr>
<tr>
<td>Lymphocytes</td>
<td>81 %</td>
</tr>
<tr>
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<td>3 %</td>
</tr>
<tr>
<td>Thrombocytes (Plt)</td>
<td>160 x 10^9 / l</td>
</tr>
<tr>
<td>10^9 / l</td>
<td></td>
</tr>
<tr>
<td>The Botkin-Humprecht bodes are found</td>
<td></td>
</tr>
</tbody>
</table>

<table>
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<th>Hemogramm № 12</th>
</tr>
</thead>
<tbody>
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</tr>
<tr>
<td>g/l</td>
<td></td>
</tr>
<tr>
<td>Erythrocytes (RBC)</td>
<td>6,3 x 10^{12} / l</td>
</tr>
<tr>
<td>10^{12} / l</td>
<td></td>
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<tr>
<td>Leucocytes (WBC)</td>
<td>6,25 x 10^9 / l</td>
</tr>
<tr>
<td>10^9 / l</td>
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<tr>
<td>Eosinophils</td>
<td>1 %</td>
</tr>
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<td>Neutrophiles:</td>
<td></td>
</tr>
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<td>0 %</td>
</tr>
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<td>0 %</td>
</tr>
<tr>
<td>band forms</td>
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</tr>
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<td>70 %</td>
</tr>
<tr>
<td>Lymphocytes</td>
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<td>Blood Parameter</td>
<td>Value</td>
</tr>
<tr>
<td>-------------------------</td>
<td>---------------</td>
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<td>Thrombocytes (Plt)</td>
<td>380 x 10⁹ / l</td>
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<td>Thrombocytes (Plt)</td>
<td>410 x 10⁹</td>
</tr>
<tr>
<td>Reticulocytes</td>
<td>5 % / l</td>
</tr>
<tr>
<td>Hemogramm № 13</td>
<td>Hemogramm № 14</td>
</tr>
<tr>
<td>----------------</td>
<td>----------------</td>
</tr>
<tr>
<td>Hemoglobin (Hgb)</td>
<td>120 g/l</td>
</tr>
<tr>
<td>Erythrocytes (RBC)</td>
<td>4.36 x 10^{12} / l</td>
</tr>
<tr>
<td>Leucocytes (WBC)</td>
<td>16.2 x 10^{9} / l</td>
</tr>
<tr>
<td>Basophils</td>
<td>0 %</td>
</tr>
<tr>
<td>Eosinophils</td>
<td>1 %</td>
</tr>
<tr>
<td>Neutrophiles:</td>
<td></td>
</tr>
<tr>
<td>myelocytes</td>
<td>0 %</td>
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<tr>
<td>methamyelocytes</td>
<td>0 %</td>
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<td>5 %</td>
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<td>37 %</td>
</tr>
<tr>
<td>Lymphocytes</td>
<td>50 %</td>
</tr>
<tr>
<td>Monocytes</td>
<td>7 %</td>
</tr>
<tr>
<td>Thrombocytes (Plt)</td>
<td>270 x 10^{9} / l</td>
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</table>

<table>
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<tbody>
<tr>
<td>Hemoglobin (Hgb)</td>
<td>102 g/l</td>
</tr>
<tr>
<td>Erythrocytes (RBC)</td>
<td>3.9 x 10^{12} / l</td>
</tr>
<tr>
<td>Leucocytes (WBC)</td>
<td>17.5 x 10^{9} / l</td>
</tr>
<tr>
<td>Basophils</td>
<td>0 %</td>
</tr>
<tr>
<td>Eosinophils</td>
<td>0.5 %</td>
</tr>
<tr>
<td>Neutrophiles:</td>
<td></td>
</tr>
<tr>
<td>myelocytes</td>
<td>4.5 %</td>
</tr>
<tr>
<td>methamyelocytes</td>
<td>16 %</td>
</tr>
<tr>
<td>band forms</td>
<td>39 %</td>
</tr>
<tr>
<td>neutrophils</td>
<td>32 %</td>
</tr>
<tr>
<td>Lymphocytes</td>
<td>4.5 %</td>
</tr>
<tr>
<td>Monocytes</td>
<td>3.5 %</td>
</tr>
<tr>
<td>Thrombocytes (Plt)</td>
<td>310 x</td>
</tr>
<tr>
<td>Reticulocytes</td>
<td>0 %</td>
</tr>
</tbody>
</table>
$10^9 / 1$
<table>
<thead>
<tr>
<th>Hemogramm № 17</th>
<th>Hemogramm № 18</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hemoglobin (Hgb)</td>
<td>40 g/l</td>
</tr>
<tr>
<td>Erythrocytes (RBC)</td>
<td>0,797 x 10¹² / l</td>
</tr>
<tr>
<td>Leucocytes (WBC)</td>
<td>3,4 x 10⁹ / l</td>
</tr>
<tr>
<td>Basophils</td>
<td>0 %</td>
</tr>
<tr>
<td>Eosinophils</td>
<td>0,5 %</td>
</tr>
<tr>
<td>Neutrophiles:</td>
<td></td>
</tr>
<tr>
<td>myelocytes</td>
<td>0 %</td>
</tr>
<tr>
<td>methamyelocytes</td>
<td>0 %</td>
</tr>
<tr>
<td>band forms</td>
<td>4 %</td>
</tr>
<tr>
<td>neutrophils</td>
<td>38 %</td>
</tr>
<tr>
<td>Lymphocytes</td>
<td>50,5 %</td>
</tr>
<tr>
<td>Monocytes</td>
<td>7 %</td>
</tr>
<tr>
<td>Thrombocytes (Plt)</td>
<td>120 x 10⁹ / l</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Hemogramm № 19</th>
<th>Hemogramm № 20</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hemoglobin (Hgb)</td>
<td>80 g/l</td>
</tr>
<tr>
<td>Erythrocytes (RBC)</td>
<td>2,8 x 10¹² / l</td>
</tr>
<tr>
<td>Leucocytes (WBC)</td>
<td>14 x 10⁹ / l</td>
</tr>
<tr>
<td>Basophils</td>
<td>1 %</td>
</tr>
<tr>
<td>Eosinophils</td>
<td>8 %</td>
</tr>
<tr>
<td>Neutrophiles:</td>
<td></td>
</tr>
<tr>
<td>myelocytes</td>
<td>0 %</td>
</tr>
<tr>
<td>methamyelocytes</td>
<td>6 %</td>
</tr>
<tr>
<td>band forms</td>
<td>16 %</td>
</tr>
<tr>
<td>neutrophils</td>
<td>53 %</td>
</tr>
<tr>
<td>Lymphocytes</td>
<td>10 %</td>
</tr>
<tr>
<td>Monocytes</td>
<td>6 %</td>
</tr>
<tr>
<td>Reticulocytes</td>
<td>18 %</td>
</tr>
<tr>
<td>Thrombocytes (Plt)</td>
<td>400 x 10⁹ / l</td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Hemogramm № 21</td>
</tr>
<tr>
<td>----------------</td>
<td>---------------</td>
</tr>
<tr>
<td></td>
<td>Hemoglobin (Hgb)</td>
</tr>
<tr>
<td>g/l</td>
<td></td>
</tr>
<tr>
<td>Erythrocytes (RBC)</td>
<td>4,2 x 10^{12} / l</td>
</tr>
<tr>
<td>Leucocytes (WBC)</td>
<td>12 x 10^9 / l</td>
</tr>
<tr>
<td>Basophils</td>
<td>1 %</td>
</tr>
<tr>
<td>Eosinophils</td>
<td>3 %</td>
</tr>
<tr>
<td>Neutrophiles:</td>
<td></td>
</tr>
<tr>
<td>myelocytes</td>
<td>0 %</td>
</tr>
<tr>
<td>methamyelocytes</td>
<td>4,5 %</td>
</tr>
<tr>
<td>band forms</td>
<td>12 %</td>
</tr>
<tr>
<td>neutrophils</td>
<td>48,5 %</td>
</tr>
<tr>
<td>Lymphocytes</td>
<td>20 %</td>
</tr>
<tr>
<td>Monocytes</td>
<td>11 %</td>
</tr>
<tr>
<td>Thrombocytes (Plt)</td>
<td>340 x 10^9 / l</td>
</tr>
<tr>
<td>Reticulocytes</td>
<td>1,2 %</td>
</tr>
<tr>
<td>Hemogramm № 23</td>
<td>Hemogramm № 24</td>
</tr>
<tr>
<td>---------------</td>
<td>---------------</td>
</tr>
<tr>
<td>Hemoglobin (Hgb)</td>
<td>95 g/l</td>
</tr>
<tr>
<td>Erythrocytes (RBC)</td>
<td>3,6 x 10^{12} / l</td>
</tr>
</tbody>
</table>
| Leucocytes (WBC) | 36 x 10^9 / 1 | Leucocytes (WBC) | 93 x 10^9 /
| Basophils | 2 % | Basophils | 4 % |
| Eosinophils | 7 % | Eosinophils | 9 % |
| Neutrophiles: | | Neutrophiles: | |
| myeloblastes | 3 % | myeloblastes | 1 % |
| promyelocytes | 6 % | promyelocytes | 6 % |
| myelocytes | 11 % | myelocytes | 20 % |
| methamyelocytes | 12 % | methamyelocytes | 20 % |
| band forms | 18 % | band forms | 13 % |
| neutrophils | 21 % | neutrophils | 12 % |
| Lymphocytes | 18 % | Lymphocytes | 10 % |
| Monocytes | 2 % | Monocytes | 5 % |
| Thrombocytes (Plt) | 300 x 10^9 / 1 | Thrombocytes (Plt) | 390 x 10^9 /
| Reticulocytes | 1,8 % | Reticulocytes | 0,4 % |

<table>
<thead>
<tr>
<th>Hemogramm № 25</th>
<th>Hemogramm № 26</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hemoglobin (Hgb)</td>
<td>68 g/l</td>
</tr>
<tr>
<td>Erythrocytes (RBC)</td>
<td>3,8 x 10^{12} / l</td>
</tr>
</tbody>
</table>
| Leucocytes (WBC) | 5,4 x 10^9 / 1 | Leucocytes (WBC) | 34 x 10^9 /
<p>| Basophils | 1 % | Basophils | 0 % |
| Eosinophils | 2 % | Eosinophils | 0 % |
| Neutrophiles: | | Neutrophiles: | |
| myelocytes | 0 % | myeloblastes | 91 % |
| methamyelocytes | 0 % | myelocytes | 0 % |
| band forms | 1 % | methamyelocytes | 0 % |
| neutrophils | 34 % | band forms | 0 % |
| Lymphoblastes | 5 % | neutrophils | 6 % |
| Lymphocytes | 50 % | Lymphocytes | 3 % |</p>
<table>
<thead>
<tr>
<th></th>
<th>Hemogramm № 27</th>
<th>Hemogramm № 28</th>
</tr>
</thead>
<tbody>
<tr>
<td>Leucocytes (WBC)</td>
<td>35 x $10^9$/l</td>
<td>2,4 x $10^9$/l</td>
</tr>
<tr>
<td>Hemoglobin (Hgb)</td>
<td>40 g/l</td>
<td></td>
</tr>
<tr>
<td>Erythrocytes (RBC)</td>
<td>1,2 $10^{12}$/l</td>
<td></td>
</tr>
<tr>
<td>Basophils</td>
<td>3 %</td>
<td>0 %</td>
</tr>
<tr>
<td>Eosinophils</td>
<td>11 %</td>
<td>0 %</td>
</tr>
<tr>
<td>Neutrophiles:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>myeloblastes</td>
<td>2 %</td>
<td></td>
</tr>
<tr>
<td>myelocytes</td>
<td>5 %</td>
<td>Neutrophiles:</td>
</tr>
<tr>
<td>methamyelocytes</td>
<td>6 %</td>
<td>myelocytes</td>
</tr>
<tr>
<td>band forms</td>
<td>12 %</td>
<td>methamyelocytes</td>
</tr>
<tr>
<td>neutrophils</td>
<td>35 %</td>
<td>band forms</td>
</tr>
<tr>
<td>Lymphocytes</td>
<td>20 %</td>
<td>neutrophils</td>
</tr>
<tr>
<td>Monocytes</td>
<td>6 %</td>
<td>16 %</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Monocytes</td>
</tr>
<tr>
<td></td>
<td></td>
<td>6 %</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Monoblastes</td>
</tr>
<tr>
<td></td>
<td></td>
<td>57 %</td>
</tr>
</tbody>
</table>
CONTROL QUESTIONS ON PATHOPHYSIOLOGY OF BLOOD:

2. Categorization of disorders of circulative blood volume (hypervolemia, hypovolemia).
9. Categorization of anemias by: etiopathogenesis, color parameter, severity of anemia, regenerative possibility, type of hematopoiesis, erythrocyte’s size.
10. Post-hemorrhagic anemia. Description, the picture of the blood under acute and chronic post-hemorrhagic anemia.
13. Hemolytic anemias. Types (congenital, autoimmune e.g.). The picture of the blood. Clinical symptoms.
15. Leukocytes. Stages of leucopoiesis. Leukocyte’s description on different stages of leucopoiesis.
16. Leukocyte’s functions (neutrophils, eosinophils, basophils, monocytes, lymphocytes).
18. Nuclear shift of leukocyte’s formula to the left, to the right.


21. Leukemia. The particularities of leukemic cells.

22. Characteristic of morphological picture of blood at acute and chronic myeloid and lymphoid leukemia.

23. Leukemia clinical syndromes.

24. Hemostasis, its components (blood vessel, platelets, plasma coagulation factors) and disorders of hemostasis (thrombophulic, hemorrhagic, thrombohemorrhagic hemostasiopathias).


27. Hemorrhagic disorders of primary hemostasis.

   - Hereditary hemorrhagic telangiectasia (Rendu-Osler-Weber Syndrome)
   - Scurvy
   - Henoch-Schonlein or anaphylactoid purpura.


   - Hemophilies (A, B).


34. Thromboelastogram at hypo- and hypercoagulation.
LESSON № 26

TOPIC: PATHOPHYSIOLOGY OF NERVOUS SYSTEM.

Aim of the lesson: to study the causes and mechanisms of nervous cell damaging, consequence of organ denervation and pathogenesis of central and peripheral paralysis.

QUESTIONS:

1. Etiology of nervous system disorders.
2. Neurone pathology Causes.
4. Denervation syndrome in somatic and internal organs.
5. Disturbances of locomotion. Central and peripheral paralysis. The causes.
Fig. 20. Motor nervous system
Fig. 21. Broun-Sequards syndrome.
Laboratory work 1. Modulation of central and peripheral paralysis in a frog.

Work description: After frog immobilization the spinal cord is destroyed in cervical part. By 5% serum acid the investigator shows students unconditioned reflexes of frog’s low extremeties. Then after separation of femoral nerve it should be cut and induction of unconditioned reflex is impossible. Peripheral paralysis occurs. Students perform differentiation of central and peripheral paralysis in a table:

<table>
<thead>
<tr>
<th>SIGNS</th>
<th>Central paralysis</th>
<th>Peripheral paralysis</th>
</tr>
</thead>
<tbody>
<tr>
<td>localization of lesion</td>
<td></td>
<td></td>
</tr>
<tr>
<td>autokinesia</td>
<td></td>
<td></td>
</tr>
<tr>
<td>unconditioning reflexes</td>
<td></td>
<td></td>
</tr>
<tr>
<td>pathological reflexes</td>
<td></td>
<td></td>
</tr>
<tr>
<td>muscular tone</td>
<td></td>
<td></td>
</tr>
<tr>
<td>distrophia</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Situational tasks:

1. Transection of dorsal white column in of the spinal cord in lumbar area in a rat has caused back extremities deafferentation. Whether the muscle tone of the extremities will change? Whether proper muscle reflexes and autokinesias of extremities will be damaged?

2. A lethal dose of tetanic toxine was injected to an animal. What movement disorder will be observed in this case?

3. The sciatic nerve of the left back extremity in the middle third of the hip was cut in a dog. Ten days after operation the wound has completely healed with primary intenion. Achilles reflex is not provoked, reaction to pricks is absent. What paralysis has developed in the dog? Is restoration of movement function of an extremity after denervation is possible?

4. In experimental rats extremity paralysis was induced by injection of curare toxine into the gastrocnemius muscle. But the conduction of impulses through the nerve fibers retained as well as extremity muscles ability to respond to direct irritation. Explain, what mechanism of paralysis development is observed in this case. Which paralysis has developed?

5. In a dog the right half of spinal cord was transected at the level of the 2nd - 3rd chest segments. Extremities of which side will be paralyzed? What sensitivity disorders have developed in this case?

6. In an experimental animal the cerebral cortex was destroyed in the area of posterior central gyrus on the right. How sensitivity in the experimental animal will be damaged?
7. 
Patient T, 32 years, complains of weakness in the right hand, difficulties in using it. Considers himself/herself a patient for 6 years when for the first time felt awkwardness of the hand, especially when writing. In the neurologic status: active movements of the right hand are limited, moderate atrophy of shoulder girdle and hand muscles. Strength of the hand is reduced, muscle tone is increased. Tendon and periostal reflexes of the right upper extremity are strong, their areas are extended. Sensitivity is not disturbed. What form motion activity disorder has the patient? What is approximate localisation of pathological process and possible mechanism of motion function disorder in this case? What caused the development of muscle atrophy of the right hand?

8. 
Patient L, 22 years, has arrived in clinic two years after the backbone and spinal cord trauma. Recently the patient is troubled by leg muscles spastisity, so he can lie only on the back. Moderate muscles hypotrophy. Babinsky bilateral symptom. Which paralysis has developed in the patient and what is its possible mechanism? What are the mechanisms of muscles spasticity in this case?

Tests:
1. What is the consequence of corticospinal path integrity infringement?
   a) hyperkinesia
   b) central paralysis
   c) peripheric paralysis
   d) ataxia

2. What is the consequence of damage of peripheral motoneurons?
   a) hyperkinesia
   b) central paralysis
   c) peripheric paralysis
   d) ataxia

3. What signs are characteristic of the central paralysis?
   a) Intensifying of reflex movements
4. What signs are characteristic of a peripheral paralysis?
   a) Absence of autokinesias
   b) Absence of reflex movements
   c) Intensifying of reflex movements
   d) Intensifying of autokinesias

5. Central paralysis is characterized by:
   a) Muscle tone increase
   b) Muscle tone decrease

6. Peripheral paralysis is characterized by:
   a) Muscle tone increase
   b) Muscle tone decrease

7. What sensitivity is changed due to damage of Gaulle and Burdach’s fascicles of the spinal cord?
   a) Absence of the proprioceptive sensitivities on the damaged side
   b) Absence of the proprioceptive sensitivities on the opposite side
   c) Absence of temperature and pain sensitivity on the damaged side
   d) Absence of temperature and pain sensitivity on the opposite side

8. What sensitivity is changed due to damage of lateral spinothalamic tract of the spinal cord?
   a) Absence of the proprioceptive sensitivities on the damaged side
   b) Absence of the proprioceptive sensitivities on the opposite side
   c) Absence of temperature sensitivity on the damaged side
   d) Absence of temperature sensitivity on the opposite side

9. What sensitivity is changed due to damage of posterior horns of the spinal cord?
   a) Absence of the proprioceptive sensitivities on the damaged side
   b) Absence of the proprioceptive sensitivities on the opposite side
   c) Absence of temperature sensitivity on the damaged side
   d) Absence of pain sensitivity on the damaged side
   e) Absence of temperature sensitivity on the opposite side
10. What change of motor function occurs due to damage of Gaulle and Burdach’s fascicles of the spinal cord?
   a) central paralysis
   b) Peripheric paralysis
   c) Hyperkinesia
   d) Sensitive ataxy

11. The cause of coordination locomotion infringement is the damage of:
   a) Cerebellum
   b) anterior horns of the spinal cord
   c) posterior horns of the spinal cord
   d) Lateral horns of a spinal cord

12. What is the reason of Broun-Sekar syndrome?
   a) full transection of the spinal cord
   b) half longitudinal transaction of the spinal cord
   c) half cross transaction of the spinal cord

13. The cause of spasms in tetanus is infringement of:
   a) mediator synthesis
   b) axon mediator transport
   c) mediator secretions in a synaptic gap
   d) interactions of a mediator with a receptor

14. The cause of spasms in strychnine poisoning is infringement of:
   a) mediator synthesis
   b) axon mediator transport
   c) mediator secretions in a synaptic gap
   d) interactions of a mediator with a receptor

15. Lack of which mediator leads to a convulsive syndrome?
   a) opioid peptides
   b) dofamin
   c) GABA
   d) neurotensin

16. Botulism toxine blocks transport of ... in a synaptic gap:
17. What is characteristic of a critical pain?

a) distinct localisation
b) indistinct localisation
c) it is brief
d) it is prolonged
e) it is weak

Answers:
1b, 2c, 3ab, 4ab, 5a, 6b, 7a, 8d, 9cd, 10d, 11ac, 12c, 13c, 14d, 15c, 16c, 17ace.

LITERATURE:
1. Lecture material.
LESSON № 27

TOPIC: PATHOPHYSIOLOGY OF HIGHER NERVOUS ACTIVITY

Aim of the lesson: to study the causes and mechanisms of higher nervous activity, and pathogenesis of neurosis, narcomania and alcoholism.

QUESTIONS:

1. Classification of types of higher nervous activity.
2. A role of the generator of pathologically enhanced excitation, pathological system in excitatory system pathology.
3. Common etiology of higher nervous activity disorders.
9. Infringements of intelligence, memory, emotions, behaviour, etc.
10. Alzheimer's disease.
12. Characteristic of manifestations and causes of sleep wakefulness cycle disorders.
Fig. 23. Synthesis of catecholamines

Tests:

1. **Following substances are concerned to narcotics:**
   a) Nicotine  
   b) Cannabis drugs  
   c) Cocaine  
   d) dichlorvos  
   e) Hallucinogens

2. **Choose true statements:**
   a) Alcohol is not produced by human organism  
   b) Alcohol is endogenous metabolite  
   c) Nicotine is not formed in human organism  
   d) Nicotine is endogenous metabolite

3. **The euphoric effect of alcohol on CNS is connected, first of all, with:**
4. Specify changes in cell membranes in acute alcohol intoxication:
   a) increased fluidity of lipidic bilayer
   b) decreased fluidity of lipidic bilayer
   c) increased cholesterol level

5. Specify metabolism changes in acute alcohol intoxication:
   a) Oppression of oxidative phosphorylation
   b) Decrease in synthesis of protein
   c) Increase in synthesis of protein

6. Death in acute alcoholic poisoning can come from:
   a) emetic masses aspiration
   b) Respiratory centre oppressions
   c) Fibrillations of heart ventricles
   d) hypoglycemic comas

7. Defence reaction in chronic alcohol abuse is:
   a) Increase of cell permeability to alcohol
   b) Decrease in cell permeability to alcohol
   c) Increase of suction of ethanol in a gastrointestinal path
   d) Weakening of oxidation of ethanol and acetic aldehyde

8. Alcoholism develops faster:
   a) At young age
   b) At mature age
   c) At all age-grades equally
   d) In people with hereditary predisposition

9. Ferment participating in oxidation of ethanol is:
   a) insulinase
   b) alcohol dehydrogenase
   c) Peptase
   d) Lactase

10. Specify stages of alcoholism (narcotisms):
11. What stage of alcoholism is characterised by the maximum tolerance to ethanol?
   a) neurotic stage
   b) drug addiction
   c) encephalopathy

12. What stage of alcoholism is characterised by organ and system decompensation?
   a) neurotic stage
   b) drug addiction
   c) encephalopathy

13. In alcoholism abstinence stage next symptoms take place:
   a) neuromediator exchange infringements
   b) tissue hypoxia deveopment
   c) alcohol dehydrogenase activation
   d) organism functions normalisation

14. The neurotic stage of alcoholism is characterised by:
   a) increase of tolerance to alcohol
   b) maximal tolerance to alcohol
   c) low tolerance to alcohol

15. The drug addiction stage of alcoholism is characterised by:
   a) maximal tolerance to alcohol
   b) Low tolerance to alcohol
   c) abstinence syndrome presence

16. The encephalopathy stage of alcoholism is characterised by:
   a) maximal tolerance to alcohol
   b) Low tolerance to alcohol
   c) polyorganic insufficiency

17. Specify manifestations of experimental neuroses:
a) phase states development
b) Simplification of development of conditioned reflexes
c) vegetative functions distress

18. Manifestations of experimental neuroses are:
a) Simplification of development of conditioned reflexes
b) Impossibility of development of new conditioned reflexes
c) hypo- and hyperkinesias, sensitivity distresses

19. How is paradoxical phase manifested?
a) Strong reaction to a weak conditioned stimulus and vice versa
b) Identical responses of nervous structure to influences of different intensity
c) Development of negative reactions in response to positive stimuli, and vice versa

20. The ultraparadoxal phase state is characterized by:
a) Strong reaction to weak conditioned stimulus and vice versa
d) Development of negative reactions in response to positive stimuli, and vice versa

21. The equalizing phase is characterized by:
a) Identical responses of nervous structure to influences of different intensity
b) Development of negative reactions in response to positive stimuli, and vice versa

22. The obstacle phase is characterised by:
a) Absence of reactions to weak and strong stimuli
b) Development of negative reactions in response to positive stimuli, and vice versa

23. The overstrain of excitatory process can be provoked by:
a) Prolonged action of strong conditioned stimuli
b) Necessity of development of subtle and complex differentiations

24. The overstrain of excitatory process can be provoked by:
a) A considerable quantity of conditioned excitators
b) Alteration of alarm value of conditioned excitators

25. *The overstrain of inhibitory process can be provoked by:*
   a) prolonged action of strong conditioned excitators
   b) Great number of conditioned excitators
   c) Necessity of development of subtle and complex differentiations
   d) Alteration of signal value of conditioned stimuli

26. *The overstrain of mobility of the excitatory processes can be provoked by:*
   a) prolonged action of strong conditioned stimuli
   b) A considerable quantity of conditioned stimuli
   c) Necessity of development of subtle and complex differentiations
   d) Alteration of signal value of conditioned stimuli

27. *Which neurosis can be modeled by overstrain of inhibitory process:*
   a) neurosis with prevalence of excitation
   b) neurosis with prevalence of inhibition
   c) neurosis with pathological mobility of the excitatory processes

28. *An overstrain of exciting process it is possible to model a neurosis:*
   a) With prevalence of inhibition
   b) With pathological mobility of the excitatory processes
   c) With prevalence of excitation

29. *Neurosis with prevalence of excitation process in animals is manifested by:*
   a) Inadequate excitement
   b) Aggressiveness and malignancy
   c) Drowsiness
   d) slow response

30. *Neurosis with prevalence of inhibition process in animals is manifested by:*
   a) Inadequate excitement
   b) Aggressiveness and malignancy
   c) Drowsiness
   d) Fussiness
31. **Neurosis with pathological mobility in animals is manifested by:**
   a) Inadequate excitement  
   b) Aggressiveness and malignancy  
   c) Drowsiness  
   d) Fussiness

32. **Following states concern to neuroses:**
   a) Neurasthenia  
   b) Schizophrenia  
   c) Hysteria  
   d) maniacal-depressive psychosis  
   e) obsessive neurosis

33. **What temperament is most often subject to neurotic distresses:**
   a) a melancholic person  
   b) a phlegmatic person  
   c) a choleric person  
   d) a sanguine person

34. **An information triad as the condition of neuroses occurrence includes:**
   a) Limitation of time  
   b) High level of motivations  
   c) Low level of motivations  
   d) Personal features  
   e) Great volume of the information which is necessary to adopt

35. **Characteristic features of neurasthenia are:**
   a) Hypererethism and irritability in combination with fast fatigability  
   b) Phobias  
   c) reduced working capacity, flaccidity  
   d) increased suggestibility

36. **Characteristic features of obsessive neurosis:**
   a) Hypererethism and irritability in combination with fast fatigability  
   b) Phobias  
   c) Delirium, hallucinations  
   d) increased suggestibility
37. Characteristic features of hysteria:
   a) Hypererethism and irritability in combination with fast fatigability
   b) Phobias
   c) Deafness, dumbness
   d) Paresis, paralyses

38. The amnesia can be observed in:
   a) Neuroses
   b) Maniacal-depressive syndrome
   c) Korsakov syndrome
   d) Alzheimer's diseases

39. Oligophrenia is a syndrome in:
   a) Diabetes
   b) Down’s syndrome
   c) Phenylketonuria
   d) Cerebral circulation infringement

40. Depression can arise in following brain disorders:
   a) Endorphins level increase
   b) Endorphins level decrease
   c) Serotonin level decrease
   d) Serotonin level increase

   Answers:
   1bce, 2bc, 3b, 4a, 5ab, 6abc, 7b, 8ad, 9b, 10ab, 11b, 12c, 13ab, 14a, 15ac, 16bc, 17ac, 18bc, 19a, 20b, 21a, 22a, 23a, 24a, 25c, 26d, 27a, 28a, 29ab, 30c, 31d, 32ace, 33a, 34abe, 35ac, 36b, 37cd, 38cd, 39bc, 40bc.

LITERATURE:
1. Lecture material.
LESSON № 28

TOPIC: PATHOPHYSIOLOGY OF ENDOCRINE SYSTEM.
PITUITARY GLAND AND ADRENAL GLAND FUNCTIONS 
DISORDERS

Aim of the lesson: to study the causes and mechanisms of hyper- and hypofunction of pituitary and adrenal glands.

QUESTIONS:

3. Classification of endocrinopathias. Concept about central (secondary and tertiary) and peripheric (primary) endocrinopathias.
11. Phaeochromocytoma.
Fig. 24. Target cells for pituitary hormones

Fig. 25. Sures of the releasing factors

Tasks:

1.
Patient A., 26 years, complained of general weakness, headaches, change of appearance, extension of hands and feet. For two years the size of footwear has increased from 39 to 42. Objectively: integration of features (massive superciliary and zygomas, big nose, lips, ears) is marked. The thorax is of the barrel-shaped form, thickened clavicles. Hands and feet are increased. Internal organs are not essentially changed. Pulse – 78 bpm, the AP – 150/90 mm Hg.

- What endocrine pathology can be suspected?

**Tests:**

1. **What hormones formation infringement takes place in pituitary gigantism?**
   a) gonadotropin
   b) somatotropinum
   c) prolactin
   d) melanocyte-stimulating hormone

2. **What infringements are characteristic of pituitary nanism?**
   a) augmentation of protein synthesis
   b) reduction of protein synthesis
   c) tissue differentiation infringement
   d) tissue differentiation is not variated

3. **What hormones formation infringement in Simmonds disease takes place?**
   a) gonadotropin
   b) somatotropin
   c) thyrotropin
   d) anterior pituitary hormones

4. **What hormones formation infringement in Shihen disease takes place?**
   a) gonadotropin
   b) somatotropinum
   c) corticotropin (ACTH)
d) anterior pituitary hormones

5. What metabolic infringements are characteristic of Somatotropin hyperproduction?
   a) augmentation of synthesis of protein
   b) lipolysis augmentation
   c) reduction of synthesis of protein
   d) lipogenesis augmentation

6. What hormones formation infringement in Itsenko-Cushing disease occurrence probably takes place?
   a) somatoliberin
   b) corticoliberin
   c) somatostatin
   d) thyroliberin

7. To what hormone formation decrease acromegaly is related?
   a) somatoliberin
   b) corticoliberin
   c) somatostatin
   d) thyroliberin

8. What change of protein metabolism characterises Itsenko-Cushing disease?
   a) augmentation of synthesis of protein
   b) augmentation of a katabolism of protein

9. What change of fat metabolism characterises Itsenko-Cushing disease?
   a) lipolysis augmentation
   b) lipogenesis augmentation

10. What changes of metabolism characterises Itsenko-Cushing disease?
    a) glyconeogenesis intensifying
    b) augmentation of fat synthesis
    c) glyconeogenesis reduction
    d) protein synthesis augmentation
    e) protein katabolism augmentation
11. What change of blood arterial pressure is characteristic of Itsenko-Cushing disease?
   a) it increases
   b) it decreases
   c) it is normal

12. What state of immune system is characteristic of Itsenko-Cushing disease?
   a) occurrence autoimmune processes
   b) activity decrease
   c) it is not broken

13. What mechanism takes place in immune system activity decrease in Itsenko-Cushing disease?
   a) intensifying of protein katabolism
   b) increase of intensity of protein synthesis
   c) lipogenesis augmentation
   d) lipolysis intensification

14. What endocrine function changes lead to diabetes insipidus?
   a) vasopressinum increase
   b) vasopressinum reduction
   c) electrocortin increase
   d) insulin lack

15. What changes are characteristic of diabetes insipidus?
   a) polyuria
   b) polydipsia
   c) dehydration
   d) oliguria
   e) hyperhydration

16. What changes are characteristic of vasopressinum surplus?
   a) polyuria
   b) polydipsia
   c) dehydration
   d) oliguria
   e) hyperhydration
17. What symptoms are characteristic of Itsenko-Cushing disease?
   a) the round-shaped face
   b) attrition
   c) low blood pressure
   d) high blood pressure

18. What change is observed in diabetes insipidus?
   a) increased water reabsorption
   b) reduction of water reabsorption
   c) glomerular filtration increase
   d) glomerular filtration reduction

19. What mechanism participates in diuresis change in Parhon syndrome (surplus of vasopressinum)?
   a) increased water reabsorption
   b) reduction of water reabsorption
   c) glomerular filtration increase
   d) glomerular filtration reduction

20. What changes of electrolytic exchange in kidney are characteristic of adrenal gland failure?
   a) increased sodium reabsorption
   b) reduction of sodium reabsorption
   c) increased secretion of potassium
   d) reduction of secretion of potassium

21. What endocrine function causes steroid diabetes?
   a) increase of mineralocorticoids
   b) reduction of glucocorticoids
   c) surplus of glucocorticoids

22. What changes in nephrons are observed in primary hyperaldosteronism?
   a) augmentation of sodium reabsorption
   b) augmentation of potassium secretion
   c) reduction of sodium reabsorption
   d) reduction of potassium secretion

23. What changes in nephrons are characteristic of hypoaldosteronism?
a) augmentation of sodium reabsorption  
b) augmentation of potassium secretion  
c) reduction of sodium reabsorption  
d) reduction of potassium secretion

24. **What changes of cell electrolite composition are characteristic of primary hyperaldosteronism?**  
a) augmentation of potassium maintenance  
b) reduction of sodium maintenance, water  
c) reduction of potassium maintenance  
d) augmentation of sodium maintenance, water

25. **What changes of blood electrolite composition are characteristic of hypoaldosteronism?**  
a) augmentation of potassium maintenance  
b) reduction of sodium maintenance  
c) reduction of potassium maintenance  
d) augmentation of sodium maintenance

26. **How does arterial pressure variate in hypoaldosteronism?**  
a) increases  
b) decreases  
c) does not variate essentially

27. **How does the arterial blood pressure variate in total hypoactivity of adrenal cortex?**  
a) increases  
b) decreases  
d) does not variate essentially

28. **What changes of carbohydrate metabolism are characteristic of total hypoactivity of adrenal cortex?**  
a) glyconeogenesis intensity augmentation  
b) glyconeogenesis intensity decrease  
c) maintenance augmentation of blood glucose  
d) maintenance decrease of blood glucose

29. **What hormones formation infringement in secondary hypergonadism takes place?**
30. **What hormones formation infringement in persistent lactorrhea syndrom takes place?**
   a) gonadotropin  
   b) somatotropin  
   c) chromatophorotropin hormone  
   d) corticotropin (ACTH)

31. **What hormones formation infringement in skin pigmentation augmentation takes place?**
   a) gonadotropin  
   b) somatotropin  
   c) prolactin  
   d) chromatophoroptropic hormone

32. **Synthesis of which biologically active substances cause pituatory nanism?**
   a) gonadotropin  
   b) somatotropin  
   c) corticotropin (ACTH)  
   d) thyrotropin  
   e) somatomedins

33. **What hormones formation infringement in adrenal cortex secondary hypoactivity takes place?**
   a) gonadotropin  
   b) somatotropin  
   c) corticotropin (ACTH)  
   d) thyrotropin

34. **What hormones formation infringement in adrenal cortex secondary hypoactivity development probably takes place?**
   a) somatoliberin  
   b) corticoliberin  
   c) somatostatin
d) thyroliberin

35. A congenital adrenogenital syndrome is characterized by:
   a) hydrocortisone augmentation
   b) adrenocorticotropic hormone (ACTH) reduction
   c) androgenic hormones augmentation
   d) hydrocortisone reduction

Answers:

1b, 2bc, 3d, 4d, 5ab, 6b, 7c, 8b, 9b, 10abe, 11a, 12b, 13a, 14b, 15abc, 16de, 17ad, 18b, 19a, 20bd, 21c, 22ab, 23cd, 24cd, 25ab, 26b, 27b, 28bd, 29a, 30c, 31d, 32be, 33c, 34b, 35cd

Literature:
1. Lecture material.
LESSON № 29

**TOPIC:** PATHOPHYSIOLOGY OF ENDOCRINE SYSTEM. PATOLOGY OF THYROID, PARATHYROID AND SEX GLANDS.

**Aim of the lesson:** to study the causes and mechanisms of hyper- and hypofunction of thyroid, parathyroid and sexual glands.

**QUESTIONS:**

1. Thyroid gland pathology. Classification of infringements (central and peripheral).
4. The causes of occurrence and the basic infringements in hyper- and Hyperparathyroidism.

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**Fig. 26. The Pathogenesis of Graves Disease**
Fig. 27. Action of parathyroid hormone on calcium/phosphate homeostasis
Fig. 28. Hyperparathyroidism

**Tasks:**

1. An experimental dog from the moment of a birth got water, free of iodine. A year and a half later the thyroid gland mass of the dog has reached 100 g while the gland mass of the control dog receiving usual water was 1 g.
   - How is the increase of the experimental dog’s thyroid gland explained?
   - What is the pathology in which the thyroid gland increase occurs due to iodine insufficiency?

2. In an experimental dog the parathyroid glands have been removed.
   - How will the calcium level in blood variate?
   - How is the pathologic state, caused by this calcium level in blood called? How is the syndrome called?
3.

Patient A, 16 years, complained of the thyroid gland enlargement. Other complains are absent. For the first time she noticed the gland enlargement 4 years ago, a year later after moving to the present district. Examination detected endemial struma.

What is struma? Name the basic mechanisms causing development of struma? Which of them takes place in this case?

4.

In a dog both adrenal glands have been removed. The day after adrenalectomy pathological changes (slackness, muscular delicacy, anorexia, vomiting, anuria) have developed. Three days after the operation the animal died. Why did the dog die?

Tests:

1. **Insufficiency of iodine in nutrition causes:**
   a) autoimmune thyroidithis
   b) hyperthyrosis
   c) hypoparathyroidism
   d) endemial struma
   e) diffuse toxic struma

2. **In severe hypothyroidism in adults leads to:**
   a) cretinism
   b) myxedema
   c) eunuchoidism
   d) nanism
   e) hypergonadism

3. **Surplus of hormones of the thyroid gland occurs in:**
   a) myxedema
   b) diffuse toxic struma
   c) endemial cretinism
   d) acromegaly
4. Surplus of which hormones is characteristic of hyperthyroidism:
   a) triiodothyronine and thyroxine
   b) prolactin
   c) hydrocortisone
   d) insulin

5. The thyrotropic hormone blood concentration augmentation in hypothyroidism testifies localisation of pathological process in:
   a) pituitary body
   b) thyroid gland
   c) parathyroid gland
   d) hypothalamus
   e) thymus

6. Hypothyroidism is characterised by:
   a) metabolism augmentation
   b) lipolysis increase
   c) metabolism decrease
   d) lipolysis decrease

   7 Hyperthyroidism is characterised by:
   a) metabolism augmentation
   b) protein katabolism intensifying
   c) lipolysis increase
   d) metabolism decrease

8. What is typical for hypothyroidism:
   a) increased irritability
   b) thinking sluggishness
   c) drowsiness
   d) decrease of intellectual and physical efficiency
   e) tremor

9. What is typical for hyperthyroidism:
   a) increased irritability
   b) drowsiness
c) tremor of dactyls of hands
 d) memory weakening

10. How does phosphorus reabsorption in nephrons tubules variate in parathyroid glands hypoactivity?
   a) increases
   b) decreases

11. How does phosphorus reabsorption in nephrons tubules variate in parathyroid glands hyperfunction?
   a) decreases
   b) increases

12. How does calcium and phosphorus maintenance in blood variates in hypoparathyroidism?
   a) calcium maintenance increases
   b) phosphorus maintenance increases
   c) calcium maintenance decreases
   d) phosphorus maintenance decreases

13. What hormones formation infringement in thyroid gland hypoactivity takes place?
   a) gonadotropin
   b) somatotropinum
   c) corticotropin (ACTH)
   d) thyrotropin

   **Answers:**
   1d, 2b, 3b, 4a, 5b, 6d, 7cd, 8abc, 9bcd, 10ac, 11a, 12a, 13bc, 14d

   **LITERATURE:**
   1. Lecture material.
LESSON № 30

**TOPIC:** INSUFFICIENCY OF BLOOD CIRCULATION. MYOCARDIAL INFARCTION.

**Aim of the lesson:** to study the basic mechanisms of systemic blood flow disturbances, heart failure and compensative and decompensative reactions during myocardial infarction.

**QUESTIONS:**

1. Insufficiency of blood circulation. General characteristic.
2. Classification of blood circulation pathology. Kinds of blood circulation insufficiency (acute and chronic, left - and right heart).
3. Etiology, pathogenesis and main symptoms of acute heart failure.
5. The basic haemodynamic indexes characterising acute and chronic insufficiency of blood circulation.
6. Mechanisms of compensation during chronic heart failure (the characteristic of cardiac and noncardiac mechanisms). Concept about heterometrical (isotonic) and homeometrical (isometric) indemnification mechanisms. Decompensation manifestations.
9. The causes of noncoronarogenic heart damages.
12. Clinical symptoms of blood circulation insufficiency and substantiation of their development mechanisms.
13. The characteristic of changes of heart hemodynamics in insufficiency of mitral valves and foramens stenoses.
Assessment tests of the functional states of cardiovascular system

**Martin-Kushelevskiy test.** A patient sits down near a table edge to the left of the doctor. On his left shoulder we fix a tonometer cuff. In a state of rest we count the frequency of heart contractions (is detected by 10 second intervals – heart rate) and measure the arterial pressure. Then a patient, without removing the cuff from the shoulder (the tonometer is disconnected), rises and carries out 20 deep squatting for 30 seconds.

At each squatting it is necessary to put both hands forward. After exercise performance the patient sits down, the doctor switches a stop watch on «0» and starts the heart rate and arterial pressure examination every 3 minutes of the regenerative period. In the first 10 seconds the doctor detects the heart rate, between 11\textsuperscript{th} and 49\textsuperscript{th} second – arterial pressure.

In quality assessment of a dynamic functional test various deviations from a normotonic reaction type are atypical. They include: asthenic, hypertensive, dystonic and reaction with step increase of arterial pressure.
Normotonic type of reaction of cardiovascular system on the exercise stress is characterised by pulse increase to 30-50 %, increase of the maximum arterial pressure to 10-35 mm Hg, decrease of the minimal arterial pressure to 4-10 mm Hg. The recovery period is 2-3 minutes.

Hypotonic (asthenic) type of reactions. It is characterised by significant, inadequate to physical activity pulse increase. Systolic arterial pressure increases a little or remains invariable. Diastolic arterial pressure increases or does not variate. Therefore, pulse pressure decreases. Thus, the increase of the minute volume of blood circulation occurs mainly due to heart rate increase and arterial pressure normalizes slowly (to 5-10 minutes). Hypotonic type of reaction is observed in children after diseases, in insufficient activity, in vegeto-vascular dystonia, hypoevolutive heart form.

Hypertensive type of reaction is characterised by significant heart rate increase, sharp increase of maximal (to 180-200 mm hg) and moderate increase of minimal arterial pressure. The regenerative period considerably elongates. Occurs in primary and symptomatic hypertensia, overtraining, exercise stress.

Dystonic type of reaction is characterised by increase of the maximum arterial pressure to 160-180 mm Hg, significant growth of heart rate (more, than on 50 %). The minimum arterial pressure considerably reduces and the phenomenon of “infinite tone» is quite often defined. The regenerative period elongates. Instability of vascular tone is observed as well as vegetative neurosises and overfatigue.

Reaction with step increase of maximal arterial pressure is characterised by the fact that maximal arterial pressure is lower after physical activity, than on the 2nd or 5th minute of rehabilitation. The expressed increase of heart rate is simultaneously observed. This reaction reflects blood circulation regulatory mechanism inferiority and is observed after infectious diseases, in fatigue, hypokinesia, insufficient physical activity.

Novaks test. Bicycle ergometer is necessary for test performing. The idea of the test consists in time during which a patient is capable to bear the increasing physical activity. The initial load is 1 Vt/kg, every 2 minute
a load increases by 1 Vt/kg until the patient refuses to perform work. At the moment of refuse the oxygen consumption is close or equal to minute volume of bloodstream, heart rate also reaches the maximal values. Test is suitable for research of both trained and not trained persons. It can be used in therapeutic physical training in the course of rehabilitation of patients. In the latter case the initial load should be 1/4 Vt/kg.

The results of Novak's test are assessed using the table:

<table>
<thead>
<tr>
<th>Power loads (Vt/kg)</th>
<th>Operating time at each stage (minutes)</th>
<th>Assessment of results testings</th>
</tr>
</thead>
<tbody>
<tr>
<td>2</td>
<td>1-2</td>
<td>Low working capacity in the untrained A</td>
</tr>
<tr>
<td>3</td>
<td>1</td>
<td>Satisfactory working capacity in the untrained B</td>
</tr>
<tr>
<td>3</td>
<td>2</td>
<td>Normal working capacity in the untrained C</td>
</tr>
<tr>
<td>4</td>
<td>1</td>
<td>Satisfactory working capacity in the trained D</td>
</tr>
<tr>
<td>4</td>
<td>2</td>
<td>Good working capacity in sportsmen E</td>
</tr>
<tr>
<td>5</td>
<td>1-2</td>
<td>High working capacity in sportsmen</td>
</tr>
<tr>
<td>6</td>
<td>1</td>
<td>Very high working capacity in sportsmen</td>
</tr>
</tbody>
</table>

Tasks:

*Guinea pigs with experimental atherosclerosis have subjected to a long exercise stress – run in running track that has caused development of a heart attack of the myocardium. Describe the mechanism of the cardiac muscle lesion?*

Tests:

1) *What pathology refers to coronarogenous myocardial blood circulation insufficiency?*

a) ischemic heart disease  
b) the valve insufficiency  
c) valvular stenosis
2) What pathology leads to noncoronary myocardial blood circulation insufficiency?
   a) ischemic heart disease
   b) the valve insufficiency
   c) valvular stenosis
   d) hypercalcemia

3) What pathology leads to myocardial blood circulation insufficiency?
   a) myocarditis
   b) the valve insufficiency
   c) valvular stenosis
   d) hearts tamponade

4) What factor is the reason of cardiac insufficiency due to its overload by blood pressure?
   a) ischemic heart disease
   b) myocarditis
   c) mitral valve insufficiency
   d) aortic stenosis

5) What factor is the cause of heart insufficiency as a result of its volume overload?
   a) ischemic heart disease
   b) myocarditis
   c) aortal valve insufficiency
   d) aortic stenosis

6) What factor leads to heart insufficiency as a result of its volume overload?
   a) ischemic heart trouble
   b) myocarditis
   c) valving foramen stenosis
   d) tricuspid valve insufficiency

7) What factor leads to heart pressure overload?
   a) ischemic heart trouble
   b) myocarditis
   c) mitral foramen stenosis
d) tricuspid insufficiency

8. What changes of hemodynamics occur in heart insufficiency?
   a) reduction of systolic outcome
   b) reduction of minute volume of blood
   c) reduction of residual volume of blood

9. What change of hemodynamics verifies heart insufficiency?
   a) augmentation of residual volume of blood
   b) augmentation of minute volume of blood
   c) augmentation of systolic outcome

10. In what kind of cardial insufficiency Frank-Starling mechanism plays the important role?
    a) in volume overload
    b) in pressure overload

11. What is characteristic of decompensated heart insufficiency induced by volume overload?
    a) increase of diastolic filling
    b) reduction of systolic outcome
    c) increase of isometric strain of myocardium

12. The heart insufficiency with pressure overload is characterized by:
    a) increase of diastolic filling
    b) increase of isometric strain of myocardium
    c) fast decompensation

13. The heart hypertrophy is characterized by:
    a) the conductive system of heart lags from increase of myocardium mass
    b) the conductive system of heart advances from increase of myocardium mass
    c) growth of vessels is behind the increase of cardiomyocytes mass
    d) growth of vessels leaves behind the increase cardiomyocytes mass

14. The heart hypertrophy in stage 2 is characterized by:
    a) the increase of myofibrill mass leaves behind the mitochondrion mass increase
b) the increase mitochondrium mass leaves behind the myofibril mass increase

c) the myofibril mass and mitochondrium mass increase equally

15. The heterometrical mechanism of the compensation arises in heart overload by:
   a) volume
   b) resistance (pressure)

16. The gomeometrical mechanism of the compensation arises in heart overload by:
   a) volume
   b) resistance (pressure)

17. In the first (emergency) stage compensatory hyperfunction of heart the synthesis of nucleic acids and proteins:
   a) increases
   b) decreases
   c) it is close to normal

18. What form of heart insufficiency causes blood congestion in the lungs?
    a) left ventricle insufficiency
    b) right ventricle insufficiency

19. Systemic circulation congestion is observed in insufficiency of:
    a) left ventricle
    b) right ventricle

20. Intracardiac compensation mechanisms in acute heart insufficiency are:
    a) tachycardia
    b) bradycardia
    c) decrease of vascular tone
    d) increase of vascular tone
    e) blood volume increase

21. Extracardiac compensation mechanisms in acute heart insufficiency are:
    a) tachycardia
22. A major factor in hypostasis development in heart insufficiency is:
   a) oncotic pressure decrease
   b) vascular permeability increase
   c) increase of osmotic pressure in kidneys
   d) hydrostatic pressure increase in veins

   **Answers:**
   1a, 2d, 3a, 4d, 5c, 6d, 7c, 8ab, 9a, 10a, 11ab, 12bc, 13ac, 14a, 15a, 16b, 17a, 18a, 19b, 20a, 21de, 22d.

   **Themes of abstracts:**
   - Pathogeny of myocardium heart attack.
   - Mechanisms of cardiogenic shock.

   **LITERATURE:**
   1. Lecture material.
LESSON № 31

TOPIC: PATHOLOGY OF HEART RATE. ARTERIAL HYPERTENSION

Aim of the lesson: to study the types and mechanisms of arrhythmias, arterial hypertension and their consequences.

QUESTIONS:
5. Factors involved in regulation of blood pressure. Severity levels of arterial hypertensia. Stages.
Fig. 30. Conduction system of the heart.

Fig. 31. Electrochemical generator in a volume conductor
**Fig. 32. Electrocardiogramm**

**Laboratory Work 1. Calcium chloride model of arrhythmia.**

Course of the work: the rat at thiopentone anaesthetic (0,1 ml of solution of 7% on 100 g of mass) is fixed to the operational plate on the back. By a skin electrodes - needles of the electrocardiograph are introduced: red – a forward right clamp, yellow – a forward left clamp, green – a back left clamp, black – a back right clamp.

After device calibration (1 mV) we manufacture record of an initial electrocardiogram in the 2nd abduction at speed moving tapes of 25 mm/seconds. After skin preparation we expose the femoral vein.

**Fig. 33. R-R interval for calculation of heart rate.**

Solution of calcium chloride is slowly introduced into the vein at a dose of 0,2 ml/100g of the mass of an animal 5%. We record of an electrocardiogram within 10-15 minutes with intervals. We study character of disorders in electrocardiogram, we analyze them and we define kinds of
rhythm infringements. We draw changes of an electrocardiogram in reports.

Fig. 34. Atrial Ectopic Beats

Fig. 35. Ventricular Tachycardia

Fig. 36. Full A-V-block

We make the conclusion.

Practical work:

WORK 1. Ortostatic test.
It is used to examine functional state of vegetative excitatory system, its sympathetic department. A patient lies for 5 minutes after which his/her pulse is taken by 10-seconds intervals, the arterial pressure is measured too. Then the patient stands up and his/her pulse is taken for 10 seconds, the arterial pressure is measured. In normal excitability of sympathetic department the heart rate increases by 20-25 % from the initial level. Higher figures indicate (unfavorable) hyperexcitability of sympathetic department of the vegetative nervous system. The AD normally isn’t
significantly changed when standing up, in comparison horizontal position. Systolic pressure is ±10 mm hg, diastolic ±5 mm hg

WORK 2. Klinostatic test.
It is used to examine parasympathetic department of vegetative nervous system. After 5 minutes of adaptation in standing position the AD and pulse are measured, then a patient lies down. Pulse and the AD are measured again. Normally bradycardia during position changing is no more than 6-12 bpm while more significant bradycardia indicates the prevalence of parasympathetic influences. Systolic AP is ±10 mm hg pressure, diastolic is ±5 mm hg.

Tasks:

1. Hypertensia model was reproduced in a rabbit. Silver rings were imposed on renal arteries which caused their constriction and renal ischemia. Describe the mechanism of arterial pressure increase in renal bloodcirculation disturbance.

2. A 17-year-old girl periodically feels palpitation attacks which last for several minutes. Pulse rate reaches 200 per 1 minute. Pulse is rhythmical. What heart disorder has developed? Describe its mechanism.

Themes of abstracts:

1. Aetiology and pathogenesis of pulmonary hypertension.

Tests:

1. What excess of systolic pressure value in the pulmonary artery leads to pulmonary hypertensia?
   a) 100 mm Hg
b) 50 mm Hg
c) 40 mm Hg
d) 30 mm Hg
e) 20 mm Hg

2. What excess of systolic pressure value of blood in the greater circulation leads to hypertensia development?
   a) 120 mm Hg
   b) 130 mm Hg
   c) 140 mm Hg
   d) 160 mm Hg

3. What excess of diastolic pressure value leads to hypertensia development?
   a) 80 mm Hg
   b) 85 mm Hg
   c) 90 mm Hg
   d) 100 mm Hg

4. What pathology is risk factor of arterial hypertensia?
   a) diabetes
   b) diabetes insipidus
   c) allergy

5. The least duration of myocardium ischemia which leads to cardiomyocytes irreciprocal damage is?
   a) 10 min
   b) 20 min
   c) 30 min
   d) 40 min
   e) 50 min

6. What is the basic energy source in the heart?
   a) noneterificite fatty acids
   b) glucose
   c) amino acids
   d) ketone bodies

7. The atrioventricular block of I degree is characterized by:
8. What is characteristic of the atrioventricular block of I degree (type of I Mobits)?
   a) elongation of P-Q interval  
   b) occurrence of the Venkebah-Samojlov periods  
   c) missed ventricular contractions  
   d) contraction of auricles independent from each other and ventricles

9. The full atrioventricular block is characterized by:
   a) elongation of P-Q interval  
   b) occurrence of the Venkebah-Samojlov periods  
   c) fallout of some ventricle contractions  
   d) contraction of auricles independent from each other and ventricles

10. In what type of blockage Morgani-Adams-Stoke attacks are most common?
   a) sinoatrium  
   b) intraatrial  
   c) atrioventricular of the I degree  
   d) atrioventricular of the II degree (type of I Mobits)  
   e) full atrioventricular block

11. At what extrasystole does full compensatory pause take place?
   a) atrial  
   b) atrioventricular  
   c) ventricular

12. Fibrillation is characterized by:
   a) chaotic excitation and contraction of cardiomyocytes  
   b) paroxismal significant increase of heart contractions  
   c) alternating of the periods of normal rhythm with tachy- and bradycardia  
   d) high frequency of correct rhythm contractions (more than 400 per minute)
13. *Trembling of auricles or ventricles is characterized by*
   a) chaotic excitation and contraction of cardiomyocytes
   b) paroxismal significant increase of heart contractions
   c) alternating of the periods of normal rhythm with tachy- and a bradycardia
   d) high frequency of contractions (more than 200 per minute)

14. *Paroxismal tachycardia is characterized by*
   a) chaotic excitation and contraction of some cardiomyocytes
   b) paroxismal significant increase of heart contractions
   c) alternating of the periods of normal rhythm, tachy- and bradycardia
   d) high frequency of correct rhythm contractions (more than 200 per minute)

15. *Sinus arrhythmia is characterized by:*
   a) chaotic excitation and contraction of some cardiomyocytes
   b) paroxismal significant increase of heart contractions
   c) alternating of the periods of normal rhythm with tachy- and a bradycardia
   e) high frequency of correct rhythm contractions (more than 200 per minute)

**d) Answers:**
1d, 2c, 3c, 4a, 5d, 6a, 7a, 8abc, 9d, 10e, 11c, 12a, 13d, 14b, 15c.

**LITERATURE:**
1. Lecture material.
LESSON № 32

TOPIC: PATHOLOGY OF RESPIRATORY SYSTEM

Aim of the lesson: to study the mechanisms of obstructive and restrictive respiratory failure, pulmonary edema, periodic breathing.

QUESTIONS:

1. Pulmonary volumes and capacities.
2. Respiratory failure (etiology, causes, symptoms).
3. Types of disturbances of alveolar ventilation. Obstructive and restrictive disturbances.
7. Pulmonary hypertension
8. Pulmonary edema.

Fig. 37. Pulmonary volumes and capacities.

TV – tidal volume
IRV – inspiratory reserve volume
ERV – expiratory reserve volume
RV – residual volume
IC – inspiratory capacity
FRC – functional residual capacity
VC – vital capacity
TLC – total lung capacity

Pulmonary volumes

• **Tidal volume** \((V_T)\) is the amount of air inspired and expired with each normal breath (500 ml).
• ° **Inspiratory reserve volume** (IRV) is the amount of air that can be inspired beyond the tidal volume (1500-3000 ml). It is used during exercise and other strenuous activities.
• ° **Expiratory reserve volume** (ERV) is the amount of air that can be expired by forceful expiration at the end of a normal tidal expiration (1100-1500 ml).
• ° **Residual volume** (RV) is the amount of air remaining in the lungs after a maximal expiration (1000-1200 ml). It cannot be measured by simple spirometry.

Pulmonary capacities

• ° **Inspiratory capacity** (IC = \(V_T + \text{IRV}\)) is the maximum volume of air a person can inspire beginning from the end of a normal expiration (3500 ml).
• ° **Functional residual capacity** (FRC = \(\text{ERV} + \text{RV}\)) is the volume of air in the lungs after a normal expiration (2300 ml).
  It cannot be measured by spirometry because it includes the residual volume.
• ° **Vital capacity** (VC = IRV + ERV + \(V_T\)) is the volume of air in lungs from maximal inspiration to maximal expiration (4600 ml).
• ° **Total lung capacity** (TLC = IRV + \(V_T\) + ERV + RV) is the maximum volume of air that the lungs can hold after the greatest possible inspiration

Minute ventilation is the sum of all the air breathed during 1 minute. **Minute ventilation (total ventilation)**, is equal to the product of the normal respiratory rate (12 breaths per minute) and the normal tidal volume (500 ml) = 6000 ml/min:

\[
\text{MV} = \text{tidal volume} \times \text{respiratory rate}
\]

**Alveolar ventilation** = (tidal volume-dead space volume) \times \text{respiratory rate} (4200 ml/min)
Fig. 38. Types of pneumothorax:
- tension pneumothorax
- secondary pneumotorax
- spontane pneumothorax

**WORK 1.** Calculation of normal minute volume of breath using the formula:

\[
\text{minute volume of breath} = S \times 3.7 \text{ (at men) or } S \times 3.2 \text{ (at women)}.
\]

At normal conditions the index compounds 6-8 litres/minutes.

The body area is detected according to the diagramm (fig. 39.).

**WORK 2.** Calculation of a normal maximal ventilation of lungs using the formula:

\[
\text{Maximal lung ventilation} = \text{Total lung capacity} \times 22
\]

On the basis of the data on lungs vital capacity calculated using the diagramm (fig. 40.) calculate maximal lungs ventilation according to the formula, normally – 70 – 120 l.
Fig. 39. The diagram for calculation of body surface area
Fig. 40. The diagram for definition of lung capacity in men and women depending on age and height
THE ASSESSMENT TESTS OF RESPIRATORY SYSTEM
FUNCTIONAL STATE

WORK 1. Stange`s test. A patient in sitting position, after a short-term rest (3-5 minutes), takes a deep inhalation and exhalation, then inhales again and holds the breath. Using stopwatch we register the time of breathholding. In men it is not less than 50 seconds, in women – not less than 40 seconds. In sportsmen this time lasts from 60 seconds to several minutes. In children of 6 years: in boys – 20 seconds, in girls – 15 seconds, at 10 years: boys – 35 s., girls – 20 s.

WORK 2. Genchi test. In sitting position after rest, a patient does inhales several times and holds the breath while exhalation. In healthy untrained the time of persons hold breath compounds – 25-30 seconds, in sportsmen – 30-90 seconds.

Stange`s and Genchi tests are used for medical control in health-improving physical training, in mass sports. In diseases of cardiovascular system, respiratory organs, anemias the time of breathhold decreases.

Tasks:

1.

Patient T., 19 years. On thy 3rd day of the disease has arrived into hospital with "acute pneumonia" diagnosis, was hospitalized. Breath was 32 per minute, superficial. Intercostal muscles participate in respiratory locomotions. Small bubbling and dry rales are heard in auscultation. Lungs radioscopy shows changes, characteristic of bilateral croupous pneumonia. Examination of external respiration efficiency detected decreased blood oxygenation– arterial blood saturation was 86 %.

- What form of external respiration disturbance is observed in the patient and what are the mechanisms of its development?
- Disturbance of what processes mostly causes decreased blood oxygenation in this case?
2. Patient K, 8 years, complains of frequent asthma attacks without obvious causes. During an attack breath becomes heavy, accompanied by cough, secretion of some viscous mucoid sputum. Sibilant rales are heard when breathing. Bronchial asthma is in anamnesis a from the age of 5.
- What type of dyspnea is characteristic of this pathology?
- What type of a lung ventilation disorder takes place in this case during asthma attacks?

3. Patient V., 56 years, arrived in neurology department due to cerebral stroke. The state is severe. The Chejn-Stoke type periodic breathing is observed. What factor has major importance in the periodic breathing pathogeny?

4. Pneumothorax kinds. Which pneumothorax is considered to be most (least) dangerous? Why? Which type (obstructive, restrictive) of ventilation disorder is observed in pneumothorax?

5. List structures through which gases (O\textsubscript{2} or CO\textsubscript{2}) diffuse from alveoles into erythrocytes and inversely. In what syndromes and lungs diseases of diffusion of gases is essentially disturbed?

6. What is normal ventilation-perfusion factor? How should it be calculated if: Minute Volume of blood flow = 5 l; Tidal volume (BV) = 500 ml; Respiratory rate (RR) = 12 in minute; volume of dead space = 150 ml/minute?

7. In a child with diphtheria aryngeal edema is observed. What kind of respiratory insufficiency develops and why? What type of breath is detected in the child? Explain the mechanism of dyspnea in this case.
8.

In one patient breathing frequency = 30, tidal volume = 300 ml; in another – breathing frequency = 15, tidal volume = 600 ml. Is the breath efficiency identical? Prove.

**Tests:**

1. What infringements can lead to external breath insufficiency?
   a) alveolar ventilation
   b) diffusion of gases through alveolo-capillary membrane
   c) transport of oxygen blood from lungs to tissues
   d) tissue oxygenation

2. What systems participate in external breath?
   a) respiratory
   b) blood circulation
   c) blood
   d) central nervous system
   e) excretion

3. Give causes of alveolar ventilation disturbances:
   a) reduction of respiratory surface of lungs
   b) constriction of respiratory tract

4. What is the mechanism of obstructive infringements of alveolar ventilation?
   a) reduction of respiratory surface of lungs
   b) infringement of passableness of respiratory tracts

5. Restrictive infringement of pulmonary ventilation are observed at:
   a) pneumonia
   b) pleurisy
   c) retropharyngeal abscess
   d) bronchial asthma

6. Obstructive infringements of pulmonary ventilation are observed in:
   a) bronchial asthma
   b) emphysema of lungs
   c) pneumonia
d) tuberculosis
e) pleurisy

7. How is the residual volume changed in obstructive type of pulmonary insufficiency?
   a) no change
   b) increased
   c) decreased

8. How is the residual volume changed in restrictive type of pulmonary insufficiency?
   a) no change
   b) increased
   c) decreased

9. The vital capacity of lungs is volume of air which can be exhaled after?
   a) the maximum breath
   b) usual exhalation
   c) usual breath

10. How is the vital capacity of lungs changed in obstructive type of pulmonary insufficiency?
    a) no change
    b) decreased
    c) increased

11. How is the vital capacity of lungs changed in restrictive type of pulmonary insufficiency?
    a) decreased
    b) no change
    c) increased

12. What is the Tiffno index?
    a) the attitude of the forced vital capacity of lungs to the first second of vital capacity (VC), expressed in percentage
    b) ratio of VC to respiratory volume
    c) ratio of VC to reserve volume of an expiration
13. How the Tiffno index variates in obstructive model of respiratory insufficiency?
   a) isn’t changed
   b) decreases
   c) increases

14. How the Tiffno index variates in restrictive model of respiratory insufficiency?
   a) decreases
   b) isn’t changed
   c) increases

15. The perfusion respiratory insufficiency is caused by?
   a) reduction of blood-groove through lungs
   b) reduction of lungs ventilation
   c) augmentation of blood-groove through lungs

16. What pathologies cause perfusion respiratory insufficiency?
   a) chronic bronchitis
   b) bronchial asthma
   c) pulmonary embolism
   d) congenital heart diseases with the shunt from right to left

17. What factors cause respiratory insufficiency of obstructive model?
   a) bronchospasm
   b) foreign bodys in respiratory paths
   c) pneumothorax
   e) thyroid gland enlargement

18. What index variates in obstructive model of respiratory insufficiency?
   a) minute volume of breath
   b) index Tiffno
   c) vital capacity of lungs

19. What pathology appears at reduction of surfactant synthesis in lungs?
   a) pneumonia
   b) hypostasis of lungs
   c) pneumofibrosis
d) pulmonary collapse

20. What pathology leads to occurrence of restrictive model of respiratory insufficiency?
   a) bronchial asthma
   b) bronchitis
   c) pneumofibrosis

21. What kind of infringement of alveolar ventilation occurs in secund pneumothorax?
   a) the obstructive
   b) restrictive

22. The restrictive infringement model is characterized by:
   a) lungs straight
   b) patency of upper airways
   c) patency of lower airways

23. What type of dyspnoe occurs in restrictive model of alveolar ventilation disturbance?
   a) inspiratory
   b) expiratory

24. What type of dyspnoe is observed in bronchiolospasm?
   a) inspiratory
   b) admixed
   c) expiratory

25. What type of dyspnoe is observed in stricture formation in tracheas and larynx?
   a) inspiratory
   b) expiratory
   c) admixed

26. What character of respiration is observed in upper respiratory paths stenosis?
   a) frequent and superficial
   b) frequent and penetrating
c) infrequent and superficial
d) infrequent and penetrating

27. What character of breath is observed in the first period of asphyxia?
   a) frequent and penetrating
   b) frequent and superficial
   c) infrequent and superficial

28. What character of breath is observed in the second period of asphyxia?
   a) frequent and penetrating
   b) frequent and superficial
   c) infrequent and penetrating
   d) infrequent and superficial

29. What character of breath is observed in the third period of asphyxia?
   a) penetrating and frequent
   b) infrequent and superficial
   c) apnoea
   d) infrequent and penetrating

30. Hemodynamics in the first period of asphyxia is characterised by:
   a) increase of minute volume of blood
   b) increase of arterial blood pressure
   c) reduction of minute volume of blood
   d) reduction of arterial blood pressure
   e) asystolia

31. Hemodynamics in the second period of asphyxia is characterised by:
   a) increase of minute volume of blood
   b) increase of arterial blood pressure
   c) reduction of minute volume of blood
   d) reduction of arterial blood pressure
   e) asystolia

32. What model of dyspnea is observed in emphysema of lungs?
   a) inspiratory
   b) expiratory
33. Cheyn-Stokes breath is characterized by:
   a) apnoea alternates with respiratory locomotions of increasing, and then decreasing depth
   b) apnoea alternates with respiratory locomotions of identical depth
   c) bradypnoea with the enhanced inhale and exhale
   d) deep and noisy breath

34. Biot breath is characterized by:
   a) apnoea alternates with respiratory locomotions of increasing, and then decreasing depth
   b) apnoea alternates with respiratory locomotions of identical depth
   c) bradypnoea with the enhanced inhale and exhale
   d) deep and noisy breath

35. Cussmaul breath is characterized by:
   a) apnoea alternates with respiratory locomotions of increasing, and then decreasing depth
   b) apnoea alternates with respiratory locomotions of identical depth
   c) bradypnoea with the enhanced inhale and exhale
   d) deep and noisy breath

Answers:
1ab, 2abcd, 3a, 4b, 5ab, 6ab, 7b, 8c, 9a, 10a, 11a, 12a, 13b, 14b, 15a, 16cd, 17abd, 18b, 19d, 20c, 21b, 22a, 23a, 24c, 25c, 26d, 27a, 28b, 29b, 30ab, 31cd, 32b, 33a, 34b, 35d.

LITERATURE:

1. Lecture material.
LESSON № 33

**TOPIC:** KIDNEY PATHOLOGY.

**Aim of the lesson:** to study the causes and mechanisms of kidney failure and its consequences.

**QUESTIONS:**
2. Pathogeny of infringements of urine formation (infringement of filtration, reabsorption, secretion and excretion).
3. The causes and mechanisms of diuresis infringements. Quantitative and qualitative infringements of urine formation (oliguria, anuria, polyuria). Hypostenuria, isostenuria, hyperstenuria. The causes.
6. Consequences of chronic glomerulonephritis.
Fig. 41. Summary of hydrostatic and colloid factors causing filtration by the glomerular capillaries. The values are estimated for a healthy person.

\[
C = \frac{UV}{P}
\]

<table>
<thead>
<tr>
<th>Symbol</th>
<th>Definition</th>
<th>Unit</th>
</tr>
</thead>
<tbody>
<tr>
<td>C</td>
<td>clearance</td>
<td>ml/min or ml/24 hr</td>
</tr>
<tr>
<td>U</td>
<td>urine concentration</td>
<td>mg/ml</td>
</tr>
<tr>
<td>V</td>
<td>urine volume/time</td>
<td>ml/min</td>
</tr>
<tr>
<td>P</td>
<td>plasma concentration</td>
<td>mg/ml</td>
</tr>
</tbody>
</table>

Fig. 42. Clearance calculation

**Clearance** - Is the ratio between excretion rate and plasma concentration for the substance. Renal clearance can also be thought of as the volume of arterial plasma completely cleared of the substance in the kidneys within one min, or the number of ml arterial plasma containing the same amount of substance as contained in the urine flow per minute.
Tasks:

1. Patient M., 16 years, presented to hospital in extremely severe shock. He was knocked down by a car. There are multiple fractures of both legs. The arterial pressure – 80/60 mm Hg, daily diuresis – 60-80 ml of urine per day. In urine: protein – 0.66 g/l, relative density of urine – 1.029. Residual nitrogen of blood – 120 mmol/l; blood urea – 35 mmol/l.
   - What mechanism of anuria is observed in the patient?

2. Patient N., 36 years, is hospitalized in nephrology department. Renal disease was detected 2 years ago when after recent acute respiratory disease face edemas developed and protein was detected in urine.
   - The urine analysis: straw-yellow colour, transparent, acid reaction, protein – 0.8 g/l, glucose is absent.
   - Precipitate: small amount of epithelium, leucocytes – 1-5, erythrocytes – 2-3 in sight, fresh and variated; hyaline cylinders – individual in sight.

Fig. 43. Artificial kidney
Zimnitsky test: comparative density of urine 1,004-1,015 at a diurnal diuresis of 2600 ml. In blood: urea – 5,1 mmol/l, kreatinine – 70 μmol/l.
- What pathological changes of urine composition are revealed in the patient?
- Are there signs indicating disturbance of renal filtration ability?
- What signs indicate disturbance of renal concentration ability?
- What is the possible mechanism of renal concentration ability disturbance in this case?

3.

Patient K, 24 years, fall ill acutely after catching a cold. Complaints to general weakness face edemas, headache, dyspnea in insignificant exercise stress.

The urine analysis: red-brown colour, cloudy, sour reaction, protein – 1,2 g/l, glucose is absent.


Zimnitsky test: comparative density of urine 1,012-1,031 at a diurnal diuresis of 780 ml. Endogenic kreatinine clearance – 56 ml/minutes
- What pathological components of urine are revealed in the patient?
- What signs indicate disturbance of renal concentration ability?
- What is the possible mechanism of renal concentration ability disturbance in this case?
- Are there signs indicating disturbance of renal concentration ability?

4.

Patient N, 45 years. Eight years ago he felt pains in lumbar area, moderate face and extremities edemas appeared. Five years later the exacerbation of the same symptoms occured. Later no symptoms were observed. Now the patient is hospitalised due to worsening of the general state. The urine analysis: yellow colour, acid reaction, protein – 0,6 g/l, glucose is absent, in deposit: moderate quantity of epithelium, leucocytes – 10-15 in sight, erythrocytes are single in a preparation, cylinders, waxy, acinose – 2-3 in sight.

Zimnitsky test: comarative density of urine 1,010-1,016 at diurnal diuresis of 860 ml.

In blood: urea – 9 mmol/l, kreatinine – 115 μmol/l.
What pathological changes the urine analysis indicates?
Are there data indicating glomerular filtration disorder?
Are there data indicating renal concentration ability disturbance?

**Tests:**

1. *The factors promoting increase of glomerular filtration:*
   a) hydrostatic pressure augmentation in capillars
   b) hydrostatic pressure reduction in capillars
   c) augmentation of oncotic pressure in capillars
   d) reduction of oncotic pressure in capillars
   e) augmentation of intrarenal pressure

2. *The factors causing decrease of glomerular filtration:*
   a) decrease in a tone of efferent arteriole
   b) decrease of oncotic pressure in capillars
   c) increase of oncotic pressure in capillars
   d) decrease in intrarenal pressure
   e) increase of intrarenal pressure

3. *The materials which are exposed to reabsorption in proximal departments of renal tubule:*
   a) glucose
   b) amino acids
   c) Na
   d) inulin

4. *The factors disturbing reabsorption of substances in proximal departments of renal tubule:*
   a) depressing of activity of ferments in renal tubule epithelium
   b) increase of activity of ferments in renal tubule epithelium
   c) surplus of reabsorption materials in primary urine
   d) tubule damage

5. *The water reabsorption in proximal department of renal tubule is caused:*
   a) active transport Na$^+$
   b) glucose reabsorption
c) reabsorption $K^+$
d) vasopressinum action

6. **Factors of water reabsorption decrease in proximal kidney tubule nephroses:**
   a) primary urine osmolar depressing
   b) glucose level increase above 10,0 mmol/l in blood
   c) electrocortin introduction
   d) furosemide Introduction

7. **The water reabsorption in distal department of renal tubule is controlled by:**
   a) vasopressinum
   b) electrocortin
   c) atrial natriuretic factor
   d) insulin

8. **Hyposthenuria is:**
   a) depressing of relative density of urine
   b) depressing of diurnal diuresis
   c) decreased urination frequency

9. **Hyposthenuria is caused by:**
   a) tubular nephron device damage
   b) Shumljansky-Boumen sheath damage
   c) acute glomerulonephritis
   d) chronic glomerulonephritis

10. **Hypersthenuria is:**
    a) increase of urination frequency
    b) increase of relative density of urine
    c) prevalence of a night diuresis over a diurnal diuresis

11. **Hypersthenuria is caused by:**
    a) tubular nephron device damage
    b) acute glomerulonephritis
    c) chronic glomerulonephritis

12. **Polyuria is augmentation:**
a) diurnal quantity of urine more than 2: 1
b) urination frequency
c) portions of urine

13. Oliguria is:
   a) hematocrite index decrease
   b) diurnal quantity of urine reduction less than 0,5/l
   c) circulating blood volume reduction
d) decreased urination frequency

14. Anuria is:
   a) augmentation of diurnal quantity of urine (it is more 1,5)
   b) reduction of diurnal quantity of urine (less than 0,5)
c) reduction of diurnal quantity of urine (less than 100 ml)

15. The renal clearance is:
   a) volume of plasma which refines from the yielded material nephroses at one minute
   b) minute diuresis
c) the volume of urine formed in one minute

16. To detect the volume glomerular filtration substances which... are used:
   a) precipitate out by a filtration in renal glomuluses
   b) are not exposed to an anatropic reabsorption
c) are exposed to an anatropic reabsorption

17. To detect glomerular volume filtration following substances are used:
   a) inulin
   b) endogenous kreatinine
   c) glucose
d) protein

18. Normal protein level in urine of the adults comprises:
   a) less than 0,033 g/l
   b) 0,33 g/l
c) 0,66 g/l
d) 1 g/l
19. *The proteinuria ekstrarenal causes are:*
   a) paraproteinemia
   b) tubular epithelium damage
   c) chronic renal insufficiency
d) diabetes insipidus
e) inflammatory processes in urinary paths

20. *The polyuria renal causes are:*
a) diabetes
b) renal insufficiency
c) chronic glomerulonephritis
d) inflammatory processes in urinary paths

21. *Basic pathogenesis links of acute diffusive glomerulonephritis are:*
a) decrease in a circulation of blood in nephroses
b) infringement of tubules secretory function
c) occurrence of antibodies against streptococcus antigens
d) lack of liposoluble vitamins

22. *Nethrotic syndrome symptoms are:*
a) decrease in quantity of lipids in blood
b) proteinuria
c) high blood pressure
d) hypoproteinemia
e) hypercholesterinemia

23. *The mechanism of filtration decrease in hypovolemic shock:*
a) decrease in effective filtration pressure
b) area reduction of the glomerular filter
c) decrease in permeability of membranes of glomuluses

24. *The mechanism of glomerular filtration decrease in urinary retention is following:*
a) decrease in effective filtration pressure
b) area reduction of the glomerular filter
c) decrease in permeability of membranes of glomuluses
25. *The mechanism of glomerular filtrations decrease the at chronic glomerulonephritis:*
   a) decrease in effective filtration pressure
   b) area reduction of the glomerular filter
   c) blood oncotic pressure increase

26. *The glucose reabsorption reduces in damage of epithelium of:*
   a) proximal tubules
   b) distal tubules
   c) the collective tubules
   d) Henle's loops

27. *The reabsorption of amino acids reduces in damage of epithelium of:*
   a) proximal tubules
   b) distal tubules
   c) the collective tubules
   d) Henle's loops

28. *Albumin-globulin factor in nephrosis:*
   a) is not variated
   b) increases
   c) decreases

29. *The protein reabsorption is disturbed in damage of epithelium of:*
   a) proximal tubules
   b) distal tubules
   c) the collective tubes
   d) Henle's loops

30. *The main symptom in the development of edemas in nephrotic syndrome is:*
   a) increase of permeability of pots
   b) augmentation of production of electrocortin
   c) hypoproteinemia
   d) augmentation of production of vasopressininum
31. **Combined increase of glomerular filtration and reduction of tubular fluid reabsorption leads to:**
   a) polyuria  
   b) oliguria  
   c) anuria

32. **Combined decrease of glomerular filtration and increase of tubular fluid reabsorption leads to:**
   a) polyuria  
   b) oliguria  
   c) anuria

33. **Increased glomerular filtration accompanied by normal tubular fluid reabsorption leads to:**
   a) polyuria  
   b) oliguria  
   c) anuria

34. **The reduction of glomerular filtration accompanied by normal tubular fluid reabsorption leads to:**
   a) polyuria  
   b) oliguria

35. **The main pathogeny link of acute renal insufficiency is:**
   a) glomerular filtration decrease  
   b) glomerular filtration increase  
   c) tubular reabsorption decrease

36. **The main pathogeny link of chronic renal insufficiency:**
   a) renal concentration ability decrease  
   b) renal concentration ability increase  
   c) tubular reabsorption increase  
   d) glomerular filtration augmentation

37. **What is observed in the first stage of acute renal insufficiency:**
   a) oliguria  
   b) pollakiuria (frequent urination)
c) polyuria

d) nycturia

38. **Mechanisms of metabolic acidosis in renal function disturbances:**
   a) renal tubules carbonic anhydrase deficiency
   b) infringement of process of deamination
   c) renal tubules transamination infringement
   d) glomerular filtration increase

39. **Azotemia mechanism in renal insufficiency is:**
   a) glomerular filtration decrease
   b) reduction of tubular reabsorption
   c) reduction of tubular secretions

40. **Anaemia mechanism in chronic renal insufficiency is:**
   a) metabolism products excretion infringement
   b) organism intoxication
   c) erythropoietin insufficiency
   d) hemolysis of erythrocytes in renal tubules

**Answers:**
1ad, 2ace, 3abc, 4acd, 5ab, 6abd, 7a, 8a, 9ad, 10b, 11b, 12a, 13b, 14c, 15a, 16ab, 17ab, 18a, 19ae, 20bc, 21ac, 22bde, 23a, 24a, 25b, 26a, 27a, 28c, 29a, 30c, 31a, 32b, 33a, 34b, 35a, 36a, 37a, 38ab, 39a, 40c.

**LITERATURE:**

1. Lecture material.
LESSON № 34

TOPIC: PATHOPHYSIOLOGY OF THE LIVER. JAUNDICE

Aim of the lesson: to study the causes and mechanisms of hepatic failure and jundance and their consequences.

QUESTIONS:

1. Studying the liver functions in the experiment. Functional trials.
Fig. 44. Studying the role of the liver in experimental conditions
а – location of vessels before operation; – б Eck’s Fistula; 
в – Eck-Pavlov's fistula

Table 6. Indices of pigmen
tal exchange in blood and ur
ine in various kinds of jaundices

<table>
<thead>
<tr>
<th>Kinds of jaundices</th>
<th>Blood Bilirubin</th>
<th>Urine Bilirubin</th>
<th>Urine Urobilinogen</th>
<th>excrement sterko-bilin excrement</th>
<th>Liver functional tests</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Direct</td>
<td>Indirect</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mechanical</td>
<td>+++</td>
<td>+</td>
<td>-</td>
<td>+++</td>
<td>-</td>
</tr>
<tr>
<td>Parehimatous</td>
<td>++</td>
<td>+</td>
<td>+++</td>
<td>+</td>
<td>+++</td>
</tr>
<tr>
<td>Hemolytic</td>
<td>±</td>
<td>+++</td>
<td>++</td>
<td>±</td>
<td>++</td>
</tr>
</tbody>
</table>

Laboratory work 1. Direct bilirubine estimation in blood plasma.

Work description. In a rat the blood is taken after occlusion of the bile duct. After blood centrifugation the plasma (1 ml) is taken to detect
quality reaction of direct billirubine. A few drops of diazoreactive are added to investigate plasma. The red color of plasma indicates the presence of direct billirubine in blood.

Students make conclusion about type of jaundice.

**Laboratory work 2. Detection of direct bilirubin in blood serum of animals in obstructive jaundice.**

Work course: Direct reaction. 0.25 ml of diazoreagent is added to 1 ml of investigated serum. In the presence of connected (direct) bilirubin a pink colouring is observed. We analyze. Make conclusions.

**Tasks:**

1.

Patient N, 46 years, complaints of weakness, absence of appetite, nausea, pains in right hypochondrium. In clinicolaboratory research the expressed yellowness of scleras and skin is revealed. In blood 71.8 μmol/l of direct bilirubin and insignificant quantity of indirect bilirubin is detected. In urine there is a high level of bilirubin and urobilin. In excrement there are marks of sterkobilin.

- Define jaundice type.
What mechanisms of hyperbilirubinemia and urobilinuria are observed in present type of jaundice?

2.

Patient D, 38 years. Approximately a day after pain attack in the area of right hypochondrium and epigastric area jaundice appeared.

Clinicolaboratory research has revealed: slightly increased liver, sensitive in palpation. Blood contains 5 μM of free and 147 μmol/l of conjugated bilirubin. Urine is dark, excrement is uncolored.

- To define jaundice type.
How is the discolouration of urine and excrement explained in this case?
3. Patient R., 33 years, has been feeling catarrhal phenomena, weekness, headaches, subfebrile temperature for a week. A day ago yellowness of scleras, dark urine was detected; he was hospitalised.

Blood test revealed free bilirubin – 27,4 μM, bound bilirubin – 51,3 μM.

Urine is of dark-brown colour, rather turbid, reaction to bilirubin – positive. Excrement is poorly coloured.

- Define jaundice type.
- What indicates high level of connected bilirubin in blood?

4. Patient K., 14 years, presented in hospital with complaints of general weekness, pains in right hypochondrium, skin yellowness.

Objectively: the bilirubin in blood serum is 51 μm, reaction with Ehrlich diazo reagent is indirect. In urine urobilin is detected. Excrement is intensively coloured.

- Detect jaundice type.
- What are hyperbilirubunemia mechanism in this jaundice type?

5. Patient A., 12 years, complaints of nausea, vomiting, dermal itch. Fell ill 8 days ago.

Examination detected: yellow scleras and skin, enlarged liver and spleen. AP – 100/60 mm Hg, pulse – 56 in a minute, rhythmical.

Bilirubin in serum – 76 μM, indirect – 20,5 μM.

Bilirubin and urobilin are detected in urine.

- Detect jaundice type.
- What bilirubin type is detected in urine of the patient?


Laboratory research detected: blood serum bilirubin – 70 μmol/l.
In urine and excrement the increased quantity of urobilin and sterkobilin is detected. Bilirubin in urine is not detected.

- Detect jaundice type.
- What bilirubin type is detected in blood serum?

7.

Patient T, 47 years, the existing dispeptic syndrome was accompanied by quickly growing jaundice. Clinicolaboratory research showed following symptoms: enlarged liver, gall bladder is accessible to palpation.

In blood there is 342 μM of bilirubin, reaction with Ehrlich diazo reagent is straight.

In urine high level of bilirubin is detected.

Excrement is decolorized.

- Detect jaundice type.
- What are the mechanisms of bilirubin increase in blood in this jaundice type?

8.

Patient V., 32 years, presented in clinic with complaints of general weakness, dyspnea, skin yellowness, dark colouring of stool and urine.

Laboratory data: bilirubin in blood serum – 68 μmol/l.

Reaction with Ehrlich diazo reagent – indirect.

In urine: reaction to bilirubin is negative, to urobilin bodies – positive.

- Detect jaundice type.
- Which bile pigments concern to urobilin bodies?

9.

Patient B., 32 years, is delivered to clinic with complaints of sudden pains in right hypochondrium, nausea, vomiting, skin yellowness. In the anamnesis there are common pain attacks in right hypochondrium within last 5 years.

Objectively: scleras and skin icteritiousness, acute morbidity and muscular strain in right hypochondrium in palpation. Body temperature – 38,7°C.

Laboratory data: blood bilirubin – 68 μM, direct.
Urine of greenish-yellow colour, reaction to bilirubin is positive, urobilin bodies are absent.
Stool is clayey, uncolored.
- Detect jaundice type.
- What can indicate absence of urobilin bodies in urine?

10.
In a patient following changes are detected: hyperbilirubinemia (indirect bilirubin – 28.3 μM), urobinogenemia, sterkobilin - and urobilingenuria (5.48 μmol/sut), hypercholic stool.
Detect jaundice type which is characterized by these changes. Explain pathogeny of the observed disorders.

11.
Hyperbilirubinemia was detected in a patient (direct bilirubin – 6.7 μM), bilirubinuria, acholia, hypercholesterinemia (13 mM).
Detect jaundice type which is characterized by these changes. Explain pathogeny of the observed disorders.

12.
Hyperbilirubinemia was detected in a patient’s blood due to direct and indirect bilirubin, in urine – direct bilirubin, urobilin, bile acids, in stool – hypocholia.
Hypocholesterinemia (1.2 mM), hypoproteinemia (crude protein – 30 g/l), decrease in coagulability of blood are detected as well.
Detect jaundice type which is characterized by these changes. Explain mechanisms of these disorders occurrence.

Themes of reports:
1. Disbolism in functional insufficiency of the liver.
2. Detoxication liver function infringement.

Tests:
1. *The use of glucose in the treatment of hepatic patients is conditioned by:*

   a) energy source
   b) stimulates ammonia neutralisation
   c) activates glyconeogenesis in a liver
   d) source for formation of glucuronic acid

2. *Name three basic indicative enzymes of the liver damage:*
   a) LDH (lactate dehydrogenase)
   b) ALT (alanine aminotransferase)
   c) AST (aspartate aminotransferase)
   d) hexokinase
   e) glycogensintase

3. *In diffusive lesion of hepatocytes following changes of protein level are observed:*
   a) hypoalbuminemia
   b) hypergammaglobulinemia
   c) hyperalbuminemia
   d) hypogammaglobulinemia

4. *How is the level of amino acids in blood variated in liver pathology?*
   a) increases
   b) it is depressed
   c) does not variate

5. *Urea level in blood in liver pathology:*
   a) increases
   b) it is depressed
   c) is not variated

6. *How is ammonia level in blood variated in liver pathology?*
   a) it is depressed
   b) raises
   c) does not variate
7. Fatty infiltrations of hepatocytes promote development of:
   a) increased mobilisation of fats from the depot
   b) lipakaine deficiency
   c) yield of fat from the liver
   d) intensifying lipolisis in the liver
   e) lack of ferments β - oxidations of fatty acids

8. What is the main factor in development of ascites in cirrhosis?
   a) decrease in osmotic pressure of blood
   b) augmentation of permeability of pots
   c) portal hypertensia
   d) Increased secretion of atrial natriuretic factor

9. Termination of bile inflow into the duodenum causes the deficiency of following vitamins:
   a) B₁
   b) A
   c) D
   d) B₁₂

10. Lipolysis disturbances are caused инвуашсие ца following substances in the intestine:
    a) bile acids
    b) bilirubin
    c) cholesterol

11. Decrease of pancreatic lipase activity is connected with disturbances in synthesis of:
    a) bile acids
    b) bilirubin
    c) cholesterol

12. Biochemical composition of blood in haemolytic jaundice is characterised by increase of:
    a) indirect bilirubin
    b) direct bilirubin
c) direct and indirect bilirubin

13. **Biochemical composition of blood in obstructive (mechanical) jaundice is characterised by maintenance increase of:**
   a) indirect bilirubin
   b) direct bilirubin
   c) direct and indirect bilirubin

14. **Biochemical composition of blood in hepatic (hepatocellular) jaundice is characterised by increased content of:**
   a) indirect bilirubin
   b) direct bilirubin
   c) direct and indirect bilirubin

15. **Biochemical composition of urine in haemolytic jaundice is characterised by presence of:**
   a) indirect bilirubin
   b) direct bilirubin
   c) urobilin

16. **Biochemical composition of urine in obstructive (mechanical) jaundice is characterised by presence of:**
   a) direct bilirubin
   b) urobilin
   c) indirect bilirubin
   d) verdoglobin

17. **Biochemical composition of urine in hepatic (hepatocellular) jaundice is characterised by presence of:**
   a) direct bilirubin
   b) indirect bilirubin
   c) urobilin

18. **Of which jaundice type cholema syndrome is characteristic?**
   a) haemolytic
   b) obstructive (mechanical)
   c) the hepatic (hepatocellular)
19. What change of heart rhythm activity is characteristic of obstructive (mechanical) jaundice?
   a) tachycardia
   b) bradycardia
   c) sinus arrhythmia

20. What causes changes in heart rhythm activity in obstructive (mechanical) jaundice?
   a) indirect bilirubin
   b) direct bilirubin
   c) bile acids
   d) cholesterol

21. Which changes of hemostasis characterize hepatic jaundice?
   a) increased hemorrhage
   b) intravascular thrombosis

22. What manifestations are characteristic of obstructive (mechanical) jaundice?
   a) increase of bile acids in blood
   b) hypercholesterinemia
   c) skin itch
   d) tachycardia
   e) decrease of direct bilirubin in blood

24. The itch in mechanical jaundice is connected with:
   a) hyperbilirubinemia
   b) choleemia
   c) Infringement of lipids exchange
   d) hyperpotassemia

23. What changes in blood are detected in hepatic jaundice?
   a) augmentation of direct bilirubin
   b) augmentation of indirect bilirubin quantity
   c) choleemia
   d) urobilinogen augmentation
   e) hyperalbuminemia

24. What is characteristic of hepatic insufficiency:
25. **What set of biochemical composition changes of blood characterizes hepatic coma?**
   a) hypoglycemia, increased level of amino acids, increased level of ammonia
   b) hyperglycemia, reduction of ammonia level
   c) hypoglycemia, reduction of amino acids level, increased level of ammonia
   d) hyperglycemia, increased amino acids level, reduction of ammonia level.

26. **Which of the following substances has the greatest value in pathogeny of hepatic coma?**
   a) bilirubin
   b) bile acids
   c) ammonia
   d) urea

27. **What clinical symptoms are caused by choleemia?**
   a) dermal itch
   b) bradycardia
   c) tachycardia
   d) diarrhoeia
   e) depressing of arterial pressure

29. **How does residual nitrogen variate in liver diseases?**
   a) does not variate
   b) raises
   c) drops

30. **Portal hypertensia develops if blood pressure in the portal vein is more than:**
   a) 36 mm Hg
   b) 26 mm Hg
   c) 16 mm Hg
   d) 8 mm Hg
   e) 0 mm Hg
31. Liver blood-flow volume in direct Eck fistula is:
   a) reduced
   b) increased
   c) not variated

32. Liver blood-flow volume in inverse Eck-Pavlov fistula is:
   a) reduced
   b) increased
   c) not variated

33. Choleemia syndrome is characterised by increase of following components in blood:
   a) bile acids
   b) cholesterol
   c) chylomicrons
   d) muriatic acid

Answers:
1ad, 2abc, 3ab, 4a, 5b, 6b, 7abe, 8c, 9bc, 10a, 11a, 12a, 13b, 14c, 15c, 16a, 17ac, 18bc, 19b, 20c, 21a, 22abc, 23b, 24abcd, 25b, 26a, 27c, 28abe, 29b, 30d, 31a, 32b, 33ab

LITERATURE:
1. Lecture material.
LESSON № 35

TOPIC: PATHOPHYSIOLOGY OF DIGESTION.

Aim of the lesson: to study the causes and mechanisms of gastritis, peptic ulcer, pancreatitis, and disbacteriosis and their consequences.

QUESTIONS:

1. Role of digestive organs. Aetiology of diseases of gastrointestinal disorders. A role of alcohol, smoking and other factors in occurrence of these disorders.
Table 7. Basal and stimulated secretion of the stomach

<table>
<thead>
<tr>
<th>Secretion state</th>
<th>Basal</th>
<th></th>
<th>Stimulated</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Quantity secret, ml/H</td>
<td>HCl mM/H</td>
<td>Quantity secret, ml/h</td>
<td>HCl mM/h</td>
</tr>
<tr>
<td>Normal</td>
<td>60±25</td>
<td>3±2,5</td>
<td>45±15</td>
<td>200±55</td>
</tr>
<tr>
<td>Hypersecretion (duodenal ulcer)</td>
<td>100</td>
<td>6</td>
<td>60</td>
<td>250</td>
</tr>
<tr>
<td>Hyposcretion (stomach cancer)</td>
<td>40</td>
<td>0,5</td>
<td>12,5</td>
<td>80</td>
</tr>
</tbody>
</table>

Table 8. Stomach secretion types

<table>
<thead>
<tr>
<th>Type</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>Moderately excitable type in reflex and chemical phases of secretion</td>
</tr>
<tr>
<td>Excitable</td>
<td>Irritability of glands in both phases of secretion (secretion and gastric acidity increase)</td>
</tr>
<tr>
<td>Inhibitory</td>
<td>Decreased excitability of glands in both phases of the secretion, decreased secretion and absence of free HCl</td>
</tr>
<tr>
<td>Asthenic</td>
<td>Excitability increases in the 1st phase, decrease of secretion and acidity in the 2nd phase</td>
</tr>
<tr>
<td>Inert</td>
<td>Decreased stomach glands secretion in the 1st phase, in the 2nd – N or secretion increase</td>
</tr>
</tbody>
</table>

Stomach and intestine ulcer experimental modelling:
- Mucosa damage by physical and chemical stimuli (hot water, fused silver nitrate, croton oil, acids, alcohol).
- Blood circulation disorders (bandaging stomach and duodenum vessels, embolism, sclerosis).
- Prolonged introduction of substances enhancing secretion of gastric juice (quinophan, histamine, pilocarpine, physostigmine, pentagastrin, etc.).
- Chronic irritation of the vagus nerve.
- Cortical mechanisms disturbances in experimental neuroses.
- Pylorus ligation (Shay method).
- Gastrotoxic serum introduction.

![Fig. 45. Acid hyposcretion and acid hypersecretion](image)
Fig. 46. Peptic ulcer. Imbalance in the defensive and aggressive forces

Fig. 47. Pathogenesis of acute pancreatitis

**Laboratory work 1.** Role of pH in protein digestion in the stomach.

**Work description.** Two Petry cups with gastric juice are used for experiment. The 1st cup pH should be about 1.0 and in 2nd – about 7.0
(arrived by 0.11 HCl or NaOH and controlled by lacmus paiper). The equal amount of bull fibrin is added to cups and after 15 min students fix results.

Students make conclusion about role of pH in protein digestion in the stomach.

**Practical work**

**WORK 1.** Detection of digestion ability of gastric juice of various acidity.

**Work course:** In Petri dishes we pour gastric juice with various acidity (increased and decreased). We put fibrin pieces in cups. We maintain constant temperature within 37°C with the help of thermostat for an hour. We estimate a state of fibrine pieces during the experiment.

**Tasks:**

1. Hidrocortizonum at a dose of 0.5-1.0 mg per 100g of weight was intramuscularly injected to rats with weight of 160-180 g. daily. After 10-15 injections all animals developed erosions or ulcers in secretory department of the stomach. Explain development mechanisms of «hydrocortizonum» stomach ulcers in experimental animals.

2. For reproduction of experimental stomach ulcers the pylorus is ligatured with retention of its patency (Shay method). Explain the mechanism of stomach ulcer occurrence in ligature imposition on pyloric stomach department.

3. Patient G, 34 years, presented to hospital for examination. Considers himself a patient for 4 months when he felt "aching" pains in epigastrium,

4.
Patient G, 53 years, was hospitalized with complains of pains in epigastric areas, especially after food intake, air eructation, nausea, sometimes vomiting, absence of appetite. In fractional stomach contents research 10 ml of fluid with slime was extracted, free hydrochloric acid – 0, total acid – 10 t.u. are taken on an empty stomach. Bazal secretion: hour strain of 25 ml, free hydrochloric acid – 5-10 t.u. Total acidity – 15-20 t.u. Debit-hour of hydrochloric acid – 0,3 milliequivalent. In response to submaximum histamine stimulation: hour strain of secretion – 45 ml, free hydrochloric acid - 15-25 t.u. Total acidity – 30-40 t.u. Debit-hour of hydrochloric acid – 0,5 milliequivalent. Characterise secretory function of the stomach on the basis of the received data.

5.
Patient Z., 63 years, complains of pains in epigastric area. After food intake pain increases, feeling of heaviness is observed. Vomiting by eaten food is common. For last 3-4 months began to weaken and loos weight considerably. Examination of stomach function by one-stage Boas-Evald method the following is revealed. Fourty five minutes after a trial breakfast 180 ml of stomach contents was extracted. The flaking quotient, which is the ratio of dense layer (the grinded bread) to liquid one equals 1:5. Free hydrochloric acid – 10 t.u., total acidity – 35 t.u. A reaction to milk acid is positive;
What is the character of stomach secretory function disorder?

6.
Patient M., 52 years, presented in clinic for examination. About 3 months ago appetite worsened, disgust to meat appeared, started to loosen weight. In the anamnesis 12 years felt unexpressed pains in epigastric area, heartburn. Fractional sounding detected: a portion on the empty stomach – 5 ml of stomach contents, free hydrochloric acid – 0, total acid – 35 t.u. A unit of Basal secretion: hour strain – 25 ml, free muriatic acid – 0, the general acidity - 12-18 t.u. A unit in response to histamine stimulation: hour strain of secretion – 20 ml, free hydrochloric acid – traces, total acid – 15-21 t. a unit.
What is the character of stomach secretory function disorder?

7.
The rat preliminarily starving for 24 h, was immobilized and put in the cold chamber with temperature +4 C for 4 hours. Results of the rat’s stomach contents analysis 24 h later: total acidity – 90 mmol/l, free HCl – 60 mmol/l. Dissection detected that the stomach mucosa was hyperemic, some erosions were detected too. Explain the mechanism of the observed changes.

8.
Patient, 38 years old, complains of burning in the tongue, heaviness in the epigastric area, air eructation, diarrhoeia, fatigability, dyspnea. Objectively: skin and mucosa is pale, tongue is crimson. In gastric juice free HCl is absent. Total acid – 12 mmol/l. In excrement not digested muscular fibres are found.

Results of the blood analysis: erythrocytes – 2,1x10^{12}/l, colour index> 0,5, anisopoikilocytosis. How can dispeptic phenomena be explained? What is pathogeny of the observed phenomena? Count a colour index.

Tests:
1. Pathological intensifying of appetite is designated by the term:
   a) hyperrexia
   b) polyphagia
   c) disphagia
   d) aphagia
   e) anorexia

2. Anorexia – is:
a) absence of appetite
b) difficulty in swallowing
c) pathologically increasing of appetite
d) increase of food intake
e) bulimia

3. *Bulimia is:*
   a) absence of appetite
   b) difficulty in swallowing
   c) pathologically increasing of appetite
   d) increase of food intake
   e) disturbance of salivation

4. *Polyphagia is:*
   a) absence of appetite
   b) difficulty in swallowing
   c) pathologically increasing of appetite
   d) increase of food intake
   e) chewing disorder

5. *Dysphagia is:*
   a) absence of appetite
   b) pathologically increasing of appetite
   c) increase of food intake
   d) difficulty in swallowing

6. *Anorexia nervosa occurs in:*
   a) enteric infection
   b) diabetes mellitus
   c) hysteria
   d) intoxication
   e) vomiting

7. *Causes of hyperrexia are:*
   a) diabetes mellitus
   b) intoxication
   c) pain syndrome
   d) suppression of food centre
   e) damage of ventrolateral nuclei hypothalami
8. Pararexia is:
   a) perversion of appetite
   b) fast satiation
   c) difficulty in swallowing
   d) increasing of appetite
   e) decreasing of appetite

9. Causes of hypersalivation are:
   a) fever
   b) parotitis
   c) sialoadenitis
   d) sialolithiasis
   e) sialolithiasis, toxicosis of pregnancy

10. Consequences of hypersalivation are:
    a) scurf on tongue
    b) lack of $K^+$
    c) caries
    d) inflammation in oral cavity
    e) xerostomia

11. Hyposalivation leads to:
    a) neutralisation of gastric juice
    b) maceration of skin around mouth
    c) hypokaliemia
    d) ptyalism (sialorrhea)
    e) xerostomia

12. What is the main effect of leptin, synthesised in adipocytes?
    a) it enhances inhibition in CNS
    b) it activates excitation in CNS
    c) it oppresses appetite
    d) it increases appetite

13. Where neuropeptid Y – the basic activator of sense of hunger - is developed?
    a) stomach
    b) small intestine
14. How does appetite change in ventrolateral nucleus of hypothalamus irritation?
   a) does not change
   b) increases
   c) reduces

15. Hyposalivation causes:
   a) acidity reduction in the stomach
   b) swallowing disorders
   c) organism dehydration
   d) gastroesophageal reflux

16. Caries is promoted by intake of:
   a) sweet
   b) salty
   c) fat

17. Incoercible vomiting causes:
   a) metabolic acidosis development
   b) development non-gaseous alkalosis
   c) increase of arterial pressure
   d) hyperglycemia
   e) hyperchlorhydria

18. Vomiting causes:
   a) hypernatriemia
   b) hypochloremia
   c) metabolic acidosis
   d) hyperosmolar dehydration

19. Vomiting has protective-adaptive value in:
   a) pregnancy toxicosis
   b) reception of substandard nutrition
   c) brain tumours
   d) emotional shock
   e) hydrocephaly
20 Stomach gases cause:
   a) hiccups
   b) vomiting
   c) eructation
   d) nausea
   e) heartburn

21. The heartburn develops in:
   a) achylia
   b) depressing of esophagus receptors sensitivity to stomach contents
   c) low intragastric pressure
   d) gastroduodenal reflux

22. In insufficiency of esofago-gastric sphincter occurs:
   a) regurgitation of stomach content into the esophagus
   b) decrease in peristalsis of the esophagus;
   c) difficulty in peristaltic activity
   d) Food ingestion disturbances
   e) Congestion and rotting of food in the esophagus.

23. What substances activate stomach secretion?
   a) somatostatin
   b) gastrin
   c) acetylcholine
   d) histamine
   e) adrenaline

24. What substances stimulate stomach motility?
   a) adrenaline
   b) noradrenaline
   c) acetylcholine
   d) motilin
   e) cholecystokinin

25. What of substances stimulate secretion of pancreatic juice?
   a) somatostatin
   b) acetylcholine
   c) secretin
26. What material stimulates secretion and motility of all departments of the gastrointestinal tract?
   a) adrenaline  
   b) acetylcholine  
   c) hydrocortisone  
   d) histamine  
   e) gastrin

27. What hormone inhibits secretion of all departments of the gastrointestinal tract?
   a) hydrocortisone  
   b) heparin  
   c) somatostatin  
   d) vasoactive intestinal peptide  
   e) serotonin

28. The absence of enzymes and hydrochloric acid in gastric juice is called?
   a) achlorhydria  
   b) acholia  
   c) achylia

29. How is the activity of peptases changed in hypoacidity state?
   a) is depressed  
   b) increases  
   c) is not changed

30. How is the activity of peptases changed in hyperacidity state?
   a) is depressed  
   b) increases  
   c) is not changed

31. What changes in the stomach are caused by excessive increase of parasympathetic nerves tone?
   a) reduction of muriatic acid formation
b) increased secretion of gastric juice  
c) reduction of histamine allocation  
d) increased histamine allocation  
e) hydrochloric acid hypersecretions

32. Specify the possible causes of stomach hypersecretion development:  
a) excessive parasympathetic stimulation of the stomach  
b) excessive sympathetic stimulation of the stomach  
c) increased gastrin synthesis  
d) gastrin synthesis deficiency  
e) increase of histamine synthesis in the stomach wall

33. Specify the possible causes of stomach hyposecretion development:  
a) stomach excessive parasympathetic stimulation  
b) decrease in gastrin synthesis  
c) increase of histamine synthesis  
d) decrease of secretin synthesis

34. Specify consequences of achlorhydria:  
a) depressing of secretin synthesis by duodeni mucosa  
b) decrease of gastric juice peptic ferments activity  
c) inhibition of food evacuation from the stomach into the intestine

35. The mechanism of stomach ulcer occurrence in a stress includes:  
a) ischemia of the mucous  
b) hyperemia of the mucous  
c) intensifying of secretion of gastric mucosa  
d) increased endorphines secretion  
e) oppression of regeneration properties of epithelium

36. Absorption of which vitamins is considerably disturbed in acholia?  
a) vitamin A  
b) vitamin B₁  
c) vitamin D  
d) vitamin E  
e) vitamin K

37. Which hormones stimulate mucous and bicarbonate secretion in the stomach?
a) prostaglandin F$_2$α  
b) prostaglandin E  
c) somatostatin  
d) hydrocortisone

38. What mechanisms promote hypersecretion and hyperchlorhydria of gastric juice?
   a) intensifying of parasympathetic influences on a stomach  
   b) intensifying of sympathetic influences on a stomach  
   c) increase formation of a somatostatin  
   d) increase formation of histamine

39. Which substances stimulate insulin development?
   a) glucose  
   b) somatostatin  
   c) lipids  
   d) antibodies

40. The intolerance of milk can cause allergy to:
   a) polysaccharides  
   b) ovalbumin  
   c) β-laktoglobulin  
   d) lactose

41. Stomach ulcer causes:
   a) high gastric acidity  
   b) low activity of factors of protection  
   c) low gastric acidity  
   d) blood-flow intensifying in stomach wall

42. Which hormones reduce the activity of protective factors in the stomach?
   a) gastrin  
   b) secretin  
   c) hydrocortisone  
   d) thyroxine

43. Factors causing the development of stomach ulcer are:
   a) increased formation of mucosa in the stomach
b) hypersecretion of bicarbonates
c) increased formation of Prostaglandins E₁ and E₂
d) depressed regeneration of mucosa

44. **Following factors lead to stomach ulcer:**
   a) blood-flow increase in stomach wall
   b) increase of PgE formation
   c) helicobacter pylori
   d) aspirin use

45. **What hormones stimulate secretion of pancreatic juice?**
   a) somatostatin
   b) acetylcholine
   c) secretin
   d) adrenaline
   e) motiline

46. **What hormones stimulate secretion and motility of all departments of the gastrointestinal tract?**
   a) adrenaline
   b) acetylcholine
   c) hydrocortisone
   d) histamine
   e) gastrin

47. **What hormone inhibits secretion of all departments of the gastrointestinal tract?**
   a) hydrocortisone
   b) heparin
   c) somatostatin
   d) vasoactive intestinal peptide (VIP)
   e) serotonin

48. **The causes of acute pancreatitis are:**
   a) organism overheating
   b) sweet nutrition
   c) cholelithiasis
   d) alcohol abuse
49. *Give pathogenic stages of acute pancreatitis:*
   a) activation of ferments in gland
   b) increase of arterial pressure
   c) increase of pressure in pancreatic ducts
   d) hypovolemic shock

50. *Name the principal cause of chronic pancreatitis:*
   a) mechanical traumas
   b) alcohol abuse
   c) cholelithiasis
   d) heritable disease

51. *More severe clinical course has obstruction of:*
   a) small intestine
   b) large intestine

52. *Malabsorbion is a syndrome caused by:*
   a) increased bile inflow into the intestine
   b) disturbed absorption of nutrients in the small intestine
   c) fasting
   d) endocrine function disturbance in the pancreas

53. *The malabsobtion syndrome is characterised by nutritive absorption disorder in:*
   a) the stomach
   b) the small intestine
   c) the thick intestine
   d) the rectum

54. *Steatoreja is:*
   a) allocation of fat with urine
   b) fat accumulation in blood
   c) allocation of fat with feces
   d) allocation stercobilin with feces
   e) allocation of urobilin with feces

55. *Steatorrhea occurs in:*
   a) gastric juice hypersecretions
   b) acholia
c) high activity of intestinal lipases
d) difficulty of the intestine motility
e) extra protein in human organism

56. Mechanical intestinal obstruction develops in:
   a) spastic stricture or paralysis of intestinal musculature
   b) thrombosis of intestinal wall vessels
   c) paresis of intestinal muscles
   d) tumors and helminthosis an intestine
   e) paralysis of intestinal wall vessels

57. The pathogenesis of intestinal autointoxication is caused by toxic influence of:
   a) products of rotting of protein in an intestine and biogenic amines (pentamethylenediamine, putrescin)
   b) indirect bilirubin
   c) ketone bodies
   d) bile acids
   e) direct bilirubin

Answers:
1a, 2a, 3c, 4d, 5e, 6c, 7a, 8a, 9e, 10b, 11e, 12c, 13d, 14c, 15b, 16a, 17b, 18b, 19b, 20c, 21d, 22a, 23bcd, 24cd, 25bc, 26b, 27c, 28c, 29a, 30b, 31bde, 32ace, 33b, 34ab, 35a, 36acde, 37b, 38ad, 39a, 40c, 41ab, 42c, 43cd, 44cd, 45bc, 46b, 47c, 48cd, 49acd, 50b, 51a, 52b, 53b, 54c, 55b, 56d, 57a.

LITERATURE:

LESSON № 36

TOPIC: SUMMING UP. COMPUTER TESTS.