MINISTRY OF HEALTH OF UKRAINE

ZAPOROZHYE STATE MEDICAL UNIVERSITY

PATHOPHYSIOLOGY DEPARTMENT

PATHOPHYSIOLOGY PRACTICAL CLASSES MANUAL

MODULE 2 Systemic Pathophysiology

Student of _____ group

medical faculty

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Zaporozhye 2015

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This manual was written according to the program of pathophysiology worked out by the Ministry of Health of Ukraine (Kiev, 2006). It includes plan of lectures and practical classes, instruction for every practical class, list of topics for independent studying, information about rating estimation of students' knowledge. Instruction for practical classes include: questions for preparing, description of experimental work, place for results registration and analysis. It can be used by the student of medical faculty while studying pathophysiology.

Навчально-методичний посібник до практичних занять з патофізіології за загальною редакцією завідувача кафедри професора Колесника Ю.М. підготували співробітники кафедри патофізіології Запорізького державного медичного університету: ст. викл., к.мед.н. О.В.Мельнікова. проф. А.В.Абрамов, доцент, к.мед.н. Жулінський В.О.

Посібник підготовлений у відповідності до програми з патофізіології для студентів вищих медичних навчальних закладів III—IV рівнів акредитації (2006 р). і містить плани лекцій та практичних занять, перелік тем для самостійного вивчення, питання до кожного практичного заняття, методику проведення експериментальних робіт, місце для обліку та оцінки результатів проведених експериментів, ситуаційні задачі для розв'язування.

Навчально-методичний посібник може бути використаний під час вивчення патофізіології студентами медичного факультету, які навчаються англійською мовою.

LECTURE PLAN (MODULE 2)

	TOPIC	Hours
1.	WBC pathology. Leukocytosis, leukopenia, leukemia: etiology, pathogenesis, clinical manifestation, laboratory findings.	2
2.	Heart pathology. Arrhythmia, myocarditis: etiology, pathogenesis, clinical manifestation, ECG signs, complications and outcomes.	2
3.	Heart pathology. Ischemic heart disease, heart failure: etiology, pathogenesis, clinical manifestation, ECG signs, complications and outcomes	2
4.	Blood vessels pathology. Arterial hypertension: classification, etiology, pathogenesis, clinical manifestation. Arterial hypotension. Atherosclerosis.	2
5.	Lungs pathology. Respiratory failure: classification, etiology, pathogenesis, clinical manifestation. Shortbreath.	2
6.	GIT and liver pathology. Gastritis, peptic ulcer disease, intestinal obstruction, malabsorption syndrome, syndromes of liver affection: classification, etiology, pathogenesis, clinical manifestation.	2
7.	Kidneys pathology. Glomerulonephritis, nephrotic syndrome, renal failure: classification, etiology, pathogenesis, clinical manifestation.	2
8.	Endocrine system pathology. Clinical manifestation of hypophysis, thyroid and adrenal glands hyper- and hypofunction	2
9.	Nervous system pathology. Violation of sensitivity and motion activity, myasthenia: classification, etiology, pathogenesis, clinical manifestation.	2
10.	Pathophysiology of extreme states. Shock, collapse, coma: classification, etiology, pathogenesis, clinical manifestation, complications and outcomes.	2

PRACTICAL CLASSES PLAN (MODULE 2)

	TOPIC	Hours
1.	RBC pathology. ESR and OER changes. Pathology of hemostasis.	3
2.	RBC pathology. Anemia and erythrocytosis.	3
3.	WBC pathology. Leukocytosis and leukopenia.	3
4.	WBC pathology. Leukemia and leukemoid reaction.	3
5.	**Submodule 4 "Blood pathology"** – checking of practical skills and test control	3
6.	Heart pathology. Arrhythmia, myocarditis.	3
7.	Heart pathology. Ischemic heart disease, heart failure.	3
8.	Blood vessels pathology. Arterial hypertension and hypotension. Atherosclerosis.	3
9.	Lungs pathology. Respiratory failure, shortbreath.	3
10.	**Submodule 5 "Pathology of cardiovascular system and lungs"** – checking of practical skills and test control	3
11.	GIT pathology. Gastritis, peptic ulcer disease, intestinal obstruction, malabsorption syndrome.	3
12.	Liver pathology. Jaundice, hepatic failure, portal hypertension.	3
13.	Kidneys pathology. Glomerulonephritis, nephrotic syndrome, renal failure.	3
14.	Pathology of endocrine system. Hyper- and hypofunction of endocrine glands	3
15.	**Submodule 6 "Pathology of GIT, liver and kidneys"** – checking of practical skills and test control	3
16.	Pathology of nervous system. Violation of sensitivity and motion activity.	3
17.	**MODULE 2 FINAL CONTROL**	2

UNIT 21
RBC PATHOLOGY. CHANGES OF RBC PHYSICO-CHEMICAL PROPERTIES. PATHOLOGY OF BLOOD CLOTTING.

1. Changes of blood circulating volume: hyper- and hypovolemia. Types and causes of appearance.
2. Qualitative changes of RBC. Regenerative and degenerative forms of RBC.
3. Osmotic resistance of erythrocytes: defining factors, normal indices, changes in pathology.
4. Definition of erythrocyte sedimentation rate (ESR), ESR mechanisms. Causes and mechanisms of ESR changes.
5. Vascular-thrombocytic hemostasis in norm. Pathology of vessels and platelets hemostasis.
6. Disturbances of blood coagulation.
7. Syndrome of disseminated intravascular coagulation (DIC-syndrome).

Experimental work.

1. **Determine erythrocytes sedimentation rate (ESR).**

Take 5% Sodium Citrate solution till mark 50 "P" with a capillary from Panchenkov's apparatus and pour on watch crystal. Treat rat's tail and cut off 1-2 mm. Take the first blood drop away with a cotton wool. Then take 2 full capillaries of blood till mark 0 "K". Pour blood on watch crystal and mix with capillary's end (ratio 1:4). Take the mixture of blood and Lithium Citrate to the capillary till mark 0 "K" and put strictly vertically in the stand for 1 hour. After this time note the level of erythrocyte sedimentation in mm. Norm of ESR: women – 2-15 mm/h, men – 1-10 mm/h.

In experimental results: sketch capillary of Panchenkov's apparatus, notate the obtained data for ESR.

In conclusions: describe mechanisms of ESR disturbances, valuate the clinical importance of the method.

2. **Determine osmotic resistance of erythrocytes.**

Prepare dilution of Sodium Chloride out of 1% solution by the following scheme:

No. of test-tube	1	2	3	4	5	6	7	8
Amount of NaCl solution , ml	2,8	2,6	2,4	2,2	2,0	1,8	1,6	1,4
Amount of distilled water, ml	2,2	2,4	2,6	2,8	3,0	3,2	3,4	3,6
Concentration of NaCl, %	0,56	0,52	0,48	0,44	0,40	0,36	0,32	0,28

General amount of solution in each test-tube should be 5 ml. With the help of pipette add to each test-tube 0,02 ml of blood from rat with anemia caused by injection of 1% muriantic phenylhydrazine. Thoroughly mix the contents of all test-tubes, and let it settle for 30 minutes. Then centrifugate the test-tubes for 5 min and note the beginning of hemolysis by the slight blushing of solution (minimal osmotic resistance), and complete hemolysis – by intense red-lacquered color of solution and absence of red sediment in the test-tube (maximal osmotic resistance).

Norms: minimal osmotic resistance – 0,48-0,46% NaCl solution, maximal osmotic resistance – 0,34-0,32% NaCl solution.

In experimental results: sketch the set of test-tubes, note maximal and minimal osmotic resistance, compare results with norm.

In conclusions: describe the mechanisms of disturbance of osmotic resistance, valuate the clinical importance of the method.

3. **Determine the blood coagulation time.**

Cut the rat's tail-end and note the time of blood appearance. Place the blood drop onto the waxed glass and deep the end of waxed glass stick in it every 30 seconds till appearance of first fibrin fibers – it conforms to the beginning of blood coagulation, and stoppage of fibers appearance – the end of coagulation.

Normal time of coagulation beginning – 5 minutes.

CONCLUSIONS

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Task 1

Fill the table "Factors influencing erythrocytes sedimentation rate"

	Increase of ESR	Decrease of ESR
RBC		
WBC		
Blood substances		
Diseases and clinical conditions		

Task 2

Draw degenerative forms of erythrocytes

Teacher's signature

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UNIT 22

RBC PATHOLOGY. ANEMIA CAUSED BY INCREASED HEMOLYSIS AND DISTURBED ERYTHROPOIESIS. ERYTHROCYTOSIS.

1. Anemia definition. Principles of anemia classification. Clinical features and blood count at anemia.
2. Acute posthemorrhagic anemia. Stages of development, blood count at different stages.
3. Chronic posthemorrhagic anemia. Stages of development, blood count at different stages
4. Hemolytic anemias principles of classification. Mechanisms of erythrocytes hemolysis.
5. Acute hemolytic anemias: causes and mechanisms of development, clinical manifestation, blood count.
6. Chronic hemolytic anemias: classification, causes and mechanisms of development, clinical manifestation, blood count.
7. Anemias caused by disturbed erythropoisesis:
    a. iron deficiency anemia;
    b. B12 and folic acid deficiency;
    c. iron refractory anemia;
    d. hypoplastic and metaplastic anemia.
8. Erythrocytosis definition, principles of classification. Clinical features and blood count at erythrocytosis. Mechanism of primary and secondary erythrocytosis development.

Experimental work.

1. **Count the quantity of erythrocytes in animal with experimental anemia.**

    Cut off the rat's tail-end, take blood into melangeur for erythrocytes until mark "0,5" and dissolve until mark "101" with 1 % Sodium Chloride solution (200 times dilution). Shake the melangeur and place the 4-5th drops from melangeur under previously lapped glass of Goryaev's chamber until the appearance of Neuton's rings. In 1 minute, using little magnification in darkened field of vision, count erythrocytes in 80 small squares and calculate their quantity in 1 microliter of blood by formula: $X = (A \times 4000 \times B)/C$, where A – the quantity of counted erythrocytes; B – degree of blood dilution; C – the quantity of counted small squares; 4000 – the volume of liquid over small square. Multiply the obtained number by 10^6 for converting of erythrocytes quantity in liter.

    Norm: man – $4-5 \times 10^{12}$/L, women – $3,9-4,7 \times 10^{12}$/L

    Experiment result:_____

2. **Determine content of hemoglobin in animal with experimental anemia.**

    Pour 0,1 n HCl solution to divided test-tube from Sali's hemometer until round mark. Then take blood into capillary pipette until mark 0,1 ml. Pour blood into the test-tube with 0,1 n HCl solution and wash the pipette with HCl three times. Shake the mixture and leave it for 5 minutes for formation of muriatic hematin. Even the color of liquid with standard color adding distillated water drop-by-drop and mixing with a glass stick. Obtain results in G% hemoglobin. Multiply the obtained number by 10 for converting into SI unit.

    Norm: men – 130-160 g/L; women – 120-140 g/L

    Anemia is diagnosed when hemoglobin amount in the blood is:

    Children from 6 months to 6 years - Hb < 110 g/L; from 6 to 14 years - Hb< 120 g/L;

    Adult man - Hb < 130 g/L; Adult woman - Hb < 120 g/L; Pregnant woman - Hb < 110 g/L

    Experiment result: _____

3. **Calculate the color index (hemoglobin content in RBC).**

    The degree of saturation of each erythrocyte with hemoglobin is determined by color index. It is calculated by formula:

    CI = (content of Hb in g/L x 0,03) / three first numbers of RBC quantity.

    Experiment result: _____

Task 1

Patient A, 54 years, 7th day after surgical operation. **Blood count**: RBC $3,6*10^{12}$/L, Hb 95 g/L, Erythrocyte's hemoglobin content (color index) 0,78 Leukocytes $16*10^9$/L, Platelets $450*10^9$/L **Blood smear**: single anizocytes, poikilocytosis, reticulocytes- 3,8%.

    1. Define RBC state and diagnose the disease.

    2. What stage of disease is diagnosed? How can you prove it?

Task 2

Fill the table describing the stages of acute blood loss.

	Stages of acute blood loss		
	1	2	3
Name the stage			
Time of duration			
Blood circulating volume			
RBC and HB quantity			
Color index			
Reticulocytes quantity			

Task 3

Patient D., 54 years, complains about general weakness, headache, giddiness, troubled sleep, frequent bloody stool.

Blood count: RBC $3,8*10^{12}$/L, Hb 68 g/L, Color index 0,54 Leukocytes $6,1*10^9$/L, **Blood smear:** hypochromic erythrocytes, microcytes, erythrocytes with basophilic granularity, single polychromatophilic normocytes, reticulocytes – 1,8%.

    1. Define RBC state and diagnose the disease and its stage.

    2. Explain the mechanism of its development

    3. Explain the mechanism of low erythrocyte's color index.

Task 4

Patient R., 54 years, arrived to clinic with complains about weakness, shortbreath after minimal physical loading, pain in tongue and fingers. **Blood count**: RBC $1,44*10^{12}$/L, Hemoglobin 66 g/L, Color index 1,4 ;Leukocytes $2,8*10^9$/L, Platelets $100*10^9$/L. **Blood smear**: expressed anizocytosis, poikilocytosis, megaloblasts, megalocytes, erythrocytes with basophilic granularity, hypersegmented neutrophiles, reticulocytes - 0,4%.

   1. Define RBC state and diagnose the disease. Explain the mechanism of its development.
   2. Explain the mechanism of high color index of erythrocytes.
   3. Why neutrophiles have hypersegmented nuclei?

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Task 5

Patient W, arrived to clinic in order to define diagnosis. Complains about weakness, dizziness, loss appetite. **Blood count**: RBC $2,7*10^{12}$/L, Hemoglobin 81 g/L, Color index 1,0; Leukocytes $7,5*10^9$/L, Platelets $230*10^9$/L. **Blood smear**: normochromic RBCs, microspherocytes, reticulocytes - 12%.

   1. Define RBC state and diagnose the disease.
   2. Explain the mechanism of its development.
   3. What type of hemolysis is activated in this case and what is the reason of its activation?

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Task 6

Patient G with chronic respiratory insufficiency. **Blood count**: RBC $6,0*10^{12}$/L, Hb 180 g/L, Color index 0,9 Leukocytes $7*10^9$/L, Platelets $200*10^9$/L. **Blood smear**: non-expressed anizocytosis, reticulocytes - 3,4%.

   1. Define RBC state and diagnose the disease.
   2. Explain the mechanism of its development.
   3. What index can you use to define the state of bone marrow regeneration?

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Task 7

A 12-year-old boy presents in the emergency room with severe chest pain. His mother reports he was doing well until he came down with a respiratory tract infection. **Blood count**: Erythrocytes $3,4*10^{12}$/L,   Hemoglobin 85 g/L, Color index 0,79; Leukocytes $5,6*10^9$/L, Platelets $210*10^9$/L, Reticulocytes 16%. **Blood smear**: anizocytosis, poikylocytosis, sickle cells.

1. Define RBC state and diagnose the disease.

2. What is the most likely cause of pain in this boy?

3. The patients with the disease usually experience anemia but not iron deficiency. Can you explain it?

Task 8

Patient F., 56 years arrived to clinic with stenocardia attack and suspicion of myocardial infarction. **Blood count**: RBC $8,5*10^{12}$/L; Hemoglobin 170 g/L; Color index 0,6; WBC $23*10^9$/L; Platelets $550*10^9$/L. **Blood smear**: hypochromic and polychromatophilic erythrocytes, anizocytosis, poikilocytosis, reticulocytes - 2,1%.

1. Define RBC state and diagnose the disease.

2. Define the state of bone marrow regeneration.

3. How can you explain the changes in WBC and platelets number?

4. Is stenocardia somehow connected with blood count changes? Explain it.

Teacher's signature

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UNIT 23
WBC PATHOLOGY. LEUKOCYTOSIS AND LEUKOPENIA

1. Definition of leukocytic formula. Regenerative and degenerative forms of leukocytes.
2. Leukopenia definition. Causes, mechanisms of development, importance for the organism:
   a) Neutropenia and agranulocytosis
   b) Lymphopenia
3. Leukocytosis definition. Classification of leukocytosis. Physiological leukocytosis causes and mechanisms of development.
4. Pathological leukocytosis different forms. Explain the mechanism of development:
   a) Eosinophilia
   b) Basophilia
   c) Lymphocytosis
   d) Neutrophilia
   e) Monocytosis
5. Definition of neutrophiles nuclear shift in the leukocytic formula. Kinds of neutrophiles nuclear shift.

Experimental work.
  1. **Calculate the leukocytic formula.**
  Place the blood smear stained by Romanovsky under the immersion objective-glass. Count no less than 100 cells moving the smear along the broken line over 3-4 fields of vision. Register numbers with the help of counter. Arrange obtained data as a table.

  2. **Determine the absolute number of each kind of leukocytes.**
  In order to determine absolute number of each kind of leukocytes divide the general quantity of white cells in liter into 100 and multiply by number of percent maintenance of each kind of leukocytes. Arrange the data as a table.

Total WBC =

Leukocytic formula

| | | | Neutrophils | | | | |
	Eosinophils	Basophils	myelo-cytes	juvenile cells	band cells	segmented cells	Lymphocytes	Monocytes
	2-4%	0-1%	0%	0-1%	2-6%	50-70%	20-40%	3-10%
%								
absolute number								

3. Fill the table with the most common causes of leukocytosis and leukopenia

	Eosinophils	Basophils	Neutrophils	Lymphocytes	Monocytes
Increased number					
Decreased number					

Task 1

Patient Tch., 27 years, has laboratory assistant of X-ray cabinet. Last month complains about weakness and frequent bleedings. Blood count: Erythrocytes $1,46*10^{12}$/L, Hemoglobin 42 g/L, Color index 0,85; Erythrocytes sedimentation rate 23 mm/hour, Leukocytes $3,1*10^9$/L, Platelets $97*10^9$/L   Leukocytes count:

Eosinophiles	Basophiles	Neutrophiles				Lymphocytes	Monocytes
		myelo-cytes	juvenile cells	bands cells	segmented cells		
1	0	0	0	2	18	68	11

Blood smear: normochromic RBCs, reticulocytes - 0,1%.
1. Define the state of WBC and diagnose the disease.
2. What is the reason of it?
3. What is the mechanism of RBC and platelets number change?

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Task 2

Patient A, 54 years, is admitted to the cardiological ward with acute myocardial infarction. Blood count: Erythrocytes $3,9*10^{12}$/L, Hemoglobin 110 g/L, Color index 0,85 Leukocytes $23*10^9$/L, Platelets $250*10^9$/L
Leukocytes count

Eosinophiles	Basophiles	Neutrophiles				Lymphocytes	Monocytes
		myelo-cytes	juvenile cells	bands cells	segmented cells		
3	1	1	2	8	64	18	3

Blood smear: normochromic RBC, reticulocytes- 1,3%.
1. Which pathology of WBC is described?
2. Does this pathology have an attitude to acute myocardial infarction? Explain.

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Task 3

A 32-year-old man presents in the allergy clinic with complaints of those of nasal pruritus (itching), nasal congestion with profuse watery drainage, sneezing, and eye irritation. The physical examination reveals edematous and inflamed nasal mucosa and redness of the ocular conjunctiva. He relates that this happens every autumn during "ragweed season."

Blood count: Erythrocytes $3,8*10^{12}$/L, Hemoglobin 120 g/L, Color index 0,86 Leukocytes $10.7*10^9$/L, Platelets $195*10^9$/L
Leukocytes count:

Eosinophiles	Basophiles	Neutrophiles				Lymphocytes	Monocytes
		myelo-cytes	juvenile cells	bands cells	segmented cells		
12	1	0	0	5	55	22	5

1. Which pathology of WBC is described?
2. What is the cause and mechanism of its development?

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Task 4

A 40-year-old man presents in the therapeutic ward with the tuberculous inflammation of the lungs. His complaints now are: cough with bloody phlegm, pain in the chest, general malaise.
Blood count: Erythrocytes $4*10^{12}$/L, Hemoglobin 125 g/L, Color index 0,85 Leukocytes $11.5*10^{9}$/L
  Leukocytes count:

Eosinophiles	Basophiles	Neutrophiles				Lymphocytes	Monocytes
		myelo-cytes	juvenile cells	bands cells	segmented cells		
3	0	0	0	1	35	53	8

1. Which pathology of WBC is described?
2. What is the cause and mechanism of its development?

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Task 5

A child 2 year old was taken to the regular medical examination. His mother says that the child has frequent respiratory infections. At the moment of examination there are no signs of infection.
Blood count: Erythrocytes $3.7*10^{12}$/L, Hemoglobin 115 g/L, Color index 0,87, Leukocytes $9*10^{9}$/L
  Leukocytes count:

Eosinophiles	Basophiles	Neutrophiles				Lymphocytes	Monocytes
		myelo-cytes	juvenile cells	bands cells	segmented cells		
3	0	0	0	2	33	55	7

1. Describe blood indices and evaluate them.
2. Explain the mechanism of the revealed changes.

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Teacher's signature

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UNIT 24
WBC PATHOLOGY. LEUKEMIA AND LEUKEMOID REACTION.

1. Leukemoid reaction: definition, causes of development. Types of leukemoid reactions.
2. Leukemia: definition, the difference between leukemia and leukemoid reaction. Principles of leukemia classification
   a. due to the maturation degree
   b. due to the type of the abnormal cell
   c. due to the total WBC count
3. Etiology of leukemia, stages of leukemia pathogenesis.
4. Common symptoms and signs of leukemia manifestation, mechanisms of their development.
5. Acute leukemias (AML, ALL, undifferentiated type) – clinical and hematological characteristic.
6. Chronic leukemias (CML, CLL) – clinical and hematological characteristic.

Experimental work.

Examine blood smears of patients with different leukemias under microscope.

Sketch and note blood smears in different leukemias and list types of WBC which are typically present

Acute Myelogenous Leukemia

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Acute Lymphogenous Leukemia

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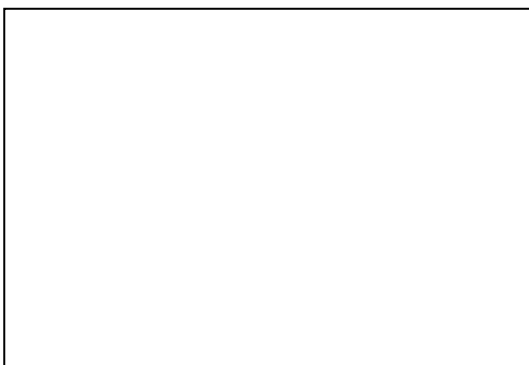
Chronic Myelogenous Leukemia

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Chronic Lymphogenous Leukemia

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Define the pathology of blood in the given clinical tasks and prove your answer.
Put the diagnosis according to all classifications.
Explain the mechanism of development of underlined symptoms and signs.

Task 1

Woman C., 38 years is in a hard state after abortion. Blood count: RBC $4.1*10^{12}$/L   Hemoglobin 129 g/L   ; Color index 0,94 WBC $36*10^9$/L,

| Eosinophils | Basophils | Neutrophils | | | | Lymphocytes | Monocytes |
		myelocytes	juvenile cells	bands cells	segmented cells		
3	1	4	7	9	51	20	5

Blood smear: single promyelocytes, <u>toxic grain in neutrophile's cytoplasm</u>, reticulocytes - 0,9%.

Task 2

Patient K., 55 years, arrived to clinic with complains about general weakness, fever, enlarged regional lymph nodes. Blood count: RBC $2,8*10^{12}$/L,   Hemoglobin 84 g/L, Color index 0,9; ESR 30 mm/hour   <u>WBC $22*10^9$/L,</u>   Platelets $142*10^9$/L

| Eosinophils | Basophils | Neutrophils | | | | Lymphocytes | Monocytes | |
		myelocytes	juvenile cells	bands cells	segmented cells			
1	0	0		1	22	61	3	88

Lymphoblasts – 3%, prolymphocytes – 9%
Blood smear: normochromic RBC, anizocytosis, poikilocytosis, reticulocytes- 0,4%, many <u>"lymphocyte's shadows" (Gumprecht cells)</u>

Task 3

Patient M., 17 years complains of plural <u>subcutaneous hemorrhages.</u> Blood count: RBC $3,6*10^{12}$/L,   Hemoglobin 100 g/L,   Color index 0,83   ESR   50   mm/hour,   WBC $6,5*10^9$/L,   Platelets $60*10^9$/L

| Eosinophils | Basophils | Neutrophils | | | | Lymphocytes | Monocytes | |
		myelocytes	juvenile cells	bands cells	segmented cells			
0	0	0	0	1	19	12	2	33

Lymphoblasts - 63%, prolymphocytes - 4%
Blood smear: normochromic RBCs, anizocytosis, poikilocytosis, reticulocytes - 0,2%.

Task 4

Patient A., 42 years, arrived to clinic with complains of general weakness, fever, <u>pains in bones and joints.</u> Blood count: RBC 2,8*10^{12}/L, Hemoglobin 84 g/L, Color index 0,9; ESR 50 mm/hour; WBC 82*10^9/L, Platelets 142*10^9/L

Eosinophils	Basophils	Neutrophils				Lymphocytes	Monocytes	
		myelocytes	juvenile cells	bands cells	segmented cells			
0	0	6	2	2	12	10	2	34

Myeloblasts - 62%, promyelocytes - 4%

Blood smear: normochromic RBCs, anizocytosis, poikilocytosis, reticulocytes- 0,4%.

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Task 5

Patient G., 34 years, complains of weakness, fatigue, sweatness, <u>pains in left subcostal region.</u> Blood count: RBC 2,9*10^{12}/L, Hemoglobin 120 g/L, Color index 0,85 ESR 50 mm/hour; <u>Leukocytes 93*10^9/L,</u> Platelets 190*10^9/L

Eosinophils	Basophils	Neutrophils				Lymphocytes	Monocytes	
		myelocytes	juvenile cells	bands cells	segmented cells			
9	4	20	20	13	12	10	5	93

Myeloblasts - 1%, promyelocytes - 6% Blood picture: normochromic RBCs, reticulocytes - 0,1%.

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Task 6

A girl, 4 years. Three weeks after quinsy becomes weak and pale. Blood count: <u>RBC 2,9*10^{12}/L,</u> Hemoglobin 89 g/L, Color index 0,9 ESR 50 mm/hour ; WBC 5,9*10^9/L, Platelets 120*10^9/L

Eosinophils	Basophils	Neutrophils				Lymphocytes	Monocytes	
		myelocytes	juvenile cells	bands cells	segmented cells			
0	0	0	0	1	16	31	2	50

Blasts cells 50% Morphological and biochemical signs of blasts cells are similar to lymphoblasts and myeloblasts.

Which type of anemia is present in the patient?

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Teacher's signature

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UNIT 25

SUB-MODULE 4 CONTROL
Checking of practical skills and theoretical knowledge «BLOOD PATHOLOGY».

The final practical class in sub-module 4 consists of estimating student's rating grade. To be allowed to pass sub-module 4 control the student should:
1. get no less than 10 grades during current practical classes;
2. have no missed lectures and practical classes to the current date;
3. have all the practical classes' protocols signed by the teacher and show the lectures notebook.

Students have an opportunity to pass sub-module control once more during two weeks. If sub-module control was not successfully passed in two weeks they will not be allowed to the following lectures and practical classes and rating mark for sub-module 4 control will be "zero".

SUB-MODULE 4 PRACTICAL SKILLS
1. Students should be able to analyze:
- Causality-effective relations (local and systemic changes, pathological and adaptive reactions, specific and non-specific signs) in pathogenesis of typical disturbances in blood system – anemia, erythrocytosis, leukocytosis, leukopenia, leukemia, coagulation disturbances);
- Regularity of peripheral blood count alterations in acute and chronic leukemia.

2. To obtain practical skills:
- in solving problem tasks and tests – definition of etiology, pathogenesis, mechanisms of clinical signs development, principles of diagnosing, prophylaxis and treatment in such clinical conditions: anemia, erythrocytosis, leukocytosis, leukopenia, leukemia, coagulation disturbances;
- to define different WBC forms count with the help of blood count;
- to define hemoglobin blood content and interpret the result;
- to calculate color index and interpret the result;
- to identify regenerative and degenerative forms of RBC and WBC in peripheral blood smear and interpret the results of findings.

UNIT 26
HEART PATHOLOGY. DISORDERS OF HEART RATE. DISEASES OF HEART MUSCLE.

1. Heart functions. Electrical conduction system of the heart.
2. Arrhythmia: definition, principles of classification.
3. Arrhythmias caused by disorders of heart automatism.
4. Arrhythmias caused by conduction abnormalities.
5. Arrhythmias caused by pathology of excitability.
6. Arrhythmias caused by pathology of contractility.
7. Myocarditis: classification, etiology, pathogenesis, clinical manifestation.

Experimental work.
Estimate the changes in given ECG recordings and define the pathology. Describe typical changes of ECG at different arrhythmias.

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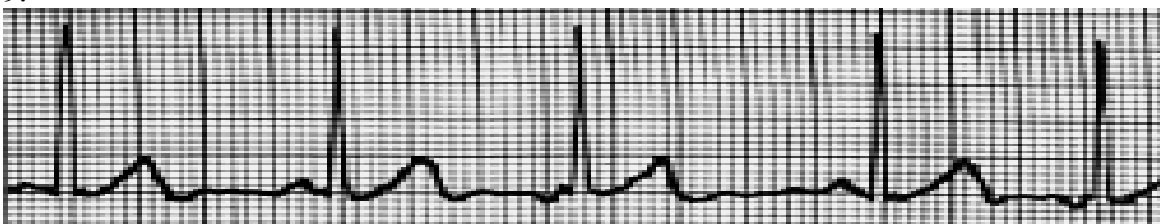

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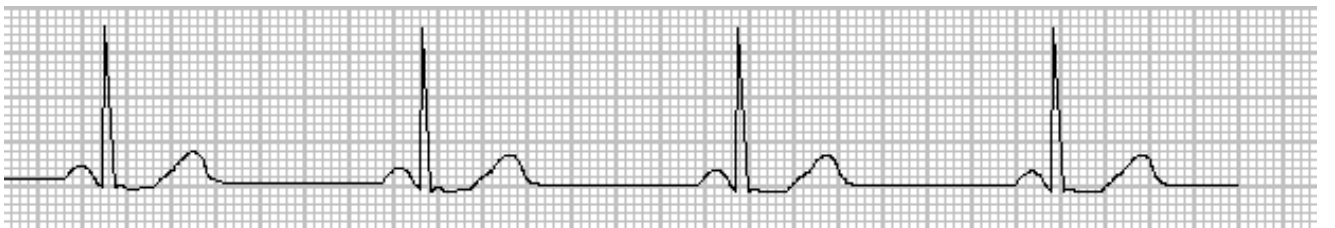
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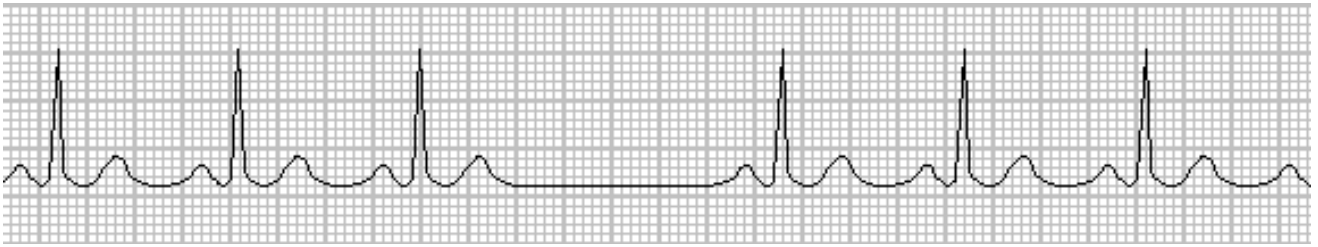
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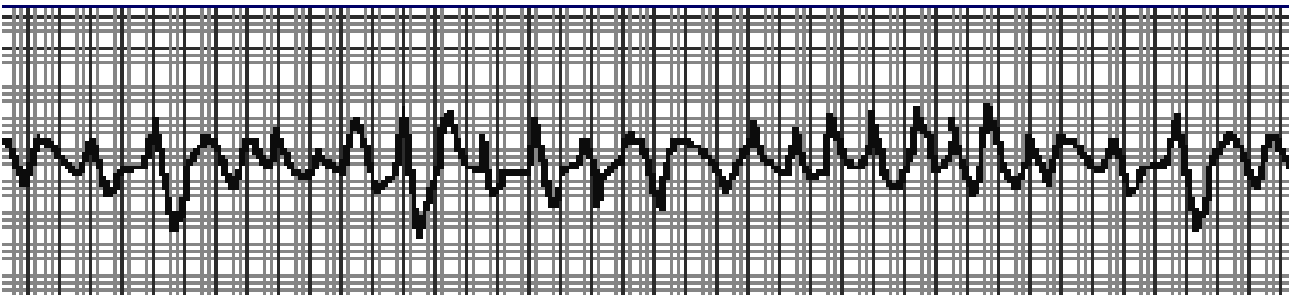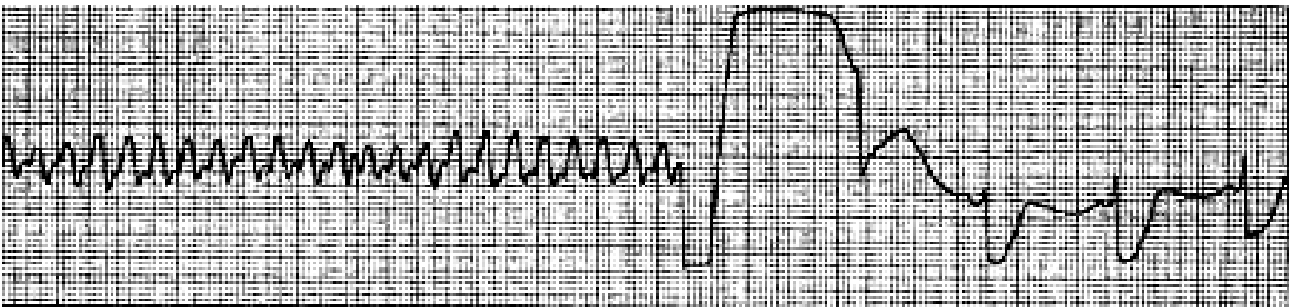
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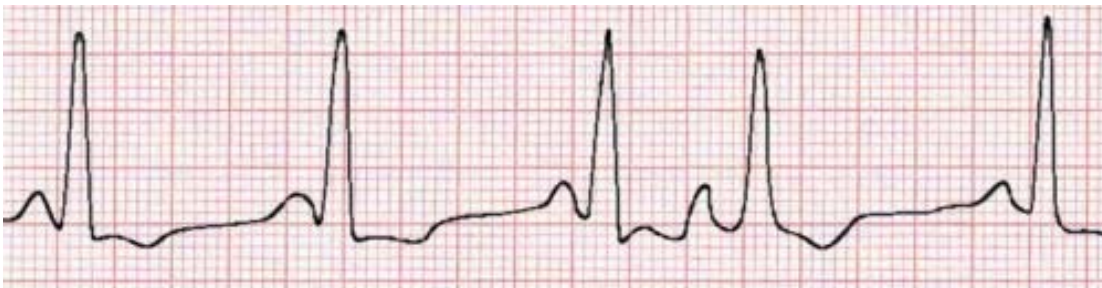
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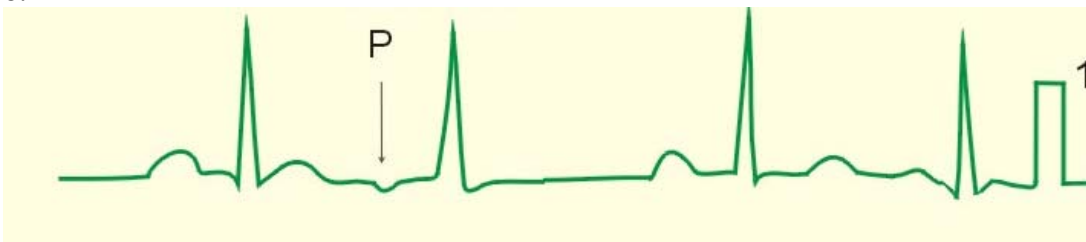
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UNIT 27
HEART PATHOLOGY. HEART FAILURE. ISCHEMIC HEART DISEASE.

1. Heart failure: definition, principles of classification.
2. Clinical manifestation of heart failure. Right-sided and left-sided heart failure.
3. Urgent mechanisms of heart failure compensation.
4. Long-term mechanisms of heart failure compensation.
5. Ischemic heart disease: etiology, pathogenesis, classification.
6. Stenocardia (angina pectoris): etiology, pathogenesis, clinical manifestation, ECG-signs.
7. Myocardial infarction: clinical manifestation. ECG-signs. Reperfusion damage of myocardial cells. Cardiogenic shock.

Experimental work. Calculate the Kerdo index of vegetative homeostasis.

Measure arterial pressure (AP) on the hand, determine the heart rate (HR).

Kerdo index (KI) is calculated by the formula:

$$IK=[1- (HR/AP_{diast})]x100$$

Norm: KI=0

Positive KI (+) testifies to prevalence of sympathetic influence upon the heart, negative (-) testifies to prevalence of parasympathetic influences. KI needs to be calculated in testing state and after physical activity. Hold this experiment with every student of the group.

Arrange obtained results as a table in the protocol, in conclusions explain the change of KI after physical activity.

Student's name	Arterial pressure	Heart rate	Kerdo index

CONCLUSION

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Task 1

Patient M., 46-year-old after intensive physical work felt severe pain behind the sternum. He experienced earlier the episodes of pain with such localization. Usually there was a relief from pain in the rest. He stopped his work, but the pain was still severe. In a few hours shortness of breath and coughing with abundant mucus appeared. The patient was hospitalized in cardiological department.

Clinical examination of the patient: pale skin with cyanotic tint. Moist rales are auscultated over left and right lung. Breath frequency – 42 per minutes. Heart rate – 120 bpm. BP – 110/70 mmHg. Arterial blood oxygenation is 85% (normal 95 to 100%).

1. What is the most likely diagnosis?
2. Which type of heart failure has developed in the patient?
3. Explain the mechanism of low blood oxygen saturation and moist rales.

Task 2

A 56-year-old woman complains of dyspnoe in the rest which increases with physical activity, legs edema, pains in the right subcostal region.

Clinical examination: pale skin with cyanotic tint, enlargement of the liver, fluid accumulation in the peritoneal cavity. Breath frequency – 38 per minute, heart rate – 136 bpm. Borders of the heart are enlarged.

1. Which type of heart failure has developed in the patient?
2. Explain the mechanism of legs edema and liver enlargement.

Task 3
Fill the table: "Urgent mechanisms of heart failure compensation".

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2.
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4.
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Task 4

A 58-year-old male teacher notices the sudden onset of "chest tightness" when he walks upstairs. The pain, which is localized over the sternum, goes away when he sits down. He does not experience any pain or discomfort at other times. He has mild hypertension, for which he is on dietary therapy. His cholesterol level is elevated. He does not smoke.

1. What is the most likely diagnosis?
2. What is the most likely mechanism for these symptoms?
3. What are the complications and prognosis for this patient?

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Task 5

A 40-year-old man presents in the emergency department complaining of substernal chest pain that is also felt in his left shoulder. He is short of breath.

His blood pressure is 148/90 mm Hg and his heart rate is 110 bpm. Body temperature $37,3^0$ C. His ECG shows an ST-elevation with T-wave inversion. He is given aspirin, morphine, and oxygen. Blood tests reveal elevated levels of creatin kinase and troponin.

1. What is the probable cause of the man's symptoms?

2. Explain the origin of the left arm pain, fever, and increased heart rate.

3. What is the significance of the ST-segment changes and elevation in creatin kinase and troponin?

4. Relate the actions of aspirin, morphine, and oxygen to the treatment of this man's condition.

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Task 6

A 26-tear-old patient presents in the infectious department complaining of throat pain, coughing, shortness of breath, shooting pain in the heart. The throat is hyperemic, tonsils are covered with fibrinous coating. His heart rate is 92 bpm, body temperature 39^0 C, breath rate – 25 per minute. Diphtheria diagnosis is supposed.

ECG shows multiply ventricle extrasystoles, amplitude of the ECG is lower than normal. Ultrasound research of the heart shows dilatation of left ventricle. Laboratory findings: total WBC - $15*10^9$/L (neutrophiles -80%), ESR – 18 mm/hour, antibodies to diphtheria's toxins are found in high amount.

1. What heart pathology has developed in the patient?

2. Which clinical signs can prove your answer?

3. Describe the connection between infectious disease and heart pathology.

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UNIT 28
BLOOD VESSELS PATHOLOGY.
ARTERIAL HYPERTENSION AND HYPOTENSION. ATHEROSCLEROSIS.

1. Mechanisms of arterial pressure regulation.
2. Definition of arterial hypertension. Classification of arterial hypertension.
3. Causes of primary and secondary hypertension development.
4. Pathogenesis of hypertension.
5. Signs, symptoms and complications of hypertension; general principles of treatment.
6. Arterial hypotension: types, causes, pathogenesis. Acute decrease of blood pressure.
7. Atherosclerosis definition, etiology, risk factors.
8. Pathogenesis and clinical manifestation of atherosclerosis.

Experimental work

To examine the indices of cardiohaemodynamics in the volunteer students of your group before and after hypoxic loading. Hypoxic loading is done as voluntary stopping of breathing for the maximal time.

	units	normal value`	Before hypoxic loading	After hypoxic loading
Heart rate	bpm	60 - 80		
Heart stroke volume	ml	60 - 75		
Heart stroke index	ml/m^2	47 - 60		
Minute blood volume	ml/min	4500 - 5000		
Heart index	ml/(min*m^2)	3600 - 4000		
Stroke volume speed	ml/sec	220 - 382		
Systolic arterial pressure	mm Hg.	110 - 120		
Diastolic arterial pressure	mm Hg	70 - 80		
Left ventricle capacity	watt	4,2 - 4,6		
Energy consumption	watt/L	9,0 - 12,5		
Total peripheral vascular resistance	din*second*cm^{-5}	1400 - 2500		
Specific peripheral vascular resistance	conventional unit	28,6 - 39,5		

The stages of hypertension

Category	Systolic BP (mm hg)	Diastolic BP (mm hg)
Normal BP	Below 130	Below 85
High-normal BP (pre-hypertension)	130-139	85-89
Stage 1 (mild) hypertension	140-159	90-99
Stage 2 (moderate) hypertension	160-179	100-109
Stage 3 (severe) hypertension	180 or higher	110 or higher

Task 1

A 47-year-old man, who is an executive in a law firm, had his blood pressure taken at a screening program and had been told that his pressure was 144/90 mm Hg. His father and older brother have hypertension, and his paternal grandparents had a history of stroke and myocardial infarction. The patient enjoys salty foods and gained about 7 kg in the past year. His physical activity is very low: he prefers watching television.

1. According to classification into what category does the patient's blood pressure fall?
2. What are his risk factors for hypertension?
3. Explain how an increased salt intake might contribute to an increase in blood pressure.
4. What type of treatment would you suggest to the patient?

Task 2

A 36-year-old woman enters the clinic complaining of headache and not feeling well. Her blood pressure is 175/90 mm Hg. Her renal tests are abnormal, and follow-up tests confirm that she has a stricture of the left renal artery.

1. What type of arterial hypertension does the woman have?
2. Explain the physiologic mechanisms underlying her blood pressure elevation.

Task 3

A group of people was in the elevator, when the electricity was turned off and elevator was stopped. A 21-year-old man suddenly felt extreme weakness, nausea and palpitations. He turned pale, his skin was covered with cold sweats and he was near-loss of consciousness. Blood pressure – 80/50 mmHg.

1. Explain the mechanism of blood pressure fall in this patient.
2. Which other causes of low blood pressure can you name?

Task 4. Fill the table "Atherosclerosis: risk factors"

Major Risk Factors	Minor Risk Factors

Task 5

A 56-year-old man complains of constant feeling of "chest tightness". When patient's BP increases he feels severe substernal pain with irradiation to left shoulder. He is smoking for 35 years. His body mass index is 33,5.

Clinical examination of the patient: heart rate – 76 bpm, BP 150/80 mm Hg.

Laboratory data: total blood cholesterol level 6,2 mmol/L, LDL – 4,1 mmol/L, HDL 0,7 mmol/L.

Normal indices of lipid metabolism:
blood cholesterol level - < 5,2 mmol/L, LDL - <3,36 mmol/L, HDL - >1,15 mmol/L

1. Define the pathology. Prove your answer.
2. What is the role of LDL and HDL in pathogenesis of the revealed disorders?
3. Describe pathogenesis of the revealed pathology?
3. Which possible complications of atherosclerosis do you know?

**Teacher's signature**

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UNIT 29
PATHOLOGY OF LUNGS. RESPIRATORY FAILURE, SHORTBREATH.

1. Respiratory failure definition, classification. Intrapulmonary and intrapulmonary causes of development.
2. Causes and mechanisms of development of:
      a. disturbances of alveolar ventilation;
      b. disorders of blood perfusion in the lungs;
      c. mismatching of ventilation/perfusion ratio;.
      d. impairment of gases diffusion in the lungs.
3. Clinical manifestation of respiratory failure: hypoxemia and hypercapnia signs and symptoms.
4. Pulmonary edema: mechanisms of development, signs and symptoms.
5. Short breath. Principles of classification.
6. Cerebral dyspnoea, periodic and agonal breathing: causes and mechanisms of development.
7. Causes and mechanisms of lungs, cardiac and hematic shortbreath development.
8. Asphyxia: stages and mechanisms of development.

Experimental work.

To study the indices of lungs function in the patients with pulmonary diseases and volunteer students and evaluate them.

Normal spirogram

Normal lung volumes
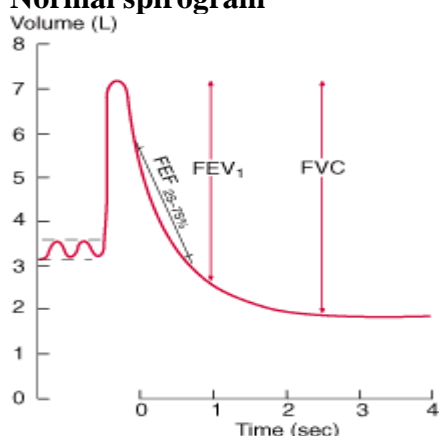
FEV_1 = forced expiratory volume in the 1st second of forced vital capacity maneuver; $FEF_{25-75\%}$ = forced expiratory flow during expiration of 25 to 75% of the FVC; FVC = forced vital capacity (the maximum amount of air forcibly expired after maximum inspiration).
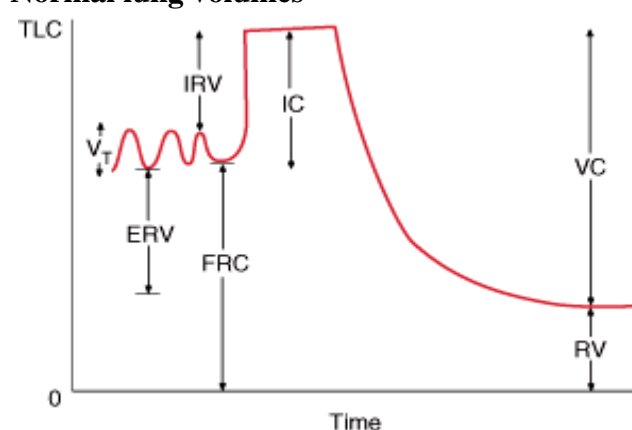
TLC = total lung capacity; VT = tidal volume; ERV = expiratory reserve volume; IRV = inspiratory reserve volume; FRC = functional residual capacity; IC = inspiratory capacity; VC = vital capacity; RV = residual volume; FRC = RV + ERV; IC = VT + IRV; VC = VT + IRV + ERV.

Difference between obstructive, restrictive and mixed disorders of alveolar ventilation

Measure	Obstructive Disorders	Restrictive Disorders	Mixed Disorders
FEV1/FVC	↓	Normal or ↑	↓
FEV1	↓	Normal ↑ ↓	↓
FVC	Normal or ↓	↓	↓
TLC	Normal or ↑	↓	↓
RV	Normal or ↑	↓	Normal ↑ ↓

Task 1

Patient A has been working at cement factory for 30 years. He complains of inability to do his usual work due to short breath which appears during physical load. Clinical examination: skin is pale, stiff breathing with dry crepitation (rales) all over the lungs are heard. Lung's X-ray picture: pneumosclerosis. Arterial blood oxygen saturation - 74 %.

1. Disturbance of which stage of external breathing is observed in the patient?
2. Why short breath develops only during physical load?

Task 2

Patient C ill with bronchial asthma suffers from frequent attacks of suffocation (asthmatic fit) without any apparent reason. The breathing become hard during this attack, it is accompanied by cough with little amount of viscous phlegm. Whistling rales during exhalation are heard.

1. What type of short breath is characteristic for such lungs pathology? Explain the mechanism of its development.
2. What type of respiration insufficiency is observed in the patient?

Task 3

Patient B. was treated in neurological ward with diagnosis "cerebral stroke". His state was hard. Breathing had a periodic pattern of Cheyne-Stokes. Two days later the type of breathing changed to Bioth type.

1. What is the leading factor in periodic breathing development?
2. How can you estimate the changes of patient's breathing? Do they have good prognostic features?

Task 4

Patient R, 20 years suffering from kidneys disease was taken to hospital in pre-coma state. Short breath with high frequency of respirations was observed. In spite of treatment the state of the patient get worse and coma developed. Now patient is unconsciousness. The breath is characterized with regularly deep inhalations and exhalations.

1. What type of short breath has developed in the patient and why?
2. What type of short breath has appeared in coma state?

Teacher's signature

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UNIT 30
SUB-MODULE 5 CONTROL «Pathology of cardiovascular system and lungs ».
Checking of practical skills and theoretical knowledge

The final practical class in sub-module 5 consists of estimating student's rating grade. To be allowed to pass sub-module 4 control the student should:
1. get no less than 12 grades during current practical classes;
2. have no missed lectures and practical classes to the current date;
3. have all the practical classes' protocols signed by the teacher and show the lectures notebook;
4. successfully passed submodule 4 control.

Students have an opportunity to pass sub-module control once more during two weeks. If sub-module control was not successfully passed in two weeks they will not be allowed to the following lectures and practical classes and rating mark for sub-module 5 control will be "zero".

SUB-MODULE 5 PRACTICAL SKILLS
1. Students should be able to define:
- typical disturbances of blood circulating system: heart failure, circulatory failure, arrhythmia, arterial hypotension and hypertension, arteriosclerosis, atherosclerosis using modern classifications;
- respiratory failure using indices of blood gases and modern classifications;

2. Students should be able to analyze:
- changes of the cardiohaemodynamic indices in the pathology;
- changes of spirogram and lungs volume.
- causality-effective relations, pathological and adaptive features in pathogenesis of the following diseases: heart failure, circulatory failure, arrhythmia, ischemic heart disease, cardiogenic shock, arterial hypotension and hypertension, arteriosclerosis, atherosclerosis, respiratory failure, lungs edema, asphyxia and shortbreath;
- the role of disturbances of alveolar ventilation, disorders of blood perfusion in the lungs, mismatching of ventilation/perfusion ratio and impairment of gases diffusion in the lungs

3. To obtain practical skills:
- in solving problem tasks and tests – definition of etiology, pathogenesis, mechanisms of clinical signs development, principles of diagnosing, prophylaxis and treatment in such clinical conditions: heart failure, circulatory failure, arrhythmia, ischemic heart disease, cardiogenic shock, arterial hypotension and hypertension, arteriosclerosis, atherosclerosis, respiratory failure, lungs edema, asphyxia and shortbreath.

UNIT 31
GIT pathology. Gastritis, peptic ulcer disease, intestinal obstruction, malabsorption syndrome

1. The main symptoms of GIT functions violations.
2. The violation of digestion in the stomach:
   a. types of gastric secretion;
   b. the role of gastric mucosal barrier damage in stomach pathology development.
3. Gastritis: classification, etiology, pathogenesis, clinical symptoms.
4. Peptic ulcer disease: etiology, pathogenesis, clinical symptoms, complications, principles of therapy.
5. The violation of the digestion in the intestines: general mechanisms of pathogenesis.
6. Intestinal obstruction: types, mechanisms of development, clinical manifestation, complications.
7. The malabsorbtion syndrome: types, clinical manifestation, complications.

Experimental work.
To analyze the results of gastric intubation and reveal the probable disturbances in stomach and intestines digestion.

Normal indices of gastric secretion

	Volume (ml.)	General acidity	Free HCl	Combined HCl	Pepsin
fasting secretion	less than 50	up to 40	up to 20		0-20
basal secretion	50-100	40-60	20-40	10-15	20-40
stimulated secretion	50-100	40-60	20-40	10-15	21-45

The result of **patient 1** gastric intubation:

	Volume (ml.)	General acidity	Free HCl	Combined HCl	Pepsin
fasting secretion	100	60	30	20	15
basal secretion	120	80	60	10	30
stimulated secretion	140	100	50	30	50

The result of **patient 2** gastric intubation:

	Volume (ml.)	General acidity	Free HCl	Combined HCl	Pepsin
fasting secretion	10	30	0	10	10
basal secretion	0	0	0	0	0
stimulated secretion	20	32	10	10	5

In conclusions you should analyze the given indices and describe the mechanisms of disturbances
CONCLUSION

Task1

Patient H. 56 years old suffers from diabetes mellitus, cholelithiasis and chronic cholecystitis. He complains of the pain in the epigastric area shortly after fat or pungent food. Also he has eructation, qualms, wind and diarrhea. Blood count: during worsening – increase of the ESR, neutrophilic leukocytosis, hypo- and dysproteinemia (increase of the globulins), moderate hyperglycemia and glucosuria, moderate increase of the pancreas's enzymes (tripsin and amylase).

1.   Do these symptoms confirm pancreas' function disorders?
2.   What is the possible mechanism of this pathology development?
3.   What is the pathogenesis of the diabetes mellitus and its progress in this case?

Task 2

Patient G. 75 years old, complains of the pain in the abdomen and dyspepsia disorders: eructation, sickness, feeling of the fulness in the stomach. Anamnesis: during 8 years patient had been suffered from rheumatoid polyarthritis. Lately he took acetylsalicylic acid and prednisolone without doctor's prescription because of worsening. Defect of the gastric's mucous was found after gastroscopy (erosion 0.5 X 0.5 cm).

1.   What pathology of the gastrointestinal tract was described in the task? Explain your answer.
2.   What is the mechanism of the gastric's mucous defect development?
3.   What are the mechanisms of the dyspepsia disorders in this case?

Task 3

Patient Z. 18 years old was cured for the "sepsis" 6 month ago. He complains of the appetite decrease, pronounced dyspepsia disorders, bad tolerance of the milk and tendency to diarrhea. Investigation of the gastric secretory function was done. Pepsin and free chlorhydric acid weren't found in the gastric juice.

1.   What pathology of the gastrointestinal tract was described in the task?
2.   Has this pathology any functional bonds with old disease?
3.   What is the pathogenesis of the milk's intolerance?

Task 4

Patient E. 35 years old suffers from Cushing's disease with hypercortisolism syndrome. She complains of the pain in the epigastric area, heartburn and vomit with gastric juice on an empty stomach in the seldom cases. Hyperacidity was found after fractional gastric probe with stimulator of the secretion (histamine).

1. What kind of the acidity determined increase of the gastric juice's acidity in this case?

2. What is the pathogenesis of the GIT pathology, according to previous disease?
3. What is the possible pathogenetic mechanism of the hyperacidity in this case?

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Task 5

Patient E. 40 years old suffers from neurotic aerophagia. She complains of feeling of the weight, pressure and repletion in the epigastric area. Also she has frequent and intense eructation with air, pain in the heart area, increasing after food and decreasing after eructation. Big air bubble in the stomach and high standing of the left diaphragm's cupola were found after X-ray examination.

   1. What disturbance of the stomach's function was described in this case?
   2. What is the cause of the cardial pain?
   3. Explain the mechanism of the eructation development in this patient.

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Task 6

Patient E. 55 years old was delivered to the surgery department in a bad condition. She complains of the intense surround pain, indomitable vomit without relief. Examination: cyanosis of the skin, tachycardia, decreasing of the BP, tongue with white spot, swollen stomach. Anamnesis: patient took a fall from stairs and took contusion of the abdomen yesterday. Blood count: leukocytosis with shift to the left, increase of the hematocrit, increase of the amylase's level. Patient had been moved to the resuscitation department and intensive antienzymatic therapy was started.

   1. What pathology of the gastrointestinal tract was described in the task? What is the cause of its development?
   2. What are the causes and pathogenesis of this pathology?
   3. Explain pathogenetic sense of the antienzymatic therapy?

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Task 7

Patient G. 68 years old was hospitalized to the surgery department with diagnosis "suppurative appendicitis, peritonitis". Examination: peristalsis of the intestines is absent, pain, flabbiness, sickness, symptoms of the intoxication (decrease of the BP, mild pyrexia, vomit). Diagnosis "bowel obstruction, bowel autointoxication" was established.

   1. What is the mechanism of the gastrointestinal tract's function disorders during peritonitis?
   2. What kind and what is the cause of the bowel obstruction in this case?
   3. What is the mechanism of the bowel autointoxication development?

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UNIT 32
Liver pathology. Jaundice, hepatic failure, portal hypertension.

1. The role of the liver in the organism. Syndromes of liver affection.
2. Normal bilirubin metabolism.
3. Jaundice: classification, mechanisms of development, clinical and laboratory findings in
   a. hemolytic jaundice;
   b. hepatic jaundice;
   c. obstructive jaundice.
4. Cholemia syndrome: mechanisms of development, clinical and laboratory findings.
5. Hepatic failure: classification, mechanisms of development, clinical and laboratory findings. Hepatic encephalopathy.
6. Portal hypertension syndrome: classification, mechanisms of development, clinical manifestation.
7. Complications of portal hypertension syndrome: ascites, splenomegaly, portosystemic shunts.
8. Hepatorenal and hepatolienal syndromes: mechanisms of development, clinical and laboratory findings.

Experimental work.
To analyze the indices of bilirubin metabolism in different types of jaundice.

Normal indices of bilirubin metabolism

Index	Blood (μmol/L)	Urine	Feces
Total bilirubin	5,1 – 17		
Direct (conjugated) bilirubin	1,7 – 5,1		
Indirect (unconjugated) bilirubin	3,4 – 12		
Urobilinogen		present	
Stercobilinogen)			present

Index	Hemolytic jaundice	Hepatic jaundice	Obstructive jaundice
Synonym			
Synonym			
Total bilirubin			
Direct bilirubin			
Indirect bilirubin			
Urine			
Feces			

Task 1.

Patient C., is treated in the clinic with diagnosis chronic hepatitis complicated by liver cirrhosis. The patient is weak, complaints about appetite loss and waste of weight. His skin is dry and pale with yellow tint and small hemorrhages on it. His belly is enlarged because of ascites. Subcutaneous veins are enlarged and well seen on the skin of belly. Concentration of proteins in the blood is lower than normal.

1. What pathology has developed in this patient and what are the reasons of it?
2. Is there connection between ascites and low blood protein level?
3. Why subcutaneous veins on the skin of belly are enlarged?

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Task 2

Patient R., complains of acute pain in the right subcostal region, itching and icteric skin, plural small hemorrhages on the skin, BP – 110/80 mmHg, heart rate – 58 bpm. Laboratory analysis of feces show increased amount of non-digested lipids. Analysis of blood coagulation – decreased prothrombine amount.

1. What is the mechanism of steatorrhea (presence of lipids in feces) development?
2. Is there pathogenic connection between steatorrhea and skin haemorrhages?
3. Explain the mechanism of decreased blood coagulation, changes of blood pressure and heart rate indices.

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Task 3

Patient L., complains of itching, icteric skin and scleras. Conjugated and non-conjugated bilirubin level is increased in the blood. Conjugated bilirubin is present in urine. Protrombin level is decreased in the blood. Patient was treated with vitamin K injections for 5 days. After last vitamin K injection prothrombine level increased on 40 %.

1. Does this result prove disturbances in protein-synthesizing liver function?
2. Can non-conjugated bilirubin be present in the urine? Why?
3. Which type of jaundice is present in this patient?

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Task 4

Patient K., 14 years old arrived to clinic with complaints about general weakness, pain in left subcostal region. Icteric skin had appeared in her from the childhood. Three months ago jaundice has strengthened and pains in liver region appeared. Blood analysis showed increased level of non-conjugated bilirubin, urine and feces are darkly colored.

1. Which type of jaundice is present in this patient?
2. What bilirubin level will be increased in this patient?
3. What is the reason of dark color of urine and feces?

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UNIT 33
Kidneys pathology. Glomerulonephritis, nephrotic syndrome, renal failure.

1. Quantitative and qualitative violations of uropoiesis: mechanisms of development, laboratory findings.

2. Pathological components of the urine: kinds of disorders, mechanisms of proteins, erythrocytes, leukocytes, renal casts and glucose appearance in the urine of:

3. Glomerular diseases classification. Etiology and pathogenesis of glomerulonephritis.

4. Nephritic and nephritic syndrome: clinical and laboratory findings, mechanisms of development.

5. Renal failure: etiology, classification, stages of development.

6. Clinical manifestation of renal failure: renal symptoms, extra-renal manifestations. Pathogenesis of renal hypertension.

7. Uremia syndrome: etiology, pathogenesis, clinical manifestation.

Experimental work.
   1. **Determine sugar content in urine.**

   Place a pinch of reactive (copper sulfate with sodium carbonate) on the object-plate with a glass stick. Put 2-3 drops of probationer's urine above. Then the object-plate is warmed up on fire until the urine begins to boil. The character and intensiveness of color indicates: blue color – no sugar, green – traces, yellowish green – about 0,5%, yellow – about 1%, brown – about 2%, brick-red – about 4% and more.

   2. **Determine content of protein in urine.**

   Pour about 1 ml of concentrated nitric acid to the test-tube, carefully add urine probe. If protein is present – the white ring appears on the border of 2 liquids. If the content of protein in the urine is 0,033% - the ring appears in 2-3 minutes. If the ring appears earlier, you need to dilute the urine 2-4-8-16 etc. times and then multiply 0,033% by the degree of urine dilution.

   3. **Determine presence of blood in urine.**

   Add 5-6 drops of 10% KOH to 3-4 ml of urine, boil. The sediment appears, that obtains brown color in the presence of erythrocytes.

   4. **Determine presence of acetone in urine.**

   Add 3-4 drops of 5% sodium nitroprusside solution and 0,5 ml of 10% caustic soda solution to 5-6 ml of urine until appearance of red or pink color. Then add 0,5-1,0 ml of ice-cold acetic acid. The color stays if the test is positive.

   In conclusions: describe possible mechanisms of determined disorders. Valuate clinical importance of investigated indices.

CONCLUSION

Task 1

The mother of the 3-year-old boy complains of his weakness, fatigue, polyphagia and polydipsia. Clinical examination of inner organs shows absence of pathology. Urine analysis: daily diuresis about 3 l., urine's specific gravity - 1,020 to 1,038. Daily glucose urine excretion 1,2 g. Blood analysis: glucose plasma level 3 mmol/L. Clinical examination of boy's brother (1,5 years) revealed the same clinical picture.

1. What kidney's function is disturbed in the patient?
2. What is the mechanism of glucosuria in this case?
3. What is the reason of glucosuria in this case?

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Task 2

An 8-year-old boy is brought to the pediatrician's office with a 2-day history of malaise, fever of 38.8°C, nausea, and vomiting. His mother reports that he has decreased urine output and that his urine is a dark, smoky color. His blood pressure is slightly elevated, and there is some swelling of his hands and feet and around his eyes. He has been in good health except for a sore throat a week or so ago.

Urine analysis: proteins 1,2 g/L, leukocytes 3-8, erythrocytes 40-100 (in the field of vision), hyalinic cylinders. Glomerular filtration – 56 ml/min (normally 110-125 ml/min).

1 What kidney's function is disturbed in the patient? What is the most likely diagnosis?
2 What mechanism is involved?
3 What is the usual clinical course?

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Task 3

Patient A., 45 years arrived the clinic with acute abundant gastric bleeding. The patient is in a hard state. BP– 85/60mmHg, daily diuresis less than 250 ml. Blood analysis: blood nitrogen – 62 mmol/L (norm 14,3 – 28,5 mmol/L), blood urea – 36 mmol/L (norm 2,5-8,3 mmol/L).

1. What kidney's pathology has developed in this patient?
2. What kidney's function is disturbed in the patient? What are the signs of such disturbance?
3. Describe the mechanism of oliguria development.

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Task 4

Patient G., 43 years was admitted to clinic a hard state. She is unconsciousness, breath smells with ammonia. She suffers from kidney disease for 16 years. Clinical examination: edema of face and legs, enlarged liver, BP –190/120 mmHg., blood nitrogen – 148 mmol/L, glomerular filtration – 12 ml/min., daily diuresis 360 ml, urine specific gravity - 1003-1007.

Blood analysis: RBC – $2,4x10^{12}$/L, Hb – 68 g/L, color index 0,85, WBC – $5,6x10^{9}$/L. leukocytic formula – without changes. In a blood smear: anizocytosis, poikilocytosis.

1. What kidney's pathology has developed in the patient?
2. Are there signs of uremia in this patient?
3. Describe blood picture and explain the reason of the changes.

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Teacher's signature

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UNIT 34
Pathology of endocrine system. Hyper- and hypofunction of endocrine glands

1. General mechanisms of endocrine system functions disturbances. General principles of endocrine diseases treatment
2. Anterior pituitary lobe disorders. Etiology, pathogenesis, clinical manifestations of:
    a. pathology of growth hormone secretion;
    b. pathology of ACTH, TSH, FSH secretion;
    c. panhypopituitarism.
3. Posterior pituitary lobe disorders (diabetes insipidus, syndrome of inappropriate ADH secretion).
4. Disturbances of thyroid gland function. Etiology, pathogenesis, clinical manifestations of hyperthyroidism and hypothyroidism.
5. Pathology of parathyroid glands. Etiology, pathogenesis, clinical manifestations of parathyroid hormone inadequate secretion.
6. Hypofunction of adrenal cortex. Etiology, pathogenesis, clinical manifestations of Addison's disease and secondary adrenal insufficiency.
7. Hyperfunction of adrenal cortex. Etiology, pathogenesis, clinical manifestations of adrenal virilism, Conn's syndrome, Cushing's syndrome.
8. Pathology of adrenal medulla.
9. General adaptation syndrome and stress. Stages of development. Biological role of general adaptation syndrome.

Experimental work
To solve situational problems.

Task 1
A 20-year-old man complains of excessive thirst and abundant urination (up to 10 L for 24 hours). Urine specific gravity -1005. Blood glucose concentration is normal, glucose is absent in the urine.
1. What endocrine pathology can be suggested?
2. Explain the mechanism of polyuria, hyposthenuria, polydypsia.

Task 2
A patient aged 23 complains of headache, changes of his appearance (increase of feet, hands, nose and lips size), hoarsening of the voice, worsening of the memory. The disease has begun 3 years late without any cause. Increase of superciliary arches, nose and tongue were found during physical examination. Blood glucose level is 6,9 mmol/L.
1. What endocrine pathology can be suggested?
2. Explain the mechanism of the given symptoms.

Task 3
A 34-year-old woman complains of increased irritability, perspiration, weakness, loss of body weight, tremor of the limbs, increased heart rate and exophtalmia. Clinical examination: body temperature 37,5^{0}C, heart rate 122 bpm, thyroid gland is increased in size. Anti-thyroid antibodies were found during blood analysis.
1. What diagnosis would this woman's history, physical, and laboratory tests suggest?
2. Explain the mechanism of the given symptoms and the role of anti-thyroid antibodies in the endocrine disorder development.

Task 4

A 76-year-old woman presents with weight gain, subjective memory loss, dry skin, and cold intolerance. On examination, she is found to have a multinodular goiter. Laboratory findings reveal a low serum T4 and elevated TSH.

1. What diagnosis would this woman's history, physical, and laboratory tests suggest?
2. Explain the possible relationship between the diagnosis and her weight gain, dry skin, cold intolerance, and subjective memory loss.

Task 5

A 45-year-old woman presents with a history of progressive weakness, fatigue, weight loss, nausea, and increased skin pigmentation (especially of creases, pressure areas). Her blood pressure is 120/78 mm Hg when supine and 105/52 mm Hg when standing. Laboratory findings revealed a serum sodium level of 120 mmol/L (normal is 135 to 145 mmol/L); potassium level of 5.9 mmol/L (normal is 3.5 to 5.0 mmol/L); low plasma cortisol levels, and high ACTH levels.

1. What diagnosis would this woman's clinical features and laboratory findings suggest?
2. Would her diagnosis be classified as a primary or secondary endocrine disorder?
3. What is the significance of her darkened skin?

Task 6.

Patient F., 26 years, complaints about muscle asthenia, headaches, thirst, night urination, convulsions of muscles of extremities and feeling of crawl of insects, pains in heart. ABP 190/110 mmHg. Borders of heart are widened to the left; at examination of eye-ground is revealed presence of spasm of arteriole and expanded venules; activity of renin in plasma is decreased, K+ - 2,9 mmol/L, Na+ - 165 mmol/L.

1. What diagnosis would these clinical features and laboratory findings suggest?
2. How can you explain increase of arterial blood pressure in the patient?

Task 7

Patient L. 30 years, in 3 months after childbirth gained weight - 7 kg per month and her attention was drawn by unusual location of fat: in the neck and face. X-ray examination revealed increased size of cella Turcica. The patient has high blood glucose level, glucose is also present in the urine.

1. What endocrine pathology can be suggested?
2. Would it be classified as a primary or secondary endocrine disorder?

Teacher's signature

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UNIT 35
SUB-MODULE 6 CONTROL "Pathology of GIT, liver, kidneys and endocrine system"
Checking of practical skills and theoretical knowledge

The final practical class in sub-module 6 consists of estimating student's rating grade. To be allowed to pass sub-module 6 control the student should:
1. get no less than 12 grades during current practical classes;
2. have no missed lectures and practical classes to the current date;
3. have all the practical classes' protocols signed by the teacher and show the lectures notebook;
4. successfully passed submodule 4 and 5control.

Students have an opportunity to pass sub-module control once more during two weeks. If sub-module control was not successfully passed in two weeks they will not be allowed to the following lectures and practical classes and rating mark for sub-module 6 control will be "zero".

SUB-MODULE 6 PRACTICAL SKILLS

1. Students should be able to define:
- typical disturbances of GIT, liver and kidneys functions: malabsorbption, peptic ulcer disease gastritis, intestinal obstruction, jaundice, hepatic failure, portal hypertension, glomerulonephritis, renal failure;
- typical disturbances of endocrine system disorders: acromegaly, gigantism, panhypopituitarism, Sheehan's syndrome, Cushing's disease and Cushing's syndrome, Grave's disease, myxedema, adrenal virilism, Conn's syndrome, Addison's disease

2. Students should be able to analyze:
- indices of stomach secretory functions and evaluate them;
- indices of bilirubin metabolism and determine jaundice type;
- qualitative and quantitative violations of uropoiesis;
- causality-effective relations, pathological and adaptive features in pathogenesis of the following diseases: peptic ulcer disease gastritis, intestinal obstruction, jaundice, hepatic failure, portal hypertension, glomerulonephritis, renal failure;
- the consequences of inadequate hormones secretion of hypophysis, thyroid, parathyroid, adrenal glands;
- biological role of general adaptation syndrome and stress, its etiology and pathogenesis.

3. To obtain practical skills:
- in solving problem tasks and tests – definition of etiology, pathogenesis, mechanisms of clinical signs development, principles of diagnosing, prophylaxis and treatment in such clinical conditions: malabsorbption, peptic ulcer disease gastritis, intestinal obstruction, jaundice, hepatic failure, portal hypertension, glomerulonephritis, renal failure, acromegaly, gigantism, panhypopituitarism, Sheehan's syndrome, Cushing's disease and Cushing's syndrome, Grave's disease, myxedema, adrenal virilism, Conn's syndrome, Addison's disease.

UNIT 36
Pathology of nervous system. Violation of sensitivity and motion activity.

1. Disorders of the sensor functions of the nervous system. Kinds of sensitivity. Qualitative and quantitative disorders of sensitivity.
2. Pain. Pain receptors. Kinds of pain, mechanisms of development. Head ache. Migraine.
3. Disorders of moving function of the nervous system. Kinds of disorders. Peripheral and central paralysis and paresis.
4. The role of vegetative nervous system in regulation of homeostasis. Vegetative vascular dysfunction: etiology, pathogenesis, features.
5. Etiology and pathogenesis of central nervous system affection. Acute and chronic disorders of cerebral circulation. Strokes. Brain edema.
6. Disorders of higher nervous activity. Neurosis.

Experimental work.

1. To solve situational problems.

Task1

A 25-year-old man is admitted to the emergency department with acute abdominal pain that began in the epigastric area and has now shifted to the lower right quadrant of the abdomen. There is localized tenderness and guarding or spasm of the muscle over the area. His heart rate and blood pressure are elevated, and his skin is moist and cool from perspiring. He is given a tentative diagnosis of appendicitis and referred for surgical consultation.

1. Describe the origin of the pain stimuli and the neural pathways involved in the pain that this man is experiencing.
2. Explain the neural mechanisms involved in the spasm of the overlying abdominal muscles.
3. What is the significance of his cool, moist skin and increased heart rate and blood pressure?

Task 2

A 21-year-old woman presents in the student health center with complaints of a throbbing pain on the left side of her head, nausea and vomiting, and extreme sensitivity to light, noise, and head movement. She also tells you she had a similar headache 3 months ago that lasted for 2 days and states that she thinks she is developing migraine headaches like her mother. She is concerned because she has been unable to attend classes and has exams next week.

1. Are this woman's history and symptoms consistent with migraine headaches?
2. Use the distribution of the trigeminal nerve and the concept of neurogenic inflammation to explain this woman's symptoms.

Task 3

A 32-year-old woman presents with complaints of drooping eyelids, difficulty chewing and swallowing, and weakness of her arms and legs that is less severe in the morning but becomes worse as the day progresses. She complains that climbing stairs and lifting objects is becoming increasingly difficult. Clinical examination confirms weakness of the eyelid and jaw muscles. She is told that she may have myasthenia gravis and is scheduled for a test using the short-acting acetylcholinesterase inhibitor edrophonium (Tensilon).

1. Explain the pathogenesis of this woman's symptoms as it relates to myasthenia gravis.

2. Explain how information from the administration of the acetylcholinesterase inhibitor edrophonium can be used to assist in the diagnosis of the disorder.

Task 4

Patient N., 49 years, is delivered to neurology department. There is limitation of voluntary movements in the left extremities, more in a hand. Tone of muscles in the left hand and leg is increased according to spastic type. There are increased local tendinous reflexes, pathologic reflexes.

1. How can you characterise the neurologic disorders of this patient?

2. Explain the mechanism of muscles tone increase in the left extremities.

Task 5

Patient L., 40 years, a month ago got the trauma of right thigh. At the examination of the neurologic status: active movements in right leg are limited because of severe pain, volume of muscles on the right leg is on 2 cm lesser, then on the left, Achilles and knee reflexes on the right side are absent. There is decreased proprioceptive sensitivity on the right leg in the region of foot.

1. How can you characterise the neurologic disorders at a patient?

2. Explain the mechanism of decreased muscles volume and reflexes absence in the patient.

Teacher's signature

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UNIT 37
FINAL MODULE 2 CONTROL.

The final module control consists of estimating student's rating grade. To be allowed to pass final module control the student should:
1. get no less than 51 grades during current practical classes;
2. have no missed lectures and practical classes to the current date;
3. successfully pass all the previous submodules.

Final module control consists of computer testing. In order to pass final module control successfully, student should obtain such theoretical and practical skills:
- to define different WBC forms count with the help of blood count;
- to define hemoglobin blood content and interpret the result;
- to calculate color index and interpret the result;
- to identify regenerative and degenerative forms of RBC and WBC in peripheral blood smear and interpret the results of findings;
- to define typical disturbances of blood circulating system: heart failure, circulatory failure, arrhythmia, arterial hypotension and hypertension, arteriosclerosis, atherosclerosis using modern classifications;
- to analyze changes of the cardiohaemodynamic indices in the pathology, changes of spirogram and lungs volume;
- to estimate the role of disturbances of alveolar ventilation, disorders of blood perfusion in the lungs, mismatching of ventilation/perfusion ratio and impairment of gases diffusion in the lungs;
- to define typical disturbances of GIT, liver and kidneys functions: malabsorbption, peptic ulcer disease gastritis, intestinal obstruction, jaundice, hepatic failure, portal hypertension, glomerulonephritis, renal failure;
- to define typical disturbances of endocrine system disorders: acromegaly, gigantism, panhypopituitarism, Sheehan's syndrome, Cushing's disease and Cushing's syndrome, Grave's disease, myxedema, adrenal virilism, Conn's syndrome, Addison's disease;
- to analyze the consequences of inadequate hormones secretion of hypophysis, thyroid, parathyroid, adrenal glands;
- to estimate biological role of general adaptation syndrome and stress, its etiology and pathogenesis;
- to define the typical disturbances of nervous system motor and sensitive function.