Pathophysiology of the Cardiovascular System.

Case 1.

A disabled woman, aged 36, was admitted to hospital with complaints of dyspnea, tachycardia, edemata of the legs, abdominal distension, quick fatigability and muscle weakness. From her medical history it is known that she has had repeated rheumocarditis, suffers from a heart disease (combined mitral valve defect with prevalence of stenosis). *Examination data*: The patient is exhausted and pale; the skin is cyanotic, cold at touch; there is apparent jaundice of the sclera. There are edemata on the legs and loin, ascites, bilateral hydrothorax. The liver projects from the costal arch by 8cm. The heart is sharply enlarged (both right and left parts). The patient has ciliary arrhythmia; heart rate is 110 – 120/min. The ejection fraction of the left ventricle is 29%. "Wedge" pressure of the pulmonary capillaries is 25 mm Hg. *In the blood:* albumin content – 29 g/l. globulin content – 30 g/l. Norepinephrine level is 3 times as high as normal. End-systolic volume (ESV) is 179 ml; end-diastolic volume (EDV) – 254 ml. Diurnal diuresis is 700 ml.

1. What is this form of cardiac insufficiency called? Confirm your diagnosis by the examination findings.
2. Explain the change in the "wedge" pressure of the pulmonary capillaries in this patient.
3. What is the prognosis of maximum life expectancy of patients with this disease?
4. What pathogenetic therapy is used for this form of cardiac insufficiency?
5. Explain the mechanism of dyspnea, tachycardia, muscle weakness and exhaustion in this patient.
6. Explain the mechanism of edemata and arrhythmia.
7. What signs are suggestive of remodeling of the patient's myocardium? What is its pathogenesis?
8. Explain the mechanism of diuresis reduction in this patient.

Case 2.

A 68-year-old man suffering from stable effort angina has been noting attacks of the disease 1 – 2 times a day during walking. Over the last week the number of attacks has increased up to 10 – 15 a day, but their severity has not changed: they ceased after a rest and, sometimes, after taking nitroglycerin. He did not apply to a physician and did not take anti-anginal medications. On the day of hospitalization, when the patient was outside his home, a severe attack of angina developed which was not relieved by nitroglycerin.

On admission: The patient is pale, scared, complains of severe chest pain. BP is 90/60 mm Hg, pulse – 100/min. *On ECG* : in leads I, aVL, V_1-V_6 - marked elevation of ST segment (monophase curve). *Echography* shows thinning of the left ventricle wall with an area of akinesia. There are increased levels of myoglobin and troponin in the blood, neutrophil leukocytosis, accelerated ESR. Body temperature is 38.4 ˚C.

1. What disease can be suspected in this patient? Confirm your diagnosis by the examination findings. What can the disease be caused by?
2. What do leukocytosis, increase in the body temperature, acceleration of ESR and results of the biochemical blood test indicate?
3. What is the mechanism of the patient's severe pain attack?
4. What is the pathogenetic therapy in this case?

Case 3.

A 68-year-old man suffering from stable effort angina has been noting attacks of the disease 1 – 2 times a day during walking. Over the last week the number of attacks has increased up to 10 – 15 a day, but their severity has not changed: they ceased after a rest and, sometimes, after taking nitroglycerin. He did not apply to a physician and did not take anti-anginal medications regularly. On the day of hospitalization, when the patient was outside his home, a severe attack of angina developed which was relieved only in the in-patient department. *On ECG:* in leads II, III, aVF,V_5,V_6 - tall widened peaked T-wave. Blood levels of myoglobin, troponin, creatinephosphokinase and aspartate-aminotranspherase are normal.

1. What pathological process in the patient's myocardium can be thought of? Confirm your supposition by the examination findings.
2. What additional investigation should be carried out to administer a proper treatment?
3. Point out the principles of prevention of myocardial infarction.

Case 4.

A 84-year-old man was admitted to hospital in an extremely severe condition with a paroxysm of ciliary tachyarrhythmia and quickly progressing pulmonary edema, tachyarrhythmia - 130 per minute on ECG, arterial hypotension – 85/60 mm Hg. ECG showed atrial fibrillation, scar changes in the posterodiaphragmal wall of the left ventricle, signs of acute myocardial infarction of the anterior wall of the left ventricle and interventricular septum. Anti-arrhythmic therapy was not effective.

10 minutes after the admission transthoracal defibrillation was performed on vital indications; sinus rhythm (94/min) was restored. Over the following 15 minutes acute left-ventricular insufficiency sharply decreased. Blood pressure became 120/70 mm Hg.

1. Why did the paroxysm of ciliary tachyarrhythmia sharply worsen the patient's condition?
2. How and why could the end-diastolic volume change at the moment of tachyarrhythmia?
3. What factors contributing to arrhythmia development arise in myocardial infarction?
4. Why did the provided treatment quickly improve the patient's condition?

Case 5.

A 75-year-old man was admitted to hospital in an extremely severe condition with a paroxysm of ciliary tachyarrhythmia (about 130 per minute), quickly progressing pulmonary edema and arterial hypertension (185/120 mm Hg). *On ECG:* frequent polytopic and group ventricular extrasystoles, scar changes in the posterodiaphragmal wall of the left ventricle, signs of myocardial infarction of the anterior wall of the left ventricle and interventricular septum. Anti-arrhythmic therapy was not effective.10 minutes after the admission transthoracal defibrillation was performed on vital indications; sinus rhythm (94/min) was restored; a diuretic and a vasodilator were introduced intravenously. Over the following 30 minutes acute left-ventricular insufficiency sharply decreased; blood pressure reduced to 120/70 mm Hg.

1. What factors provoked the development of acute left-ventricular insufficiency in this patient?
2. How did the contractile function of the left ventricle myocardium change and why?
3. How could the indices of intracardiac hemodynamics change and why?
4. Why did the provided treatment quickly improve the patient's condition?

Case 6.

A 35-year-old woman presented with persistent recurrent headaches and attacks of palpitations which at times interfere with her sleep and moving. She sometimes complains of dizziness, tingling in the ears, burning sensation in her arms and legs, white or black "floaters" in the eyes. She has a 9-year history of these symptoms. 12 years ago the patient's blood pressure measurement in an out-patient clinic showed 160/80 mm Hg. She was periodically treated with various hypotensive drugs. *On examination:* the heart is moderately enlarged to the left; pulse – 72 – 96/min, slightly tense. While the patient was staying in hospital her blood pressure varied: 170/100; 160/100, 145/90 and even 125/80 mm Hg. The patient's mood is very unstable. *ECG* reveals signs of left ventricle hypertrophy; PQ interval – 0.22 sec. Examination of the eye fundus indicates narrowing of the arteries due to organic changes in them. The patient's diagnosis – arterial hypertension.

1. Calculate the variants of the patient's mean blood pressure.
2. Classify this hypertension according to BP level.
3. What stage of arterial hypertension does this patient have? Justify your conclusion.
4. What types of arterial hypertension do you know? What type may this patient have? What investigations should be performed to specify the diagnosis?
5. Explain the mechanism of the left ventricle hypertrophy.

Case 7.

A disabled woman, 38 years old, was admitted to hospital with complaints of dyspnea, tachycardia, leg edemata and abdomen distension. From her medical history it is known that she has had repeated rheumocarditis, suffers from a heart disease (combined mitral valve defect with prevalence of stenosis). *Examination data*: The patient is exhausted; her skin is pale and cold at touch; she has acrocyanosis, jaundice of the sclera. There are edemata on the legs and loin, ascites, bilateral hydrothorax. The liver projects from the costal arch by 8cm. The heart is sharply enlarged (both right and left parts). The patient has ciliary arrhythmia, heart rate is 110 – 120/min. Diurnal diuresis is 700 ml.

1. What is this form of cardiac insufficiency called?
2. Confirm your diagnosis by examination findings.
3. What could hemodynamic indices in this patient be (cardiac output, cardiac index, arterio-venous (A-V) oxygen difference, oxygen utilization coefficient, peripheral vascular resistance, central venous pressure, cathecholamine level)?
4. Explain your answer.

Case 8.

A disabled woman, 39 years old, was admitted to hospital with complaints of dyspnea, tachycardia, leg edemata, abdomen distention, increased fatigability and muscle weakness. From her medical history it is known that she has had repeated rheumocarditis, suffers from a heart disease (combined mitral valve defect with prevalence of stenosis). *Examination data*: The patient is exhausted; her skin is pale with a cyanotic tint, jaundice of the sclera is noted. There are edemata on the legs and loin, ascites, bilateral hydrothorax. The liver projects from the costal arch by 8cm. The heart is sharply enlarged (both right and left parts). The patient has ciliary arrhythmia, heart rate is 110 – 120/min. Left ventricle indices: stroke volume – 77 ml, end-systolic volume – 179 ml, end-diastolic volume – 254 ml. Diurnal diuresis – 800 ml.

1. What is this form of cardiac insufficiency called?
2. Confirm your diagnosis by the examination findings.
3. What hemodynamic index can confirm a decrease in left ventricle myocardium contractility in this patient?
4. How and why could the indices of compliance and relaxation of the patient's myocardium change?

Case 9.

A disabled woman, 42 years old, was admitted to hospital with complaints of dyspnea, tachycardia, leg edemata, abdomen distention, increased fatigability and muscle weakness. From her medical history it is known that she has had repeated rheumocarditis, suffers from a heart disease (combined mitral valve defect with prevalence of stenosis). *Examination data*: The patient is exhausted; her skin is pale with a cyanotic tint, jaundice of the sclera is noted. There are edemata on the legs and loin, ascites, bilateral hydrothorax. The liver projects from the costal arch by 8cm. The heart is sharply enlarged (both right and left parts). The patient has ciliary arrhythmia, heart rate is 110 – 120/min. Diurnal diuresis – 400 ml.

1. What is this form of cardiac insufficiency called?
2. Confirm your diagnosis by the examination findings.
3. What changes of the intracardiac hemodynamic indices are most likely to be found in this patient (stroke volume [SV], cardiac index [CI], ejection fraction [EF], end-diastolic volume [EDV], end-systolic volume [ESV], filling pressure in the left and right heart)? Compare these results with the norm (higher, lower or within the norm).
4. Explain the pathogenesis of the skin symptoms and myocardium hypertrophy.

Case 10

A disabled man, 58 years old, only recently worked as an engineer. He presented with complaints of periodic palpitations, seeing a "net" in front of the eyes, severe headaches sometimes accompanied by vomiting. 9 years ago the patient was first noticed to have blood pressure of 180-190/100 mm Hg. He periodically took hypotensive drugs. Over the last 3 years he had been having nocturnal attacks of asphyxia. Half a year ago he had right-sided hemiparesis. *Examination data*: The patient is edematous, pale, a little euphoric, talkative; his memory is markedly decreased. Heart boundaries are expanded both to the left and to the right. The pulse is 64-68 per minute, tense; blood pressure – from 200/120 to 180/90 mm Hg. *On ECG:* hypertrophy of the left ventricle and slowing down of the intraventricular conductivity. Eye fundus examination revealed sharp narrowing of the retinal arteries, sometimes with thickening of their walls, pinpoint hemorrhages. The kidneys are almost unchanged; slight albuminuria – 0.06 %.

1. Calculate fluctuations of the patient's mean BP.
2. Classify this hypertension according to the level of BP.
3. What stage of arterial hypertension does the patient have? Justify your conclusion.
4. Point out the target organs of the arterial hypertension in this patient.
5. Explain the pathogenesis of the patient's symptoms associated with his main disease.
6. What are pathogenetically-grounded recommendations for treatment of this patient?

Case 11.

Patient K., 53 years old, was hospitalized with a diagnosis: acute anteroseptal myocardial infarction of the left ventricle. He was given standard therapy but his condition suddenly deteriorated: he developed dyspnea with respiratory frequency of 40/min, cough with foamy pink sputum. Moist rales were auscultated all over the lungs, heart sounds were muffled with an accent of sound II over the pulmonary artery.

1. What complication of myocardial infarction developed in this patient? Justify your answer. Describe the pathogenesis of this complication.
2. What ECG sign indicates the development of an ischemic lesion of the myocardium? Describe its mechanism.
3. Point out the principles of pathogenetic therapy for this patient.

Case 12.

Patient N., 56 years old, was hospitalized with a diagnosis: hypertensive crisis.
History data: The patient has been suffering from arterial hypertension for 10 years. When her blood pressure rose up to 240/130 mm Hg, she developed asphyxia, gurgling rales all over the lungs, cardialgia, tachycardia. At the moment of the attack ECG showed a negative symmetrical T-wave in leads V_2 - V_5.

1. What complication of hypertensive crisis developed in this patient? Justify your answer.
2. What does a negative symmetrical T-wave in the chest leads indicate? Explain the mechanism of its formation.
3. Explain the pathogenesis of this complication.
4. Point out the principles of pathogenetic therapy for this patient.

case

Calculate the cardiac ejection fraction (EP). if it is known, that the end-dlastolic volume is 120 ml, the end-systolic volume is 50 ml. Estimate your result.

case

Calculate the load (work) of the right ventricle, if it is known that its stroke volume is about 80 ml and average lung arteria pressure is about 20 mm Hg.Heart rate- 100/ min. Estimate your result.

case

Calculate average arterial pressure, if it is known that pulse pressure is about 50 mm Hg and systollc pressure is about 165 mm Hg.

case

Patient, 46 years old, has area of her body surface - 1,5 m , cardiac output - 4,5 1/min, the end-dlastolic volume of left ventricle - 100 ml, the end-systolic volume of left ventricle - 30 ml. Calculate stroke volume of left ventricle, the cardiac index and the cardiac ejection fraction.

case

Calculate the blood oxygen utilization index, if it is known; the content of O_2 in arterial blood - 18 vol.%, the content of O_2 in venous blood is about 10 vol.%. Estimate the result of your calculation.

case

Calculate the volume of circulating blood, if it is known, that 10 minutes after 10 mg blue Evance intravenous injection its average concentration was been about 5 mg/per liter. Estimate your result. The weight of patient is about 56 kg, his hematocrite is 40 %.

case

Calculate the cardiac output in the patient,which arterial blood contains of 18 vol.% of O_2, the venous blood - 10 vol.% of O_2.

The patient intakes of 320 ml O_2 per minute

case

Which form of heart pathology would you suppose, if it is known, that in patient:

1. aortic pressure is about 110/80 mm Hg
2. Into the left ventricle the end-diastolic volume is about 300 ml, the end-systolic volume is about 150 ml, the end-diastolic pressure is about 20 mm Hg
3. the lung capillary pressure is about 22 ram Hg ?

case

Calculate the cardiac index in patient of 75 kg weight and of 180 cm height. His cardiac output is about 5,5 liter (use a nomogramm).

case

Which form of heart pathology would you suppose, if it is known, that in patient:

1. aortic pressure is about 110/70 mm Hg
2. into the left ventricle the end-diastolic volume is about 90 ml, the end-systolic volume is about 45 ml
3. the lung capillary pressure is about 25 mm Hg ?

case

Calculate the heart output, if it is known, that heart stroke volume is about 60 ml, pulse rate is 80/min.

case

Which form of heart pathology would you suppose, if it is known, that in patient:

1. aortic pressure is about 160/65 mm Hg
2. into the left ventricle:
   the end-diastolic volume is about 300 ml, the end-systolic volume is about 120 ml, the end-diastolic pressure is about 16 mm Hg
3. the lung capillary pressure Is about 13 mm Hg ?