Colloquium 5
Cases

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 attended his dentist for check-up, 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 35-year-old woman was admitted to hospital with unstoppable bleeding after tooth extraction, headache. She sometimes complains of dizziness, tingling in the ears, burning sensation in her arms and legs, white or black "floaters" in the eyes and attacks of palpitations which at times interfere with her sleep and moving. 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 4.

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 5

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 6.

Patient V., 60 years old, height -160 cm, body mass – 80 kg, was admitted to hospital with complaints of acute weakness, dyspnea, palpitations, cough with "rusty" sputum, headache, drowsiness, decreased appetite, temperature rise up to 38 – 39˚C with marked chills. She became ill several days ago.

Examination findings: body temperature – 38.5˚C; leukocyte count – 13 x 10^9 /l; ESR – 20 mm/h. On auscultation in the left lung crepitation and pleural friction murmur are heard. Pa O_2 – 60 mm Hg, Pa CO_2 – 50 mm Hg; diffusing lung capacity (DLCO) – 10 ml/ 1 mm Hg/min.

Ventilation indices: breathing rate (BR) – 30/min; tidal volume (TV) – 0.25liters; reserve volume of inspiration (RV_{ins}) -1 liter; vital capacity of lungs (VC) –
2.5 litres; forced vital capacity (FVC) – 2.3 liters ; forced expiratory volume (FEV_{1sec}) – 2 liters; total lung capacity (TLC) – 3.7 liters, dead space volume (DSV) – 150 ml.

1. Calculate and assess minute respiratory volume (MV), minute alveolar ventilation (MAV) and Tiffeneau index.
2. What type of respiration does the patient have?
3. What is the type of lung ventilation disturbance?
4. Is gas diffusion in the patient's lungs disturbed? Confirm your viewpoint.
5. What disease can be thought of?
6. Explain the pathogenesis of the symptoms.

Reference (predictive values): ***Pred*MV = 6.7 l/min; *Pred*VC = 3.2 l; *Pred*MAV = 4.3 – 4.5 l/min**

Case 7.

Patient K., 45 years old, height – 175 cm, complains of cough with sputum which has been disturbing him for the last three years, expiratory dyspnea, palpitations,

increased fatigability, headache. He has been smoking since the age of 15, smokes 2 packs of cigarettes a day.

Examination findings: body temperature is 36.7°C; ESR – 7 mm/h; barrel chest; bandbox sound on percussion. Diffusing lung capacity (DLCO) is 12 ml/1mm Hg/ min. Forced expiratory flow rate: peak forced expiratory flow rate (PFEFR), maximum forced expiratory flow rate - $MFEFR_{25}$, $MFEFR_{50}$, $MFEFR_{75}$ are less than predictive values.

Ventilation indices: BR – 30/min; VC – 3.8 liters; FVC – 3.3 liters; FEV_{1sec} – 1.4 liters; TLC – 6.6 liters; RV_{ins} – 1litre.

1. Calculate and assess Tiffeneau index and residual lung volume (RLV).
2. Determine the type of lung ventilation disturbance.
3. Determine the type of respiratory insufficiency according to its pathogenesis.
4. What disease can be thought of?
5. Explain the pathogenesis of this disease.
6. What are the symptoms of the disease conditioned by?

***Reference (predictive values): Pred*VC = 4.65 l; *Pred*RLV = 1.5 l**

Case 8.

Patient N., a 20-years-old woman, height – 164 cm, body mass – 65 kg; complains of periodic attacks of asphyxia with difficulty in expiration which are accompanied by excretion of viscous glassy sputum. The attacks began two years ago after the family had acquired a dog. The attacks are often triggered by inhaling cold air or strong emotions. Her mother suffers from urticaria, her brother has pollinosis.

Examination findings: Diffusing lung capacity (DLCO) – 20 ml/ 1 mm Hg/ min. The results of the test "flow/ volume curve": peak forced expiratory flow rate (PFEFR) and maximum forced expiratory flow rate $MFEFR_{25}$ are not changed, $MFEFR_{50}$ and $MFEFR_{75}$ are less than the predictive values.

Ventilation indices: BR – 20/min; tidal volume (TV) – 0.4 liters; maximum voluntary ventilation (MVV) – 60 l/min; vital capacity of the lungs (VL) – 3.7 liters; forced vital capacity (FVC) – 3.4 liters; forced expiratory volume (FEV_{1sec}) – 2 liters; residual lung volume (RLV) – 1.8 liters; inspiration/ expiration ratio – 1: 1.5.

1. Calculate and assess respiratory reserve (RR), Tiffeneau index and total lung capacity (TLC).
2. What type of lung ventilation disturbance does the patient have?
3. What is the type of respiratory insufficiency according to its pathogenesis?
4. What is this disease called?
5. What long-term consequence can develop in this patient if the disease is progressing? What is the mechanism of its development?
6. Is it reasonable to use M-cholinoblockers in the complex treatment of this patient? Why?

***Reference (predictive values): Pred*MV= 6.3 l/min; *Pred*VC= 3.7 l; *Pred*RR = 81.4 l/min; *Pred*TLC = 5.2 l.**