МИНИСТЕРСТВО ЗДРАВООХРАНЕНИЯ РЕСПУБЛИКИ БЕЛАРУСЬ
БЕЛОРУССКИЙ ГОСУДАРСТВЕННЫЙ МЕДИЦИНСКИЙ УНИВЕРСИТЕТ
КАФЕДРА ПРОПЕДЕВТИКИ ВНУТРЕННИХ БОЛЕЗНЕЙ

ОСТРАЯ И ХРОНИЧЕСКАЯ СЕРДЕЧНАЯ НЕДОСТАТОЧНОСТЬ. НАРУШЕНИЯ СЕРДЕЧНОГО РИТМА И ПРОВОДИМОСТИ

ACUTE AND CHRONIC HEART FAILURE. CARDIAC ARRHYTHMIAS AND CONDUCTION DISORDERS

Учебно-методическое пособие

Минск БГМУ 2026

УДК 616.12-008.46.616.12.008.3.616.127(075.8)=111
ББК 54.101я73
О-76

Рекомендовано Научно-методическим советом университета в качестве учебно-методического пособия 20.05.2026 г., протокол № 9

А в т о р ы: Э. А. Доценко, М. В. Шолкова, Ю. В. Репина, М. Н. Антонович

Р е ц е н з е н т ы: д-р мед. наук, проф., гл. науч. сотр. лаборатории сердечно-сосудистой хирургии, консультант 3-го кардиологического отделения Республиканского научно-практического центра «Кардиология» Л. Г. Гелис; каф. пропедевтики внутренних болезней Витебского государственного ордена Дружбы народов медицинского университета

Острая и хроническая сердечная недостаточность. Нарушения сердечного О-76 ритма и проводимости = Acute and chronic heart failure. Cardiac arrhythmias and conduction disorders : учебно-методическое пособие / Э. А. Доценко, М. В. Шолкова, Ю. В. Репина, М. Н. Антонович. – Минск : БГМУ, 2026. – 31 с.

ISBN 978-985-21-2279-5.

Содержит сведения об этиологии, патогенезе, клинических проявлениях, принципах лечения острой и хронической сердечной недостаточности и нарушениях сердечного ритма и проводимости.

Предназначено для специальности «Лечебное дело» по учебной дисциплине «Пропедевтика внутренних болезней».

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LIST OF ABBREVIATIONS

ACEi — angiotensin-converting enzyme inhibitor
AF — atrial fibrillation
AHF — acute heart failure
ARNI — angiotensin receptor-neprilysin inhibitor
ARB — angiotensin II receptor blocker
AV — atrioventricular
BB — beta-blocker
BNP — brain natriuretic peptide
BP — blood pressure
CHF — chronic heart failure
ECG — electrocardiography
EF — ejection fraction
ESC — European Society of Cardiology
HF — heart failure
HFmrEF — heart failure with mildly reduced ejection fraction
HFpEF — heart failure with preserved ejection fraction
HFrEF — heart failure with reduced ejection fraction
HR — heart rate
IV — Intravenous
LA — left atrium
LV — left ventricle
LVEF — left ventricular ejection fraction
MRA — mineralocorticoid receptor antagonist
NYHA — New York Heart Association
RA — right atrium
RAAS — renin-angiotensin-aldosterone system
RV — right ventricle
S1 — first heart sound
S2 — second heart sound
S3 — third heart sound
SBP — systolic blood pressure
SNS — sympathetic nervous system
SpO_2 — peripheral oxygen saturation
SVT — supraventricular tachycardia
VT — ventricular tachycardia

MOTIVATIONAL CHARACTERISTICS OF THE TOPIC

Topic: "Acute and Chronic Heart Failure. Cardiac Arrhythmias and Conduction Disorders."

Duration of the lesson: 3 hours.

Goals. To understand the definitions, etiology, pathogenesis, clinical presentations, and classifications of acute and chronic heart failure, as well as cardiac arrhythmias and conduction disorders. To learn diagnostic approaches and treatment principles.

Objectives:

1. Define heart failure. Define acute vascular insufficiency.
2. Describe the etiology and pathogenesis of heart failure.
3. Classify acute and chronic heart failure and characterize their clinical manifestation.
4. Recognize laboratory and instrumental methods for diagnosing heart failure.
5. Understand treatment principles and prophylaxis for heart failure.
6. Describe clinical manifestation and management of syncope, collapse, and shock.
7. Explain mechanisms of heart rhythm disorders.
8. Identify clinical and ECG signs of extrasystole, paroxysmal tachycardia, atrial flutter, atrial fibrillation, AV block, and His bundle branch block.

The student must be able to:

1. Define heart failure and its types.
2. Describe causes and pathogenic mechanisms of heart failure.
3. Classify and characterize acute and chronic heart failure based on symptoms and functional classes.
4. Interpret laboratory and instrumental diagnostic findings for heart failure.
5. Explain treatment strategies for acute and chronic heart failure.
6. Define acute vascular insufficiency and describe clinical signs of syncope, collapse, and shock. Provide first aid for syncope.
7. Outline mechanisms of heart rhythm disorders.
8. Identify clinical and electrocardiographic features of: extrasystole, paroxysmal tachycardia, atrial flutter, atrial fibrillation, AV block, His bundle branch block.

The student should know:

– pathophysiological mechanisms of heart failure and arrhythmias;

– clinical manifestations distinctive to right and left ventricular failure;

– ECG characteristics of common arrhythmias and conduction disturbances;

– principles of emergency management in acute vascular insufficiency.

Initial level of knowledge. Familiarity with cardiac anatomy, physiology of ventricular function, and basics of cardiac electrophysiology.

Control questions from related disciplines:

– *anatomy:* heart chambers, conduction system anatomy including the sinoatrial node, AV node, and His–Purkinje system. The right heart receives

deoxygenated blood and pumps it to the lungs; the left heart receives oxygenated blood and pumps it into the systemic circulation;

– *physiology:* cardiac contractility, the Frank–Starling mechanism (stroke volume increases with increased preload), electrophysiological properties of cardiac tissue (automaticity, excitability, conductivity, refractoriness), and regulation of cardiac output;

– *biochemistry:* biomarkers in heart failure (BNP, troponins), factors affecting myocardial metabolism (oxygen consumption, substrate utilization, calcium handling).

Control questions:

1. Definition of the term “heart failure.” Etiology and pathogenesis of heart failure.
2. Classification heart failure.
3. Clinical manifestation of acute right ventricular heart failure.
4. Clinical manifestation of acute left ventricular heart failure.
5. Laboratory and instrumental diagnosis of acute heart failure.
6. Principles of acute heart failure (right and left ventricular) treatment.
7. Clinical manifestation of chronic heart failure (according to stages and functional classes).
8. Laboratory and instrumental diagnosis of chronic heart failure.
9. Principles of chronic heart failure treatment.
10. Heart failure prevention.
11. Mechanisms of heart rhythm disorders.
12. Extrasystole: definition, classification, clinical and electrocardiographic signs.
13. Paroxysmal tachycardia: definition, classification, clinical and electrocardiographic signs.
14. Atrial flutter: definition, clinical and electrocardiographic signs.
15. Atrial fibrillation: definition, clinical and electrocardiographic signs.
16. Mechanism of heart conduction disorders.
17. AV block: definition, classification, clinical and electrocardiographic signs.
18. His bundle branch block: definition, classification, clinical and electrocardiographic signs.

TERMINOLOGY

Heart failure — a clinical syndrome resulting from a structural and/or functional cardiac abnormality, leading to reduced cardiac output and/or elevated intracardiac pressures at rest or during stress, manifested by typical symptoms such as breathlessness, ankle swelling, and fatigue.

Acute heart failure (AHF) — a rapid onset or worsening of signs and symptoms of heart failure, requiring urgent evaluation and treatment; it is a life-threatening condition that usually leads to emergency hospitalization.

Chronic heart failure (CHF) — a clinical syndrome characterized by typical symptoms (breathlessness, ankle swelling, fatigue) that may be accompanied by signs (elevated jugular venous pressure, pulmonary crackles, peripheral edema) caused by a structural and/or functional cardiac abnormality.

Cardiac output (CO) — the volume of blood ejected by the heart per unit time, usually expressed in liters per minute.

Ejection fraction (EF) — the fraction of blood in the ventricle that is ejected with each contraction; normal LVEF is ≥ 50 %.

Cardiac asthma — a stage of acute left ventricular failure characterized by interstitial pulmonary edema, presenting with paroxysmal nocturnal dyspnea and wheezing.

Pulmonary edema — a stage of acute left ventricular failure characterized by fluid accumulation in the alveoli, causing severe dyspnea and the production of pink frothy sputum.

Orthopnea — dyspnea in the supine position, relieved by sitting upright.

Gallop rhythm — an audible third heart sound (S3), indicative of rapid ventricular filling in the context of a dilated, non-compliant ventricle; suggestive of heart failure.

Arrhythmia — any deviation from the normal sinus rhythm of the heart, including abnormalities in rate, regularity, site of origin, or conduction of the cardiac electrical impulse.

Extrasystole (premature contraction) — a premature cardiac contraction arising from an ectopic (heterotopic) focus in the atria, atrioventricular junction, or ventricles, occurring before the expected normal beat.

Paroxysmal tachycardia — a sudden onset and termination of a rapid heart rate exceeding 100 beats per minute, arising from a focus outside the sinus node.

Atrial fibrillation (AF) — chaotic, irregular electrical activity of the atria with a rate of 350–600 per minute, resulting in irregularly irregular ventricular contractions and the absence of coordinated atrial contraction.

Atrial flutter — a rapid, regular atrial rhythm (200–500 per minute) with a characteristic “sawtooth” pattern on ECG, often conducted to the ventricles in fixed ratios (2 : 1, 3 : 1, 4 : 1).

Atrioventricular (AV) block — a delay or interruption in the conduction of electrical impulses from the atria to the ventricles through the AV node and/or His–Purkinje system.

His bundle branch block — a conduction delay or block in one or more branches of the His bundle (right or left), resulting in prolongation and altered morphology of the QRS complex on ECG.

ACUTE HEART FAILURE

Definition. Heart failure is a condition in which the heart muscle is unable to pump enough blood to meet the body’s needs for blood and oxygen. More formally, heart failure is a clinical syndrome characterized by typical symptoms

(breathlessness, ankle swelling, and fatigue) that may be accompanied by signs (elevated jugular venous pressure, pulmonary crackles, and peripheral edema) caused by a structural and/or functional cardiac abnormality, resulting in a reduced cardiac output and/or elevated intracardiac pressures at rest or during stress.

Heart failure is not a single disease but rather a syndrome that may result from a wide variety of cardiac and non-cardiac disorders. It represents the final common pathway for many forms of heart disease.

Etiology. The principal etiologies of heart failure include:

– myocardial dysfunction: coronary heart disease (ischemic cardiomyopathy), myocarditis, dilated cardiomyopathy, hypertrophic cardiomyopathy;

– pressure overload (increased afterload): arterial hypertension, aortic valve stenosis, pulmonary hypertension;

– volume overload (increased preload): valvular regurgitation (mitral, aortic), intracardiac shunts, high-output states (thyrotoxicosis, anaemia);

– impaired ventricular filling: constrictive pericarditis, restrictive cardiomyopathy, cardiac tamponade;

– heart rhythm and conduction disorders: tachyarrhythmias (e.g., atrial fibrillation), bradyarrhythmias, complete AV block;

– toxic damage to the myocardium (alcohol, drugs).

Coronary heart disease and arterial hypertension together account for the majority of cases of heart failure in developed countries.

Pathogenesis. The pathogenesis of heart failure involves an interplay of hemodynamic, neurohormonal, and cellular mechanisms.

Hemodynamic mechanisms. When the heart's pump function is impaired, cardiac output decreases. According to the Frank–Starling mechanism, an increase in end-diastolic volume (preload) initially enhances myocardial fiber stretch and augments contractile force, thereby maintaining stroke volume. However, beyond a critical point, further increases in preload lead to overstretching of myocardial fibers, and stroke volume begins to decline. The ventricle becomes dilated, wall stress increases, and ventricular function deteriorates further.

Neurohormonal activation. In response to reduced cardiac output, several neurohormonal compensatory systems are activated:

Sympathetic nervous system: activation leads to release of catecholamines (norepinephrine, epinephrine), causing tachycardia, increased myocardial contractility, arterial vasoconstriction (to maintain blood pressure), and venous constriction (to increase venous return). Chronically, excessive sympathetic drive promotes myocardial remodelling, arrhythmias, and increased oxygen demand.

Renin-angiotensin-aldosterone system (RAAS): renal hypoperfusion stimulates renin release from juxtaglomerular cells, leading to formation of angiotensin II. Angiotensin II causes potent vasoconstriction, stimulates aldosterone secretion (promoting sodium and water retention), and promotes myocardial fibrosis and hypertrophy. Aldosterone also contributes to potassium loss and fibrosis.

Antidiuretic hormone (ADH/vasopressin): its secretion is increased in HF, contributing to water retention and hyponatraemia.

Natriuretic peptides (BNP): released in response to myocardial wall stress, these peptides promote natriuresis, diuresis, and vasodilation, partially counteracting the effects of the RAAS and Sympathetic nervous system. Elevated levels of BNP serve as important diagnostic and prognostic biomarkers.

Myocardial remodeling. Chronic neurohormonal activation and hemodynamic overload trigger structural changes in the myocardium known as remodeling. Remodeling includes myocardial hypertrophy (concentric in pressure overload, eccentric in volume overload), interstitial fibrosis, cardiomyocyte apoptosis, and progressive ventricular dilation. These changes initially serve as compensatory mechanisms but ultimately contribute to further impairment of ventricular function.

Classification of heart failure:

1. By time course:

a) acute heart failure (AHF). Rapid onset or worsening of symptoms and signs of HF, requiring urgent evaluation and treatment;

b) chronic heart failure (CHF). Long-standing clinical syndrome with persistent symptoms and/or signs due to structural or functional cardiac abnormality.

2. By predominant ventricle involved:

a) left ventricular heart failure. Predominantly pulmonary congestion (dyspnoea, orthopnoea, pulmonary rales);

b) right ventricular heart failure. Predominantly systemic venous congestion (peripheral edema, hepatomegaly, jugular venous distension);

c) biventricular (global) heart failure. Combined features of left and right ventricular failure.

3. By damaged function:

a) **systolic heart failure.** The heart cannot pump with enough force to push sufficient blood into systemic circulation (reduced ejection fraction);

b) **diastolic heart failure.** The heart loses its ability to relax normally due to muscle stiffness (ejection fraction is preserved or mildly reduced).

4. By left ventricular ejection fraction:

a) heart failure with reduced ejection fraction (HFrEF): LVEF $\leq$ 40 %;

b) heart failure with mildly reduced ejection fraction (HFmrEF): LVEF 41–49 %;

c) heart failure with preserved ejection fraction (HFpEF): LVEF $\geq$ 50 %.

5. New York Heart Association (NYHA) functional classification (the NYHA classification grades the severity of HF symptoms based on functional limitation):

a) **NYHA Class I** — no limitation of physical activity. Ordinary physical activity does not cause undue breathlessness, fatigue, or palpitations;

b) **NYHA Class II** — slight limitation of physical activity. Comfortable at rest. Moderate physical exertion leads to breathlessness, fatigue, or palpitations;

c) **NYHA Class III** — marked limitation of physical activity. Comfortable at rest. Minimal exertion leads to breathlessness, fatigue, or palpitations;

d) **NYHA Class IV** — inability to carry on any physical activity without discomfort. Symptoms present at rest and worsen with any physical exertion.

6. Staging classification (Strazhesko–Vasilenko, as adapted in the Russian and Belarusian traditions):

a) **stage I** — no symptoms at rest. Only intensive physical exertion leads to shortness of breath on exertion;

b) **stage IIA** — congestion in one circle of blood circulation (pulmonary ***or*** systemic);

c) **stage IIB** — congestion in both circles of blood circulation (pulmonary ***and*** systemic);

d) **stage III** — structural irreversible changes in target organs (cardiosclerosis, pulmonary fibrosis, liver cirrhosis, ascites, anasarca).

ACUTE RIGHT VENTRICULAR HEART FAILURE

Symptoms are the following: sudden onset of dyspnea, weakness and fatigue, heaviness and pain in the right hypochondrium (hepatic congestion), abdominal distension, peripheral edema (rapidly progressing).

Objective examination: inspection shows cyanosis (may be central or peripheral), jugular venous distension (markedly elevated central venous pressure), peripheral edema of the lower extremities (pedal edema), ascites, anasarca.

Palpation reveals weak pulse (due to low cardiac output), tachycardia, hepatomegaly (tender, enlarged liver with a smooth surface and rounded edge, enlargement of liver dullness), positive hepatojugular reflux, epigastric pulsation (due to an enlarged, hypertrophied RV).

Percussion can find dilation of right ventricle and right relative heart border displaced to the right, changes in pulmonary sound according lung pathology.

Auscultation shows accentuation of the S2 over the pulmonary artery, possible tricuspid regurgitation murmur, right-sided S3 gallop, possible lowered BP.

ACUTE LEFT VENTRICULAR HEART FAILURE

Acute left ventricular failure occurs when the left ventricle muscle is suddenly weakened, rendering the heart unable to pump oxygen-rich blood from the lungs through the body.

Causes of increased pulmonary capillary pressure (normal is 2–10 mm Hg):

– Increased diastolic pressure in the LV (ischaemic heart disease, myocardial infarction, valvular heart disease, hypertensive crisis).

– High load on the myocardium (thyrotoxicosis, anaemia, arrhythmias, IV infusion of large fluid volumes, constrictive pericarditis).

– Increased pressure in the left atrium (mitral stenosis, myxoma of the left atrium).

Acute left ventricular failure symptoms and physical examination are in Table 1.

Table 1

Symptoms and objective examination in case of acute left ventricular failure

Method	Result	
	Cardiac asthma	**Pulmonary edema**
Symptoms	Shortness of breath (dyspnea). Orthopnea. Chest pain (tightness). Wheezing or gasping.	
	Cough (dry)	Cough (with white or pink foamy sputum)
Visual examination	Pale skin or cyanosis, tachypnea	
Palpation	Pulse is weak or/and irregular, tachycardia	
Percussion	Relative heart dullness left border is shifted to the left	
Auscultation	Heart sounds weaken. S3 at the apex (gallop rhythm), murmurs. S2 louder at pulmonary artery. Weaken vesicular breathing	
	Dry rales (wheezing)	Wet rales (crackles), crepitation
Laboratory examination	Biochemical blood test shows increased level of BNP (brain natriuretic peptide)	
Instrumental examination	ECG — left ventricle hypertrophy, arrhythmia, conduction block, non-specific ST/T wave changes. Pulse oximetry — low SpO_2. Echocardiogram — abnormal heart valves, low myocardial contractility (↓EF), high pressure in pulmonary artery	
	Chest X-ray — congestion, pleural effusion, increased cardio-thoracic ratio	
	Cephalization of the lung vasculature is presence of symmetrical homogeneous shadows in the lung roots (“bat wing”)	Bilateral multiply diffuse shadows of varying intensity

The two principal stages are cardiac asthma (A) and pulmonary edema (B):

A — cardiac asthma (interstitial pulmonary edema). High hydrostatic pressure in the lung capillaries leads to fluid movement from the capillaries into the interstitial space.

Symptoms: dyspnea (sudden onset, often nocturnal — paroxysmal nocturnal dyspnea), orthopnea, chest tightness, wheezing, dry cough.

Objective examination: pale skin or cyanosis, tachypnoe, weak or irregular pulse, tachycardia. On percussion: left border of relative cardiac dullness shifted to the left. On auscultation: weakened heart sounds, S3 at the apex (gallop rhythm), possible murmurs; accentuated S2 over the pulmonary artery; weakened vesicular breathing with dry rales (wheezing).

B — pulmonary edema (alveolar edema). When fluid enters the alveolar space, pulmonary edema develops.

Complaints: severe dyspnea, orthopnea, cough producing white or pink foamy sputum.

Objective examination: in addition to findings listed above — wet rales (crackles) and crepitation bilaterally, extensive bubbling rales, severe cyanosis, cold diaphoresis. In auscultation of the lungs: bilateral wet rales, crepitation. Heart sounds are weak, S3 gallop rhythm.

LABORATORY AND INSTRUMENTAL DIAGNOSIS

Laboratory investigation. The diagnosis of heart failure can be challenging because of the frequent overlap of the symptoms of dyspnea and fluid retention with other conditions.

BNP is an effective, rapid laboratory test used to support a heart failure diagnosis, particularly in emergency settings**.** Elevated levels confirm the diagnosis of HF (BNP > 100 pg/mL strongly suggest HF).

Complete blood count is used to exclude anemia and infection.

Electrolytes (K^+, Na^+, Mg^{2+}) are used to identify life-threatening electrolyte disturbances.

Liver function tests (ALT, AST) may be elevated in hepatic congestion.

Troponin is used to detect acute coronary syndrome as a cause of HF.

Instrumental investigation. ***ECG*** may show LV hypertrophy (Sokolow–Lyon index: S(V1) + R(V5 or V6) ≥ 35 mm, R in aVL ≥ 11 mm, left axis deviation), arrhythmias, conduction block, ischemia (ST depression, T wave inversion in lateral leads (I, aVL, V5, V6)) or other abnormalities. A completely normal ECG makes HF unlikely.

Pulse oximetry is a reliable tool in assessing oxygen saturation in patients with HF of different severity. Low SpO_2 (< 94 %) indicates respiratory compromise.

Echocardiography is a critical, non-invasive imaging tool for diagnosing, staging, and managing heart failure by visualizing heart structure and function. It assesses LV systolic function (EF), diastolic function, valvular pathology, chamber dimensions, and estimates pulmonary artery pressure.

Chest X-ray may reveal pulmonary congestion, pleural effusion, increased cardiothoracic ratio, cephalisation of pulmonary vasculature, and "bat wing" alveolar shadowing in pulmonary edema.

TREATMENT

Treatment of AHF requires immediate action and is guided by the clinical signs, hemodynamic profile, and the cause.

General measures:

1. Positioning. The patient should be placed in a sitting or semi-recumbent position to reduce venous return and improve respiratory mechanics.

2. Oxygen therapy. Administer supplemental oxygen to maintain SpO_2 ≥ 95 % (≥ 90 % in patients with COPD).

3. Non-invasive ventilation (NIV) should be initiated early in patients with acute pulmonary edema and respiratory distress. NIV reduces the need for intubation and decreases mortality.

4. Continuous monitoring ECG, blood pressure, respiratory rate, SpO_2, urine output.

AHF requires immediate intravenous (IV) access to administer fast-acting, life-saving therapies.

Treatment of acute left ventricular failure:

1. IV loop diuretics, most common *Furosemide* initial dose 20–40 mg IV bolus, dose should be corrected according urine output. Monitor renal function and electrolytes during therapy.

2. Vasodilators may be considered in patients with SBP > 110 mm Hg to relieve congestion and reduce afterload. *Nitroglycerin*: sublingual 0.5 mg (may repeat every 5–10 minutes), or IV infusion starting at 10–20 mcg/min, titrated up to 200 mcg/min. *Sodium nitroprusside*: IV 0.3–5 mcg/kg/min (primarily for hypertensive emergencies with AHF); requires continuous BP monitoring.

3. Morphine (with caution): 2.5–5 mg IV may be considered in severe anxiety or agitation, but routine use is not recommended due to the risk of respiratory depression and hypotension.

4. Inotropes are considered in patients with severe systolic dysfunction and hypotension causing organ hypoperfusion: *Dobutamine*: 2–20 mcg/kg/min IV. *Levosimendan*: loading dose 6–12 mcg/kg over 10 minutes, then 0.05–0.1 mcg/kg/min for 24–48 hours (may be considered if beta-blocker effect contributes to hypotension). *Milrinone*: 0.375–0.75 mcg/kg/min IV.

5. Vasopressors can be used for persistent hypotension despite fluids and inotropes: *Dopamine*: 3–5 mcg/kg/min (renal dose effect) or > 5 mcg/kg/min (vasopressor effect). *Epinephrine*: 0.05–0.5 mcg/kg/min IV (reserved for refractory shock or cardiac arrest).

6. Mechanical circulatory support is used in case of refractory cardiogenic shock, temporary mechanical circulatory support (intra-aortic balloon pump, extra corporeal membrane oxygenation — ECMO) should be considered as a bridge to recovery, decision, or transplantation.

Treatment of acute right ventricular failure. Treatment of the underlying cause is the most important task (e.g., thrombolysis or thrombectomy for massive pulmonary embolism).

Volume management is used (cautious IV fluid challenge if signs of hypovolemia are present).

Inotropes and vasopressors can be used (see above) for hemodynamic support.

Inhaled nitric oxide or IV prostacyclin may be considered to reduce pulmonary arterial pressure.

CHRONIC HEART FAILURE

LEFT VENTRICULAR HEART FAILURE

Left ventricular failure leads to pulmonary congestion.

Symptoms. Shortness of breath (initially on exertion, later at rest; sensitivity of 89 %), orthopnea — dyspnea in the supine position (specificity of 89 %), paroxysmal nocturnal dyspnea, dry cough or cough with sputum.

Physical examination:

– orthopnea (sitting position to relieve dyspnea);

– pale skin or acrocyanosis, tachypnoea;

– pulse: weak, possibly irregular, tachycardia;

– percussion: left border of relative cardiac dullness shifted to the left;

– auscultation: weakened heart sounds, S3 at the apex (gallop rhythm), murmurs; accentuated S2 over the pulmonary artery; weakened vesicular breathing, dry rales (wheezing), wet rales (crackles), crepitation bilaterally at the lower lung lobes.

RIGHT VENTRICULAR HEART FAILURE

Right ventricular failure results from the RV being unable to pump enough blood into the pulmonary circulation, causing blood to accumulate in the systemic veins.

Symptoms. Pedal edema (increases in the evening; pale or cyanotic skin, cold temperature), heaviness in the right hypochondrium (due to liver congestion and hepatomegaly), abdominal distension (ascites), decreased daily urine output with nocturia.

Physical examination:

– jugular vein distension;

– hepatomegaly: liver is enlarged, surface is smooth, edge is rounded and painful on palpation. Positive hepatojugular reflux;

– peripheral edema, ascites, anasarca (in advanced cases);

– pulse: weak, asymmetric, or irregular, tachycardia;

– percussion: right border of relative cardiac dullness shifted to the right;

– auscultation: weakened heart sounds, S3, murmurs.

BIVENTRICULAR (GLOBAL) HEART FAILURE

Since the most common cause of heart failure is damage to the left ventricle, patients typically develop left ventricular failure first, after which right ventricular failure joins, and biventricular heart failure develops. This stage combines features of both pulmonary and systemic congestion.

CORRELATION OF NYHA CLASSES AND STAGES

NYHA I / Stage I: asymptomatic at rest; only intensive exertion causes symptoms.

NYHA II / Stage IIA: moderate exertion causes breathlessness; congestion in one circle of blood circulation.

NYHA III / Stage IIB: minimal exertion causes symptoms; congestion in both circles of blood circulation.

NYHA IV / Stage III: symptoms at rest, worsening with any exertion; irreversible structural changes in target organs (cardiosclerosis, pulmonary fibrosis, liver cirrhosis, ascites, anasarca).

LABORATORY AND INSTRUMENTAL DIAGNOSIS

Laboratory investigations:

– BNP / NT-proBNP: Elevated levels confirm the diagnosis (BNP > 35 pg/mL suggest HF in chronic ambulatory setting; higher thresholds in acute setting);

– thyroid function (TSH) is used to exclude thyroid disease as a reversible cause;

– complete blood count (anemia can provoke HF);

– renal function (creatinine, urea, eGFR): assess for cardiorenal syndrome;

– electrolytes (Na^+, K^+, Mg^{2+}) should be assessed with diuretic therapy;

– liver function tests (AST, ALT) are assessed in case of hepatomegaly;

– ferritin and transferrin are used to assess iron status (iron deficiency is a common comorbidity).

Instrumental investigation:

– pulse oximetry can show low SpO_2;

– chest X-ray usually reveals pulmonary congestion, pleural effusion, increased cardiothoracic ratio;

– ECG may show LV hypertrophy, arrhythmia, conduction disorders, or other abnormalities. A completely normal ECG makes HF unlikely;

– echocardiography is the gold standard for assessment of HF. Evaluates LVEF (reduced, mildly reduced, or preserved), chamber sizes, wall thickness, valvular function, diastolic function, right heart pressures, and presence of pericardial effusion;

– cardiac MRI may be used to characterize myocardial tissue (fibrosis, infiltration, inflammation);

– coronary angiography is used in patients with suspected ischemic etiology of HF.

TREATMENT

Non-pharmacological measures:

– exercise training / cardiac rehabilitation is recommended for stable patients to improve functional capacity and quality of life;

– dietary advice: sodium restriction (< 2 g/day), fluid restriction in patients with refractory congestion;

– weight monitoring should be regular; patients should report weight gain > 2 kg in 3 days.

Pharmacological measures. ***RAAS blockers*** reduce blood pressure and afterload, decrease sodium and water retention, limit myocardial remodeling. Always start with low doses and increase gradually, monitoring blood pressure, creatinine and potassium.

RAAS blockers include:

– ACE inhibitors (*Enalapril, Ramipril, Perindopril*);

– ARNI (Angiotensin Receptor-Neprilysin Inhibitor), e.g. *Sacubitril/Valsartan*;

– ARB (Angiotensin II receptor blocker), e.g. *Valsartan. Losartan, Candesartan.*

Beta blockers (*Bisoprolol, Carvedilol, Metoprolol, Nebivolol*) slow the heart rate, decrease oxygen demand, protect myocardium from chronic sympathetic overstimulation. Start with low dose and titrate slowly under the pulse rate monitoring.

Mineralocorticoid receptor antagonists **(MRAs)**, e.g. *Spironolactone, Eplerenone.*

Block aldosterone, reducing sodium/water retention and fibrosis. Important to monitor potassium and kidney function to avoid hyperkaliemia.

SGLT2 inhibitors (*Dapagliflozin, Empagliflozin*) were initially antidiabetic medicine, now shown to reduce mortality and HF even in patients without diabetes. Simple regimen: usually one tablet once daily, minimal need for titration.

Loop diuretics (*Furosemide, Torasemide*) are the main medicines for relief of congestion: decrease edema, dyspnea, body weight. Dose is individual and may change depending on symptoms and kidney function. Do not improve survival, but crucial for quality of life.

Other symptomatic options in selected patients include *Ivabradine* (can be used if sinus rhythm and heart rate remains high despite beta blocker), *Digoxin* (sometimes added in patients with sinus rhythm and persistent symptoms or with atrial fibrillation to control ventricular rate).

Surgical treatment for heart failure aims to improve heart function, enhance quality of life, and prolong survival when medication is insufficient. Key options include coronary artery bypass grafting, valve repair/replacement, left ventricular assist devices, and heart transplantation. These procedures are tailored based on the underlying cause, such as coronary artery disease or structural valve issues.

HEART FAILURE PREVENTION

Primary prevention (for patients at risk):

1. Control of hypertension (target BP < 130/80 mm Hg in most patients).
2. Management of diabetes mellitus with SGLT2 inhibitors (which reduce incident HF even before symptoms develop).
3. Treatment of coronary artery disease and dyslipidemia (statins).

4. Smoking cessation.

5. Limitation of alcohol intake.

6. Maintenance of healthy body weight and regular physical activity (at least 150 minutes/week of moderate aerobic exercise).

7. Adequate sleep (7–9 hours/night).

8. Management of obesity and obstructive sleep apnea.

9. These correspond to the AHA's "Life's Essential 8" factors: blood pressure, cholesterol, blood sugar, diet, physical activity, weight, smoking cessation, and sleep.

Secondary prevention (for patients with structural heart disease) — proper treatment of disease.

Tertiary prevention (for patients with HF):

– adherence to medical therapy;

– self-management education (daily weight monitoring, recognition of decompensation signs, medication adherence);

– vaccinations: influenza, pneumococcus, COVID-19;

– cardiac rehabilitation programs;

– avoidance of cardiotoxic substances (NSAIDs, certain chemotherapy agents, excessive alcohol).

ARRHYTHMIAS

Any deviations from the normal rhythm of the heart are called arrhythmias. Cardiac arrhythmias are common in many organic and functional disorders of circulatory system. Arrhythmias are often a manifestation of structural heart disease but may also occur because of abnormal conduction or depolarization in an otherwise healthy heart due to other disorders (endocrine diseases, infection, electrolyte imbalances, etc.).

Standard 12-lead electrocardiography is the primary method for the initial diagnosis of arrhythmias and conduction disorders, allowing precise identification of the type of rhythm disturbance and block.

Echocardiography assesses cardiac structure and function, reveals possible organic causes of arrhythmias and conduction disorders, and evaluates their haemodynamic significance.

24-hour ECG monitoring (Holter ECG) records cardiac rhythm during daily activities, enabling detection of transient or infrequent episodes of arrhythmias and conduction disorders and establishing their relationship with the patient's symptoms.

MECHANISMS OF HEART RHYTHM DISORDERS

Cardiac arrhythmias are common in many organic and functional disorders of the circulatory system. Arrhythmias are often a manifestation of structural heart disease but may also occur because of abnormal conduction or depolarisation in

an otherwise healthy heart due to other disorders (endocrine diseases, infection, electrolyte imbalances, etc.).

The three fundamental electrophysiological mechanisms of arrhythmias are:

1. **Disorders of impulse formation (abnormal automaticity).** *Enhanced automaticity:* an increase in the rate of spontaneous (Phase 4) depolarization in normal or latent pacemaker cells, or the emergence of abnormal automaticity in non-pacemaker cells (e.g., damaged atrial or ventricular myocardium). This mechanism underlies sinus tachycardia, some atrial tachycardia, and accelerated idioventricular rhythms.

Triggered activity: oscillations in membrane potential that occur during or after repolarization (early or delayed depolarizations). If they reach threshold, they trigger a premature action potential. Early depolarizations are associated with long QT syndrome and *torsades de pointes*. Delayed depolarizations are associated with digitalis toxicity and catecholamine excess.

2. **Re-entry mechanism.** The most common mechanism of sustained arrhythmias. It requires: 1) two functionally distinct pathways; 2) unidirectional block in one pathway; 3) slow conduction through the other pathway allowing the originally blocked pathway to recover excitability. This mechanism underlies AV nodal re-entrant tachycardia, atrioventricular re-entrant tachycardia (WPW syndrome), atrial flutter, and many ventricular tachycardias.

3. **Conduction blocks.** Delayed conduction or complete absence of conduction in any part of the cardiac conduction system (SA node, AV node, His bundle, bundle branches). This mechanism underlies heart blocks (sinoatrial block, AV block, His bundle branch block).

COMMON CLINICAL SYMPTOMS OF ARRHYTHMIAS

General symptoms of arrythmia are palpitation, abnormally strong or forceful heartbeats, an irregular heartbeat, chest pain, change in blood pressure, difficulty breathing, dizziness, fainting, anxiety.

Palpitation (awareness of the heartbeat) due to an arrhythmia may be accompanied by weakness, dyspnea, or light-headedness. Hemodynamic disorders are manifested by dizziness, syncope, arterial hypotension, dyspnea, acute heart failure. Changes of rate and regularity of cardiac rhythm are detected by pulse examination, heart auscultation.

EXTRASYSTOLE

Extrasystole (premature contraction) is a premature beat arising from an ectopic focus in the atria, AV junction, or ventricles, occurring before the completion of the normal diastolic pause.

Symptoms. Palpitations, a sensation of accelerated, skipped, or irregular heartbeat. Some patients describe a “flip-flop” sensation or a feeling that the heart “stops” momentarily, followed by a strong beat.

Objective examination. ***Physical examination.*** Extrasystolic beat produces a weaker pulse wave followed by a prolonged (compensatory) pause. If the extrasystole occurs too early, the LV may not have filled sufficiently, and no pulse wave is generated ("missing pulse" or pulse deficit). Auscultation in case of premature contraction reveals one sudden loud first heart sound (due to small diastolic filling of the ventricles).

ECG signs:

1. Premature appearance of the QRS complex.
2. Elongated pause between the extrasystolic and the subsequent normal contraction.
3. Compensatory pause — the sum of pre-extrasystolic and post-extrasystolic intervals.

Complete compensatory pause = 2 normal R-R intervals (typical for ventricular extrasystoles).

Incomplete compensatory pause < 2 normal R-R intervals (typical for atrial extrasystoles).

Premature atrial contraction (atrial extrasystole) (Fig. 1):

1. Premature beat with a P wave and a following QRS complex.
2. The P wave is deformed (different morphology from sinus P wave).
3. The premature QRS complex is unchanged, similar in shape to normal QRS complexes of sinus origin.
4. Incomplete compensatory pause.

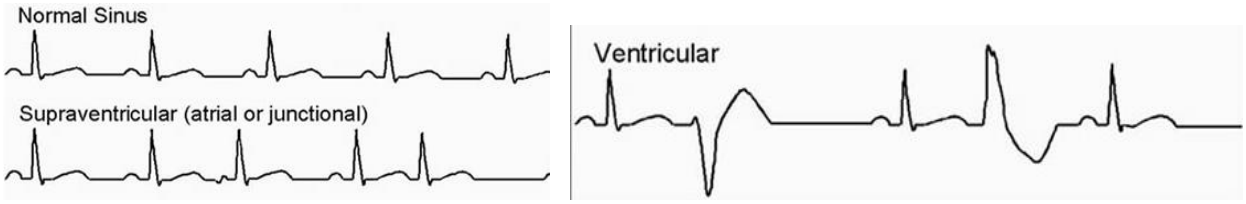


Fig. 1. Premature contraction:
a — atrial extrasystole; *b* — ventricular extrasystole

Premature ventricular contraction (ventricular extrasystole):

1. Absence of a P wave before the premature contraction.
2. The premature QRS complex is deformed and prolonged (> 0.12 s).
3. The ST segment and T wave of the premature ventricular contraction are discordant (opposite in direction) to the main wave of the QRS complex.
4. Complete compensatory pause.

Left-ventricular extrasystole: high R wave in lead III and deep S wave in lead I; high R wave in the right chest leads and broad/deep S wave in the left chest leads.

Right-ventricular extrasystole: high R wave in lead I and deep S wave in lead III; deep S wave in the right chest leads and high R wave in the left chest leads.

PAROXYSMAL TACHYCARDIA

Paroxysmal tachycardia refers to a sudden onset and termination of rapid heart rate, typically exceeding 100–150 beats per minute, which can occur in episodes lasting from seconds to several hours. This condition can arise from various mechanisms including re-entry circuits, automaticity, or triggered activity.

Paroxysmal tachycardia may be atrial, junctional and ventricular, on depends of source of ectopic pacemaker. Atrial and junctional tachycardia are called *Supraventricular paroxysmal tachycardia.*

Causes of paroxysmal tachycardia includes electrolyte imbalances (hypokalemia), ischemic heart disease, stimulants, hyperthyroidism, anxiety or stress.

Symptoms: sudden onset of palpitations, chest pain, dizziness, anxiety, dyspnea. In severe cases: presyncope or syncope, hemodynamic instability.

Physical examination:

1. Inspection: agitation, anxiety, pallor or cyanosis (if hemodynamically unstable).

2. Palpation: rapid pulse (regular or rarely irregular), weak; apex impulse may be displaced left or difficult to palpate due to tachycardia.

3. Auscultation: heart sounds may be difficult to discern; rhythm is regular or irregular depending on type; a “gallop” rhythm may be heard in cases of HF.

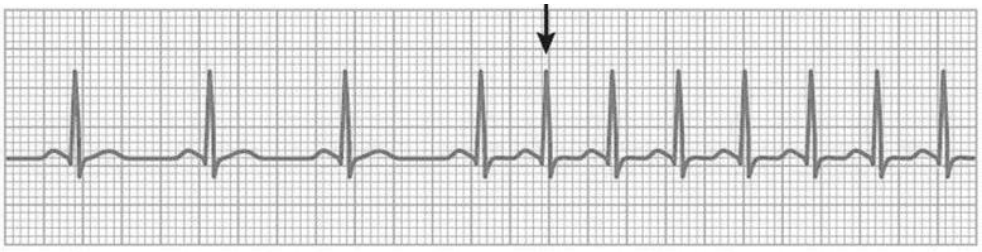
Laboratory findings: electrolyte levels (↓ potassium, ↓ magnesium); thyroid function tests (↓ TSH in hyperthyroidism).

Supraventricular tachycardia (SVT) signs (Fig. 2, *a*):

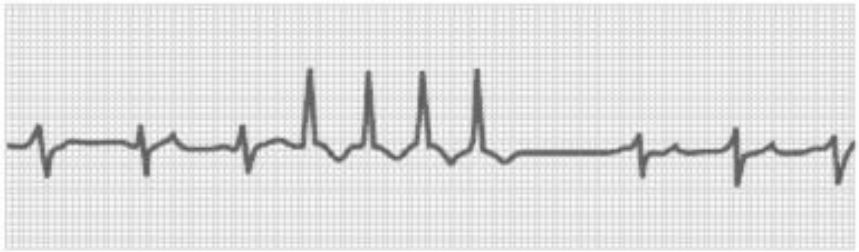
– heart rate typically > 150 bpm;

– narrow QRS complexes (< 0.12 s) with a regular rhythm;

– P waves may be absent or abnormal (hidden within or after QRS/T wave).



a



b

Fig. 2. Paroxysmal tachycardia:
a — supraventricular; *b* — ventricular

Ventricular tachycardia (VT) signs (Fig. 2, *b*):

– heart rate typically 120–250 bpm;

– wide QRS complexes (> 0.12 s) with a regular rhythm;

– may be monomorphic or polymorphic.

P waves are frequently hidden within the broad ventricular complexes, although they can sometimes be identified as bumps or notches (AV dissociation).

Emergency management of paroxysmal SVT. Treatment is based on the duration of the paroxysm, hemodynamic stability, and prior medication response:

1. Vagal maneuver (first-line): carotid sinus massage, Valsalva maneuver (straining), and cold water immersion.

2. Adenosine — 6 mg IV bolus; if ineffective, repeat 12 mg IV bolus (contraindicated in acute coronary syndrome and bronchial asthma).

3. Verapamil 0.25 % — 2–4 mL (5–10 mg) IV slowly.

4. Metoprolol 0.1 % — 2.5–5 mg IV at 1–2 mg/min; may be repeated at 5-minute intervals up to 10–15 mg (max 20 mg).

5. Procainamide — 500–1000 mg (up to 17 mg/kg) IV infusion over 10 minutes.

6. Propafenone — 0.5–1 mg/kg IV over 10–20 minutes (in the absence of structural heart disease).

7. Amiodarone — 300 mg (5 mg/kg) IV over 20 minutes, then infusion up to 1000–1200 mg/day (if above treatments are ineffective).

8. Electrical cardioversion — at 50–360 J (if pharmacological therapy fails or hemodynamic instability is present).

ATRIAL FLUTTER

Atrial flutter is characterized by a rapid, regular atrial rhythm, but faster than usual, with the atria beating more often than the ventricles due to limited conduction through the AV node.

Symptoms: palpitations, fatigue, exertional dyspnea, dizziness, presyncope or syncope.

ECG signs (Fig. 3):

– atrial F waves: Frequent (200–500 per minute), regular, similar to each other, with a characteristic sawtooth shape, best seen in leads II, III, aVF, V1, V2;

– in most cases, a regular ventricular rhythm with regular R-R intervals;

– QRS complex has a normal, unchanged shape;

– each QRS complex is preceded by a certain (often constant) number of F waves (2 : 1, 3 : 1, 4 : 1, etc.);

– in typical 2 : 1 flutter with atrial rate of ~300/min, ventricular rate is ~150/min.

Treatment principles:

1. Rate control: beta-blockers, non-dihydropyridine calcium channel blockers (verapamil, diltiazem) or digoxin.

2. Rhythm control: catheter ablation (specifically cavotricuspid isthmus ablation for typical flutter) is often the preferred definitive treatment due to high success rates.

3. Anticoagulation: the same stroke risk assessment (CHA_2DS_2-VASc score) and anticoagulation recommendations as for AF apply.

4. Electrical cardioversion: effective for termination of flutter episodes.

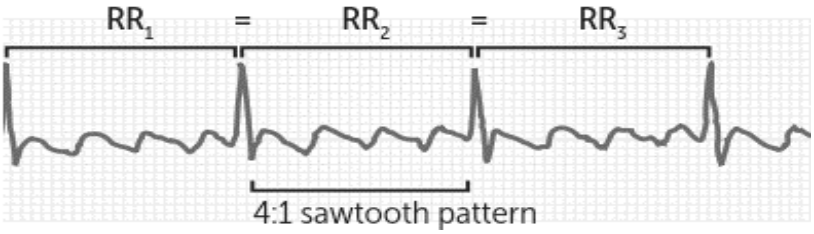


Fig. 3. Atrial flutter

ATRIAL FIBRILLATION

Atrial fibrillation (AF) is the most common sustained cardiac arrhythmia worldwide. In AF, the atria beat irregularly due to chaotic electrical activity.

Symptoms: palpitations, fatigue, exertional dyspnea, dizziness, presyncope or syncope. Some patients with AF are asymptomatic.

ECG signs (Fig. 4):

– P wave is absent in all leads;

– irregular f (fibrillatory) waves of various shape and amplitude with a frequency of 350–600 per minute;

– f waves are best visible in leads V1, V2, II, III, and aVF;

– QRS complex is irregular — all R-R intervals are different (irregularly irregular rhythm);

– QRS complex usually has a normal, unchanged shape (narrow, unless aberrant conduction is present).

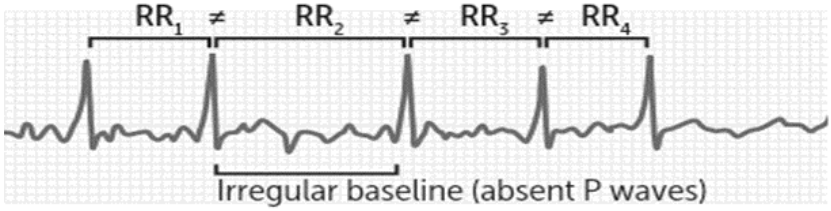


Fig. 4. Atrial fibrillation

Treatment principles:

1. Oral anticoagulants are prescribed for prevention stroke and thromboembolism in patients with AF. Assess stroke risk using the CHA_2DS_2-VASc score. This score is a clinical tool used to estimate the risk of ischemic stroke in patients with non-valvular atrial fibrillation and to guide decisions about

oral anticoagulation therapy. Oral anticoagulation (*Apixaban, Rivaroxaban, Dabigatran*) are recommended for male patients with CHA_2DS_2-VASc ≥ 2 and women with ≥ 3.

2. Rate control: beta-blockers or non-dihydropyridine calcium channel blockers (*Diltiazem, Verapamil*) as first-line; digoxin as second-line. Target resting HR < 110 bpm (lenient strategy) or < 80 bpm (strict strategy, if symptoms persist).

3. Rhythm control: antiarrhythmic drugs (Flecainide, Propafenone — in absence of structural heart disease; Amiodarone — if structural heart disease is present; Dronedarone — in patients without severe HF). Catheter ablation has been upgraded to a first-line rhythm control option in patients with paroxysmal AF. Electrical or pharmacological cardioversion is used for symptomatic episodes.

VENTRICULAR FIBRILLATION

Ventricular fibrillation (VF) is a life-threatening cardiac arrhythmia characterized by rapid, erratic electrical impulses in the ventricles, leading to ineffective quivering of the heart muscle. This results in cessation of effective blood circulation and, if not promptly treated, leads to sudden cardiac arrest and death.

Symptoms: sudden loss of consciousness, absence of pulse, absent breathing, absent heart sounds.

ECG signs (Fig. 5):

– chaotic, irregular waves of varying amplitude;

– no identifiable P waves, QRS complexes, or T waves;

– rate: 150–500 per minute;

– ventricular fibrillation is the main cause of sudden cardiac death.

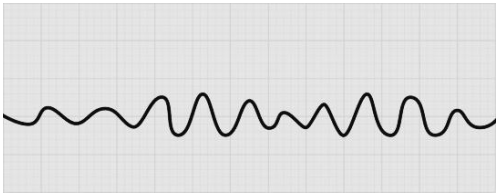


Fig. 5. Ventricular fibrillation

Emergency management of VF:

1. Ensure the patient is supine on a firm surface.

2. Initiate high-quality CPR immediately (compression-to-ventilation ratio 30 : 2).

3. Use an automated external defibrillator as soon as available.

4. **Epinephrine 0.1 %:** 1 mg IV every 3–5 minutes during resuscitation.

5. **Amiodarone 5 %:** 300 mg IV first dose, followed by 150 mg IV if VF persists.

6. **Lidocaine 2 %:** 1–1.5 mg/kg IV can be used in case of allergy to amiodarone or absence of effect with additional doses of 0.5–0.75 mg/kg every 5–10 minutes (maximum total 3 mg/kg).

ATRIOVENTRICULAR BLOCK

Atrioventricular (AV) block is a delay or failure of conduction of electrical impulses from the atria to the ventricles through the AV node and/or His–Purkinje system. AV block is caused by interference with the heart's electrical signals, commonly driven by age-related fibrosis, coronary artery disease, medications (beta-blockers, digoxin), infections (Lyme disease), or congenital heart defects.

First-degree AV-block. ***Symptoms:*** usually absent (asymptomatic).

ECG: PR interval is fixed and prolonged > 0.20 s; all normal P waves are followed by QRS complexes (Fig. 6, *a*).

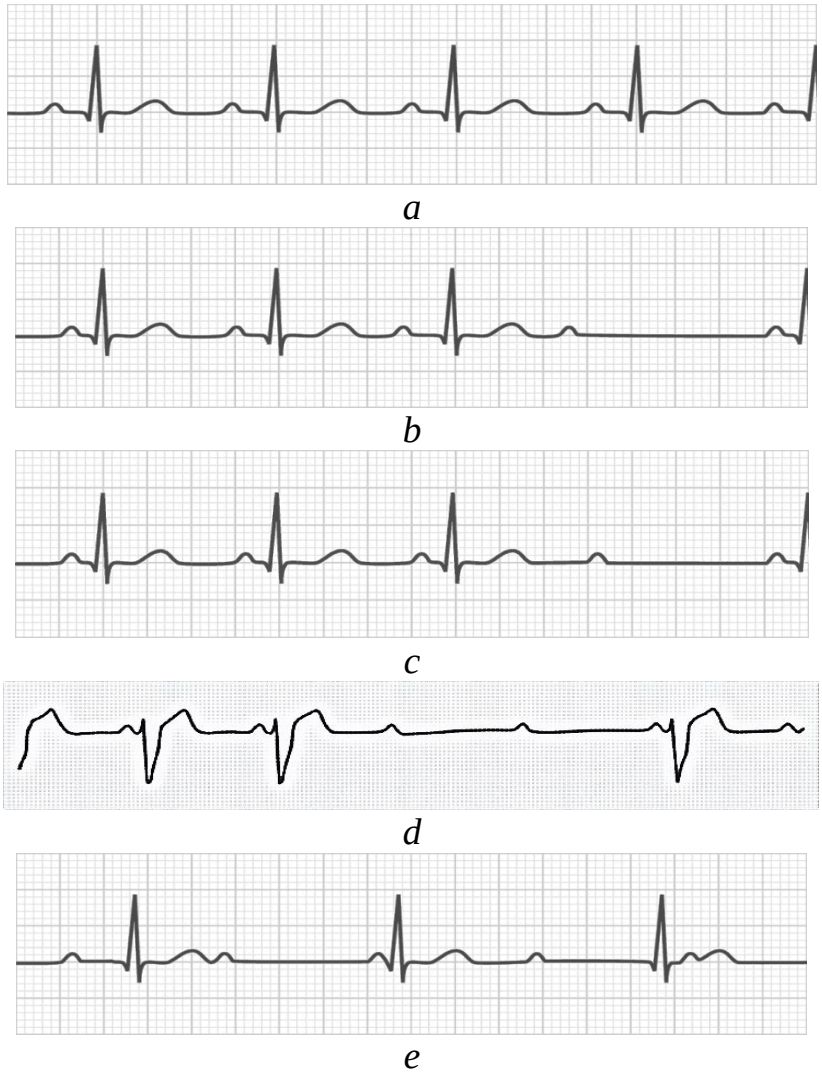


Fig. 6. Types of AV-block:
a — 1st degree AV-block; *b* — 2nd degree AV-block (type 1 (Mobitz 1)); *c* — 2nd degree AV-block (type 2 (Mobitz 2)); *d* — 2nd degree AV-block (high-grade AV block); *e* — 3rd degree AV-block (complete)

Second-degree AV-block. ***Type 1 (Mobitz I / Wenckebach):***

– symptoms: periodic sensation of a missed heartbeat and pulse beat. Usually benign;

– ECG: PR interval progressively lengthens until a nonconducted P wave occurs (one beat is “dropped”) — the Wenckebach phenomenon (Fig. 6, *b*).

Type 2 (Mobitz II):

– symptoms: periodic missing of heartbeats. Dizziness, presyncope, and syncope if several consecutive beats are dropped;

– ECG: PR interval remains constant (normal or prolonged). Beats are intermittently nonconducted (dropped QRS complexes). Block may present as a single nonconducted P wave or a repetitive pattern (2 : 1, 3 : 1, etc.) (Fig. 6, *c*).

High-grade AV-block: two or more consecutive blocked P waves or every second heartbeats are missed (Fig. 6, *d*).

Third-degree (complete) AV-block:

– symptoms: dizziness, fatigue, confusion, syncope (Morgagni–Adams–Stokes attacks), chest pressure or pain, dyspnea;

– physical examination: slow pulse (typically 20–40 bpm), possible cyanosis, cold/clammy skin, signs of heart failure if severe;

– ECG (Fig. 6, *e*);

– complete dissociation between P waves and QRS complexes (AV dissociation);

– P waves occur at a normal atrial rate (60–100/min);

– more P waves than QRS complexes;

– regular QRS complexes at a slower rate (typically 20–40/min);

– QRS morphology: narrow if escape rhythm is junctional; wide if escape rhythm is ventricular;

– constant PR interval is absent (PR varies randomly because P and QRS are independent);

– P waves may occur on any part of the ECG tracing.

Emergency management of third-degree AV-block:

1. Positioning: supine position to optimise venous return. Prepare for CPR if needed. Continuous cardiac monitoring. IV access. Oxygen therapy.

2. **Atropine:** 0.5 mg IV every 3–5 minutes as needed, up to a total of 3 mg.

3. **Dopamine:** 2–20 mcg/kg/min IV infusion, or **Epinephrine:** 1 mg IV every 3–5 minutes if associated cardiac arrest or severe bradycardia.

4. Definitive treatment:

– temporary transvenous pacing if medical therapy is ineffective;

– permanent pacemaker implantation for most cases.

HIS BUNDLE BRANCH BLOCK

His bundle branch block is a conduction delay or block in the right or left bundle branch, resulting in altered ventricular depolarization and characteristic QRS changes.

Symptoms: typically, absent (asymptomatic). Bundle branch block is usually discovered incidentally on routine ECG. Symptoms, if present, are related to the underlying cardiac disease rather than the conduction defect itself.

General ECG signs of bundle branch blocks:

– P wave is unchanged;

– ventricles contract rhythmically by the impulse from the sinus node;

– QRS complexes are markedly prolonged ≥ 0.12 s (complete block) or 0.10–0.12 s (incomplete block);

– the shape of the ventricular complexes depends on which bundle branch is blocked.

Right bundle branch block (RBBB):

– the QRS complex appears as the letter "M" (rSR', rsR') in leads V1–V2. The second R-wave is always larger than the first R-wave;

– S-wave is wide and notched in leads V5, V6, and aVL;

– V1–V2 leads show downsloping ST-segments and inverted T-waves (discordant to the terminal QRS deflection);

– complete RBBB: QRS > 0.12 s. Incomplete RBBB: QRS 0.11–0.12 s (Fig. 7, *a*).

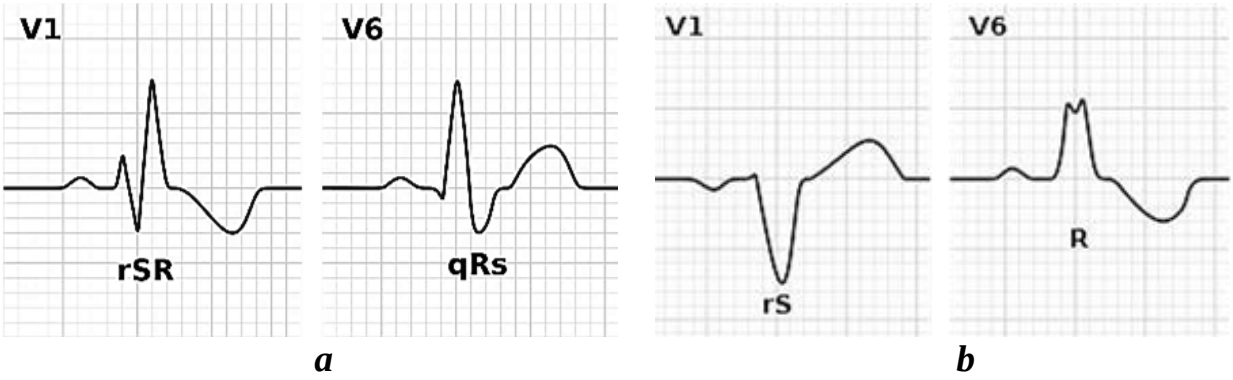


Fig. 7. His bundle branch block:
a — right bundle branch block; *b* — left bundle branch block

Left bundle branch block (LBBB):

– complete LBBB: QRS > 0.12 s;

– wide, notched R wave in leads I, aVL, V5, and V6;

– QRS complex has the shape QS or rS in leads V1, V2, III, aVF (S wave is wide and notched);

– absence of Q wave in leads I, V5, and V6;

– ST segment and T wave are generally opposite in direction to the main QRS deflection (discordant);

– the mean QRS axis turns to the left (sometimes to the right) (Fig. 7, *b*).

SELF-MONITORING OF TOPIC ACQUISITION

TESTS

1. Heart failure is best defined as:

a) any arrhythmia with heart rate > 100/min;

b) a condition in which the heart cannot pump enough blood to meet the body's needs at rest or during stress;

c) any structural abnormality of heart valves;

d) any increase in blood pressure above 140/90 mm Hg;

e) a transient reduction of cerebral blood flow leading to syncope.

2. Which combination correctly describes the main clinical manifestations of left ventricular heart failure:

a) orthopnea, paroxysmal nocturnal dyspnea, pulmonary crackles;

b) peripheral edema, hepatomegaly, ascites;

c) polyuria, polydipsia, weight loss;

d) claudication, cold extremities, absent peripheral pulses;

e) jaundice, pruritus, dark urine?

3. Which statements regarding heart failure classification by LVEF are correct:

a) HFrEF is defined as LVEF ≤ 40 %;

b) HFmrEF is defined as LVEF 41–49 %;

c) HFpEF is defined as LVEF ≥ 50 % with evidence of structural heart disease and/or diastolic dysfunction;

d) HFrEF is defined as LVEF ≥ 50 %?

4. Typical findings of right ventricular heart failure on physical examination include:

a) jugular venous distension;

b) hepatomegaly with a smooth, tender liver edge;

c) positive hepatojugular reflux;

d) bilateral basal crackles as the only sign;

e) peripheral edema, ascites in advanced cases.

5. Which statements about BNP in heart failure are true:

a) elevated levels support the diagnosis of heart failure;

b) BNP levels decrease during acute decompensation;

c) BNP reflect myocardial wall stress;

d) normal natriuretic peptide levels make HF very unlikely;

e) BNP is specific only for renal failure?

6. Which statements about cardiac asthma are correct:

a) it is a manifestation of acute left ventricular failure with interstitial pulmonary edema;

b) it typically presents with paroxysmal nocturnal dyspnea and orthopnea;

c) cough with pink frothy sputum is the main early symptom;

d) on auscultation, dry wheezing rales may be heard over the lungs;

e) it is caused by a sudden decrease in right ventricular contractility?

7. ECG signs characteristic of atrial fibrillation include:

a) absence of P waves in all leads;

b) irregular f-waves with frequency 350–600/min;

c) regular R–R intervals;

d) normal, narrow QRS complexes in most cases;

e) typical F-waves in leads II, III, aVF.

8. Which ECG features are typical for ventricular extrasystole (premature ventricular contraction):

a) premature QRS complex without preceding P wave;

b) narrow, normal-shaped QRS complex;

c) wide, deformed QRS complex (≥ 0.12 s);

d) ST segment and T wave discordant to the main QRS deflection;

e) incomplete compensatory pause equal to < 2 normal R–R intervals?

9. Which statements about third-degree (complete) AV-block are correct:

a) there is complete dissociation between P waves and QRS complexes;

b) atrial rate is faster than ventricular rate;

c) every P wave is followed by a QRS complex with constant PR interval;

d) ventricular rate is typically 20–40/min;

e) this condition often requires pacemaker implantation as definitive treatment?

10. Which clinical features are most typical for cardiogenic shock due to acute heart failure:

a) severe hypotension with signs of peripheral hypoperfusion (cold, clammy skin, cyanosis);

b) warm, flushed skin with bounding pulse;

c) oliguria, elevated lactate;

d) moist skin, tachycardia, weak heart sounds on auscultation;

e) stable blood pressure with isolated tachycardia?

Correct answers: 1 — b; **2** — a; **3** — a, b, c; **4** — a, b, c, e; **5** — a, c, d; **6** — a, b, d; **7** — a, b, d; **8** — a, c, d; **9** — a, b, d, e; **10** — a, c, d.

CLINICAL CASES

Clinical case 1. A 23-year-old female student is brought to the clinic after a brief loss of consciousness in a crowded, poorly ventilated bus. Just before the episode, she noted a feeling of heat, sweating, nausea, dimming of vision, and ringing in the ears. She regained consciousness spontaneously after a few seconds; there were no convulsions. After the episode, she feels weakness and cold sweat. On examination: skin is pale, BP 100/60 mm Hg, HR 68/min, respiratory rate 16/min, no focal neurological signs. ECG: sinus rhythm, no pathological changes.

Questions:

1. Formulate a preliminary diagnosis.
2. List the main causes of this condition.
3. What is the management strategy and first aid in this situation?

Clinical case 2. A 67-year-old man complains of dyspnea when walking on flat ground for 100–150 m, intermittent ankle swelling in the evening, palpitations, and easy fatigability. History: anterior myocardial infarction 5 years ago, arterial hypertension. On examination: orthopnea, pale skin, acrocyanosis, edema of the lower legs and feet, respiratory rate 22/min, HR 88/min, regular rhythm, BP 130/80 mm Hg. Lungs: decreased vesicular breath sounds, a few moist rales in the lower lung fields. The liver protrudes 3 cm below the costal margin. ECG shows signs of previous anterior MI; echocardiography reveals a left ventricular ejection fraction of 35 %.

Questions:

1. Formulate a preliminary diagnosis.
2. Which additional laboratory and instrumental tests are indicated?
3. What is the management strategy in this situation?

Clinical case 3. A 74-year-old woman complains of sudden-onset palpitations, dyspnea on mild exertion, irregular heartbeat, and weakness. History: long-standing arterial hypertension, type 2 diabetes mellitus, ischemic stroke 3 years ago. On examination: moderate general condition, pale skin, respiratory rate 20/min. Pulse 130/min, irregular, pulse deficit present. BP 140/85 mm Hg. On auscultation: heart sounds are muffled, rhythm is completely irregular. ECG: absence of P waves, presence of f waves, irregular R–R intervals, ventricular rate about 130/min.

Questions:

1. Formulate a preliminary diagnosis.
2. Assess the stroke risk using the CHA_2DS_2-VASc score (list the risk factors present in this patient).
3. What is the management strategy in this situation?

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CONTENTS

Учебное издание

Доценко Эдуард Анатольевич
Шолкова Мария Владимировна
Репина Юлия Викторовна
Антонович Мария Николаевна

ОСТРАЯ И ХРОНИЧЕСКАЯ СЕРДЕЧНАЯ НЕДОСТАТОЧНОСТЬ. НАРУШЕНИЯ СЕРДЕЧНОГО РИТМА И ПРОВОДИМОСТИ

ACUTE AND CHRONIC HEART FAILURE. CARDIAC ARRHYTHMIAS AND CONDUCTION DISORDERS

Учебно-методическое пособие

На английском языке

Ответственный за выпуск Э. А. Доценко
Переводчик М. В. Шолкова
Компьютерная вёрстка Н. М. Федорцовой

Подписано в печать 05.06.26. Формат 60×84/16. Бумага писчая «Марафон Бизнес».
Ризография. Гарнитура «Times».
Усл. печ. л. 1,86. Уч.-изд. л. 1,56. Тираж 70 экз. Заказ 297.

Издатель и полиграфическое исполнение: учреждение образования
«Белорусский государственный медицинский университет».
Свидетельство о государственной регистрации издателя, изготовителя,
распространителя печатных изданий № 1/187 от 24.11.2023.
Ул. Ленинградская, 6, 220006, Минск.