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EMERGENCY IN INTERNAL MEDICINE

Учебно-методическое пособие

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ABBREVIATIONS

ABC — airway, breathing, circulation
ACE — angiotensin-converted enzyme
BNP — brain natriuretic peptide
BP — blood pressure
CAD — coronary artery disease
CPR — cardiopulmonary resuscitation
CPR — cardiopulmonary resuscitation
ECG — electrocardiogram
GIB — gastrointestinal bleeding
HR — heart rate
ICU — intensive care unit
IV — intravenous
LV — left ventricle
MI — myocardial infarction
NSTEMI — non-ST-segment elevation myocardial infarction
SpO_2 — oxygen saturation
STEMI — ST-segment elevation myocardial infarction
TSH — thyroid stimulating hormone
VF — ventricular fibrillation

EXPLANATORY NOTE

Total duration of classes: 9 hours.

The purpose of the class: to teach students the most common emergency in internal medicine; to teach diagnostics and treatment in emergency cases.

Objectives of the class. In the dynamic field of internal medicine, the ability to respond effectively to emergencies is paramount. Physicians are often at the forefront of patient care, managing complex cases that require swift decision-making and a comprehensive understanding of various medical conditions. The urgency of emergencies in internal medicine not only tests the clinical acumen of healthcare professionals but also highlights the critical importance of teamwork, communication, and continuous learning.

Emergencies in internal medicine encompass a wide range of scenarios, from acute exacerbations of chronic diseases to life-threatening conditions such as myocardial infarction, asthma attack and gastrointestinal bleeding. Each situation

demands a thorough assessment, rapid intervention, and the ability to prioritize care based on the severity of the patient's condition. This fast-paced environment fosters a culture of resilience and adaptability, essential traits for any aspiring physician.

Moreover, the management of emergencies in internal medicine emphasizes the significance of a holistic approach to patient care. Physicians must consider not only the immediate medical needs but also the psychological and social factors that may impact patient outcomes. This comprehensive perspective is vital in ensuring that patients receive the best possible care during critical moments.

The field of internal medicine is continuously evolving, with advancements in technology and treatment protocols. Staying abreast of the latest developments is crucial for practitioners, as it directly influences their ability to provide high-quality care in emergencies.

In conclusion, the realm of emergencies in internal medicine is both challenging and rewarding. It offers a unique opportunity for healthcare professionals to make a significant impact on patient lives during their most vulnerable moments. By embracing the challenges of this field, aspiring internal medicine physicians can cultivate their skills, enhance their knowledge, and ultimately contribute to the advancement of patient care in emergency settings.

Issues of discussion:

1. Bronchial asthma attack: clinical manifestations, principles of treatment.
2. Hemoptysis and pulmonary hemorrhage: principles of treatment and prevention.
3. Foreign body in the airways.
4. Acute respiratory failure: clinical manifestations, principles of treatment.
5. Hypertensive crisis: clinical manifestations, principles of treatment.
6. Angina pectoris: clinical manifestations, principles of treatment.
7. Acute left ventricular failure: clinical manifestations, principles of treatment.
8. Myocardial infarction: clinical manifestations, principles of treatment.
9. Acute circulatory failure: fainting, collapse: clinical manifestations, principles of treatment.
10. Life-threatening cardiac rhythm and conduction disorders (ventricular fibrillation, asystole, paroxysmal tachycardia, third-degree atrioventricular block).
11. Cardiopulmonary resuscitation.
12. Gastrointestinal bleeding: clinical manifestations, principles of treatment.
13. Hyperglycemic coma: clinical manifestations, principles of treatment.
14. Hypoglycemic coma: clinical manifestations, principles of treatment.
15. Acute urticaria, Quincke's edema: clinical manifestations, principles of treatment and prevention.
16. Anaphylactic shock: clinical manifestations, principles of treatment and prevention.

ASTHMA ATTACK

An asthma attack is a sudden worsening of asthma symptoms (Table 1). Status asthmaticus is a severe, prolonged asthma exacerbation that is unresponsive to repeated doses of standard bronchodilators and may lead to respiratory failure.

Patient's complaints (symptoms):

1. Expiratory dyspnea. Orthopnea (difficulty breathing when lying flat).
2. Chest tightness or pressure (often described as a constricting sensation).
3. Wheezing (although, as we'll discuss, the absence of wheezing can be a concerning sign).
4. Cough (may be productive of thick, tenacious sputum, or non-productive).
5. Difficulty speaking in full sentences.

Table 1

Objective examination in case of asthma attack

Method	Result
Inspection	The patient typically appears anxious, distressed, and diaphoretic (sweating profusely). Forced position: (leaning forward with hands on knees or a table) in an attempt to maximize accessory muscle use and lung expansion. They may be unable to lie flat (orthopnea). Pallor (paleness) or cyanosis (bluish discoloration, especially of the lips and nail beds) skin and mucous membranes (indicates hypoxemia). Chest configuration: may appear hyperinflated (barrel chest). Marked use of accessory muscles of respiration (sternocleidomastoid muscles, intercostal muscles (nasal flaring)
Palpation	Decreased or absent vocal/tactile fremitus due to hyperinflation and air trapping
Percussion	Hyperresonance percussion sound bilaterally. Reduced chest expansion bilaterally
Auscultation	Wheezing is present, but in severe status asthmaticus, breath sounds may be markedly diminished or even «absent» («silent chest»). Prolonged expiratory phase. Tachycardia
Instrumental test	Pulse oximetry: low oxygen saturation ($SpO_2 < 90$ % on room air). ↓BP

Emergency management algorithm:

1. Position the patient upright: elevate the patient to a sitting or semi-Fowler's position to optimize lung expansion.

2. Oxygen: administer high-flow supplemental oxygen via non-rebreather mask to maintain $SpO_2 \geq 90$ %.

3. Administer rapid-acting bronchodilator: prescribe one puff of a short-acting beta-2 agonist inhaler (albuterol/salbutamol) every 30–60 seconds, up to a maximum of 10 puffs. Nebulized form of bronchodilator can be used.

4. Anticholinergics: ipratropium bromide provides additional bronchodilation. Administer 0,5 mg via nebulizer every 20 minutes for the first hour.

5. Corticosteroids: methylprednisolone intravenously (IV): 1–2 mg/kg IV bolus, then 0,5–1 mg/kg IV every 6 hours. Alternatives include IV hydrocortisone, prednisone orally (if IV access is difficult).

6. Magnesium Sulfate 2 g IV over 20 minutes. May provide bronchodilation, particularly in severe cases or those not responding to initial treatment.

7. Epinephrine intramuscularly 0,3–0,5 mg may be used as a temporizing measure while other therapies are being initiated, especially if there is concern for anaphylaxis.

HEMOPTYSIS AND PULMONARY HEMORRHAGE

Pulmonary hemorrhage and hemoptysis mean the expectoration of blood or blood-tinged sputum from the respiratory tract, originating from the larynx, trachea, bronchi, or lung parenchyma. It is crucial to differentiate hemoptysis from pseudohemoptysis (bleeding from the upper respiratory tract or gastrointestinal tract that is coughed up) and hematemesis (vomiting of blood).

Pulmonary hemorrhage (more than 50 ml of blood in 24 hours): a more general term referring to bleeding within the lung parenchyma, which may or may not manifest as hemoptysis. It can range from microscopic bleeding (diffuse alveolar hemorrhage) to massive, life-threatening bleeding.

Causes of hemoptysis: infectious (bronchitis, bronchiectasis, pneumonia, tuberculosis), neoplastic (bronchogenic carcinoma, pulmonary metastasis from other sites), cardiovascular (congestive heart failure, mitral stenosis, pulmonary embolism/infarction, pulmonary arteriovenous malformation), miscellaneous (idiopathic (as much as 20 % to 30 % of cases), lung contusion, Goodpasture syndrome).

Patient complaints (symptoms):

1. Expectoration of blood or blood-tinged sputum (hemoptysis). Amount of blood may be small or large; massive hemoptysis is defined most commonly as a loss of more than 600 ml of blood in 24 hours.
2. Cough (may be dry or productive). When hemoptysis occurs, it is often associated with a cough.
3. Dyspnea (shortness of breath).
4. Chest pain (may suggest underlying lung disease or pulmonary embolism).
5. Fever (may suggest infection).
6. Dizziness or lightheadedness (if significant blood loss has occurred).
7. Fatigue and weakness. Anxiety.

Objective examination in case of pulmonary hemorrhage presented in Table 2. The differential diagnosis of pulmonary hemorrhage versus gastrointestinal bleeding presented in Table 3.

Table 2

Objective examination in case of pulmonary hemorrhage

Method	Result
Inspection	Confusion in case of hypoperfusion. Patient position: may be sitting upright, leaning forward, or lying on the affected side to minimize aspiration of blood into the unaffected lung. Pallor (paleness) or cyanosis (bluish discoloration) skin and mucous membranes may indicate significant blood loss and hypoxemia. Respiratory rate (tachypnea or bradypnea). Chest wall symmetry and expansion. Signs of underlying lung disease (e.g., barrel chest in COPD)
Palpation	Increased over areas of lung consolidation (e.g., pneumonia) or decreased over areas of pleural effusion or pneumothorax
Percussion	Areas of dullness (consolidation, effusion) or hyperresonance (pneumothorax)
Auscultation	Crackles or rhonchi may be present in the area of the bleeding and can help localize the site of bleeding. Tachycardia. ↓BP
Sputum test	typically appears bright red and may be frothy or bubbly due to the presence of air and mucus. Color may vary from pink to rust-colored, depending on the amount and timing of the bleeding. RBC, WBC (neutrophils in infections), and macrophages may be present. The presence of hemosiderin-laden macrophages can indicate prior bleeding
Instrumental test	Chest X-ray: underlying lung pathology (e.g., pneumonia, lung cavity, pulmonary edema). Bronchoscopy allows direct visualization of the airways to identify the site of bleeding, allows for collection of bronchoalveolar lavage fluid for cytology, microbiology, and cell count

Table 3

The differential diagnosis of pulmonary hemorrhage versus gastrointestinal bleeding

Criteria	Pulmonary hemorrhage	Gastrointestinal bleeding
Source of bleeding	Lungs and airways	Gastrointestinal tract (esophagus, stomach)
Common causes	Trauma. Infections (pneumonia, tuberculosis). Coagulation disorders. Pulmonary embolism. Autoimmune diseases (e.g., vasculitis)	Oral cavity: trauma, dental issues, Peptic ulcers. Esophageal varices. Malignancies. Inflammatory bowel disease. Angiodysplasia

End of the Table 3

Criteria	Pulmonary hemorrhage	Gastrointestinal bleeding
	Symptoms	
Color of blood	Bright red	Oral cavity bleeding: bright red liquid, may be mixed with saliva. Esophageal venous bleeding: dark red (due to venous blood)
pH of blood	> 7 (alkali)	< 7 (acidic)
Texture of blood	Frothy or bubbly (due to mixing with air)	Esophageal venous bleeding: liquid, may be mixed with food particles
Discharge the blood	Cough	Vomiting
Associated symptoms	Shortness of breath. Chest pain. Wheezing	Abdominal pain. Melena
Instrumental investigations	Chest X-ray or CT scan. Bronchoscopy	Liver function tests. Gastroscopy

Emergency management algorithm. The initial management focuses on stabilizing the patient, protecting the airway, identifying the source of bleeding, and controlling the hemorrhage:

1. Airway Management:

– assess and secure the airway. Intubation may be necessary if there is massive hemoptysis or if the patient is unable to protect their airway;

– place the patient in the lateral decubitus position with the affected lung down to prevent aspiration of blood into the unaffected lung;

– suction the airway to remove blood and secretions.

2. Oxygenation: administer supplemental oxygen via nasal cannula or face mask to maintain $SpO_2 \geq 90$ %.

3. Monitor vital signs, oxygen saturation, and level of consciousness.

Pharmacological interventions:

1. Hemodynamic stabilization: IV fluids (e.g., normal saline) to restore intravascular volume. Transfuse blood products (packed red blood cells, fresh frozen plasma, platelets) as needed to correct anemia and coagulopathy.

2. Antifibrinolytic agents: *tranexamic acid* 1 g IV slowly over 10 minutes, followed by 1 g IV every 8 hours. May also be administered via nebulizer (500 mg in 20 mL normal saline).

3. Vasopressors (if hypotension is present). *Norepinephrine* initial dose 0.1–0.5 mcg/kg/min IV, titrate to effect.

4. Bronchodilators (if wheezing is present). *Salbutamol* 2.5–5 mg nebulized every 20 minutes or continuously.

Bronchoscopy allows suctioning of blood and secretions, instillation of vasoconstrictors (e.g., epinephrine), balloon tamponade, endobronchial coagulation. In severe cases surgical interventions is needed.

FOREIGN BODY IN THE AIRWAYS

A foreign body in the airways is defined as any object of organic or inorganic nature that has entered the lumen of the respiratory tract, sufficiently large in size to potentially disrupt the functions of the respiratory system.

Foreign bodies in the trachea and bronchi can be categorized by their origin as exogenous (such as coins, dental crowns, and needles) or endogenous (like fragments of removed tissues and teeth), and further classified as organic (including nuts, seeds, and food) or inorganic (such as metal and glass). Sharp or thin objects may lodge in the laryngeal or tracheal walls, leading to cough, significant pain, and potential inflammatory processes or sclerosis at the penetration site.

Clinical manifestations can vary based on form, size, and nature of the foreign body; location of it; age of the patient, and presence of comorbidities.

Inhalation of a foreign body in adults may lead to coughing, dyspnea, and hemoptysis. A notable symptom of obstruction in the larynx and trachea is stridor: a coarse sound caused by turbulent airflow through a narrowed segment. A key indicator of a foreign body in the trachea is the "clapping" symptom, audible during a cough reflex as the foreign body strikes the vocal cords. If the foreign body is located in the bronchi, symptoms may be mild and resemble.

Emergency care:

1. Self-help techniques: after aspiration, the individual should attempt 4–5 strong coughs without deep breaths. If ineffective, they can perform 4 sharp inward and upward thrusts by placing a fist above the upper abdomen, or bend forward sharply against a solid surface to increase pressure and expel the foreign body.

2. Assistance for others: if self-help is not possible (e.g., in children or weakened adults), assistance is required. Two first aid methods include the "Burattino" method and the Heimlich maneuver.

The "Burattino" method involves positioning a chair with its back against the chest of the victim. The rescuer then grasps the victim by the waistband and collar, flipping them over the back of the chair to create pressure that may help expel the foreign body from the airways.

The Heimlich maneuver involves standing behind a sitting or standing person, wrapping your arms around their waist, and applying pressure to the abdomen (between the navel and xiphoid process) with quick upward thrusts. This action increases intra-abdominal pressure to help expel a foreign body obstructing the airway.

3. Immediate action for laryngeal obstruction: if a foreign body is lodged in the larynx, immediate internal finger exploration via the oral cavity is necessary. The tongue should be grasped and pulled outward to facilitate removal or to push the object into the trachea for airflow.

4. Emergency procedures: if removal attempts fail within 2–4 minutes and asphyxia progresses, emergency tracheotomy or cricothyrotomy should be performed to ensure airflow. In cases of absent spontaneous breathing, artificial ventilation and resuscitation protocols must be initiated.

ACUTE RESPIRATORY FAILURE

Acute respiratory failure (ARF) is a life-threatening condition characterized by the lungs' inability to adequately perform their primary functions of oxygenation and carbon dioxide removal, leading to hypoxemia ($PaO_2 < 60$ mmHg) and/or hypercapnia ($PaCO_2 > 50$ mmHg), often with accompanying acidemia (pH < 7.35). This gas exchange impairment occurs acutely, often developing within minutes to hours. The clinical presentation of ARF is variable, dependent on the underlying etiology, the speed of onset, and the patient's pre-existing respiratory and cardiac function. A comprehensive assessment is crucial for accurate diagnosis and management.

Patient's complaints (symptoms):

1. Dyspnea, ranging from mild exertional dyspnea to severe breathlessness at rest.
2. Orthopnea (difficulty breathing when lying flat).
3. Chest pain (may suggest cardiac ischemia or pulmonary embolism).
4. Cough (may be productive or non-productive).
5. Fatigue and weakness.
6. Anxiety and restlessness (often early signs of hypoxemia).

Physical examination, laboratory and instrumental examination in case of acute respiratory failure present in Table 4.

Table 4

Objective examination in case of acute respiratory failure

Method	Result
Inspection	Level of consciousness: signs of confusion, agitation, lethargy, or coma. Forced position: may be sitting upright or leaning forward (tripod position) to maximize lung expansion. They may be unable to lie flat (orthopnea). Pallor (paleness) or cyanosis (bluish discoloration, especially of the lips and nail beds) skin and mucous membranes (indicates hypoxemia).

End of the Table 4

Method	Result
	Depth of respiration (shallow or deep). Use of accessory muscles of respiration (sternocleidomastoid, scalene, intercostal retractions, nasal flaring). This indicates increased work of breathing. Chest wall symmetry and expansion (asymmetry in case of pneumothorax or pleural effusion)
Palpation	Increased vocal fremitus suggests lung consolidation (e.g., pneumonia); decreased tactile fremitus suggests pleural effusion or pneumothorax. Respiratory rate (tachypnea > 20 breaths/min is common; bradypnea < 12 breaths/min is a sign of impending respiratory arrest)
Percussion	Dullness in case of pneumonia, pulmonary edema, pleural effusion. Hyperresonant sound in case of emphysema, pneumothorax
Auscultation	The results will depend on the disease. Wheezing in case of bronchial obstruction (asthma). Week vesicular breath sounds in case of emphysema, pneumothorax. Crackles in case of pneumonia, pulmonary edema. Tachycardia, ↓BP
X-ray	The results will depend on the disease

Emergency management algorithm:

1. Ensuring airway patency. Place the patient in an upright position (if tolerated) to maximize lung expansion. If hypotensive, supine with legs elevated.

Normalization of sputum drainage: stimulation of natural expectoration and artificial removal of sputum (postural drainage, massage, stimulation and imitation of coughing, lung lavage, and suctioning of sputum).

2. Continuous monitoring of vital signs (heart rate, BP, respiratory rate, SpO_2, level of consciousness) is essential.

3. Oxygen therapy: supplemental oxygen via nasal cannula or face mask. Titrate oxygen flow to achieve a target SpO_2 of 90–94 %. If the patient is deteriorating despite initial interventions, prepare for intubation and mechanical ventilation.

4. Artificial and assisted lung ventilation. Intubation and mechanical ventilation is done in case of failure of non-invasive measures, worsening respiratory distress, altered mental status, hypoxemia and hypercapnia with respiratory acidosis.

Pharmacological interventions:

1. Rapid-acting bronchodilator: short-acting beta-2 agonist (albuterol/salbutamol) every 30–60 seconds, up to a maximum of 10 puffs.

2. Anticholinergics: *ipratropium bromide* 0,5 mg via nebulizer every 20 minutes for the first hour.

3. Corticosteroids: *methylprednisolone* IV 1–2 mg/kg IV bolus, then 0,5–1 mg/kg IV every 6 hours. Alternatives include IV *hydrocortisone, prednisone* orally.

4. Diuretics (for pulmonary edema): *furosemide* 20–40 mg IV.

5. Antibiotics (for pneumonia): select antibiotics based on suspected pathogen.

6. Vasopressors (for hypotension): *norepinephrine* initial dose 0.1–0.5 mcg/kg/min IV, titrate to effect.

7. Sedatives (for agitation): *lorazepam* 0.5–2 mg IV for anxiety or agitation.

HYPERTENSIVE CRISIS

Hypertensive crisis is a complication of hypertension, when blood pressure rises quickly and severely, as a systolic blood pressure > 180 mm Hg or a diastolic blood pressure > 120 mm Hg.

Hypertensive crisis can be classified depending on end-organ involvement including cardiac, renal, and neurologic injury as follows:

– hypertensive urgency (absence of acute target organ disease);

– hypertensive emergency (with signs of damage to target organs).

Patient's complaints and objective examination present in Table 5.

Table 5

Symptoms and objective examination in case of hypertensive crisis

System	Symptoms	Objective examination
Hypertensive urgency		
Neurological system	Headache (can be severe). Dizziness. Blurred vision	Alert and oriented to person, place, and time. No focal neurological deficits observed. Pupil reaction normal, no signs of papilledema
Cardiovascular system	Palpitations	Elevated blood pressure (typically ≥ 180/120 mmHg). Heart rate may be elevated (tachycardia) but usually within normal limits. Heart sounds normal, accent S2 over the aorta, no gallops or murmurs appreciated. Peripheral pulses intact and symmetric. No signs of heart failure (e.g., no jugular venous distension). No edema or cyanosis
Respiratory system	Shortness of breath (may occur but less common)	Respiratory rate is normal unless there is associated anxiety. Lung auscultation: vesicular breath sounds No signs of respiratory distress or wheezing
Renal system	Mild changes in urine output	Normal creatinine level
Gastrointestinal system	Nausea (rarely vomiting)	Abdomen on palpation is soft, non-tender Normal bowel sounds

End of the Table 5

System	Symptoms	Objective examination
Hypertensive emergency		
Neurological system	Severe headache. Blurred vision due to retinopathy (e.g., retinal hemorrhages, exudates). Confusion, altered mental status or loss of consciousness. Focal neurological deficits (e.g., weakness some musculus/ extremities)	Altered level of consciousness or confusion may be present. Possible focal neurological deficits (e.g., weakness or sensory loss). Signs of papilledema may be observed on fundoscopic examination
Cardiovascular system	Chest pain (may indicate acute coronary syndrome). Arrhythmias. Heart failure symptoms (e.g., edema, severe dyspnea)	Blood pressure often exceeds 180/120 mmHg and may be significantly higher. Heart rate: accent and splitting S2 over the aorta, tachycardia may be present. Possible presence of S3 gallop or murmur indicating heart failure. Jugular venous distension may be evident, suggesting right heart strain. Peripheral pulses may be diminished/asymmetric. Peripheral edema may be present, especially in cases of congestive heart failure
Respiratory system	Severe shortness of breath or acute pulmonary edema	Respiratory rate may be elevated in cases of distress. Adventitious lung sounds (e.g., crackles) may be present if pulmonary congestion is evident. Signs of respiratory distress, such as use of accessory muscles
Renal system	Oliguria or anuria	Elevated creatinine levels indicating acute renal failure
Gastrointestinal system	Gastrointestinal bleeding (in severe cases)	Possible tenderness in cases of acute abdomen or splenic involvement. Increased bowel sounds if there is associated gastrointestinal bleeding

The primary goal of Hypertensive crisis management is to safely reduce the blood pressure. Blood pressure should be decreased slowly, by 25 % over the first hour, then to 160/100 mmHg over next 2–6 hours, then to normal range over 24–48 hours (Table 6). Hypertensive urgency usually treated with oral or sublingual antihypertensive medicines, hypertensive emergency with intravenous injection or infusion of medicines.

Table 6

Hypertensive crisis management

Treatment method	Practical measures
Hypertensive urgency	
Initial assessment	Measure blood pressure, heart rate, respiratory rate, and temperature. Check for any signs of neurological deficits or altered mental status. Assess heart sounds and peripheral circulation
Positioning	Place the patient in a comfortable position, often sitting or semi-Fowler's
Immediate treatment	Administer oral antihypertensive medications: **ACE inhibitors:** captopril 25–50 mg oral/sublingual. **Calcium channel blockers:** amlodipine 5 mg or nifedipine long acting 10–30 mg oral. **β-blockers:** propranolol 10–20 mg oral/sublingual, atenolol 25 mg oral Clonidine 0,075 mg oral/sublingual Pre-existing hypertension: increase doses of current medications
Monitoring	Continuous blood pressure monitoring every 5–15 minutes until stabilized
Education	Inform the patient about lifestyle modifications and the importance of medication compliance. Schedule follow-up appointments to reassess blood pressure and medication effectiveness
Hypertensive emergency	
Initial assessment	Measure blood pressure, heart rate, respiratory rate, and temperature. Check for any signs of neurological deficits or altered mental status. Assess heart sounds and peripheral circulation. Conduct a thorough neurological assessment (checking for altered consciousness, focal deficits). Assess for signs of heart failure or arrhythmias
Positioning	Place the patient in a semi-Fowler's position to ease breathing and support circulation
Immediate treatment	Administer IV antihypertensive medications: **β-blockers:** labetalol started at 20 mg/h infusion. **Calcium channel blockers:** nicardipine started at 3–5 mg/h Nitroprusside started at 0.5–1.5 mcg/kg/min
Monitoring	Continuously monitor vital signs: blood pressure, heart rate, respiratory rate and oxygen saturation. Obtain ECG to assess for arrhythmias or ischemic changes
Specific complication management	Consider CT or MRI if signs of stroke or intracranial hemorrhage are present. Acute coronary syndrome: IV glyceryl trinitrate started at 15–20 mcg/min, aspirin 75–100 mg. Left ventricle heart failure: IV furosemide 40–80 mg. Assess renal function and consider nephrology consultation if acute kidney injury is suspected

ANGINA PECTORIS

Angina pectoris is a clinical syndrome characterized by chest pain or discomfort due to myocardial ischemia. It typically occurs when the heart muscle does not receive enough blood and oxygen, most often related to coronary artery disease (CAD).

Angina pectoris can be classified into several types based on its characteristics, triggers, and underlying pathophysiology. The main classifications see in Table 7. Clinical signs in case of angina pectoris presents in Table 8.

Table 7

Angina pectoris classification

Type	Clinical features
Stable angina	Occurs predictably with exertion or emotional stress and is relieved by rest or nitroglycerin. Episodes typically last a few minutes and have a consistent pattern in terms of frequency, duration, and intensity. It is often associated with fixed coronary artery stenosis
Unstable angina	Is characterized by an increase in the frequency, duration, or intensity of angina episodes, or occurs at rest. This type is more severe and unpredictable, indicating a higher risk of myocardial infarction. It may last longer than stable angina and is not reliably relieved by rest or nitroglycerin
Variant (prinzmetal's) angina	Is caused by coronary artery spasm, leading to temporary ischemia. Episodes often occur at rest, typically in cycles, and may be associated with transient ST-segment elevation on the ECG. It can occur in patients with or without significant coronary artery disease

Table 8

Angina pectoris clinical signs

Type of clinical data	Details
Patient complaints (symptoms)	Sensation of pressure, squeezing, or pain in the chest (retrosternal area), which may radiate to the left shoulder, arm, neck, jaw, or back. The episodes are often triggered by physical exertion, emotional stress, or heavy meals and usually last from a few seconds to 15 minutes. Patients may also experience shortness of breath, fatigue, sweating, nausea, dizziness, or palpitations
Objective signs	During a clinical examination, vital signs may be normal between episodes, but hypertension or tachycardia can be present during an angina attack. Physical examination may reveal signs of associated heart conditions, such as heart murmurs or peripheral edema
ECG changes	During an angina episode, the ECG may show transient ST-segment depression or T-wave inversion, indicating myocardial ischemia (Fig. 1). Returns to baseline often occur after the episode resolves. In stable angina, resting ECG might be normal

End of the Table 8

Type of clinical data	Details
Laboratory and instrumental data	Cardiac biomarkers (troponin, creatinine kinase MB-fraction (CK-MB), myoglobin), are typically normal in stable angina but may be elevated in unstable angina or myocardial infarction. Imaging studies, such as stress tests (exercise or pharmacologic) and coronary angiography, can assess coronary artery patency and ischemic areas

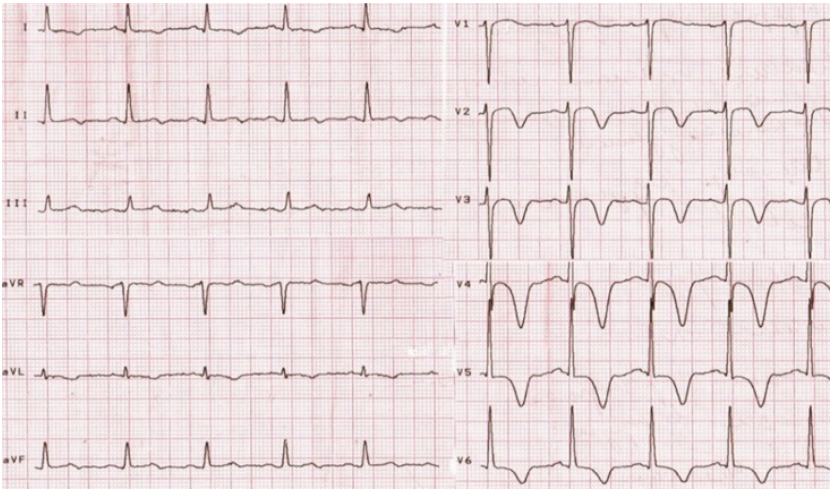


Fig. 1. ECG with T-wave inversion

Angina pectoris management includes both pharmacological and non-pharmacological strategies (Table 9). In summary, angina pectoris is a critical manifestation of coronary artery disease requiring timely diagnosis and comprehensive management to alleviate symptoms and improve quality of life while reducing the risk of myocardial infarction.

Table 9

Angina pectoris management

Treatment method	Practical measures
Lifestyle modifications	Dietary changes (restrict cholesterol consumption), regular physical activity, smoking cessation, weight management
Cardiac rehabilitation	A structured program involving exercise and education supports recovery and promotes heart health

End of the Table 9

Treatment method	Practical measures
Pharmacological treatment:	**Nitrates:** – short-acting nitrates (nitroglycerin 0.5 mg sublingually) provide rapid relief during acute episodes; – long-acting nitrates (isosorbide mononitrate 30–60 mg per os one time a day at morning) are used for maintenance therapy. **Beta-blockers** reduce myocardial oxygen demand by decreasing heart rate and contractility: (metoprolol 25–50 mg per os 2–3 times a day, bisoprolol 5–10 mg once a day). **Calcium channel blockers** help relieve angina by promoting vasodilation and reducing myocardial workload (amlodipine 5–10 mg once a day). **Antiplatelet agents:** aspirin 75–150 mg once a day or clopidogrel 75 mg once a day. **Statins** to manage dyslipidemia and reduce cardiovascular risk: – atorvastatin 10–40 mg once a day; – rosuvastatin 5–20 mg once a day
Revascularization procedures	In cases of significant coronary artery disease, percutaneous coronary intervention or coronary artery bypass grafting may be indicated

ACUTE LEFT VENTRICULAR FAILURE

Acute left ventricle heart failure occurs when the left ventricle muscle is suddenly weakened. In such a case, the heart is unable to pump oxygen-rich blood from the lungs through the body. Stages of left ventricle heart failure are following:

– cardiac asthma (fluid in the interstitial tissue);

– pulmonary edema (fluid in the alveoli).

High hydrostatic pressure in the lung capillaries leads to fluid movement from the capillaries into the interstitial space. It happens at stage of cardiac asthma. Then fluid goes into the lumen of the alveoli pulmonary edema develops. Normal pressure in the lung capillaries is 2–10 mm Hg. Acute left ventricular failure examination data are the following (Table 10). Cardiac causes of an increase pressure are the following:

– increased diastolic pressure in the left ventricle (ischemic heart diseases, myocardial infarction, valvular heart diseases, hypertensive crisis);

– high load on the myocardium (thyrotoxicosis, anemia, arrhythmias, intravenous infusions of large fluid volume, constrictive pericarditis);

– increased pressure in the left atrium: mitral stenosis, myxomas of the left atrium.

The management of acute left ventricle heart failure focuses on alleviating symptoms, improving hemodynamics, and addressing the underlying cause. The treatment approach typically includes pharmacological interventions, non-pharmacological measures, and sometimes invasive procedures (Table 11).

Table 10

Examination in case of acute left ventricular failure

Method	Result	
	Cardiac asthma	**Pulmonary edema**
Complaints (symptoms)	Shortness of breath (dyspnea), orthopnea, chest pain (tightness) Wheezing or gasping	
	Cough (dry)	Cough with white or pink foamy sputum
Inspection	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 tests	Biochemical blood test: ↑ 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

Table 11

Acute left ventricle heart failure management

Treatment method	Practical measures
Non-pharmacological treatment	Treatment is done in ICU Department Continuous monitoring of vital signs, oxygen saturation, and urine output is essential to assess the response to treatment and adjust therapy accordingly
Oxygen therapy	Administer supplemental oxygen (oxygen mask or nasal cannula) to maintain oxygen saturation above 90 %
Positioning	Elevate the head of the bed to improve respiratory comfort and venous return

End of the Table 11

Treatment method	Practical measures
Pharmacological treatment	**Diuretics:** furosemide (lasix) initial dose of 20–40 mg IV bolus; may be increased by 20 mg every 1–2 hours until the desired diuresis is achieved. **Nitroglycerin** initial dose 5 mcg/min IV infusion; may increase by 5 mcg/min every 3–5 minutes to a maximum of 200 mcg/min based on blood pressure and symptom relief. **Morphine** 2–4 mg IV for symptomatic relief of dyspnea; may repeat every 5–30 minutes as needed. Administration: IV route; monitor for respiratory depression. **Sodium nitroprusside:** start at 0.5–1 mcg/kg/min IV infusion; titrate to effect, with a maximum dose of 10 mcg/kg/min, monitor closely for hypotension. **Inotropes** (if needed for low cardiac output): dobutamine. Dosage: start at 2–5 mcg/kg/min IV; may increase to 20 mcg/kg/min based on clinical response. **Anticoagulation** (if indicated): heparin initial bolus of 60–80 units/kg IV (maximum 5,000 units), followed by a continuous infusion of 12–15 units/kg/hour, adjusted based on activated partial thromboplastin time
Mechanical circulatory support	In cases of severe left ventricle failure with cardiogenic shock, consider intra-aortic balloon pump or ventricular assist devices
Revascularization	If acute left ventricle heart failure is due to acute coronary syndrome, urgent coronary angiography and possible percutaneous coronary intervention may be warranted

MYOCARDIAL INFARCTION

The term *"myocardial infarction"* (MI) means ischemic myocardial necrosis due to prolonged ischemia resulting from coronary artery occlusion. Main cause is decreased blood flow in the coronary arteries such that part of the myocardium dies. Myocardial infarction usually results from atherosclerosis, when cholesterol plaques build up in the walls of coronary arteries. MI occurs when a vulnerable atherosclerotic plaque ruptures, leading to platelet activation and aggregation, resulting in the formation of intracoronary thrombus (Fig. 2.). Ischemia may start due to increased oxygen demand (for example, hypertension), or decreased supply (e.g., coronary artery spasm or embolism, arrhythmia, hypotension). Other types of MI include sudden unexpected cardiac death, complications of coronary intervention (percutaneous coronary intervention, stent thrombosis or coronary artery bypass grafting). MI affects predominantly the left ventricle (LV), but damage may extend into the right ventricle (RV) or the atria. Risk factors of Myocardial Infarction are as follows (Table 12).

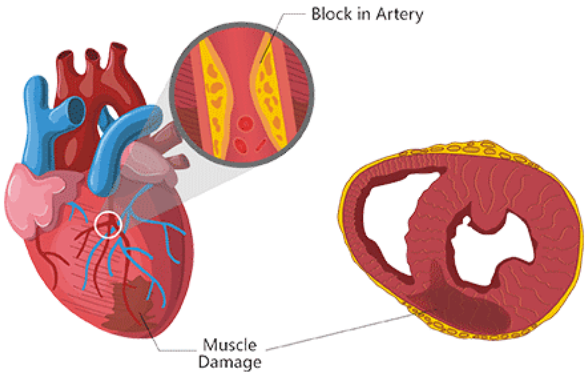


Fig. 2. Myocardial Infarction

Table 12

Risk factors of myocardial infarction

Non-modified	Modified
Age. Family history. Male sex. Race	Smoking. Hypertension. Dyslipidemia. Diabetes mellitus. Obesity, high calorie intake diet. Physical inactivity (sedentary lifestyle). Psychological stress

There are two types of myocardial infarction according ECG-changes:

– *ST-elevation myocardial infarction* (STEMI), when ST-segment at ECG elevates more than 0.1 mV in two or more contiguous leads;

– *non-ST-elevation myocardial infarction* (NSTEMI), this type of MI may have other ECG changes, such as ST-segment depression or T-wave inversion.

MI may be classified by extent:

– *transmural* (it involves the whole thickness of myocardium from epicardium to endocardium and are usually characterized by abnormal Q waves on ECG) (Fig. 3);

– *nontransmural* (infarcts do not extend through the ventricular wall and cause only ST-segment and T-wave (ST-T) abnormalities. Special type of nontransmural MI is subendocardial infarct, it usually involve the inner one third of myocardium, where wall tension is highest and myocardial blood flow is most vulnerable to circulatory change.

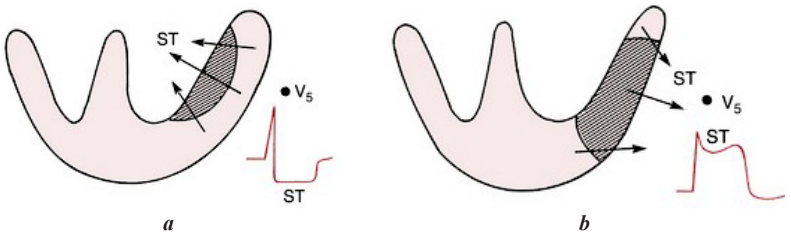


Fig. 3. Myocardial infarction:
a — nontransmural (subendocardial); *b* — transmural

Patient's complaints (symptoms):

1. Retrosternal pain has following characteristics:
   – duration at least 30 min;
   – radiation to the neck, arms, jaw, epigastrium or back (left scapular area);
   – the character (type) of pain is described as crushing, heaviness or like a tight band. Patients classically clinch their fist and hold it on their chest to describe the pain, this gesture called Levine's Sign;
   – worse with physical or emotional exertion;
   – not relieved by rest;
   – nitrate spray (within a couple of minutes) may not always relieve the pain.
2. Anxiety and fear of impending death.
3. Nausea and vomiting.
4. Breathlessness.
5. Collapse/syncope.

Objective examination in case of MI see Table 13.

Table 13

Objective examination in case of myocardial infarction

Method	Result
Visual examination	Skin has a pale color, sweating. Extremities are cold. Low-grade fever develops after several days (resorption of necrotic tissue)
Palpation	Pulse: tachycardia or bradycardia, arrhythmia. Hypotension, low pulse pressure
Percussion	Heart borders may be displaced according previous condition (hypertension, left ventricle hypertrophy)
Auscultation	Heart sounds are weak, especially S1 at the apex, Gallop rhythm (third heart sound). Bilateral lower-lobes lung crepitation due to acute heart failure (lung congestion)

End of the Table 13

Method	Result
Laboratory examination	**CBC:** leukocytosis, increased erythrocytes sedimentation rate. **Biochemical blood test:** cardiac enzymes (leakage of protein from injured cardiac myocytes): – *troponin* rises in 6 hours, peaks in 1–2 days, lasts 2 weeks. It has high sensitivity and specificity; – *creatinine kinase MB-fraction* (CK-MB) rises in 4–6 hours, peaks at 12 hours and at 2 days drops off; – *myoglobin* rises in 2 hours. It has high sensitivity, but low specificity. **Markers of atherosclerotic process:** – total cholesterol level more than 5,2 mmol/L; – low-density lipoprotein level more than 3,5 mmol/L. **ECG:** – ST segment elevation or depression; – pathological Q wave; – T wave flattening or negative
Instrumental examination	Echocardiography shows regional heart wall motion abnormalities. Coronary angiography

MI management includes first aid (oxygen therapy, antiplatelets medicines, nitrates and painkillers), reperfusion therapy, pharmacological and non-pharmacological long-term treatment (Table 14).

Table 14

Myocardial infarction management

Triage to appropriate medical center	Coronary care unit/intensive cardiac care unit
Oxygen therapy	Administer supplemental oxygen (oxygen mask or nasal cannula) to maintain oxygen saturation above 90 %
Antiplatelets	Aspirin 150–300 mg orally, followed 75–100 mg once a day
	Clopidogrel 300–600 mg orally, followed 75 mg once a day
Nitrates	Sublingual nitroglycerine 0,5 mg. Isosorbide dinitrate 0,1 % — 10 ml intravenously slowly (10 mcg/min)
Pain relief	Morphine 5–10 mg intravenously
Reperfusion therapy	*Percutaneous coronary intervention* Coronary angiography is obtained urgently for patients with STEMI. Patients with NSTEMI undergo angiography within the first 24 to 48 hours. Percutaneous coronary intervention (angioplasty, stent placement) restores blood flow. Coronary artery bypass grafting can be done in some cases

End of the Table 14

Triage to appropriate medical center	Coronary care unit/intensive cardiac care unit
	Fibrinolytic therapy (STEMI) Fibrinolytic therapy initiation should not be delayed by waiting for the results of cardiac biomarker testing. The goal is to start fibrinolytic therapy within 10 min of the STEMI diagnosis *(tenecteplase, alteplase, or reteplase)*
Anticoagulants	Enoxaparin 1 mg/kg twice a day subcutaneously or Fondaparinux 2.5 mg/d subcutaneously
Lipid-lowering therapy	Statins: atorvastatin 40–80 mg once a day orally. Ezetimibe 10 mg once a day orally
Beta-blockers	Metoprolol (with no signs of acute heart failure — intravenously), followed 50–100 mg 1–2 times a day orally. Bisoprolol 2,5 mg once a day, carvedilol 3,125 mg twice a day. Dosage increases slowly, according HR
ACE inhibitors	Initial dosage: enalapril 2,5 mg twice a day, lisinopril 2,5 mg twice a day, ramipril 1,25 mg once a day, perindopril 2 mg once a day. Dosage increases slowly, according BP
Non-pharmacological treatment	Smoking cessation, heathy diet, regular exercise, healthy weight, psychosocial management

ACUTE CIRCULATORY FAILURE

Acute circulatory failure is a pathologic state developed due to decreased vessel's tone and arterial hypotension. It can be realized like syncope, collapse or shock.

Syncope (fainting or passing out) is sudden short-term loss of consciousness caused by insufficient blood flow to the brain. Syncope can occur as a result of stress, severe pain, cardiac arrhythmias, structural heart disease (heart valve diseases, ischemic heart diseases, cardiomyopathy), stimulation of carotid sinus, etc. Fainting lasts from a few seconds to one minute, rarely longer.

On examination, the patient's skin is pale, the pulse is rare and faint, blood pressure is low. Heart auscultation can show tachycardia, irregular heartbeats (in case of arrhythmias) or murmurs (in case of heart valve diseases).

Collapse and shock are the forms of acute vascular insufficiency, based on cardiac output decrease. In such a case the cardiac output is not sufficient for normal tissue supply. The causes of collapse and shock are the following: severe bleeding, myocardial infarction, heart rhythm disorders, severe infection, poisoning, trauma.

Collapse is a moderate form of vascular insufficiency (hypotension is the main symptom). Consciousness is always present in case of collapse, but can be confused. Skin is pale and cold, acrocyanosis can present. Blood pressure is low, pulse is frequent and weak.

Shock is the most severe form of vascular insufficiency. Shock leads to the acute metabolic disorders, microcirculation, the organs and systems disturbance. With any type of shock severe hypotension is present; the patient's skin is moist, cold and cyanotic, sometimes with a "marble" pattern. The patient's consciousness is inhibited, but is present, pulse is frequent and thread, breathing is shallow. Heart sounds are weak in auscultation, tachycardia, irregular heartbeats, murmurs or "gallop" can be head.

Acute circulatory failure management. Early and appropriate treatment of acute circulatory failure influences outcomes in critically ill patients. Treatment aims to optimize oxygen delivery and reverse hypoperfusion through volume resuscitation, vasoactive agents for refractory hypotension due to vasodilation, management of cardiac dysfunction, and treatment of the underlying cause (Table 15). Management of shock is best undertaken in a critical care unit.

Table 15

Acute circulatory failure signs and management

System	Clinical signs	Laboratory parameters	Treatment
Respiratory	Tachypnea, shallow breathing, cyanosis	SpO_2 < 94 %	O_2 therapy Invasive ventilation
Cardiovascular	Tachycardia or bradycardia, arrhythmias, pallor, weak radial pulse, hypotension. Heart rate > 100 beats/minute. Respiratory rate > 22 breaths/minute. Hypotension (systolic blood pressure < 90 mm Hg)	SpO_2 < 94 %. BNP > 400 pg/mL	Infusion of IV solution (normal saline, etc.). Dopamine 1–5 μg/kg/min. Epinephrine 0.01–0.03 μg/kg/min. Norepinephrine 0.01–1 μg/kg/min. Levosimendan 6–12 μg/kg for 10 min, then 0.05–0.1 μg/kg/min for 24 to 48 h
Renal	Oliguria, anuria	Creatinine elevation (doubling of creatinine from admission)	Infusion of IV solution (normal saline, etc.). Furosemide 20–60 mg IV
Neurologic	Agitation, anxiety, later dizziness or loss of consciousness	Glasgow Coma Scale < 6	Anesthesia. Immobilization

End of the Table 15

System	Clinical signs	Laboratory parameters	Treatment
Gastrointestinal	Abdominal cramps and spasms, feeding intolerance, GI bleeding	–	Tube feeding
Hepatic	Right upper quadrant tenderness, hepatomegaly, jaundice	Alanine aminotransferase (ALT), aspartate aminotransferase (AST) > 200 IU/L	Hepatoprotective drugs (silymarin)
Hematologic	Endothelial and platelet activation. Hemorragic rash (red and flat: petechiae, purpura, ecchymoses)	Platelets < 50 × 10⁹. Fibrinogen < 100 mg/dL or > 400 mg/dL; D-dimers	Symptomatic treatment
Metabolic	Acidosis, hyperlactatemia	Lactate level	Symptomatic treatment

LIFE-THREATENING CARDIAC RHYTHM AND CONDUCTION DISORDERS

Paroxysmal Tachycardia

Paroxysmal tachycardia refers to a sudden onset of rapid heart rate, typically defined as a heart rate exceeding 100 beats per minute, which can occur in episodes lasting from a few seconds to several hours (Table 16). This condition can arise from various mechanisms, including reentry circuits, automaticity, or triggered activity, and may lead to hemodynamic instability if not promptly managed.

Caused: atrial fibrillation or flutter, electrolyte imbalances, ischemic heart disease, stimulants, hyperthyroidism, anxiety or stress.

Patient Complaints (Symptoms): patients may report sudden loss of consciousness, absence of pulse, and may exhibit signs of impending doom prior to the event. Some may experience chest pain or palpitations before the onset.

Table 16

Objective examination in case of paroxysmal tachycardia

Method	Result
Inspection	Agitation, anxiety, dizziness or loss of consciousness due to hypoperfusion. Patient position: patients may present in a sitting or supine position Skin color: pallor or cyanosis, particularly if hemodynamically unstable
Palpation	Pulse: rapid (rare irregular), weak. Apex impulse may be displaced left or difficult to palpate due to tachycardia

End of the Table 16

Method	Result
Percussion	Typically, not significant in acute assessment
Auscultation	Heart sounds: may be difficult to discern due to tachycardia. Rhythm: regular or irregular depending on the type of tachycardia; may hear a «gallop» rhythm in cases of heart failure
Laboratory test	Electrolyte levels (↓potassium, ↓magnesium). Thyroid function tests (↓TSH in case of hyperthyroidism)
Instrumental test	ECG: rate typically > 100 beats per minute. Characteristic features depend on the type of tachycardia: – **supraventricular tachycardia** (Fig. 4): narrow QRS complexes with a regular rhythm, P waves may be absent or abnormal; – **ventricular tachycardia** (Fig. 5): wide QRS complexes 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 in the ventricular cycles

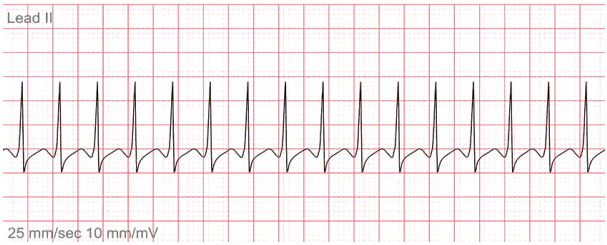


Fig. 4. Supraventricular tachycardia

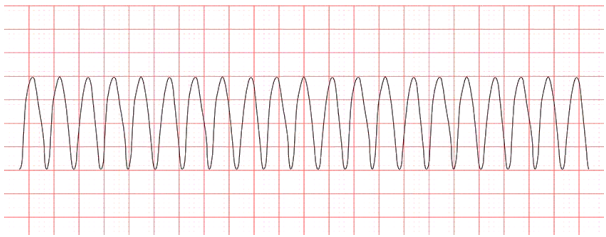
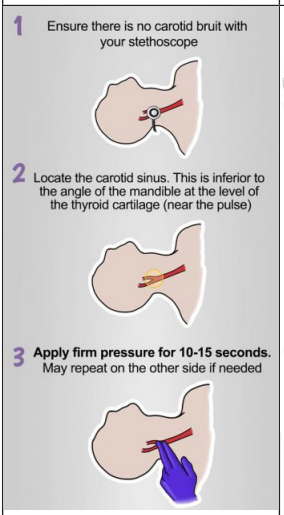
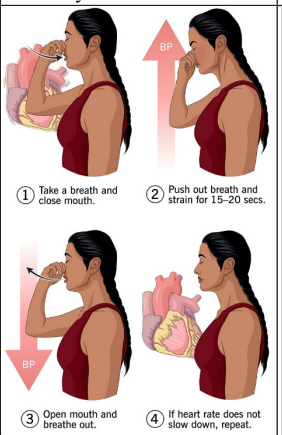
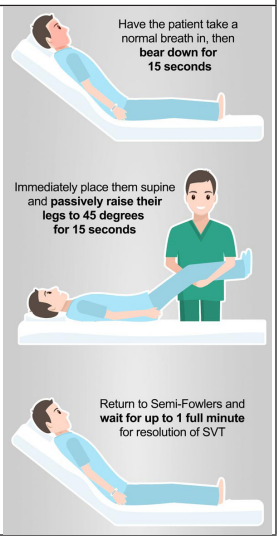


Fig. 5. Ventricular tachycardia

Emergency management algorithm. Positioning: place the patient in a comfortable position, preferably supine, to optimize venous return. **Vagal maneuvers** are techniques used to stimulate the vagus nerve, aiming to terminate certain types of supraventricular tachycardia by slowing down the heart rate (Table 17).

Table 17

Vagal maneuvers

Carotid sinus massage	**Valsalva maneuver**	**Modified Valsalva maneuver**
The patient is positioned supine or sitting comfortably. Doctor locates the carotid sinus, typically located at the level of the thyroid cartilage, just lateral to the trachea. Gentle pressure is applied to one carotid artery for 5–10 seconds while monitoring the patient's heart rate and blood pressure	The patient is instructed to take a deep breath and then forcibly exhale against a closed airway (e.g., by pinching the nose and keeping the mouth closed) for about 10–15 seconds. This increases intrathoracic pressure, which can stimulate the vagus nerve and potentially restore normal heart rhythm	The patient is positioned supine and instructed to perform the Valsalva maneuver as described above. After 15 seconds, the patient is quickly returned to an upright position, which may enhance the effectiveness of the maneuver
		

Treatment of supraventricular tachycardia. Treatment is conducted based on the duration of the paroxysm, hemodynamic stability, and the effectiveness of medications in the patient's history:

1. Verapamil 0.25 % solution, 2–4 mL (5–10 mg) administered slowly IV.

2. Metoprolol 0.1 % solution — initially 2.5–5 mL (2.5–5.0 mg at a rate of 1–2 mg/min); if necessary, the dose may be repeated at 5-minute intervals until a therapeutic effect is achieved or up to 10–15 mg (maximum dose — 20 mg); alternatively, metoprolol 25–50 mg/day orally.

3. Procainamide 500–1000 mg (up to 17 mg/kg) administered IV by infusion over 10 minutes; if there is a risk of hypotension, it may be combined with 1 % mezaton — 0.3–0.5 mL.

4. Propafenone — 0.5–1 mg/kg IV over 10–20 minutes (if necessary, the dose may be repeated for a total of up to 2 mg/kg IV) or propafenone 150–300 mg as a single dose (if necessary, may be repeated after 1–2 hours) in the absence of structural heart disease.

5. Etacizine 50–100 mg as a single dose (if necessary, may be repeated after 1–2 hours) in the absence of structural heart disease, with the addition of a beta-blocker (metoprolol 25–50 mg, bisoprolol 2.5–5 mg).

6. Adenosine — 6 mg administered as a bolus IV; if ineffective, repeat with 12 mg as a bolus IV (contraindicated in acute coronary syndrome/myocardial infarction and bronchial asthma).

7. If the above treatments are ineffective — amiodarone 300 mg (5 mg/kg) over 20 minutes, then IV by infusion at a rate of up to 1000–1200 mg/day.

8. Electrical cardioversion at 50–360 J.

Third-degree atrioventricular block

Third-degree AV block is a severe cardiac conduction disorder characterized by complete dissociation between atrial and ventricular activity, where no atrial impulses conduct to the ventricles (Table 18, Fig. 6). The ventricles are controlled by an escape rhythm originating either from the AV junction or the ventricles.

Caused: age-related fibrosis of the conducting system, ischemic heart disease, MI, myocarditis, cardiac surgery complications, catheter ablation procedures, medication-induced (beta-blockers, calcium channel blockers, digoxin, antiarrhythmics), congenital heart defects, hyperkalemia or hypocalcemia.

Patient Complaints (Symptoms): shortness of breath, chest pain or discomfort, palpitations, dizziness or syncope, fatigue and weakness.

Table 18

Objective examination in case of third-degree AV block

Method	Result
Inspection	Dizziness or loss of consciousness due to hypoperfusion. Patients may present in a supine position due to weakness or syncope. Skin color: cyanosis, particularly in severe cases. Cold, clammy skin in severe cases
Palpation	Pulse: irregular, slow pulse (typically 20–40 beats/minute). Apex impulse: difficult to palpate due to decreased cardiac output
Percussion	Dullness may be noted if there is associated heart failure or pericardial effusion

End of the Table 18

Method	Result
Auscultation	Heart sounds: bradycardia. Murmurs may be present if there is underlying valvular disease
Laboratory test	Cardiac biomarkers (↑troponin, ↑CK-MB) in case of ischemic etiology (MI). Electrolyte levels (↑potassium,↑magnesium). Thyroid function tests (↑TSH in case of hyporthyroidism)
Instrumental test	ECG: complete dissociation between P waves and QRS complexes. P waves at normal rate (60–100/min). More P waves than QRS complexes. Regular QRS complexes at slower rate (typically 20–40/min). QRS morphology: narrow if escape rhythm is junctional, wide if escape rhythm is ventricular. Constant PR interval absent

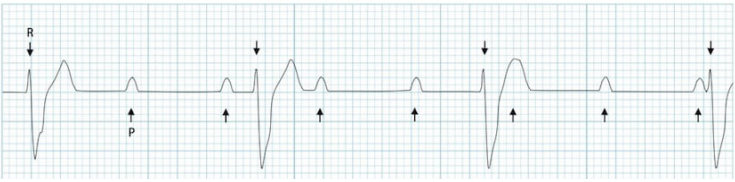


Fig. 6. Third-degree (complete) atrioventricular block

Emergency management algorithm. Positioning: place the patient in a comfortable position, preferably supine, to optimize venous return. Ready for CPR if needed. Continuous cardiac monitoring. IV access establishment. Oxygen therapy if needed.

Pharmacological interventions:

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

2. *Dopamine* 2–20 mcg/kg/min IV infusion to support blood pressure and cardiac output or Epinephrine 1 mg IV every 3–5 minutes if there is associated cardiac arrest or severe bradycardia.

3. Definitive treatment:

– temporary transvenous pacing if medical therapy ineffective (artificial stimulation of cardiac contraction) to stabilize the heart rate;

– permanent pacemaker implantation for most cases.

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 the cessation of effective

blood circulation and, if not promptly treated, can lead to sudden cardiac arrest and death (Table 19).

Caused: damage to the heart muscle due to ischemia, heart failure, congenital heart disease. Other causes may include electrolyte imbalances, hypoxia, and trauma to the heart.

Patient Complaints (Symptoms): sudden loss of consciousness, absence of pulse. Some may experience chest pain or palpitations before the onset.

Table 19

Objective examination in case of ventricular fibrillation

Method	Result
Inspection	Typically, the patient is found in a supine position due to loss of consciousness. Skin color: cyanosis. Cold, clammy skin
Palpation	Pulse is no palpable due to the ineffective contraction of the ventricles. Apex impulse is absent
Percussion	Percussion may reveal a dull sound if there is associated fluid accumulation, but this is less relevant in VF
Auscultation	Heart sounds are absent
Laboratory test	Blood tests may reveal metabolic derangements, electrolyte imbalances (such as hyperkalemia or hypomagnesemia), and cardiac biomarkers may be elevated if there is MI
Instrumental test	ECG (Fig. 7): – chaotic irregular waves of varying amplitude; – no identifiable P waves, QRS complexes, or T waves; – rate 150 to 500 per minute; – ventricular fibrillation is the main cause of sudden death

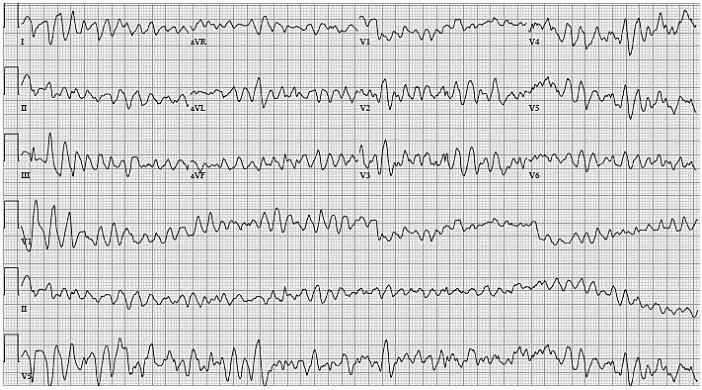


Fig. 7. Ventricular fibrillation

Emergency management algorithm:

1. Positioning: ensure the patient is in a supine position on a firm surface to facilitate cardiopulmonary resuscitation (CPR).

2. Initiate high-quality **CPR immediately,** with a compression-to-ventilation ratio of 30 : 2.

3. Use an automated external defibrillator as soon as available to assess the rhythm and **deliver a shock if indicated**.

4. *Epinephrine* 0,1 %: 1 mg IV every 3–5 minutes during resuscitation.

5. *Amiodarone* 5 %: 300 mg IV as the first dose, followed by a second dose of 150 mg if VF persists.

6. *Lidocaine* 2 %: An alternative to amiodarone, 1–1.5 mg/kg IV with additional doses of 0.5–0.75 mg/kg every 5–10 minutes (maximum total dose of 3 mg/kg).

Asystole

Asystole is defined as the absence of electrical activity in the heart, resulting in a complete lack of cardiac output (Table 20). It is characterized by a flat line on the ECG and is considered a critical condition requiring immediate medical intervention.

Caused: myocardial ischemia, severe electrolyte imbalances, hypoxia, acidosis, drug overdose (certain medications, particularly those affecting cardiac conduction (e.g., beta-blockers, calcium channel blockers, and opioids), severe hypothermia, cardiac tamponade, pulmonary embolism.

Symptoms: sudden collapse with loss of consciousness, loss of movement, unresponsive, no spontaneous respirations, no palpable pulse.

Table 20

Objective examination in case of asystole

Method	Result
Inspection	Typically, the patient is found in a supine position due to loss of consciousness. Skin color: cyanosis. Cold, clammy skin
Palpation	Pulse is not palpable. Apical impulse is absent
Percussion	Percussion may reveal a dull sound if there is associated fluid accumulation, but this is not relevant for asystole
Auscultation	Heart sounds are absent
Laboratory test	Blood tests may reveal metabolic derangements, electrolyte imbalances (such as hyperkalemia or hypomagnesemia), and cardiac biomarkers may be elevated if there is MI

End of the Table 20

Method	Result
Instrumental test	ECG (Fig. 8): – flat line (asystole) with no discernible electrical activity; – no P waves, QRS complexes, or T waves present. Chest X-ray may be performed to evaluate for cardiac tamponade or pulmonary embolism

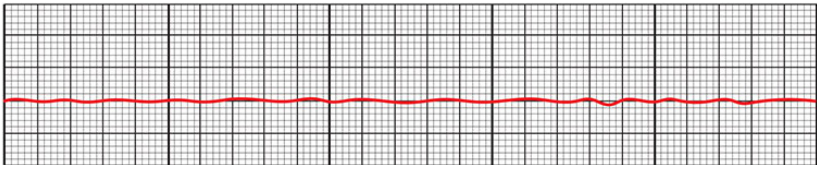


Fig. 8. Asystole

Emergency management algorithm:

1. Ensure the patient is in a supine position on a firm surface to facilitate CPR. Initiate high-quality **CPR immediately**, with a compression-to-ventilation ratio of 30 : 2. Use an automated external defibrillator as soon as available to assess the rhythm and deliver a shock if indicated.

2. *Epinephrine* 0,1 % —1 mg IV every 3–5 minutes during resuscitation.

3. *Atropine* 0,1 % — 1 mg IV

4. Other medicines: *sodium bicarbonate* 1 mEq/kg IV if metabolic acidosis is suspected. *Calcium chloride* or *calcium gluconate* if hyperkalemia is suspected (1 g IV).

CARDIOPULMONARY RESUSCITATION ALGORITHM

Cardiopulmonary resuscitation (CPR) is a technique that any doctor has to know. A person can be revived after cardiac arrest, particularly if treatment is started immediately. CPR has an aim to support circulation at minimal level till the heart function restores. The more time that passes without blood being pumped to the brain, the less likely it is that the person can be revived, and, if revived, the more likely it is that the person will have permanent brain damage. That's' why we need to start CPR immediately after cardiac arrest. Unresponsive adult protocol of CPR you can in Table 21.

The ABC steps: airway, breathing, and circulation, are the foundational steps in performing CPR.

1. Airway: open the victim's airway using a head-tilt chin-lift or jaw thrust maneuver.

2. Breathing: provide rescue breathing.

3. Circulation: perform chest compression to restore the blood circulation.

First step (A or C) should be used according local guidelines.

Table 21

CPR unresponsive adult protocol

Step	Practical measures
Ensure the scene is safe	Direct someone to obtain an Automated external defibrillator
Recognize cardiac arrest	Check for responsiveness: tap the person and shout. Call for help if unresponsive
Activate emergency response	Call emergency services. If available, send someone to get an Automated external defibrillator. If multiple rescuers are available, assign roles (compressions, breaths, others)
Check carotid pulse and breathing simultaneously (do not take >10 seconds)	If pulse present, breathing normal: monitor until emergency services arrive. If pulse present, breathing abnormal: manage airway, begin rescue breathing (1 breath every 5–6 seconds). If no pulse, breathing abnormal: Start chest compressions (CPR ratio: 30 : 2)
	Provide rescue breaths After 30 compressions, give 2 rescue breaths. Open the airway: use the head-tilt/chin-lift maneuver. Pinch the patient's nose closed. Inhale, then place your mouth over the patient's mouth. Breathe into the patient's mouth for 1 second, watching for chest rise. Remove your mouth to allow air to escape, then give a second breath. Continue with cycles of 30 compressions and 2 breaths
Start chest compressions	**One Rescuer:** CPR ratio: 30 compressions to 2 breaths. **Two Rescuers:** 1st rescuer: 30 compressions. 2nd rescuer: 2 breaths. Change positions every 2 minutes
	The technique of chest compressions Position the heel of one hand on the center of the chest (lower half of the sternum). Place the other hand on top and interlock fingers. Compress at a rate of 100-120 compressions per minute. Focus on minimizing interruptions in chest compressions. Compress to a depth of at least 5 cm for adults. Allow full chest recoil between compressions
Defibrillator arrives	Stop chest compressions. Turn on Defibrillator and attach pads to the bare chest. Ensure no one is touching the patient during the analysis. If a shock is advised, ensure everyone is clear and deliver the shock. Resume CPR immediately after the shock for 2 minutes (5 cycles of 30 : 2)

Signs of restored ventilation/circulation are as follows: improving skin color, swallowing attempts, struggling movements, return of or strong pulse, return of systemic blood pressure. You have to continue CPR till the time when pulse and respiration returns or Emergency medical help arrives or signs of biological death appear despite CPR. **In case of ineffectiveness CPR is done at least for 30 min!**

Post-resuscitation care. Once the patient is stabilized, monitor vital signs and provide appropriate post-resuscitation care. Transport to a medical facility for further evaluation and treatment.

GASTROINTESTINAL BLEEDING

Gastrointestinal bleeding (GIB) refers to any hemorrhage occurring within the gastrointestinal tract, from the esophagus to the anus. GIB can be classified as Upper gastrointestinal bleeding (UGIB) — from oral cavity to the end of duodenum, Lower Gastrointestinal Bleeding (LGIB) — from jejunum to rectum, and Obscure Gastrointestinal Bleeding with no identified source (Table 22). Objective examination in case of gastrointestinal bleeding see in Table 23.

Table 22

Classification and symptoms of GIB

Sign	UGIB	LGIB	Obscure
Definition	Bleeding originating proximal to the ligament of Treitz, including the esophagus, stomach, and duodenum	Bleeding originating distal to the ligament of Treitz, including the jejunum, ileum, colon, rectum, and anus	Persistent or recurrent gastrointestinal bleeding with no identified source after initial endoscopy. May require further investigation
Symptoms	Hematemesis: vomiting of blood (bright red or «coffee grounds»)	Hematochezia: passage of bright red blood per rectum	May require capsule endoscopy or angiography for diagnosis
	Melena: black, tarry stools (indicates digestion of blood in the upper GI tract)	Changes in bowel habits (diarrhea, constipation)	
	Epigastric pain or discomfort	Abdominal pain or cramping	
	Heartburn or acid reflux	Tenesmus (feeling of incomplete emptying of the bowel)	
	General symptoms (both UGIB and LGIB): syncope (fainting), dyspnea, weight loss		

Table 23

Objective examination in case of gastrointestinal bleeding

Method	Result
Vital signs	Tachycardia, hypotension, orthostatic hypotension (drop in blood pressure upon standing) is an early sign of volume depletion. Tachypnea. Fever (may suggest infection as an underlying cause, but is less common)
Inspection	The patient typically appears anxious, distressed, and diaphoretic (sweating profusely). Patient position: may be restless or lying still, depending on the severity of bleeding. Pallor (paleness) skin and mucous membranes. Assess for abdominal distension, visible peristalsis, and surgical scars. Evaluate for signs of chronic liver disease (e.g., jaundice, ascites, spider angiomas, palmar erythema, gynecomastia). Assess for signs of coagulopathy (e.g., petechiae, purpura, ecchymoses)
Palpation	Assess for tenderness, guarding, and rebound tenderness (signs of peritonitis). Palpate for hepatomegaly/splenomegaly (liver disease or portal hypertension). Palpate for abdominal masses. Rectal examination (hemorrhoids, fissures, or other anorectal lesions)
Percussion	Assess for tympany (suggests bowel obstruction) or dullness (suggests ascites or organomegaly)
Auscultation	Hyperactive bowel sounds may be present in cases of gastroenteritis; absent bowel sounds may indicate ileus or peritonitis. ↓BP
Laboratory and instrumental investigations	CBC: ↓RBC, ↓Hb, ↓Ht. ↓Coagulation. Fecal occult blood test is positive. ECG: cardiac ischemia or arrhythmias. **Upper endoscopy:** – allows direct visualization of the esophagus, stomach, and duodenum to identify the source of bleeding (e.g., ulcers, varices, erosions, tumors); – allows for therapeutic interventions (e.g., injection sclerotherapy, band ligation, thermal coagulation, clipping). **Colonoscopy, sigmoidoscopy:** – allows direct visualization of the colon and rectum to identify the source of bleeding (e.g., diverticulosis, angiodysplasia, polyps, tumors, inflammatory bowel disease); – allows for therapeutic interventions (e.g., polypectomy, thermal coagulation, clipping). **Capsule endoscopy:** – a wireless capsule containing a camera is swallowed by the patient and transmits images of the small bowel to a recorder; – useful for evaluating obscure GIB when upper and lower endoscopy are negative; – contraindicated in patients with known or suspected bowel obstruction or strictures

Emergency management algorithm. The immediate goals of emergency management are to stabilize the patient, assess the severity of bleeding, and identify the source of bleeding.

Initial actions:

1. Assess and secure the airway. Suction the airway to prevent aspiration.

2. Monitoring of vital signs, oxygen saturation, and level of consciousness is essential. Monitor urine output to assess renal perfusion.

3. Dietary instructions:

– 24 hours: no food or drink;

– next 24 hours: clear liquids only;

– 48 hours: light diet (if no recurrent bleeding).

Pharmacological interventions:

1. IV fluids (normal saline, lactated Ringer's solution) to restore intravascular volume.

2. Transfuse blood products (packed red blood cells, fresh frozen plasma, platelets) as needed to correct anemia and coagulopathy.

3. Proton pump inhibitors for suspected UGIB: *pantoprazole, omeprazole*: 80 mg IV bolus followed by 8 mg/hr continuous infusion. Reduce gastric acid secretion and promote ulcer healing.

4. Specific treatment: in case of variceal bleeding — *octreotide, vasopressin*, in case of coagulopathy due to warfarin — *vitamin K*, in case of fibrinolysis — *tranexamic acid*.

Endoscopic interventions:

1. Upper endoscopy: injection sclerotherapy, band ligation (placing a rubber band around the bleeding varix), thermal coagulation, clipping.

2. Colonoscopy: polypectomy, thermal coagulation, clipping.

HYPERGLYCEMIC AND HYPOGLYCEMIC COMAS

Hyperglycemic coma, often referred to as diabetic coma, is a life-threatening condition characterized by severely elevated blood glucose levels, typically exceeding 600 mg/dL (33.3 mmol/L), leading to *hyperosmolar hyperglycemic state* or *diabetic ketoacidosis*, in rare cases *hyperlactacidemic coma*. This condition results in significant dehydration, electrolyte imbalances, and altered mental status, necessitating immediate medical intervention.

Hypoglycemic coma is a severe neurological condition resulting from critically low blood glucose levels, leading to altered mental status, loss of consciousness, and potential irreversible brain damage if not promptly treated (Table 24).

Table 24

Clinical manifestations of hyperglycemic and hypoglycemic comas

Signs	Hyperglycemic comas			Hypoglycemic
	Ketoacidotic	Hyperosmolar	Hyperlactacidemic	
Onset	Gradual	Gradual	Rapid	Rapid
Causes	Insulin deficiency, nutritional disorders, stress, infection	+ vomiting, diarrhea, dehydration, diuretics	Infection, biguanides, hepatic and renal failure, infarction	Insulin overdose, alcohol, physical exertion, missed meals
Behavior	Passive	Passive	Passive	Psychomotor agitation
Breathing	Kussmaul	Normal	Kussmaul	Normal
Acetone odor	Present	Absent	Absent	Absent
Skin	Dry, cold, decreased turgor	Dry, decreased turgor	Dry, pale	Moist
Eyeball tone	Decreased	Severely decreased	Normal	Normal
Pupils	Constricted	Normal	Normal	Dilated
Muscle tone	Decreased	Decreased	Normal	Increased
Convulsions	Absent	Absent	Absent/Present	Present
Reflexes	↓/Normal	Normal	↓/Normal	↑
Body temperature	↓	Normal	Normal	Normal
Blood pressure	↓	↓↓↓	↓	Normal
Pulse	Rapid, weak	Rapid, weak	Rapid	Normal
Abdomen	Tense	Normal	Pain, not tense	Normal
Complete blood count	Leukocytosis, ↑ESR	↑ESR	Leukocytosis, ↑ESR	Normal
Urinalysis	Proteinuria, cylindruria	Proteinuria, cylindruria	Normal	Normal
Urine acetone	Present	Absent/+	Absent	Absent
Glycemia	> 13 mmol/L	> 33 mmol/L	↑	< 3 mmol/L
Blood pH	< 7,3	> 7,3	< 7,3	Normal
Blood urea	↑	↑	Normal/↑	Normal
Sodium	↑/↓	↑	Normal	Normal
Potassium	↓	↓	Normal	Normal

Ketoacidotic management protocol:

1. Positioning: place the patient in a supine position to ensure adequate venous return and monitor vital signs.

2. Monitoring: continuous cardiac monitoring and vital signs assessment.

3. Fluid replacement: initiate IV fluid therapy with isotonic saline (0.9 % NaCl) to address dehydration.

4. Insulin: administer IV insulin (e.g., regular insulin) at a rate of 0.15 units/kg/hour, adjusting based on blood glucose levels. Continue until blood glucose levels stabilize (typically < 11.0 mmol/L) and the patient is able to resume oral intake.

5. Electrolyte replacement: administer potassium chloride (KCl) if serum potassium is < 5 mmol/L at a rate of 1–2 g/h in IV fluids, adjusting based on serum potassium levels. Continue until the patient is rehydrated and electrolyte levels are normalized.

6. Bicarbonate: consider sodium bicarbonate if severe acidosis (pH < 7.0) is present, typically administered at 1–2 mEq/kg IV.

Hypoglycemic management protocol:

1. Positioning: place the patient in a lateral recumbent position to prevent aspiration if unconscious.

2. Glucose administration:

– if the patient is conscious and able to swallow, provide oral carbohydrates (e.g., glucose tablets, fruit juice);

– **dextrose:** 50 % — administer 25 g (50 mL) IV push for rapid correction of hypoglycemia;

– **glucagon:** if IV access is not available and the patient is unconscious, administer 1 mg IM or SC.

Monitor blood glucose levels every 15 minutes until stable, and adjust treatment as necessary. Continue monitoring and supportive care until the patient is fully conscious and able to maintain adequate oral intake.

Hyperosmolar management protocol:

1. Positioning: place the patient in a supine position to ensure adequate venous return and monitor vital signs.

2. Fluid administration: administer 1 liter of 0.9 % sodium chloride solution IV during the first hour; 500 ml of 5 % glucose solution IV. If BP is < 80/50 mmHg, infusion can be faster, colloids solutions can be used.

3. Insulin: IV *short-acting insulin* at a rate of 0,05 units/kg/hour, adjusting based on blood glucose levels. Continue until blood glucose levels stabilize (typically < 14.0 mmol/L) and the patient is able to resume oral intake.

4. Electrolyte replacement: *potassium chloride* if serum potassium is < 5 mmol/L at a rate of 1–2 g/h in IV fluids, adjusting based on serum potassium levels.

Hyperlactacidemic management protocol:

1. Positioning: place the patient in a supine position to ensure adequate venous return and monitor vital signs. Continuous cardiac monitoring and vital signs assessment.

2. Reduction of lactate production: administer intravenous short-acting insulin at a dose of 2–5 units/hour with 5 % glucose solution at a rate of 100–125 mL/hour, while monitoring blood glucose levels. Removal of excess lactate: perform hemodialysis using a non-lactate buffer.

3. Restoration of acid-base balance:

– provide oxygen therapy and mechanical ventilation in hyperventilation mode until pCO_2 reaches 25–30 mmHg;

– administer sodium bicarbonate only if pH < 6.9, at a maximum of 100 mL of 4 % solution intravenously and slowly, followed by an increase in lung ventilation to eliminate excess CO_2.

Identify and treat any underlying conditions that have provoked lactic acidosis.

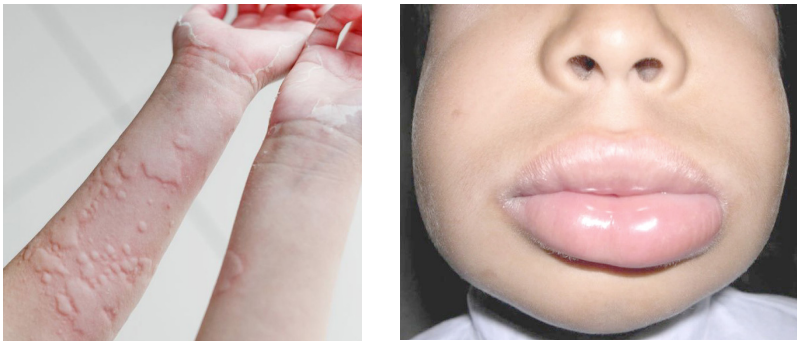
ACUTE URTICARIA, QUINCKE'S EDEMA

Urticaria (hives) and Quincke's edema (angioedema) are types of allergic reaction. Patient's complaints (symptoms) of urticaria and angioedema are the following (Table 25, Fig. 9)

Table 25

Symptoms of urticaria and angioedema

Urticaria	Quincke's edema
Skin rash anywhere on the body, superficial skin-colored or pale skin swelling, surrounded by erythema (redness), very itchy rashes with irritation and burning pain	Deeper swelling within the skin or mucous membranes and can be skin-colored or red, sometimes pain rather than itch. Swollen lips, tongue, or uvula, swelling of the hands, feet or throat. Gastrointestinal tract involvement, such as abdominal pain, nausea, or vomiting, cramps. Hypotonia (collapse), syncope. Chest tightness, stridor (a high-pitched inspiratory noise due to upper airway obstruction), wheezing, shortness of breath, and even death due to suffocation (in case of edema of upper airways)
Skin returning to its normal appearance, usually within 30 min to 24 hours	Skin returning to its normal appearance, usually within several hours to 72 hours



a *b*

Fig. 9. Skin signs of allergy:
a — urticaria; *b* — angioedema

Emergency management algorithm:

1. Avoidance of eliciting factors (discontinue medication that is suspected to provoke the disease).

2. H_1-antihistamines (cetirizine 10 mg once daily or loratadine 10 mg once daily or chloropyramine 25 mg per os every 4–6 hours or intramuscular 20–40 mg).

3. In case of stridor — upright position, oxygen inhalation (high-flow supplemental oxygen via non-rebreather mask to maintain $SpO_2 \geq 90\ \%$), corticosteroids inhalation (budesonide 200 mcg).

4. In case of hypotension (systolic blood pressure less than 90 mmHg) — epinephrine 0,5 ml intramuscular, systemic corticosteroids (prednisolone 60–90 mg intramuscular/intravenous).

5. In case of dyspnea and whizzing (asthma attack) — β-2-agonists inhalation (salbutamol 200 mcg).

ANAPHYLACTIC SHOCK

Anaphylactic shock is a severe allergic reaction (anaphylaxis). The most common triggers are food, drugs and venom. Anaphylaxis is a potentially life-threatening allergic reaction characterized by sudden onset and rapid progression of airway, breathing and circulation (ABC) problems (Table 26).

Emergency management algorithm:

1. Remove trigger if possible (stop infusion of medicine, etc.).
2. Call resuscitation teem or ambulance.
3. Position of the patient lying flat (with or without feet elevated. Sitting position may make breathing easier). Do NOT allow to stand or walk. If unconscious place in recovery position.

4. Epinephrine (adrenaline) 0.1 % — 0,5 ml (0.5 mg) intramuscular, into outer mid-thigh, repeat after 5 minutes.

5. Check the vital signs. Ensure the airway is free, check the pulse. In case of absence of pulse and respiration, start CPR.

6. Provide oxygen therapy (open the window, at hospital — oxygen 6–8 L/min through oxygen mask, titrate to SpO_2 94–98 %).

7. Monitor BP, ECG, pulse oximetry.

8. Establish venous access and start intravenous infusion of fluid (normal saline solution 0,9 % NaCl).

9. H_1-antihistamines (chloropyramine 20–40 mg) intravenous.

10. Corticosteroids (prednisolone 90–120 mg, hydrocortisone 100 mg) intravenous.

11. In case of dyspnea and whizzing — β-2-agonists inhalation (salbutamol 200 mcg).

12. In case of stridor — corticosteroids inhalation (budesonide 200 mcg).

Table 26

Clinical signs of anaphylactic shock

Airway problems	Breathing problems	Circulation problems
Airway swelling (throat and tongue swelling causing difficulty in breathing/ swallowing; patients may feel their throat is closing). Hoarse voice. Stridor (a high-pitched inspiratory noise caused by upper airway obstruction)	Increased work of breathing. Bronchospasm (wheeze) and/ or persistent cough. Patient becoming tired with the effort of breathing (fatigue). Hypoxemia ($SpO_2 < 94$ %) which may cause confusion and/or central cyanosis. Respiratory arrest	Signs of shock: – pale, clammy skin; – significant tachycardia; – hypotension. Dizziness, decreased conscious level or loss of consciousness. Arrhythmia. Cardiac arrest

SELF-CONTROL QUIZ

1. What is the first-line treatment for acute urticaria:

a) antihistamines;
b) corticosteroids;
c) epinephrine;
d) antibiotics?

2. What is the most critical first step in managing anaphylactic shock:

a) administering antihistamines;
b) providing oxygen;
c) administering epinephrine;
d) starting intravenous fluids?

3. Which medication is commonly used as a rescue inhaler during a bronchial asthma attack:

a) long-acting beta-agonists;
b) short-acting beta-agonists;
c) corticosteroids;
d) leukotriene modifiers?

4. What is the most appropriate initial diagnostic test for a patient presenting with hemoptysis:

a) chest X-ray;
b) CT scan of the abdomen;
c) bronchoscopy;
d) complete blood count?

5. What is the target blood pressure reduction in a hypertensive crisis:

a) 10–15 % in the first hour;
b) 25 % in the first hour;
c) 50 % in the first hour;
d) no reduction is necessary?

6. What medicines can be used for stable angina pectoris treatment:

a) beta-blockers;
b) calcium channel blockers;
c) morphine;
d) nitrates;
e) aspirin?

7. Which medication is commonly administered immediately upon suspicion of a myocardial infarction:

a) warfarin;
b) aspirin;
c) amiodaron;
d) salbutamol?

8. What is the most important initial step in the management of a patient with gastrointestinal bleeding:

a) administering IV fluids;
b) performing an endoscopy;
c) administering proton pump inhibitors;
d) obtaining a complete blood count?

9. What is the primary treatment for a patient in hyperglycemic coma:

a) insulin therapy;
b) oral hypoglycemics;
c) glucagon;
d) sodium bicarbonate?

10. What is the primary goal in the management of acute circulatory failure:

a) administering diuretics;
b) restoring adequate tissue perfusion;
c) reducing blood pressure;
d) increasing heart rate?

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

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