MINISTRY OF HEALTH OF UKRAINE

ODESA NATIONAL MEDICAL UNIVERSITY

Departments of Pediatrics №2

CONFIRMED by

Vice-rector for research and educational work

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METHODOLOGICAL RECOMMENDATIONS ON PRACTICAL CLASSES FOR STUDENTS

International Medical Faculty, course 6

Educational discipline "**PEDIATRICS**"

Approved
at the meeting of the department of Pediatrics №2
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1. **Topic** № 17
   Differential diagnosis of heart rhythm and conduction disorders in children. Leading clinical symptoms and syndromes in heart rhythm disorders: extrasystole, paroxysmal tachycardia, atrial fibrillation, complete atrioventricular block. Clinical variants of the course of paroxysmal tachycardia and atrial fibrillation in children. Patient management tactics for heart rhythm and conduction disorders in children. Data of instrumental studies in extrasystole, paroxysmal tachycardia, atrial fibrillation, complete atrioventricular blockade. Differential diagnosis of extrasystole, paroxysmal tachycardia, atrial fibrillation and complete atrioventricular block. Emergency care for paroxysmal rhythm disturbances and Morgagni-Adams-Stokes syndrome. Prevention of heart rhythm and conduction disorders in children.
2. **Relevance of the topic. Heart rhythm disorders** (HRD) a**s v**iolation of the heart rhythm and conduction in children is one of the most common pathologies in childhood; it can have a functional and organic nature, and remain asymptomatic for a long time. The danger of these conditions is that they can cause sudden cardiac arrest and sudden cardiac death. There are 5 types of arrhythmias most common in children:

1. Supraventricular tachyarrhythmias.
2. Ventricular tachyarrhythmias
3. Sick sinus syndrome (SSS)
4. Supraventricular extrasystole
5. Ventricular premature beats.

In recent years, there has been a tendency towards an increase in the frequency of arrhythmias among children of different ages; this is facilitated by the instability of the child's heart rate, due to the anatomical and physiological characteristics of the cardiovascular system, imperfection of regulatory mechanisms, ecosocial ill-being. In children, rhythm disturbances are mainly of a functional nature, and do not require the appointment of antiarrhythmic drugs. At the same time, the sudden appearance of paroxysms and the rate of development of heart failure pose a danger to the child's life, requiring an immediate complex of urgent measures.

3. **Objectives of the lesson:**

3.1. General goals: To get acquainted with the modern definition of the concept of HRD, their etiology, clinical signs, to be able to diagnose in children, to draw up a plan of therapeutic and preventive measures. To get acquainted with the modern definition of HF, etiology, clinical signs, be able to diagnose in children, draw up a plan for therapeutic and preventive measures.

3.2. Educational goals: To get acquainted with the contribution of domestic and foreign scientists to the study of HRD problems, to compare the percentage of morbidity in different regions of Ukraine, to highlight the factors that contribute to the development of diseases, to determine the need for prevention of HRD in children. To get acquainted with the contribution of domestic and foreign scientists to the study of HF problems, to compare the percentage of morbidity in different regions of Ukraine, to highlight the factors that contribute to the development of diseases, to determine the need for prevention of HF in children.

3.3. Specific objectives:

- to know:

1. Etiology and pathogenesis of HRD
a) the reasons for the occurrence of HRD;
b) the pathogenesis of HRD.

2. Clinic of the HRD
a) classification of the HRD;
b) clinical manifestations of tachycardia;
c) clinical manifestations of bradycardia;
d) clinical manifestations of extrasystole.

3. Methods for diagnosing HRD:
a) the role of additional research methods (laboratory: general blood test, general urine analysis, biochemical blood tests; instrumental: EKG, EchoKG, radiography of the OGK).
b) differential diagnosis.

4.Treatment methods:
a) medication;
b) surgical.

5. Prevention of HRD.

6. Etiology and pathogenesis of heart failure (HF)
a) the causes of heart failure;
b) the pathogenesis of HF.
7. CH clinic
a) classification of CH;
b) clinical manifestations of acute and chronic heart failure.

8. Methods for diagnosing HF:
a) the role of additional research methods (laboratory: general blood test, general urine analysis, biochemical blood tests; instrumental: EKG, EchoKG, radiography of the OGK).
b) differential diagnosis.

9.Treatment methods:
a) medication;
b) surgical.

3.4. Based on theoretical knowledge of the topic:
- master the techniques / be able to /:
1. Distinguish the clinical signs of tachy -, bradycardia and extrasystole, HF, sudden cardiac death, cardiac arrest based on the interpretation of the collected complaints, anamnesis of life and illness, data of objective and laboratory research methods.

2. Formulate a clinical diagnosis and prescribe treatment.

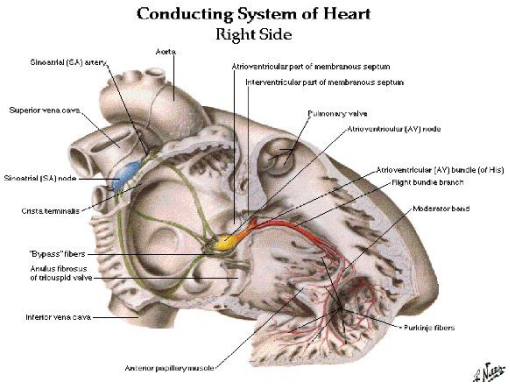
3. Develop preventive measures.

4. Materials for classroom self-study (interdisciplinary integration).

№	Disciplines	To know	To be able to do
1	**2**	**3**	**4**
1.	Previous disciplines 1. Anatomy.	Anatomical structure of the cardiovascular system (CVS).	To mark the features of the anatomical structure in children

	2. Physiology. 3. Biochemistry. 4. Propedeutics of childhood diseases.	Physiology of CVS. Features of the state of the hemostasis system. Basic concepts about HRD. AFO SSS.	To note the features of the physiological regulation of metabolic processes in the myo-, peri- and endocardium. To note the pathology of metabolic processes at different levels. Conduct a clinical examination of a child with HDS.
2.	The following disciplines 1.Hospital pediatrics	Methods for the diagnosis and differential diagnosis of CVD diseases in children.	Carry out a differential diagnosis with other nosoforms.
3.	Intra-subject integration 1. Tachycardia 2.Bradycardia 3.Extrasystole	Features of development, clinical and morphological changes in HRD.	Conduct a clinical examination of the patient, evaluate the data of the clinical examination, radiological and echocardiographic signs, laboratory changes for the diagnosis of the disease.

4. Content of the lesson.



A healthy heart beats in a regular, coordinated manner due to the fact that electrical impulses in the heart are generated and propagated by myocytes with unique electrical properties that trigger a consistent and orderly myocardial contraction. Diseases associated with disturbances in rhythm and conduction are caused by improper formation and / or conduction of these impulses.

Arrhythmia:

• change in heart rate;

• change of rhythmic regularity and source of excitation of the heart muscle;

• incorrect sequence or disrupted connection between the activation of both the cardiac ventricles and the atria.

The most common rhythm disturbances in children are:

1. Supraventricular tachyarrhythmias.
2. Ventricular tachyarrhythmias
3. Sick sinus syndrome (SSS)
4. Supraventricular extrasystole
5. Ventricular premature beats

Life-threatening arrhythmias in children:

1. Long QT syndrome (LQT)
2. SSS (III and IV options)
3. Paroxysmal tachycardia
4. Ventricular extrasystole of high gradations
5. Blockades of high grades

Classification of arrhythmias (N.A. Belokon)		
Impulse formation disorders	**Conduction disturbances**	**Combined arrhythmias**
A. Nomotopic rhythm disturbances 1) sinus arrhythmia 2) sinus bradycardia 3) sinus tachycardia 4) pacemaker migration *B. Heterotopic (ectopic) disorders* 1) Extrasystoles: - supraventricular - ventricular; 2) Paroxysmal tachycardia: - supraventricular, - ventricular; 3) Non-paroxysmal tachycardia: - atrial, - from the atrioventricular connection, - ventricular; 4) Atrial flutter and atrial fibrillation (atrial fibrillation); 5) Flutter and fibrillation of the ventricles.	sinoatrial blockade intra atrial block atrioventricular block intraventricular block	sick sinus syndrome atrioventricular dissociation premature ventricular excitation syndrome

Clinical classification of cardiac arrhythmias		
TACHYCARDIA	**BRADICARDIA**	**ARRHYTHMIA**
Supraventricular: sinus, atrial, nodular, atrial fibrillation, atrial flutter **Ventricular:** tachycardia, atrial fibrillation, ventricular flutter	**Supraventricular:** sinus, sinus node failure, SA block, junctional rhythm **Ventricular:** AV block	Extrasystoles Absolute arrhythmia with atrial fibrillation or atrial flutter with changing atrioventricular block Partial AV block with loss of individual ventricular contractions or a change in the degree of block CA blockade with loss of individual ventricular contractions or a change in the degree of blockade Atrioventricular dissociation with interference Parasystole

Classification of arrhythmias according to their clinical significance
(Kozyreva O.A., Bogacheva R.S., 1998)

Insignificant arrhythmias are unstable, have no clinical significance, do not affect the patient's well-being and prognosis	Significant arrhythmias are persistent arrhythmias affecting the patient's condition and having prognostic value
• supraventricular extrasystole • rare ventricular premature beats - up to 10 per hour • migration of the pacemaker during night sleep • sinus bradycardia and tachycardia, without clinical manifestations	• frequent extrasystole - more than 10 per minute. or 100 per hour • polytopic extrasystole, paroxysmal heart rhythm disturbances • SSS • LQTS • WPW syndrome

ETIOLOGY

Among the causes of HRD are:

1. *Cardiac* (Wolff-Parkinson-White phenomenon, myocarditis, ischemic disorders in the myocardium, congenital heart disease, CMP)
2. *Extracardiac* (intoxication of various origins - medicinal, alcohol, caffeine; diphtheria heart disease, sepsis, damage to the central nervous system or VNS, hypokalemia or hypomagnesemia, hormonal disorders - hypothyroidism, hyperthyroidism; release of catecholamines during stress, exercise, fever, infections, pain)
3. *Mixed*

In the absence of organic defects, rhythm disturbances are considered "idiopathic"

PATHOPHYSIOLOGY

Rhythm disturbances occur as a result of impaired formation and / or impulse conduction. Bradyarrhythmias occur as a result of a decrease in their own pacemaker function or blockade of conduction, mainly in the AV node or the His-Purkinje system. Most tachyarrhythmias are caused by the reentry mechanism; some as a result of an increase in the normal or pathological mechanisms of automatism.

Arrhythmia development mechanisms		
Trigger (launch, induced) activity	*Abnormal automatism*	*Reentry mechanism*
activation of latent pacemakers due to an increase in their automatism or inhibition of the sinus node function: • *ventricular tachycardia caused by physical activity in patients without organic heart disease* • *sinus tachycardia in patients with left ventricular hypertrophy* • *trigger rhythms associated with digitalis intoxication*	The ectopic rhythm is formed in cells that, under normal conditions, do not have the function of automatism, i.e. cannot be a source of rhythm, or when cells capable of automatic activity become centers of increased activity: • parasystole • focal atrial tachycardia • some forms of ventricular tachycardia	circular propagation of an impulse around 2 interconnected pathways with different conduction characteristics and refractory periods. The impulse moves in a closed circle, returning to the place of its origin and repeats the movement. There may be several such circles in the atria, and the smallest of them turns out to be the leading one: • *atrial and ventricular fibrillation and flutter* • *tachycardia in WPW syndrome*

An example of the mechanism of AV nodal tachycardia. The two paths connect the same points. Pathway A has a slow conduction and a short refractory period. Pathway B conducts normally, has a longer refractory period.

L. Brent Mitchell, MD, Libin Cardiovascular Institute of Alberta, University of Calgary

I. Normally, the impulse propagates through the A and B paths (1). Conducting through path A is slower and the impulse arrives at the already depolarized path B and thus refractory (2). The result is a normal sinus rhythm.

II. The premature impulse fiHRD pathway B refractory and blocked, but can be carried through pathway A because its refractory period is shorter. Reaching the 2nd, the impulse continues to go forward and back to path B, where it is blocked by tissue that is difficult to treat, until the 3rd. The result is a premature supraventricular rhythm with an increase in the PR interval.

III. If the conduction through path A is slow enough, then the premature impulse can propagate retrograde through path B, the refractory period of which has already ended. If the refractory period of path A has also ended, the impulse can re-enter path A and pass in a circle, sending an impulse of each cycle to the ventricle (4) and retrograde to the atria (5), creating a stable reentry tachycardia.

3 conditions contribute to the reentry phenomenon:
1. Reduction of the period of tissue refractoriness (stimulation of the sympathetic system).
2. Elongation of the conduction pathway (hypertrophy or abnormal pathways).
3. Slowdown during impulse conduction (ischemia).

Features of heart rhythm disturbances in children
• Arrhythmias of a functional nature prevail, often caused by autonomic dysfunctions with a predominance of vago- or sympathicotonia.
• Syndromes of cardiac arrhythmias are often associated with congenital heart defects.
• Arrhythmias associated with impaired impulse formation (nomotopic and heterotopic) predominate.
• Characterized by the sudden appearance of paroxysms.
• Rapid development of heart failure is observed.

CLINIC
Symptoms depend on heart rate, underlying heart disease, duration of arrhythmia and individual patient sensitivity to arrhythmia.
• heartbeat, a feeling of interruption in the work of the heart
•fatigue
• dizziness, headache, behavioral disturbances, sleep disorders
• feeling of discomfort in the chest
• shortness of breath, feeling short of breath
• feeling of heat in the chest and throat
• presyncopal states or fainting
• lag in physical development
• polyuria (release of atrial natriuretic peptide into the bloodstream during prolonged supraventricular tachycardia.).

Most often, the character is paroxysmal (sudden onset and sudden cessation), less often - continuous (it is long, alternates with sinus rhythm, takes > 50% of the day).

Physical examination: palpation of the pulse and auscultation of the heart can determine the frequency of ventricular contractions, regularity or randomness. Palpation of pulse waves in the cervical veins can help diagnose AV blocks and tachyarrhythmias. For example, in complete AV block, the atria contract intermittently when the AV valves are closed, causing large (fast) pulse waves to appear in the jugular veins. There are few other physical examples of arrhythmias.

DIAGNOSTICS

TEST PLAN FOR CHILDREN WITH RHYTHM DISORDERS

1. Evaluation of clinical, anamnestic and genealogical data. NB! *For cases of syncope and sudden death in the family.*
2. ECG examination, including ECG of parents and siblings. It is shown conducting an ECG survey in three positions: wedge (lying), ortho (standing) and after a small physical one. NB! *If you suspect a rhythm disturbance, you must also record a long ECG tape.*
3. Holter monitoring (HM). Today the method is the leading one in the examination of children with LDCs.

Stress tests (bicycle ergometry - VEM, treadmill): determination of exercise tolerance, prognosis of LDC, identification of latent rhythm and conduction disturbances:

- with differentiation of extrasystoles (if extrasystoles are caused by an increase in the tone of the vagus, then under load they disappear or their number decreases);
- to clarify the origin of conduction disturbances (if the latter are caused by an increase in the tone of the vagus, then under load they disappear, if they are associated with organic damage to the myocardium, they will remain without noticeable changes);
- to assess the increase in heart rate in bradycardia;
- to assess the functional activity of the sinus node with its dysfunctions;
- when interpreting changes in the phase of myocardial repolarization, identifying disorders of coronary circulation (with organic lesion, during exercise, a sharp flattening and inversion of T waves appears on the ECG, displacement of the ST segment).
- NB! Life-threatening arrhythmia can be triggered by exercise. Stress tests are allowed only if it is possible to carry out resuscitation measures, with the obligatory presence of a defibrillator!

1. Medicinal electrocardiographic tests (atropine, potassium-obsidane tests)
2. Neurophysiological studies (EEG)
3. Assessment of central hemodynamics, state of cerebral and peripheral circulation
4. EchoCG
5. Virological examination.

TREATMENT

The need for treatment depeHRD on the symptoms and the risk of arrhythmia. Asymptomatic arrhythmias without serious risks do not require treatment, even if they impair quality of life. Symptomatic arrhythmias may require treatment to improve quality of life. Potentially life-threatening arrhythmias require treatment.

Treatment depeHRD on the type of HSP.

1. Treatment of the cause of the disease
2. Antiarrhythmic drugs
3. Cardiac pacing, implantation of a cardioverter-defibrillator, catheter ablation
4. Surgical intervention is indicated for arrhythmias refractory to ablation, or in the presence of indications for combined surgical treatment, especially in patients with atrial fibrillation who need

replacement or plastic heart valves or in patients with VT who require revascularization or resection of an LV aneurysm.

Principles of therapy for cardiac arrhythmias
1. If possible, eliminate the cause of the arrhythmia.
2. Assess the state of hemodynamics.
3. To identify the main and concomitant diseases.
4. In the case of organic heart disease, treatment of the underlying disease is mandatory.
5. In case of autonomic dysfunctions, treatment should be carried out taking into account their type.
6. Remediation of foci of chronic infection.
7. Follow a regimen and a diet rich in vitamins, potassium and other minerals.

Indications for the appointment of antiarrhythmic drugs:
1. Availability of relevant complaints.
2. Persistent sinus tachycardia.
3. Ventricular extrasystoles.
4. Attack of paroxysmal tachycardia.
5. Atrial fibrillation.
6. Violation of hemodynamics.
7. To maintain hemodynamics (in infants, the frequency of ventricular contractions should be at least 60 per 1 min, for older children - at least 45-50 per 1 min).

Antiarrhythmic drugs (AAP) (classification by Vaughan Williams).

Class I: blockers of sodium channels (membrane stabilizing drugs) block fast sodium channels, slowing down conduction in the corresponding parts of the myocardium (working cardiomyocytes of the atria and ventricles, His - Purkinje system).

Class II: β-blockers, which predominantly act on tissues with slow conduction (sinoatrial [SA] and atrioventricular [AV] node), where they decrease the frequency of automatism, slow down conduction and increase refractoriness.

Class III: potassium channel blockers, which increase the duration of action potential and refractoriness in slow and fast channels.

Class IV: Calcium channel blockers, which inhibit the calcium-dependent action potential in the slow channels, hence decrease automatism, slow down the rate of electrical conduction and increase refractoriness.

Digoxin and adenosine are not included in this classification.

Vaughan Williams Classification			
Medicinal product	*Dosage*	*Selective side effects*	*Notes*
Class Ia *Application: suppression of RPE, PVC, NVT, VT, AF or TP, VF*			
Disopyramide	Intravenous: Initially 1.5 mg / kg for> 5 min, then	Anticholinergic effects (urinary retention, glaucoma, dry mouth,	The drug should be used with caution in patients with impaired LV function.

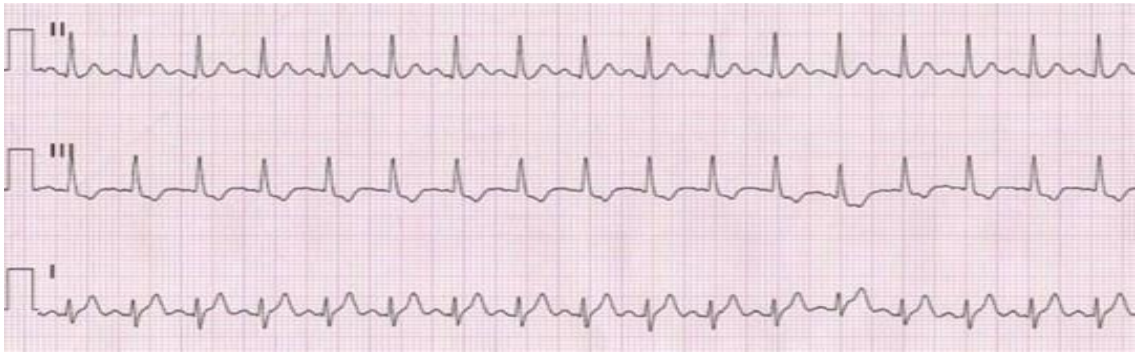
	infusion of 0.4 mg / kg / hour Inside: <2 years: 20-30 mg / kg / s every 6-12 hours; 2-10 years: 9-24 mg / kg / s every 6-12 hours; 11 years and <- 5-13 mg / kg / s every 6-12 hours	blurred vision, intestinal disorders), hypoglycemia, bidirectional VT, negative inotropic effects (which may aggravate heart failure or lead to hypotension)	The dosage should be reduced in patients with renal impairment. Side effects can occur if the treatment regimen is not correct. When the QRS complex is widened (> 50% if the initial interval is <120 m / sec, or> 25% if the initial interval is> 120 m / sec), or if the corrected QT interval is prolonged> 550 m / sec, the infusion rate or dose the administration of the drug should be reduced or suspended.
Procainamide *	Intravenous, 3-6 mg / kg, jet (5 min), repeated dose up to 15 mg / kg, followed by continuous intravenous infusion at a rate of 1-4 mg / min Inside: 15-50 mg / kg / s every 4 hours	Hypotension (with IV infusion), serological abnormalities (especially ANA) occur in almost 100% of cases when the drug is taken> 12 months, drug-induced lupus (arthralgia, fever, pervral effusion) occurs in 15-20%, agranulocytosis in <1% cases, polymorphic VT	There is no need for frequent dosing, as the active ingredient is slowly excreted. When the QRS complex is widened (> 50% if the initial interval is <120 m / sec, or> 25% if the initial interval is> 120 m / sec) or if the corrected QT interval is prolonged> 550 m / sec, the infusion rate or dose should the introduction of the drug should be reduced or suspended.
Quinidine *	By mouth: 20-60 mg / kg / s every 6 (sulfate) / 8 (gluconate) hours	Diarrhea, colic, flatulence, fever, abnormal liver function, ventricular tachycardia "torsades de pointes", the overall incidence of side effects is 30%	If the QRS interval is lengthened (> 50% if the initial interval is <120 m / s, or> 25% if the initial interval is> 120 m / s) or if the corrected QT interval is prolonged> 550 m / s, the infusion rate or dose should the introduction of the drug should be reduced or suspended.
Class Ib *Use: suppression of ventricular arrhythmias (ventricular ES, VT, VF)*			
Lidocaine	Intravenous: 1 mg / kg for 5 minutes, 2 times, then there is continuous infusion	Tremors, convulsions; if administered too quickly there are drowsiness, delirium, paresthesia; possible increase in the risk of developing	The dose or rate of administration should be reduced to 2 mg / min to reduce the risk of intoxication, after 24 hours.

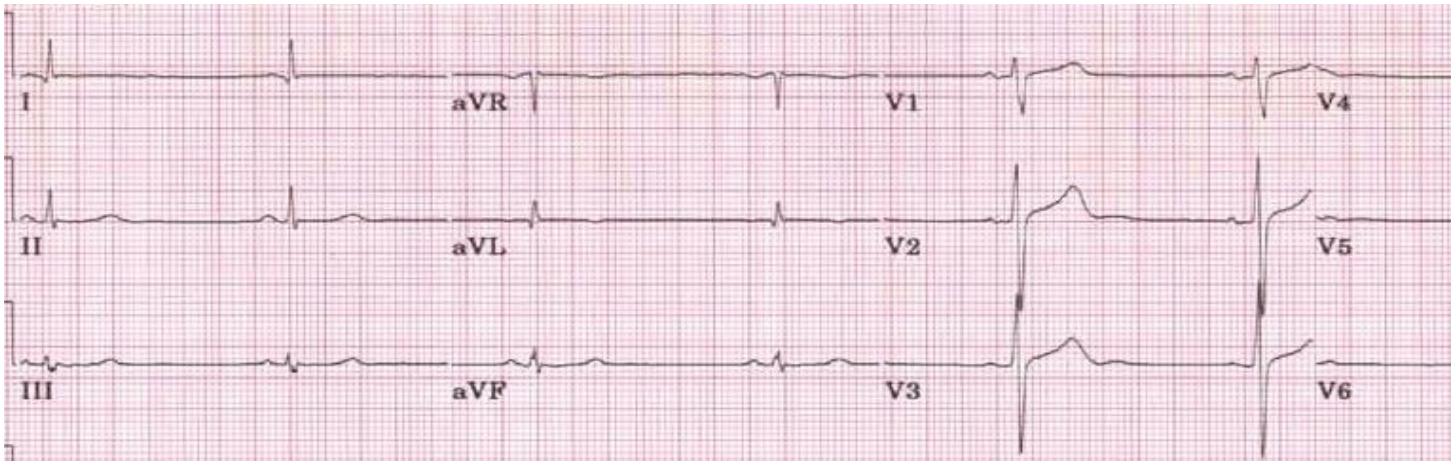
		bradyarrhythmias after acute myocardial infarction	Pronounced metabolism in the liver during the initial passage.
Class Ic *Application: suppression of RPE, PVC, NVT, VT, AF or TP, VF*			
Flecainide	Inside: 3-10 mg / kg / s every 8 hours Intravenous: 1-2 mg / kg over 10 minutes	Occasionally visual impairment and paresthesia	If the QRS interval is prolonged (> 50% if the initial interval is <120 m / s, or> 25% if the initial interval is> 120 m / s), the dose should be reduced or the drug should be suspended.
Propafenone	Inside: 150 - 300 mg / m2 / s every 8 hours; Intravenous: 2 mg / kg, jet; subsequently - introduction at a rate of 2 mg / min	Beta-blocking activity, respiratory distress is possible; rarely gastrointestinal disorders	Pharmacokinetics is nonlinear; the dose increase should not be more than 50% of the previous one. Bioavailability and protein binding are variable; the drug has a cumulative effect.
Class II (beta blockers) *Uses: supraventricular tachyarrhythmias (RPE, sinus tachycardia, IVT, atrial flutter and atrial fibrillation) and ventricular tachyarrhythmias (often for prophylaxis)*			
Carvedilol	Oral: Initially, 6.25 mg 2 times / day, followed by titration to 25 mg 2 times / day	Beta blockers do not have a recommended dosage; titration is carried out until the heart rate decreases by more than 25% Gastrointestinal disorders, insomnia, nightmares, lethargy, erectile dysfunction, abnormalities in AV conduction are possible	Beta blockers are contraindicated in patients with bronchospastic disorders.
Esmolol	Intravenous: 50-200 mcg / kg / min		
Propranolol	Inside: 1-4 mg / kg / s 4 times / day IV: 0.1-0.15 mg / kg mg (the injection can be repeated after 5 minutes if necessary)		
Class III (membrane stabilizing drugs) *Application: any tachyarrhythmias other than fusiform VT*			
Amiodarone	Inside, 10 mg / kg / s mg / day 1-2 r / d for 4-14 days, then dose reduction to 5 mg / kg / s for 3 weeks, followed by a maintenance dose of 2.5 mg / kg / s Intravenous: 2.5-5 mg / kg in 30-60	Fibrosis of the lung tissue (up to 5% of patients treated for> 5 years) can be fatal; lengthening of the QT interval; polymorphic VT (rare); bradycardia; gray and cyanotic skin pigmentation; photosensitivity; liver	The drug has a non-competitive beta-blocking effect, the properties of a calcium and sodium channel blocker with a delayed effect. By lengthening the refractoriness, the drug can lead to a homogeneous state

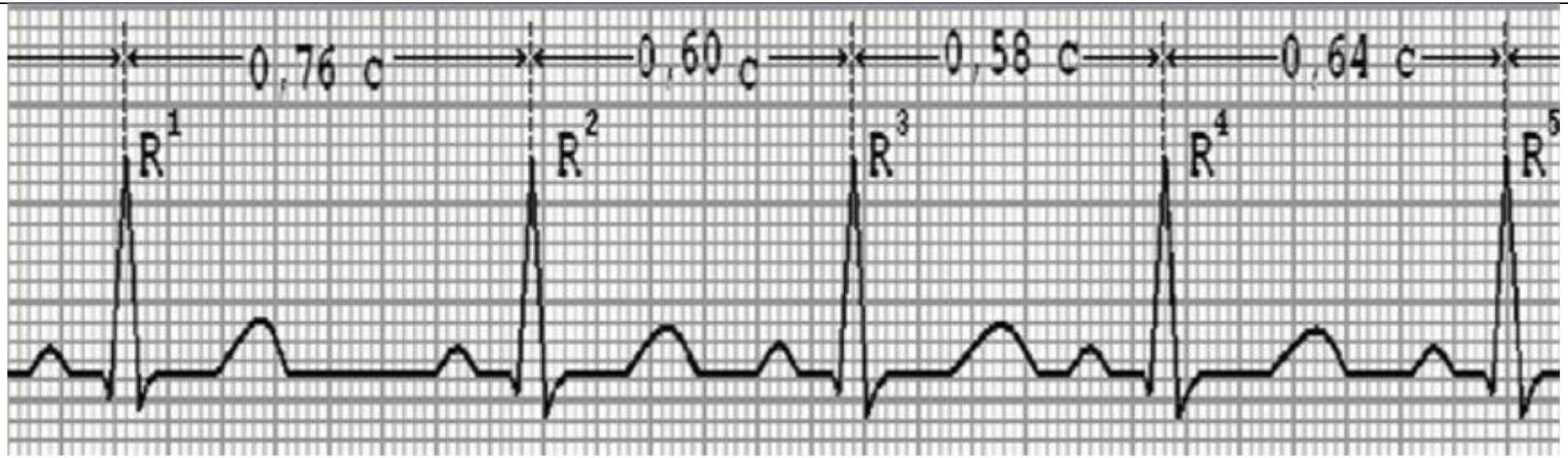
	minutes, can be repeated 3 times with the introduction of a further maintenance dose of 2-10 mg / kg / s	dysfunction; peripheral neuropathy; micro deposits on the cornea (in almost all patients) - usually without serious visual impairment, they disappear after stopping treatment; changes in thyroid function; raising the level of creatinine up to 10% without changing the glomerular filtration rate; slow clearance, possibly prolonging the duration of side effects	of repolarization of the heart. Intravenous forms can be used for rhythm conversion.
Bretilius *	Intravenous: Initially, 5 mg / kg, followed by continuous infusion at a rate of 1-2 mg / min IM: Initially, 5-10 mg / kg, administration can be repeated until a concentration of 30 mg / kg is reached I / m maintenance dose 5 mg / kg every 6-8 hours	Hypotension Possesses the properties of drugs of class II.	Effects can last 10-20 minutes. The drug is used to treat potentially life-threatening persistent ventricular tachyarrhythmias (intractable VT, recurrent VF), in which it usually acts within 30 minutes after administration.
Ibutilid	Intravenous: for patients weighing more than or equal to 60 kg, 1 mg infusion or, for patients weighing less than 60 kg, 0.01 mg / kg in 10 minutes, with re-administration after 10 minutes if the first infusion is unsuccessful	Fusiform VT (in 2%)	The drug is used to arrest atrial fibrillation (approximately 40% successful) and atrial flutter (effective in almost 65% of patients).
Sotalol .	Inside: 80-160 mg every 12 hours Intravenous: 10 mg every 1-2 minutes	Similar to class II; possible impairment of left ventricular function and fusiform VT	The racemic [d-l] form has class II (beta-blocking) properties, the [d] form does not. Both forms are similar in activity to class III. Only racemic Sotalol is available for clinical use.

			The drug should not be used in patients with renal недостаточностью
Class IV (calcium channel blockers) *Application: relief of supraventricular tachycardia and heart rate control in atrial fibrillation and atrial flutter*			
Diltiazem	Prolonged oral form (Diltiazem CD): 120-360 mg once / day Intravenous: 5-15 mg / h for up to 24 hours	Possible ventricular fibrillation in patients with ventricular tachycardia, negative inotropic effect	The intravenous form is most commonly used to reduce the ventricular rate in AF and AT.
Verapamil (only not with ERW !!!)	Inside, 2-7 mg / kg / s mg 3 times / day; Intravenous, 0.1-0.2 mg / kg, 2 doses 20 min apart Oral prophylaxis, 40-120 mg, 3 times / day	Possible ventricular fibrillation in patients with ventricular tachycardia, negative inotropic effect	The IV form is used to relieve tachycardia with a narrow QRS complex, including the junctional one (efficiency is almost 100% when administered intravenously 5-10 mg in 10 minutes).
Other antiarrhythmic drugs			
Adenosine	50-300 mcg / kg, start at 50 mcg / kg, increase by 50-100 mcg / kg / dose, if there is no effect, intravenous bolus	Transient breathing disorders, chest discomfort, and (in 30-60%) transient bronchospasm	The drug slows down or blocks conduction in the AV junction. The duration of action is extremely short. Contraindications include asthma and high-grade atrioventricular block. Dipyridamole enhances the effect of the drug.
Digoxin (only not with ERW !!!)	IV ¾ oral dose Inside 40 mcg / kg, give ½ dose then ¼ dose after 8-12 hours 2 times maintenance dose: 10 mcg / kg / s every 12 hours	Anorexia, nausea, vomiting and, often, serious arrhythmias (ventricular extrasystoles and tachycardias, atrial extrasystoles and tachycardias, atrioventricular blockade 2 and 3 degrees, combinations of these rhythm disturbances)	Contraindications include the presence of an antegrade-conducting additional conduction pathway (manifesting Wolff-Parkinson-White syndrome), since the occurrence of atrial fibrillation will lead to severe ventricular tachysystole (digoxin shortens the refractory

			period of the additional atrioventricular junction).
AF = atrial fibrillation; ANA = antinuclear antibodies: RPE = atrial premature beats; AV = atrioventricular; CrCl = creatinine clearance; LV = left ventricle; QTk = corrected QT interval; IVT = supraventricular tachycardia; VF = ventricular fibrillation; PVC = premature ventricular contraction; VT = ventricular tachycardia.			

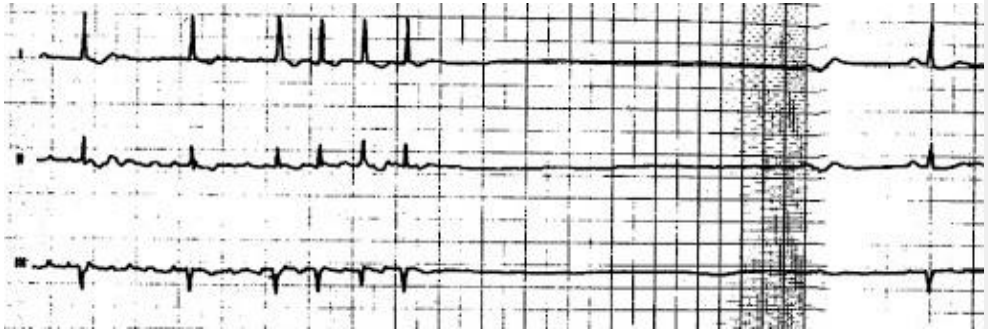
HRD type	***Diagnostic criteria***			***Treatment***
	Etiology, pathogenesis, classification	Clinical	Paraclinical	
Violation of the formation of an impulse in the sinus node (nomotopic)				
Sinus tachycardia (CT) an increase in heart rate in 1 min compared to the age norm, the pacemaker is the sinus node. CT adversely affects cardiac hemodynamics: diastole is shortened, cardiac output decreases, and myocardial oxygen demand increases.	SVD by sympathicotonic type Thyrotoxicosis Arterial hypotension IDA Hypoxia Fever, intoxication of infectious origin Myocarditis, heart failure. Constitutional, hereditary ST. Exercise stress Atropine, adrenaline, caffeine, ACTH, GCS	Complaints are minor: palpitations, a feeling of heaviness in the chest. More often, children do not make complaints Gradual start and end Correct frequent rhythm Auscultatory rapid rhythm with preserved heart melody. Strengthening and splitting the I tone, weakening the II tone Pendulum rhythm, embryocardia	Increased heart rate, maintaining the correct sinus rhythm; positive P wave in leads I, II, aVF, V4-V6; constant interval P-R 0.12-0.2 s. constant P waveform in all leads	Elimination of the cause of ST is treatment of the underlying disease (anemia, arterial hypotension, thyrotoxicosis, etc.). The use of sedatives, electrosleep, β-blockers in small doses. For myocarditis with ST: NSAIDs, potassium preparations, cocarboxylase. In the presence of heart failure, cardiac glycosides.
				
Sinus bradycardia (SB) slowing down of cardiac activity in comparison with the age norm, while the pacemaker is the sinus node	Physiological SB in athletes during sleep SVD by vagotonic type Myocarditis, MKD Food and drug intoxication (cardiac glycosides, antihypertensive drugs, potassium preparations, β-blockers) Pronounced SAT can be a manifestation of the SSSU (1 version of the SSSU)	Usually, children do not present complaints, with severe SB, weakness, dizziness, palpitations, depression, fainting may appear The rhythm is correct, rare	ECG: decrease in heart rate Maintaining the correct sinus rhythm. Positive P wave in leads I, II, aVF, V4-V6	Children with severe vagotonia are prescribed drugs that reduce the activity of the vagus (amizil, bellataminal). In case of poisoning, appropriate antidotes are used: in case of poisoning with cardiac glycosides - unitiol, in case of an overdose of potassium - calcium

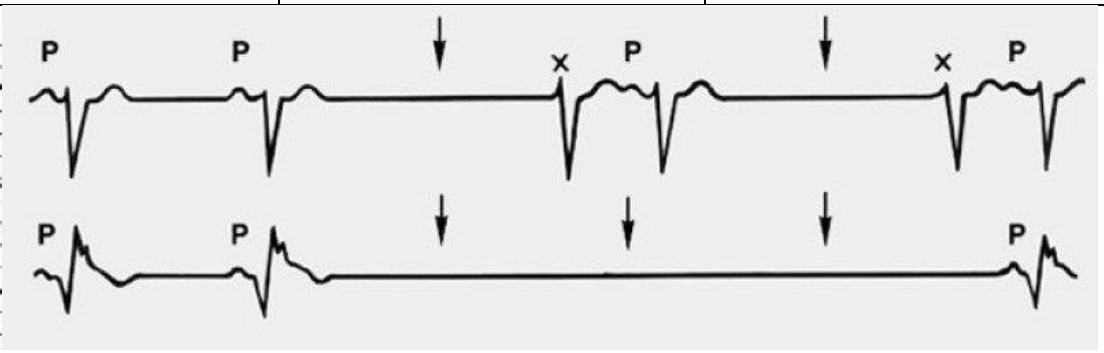
Moderate SB does not cause hemodynamic disturbances.	CNS damage (meningoencephalitis, brain tumors, cerebral hemorrhages)	Gradual start Variability of heart rate with irritation of the vagus and sympathetic nerves Auscultatory, the melody of the heart is preserved, the pauses between the tones are lengthened.	constant interval P-R 0.12-0.2 s. constant P waveform in all leads	preparations. In other cases, treatment of the underlying disease is required.
				
Sinus arrhythmia (SA)	Irregular sinus pacemaker	In an isolated form, SA is asymptomatic more often indicates the activity of parasympathetic influences on the heart rhythm. Different pauses between tones are audible on auscultation.	a complex of ECG and clinical symptoms caused by a decrease in the automatism of the SU and a violation of the sinoatrial (SA) - conduction	There is no specific treatment for sinus arrhythmia. Treatment of the underlying disease is necessary

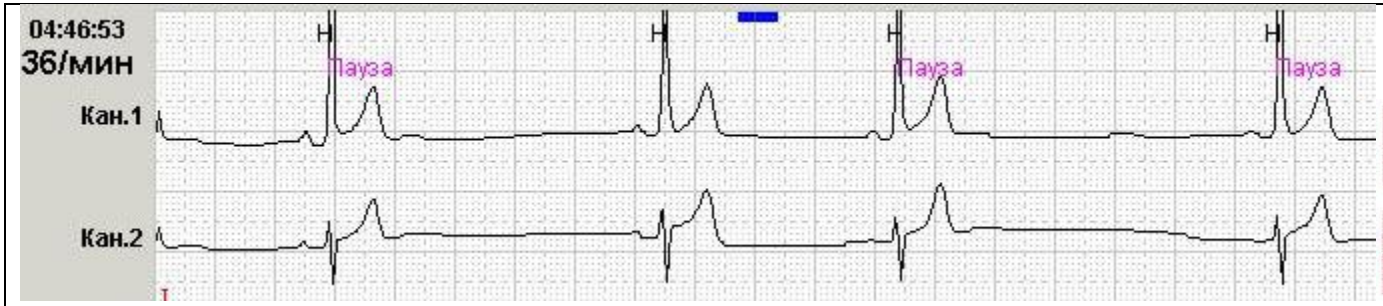


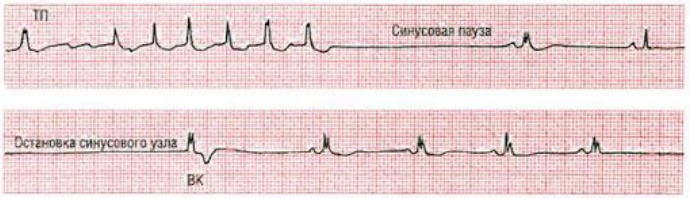
Sick sinus syndrome Sinus node dysfunction includes: 1. Unexplained sinus bradycardia 2. Alternating bradycardia and atrial tachyarrhythmias (bradycardia-tachycardia syndrome) 3.Sinus pause or stop 4.Sinoatrial (SA) exit block	**Primary:** organic lesions of the CA-zone with: a) cardiac pathology - ischemic heart disease, hypertension, CMP, heart defects, myocarditis, surgery and heart transplantation; b) idiopathic fibrosis of the CA-node; c) hypothyroidism, amyloidosis, sarcoidosis, scleroderma heart, etc. **Secondary:** exogenous factors (hyperkalemia, hypercalcemia, β-blockers, sotalol, amiodarone,	Dizziness Fainting Sometimes convulsions (attacks of Morgagni-Adams-Stokes). With persistent bradycardia - symptoms of CHF or coronary insufficiency Poor exercise tolerance Inadequate increase in heart rate (chronotropic insufficiency). **Flow forms:** 1. Compensated: bradystolic, brady / tachysystolic variant 1. Decompensated: bradystolic, brady / tachysystolic variant 2. Permanent form of atrial fibrillation: tachysystolic, bradystolic variant	Slow, irregular rhythm. 1. Persistent pronounced sinus bradycardia. 2. Bradysystolic form of atrial fibrillation. 3. Migration of the atrial pacemaker. 4. Stopping the sinus node. 5. Sinoauricular block.	The main treatment for sinus node dysfunction is the implantation of a pacemaker. Theophylline and hydralazine increase heart rate in healthy young patients with bradycardia without syncope.

	verapamil, diltiazem, procainamide, cardiac glycosides, dopegit, reserpine, etc .; autonomic dysfunction)		6. Pauses> 2.5 s due to sinus node arrest, SA blockade, or rare replacement rhythms. 7. Takhi-brady syndrome, alternation of periods of tachycardia and bradycardia. 8. Rarely - attacks of ventricular tachycardia and / or ventricular fibrillation. 9.Slow restoration of sinus node function after extrasystoles, paroxysms of tachycardia and fibrillation 10. Inadequate slowing down of the rhythm when using even small doses of β-blockers.	

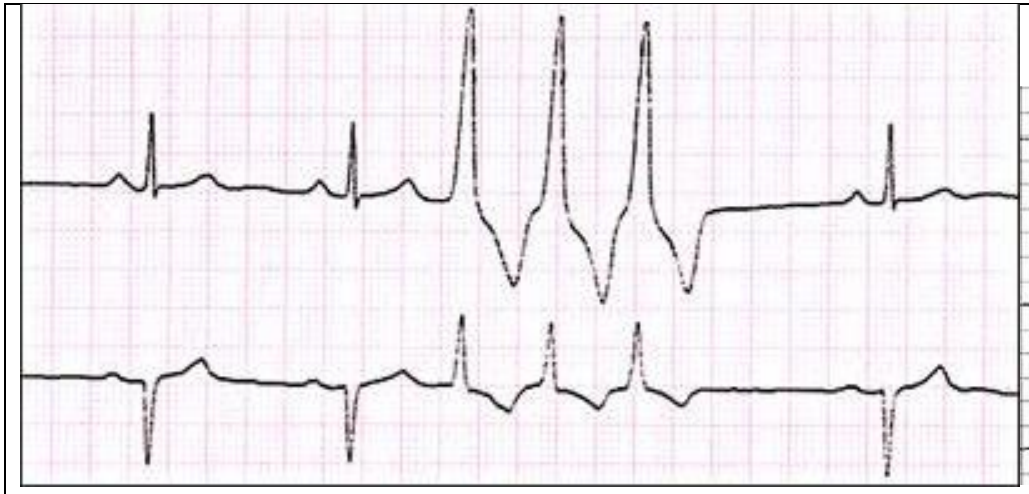


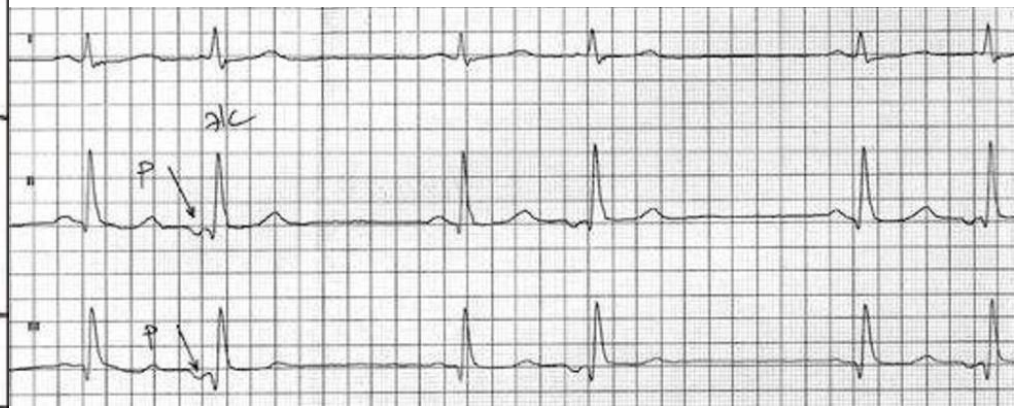




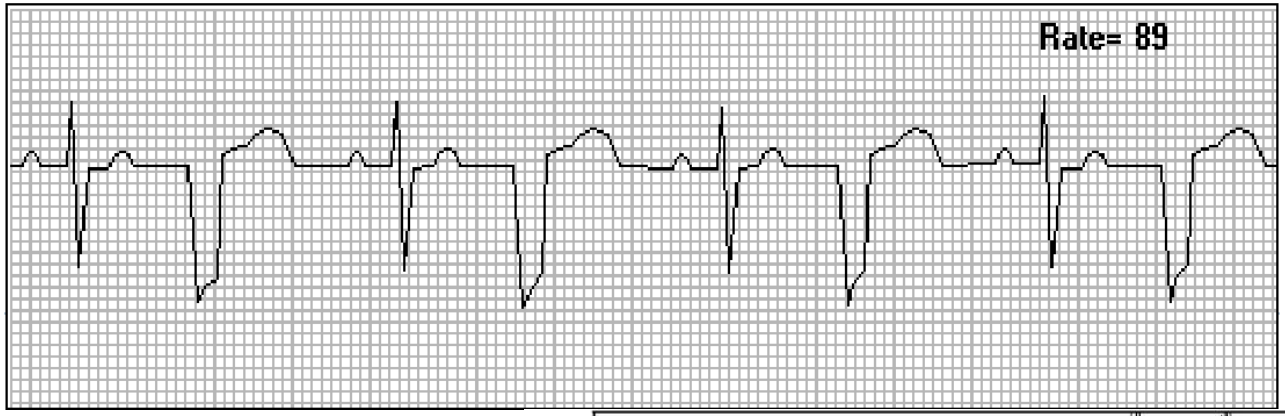


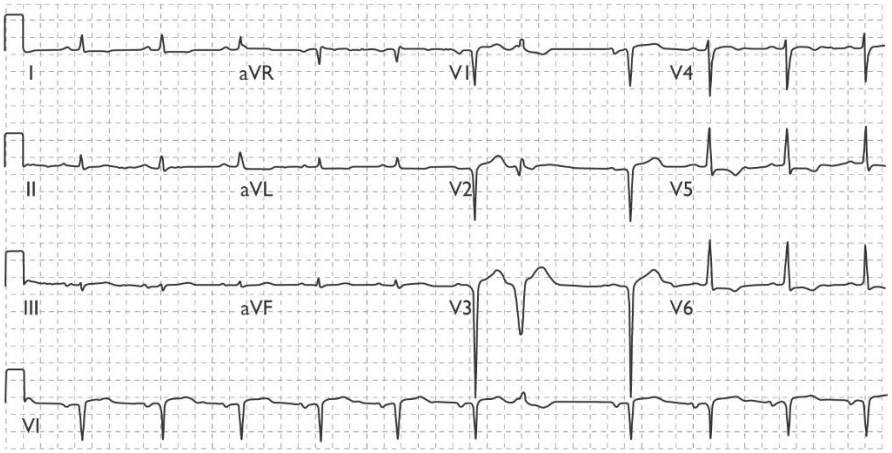
Extrasystole (ES) Premature excitation and contraction of the myocardium, which occurs against the background of sinus rhythm. The most commonly identified: 1. at 3-5 years old; 2. at 11-13 years old - in girls; at 12-15 years old - in boys. Prognostically unfavorable ES: group, frequent, against the background of lengthening of the QT interval, polytopic and polymorphic, as well as early and very early. Early and very early ventricular ES are especially dangerous.	1. Single (separately located) <u>2. Paired and</u> 3. Group (3 or more ES can be called an attack of ectopic tachycardia). <u>Allorhythmic ES is</u> rhythmic in the correct sequence (bigeminia, trigeminia, quadrheminia). The correct alternation of the next complexes with the ES groups is the group allorhythmic ES. Depending on the time of occurrence, late, early and ultra-early extrasystoles are distinguished	Often there are no complaints "Interruptions" or "sinking" in the heart. Auscultatory, a premature tone and a pause after it are heard.	Diastole shortening before ES and compensatory pause after it. The shape of the ectopic complex depeHRD on the place of occurrence of ES (slightly altered narrow complexes in supraventricular ES, wide, with a discordant ST-T position in ventricular ES). ES from one source - homotopic, from different sources - polytopic QRS complexes of the same form are monomorphic, and QRS complexes differing in form are polymorphic.	The reason for the occurrence of ES, its type and form is taken into account. With organic lesions, treatment of the underlying disease is required. In the presence of adverse ES, the drugs of choice are ethazizin and etmozin. Cardiotrophic therapy. For supraventricular ES, isoptin (finoptin, verapamil) is recommended, for ventricular ES - etacizin, etmozin.

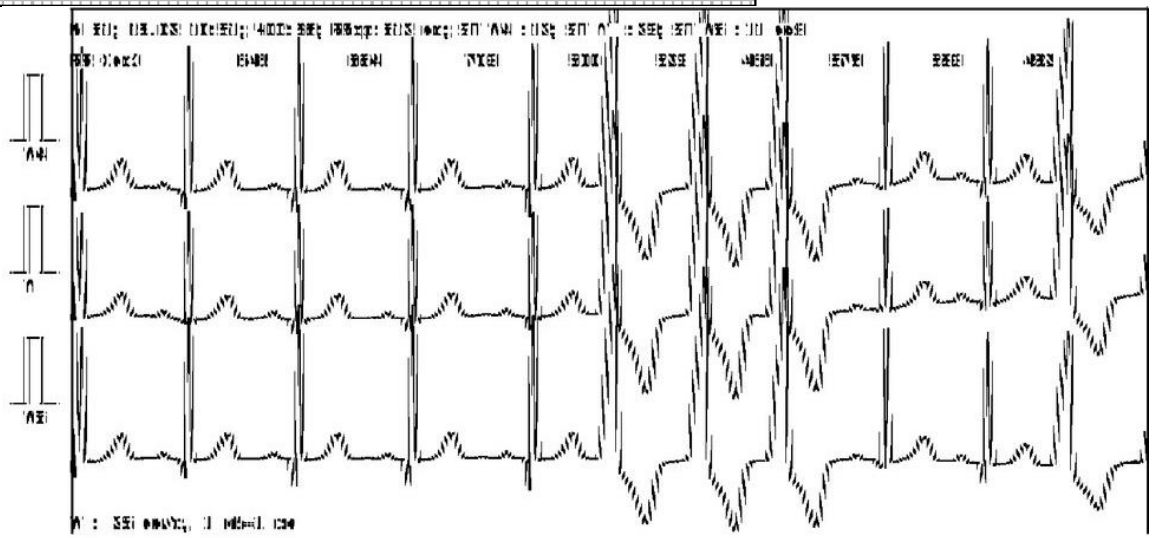




Ventricular premature beats	Provoking factors: anxiety, stress, alcohol, caffeine, sympathomimetics, hypoxia, electrolyte disturbances.	Missing or missed hits; PVCs alone are not felt, but subsequent sinus contraction is felt. Frequent PVC causes moderate hemodynamic impairment. Preexisting systolic murmurs in the heart can be significantly increased due to increased filling and increased contraction after a compensatory pause.	A wide QRS complex without a preceding P wave, usually with a full compensatory pause.	PVCs in patients without heart disease are insignificant and do not require special treatment, except for the elimination of provoking factors. Beta blockers or ablation are only offered if symptoms are unbearable or if PVCs are very frequent and induce heart failure. Other antiarrhythmic drugs that suppress PVCs, in turn, can provoke other serious cardiac arrhythmias. Indications for the appointment of AARP: 1) Malignant arrhythmias (paired, group, polymorphic, early and very early, ES against the background of prolonged int. QT). 2) Arrhythmogenic disorders of hemodynamics.

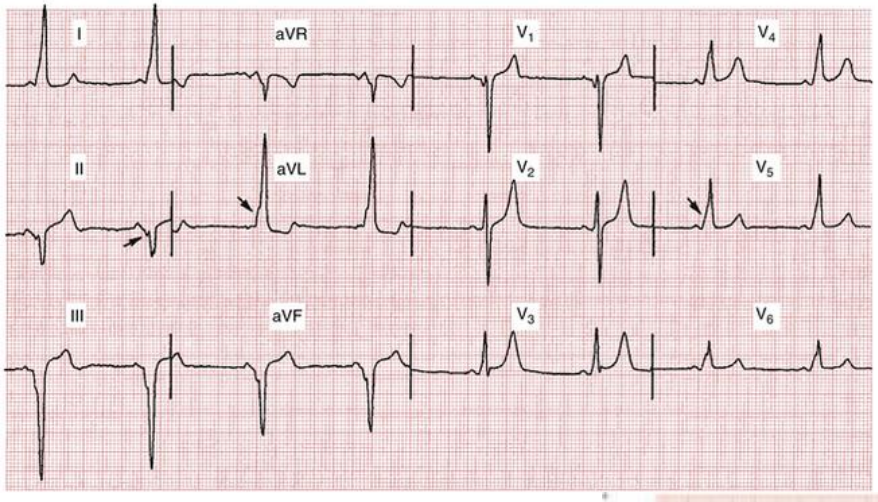


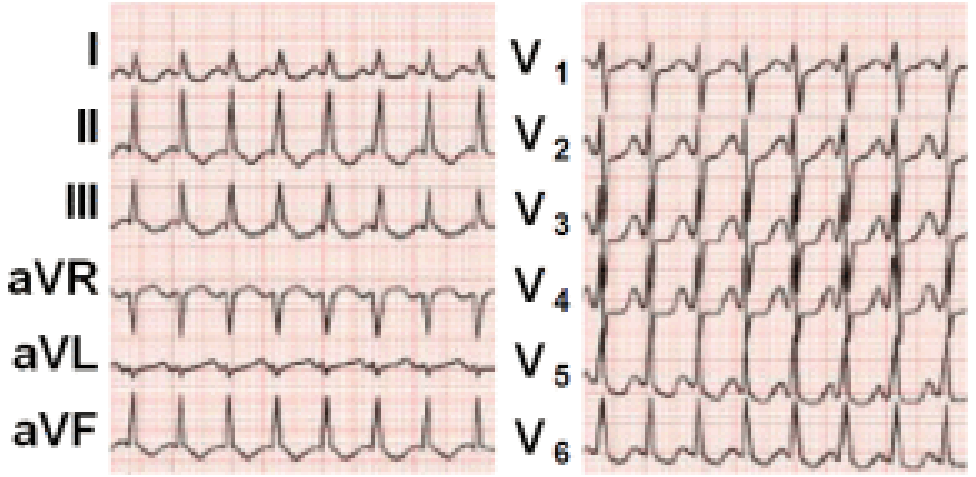


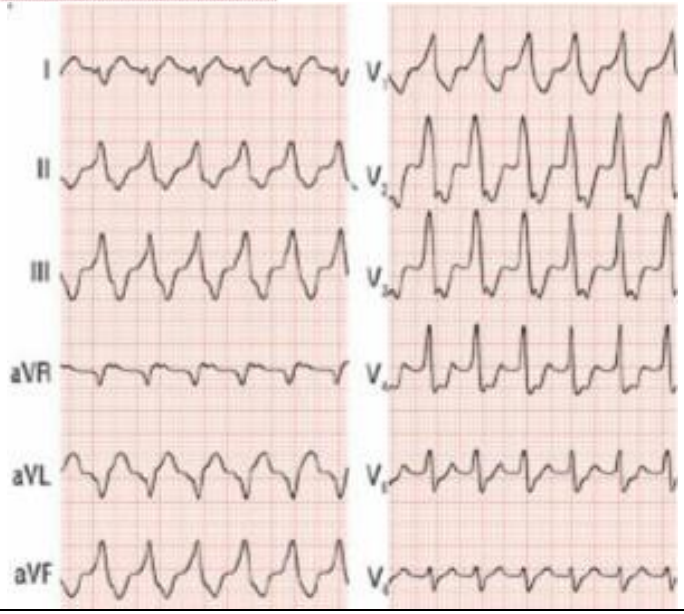


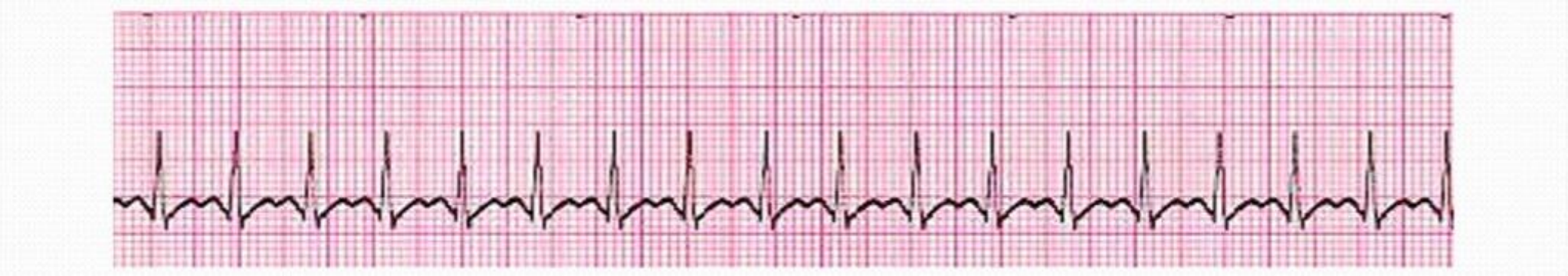
"WPW syndrome": a set of ECG manifestations and clinical manifestations in the form of paroxysms of supraventricular tachycardia **"WPW phenomenon":** ECG manifestation without attacks of paroxysmal tachycardia. The WPW phenomenon is spoken of if a patient has signs of ventricular	1. Constant (ECG symptoms of the syndrome are recorded constantly); 2. Transient (both ways of conduction function alternately - normal, and additional - along the Kent's bundle). The ECG shows either normal or deformed complexes). 3. Latent - occurs during provocation (for example, obsidan breakdown); 4. Hidden - it must be caused by special	Attacks of rapid rhythmic heartbeat, which starts and stops suddenly, accompanied by palpitations, dizziness, lightheadedness, fainting. With the manifestation of SVC syndrome, antegrade conduction is carried out through an additional conduction route. When atrial fibrillation (AF) occurs, ventricular fibrillation and sudden death can occur. Patients with latent WPW syndrome are not at risk because additional pathways do not conduct excitation in the antegrade direction.	1. Δ wave 2.shortening the P-R (P-Q) interval 3.expansion of the QRS complex ECG during tachycardia in WPW syndrome Orthodromic tachycardia: 140-240 beats / min. The QRS is usually narrow, in which case P waves are visible after the end of the ventricular complex with an R-P <P-R characteristic. Antidromic	The therapy of choice for SVC syndrome is direct current cardioversion. Conventional drugs used to control heart rate in atrial fibrillation are not effective, and digoxin and nondihydropyridine calcium channel blockers (eg, verapamil, diltiazem) are contraindicated because they can increase the rate of ventricular contraction and lead to ventricular fibrillation. If cardioversion is not possible, then drugs that increase the refractory period of the accessory pathway should be used. Intravenous administration of procainamide or amiodarone is preferred, but

pre-excitation on the surface ECG against the background of sinus rhythm, but there is no history of clinical manifestations of AVRT	electrophysiological methods.		AVRT has a wide QRS complex and P waves are either not visible or preceding the QRS complex. Intracardiac electrophysiological examination (EPI) is an obligatory stage of preoperative topical diagnosis.	antiarrhythmic drugs of classes Ia, Ic, and III can also be used.

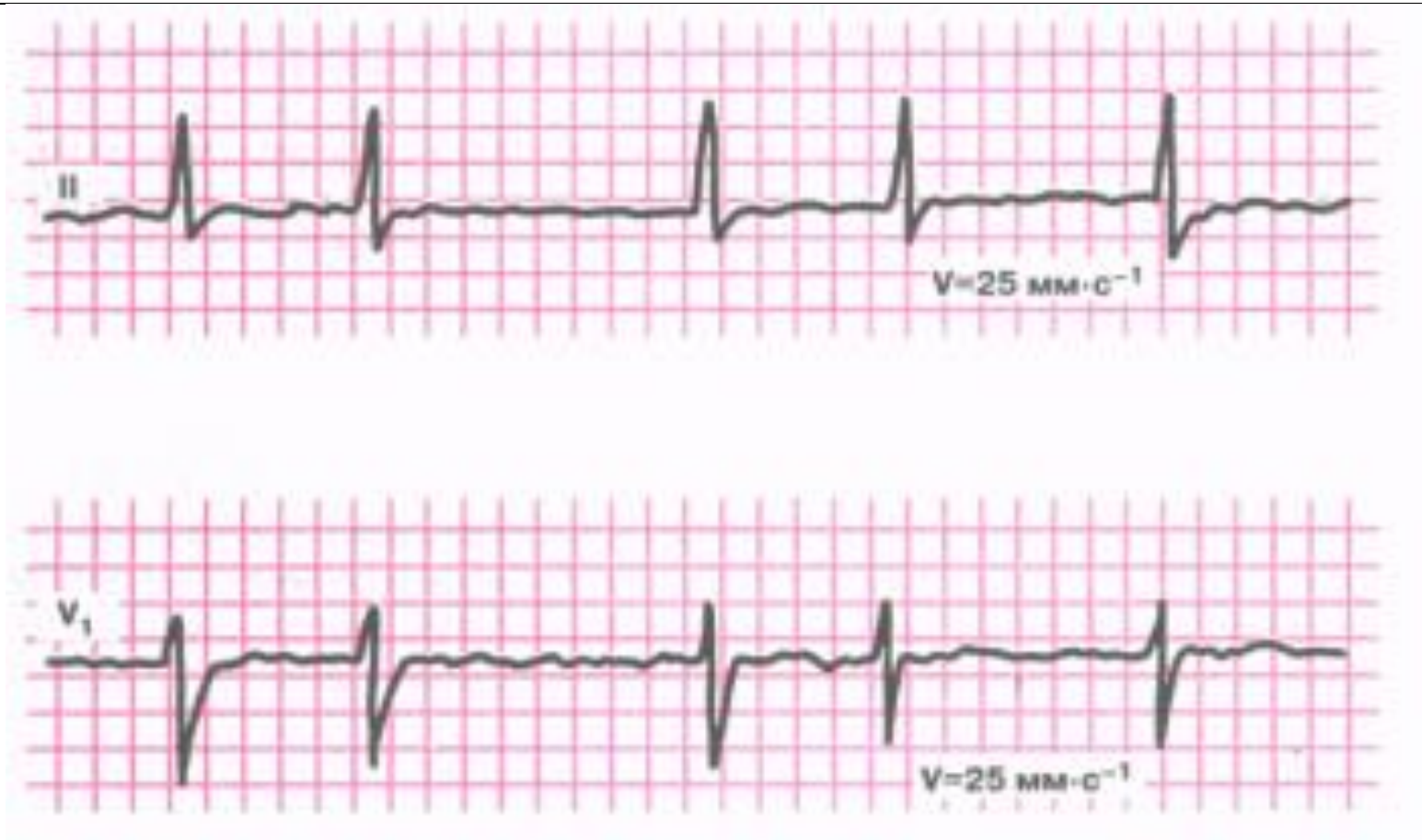






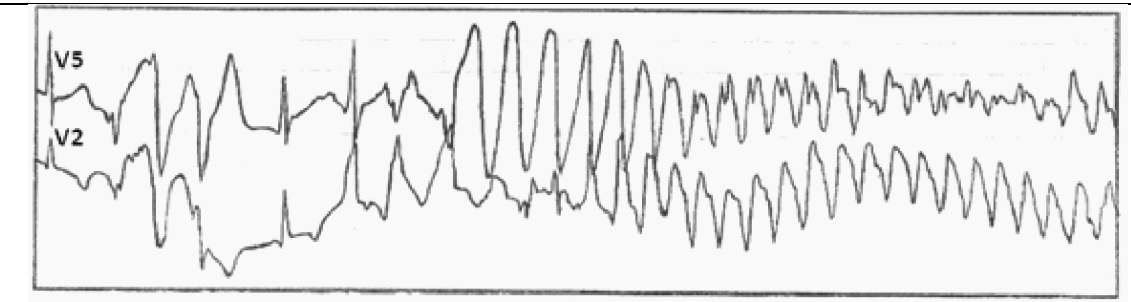
Paroxysmal supraventricular tachycardia (SVT)	The reentry path is inside: 1. Atrioventricular (AV) node (approximately 50%) in people without other diseases. It is most commonly caused by atrial premature beats. 2.Additional workaround (40%) - with SVC syndrome 3.Atrial or sinoatrial (SA) node (10%)	Episodes of sudden and ending, frequent, rhythmic and often hemodynamically significant heartbeats Dyspnea Chest discomfort Dizziness Attacks can last from a few secoHRD to several hours (rarely> 12 hours). In newborns and young children, shortness of breath, malnutrition, or rapid precordial pulsation may occur sporadically, and may be accompanied by manifestations of heart failure.	Frequent, rhythmic tachycardia. Possibly a manifest type of WPW syndrome. The P waveform can be different. In most cases of AV nodal tachycardia, P waves are located in the terminal part of the QRS complex; in about a third of cases, immediately after the QRS complex and, very rarely, before it. In the case of orthodromic reciprocal tachycardia, P-waves always follow the QRS complex. QRS complex narrow Wide complex tachycardia should be differentiated from ventricular tachycardia	Treatment 1. Vagus tests. Patients can use them on their own. Vagotonic techniques (Valsalva test, unilateral massage of the carotid sinus, immersion of the face in cold water, drinking ice water) can stop tachyarrhythmias in the earliest period. 2. Adenosine 3. Verapamil or diltiazem for tachycardia with narrow complexes. If the mechanism of tachycardia is unknown and ventricular tachycardia cannot be ruled out, intravenous administration of procainamide or amiodarone. 4. With frequent relapses - ablation Avoiding the use of beta-blockers, with wide-complex tachycardia, it is possible to use electrocardioversion, or the introduction of procainamide, amiodarone
				
Atrial fibrillation (AF) is a fast, irregular atrial rhythm. Isolated AF is AF for no known cause.	Causes: Arterial hypertension Cardiomyopathy Mitral or tricuspid defects Hyperthyroidism Excessive consumption of alcohol ("holiday of the heart" syndrome) Pulmonary embolism	Interruptions in the work of the heart Rapid heartbeat Weakness Decreased exercise tolerance, Shortness of breath Presyncopal states. Thrombus formation in the atrial cavity, Reliable risk of embolic stroke.	ECG. Missing P waves The presence of f waves between QRS complexes; waves f are irregular in time, have an irregular morphology; atrial rhythm> 300 beats / min; Irregular irregular R-R intervals.	Rhythm control 1. Synchronized cardioversion (100 J, then 200 and 360 J if necessary) 2. Drugs (procainamide, quinidine, disopyramide), Ic (flecainide, propafenone) and class III (amiodarone, dofetilide, dronedarone,

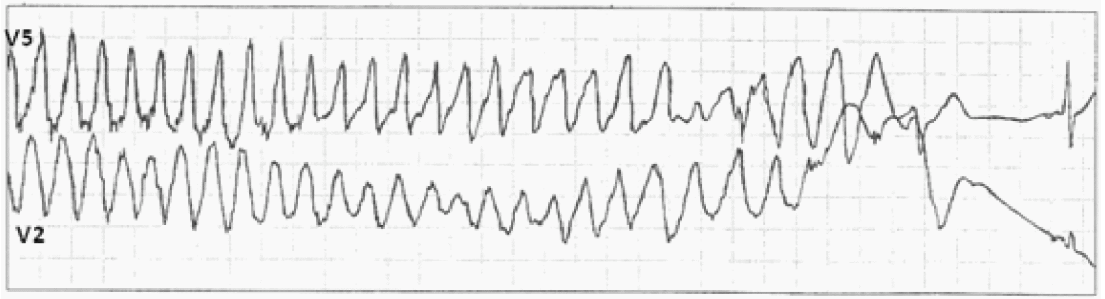
	Atrial septal defects and other congenital heart defects COPD Myocarditis Pericarditis **Classification** **1. Paroxysmal AF <1 week** **2. Persistent AF> 1 week.** **3. Long-term persistent AF>** 1 year, the possibility of restoration of sinus rhythm. **4. Permanent form: sinus rhythm cannot be restored.**	Pulse irregular with loss of jugular venous pulse waves. Pulse deficiency	Wide QRS is with WPW syndrome. Echocardiography Examination of thyroid function.	ibutilide, sotalol) Should not be used while heart rate is controlled with beta-blockers or nondihydropyridine calcium channel blockers. Calcium antagonists should not be used in patients with WPW syndrome (wide QRS complexes); possible ventricular fibrillation. 3. ACE inhibitors, ARB-II and aldosterone blockers slow down myocardial fibrosis, which is a substrate for AF in patients with heart failure. 4. Prevention of thromboembolism during rhythm restoration If the onset of current AF cannot be recognized within 48 hours, the patient should be treated with anticoagulants for 3 weeks before and at least 4 weeks after cardioversion. 5. Ablation procedure for atrial fibrillation, ineffective or impossible heart rate control

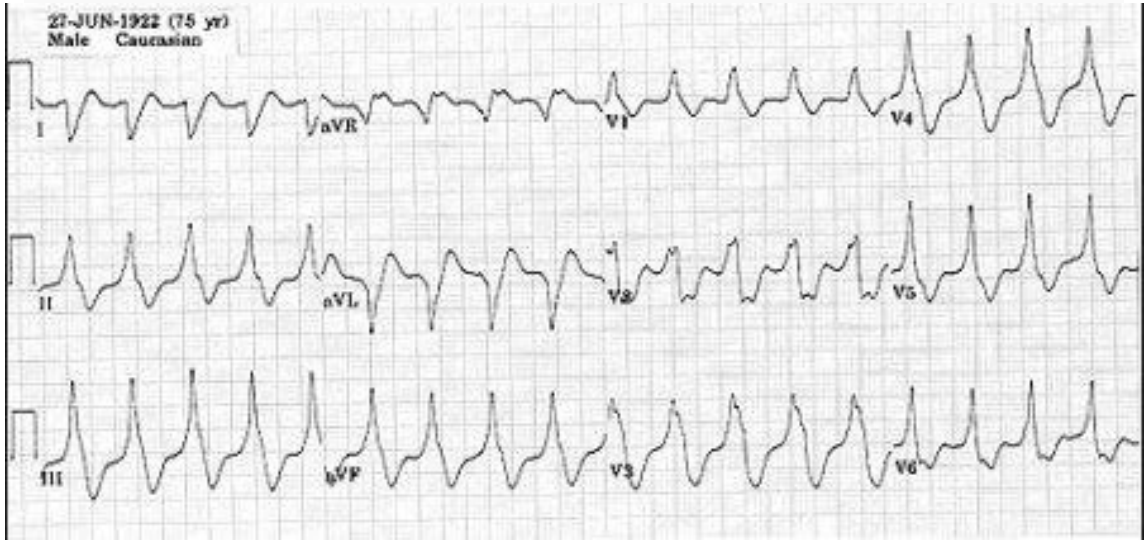


Ventricular tachycardia (VT) ≥ 3 consecutive ventricular beats at a rate of ≥ 120 beats / min.	Causes: • KMP • Electrolyte disturbances (hypokalemia, hypomagnesemia), acidemia, hypoxemia • Long QT syndrome **Monomorphic VT:** single pathologic focus or reentry mechanism and regular, identical QRS. **Polymorphic VT:** several different foci or accessory pathways, irregular QRS complexes	VT becomes ventricular fibrillation and thus causes cardiac arrest. May be asymptomatic. Sustained VT is almost always symptomatic, causing palpitations, HF symptoms, or sudden cardiac death.	Any wide QRS tachycardia ≥0.12 sec should be considered VT until a different tachycardia is proven. Dissociation of P waves, confluent complexes or supraventricular "captures", vectors of QRS concordance in the chest leads with a discordant T-wave vector.	Acute VT: 1. Pulseless VT: defibrillation with a discharge energy ≥ 100 joules. 2. Stable sustained VT: synchronized DC electrical cardioversion with an energy of ≥100 J. OR intravenous AARP I or III classes. Lidocaine is fast acting but often ineffective. If lidocaine is ineffective, a class IV drug, procainamide, can be used. Failure of intravenous procainamide or intravenous

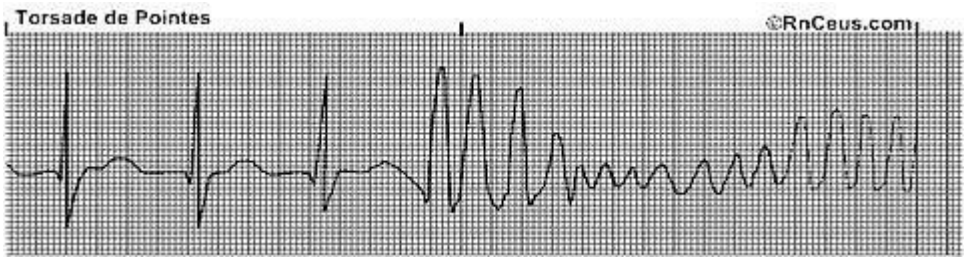
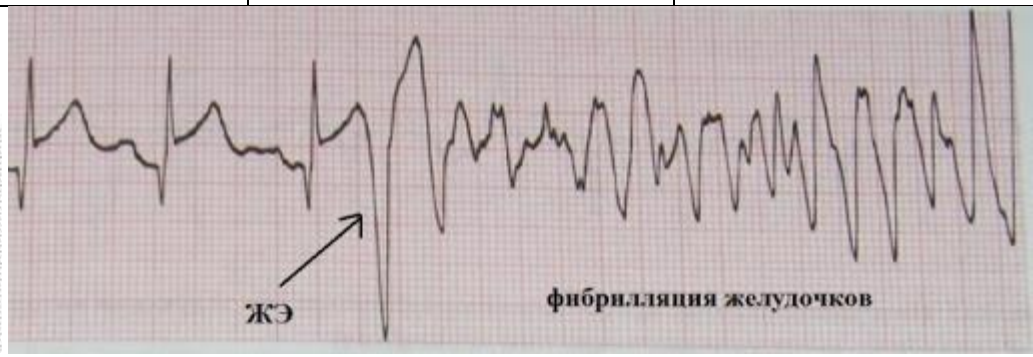
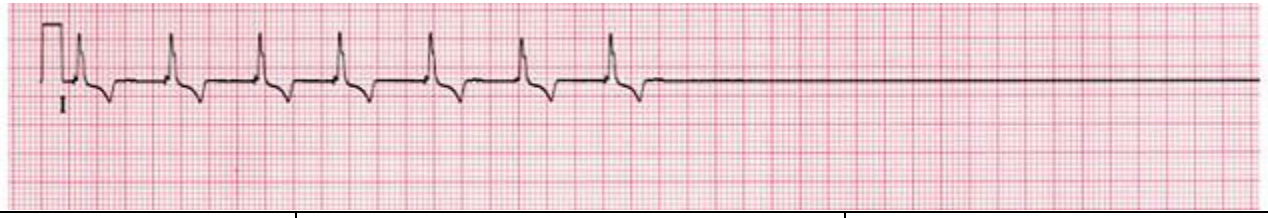
	Volatile VT: <30 sec **Sustained VT: ≥ 30 sec**			amiodarone is an indication for cardioversion. Prolonged VT: 1. Implantable cardioverter defibrillator (ICD). Long-term treatment is not required after a transient cause (acid-base disorders, electrolyte disturbances, proarrhythmic drugs).

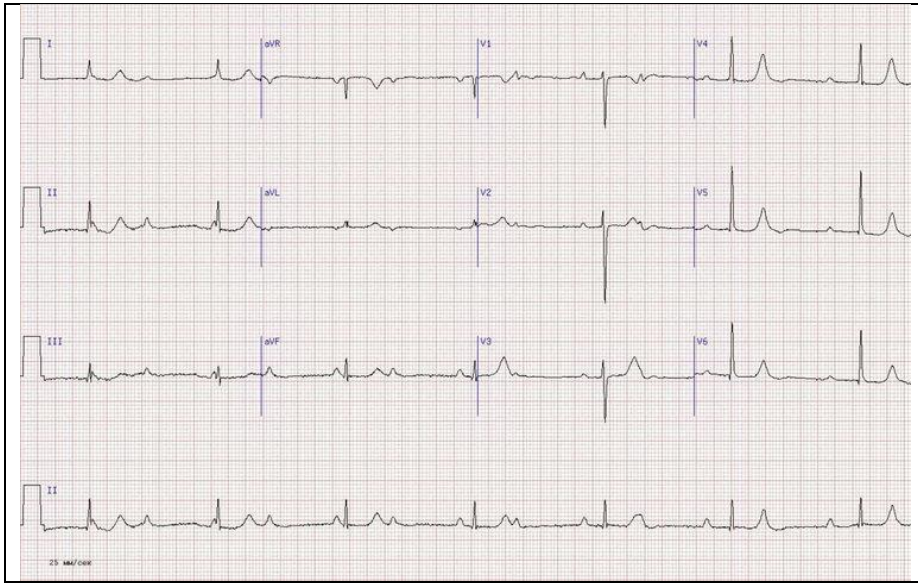






Ventricular fibrillation (VF)	Various diseases. The risk of VF in many diseases increases with electrolyte disturbances, acidosis, hypoxemia, or ischemia. Less common is among infants and children, in whom asystole is a more	***immediate syncope and death within minutes.***	very fast waves, irregular in time and morphology	1 CPR. Defibrillation The probability of success with immediate (within 3 minutes) defibrillation is about 95%, provided that there is no preexisting insufficiency of the pumping function of the heart. 2.Implantable cardioverter defibrillator

	common cause of circulatory arrest (Fig. 3).			
  				
Atrioventricular block partial or complete interruption of the impulse from the atria to the ventricles.	Causes: Idiopathic fibrosis and sclerosis of the vascular system Drugs (beta blockers, calcium channel blockers, digoxin, amiodarone) Increased vagus tone Heart valve pathology Congenital heart disease, genetic or other disorders AV block can be partial or complete. Blockade of the first degree, second degree - these are partial forms. The third degree of blockade is complete.	Grade I AV block can be physiological in young patients with a pronounced vagotonic reaction of the nervous system and in well-trained athletes. Grade I AV block is rarely symptomatic and does not require treatment. Mobitz type II AV block II degree is always pathological. III degree AV block: fatigue, postural dizziness, decreased exercise tolerance, light-headedness, fainting, heart failure, blood pressure fluctuations, changes in the 1st heart sound (S1)	1st degree AV block: conduction is slowed down. All normal P waves precede QRS complexes, but the PR interval is greater than normal (> 0.2 s). Mobitz type I 2nd degree AV block: the PR interval gradually lengthens with each contraction, followed by the absence of QRS (Samoilov-Wenckebach phenomenon. Mobitz type II AV block II degree: the PR interval remains constant. Loss of some QRS is usually in a repetitive cycle (3: 1 block) or (4: 1 block). III degree AV block: there is no relationship between P-waves and QRS complexes (AV dissociation). Replacement nodal or idioventricular rhythm.	Most patients require pacemaker implantation With a complete blockade: Hospitalization in the intensive care unit. A 0.1% solution of atropine sulfate (0.1 mg per 1 kg of body weight), steroid hormones are injected intravenously. With a short attack of asystole, sublingual izadrin is given 1 / 2-1 tablet or 0.2% norepinephrine solution is injected with hydrotartrate 0.5-1 ml, or 0.05% alupent solution (0.1 ml for 1 year life - no more than 1 ml per 200 ml of 5% glucose solution) under ECG control.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aVR
V1
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aVL
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III
aVF
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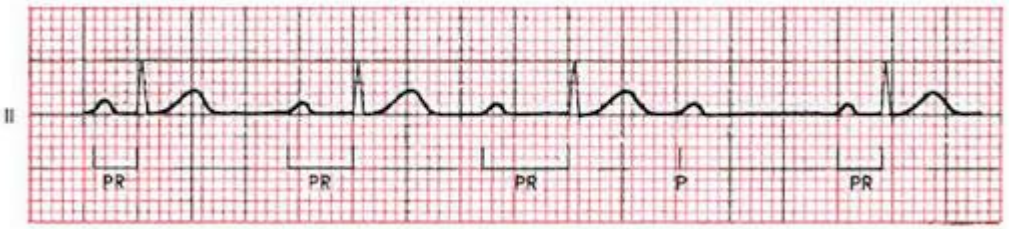
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Heart failure (HF) is a syndrome of dysfunction of the ventricles of the heart.
The most common classification of HF today is:

1. Heart failure with reduced ejection fraction ("systolic HF"). LVEF ≤ 40%.
2. Heart failure with preserved ejection fraction ("diastolic HF"). LVEF ≥ 50%.
3. HF with an average EF: LVEF - 40 - 50%.

The traditional distinction between left and right ventricular failure is somewhat erroneous, pathological changes in one chamber ultimately affect the work of the whole heart. However, these terms define the location of the greatest lesion leading to heart failure and may be useful for initial diagnosis and treatment.
Other, most commonly used terms used to describe HF: acute and chronic; high or low cardiac output; ischemic, hypertensive, or idiopathic dilated cardiomyopathy.

Age	**Possible cause of HF**
Intrauterine	Chronic anemia followed by hypersystolic HF Large systemic arteriovenous fistulas (eg, Galen cerebral vein shunt) Myocardial dysfunction secondary to myocarditis Persistent intrauterine tachycardia
From birth to the first few days	Any of the above Critical aortic stenosis or critical coarctation of the aorta Ebstein's anomaly with severe tricuspid and / or pulmonary regurgitation Left heart hypoplasia syndrome Neonatal paroxysmal supraventricular tachycardia Metabolic disorders (eg, hypoglycemia, hypothermia, severe metabolic acidosis) Perinatal asphyxia with myocardial injury Severe intrauterine anemia Complete abnormal drainage of pulmonary veins with severe obstruction (usually of the infracardiac type)
Up to 1 month	Any of the above Coarctation of the aorta with or without associated abnormalities Complete heart block is associated with structural abnormalities of the heart Large left-to-right shunt in premature babies Transposition of the great arteries with a large ventricular septal defect
Infancy (especially 6 to 8 weeks)	Bronchopulmonary dysplasia (right ventricular failure) Complete atrioventricular septal defect Patent ductus arteriosus Common arterial trunk Rare metabolic disorders (eg, type 2 glycogen storage disease - Pompe disease) Single ventricle Supraventricular tachycardia Ventricular septal defect
Childhood	Acute cor pulmonale Acute rheumatic fever with carditis Acute severe hypertension (eg, with acute glomerulonephritis) Total abnormal pulmonary venous return (non-obstructive) Bacterial endocarditis Anemia (severe) Dilated cardiomyopathy

	Excess iron due to altered iron metabolism (hereditary hemochromatosis) or due to frequent transfusions (for example, with erythroblastic thalassemia) Protein-energy deficiency Valvular heart disease due to congenital or acquired heart disease Viral myocarditis Volume overload in extracardiac disorders

Symptoms and signs of HF in infants:

1. Tachycardia;
2. Tachypnea;
3. Shortness of breath when feeding;
4. Diaphoresis, especially when feeding;
5. Anxiety, irritability;
6. Hepatomegaly;
7. Inadequate nutrition and low growth rates;
8. In most cases, there are no dilated neck veins and edema (unlike adults and older children), but they sometimes have a pronounced edema of the periorbital region.

Symptoms in older children with heart failure are similar to those in adults.

Diagnosis is clinical, confirmed by chest x-ray, echocardiography, and plasma levels of natriuretic peptides.

Treatment:

- ✓ patient education;
- ✓ diet: limiting salt intake, with critical CHD, it may be recommended to abstain from feeding - minimizing the risks of necrotizing enterocolitis; shunts from left to right - increased calorie content (↑ calorie supply ↓ risk of volume overload); some babies require tube feeding;
- ✓ diuretics;
- ✓ ACE inhibitors;
- ✓ BRA-II;
- ✓ beta blockers;
- ✓ Digoxin: Used less frequently, with significant left-to-right shunts and in some postoperative patients with congenital heart disease (reduces mortality among patients with a single ventricle after the Norwood procedure, as well as before two-stage surgery). It decreased with the treatment of supraventricular tachycardia in newborns, as it leads to higher mortality than with propranolol;
- ✓ aldosterone antagonists;
- ✓ neprilisin inhibitors;
- ✓ in some cases, supplemental oxygen or prostaglandin E1; specialized implantable pacemakers / defibrillators, etc.
- ✓ correction of the causes of HF;
- ✓ radical therapy usually requires correcting the underlying problem.

Features of HF therapy in children of certain age categories	
Newborn	**Infants and young children**
• Requires emergency medical attention • Provide safe vascular access, preferably through an umbilical venous catheter.	Diuretics: furosemide 0.5-1 mg / kg IV or 1-3 mg / kg orally every 8-24 hours, titrated if necessary, increasing the dose.

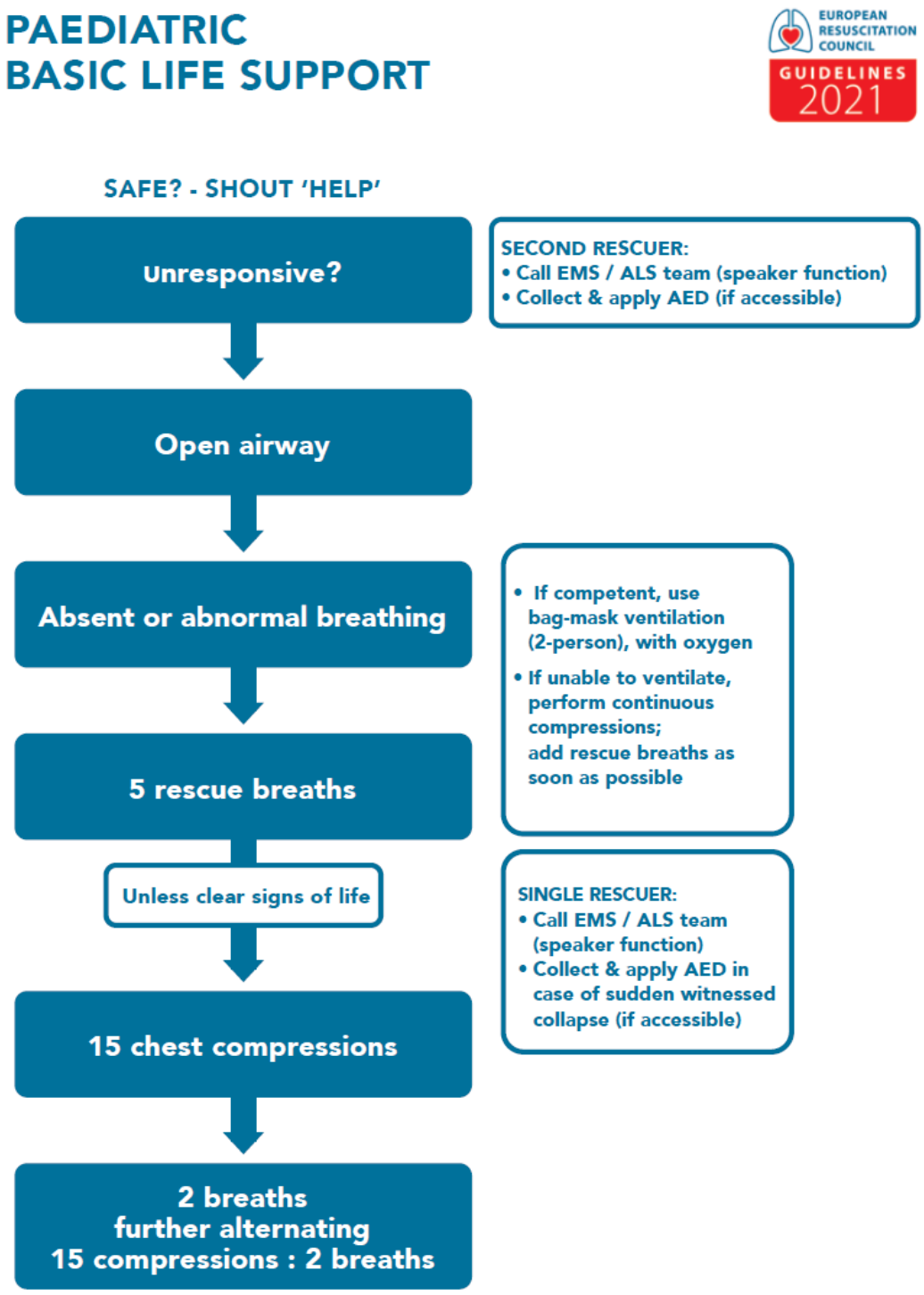
• If critical CHD is suspected: IV prostaglandin E1 at a dose of 0.05–0.1 mcg / kg / min. • mechanical ventilation: prescribe reasonably or even postpone (for example, in the syndrome of hypoplasia of the left heart, ductus-dependent defects). • Diuretics: furosemide - initial bolus 1 mg / kg IV, then titrated based on urine volume. • Inotropic drugs: dopamine / dobutamine can maintain blood pressure, ↑ heart rate and afterload, ↑ myocardial oxygen consumption, rarely used in children with CHD. Milrinone: postoperative patients with congenital heart disease, positive inotropic effect, vasodilator. Dopamine, dobutamine, and milrinone increase the risk of arrhythmias. • Nitroprusside: postoperative hypertension - from 0.3-0.5 mcg / kg / min to the desired effect (the usual maintenance dose is about 3 mcg / kg / min).	Potassium-sparing diuretics: spironolactone 1 mg / kg orally 1-2 times / day, titrated to 2 mg / kg / dose if necessary ACE inhibitors: captopril 0.1–0.3 mg / kg orally 3 times / day. Beta-blockers (carvedilol, metoprolol): CHF.

Emergency care and resuscitation measures in children with acute heart failure

Pulmonary edema - acute severe left ventricular failure with pulmonary venous hypertension and alveolar edema	**Symptoms:** severe shortness of breath, feeling short of breath, sweating pallor, cyanosis, fussiness and anxiety, wheezing sometimes frothy sputum, stained with blood, some patients have foam at the mouth, the pulse is fast, with low filling, BP: hypertension (significant cardiac reserve) hypotension is a threatening sign, wheezing, inspiratory crepitus, scattered along the anterior and posterior surfaces over all pulmonary fields, gallop rhythm (III (S3) + IV (S4)) signs of pancreatic insufficiency (swelling of the	**Therapeutic tactics:** 1. Treatment of the identified cause. 2. 100% oxygen in oxygen mask with valve, stay upright. 3. Intravenous diuretic: furosemide 0.5–1.0 mg / kg intravenously or by continuous infusion of 5–10 mg / h; 4. Nitrates: nitroglycerin 0.4 mg sublingually every 5 min, followed by intravenous drip at 10–20 mg / min with an increase in the dose by 10 mg / min every 5 min, if necessary up to a maximum dose of 300 mg / min, if systolic blood pressure is > 100 mm Hg. Art. 5. Inotropes intravenously 6. Morphine 1-5 mg intravenously once or twice a day has been used for a long time to reduce anxiety and ease breathing, but it is being used less and less. 7. Ventilation support is BiPAP. Tracheal intubation and mechanical ventilation are necessary in the event

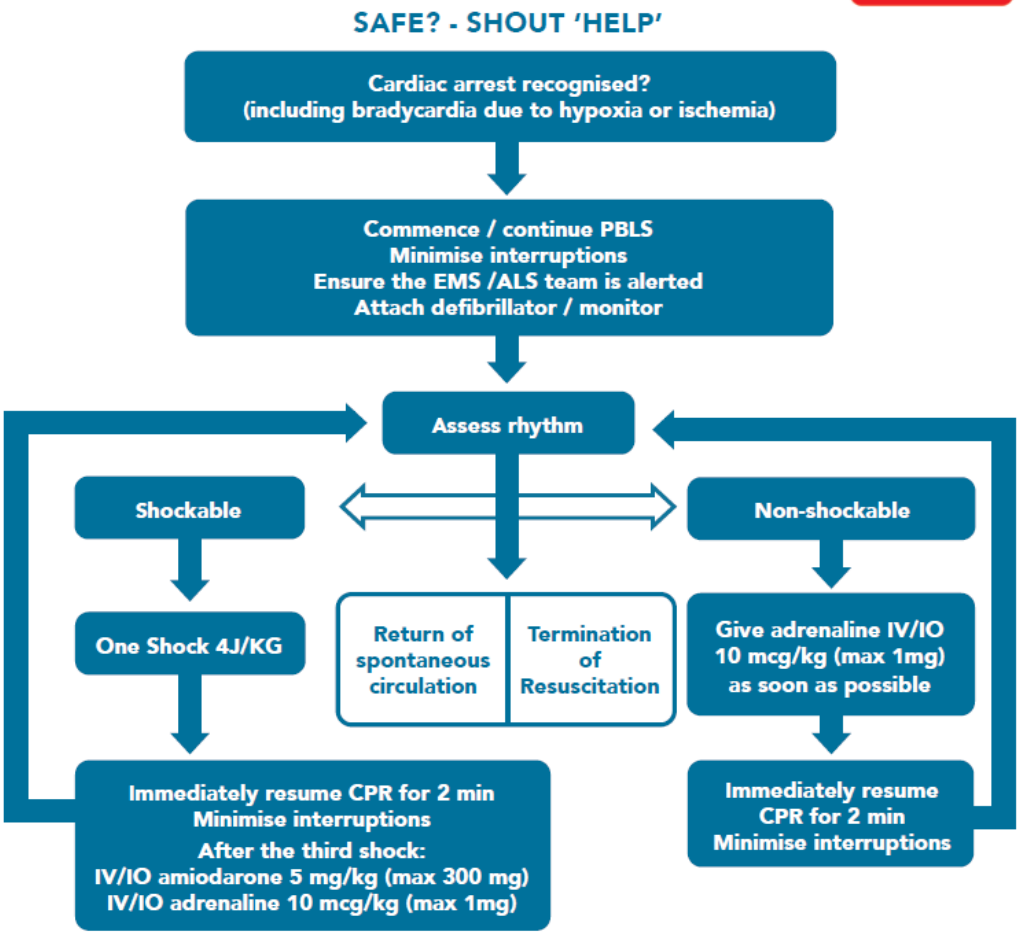
	veins of the neck, peripheral edema). Differential diagnosis: exacerbation of COPD (usually long history of COPD, excessive shortness of breath)	of CO2 retention or patient retardation. 8. Cardioversion is the preferred method in severe atrial fibrillation. For supraventricular or ventricular tachycardia, direct current cardioversion is performed. 9. There are intravenous beta-locators, intravenous digoxin, or careful intravenous administration of calcium channel blockers to slow the ventricular rate. 10. There are intravenous dobutamine and intra-aortic balloon counterpulsation develops is hypotension <100 mm Hg. Art. or shock
Common reasons: • acute coronary ischemia, • decompensation of primary heart failure, • arrhythmias, • acute dysfunction of valves • acute volume overload.	**Diagnostics:** Clinical picture Chest x-ray: a picture of interstitial edema Pulse Oximetry ECG Determination of serum BNP or NT-pro-BNP, cardiac markers and other tests to establish etiology as needed	
Cardiac arrest is the termination of its mechanical activity, lack of blood circulation. **Sudden cardiac arrest** is an unexpected cessation of circulation within a short period of time after the first symptoms appear (sometimes there are no foreshadowing signs). **The predominant causes of sudden cardiac arrest** are respiratory failure due to respiratory distress, injury and poisoning.	**Symptoms:** (in terminal patients preceded by a period of clinical deterioration with rapid, shallow breathing, arterial hypotension and a progressive decrease in mental activity): ✓ unconsciousness ✓ apnea / agonal breathing pattern ✓ pallor or deep cyanosis ✓ lack of response to pain (coma) ✓ lack of pulse ✓ BP is not measurable ✓ pupils dilate and do not respond to light after a few minutes **Diagnostics** ✓ Clinical evaluation ✓ Heart rate monitoring ✓ ECG ✓ Sometimes EchoCG, chest x-ray / ultrasound The patient's condition is assessed in relation to potentially curable causes; in this case, the so-called "T" - reasons and "G" - reasons serve as a reminder:	**CPR for children and infants should begin with chest compressions (C-A-B, not A-B-C sequence) if one resuscitator is 30, if two are 15.** **https://www.heartaHRDtroke.ca/-/media/pdf-files/canada/cpr-2017/ecc-highlights-of-2015-guidelines-update-for-cpr-ecclr.ashx;** **https://eccguidelines.heart.org/wp-content/uploads/2015/10/2015-AHA-Guidelines-Highlights-Russian.pdf**

	"D" causes: hypoxia, hypovolemia, acidosis, hyperkalemia / hypokalemia, hypothermia, hypoglycemia; "T": poisoning with pills or toxins, cardiac tamponade, tension pneumothorax, thromboembolism, trauma	



PAEDIATRIC ADVANCED LIFE SUPPORT

EUROPEAN RESUSCITATION COUNCIL GUIDELINES 2021



DURING CPR

- Ensure high-quality CPR: rate, depth, recoil
- Provide bag-mask ventilation with 100% oxygen (2-person approach)
- Avoid hyperventilation
- Vascular access (intravenous, intraosseous)
- Once started, give adrenaline every 3-5 min
- Flush after each drug
- Repeat amiodarone 5 mg/kg (max 150mg) after the 5th shock
- Consider an advanced airway and capnography (if competent)
- Provide continuous compressions when a tracheal tube is in place. Ventilate at a rate of 25 (infants) – 20 (1-8y) – 15 (8-12y) or 10 (>12y) per minute
- Consider stepwise escalating shock dose (max 8J/kg – max 360J) for refractory VF/pVT (≥6 shocks)

CORRECT REVERSIBLE CAUSES

- Hypoxia
- Hypovolaemia
- Hyper/hypokalaemia, -calcaemia, -magnesemia; Hypoglycaemia
- Hypothermia - hyperthermia
- Toxic agents
- Tension pneumothorax
- Tamponade (cardiac)
- Thrombosis (coronary or pulmonary)

ADJUST ALGORITHM IN SPECIFIC SETTINGS (E.G. TRAUMA, E-CPR)

IMMEDIATE POST ROSC

- ABCDE approach
- Controlled oxygenation (SpO_2 94-98%) & ventilation (normocapnia)
- Avoid hypotension
- Treat precipitating causes

Advanced resuscitation in pediatrics.

Assessment of a seriously ill or injured child - preventing cardiac arrest and respiratory arrest.

The assessment and intervention procedure for any seriously ill child follows the ABCDE guidelines.
• A - airway patency.
• B - breathing.
• C - blood circulation.
• D - neurological status.
• E - appearance.
Items D and E are outside the scope of these guidelines.

Diagnosis of Respiratory Failure: Assessment A and B.
Evaluation of potentially critically ill children begins with an assessment of the airways (A) and breathing (B). Signs of respiratory failure may include:
• A breathing rate that is higher than normal for the child's age.
• Initially, increased work of breathing, which can develop into inadequate / reduced work of breathing, as the child gets tired or his compensatory mechanisms are depleted.
• Additional breathing souHRD such as stridor, wheezing, crepitus, snorting, or no breathing souHRD.
• Decreased tidal volume, manifested by shallow breathing, decreased pulmonary excursion, or decreased air intake on auscultation.
• Hypoxemia (with or without oxygen) is mainly manifested by cyanosis, but it is often possible to detect it before using pulse oximetry.

At stage C, the following signs can be identified:
• Increased tachycardia (compensatory mechanism that increases the delivery of oxygen to the tissues).
• Pallor.
• Bradycardia (a formidable harbinger of the exhaustion of the possibilities of compensatory mechanisms).
The transition from the state of compensation to decompensation can occur in an unpredictable manner. Thus, the child should be monitored quickly
identify and correct any deterioration in its physiological parameters.

Treatment for cardiac / respiratory arrest
Airway and breathing.
• Open the airways.
• Optimize ventilation.
• Ensure adequate oxygenation, start with 100% oxygen.
• Start monitoring respiration (SpO2).
• Obtain adequate ventilation and oxygenation - this may require the use of airway devices ± bag and mask ventilation, the use of a laryngeal mask or other supraglottic airway devices, or tracheal intubation.

Circulation.

• Start monitoring respiration (SpO2), ECG, non-invasive blood pressure measurement.

• Provide intravenous / intraosseous access.

• Give bolus fluid (20 ml / kg) and / or drugs (eg, inotropics, vasopressors, antiarrhythmics).
With great caution, the liquid should be used as a bolus for primary cardiac dysfunction

(myocarditis, cardiomyopathy). Isotonic crystalloids are recommended as the first fluid for resuscitation in young and middle-aged children with any type of shock, including septic shock.

• Repeat the examination of the child continuously, each time starting with the respiratory tract, then breathing, and then blood circulation. Measuring blood gases and lactate can be helpful.

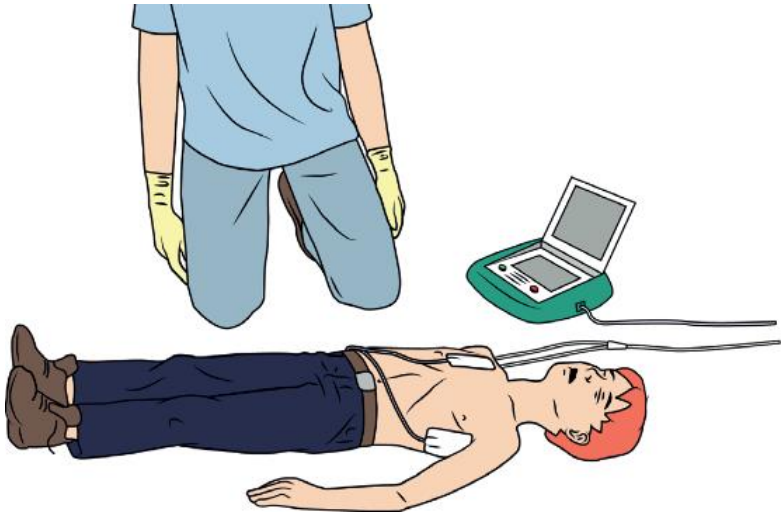
• During primary resuscitation, oxygen should be given at the highest concentration (i.e 100%). Once the child is stabilized and / or effective circulation is restored, the inspired oxygen fraction (FiO2) should be titrated until normoxemia is achieved, or at least (if arterial blood gas analysis is available) maintain the SpO2 in the 94–98% range.

• Epinephrine (epinephrine) plays a central role in treatment algorithms for cardiac arrest with rhythms that should and should not be treated with shock. In case of cardiac / respiratory arrest in children, the recommended intravenous / intraosseous dose of adrenaline for the first and subsequent administration is 10 mg / kg. The maximum single dose is 1 mg. The use of higher single doses (more than 10 mg / kg) is not recommended, as it does not improve either survival or neurological outcome.

• Amiodarone in children resistant to VF / VFBP discharge. It is administered as a bolus of 5 mg / kg after the third shock (can be repeated after the fifth shock). When treating other cardiac arrhythmias, amiodarone should be administered slowly (over 10–20 minutes) under the control of systemic blood pressure and ECG to avoid hypotension. This side effect is less common with aqueous solutions.

Most cases of cardiac / respiratory arrest in children and adolescents are of respiratory origin. Therefore, in this age group, it is imperative that CPR be performed immediately before looking for an AED or manual defibrillator, as immediate availability does not improve the outcome of respiratory arrest.

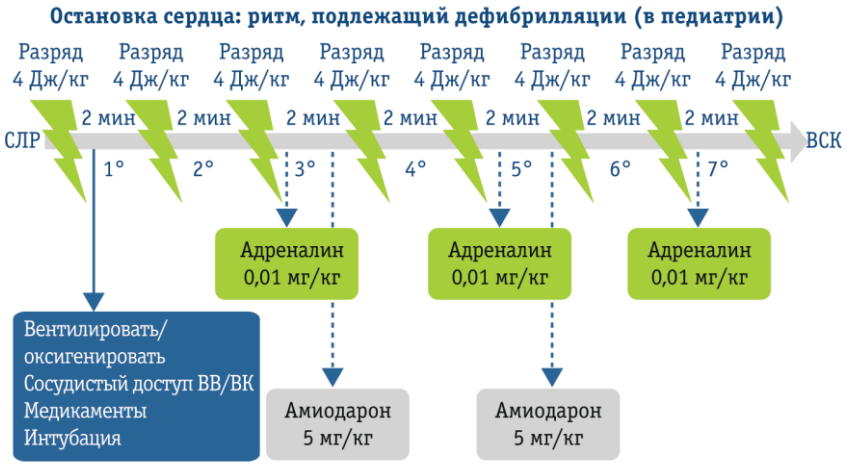
Rhythms not eligible for defibrillation	Shock defibrillation rhythms
Asystole EMD (presence of electrical activity on the ECG in the absence of pulse), after a period of hypoxia or myocardial ischemia. Sometimes it can have a removable cause (one of the four G or T).	Primary VF VT Secondary VF



Defibrillation electrode locations

Рис. Расширенные реанимационные мероприятия в педиатрии

Resuscitation algorithm for non-defibrillable rhythms



Resuscitation algorithm for rhythms subject to defibrillation

Extracorporeal life support: in children with cardiac arrest, refractory conventional CPR with a potentially recoverable cause, if the arrest occurs where there is experience, resources and a system to start it quickly.

General approaches to emergency care for unstable arrhythmias

Check for the presence / absence of signs of life and central pulse;

If there are no signs of life, treat as cardiac / respiratory arrest.

If the child has signs of life and has a central pulse, it is necessary to assess the hemodynamic status.

In any case of hemodynamic disorders, it is first of all necessary:

1. Open the airways:

2. Provide oxygen and auxiliary ventilation as needed.

3. Connect the ECG monitor and defibrillator, assess the heart rate.

4. Assess the compliance of the speed (too fast or too slow) of the rhythm with the age of the child.

5. Assess the regularity / irregularity of the rhythm.

6. Measure the QRS complex (narrow complexes: <0.08 sec; wide complexes> 0.08 sec).

7. The choice of treatment tactics depeHRD on the hemodynamic stability of the child.

Bradycardia	Narrow complex tachycardia	Wide-complex tachycardia

Cause: hypoxia, acidosis and / or severe hypotension If circulatory failure → 100% oxygen / ventilator (if necessary) If heart failure + heart rate <60 / min + no rapid response to oxygen → chest compressions (CHC) and inject epinephrine Pacing in cases of AV block or sinus node dysfunction without a response to oxygenation, ventilation, CHC and other types of drugs Pacing **is not effective** in asystole or arrhythmias caused by hypoxia or ischemia	If hemodynamics is stable → Valsalva, IV bolus adenosine, saline bolus If signs of decompensated shock with depression of consciousness / no vascular access / with the help of adenosine the rhythm could not be restored → immediately try electrical cardioversion (1st dose -1 J / kg, 2nd - 2 J / kg). If the rhythm is not restored → amiodarone or procainamide as directed by a pediatric cardiologist or resuscitator - before attempting a third attempt at cardioversion	It rarely occurs in children. In origin, it is more likely supraventricular than ventricular. In hemodynamically unstable children, **VT** should be considered until proven otherwise. **Causes of VT**: after heart surgery, cardiomyopathy, myocarditis, electrolyte disturbances, prolongation of the QT interval, central intracardiac catheter. Synchronized cardioversion. If the second cardioversion is unsuccessful or VT recurs, antiarrhythmic therapy should be considered.
Stable arrhythmias: ✓ maintaining the patency of the airways, breathing and blood circulation; ✓ consultation with an expert before starting treatment; ✓ depending on the clinical history, symptomatology, and ECG findings, the child with stable wide QRS tachycardia may be treated as NVT and vagal or adenosine may be used.		
Pulmonary hypertension: ✓ the risk of cardiac arrest is increased; ✓ routine resuscitation protocols ✓ increased attention to high FiO2 and alkalosis / hyperventilation ✓ inhalation of nitric oxide to reduce pulmonary vascular resistance.		

Postresuscitation treatment should be multidisciplinary and include all types of treatment needed for complete neurological recovery:

1. Infusion therapy and vasoactive drugs (epinephrine, dobutamine, dopamine, and norepinephrine) can improve the hemodynamic status of the child after stopping and should be titrated to maintain systolic blood pressure at least 5% above age norm.

2. The goal of oxygenation and ventilation after restoration of blood circulation and stabilization of the patient is to maintain PaO2 within the normal range (normoxemia).

3. After circulatory restoration, mild hypothermia has an acceptably safe profile in neonates. In children, both hypothermia (32–34 ° C) and controlled normothermia (36–37.5 ° C) can be used. Temperature should be strictly controlled to avoid hyperthermia (> 37.5 ° C) and severe hypothermia (<32 ° C).

4. Monitor glucose levels and avoid hypoglycemia / hyperglycemia.

5. There is no simple guide to help you determine when resuscitation attempts become hopeless. Considerations for deciding whether to continue CPR include the reason for the arrest, the patient's baseline, age, where the arrest occurred, whether the arrest occurred in front of bystanders, the duration of untreated cardiac / respiratory arrest (“no blood flow” time), the presence of a rhythm to be treated with a shock as the first or subsequent rhythm, and association with special circumstances (eg, drowning in ice water, exposure to toxic drugs). The role of EEG as a prognostic factor is still unclear.

6. The presence of parents. In some Western countries, most parents want to be present during their child's resuscitation. The presence of the parents at the death of their child showed in the future their better controllability, they more steadfastly endured the grief that befell them. The data on the presence of parents during resuscitation are obtained from some countries and, probably, they cannot be extended to all countries of Europe, in which there may be different socio-cultural and ethical perceptions.

7. After drug stabilization of acute symptoms of heart failure or cyanosis, most children require surgical or transcatheter treatment. Transcatheter procedures:

• balloon atrioseptostomy (palliative treatment of severe cyanosis in newborns with transposition of the great arteries);

• balloon dilation of severe aortic valve stenosis or pulmonary valve stenosis;

• transcatheter closure of cardiac shunts (more often atrial septal defect and patent ductus arteriosus).

Sudden cardiac death in junior athletes.

An estimated 1–3 out of 100,000 apparently healthy young athletes develop sudden ventricular tachycardia or ventricular fibrillation and die suddenly while playing sports (m> d, more commonly soccer, basketball, soccer players).

Causes of sudden cardiovascular death among juniors

Hypertrophic cardiomyopathy
Commotio cordis (sudden ventricular tachycardia or fibrillation following a blow to the precordial region) is a hazard to athletes with thin, pliable chest walls, even in the absence of cardiovascular disorders. Cardiac dysfunction can occur when struck with moderate force (eg, a baseball, hockey puck, lacross ball) or colliding with another player during the vulnerable phase of myocardial repolarization.
Coronary artery anomalies (abnormal origin of the left coronary artery, right coronary artery, coronary artery hypoplasia)
Myocarditis
Aortic aneurysm rupture
Arrhythmogenic right ventricular dysplasia
Tunneling of the anterior interventricular branch of the left coronary artery
Aortic stenosis
Early coronary atherosclerosis
Dilated cardiomyopathy
Myxomatous degeneration of the mitral valve
Long QT syndrome

Hypertrophic cardiomyopathy
Brugada syndrome
Wolff-Parkinson-White syndrome (antegrade conduction only)
Catecholaminergic polymorphic tachycardia
Tachycardia from the outflow tract of the right ventricle
Coronary vasospasm
Sarcoidosis of the heart
Trauma to the heart
Rupture of a cerebral aneurysm

Other conditions: bronchial asthma, heatstroke, complications associated with the use of illegal or performance-enhancing drugs, ventricular tachycardia or fibrillation.

Risk factors include:

- ✓ chest pain or discomfort;
- ✓ fast or irregular heartbeat;
- ✓ fainting or light-headedness;
- ✓ fatigue and shortness of breath, especially if these symptoms occur during intense physical exertion;
- ✓ family members have cases of fainting and death during physical activity or cases of sudden death before the age of 50;
- ✓ drug use.

Symptoms are similar to those of cardiovascular collapse, the diagnosis is obvious.

Emergency treatment with maintenance of vital functions is successful in less than 20% of cases. This figure may increase as the proliferation of commonly available automated external defibrillators expaHRD.

Surviving athletes are monitored, treated or corrected for symptoms of the underlying disease. In some cases, mandatory implantation of a cardioverter-defibrillator may be required.

Screening of the state of the cardiovascular system to determine the possibility of playing sports: screening before starting sports, which is repeated every 2 years (for athletes of school age) or every 4 years (for students and older athletes), in Ukraine - every 6 months.
Screening recommendations in the US for college-age young adults, as well as for children and adolescents, include the following:

- • Collection of medical, family and drug history (including the use of doping and drugs that predispose to the manifestation of long QT syndrome)
- • Objective examination (including measurement of blood pressure and auscultation of the heart in the supine and standing positions)

• Selected studies, depending on the circumstances elucidated by history or physical examination *

** History and examination are neither sensitive nor specific; false-negative and false-positive results are common because the prevalence of cardiac abnormalities in the otherwise healthy population is very low. The use of a screening ECG or echocardiography would improve the detection of diseases, but would lead to an additional increase in the number of false positive diagnoses, and in addition, at the population level, their implementation is impractical.*

• European guidelines recommend a screening electrocardiogram (ECG) for all children, adolescents and college-age athletes.
• Genetic testing to diagnose hypertrophic cardiomyopathy or long QT syndrome is not recommended.
• Athletes with a family history of hypertrophic cardiomyopathy, long QT syndrome, or Marfan syndrome, or with symptoms of these conditions, should be considered for additional testing, usually including ECG and / or echocardiography. Confirmation of any of these conditions prohibits sports.
• Athletes with pre-syncope and syncope may be evaluated for coronary artery abnormalities (coronary angiography).
• If AV block of II degree of Mobitz type 1, complete transverse heart block, true right bundle branch block or left bundle branch block, or clinical or ECG signs of supraventricular or ventricular arrhythmias are observed, it is necessary to find out the presence of heart disease.
• If echocardiography shows enlargement of the aorta, further testing is necessary.

Recommendations

• Athletes should be warned against the use of prohibited medications.
• Patients with mild to moderate valvular heart disease can engage in vigorous physical activity, but patients with severe valvular heart disease, especially those with stenotic form, should not participate in competitive sports or high-intensity active sports.
• Patients with most structural or arrhythmogenic heart diseases (eg, hypertrophic cardiomyopathy, coronary artery abnormalities, arrhythmogenic right ventricular dysplasia) should not participate in competitive sports or high-intensity recreational sports.

6. Materials for methodological support of the lesson.

6.1. Tasks for self-examination of the initial level of knowledge and skills (tests, tasks)

Tests

Child L, 12 years old, who is registered with a cardio-rheumatologist for an acquired heart defect, has stenosis of the left venous orifice; the disappearance of the presystolic murmur was recorded on the phonocardiogram. A typical rhythm disturbance in this case is?
A. sinus tachycardia
B. sinus bradycardia
C paroxysmal tachycardia
D. atrial fibrillation
E. Wolff-Parkinson-White syndrome

2. Child I., 10 years old, makes no complaints. During the preventive examination, the ECG revealed an increase in heart rate with equal intervals between them, a shortened normal heart complex, a decrease in the distance between the T - P waves. The PQ interval is also shortened. What type of cardiac dysfunction does this child have?
A. Sinus extrasystole
B. Sinus bradycardia
C. Sinus tachycardia

D. Paroxysmal tachycardia
E. Intra-atrial block

3. In a patient with acquired heart disease, mitral valve stenosis was detected, an extended bacticular tooth P was found on the ECG. How to interpret this symptom?
A. Atrioventricular extrasystoles
B. Intra-atrial block
C Atrioventricular block
D. Blockade of the left bundle branch
E. Wolff-Parkinson-White syndrome

4. On admission to the hospital, child I., 10 years old, complains of discomfort in the chest. Dizziness, pallor of the skin, there are increased pulsation and swelling of the jugular veins in the neck. It is impossible to determine the pulse on the radial artery. Heart souHRD are loud and heart rate is uncountable. On the electrocardiogram, a heart rate of more than 200 per minute is determined, the QRS complex is preceded by a P wave, a slightly lengthened P - R interval. How to interpret this patient's condition?
A. Paroxysmal supraventricular tachycardia
B. Sinus tachycardia
C Atrial flutter
D. Ventricular paroxysmal tachycardia
E. Sinus bradycardia

5. After ineffective use of sedatives and psychotherapeutic methods, child L., 14 years old with paroxysmal tachycardia, was prescribed isoptin (verapamil). What is the dose of this drug per kg of body weight.
A. 0.1 mg
B. 0.2 mg
C. 0.3 mg
D. 0.4 mg
E. 0.5 mg

6. On the basis of the following ECG signs, an extended QRS complex, a heart rate of 170 per minute, a stable P - R interval, deviations of the S - T interval, a sudden onset and end of tachycardia were identified. The child is 10 years old. There are complaints of shortness of breath, pain in the heart, a feeling of heaviness in the chest. You can approximately diagnose:
A. Supraventricular paroxysmal tachycardia
B. Ventricular paroxysmal tachycardia
C. Sinus tachycardia
E. Atrial flutter
E. Sinus bradycardia
7. Anya, 15 years old, suddenly had an attack of rapid heartbeat with dizziness and severe anxiety. Heart rate is 220 beats / min. The ECG result shows that in all leads, the P wave is located in front of the ventricular complex, the ST wave is below the isoline, and the T wave is isoelectric. What rhythm disturbance is there?
A. Ventricular paroxysmal tachycardia
B. Paroxysmal atrial tachycardia.
C. Sinoauricular block
D. atrioventricular block
E. Sinus arrhythmia

8. A 13-year-old child has clinical ECG signs of paroxysmal tachycardia. What ECG changes are characteristic for this rhythm disturbance?
A. Extension of the P wave
B. Decrease in voltage of ECG waves
C. Deformation of the QRS complex
D. Increased heart rate up to 200 beats / min, P wave - in front of the QRS complex, ST and T deviation
E. None of the above

9. A 14-year-old girl has an attack of supraventricular paroxysmal tachycardia. Reflex influences were ineffective. What is the further algorithm of action?
A. Isoptin under ECG control
B. Novocainamide
C. Strofantin
D. Obzidan
E. Sequential staged prescribing of the above drugs.

10. In a 13-year-old girl, a planned ECG examination revealed a rhythm disturbance in the form of extrasystole. What are the main causes of heterotrophic rhythm disturbances?
A. Organic diseases (inflammatory, degenerative and others)
B. Toxic factors.
C. Vegetative dysfunction
D. Hypoxia
E. All of the above

11. Sergey is 11 years old. The child complains of dizziness, interruptions of cardiac activity in the evening. The ECG revealed signs of atrial extrasystole. What are the most characteristic changes that will be registered?
A. Premature occurrence of the P wave and QRS complex
B. Change in the morphology of the P wave
C. Incomplete compensatory pause
E. None of the above
E. All of the above

12. An 11-year-old Vasya had signs of blockade of the anterior branch of the left bundle branch of the His on a preventive ECG examination. What kind of cardiac pathology is this type of blockade observed?
A. Congenital heart defects
V. Cardite
C. Pulmonary emphysema
E. Pathology of the coronary vessels
E. All of the above

13. Galya, 14 years old, was admitted to the cardiology department with a diagnosis of cardiac arrhythmias. There is atrial premature beats. What groups of antiarrhythmic drugs will be included in the treatment plan?

A. Sodium channel blockers

B. Adrenergic blockers

C. Drugs that slow down repolarization

D. All of the above

E. Picamilon

Tasks.

<table>
<tr><td colspan="2">1. Maxim is 15 years old. During the examination on the ECG, sinus bradycardia is noted. The boy complains of rare dizziness, sweating, motion sickness in transport. Heart rate is 52 / min. AD is 90/60 mm. rt. Art.</td></tr>
<tr><td>Exercise
1. What ECG changes are characteristic of sinus bradycardia?
2. What are the main reasons for ECG changes in the patient?
3. Are these changes related to rhythm disturbances?
4. What additional examination methods should be recommended to the boy?
5. Make a program of treatment and rehabilitation measures.</td><td>Sample answer.
1. Sinus rhythm, 15% or more less often than the age norm. The PQ interval is within normal limits, the ventricular complex is not changed. RR intervals are greater than 1.0 secoHRD. The ST segment is 1 mm above the isoline and turns into a high-amplitude T wave. The P wave is flattened.
2. Autonomic dysfunction of the vagotonic type.
3. Violation of the automatism of the sinus node (nomotopic arrhythmias).
4. Cardiorhythmic intervalography, echocardiography, echoencephalography, electroencephalography, transcranial rheodoplerography.
5. Adaptogens, vitamin therapy, electrophoresis on the collar zone with calcium chloride, caffeine, mezaton, electrosleep.</td></tr>
<tr><td colspan="2">2. Roman, 14 years old, was hospitalized with complaints of pain in the region of the heart, sudden palpitations, discomfort in the chest, dizziness. Objectively: the skin is pale, covered with cold sweat. On the electrocardiogram - the heart rate is more than 200 per minute; the QRS complex is preceded by the P wave, a slightly lengthened P - R interval.</td></tr>
<tr><td>Exercise
1. How can this condition be interpreted?
2. What is the main mechanism for the development of this rhythm disturbance?
3. What other forms of this rhythm disturbance do you know?
4. Check additional diagnostic methods?
5. Algorithm of emergency therapy.</td><td>Sample answer.
1. Atrial paroxysmal tachycardia.
2. The causes of paroxysmal tachycardia can be autonomic dysfunction with a predominance of its sympathetic division, damage, myocardial inflammation, potassium deficiency, Wolff-Parkinson-White syndrome. Paroxysmal tachycardia is a continuous extrasystole from one focus of the cardiac conduction system. Another theory explaining paroxysmal tachycardia is the theory of re-entry, the circular circulation of the excitation wave from the atria to the atrioventricular node and back.
3. Atrial, atrioventricular, ventricular paroxysmal tachycardia.
4. Holter monitoring, echocardiography.
5. Algorithm of emergency care:
• Reflex effect (Valsalva test)
• Intravenous jet injection of calcium channel blockers (veropamil), novocainamide, cordaron, obzidana, panangina. If the rhythm cannot be restored, electro-pulse therapy is used.</td></tr>
<tr><td colspan="2">3. Dasha, 12 years old, complains of dizziness, discomfort in the region of the heart, interruptions in cardiac activity. On the ECG, signs of atrial extrasystole are recorded.</td></tr>
<tr><td>Exercise
1. What changes will be noted on the ECG?</td><td>Sample answer.
1. Atrial premature beats are characterized by:
• Premature atrial complex.
• The presence of a P wave in front of the QRS complex.</td></tr>
</table>

2. What are the main causes of extrasystole? 3. Classification of extrasystoles depending on the location of the heterotopic focus. 4. The main methods of examination of children with extrasystole. 5. What are the main drugs for the treatment of extrasystoles.	• The morphology and / or polarity of the P wave of the extrasystole is different from the sinus one. It can be reduced (upper atrial premature beats), flattened (mid-atrial), or negative (lower atrial). • PR of the atrial EC can be shortened, lengthened and normal. • The shape of the gastric QRS complex, as a rule, is not changed. • The compensatory pause is incomplete, but there may be a full compensatory pause with aberrant QRS. 2. Extrasystole is a multifactorial pathology: • Hereditary characteristics of the cardiac conduction system. • Violations of autonomic regulation. • Often EC appears in people with minimal cerebral dysfunction, with pathology of the spine and segmental disorders of the cervical spinal cord. • Among the organic causes, it is worth paying attention to cardiomyopathies, myocarditis. 3. Supraventricular and ventricular. 4. Algorithm of examination • Evaluation of clinical, anamnestic and genealogical data. • Electrocardiographic examination, including ECG of parents and siblings. • Neurophysiological examination and assessment of neurotrophic function. • Holter monitoring with assessment of rhythm variability. • Echocardiographic examination. • Virological examination. • Registration of late potentials, indicative of electrical instability of the myocardium. • 5. Antiarrhythmic drugs: calcium channel blockers, adrenergic blockers, potassium preparations, vegetative-correcting drugs.

6.2. The information necessary for the formation of knowledge and skills can be found in the textbooks:

- *basic:*

1. Volosovets O.P, Snisar V.I. Recommendations for cardiopulmonary resuscitation in children. Methodical manual. Dnepropetrovsk: ART-PRESS, 2015. 48 p.
3. Differential diagnosis of the most common diseases of childhood. Textbook / ed. V.M. Dudnyk, 1st edition. Vinnytsia: Nilan Ltd., 2017. 560 p.
4. Karen J. Markdante, Robert M. Kligman. Fundamentals of pediatrics according to Nelson: translation of the 8th English. edition: in 2 volumes. Volume 1. Kyiv: VSV "Medicine", 2019. XIV, 378 p.
5. Karen J. Markdante, Robert M. Kligman. Fundamentals of pediatrics according to Nelson: translation of the 8th English. edition: in 2 volumes. Volume 2. Kyiv: VSV "Medicine", 2019. XIV, 426 p.
7. Emergencies in pediatric practice: Textbook. way. for students. physicians. institutions of higher education, interns. - 2nd type. Recommended by the Ministry of Education and Science,

Recommended by the Academic Council of NMU. O.O. Bogomolets / Marushko Y.V, Chef G.G etc. Kyiv: VSV "Medicine", 2020. 440 p.
8. Pediatrics: a national textbook: in 2 volumes / Ed. prof. Berezhnogo V.V Kyiv, 2013. Vol.1. Kyiv, 2013. 1040 p.
9. Pediatrics: a national textbook: in 2 volumes / Ed. prof. Berezhnogo V.V Kyiv, 2013. Vol.2. Kyiv, 2013. 1024 p.
10. Pediatrics: textbook: in 2 volumes / M.L Aryaev, N.V Kotova, N.Y Gornostaeva (etc.), edited by: M.L Aryaeva, N.V Kotova, Odessa National Medical University. - Odessa, 2014. - 311 pp., Ill., Tab.

- *additional*

1. Radmilovic J, D'Andrea A, D'Amato A, et al. Echocardiography in Athletes in Primary Prevention of Sudden Death. J Cardiovasc Echogr. 2019;29(4):139-148. doi:10.4103/jcecho.jcecho_26_19
2. Erickson CC. Discrimination of the "Athlete's Heart" from real disease by electrocardiogram and echocardiogram. Cardiol Young. 2017;27(S1):S80-S88. doi:10.1017/S1047951116002286
3. https://www.heartaHRDtroke.ca/-/media/pdf-files/canada/cpr-2017/ecc-highlights-of-2015-guidelines-update-for-cpr-ecclr.ashx; https://eccguidelines.heart.org/wp-content/uploads/2015/10/2015-AHA-Guidelines-Highlights-Russian.pdf
4. de Caen AR, Maconochie IK, Aickin R, Atkins DL, Biarent D, Guerguerian AM, Kleinman ME, Kloeck DA, Meaney PA, Nadkarni VM, Ng KC, Nuthall G, Reis AG, Shimizu N, Tibballs J, Veliz Pintos R; on behalf of the Pediatric Basic Life Support and Pediatric Advanced Life Support Chapter Collaborators. Part 6: pediatric basic life support and pediatric advanced life support: 2015 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science With Treatment Recommendations. Circulation.
5. Berg MD, Schexnayder SM, Chameides L, Terry M, Donoghue A, Hickey RW, Berg RA, Sutton RM, Hazinski MF. Part 13: pediatric basic life support: 2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. Circulation. 2010;122(suppl 3):S862–S875. doi: 10.1161/ CIRCULATIONAHA.110.971085. 2015;132 (suppl 1):S177–S203. doi: 10.1161/CIR.0000000000000275.
6. Maconochie IK, de Caen AR, Aickin R, Atkins DL, Biarent D, Guerguerian AM, Kleinman ME, Kloeck DA, Meaney PA, Nadkarni VM, Ng KC, Nuthall G, Reis AG, Shimizu N, Tibballs J, Veliz Pintos R; on behalf of the Pediatric Basic Life Support and Pediatric Advanced Life Support Chapter Collaborators. Part 6: pediatric basic life support and pediatric advanced life support: 2015 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science With Treatment Recommendations. Resuscitation. 2015.
7. Recommendations for the implementation of resuscitation measures of the European Resuscitation Council (revision 2015). Ed. Corresponding member RAS VV Moroz 3rd edition, revised and enlarged. - M .: NIIOR, NSR, 2016 .-- 192 p. https://cprguidelines.eu/sites/573c777f5e61585a053d7ba5/content_entry573c77e35e61585a053d7baf/589d9b914c848614cf360a9e/files/Russian_translation_guidelines.pdf

6.3. Materials for methodological support of self-training of students: an orientation map for the organization of independent work of students with educational literature.

№№	Main goals	Directions	Answers
1	2	3	4
1.	To study the concepts: takhi -, bradycardia, extrasystole.	To give a definition of the HRD.	To point out what lies at the heart of the HRD.
2.	Etiology.	To point out the causes of the disease.	To note that physical and emotional activity,

			medication, etc., are essential.
3.	Pathogenesis.	Highlight the main links of pathogenesis.	
4.	Clinic.	To provide a description of the clinical manifestations in HRD.	
5.	Differential diagnosis.	To provide a description of the most frequent disease requiring differential diagnosis.	Determine the clinical manifestations of various HRD.
6.	Treatment.	To know the principles of treatment for various types of HDS.	
7.	Prevention.	Indicate the basic principles of prevention.	

7. Materials for self-control of the quality of training (questions):

1. What factors contribute to the development of tachy -, bradycardia and extrasystoles?
2. Give the clinical characteristics of tachycardia.
3. Give the clinical characteristics of bradycardia.
4. Treatment of extrasystole.
5. How is the prevention of HRD in children carried out?

8. Materials for classroom self-study

8.1. The list of educational practical tasks that must be completed during the practical lesson:

1. Work at the patient's bedside.
2. Make a clinical diagnosis.
3. Prescribe treatment.
4. Outline preventive measures.

9. Guidance materials for mastering professional skills

9.1. Methodology for performing work, stages of implementation.

1. Collect complaints, anamnesis of life and illness.
2. To assess the data of anamnesis, life and illness, risk factors contributing to the development of the disease.
3. Conduct a clinical examination of CVS in patients with HSR.
4. Make a plan for additional survey methods.
5. Evaluate the results of laboratory and instrumental data.
6. Formulate a clinical diagnosis.
7. Provide emergency care when needed and determine the following therapeutic measures.

10. Materials for self-control of mastering knowledge, abilities, skills provided for by this work.

10.1. Tests of different levels (see above).