RHYTHMAL AND CONDUCTIVITY DISORDERS IN CHILDREN

Recommendations for V–VI students Higher medical education institutions of the III–IV accreditation levels studying in English

МІНІСТЕРСТВО ОХОРОНИ ЗДОРОВ'Я УКРАЇНИ
Харківський національний медичний університет

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ПОРУШЕННЯ РИТМУ ТА ПРОВІДНОСТІ СЕРЦЯ У ДІТЕЙ

Методичні вказівки для студентів V–VI курсів вищих медичних закладів освіти III–IV рівнів акредитації, що навчаються англійською мовою

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Criteria for the diagnosis of tachycardia in children of different ages

Tachyarrhythmia is the most common and clinically significant cardiac arrhythmia in children. According to modern ideas, all tachyarrhythmias are divided into sinus, supraventricular and ventricular; paroxysmal and non-paroxysmal; reciprocal and automatic (ectopic); congenital and acquired; primary (idiopathic) and secondary, observed in pathology of the cardiovascular or other system.

School ID tachycardia realize an increase in heart rate (HR) above the age norm for 10–60 %. In children, heart rate depends on age (*Table 1*). There are sinus tachycardia:

1) moderate (I degree) - an increase in heart rate by 10–20 % above the age norm;

2) average (II degree) – by 20–40 %;

3) expressed (III degree) – by 40–60 %.

Table 1

The value of heart rate in a age groups from 0 to 18 years according to "ECG screening children and adolescents"; percentile distribution (Shkolnikova MA, Egorova DF, 2012)

Age	N	Percentile				
		2	5	50	95	98
0–7 days	262	100	110	139	170	180
1 week-2 months	378	115	121	153	186	200
3–5 months	237	106	114	136	171	183
6-12 months	324	99	105	127	170	185
1–2 years	207	80	92	123	188	195
2–3 years	135	74	88	112	150	171
3–4 years	200	76	83	101	128	149
4–5 years	207	76	80	98	118	125
5–6 years	250	71	74	92	115	127
6–7 years	252	70	71	92	115	120
7–8 years	433	65	70	88	114	125
8–9 years	337	62	66	87	114	125
9–10 years	267	60	65	83	109	115
10–11 years	309	60	63	81	108	117
11–12 years	278	57	60	81	111	120
12–13 years	215	53	60	81	106	115
13–14 years	263	56	61	80	107	111
14–15 years	362	56	61	80	109	115
15–16 years	241	54	60	78	103	110
16–18 years	220	49	56	72	107	114

The lower limit of normal heart rate (2 percentile) in the first year of life ranges from 100–115 per minute. Then there is a decrease in heart rate to 16 years. The value of the upper limit of heart rate (98th percentile) is maximum in the first year of life. In the future, heart rate decreases by 22–24 beats per minute

annually for up to 4 years. Subsequently, the heart rate is 120–125 beats per minute for up to 9 years. Then, by the age of 18, the value of the 98th percentile is reduced by 10 beats per minute. Tachyar itrmia – excess heart rate above 98 percentile. Tachyarrhythmias are divided into supraventricular (SVT) and ventricular (PT).

Heart rate (HR) is the number of electrical excitations of the myocardium that leads to further contraction of the heart muscle in 1 minute. The value of heart rate in a ikovyh groups from 0 to 18 are presented in *Table 1*.

Etiology.

Tachyarrhythmias can develop in organic heart disease (congenital heart disease, carditis, cardiomyopathy) and in the absence of such (CNS lesions, systemic connective tissue diseases, metabolic diseases, endocrine pathology, high-achievement sports, psychosomatic personality traits).

95 % of SVT is found in children with an anatomically normal heart. CT in 90 % of cases are found in organic heart disease.

Supraventricular tachyarrhythmia.

Headings for ICD-10

147.1 – Paroxysmal supraventricular tachycardia, AV-nodal tachycardia, ectopic (focal) atrial tachycardia;

145.6 – Wolff-Park and Nson-White syndrome;

148 – Atrial fibrillation.

SVT – three or more consecutive heartbeats rate with a frequency greater than the upper limit of normal. SVT includes tachycardias that occur on the sinus node, atrial myocardium, AV node, trunk of the His bundle, which come from the mouth of the vena cava and pulmonary veins.

Clinical and electrophysiological classification of supraventricular tachycardias:

I. Clinical variants of SVT:
  1. Paroxysmal tachycardia:
    – resistant (attack duration 30 s and more)
    – unstable (attack duration less than 30 s)
  2. X ronichna tachycardia:
    – constant
    – constant-reversive

II. Clinical forms and SVT:
  1. Sinus tachycardia:
    – sinus tachycardia (functional)
    – chronic sinus tachycardia
    – synoatryalna reciprocal and tachycardias I

2. Atrial tachycardia :
– focal atrial tachycardia
– multifocal or chaotic atrial tachycardia
– incisional atrial tachycardia
– atrial fibrillation
– atrial flutter
3. Tachycardia with AV connection :
– atrioventricular nodular reciprocal tachycardia
• typical
• atypical
– focal tachycardia with AV connection
• postoperative
• congenital
• "adult" form
4. Tachycardia involving additional conduction pathways (Wolf-Parkinson-White syndrome, atriofascicular tract and other STDs):
– paroxysmal orthodromic AV- reciprocal tachycardia with DPS
– chronic orthodromic AV- reciprocal tachycardia with slow DPS
– paroxysmal antidromic AV - reciprocal tachycardia with DPS
– paroxysmal AV- reciprocal tachycardia with pre-excitation

Mechanisms of supraventricular tachycardia.

Causes of SVT may be impaired pulse formation (increased automatism; residual or latent pacemakers, such as sinus tachycardia; pathological automatism of ectopic foci in the atrial myocardium – focal atrial tachycardia or tachycardia with AV connection; trigger activity – additional occurrence, occurrence which are associated with the previous action potential) and impulse conduction (re-entry), reciprocal tachycardia, for example, in the presence of additional conduction pathways (DPS).

Clinical manifestations, diagnosis and examination plan.

Sinus (non-paroxysmal) tachycardia can be physiological or pathological. Physiological tachycardia occurs during psycho-emotional stress, the transition to orthostasis, increased ambient temperature, after a hearty meal. Extracardiac pathological sinus tachycardia occurs with fever, acidosis, hypoglycemia, thyrotoxicosis, hypoxemia, anemia, pheochromocytoma, neurotoxicosis, when taking (overdose) drugs (adrenaline, isadrine, euphyllin etc.). Cardiac pathological sinus tachycardia is a manifestation of heart failure, characterized by a constant increase in heart rate at rest. The pathophysiological mechanism of sinus tachycardia is based on two main mechanisms: increased automaticity of the sinus node (SN) and violation of the autonomic regulation of SN with increased sympathetic and decreased parasympathetic tone.

Paroxysmal SVT tachycardia is characterized by the sudden onset of a tachycardia attack and the same sudden end spontaneously or as a result of drug or non-drug (vagus) effects. Children in the first year of life during the attack may experience anxiety, lethargy, paleness, pulsation of the vessels of the neck. In school-age children, seizures are provoked by physical or emotional stress. Children complain of palpitations, weakness, dizziness, darkening of the eyes, etc.

In 10–15 % of cases of SVT there is a loss of consciousness. The reason may be a high heart rate in atrial fibrillation with AV conduction for DPS in the syndrome of WPW, congenital heart disease (aortic stenosis), KMP (often hypertrophic), cerebrovascular insufficiency.

Chronic sinus tachycardia is more often detected accidentally when performing an ECG with increased levels of cortical activation (excluding other reasons described above). Chronic forms of tachycardia are asymptomatic.

Methods of examination in the detection of SVT:

1. History taking (complaints of recurrent heartbeat, syncope and presyncopal states, recurrent episodes of sudden weakness, lethargy in infants and young children, detection of tachycardia during examination of the child, time of onset of the attack, than the presence of ECG in parents, sudden death relatives, surgical correction of the congenital heart disease, signs of heart failure in a child).
2. ECG at in 12 leads.
3. ECG during a tachycardia attack.
4. Daily and longer ECG monitoring.
5. Tests with dosed exercise.
6. Esophageal electrophysiological study.
7. Endocardial electrophysiological examination (according to the indications, necessarily at WPW).
8. Electroencephalography.
9. Neurosonography.
10. Psychological tests.
11. Ultrasound of the thyroid gland.
12. Stress tests of ECG diagnostics of supraventricular tachycardias (bicycle ergometry, treadmill test).
13. Blood pressure control.

To establish the type of SVT you need to know:

1. Width of QRS complexes: tachycardia with wide QRS complexes, with narrow QRS complexes (ECG tachycardia criteria are presented in *table 2*).
2. Regularity of heart rhythm during tachycardia: regular, irregular.
3. Heart rate ratio of teeth P'i QRS: AV conduction 1 : 1; 2 : 1, etc.; AV dissociation.
4. Morphology and location of the teeth P and the duration of the interval RP'.
5. The ratio of the intervals RP' and P'R, their stability, there are three groups of tachycardias: with RP' < P'R, with RP' > P'R and with a variable interval RP'.

6. Change in the length of the tachycardia cycle (RR intervals during tachycardia) in the event of transient complete blockade of the leg of the His bundle.

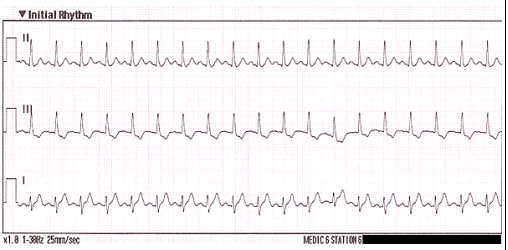
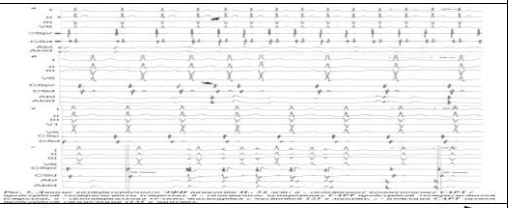
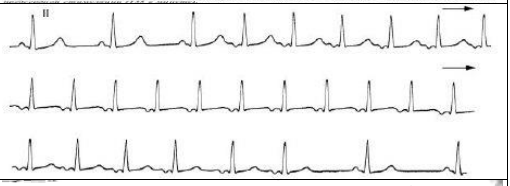
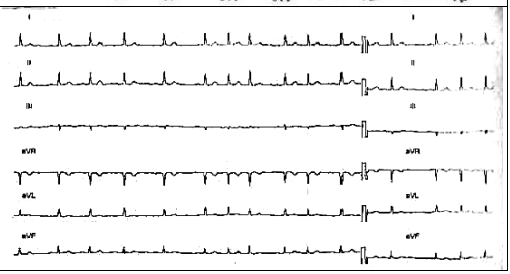
7. The moment of spontaneous onset of tachycardia or provocation of tachycardia during CSEFD.

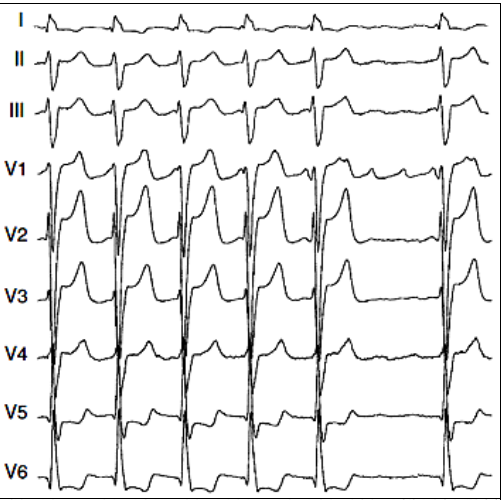
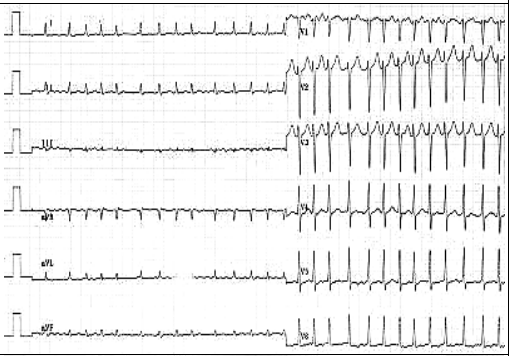
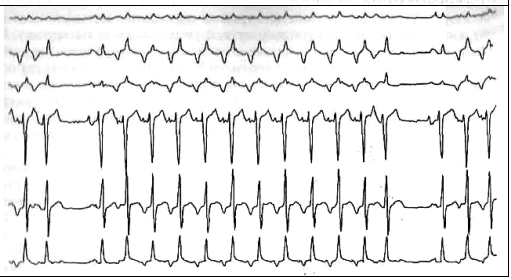
8. Methods of purchasing tachycardia (spontaneous, vagal techniques, atrial electrical stimulation, antiarrhythmic drugs, EIT).

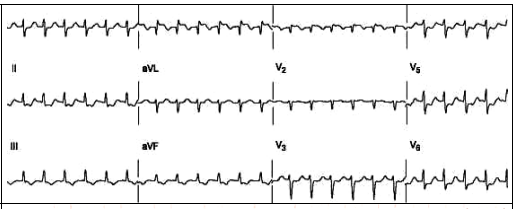
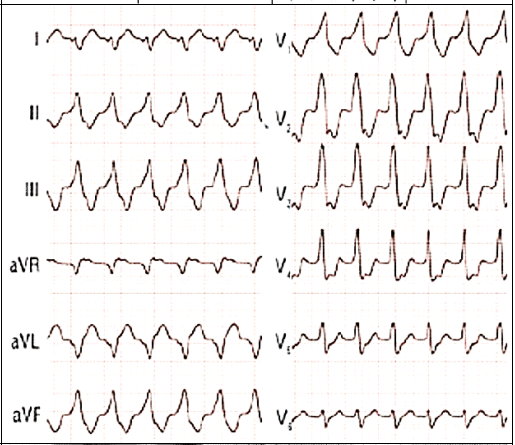
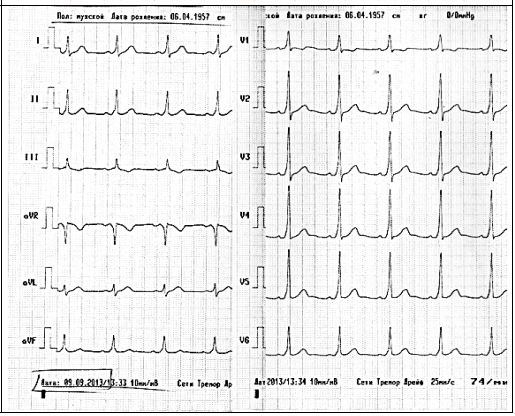
9. The nature of the tachycardia: paroxysmal or chronic.

10. Hemodynamic disorders and subjective tolerability of tachycardia.

Table 2

Sinus tachycardia 1. Acceleration of sinus rhythm (the presence of the P wave and its normal morphology before each QRS complex at the level of 95–98 ‰ age norm (*table 1)*. 2. QRS complexes are narrow. 3. Gradual onset and end of tachycardia.	
Chronical sinus tachycardia 1. Constant sinus tachycardia with accelerated pulse during exercise. 2. Possible normalization of heart rate during sleep. 3. The morphology of the P wave before each QRS complex is not disturbed.	
Sinus node re-entry tachycardia 1. Sudden onset and end of an attack in. 2. Normal morphology of the P wave before each QRS complex.	
Focal atrial tachycardia 1. Heart rate – 120–130 per minute. 2. Probe P is not of sinus origin (different morphology), registered before each QRS complex.	
Multifocal chaotic SVT 1. One lead recorded at least 3 g iznyh in morphology teeth R. 2. Irregular intervals PR, PP, RR.	

Atrial fibrillation 1. Proper regular atrial rhythm with a frequency of 250-450 hit in a minute. 2. Instead of teeth P are registered "sawtooth wave".	
Atrial flutter 1. Chaotic electrical activity of the atria with a frequency of 300–700 beats per minute. 2. Different amplitude and configurations wave f without isoline between them.	
Paroxysmal atrioventricular nodal reciprocal tachycardia (typical form) 1. Narrow QRS complexes. 2. Heart rate 150–250 hit in a minute. 3. Regular rhythm. 4. The tooth P is often not defined, or is represented by pseudo r' in lead V1 and / or pseudo S' in II, III and aVF leads. 5. The P wave can be defined in II, III, aVF negative (retrograde). 6. The RP 'interval is less than 100 ms.	

Paroxysmal atrioventricular nodal reciprocal tachycardia (atypical form) 1. The P wave is located between two QRS complexes. 2. Heart rate does not exceed 150 beats a minute. 3. Often non-paroxysmal chronic course.	
Syndrome Wolf-Parkinson-White (macro-re-entry) 1. Heart rate in infants 260–300 beats per minute, adolescents – 180–220 beats per minute. 2. Correlation RP' < P'R. 3. The retrograde tooth P' is located on the QRS complex on the ST segment or at the beginning of the T wave, and PR' > 70 ms. Sometimes the P wave is hidden in the T-wave. 4. QRS complexes are narrow, at aberrant carrying out (blockade of legs of a bunch of His) – wide.	
Syndrome Wolf-Parkinson-White (outside attack) 1. Reduction of the PR interval < 120 ms. 2. The presence of a delta wave in front of the QRS complex. 3. Expansion of the QRS > complex 100 ms. 4. Secondary ST-T changes.	

Wolf-Parkinson-White syndrome (ventricular preexcitation syndrome)

Ventricular preexcitation is associated with the presence of additional conduction pathways (ACP), which are represented by specific ECG phenomena (characteristic ECG pattern without tachycardia) and can cause the appearance of AV-reciprocal tachycardia (WPW syndrome).

ACP are short muscle bundles 1.3 mm in diameter that connect the myocardium of the atria and ventricles (muscle bridges).

At a syndrome of WPW the paroxysmal orthodromic AV reciprocal tachycardia (macro-re-entry) most often meets (90 %). ECG criteria presented in *table 2*. In a small number of patients with WPW syndrome occurs paroxysmal reciprocal and tachycardias I, in which the pulse passes through the additional AV connection (DAVZ) from the atria to the ventricles, and returns to the AV node.

ECG criteria for small tachycardia:

1. QRS wide, deformed.

2. Retrograde teeth P' are usually located in the second half of the tachycardia cycle, ie RP' > P'R and inverted in II, III and aVF leads.

Outside the attack of tachycardia ECG criteria are described in *table 2*.

An important method of diagnosing SVT and the phenomenon of ventricular excitability (phenomenon, WPW syndrome) is esophageal electrophysiological examination (TSEFD). TV Kruchina and DF Egorov in their monograph "Supraventricular tachycardia in children" (St. Petersburg, 2011, p. 258) in order to diagnose paroxysmal SVT offer the following indications for CSEFD:

Class I:

1. Children with heart attacks not recorded on the ECG, to obtain information about the presence of paroxysmal tachycardia and its possible mechanism.

2. Children with phenomena of ventricular excitability, engaged in sports, to assess the properties of additional conduction pathways, the risk of life-threatening arrhythmias.

Class II:

1. Children with registered tachycardia attacks without signs of ventricular excitability on the ECG, to clarify the mechanism of paroxysmal tachycardia.

2. Children with phenomena of ventricular excitability, not engaged in sports, to assess the properties of additional conduction pathways, the risk of life-threatening arrhythmias.

Class III:

1. Children with WPW syndrome, ie with signs of ventricular excitability and registered tachycardia on the ECG.

Treatment

Treatment of SVT is divided into the provision of emergency care (during the attack), pathogenetic and symptomatic therapy outside the attack.

Drug therapy includes the introduction of antiarrhythmic drugs, tranquilizers and diuretics. Non-drug – vagus tests, esophageal tachycardia.

Immediate therapy for the purchase of SVT should begin with oscillating methods of exposure: turning upside down in young children, Valsalva test – tension on exhalation with a closed nose, increasing the tone of the abdominal muscles, pressing on the root of the tongue, kidney reflex – immersion of the face in cold water, applying a cold warmer (water) to the lower third of the face (through a diaper).

Vagal tests are effective during the first 20–25 minutes of the attack, in the future it is necessary to use antiarrhythmic drugs in parallel with sedative drugs (phenibut – ½ daily adult dose, carbamazepine – 3 mg/kg once). In case of a prolonged attack, diuretics are used.

The algorithm for the treatment of paroxysmal tachycardia in a stable state of hemodynamics is given in *table 3*.

Table 3

Immediate therapy of paroxysmal SVT in a stable condition with a narrow QRS complex, as well as with a wide QRS complex as a result of functional blockade of the legs of the His bundle begins with intravenous adenosine – a drug that has pronounced antiarrhythmic properties. If the injection is ineffective, it can be repeated twice more with an interval of several minutes (at least 2 minutes). When administered intravenously, adenosine slows conduction through the AV node, interrupts the re-entry mechanism and helps to restore sinus rhythm. The antiarrhythmic effect of the drug is associated with a slowing of the automatism of pacemaker cells, slowing down the conduction in the AV node, reducing atrial contractility, inhibition of norepinephrine release. If adenosine is ineffective three times in a row, further administration of the drug is impractical and the acquisition of tachycardia paroxysm is continued with the introduction of verapamil (isoptin) – a class IV antiarrhythmic drug that acts on slow calcium channels, inhibits sinus node automatism and depolarization in the AV node. When administered intravenously, the drug is rapidly metabolized. The drug is contraindicated in children under 1 year of age. While maintaining tachycardia, intravenous cordarone is recommended, the properties of which are the prolongation of the duration of action potential and refractoriness of all myocardial tissue, including additional conduction pathways, due to blockade of potassium channels; inhibition of fast input sodium current; local antiadrenergic (both α- and β-blocking) action at the level of the myocardium, which does not extend to other organs and systems; blockade of calcium channels, resulting in a decrease in rhythm and slow conduction in the AV node; dilatation of peripheral and coronary vessels. The peak concentration of the drug in the serum is reached within 30 minutes. If necessary, the drug can be administered for several days (not more than 5 days).

Cordarone can also be administered in bolus at 5 mg/kg for 5–10 minutes, followed by a long infusion at a dose of 10 mg/kg/day.

In paroxysms of atrial fibrillation, ectopic and re-entry atrial tachycardia, orthodromic AV reciprocal tachycardia, the onset of seizures in older children (7–18 years) is possible with the introduction of procainamide (novocainamide), which belongs to class IA antiarrhythmic drugs. in the membranes of rhythm driver cells, reducing the rate of depolarization in phase 0 of the action potential;

reduces conduction in most myocardial tissues – in the atria, the conduction system of the heart distal to the AV node, in the ventricles (to a lesser extent affects the conduction in the AV node), inhibits antegrade and retrograde conduction by additional conduction pathways. If the tachycardia attack persists on the background of complex drug therapy and the presence of conditions, it is possible to conduct transesophageal pacing to purchase paroxysms. In children with hemodynamically unstable paroxysmal tachycardia, as well as with atrial fibrillation, the method of choice is synchronized cardioversion with an initial energy of 0.5 J/kg and a subsequent increase if necessary to 1 J/kg.

First antyary tmichn and Therapy I paroxysmal SVT with NEST abilnomu state (hemodynam ichno ineffective max and arrhythmias and I develop collapse, syncope), which is due to atrial fibrillation, including antehra dnym on the pulse DPSH and ventricular tachycardia requires urgent electropulse therapy on the background of constant oxygenation. With anti-arrhythmic drugs (including anticipated type of arrhythmia) class I used drugs procainamide (novokainamid), lidocaine (with polymorphic second shlunoch ing th tachycardia or ventricular fibrillation). Saving entry tachycardia shown Cordaron in, with a possible claim idvyschennyam dose of 5 to 15 mg/kg (in infants) and preparations of magnesium. With regard to hemodynamic instability, marked myocardial hypoxia, electrolyte imbalance central hemodynamic support shown entering adreno rhichnyh drugs polarize Choi mixture and antioxidants (meksydol, 2 mg/kg to tively).

Prolonged hemodynamically significant antidromic paroxysmal SVT, as well as attacks of atrial flutter with DPS conduction require immediate radiofrequency catheter destruction of abnormal additional atrioventricular junction.

Against recurrent tachyarrhythmias based therapy in the treatment of disease caused by arrhythmia (CHD, KMP, myocarditis) and normalization korkovo-during cortical relationships.

Rational (anti-relapse) therapy is carried out taking into account the correction of the main pathophysiological mechanisms of their development and includes the impact on the neurovegetative basis of arrhythmia and the specific electrophysiological mechanism of its development. The purpose of drug therapy for paroxysmal SVT – to prevent further paroxysms of tachycardia; with nonparoxysmal SVT – to restore sinus rhythm.

Rational therapy of paroxysmal SVT in the period between attacks differs depending on the frequency of attacks, their circadian (day, night, mixed), electrophysiological variant of arrhythmia. In the period between attacks for children with frequent paroxysms is characterized by a decrease in the impact of the sympatho-adrenal department on heart rate, which is expressed in lower than in healthy and children with rare attacks, values of day and night heart rate combined with low functional reserve of heart rate adaptation. Thus, the stabilization of rhythm in children with paroxysmal tachycardia should be influenced by increasing the reserves of sympatho-adrenal rhythm regulation in the period between attacks. It is shown that seizures mainly occur during

periods of the day with a steady decrease in the adaptive capacity of the heart rate. Basic neurometabolic therapy is carried out 2 times a year for 3 months. It affects the neurogenic basis of arrhythmia, contributing to the normalization of neurovegetative imbalance in the regulation of heart rhythm, responsible for the implementation of the abnormal electrophysiological mechanism of myocardial excitation and the development of paroxysms. Nootropic and nootropic drugs have a trophic effect on the autonomic centers of regulation, enhance metabolic activity of cells, mobilization of energy reserves of cells, regulation of cortical-subcortical relationships, have a mild and lasting stimulating effect on sympathetic regulation of the heart. Assigned one drug per month, the duration of the course 1–2 months, followed by replacement with another drug in this group:

• *Amynalon* 50 mg 2 times in a day (up to 3 years), ½ tab. 2 times in a day (up to 7 years), 1-tab. 2–3 times and on the day (over 7 years);

• *Glutamic acid* 50 mg – 2 times a day (up to 3 years), 125 mg – 2–3 times a day (up to 7 years), 250 mg-3 times a day (up to 12 years), 500 mg – 2 times a day (over 12 years);

• *Encephabol* 25 mg – 2 times a day (up to 3 years), 50 mg – 2 times a day (up to 7 years), 100 mg – 2–3 times a day (over 7 years);

• *Semak* 0.1% solution (drops in no s , in each nostril) 1 drop – 2 times a day (up to 3 years), 2 drops – 2 times a day (up to 7 years), 3 drops – 2 times a day (up to 10 years), 5 drops – 2 times a day (over 10 years);

• *Pantogam* 50 mg – 2 times a day (up to 3 years), 125 mg – 2 times a day (up to 7 years), 250 mg – 2 times a day (up to 10 years), 500 mg – 2 times a day (older than 10 years).

• *Cortexine* 10 mg, intravenously № 10 (up to 7 years), 20 mg, intravenously; № 10 (over 10 years).

In psycho-emotional disorders, the occurrence of paroxysms on the background of psycho-emotional stress, prescribe the tranquilizer phenibut , which has a sedative, anxiolytic effect and has elements of nootropic activity.

• *Fenibut* 50 mg – 2 times a day (up to 3 years), 125 mg – 2 times a day (up to 7 years), 250 mg – 2 times a day (up to 10 years), 250 mg – 3 times a day day (after 10 years).

• Picamilon 10 mg – 2 times a day (up to 3 years), 20 mg – 2 times a day (up to 7 years), 50 mg – 2 times a day (up to 10 years), 50 mg – 3 times a day (over 10 years).

At frequent paroxysmal SVT (monthly attacks) and impossibility to carry out interventional treatment of an arrhythmia (small age of the patient, localization of an electrophysiological substrate in close proximity to structures of conducting system of heart or epicardially) steady antiarrhythmic effect can be provided by anticonvulsant drug *carbamazepine* (finlepsin-10)/kg/day (up to 15 mg/kg/day in children under one year) in 2–3 doses lasted, which has antidepressant, membrane-stabilizing and antiarrhythmic effect due to inactivation

of the input sodium current.

In paroxysmal SVT children continued appointment of classic anti-arrhythmic drug has significant limitations and justified mainly for purchase of paroxysm; their use in the period between attacks is accompanied by an increase in basal rhythm depression and, in some cases, suppression of sympathoadrenal function, which, along with the antiarrhythmic effect, exacerbates neurovegetative rhythm regulation, which is an important pathophysiological mechanism of paroxysmal SVT. Prolonged use of antiarrhythmic drugs adversely affects the long-term prognosis of paroxysmal SVT. In cases where tachycardia is an avid nature and requires connection of classic anti- arrhythmic drugs of choice are radiofrequency catheter ablation (RCHA). In determining the indications should follow a "reasonable" conservatism in young children, which is associated with a high probability of spontaneous disappearance of arrhythmias before 18 months of age. However, in 30 % of them the arrhythmia subsequently recurred, which requires observation and decision-making on further treatment tactics. Despite the constant development and improvement of technologies, diagnostic and therapeutic catheter electrodes, children in early and preschool age have a higher risk of complications than in the older age group. In children older than 10 years, the indications for interventional treatments for tachyarrhythmias are comparable to those for adult patients. The efficiency of radiofrequency ablation of supraventricular tachycardias is according to various authors from 83 % to 96 % and depends on the type of arrhythmia, technical capabilities and clinical experience. In preschool children with frequent and/or hemodynamically unstable attacks of paroxysmal SVT on the background of drug-based therapy and ineffectiveness of finlepsin may be a course of antiarrhythmic drugs:

• *Amiodarone* per os at a dose of 5–7–10 mg/kg (in children under one year up to 15 mg/kg/day);

• *Propafenone* per os 10–15 mg/kg/day;

• *Sotalex* 1–2 mg/kg/day;

• *Verapamil* 2 mg/kg/day;

• *Allapamine* 1 mg/kg/day (in atrial fibrillation, atrial flutter, bradisystolyc forms).

Rational drug therapy of nonparoxysmal SVT aims to correct neurovegetative disorders that contribute to the functioning of abnormal electrophysiological mechanisms of myocardial excitation (basic therapy); direct effect on the electrophysiological substrate of arrhythmia (antiarrhythmic drugs). Basic therapy helps to restore the protective function of the sympathoadrenal system and has a trophic effect on the autonomic centers of regulation, restoring the balance of autonomic regulation of heart rhythm, shifted in children with nonparoxysmal SVT towards the relative predominance of parasympathetic influences. For this purpose, children with nonparoxysmal SVT are prescribed nootropic and vegetotropic drugs with a stimulating component of action *(aminalone, glutamic acid, encephabol, semax, cortexin, cerebrolysin).*

Neurometabolic stimulants have antiasthenic, sympathomimetic, autonomic, antidepressant and adaptogenic (improve tolerance to exogenous stressors) effects to varying degrees. In children with nonparoxysmal SVT, these drugs are prescribed alternately for 2–3 months. each (total duration of the first course 6 months). With a significant reduction in the severity of arrhythmia after the first course, a second course is prescribed for 3 months.

As an antiarrhythmic agent in children with non-paroxysmal SVT, the effective anticonvulsant drug *carbamazepine*, which has antidepressant, membrane stabilizing and antiarrhythmic effects due to inactivation of the incoming sodium current.

Children with non-paroxysmal SVT of constant type, SVT of repeated and high representation of heterotopic rhythm (more than 80 %) per day at detection of signs of arrhythmogenic cardiomyopathy and impossibility of carrying out interventional treatment of arrhythmia are appointed antiarrhythmic drugs: *amiodarone, propafenone*, co. In the presence of arrhythmogenic myocardial dysfunction (symptoms of heart failure), high frequency heterotopic rhythm is prescribed *digoxin* in a maintenance dose of 0.005 mg/kg/day (2 doses) or *capoten* at a dose of 0.5 mg/kg/day (3 doses).

When signs of diastolic myocardial dysfunction are detected according to echocardiography, disorders of the repolarization process according to ECG data, stress tests, metabolic therapy is performed. For this purpose, prescribe antihypoxants and antioxidants, vitamins and vitamin-like agents, macro-and micronutrients (consecutively 1 drug per month, lasting 1 month):

- *Levocarnitine* per os 50–100 mg/day,
- *Kudesan* per os 7–15 drops/day,
- *Actovegin* intravenously 20–40 mg for 5–10 days,
- *Preduktal* – 1 tab. – 2 times a day,
- *magnesium* preparations (Magnerot, Magne B_6),
- *Riboflavin mononucleotide*,
- *Cytochrome C,*
- *Mildronate* .

Indications for surgical treatment of supraventricular tachycardia in children.

In 2002, guidelines for radiofrequency ablation (RFA) in children were published by the North American Society for Pacemaking and Electrophysiology (NASPE, 2002) (*Table 5*).

Table 5

Recommendations for radiofrequency ablation in children (NASPE, 2002)

Class I

1. WPW syndrome , episode of RCC.

2. WPW syndrome, syncope, short RR interval during AF (RR < 25 0 ms) or anterograde ERP DAVZ < 250 ms.

3. Chronic ab recurrent SVT with ventricular dysfunction.

4. Recurrent ventricular tachycardia with hemodynamic disturbances with the possibility of eliminating tachycardia by RFA.

Class II

Class II (A)

1. Retsydyvyruyu s and/or symptomatic SVT, refractory to medical therapy, age > 4 years.

2. Future surgery for the BBC, which may further complicate access to blood vessels and chambers of the heart.

3. Chronic (lasting more than 6–12 months) SVT, normal PV.

4. Chronic or often repeat Wang vnutrishnoperedserdn a reciprocal and tachycardias me.

5. History of heartbeat with induction of stable SVT during electro-physiological study.

Class II (B)

1. WPW phenomenon , age > 5 years.

2. SVT, age > 5 years, the choice of RFA in possible chronic anti-arrhythmic therapy.

3. SVT, age < 5 years, ineffectiveness or side effects of antiarrhythmic therapy, including sotalol and amiadarone.

4. Intraatrial reciprocal tachycardia, rare episodes (1–3 per year) that require medical intervention to eliminate them.

5. Ablation of the AV node and implantation of EX as a possible method of treatment of recurrent or resistant to treatment of atrial reciprocal tachycardia.

6. A single episode of ventricular tachycardia with hemodynamic disorders with the possibility of eliminating tachycardia by RFA.

Class III

1. WPW phenomenon , age < 5 years.

2. SVT, age < effectiveness of antiarrhythmic therapy.

3. Unstable paroxysmal ventricular tachycardia without hemodynamic disorders.

4. Episodes of unstable SVT that do not require therapy and/or have minimal clinical manifestations.

Long-term antiarrhythmic and treatment is carried out only when it is impossible radical treatment, especially in young children. Indications for long-term use of antiarrhythmic drugs are:

1. The emergence of SVT in fetal in and neonatal period.

2. Children Early oho age with frequent trudnokupu is of ymi accompanied by hemodynamic disorders SVT attacks.

3. The emergence of SVT on the background of the current city iokardytu.

4. Inefficiency or impossibility of RCA.

5. Refusal of RCA in older children with frequent, hemodynamically

significant SVT.

Appointment of antiarrhythmic drugs is carried out by the method of selection, starting with drugs that have the lowest risk of complications after determining the type and mechanism of tachyarrhythmia in the hospital. When prescribing antiarrhythmic therapy, it is necessary to assess the initial interval of QT and QTc. In children younger than one year, QTs should not exceed 470 ms., In children older than one year – QTs should not exceed 460 ms.

Drugs that have the lowest risk of complications include *digoxin* and *β-blockers*. It should be remembered that digoxin is not prescribed to children with WPW syndrome and tachycardias with broad complexes (until their mechanism is established), as well as *verapamil* for WPW syndrome (shortens the effective refractory period of DAVZ).

Next we present a brief description of the main antiarrhythmic drugs (*Table 6*).

Table 6

Brief description of the main antiarrhythmic drugs used to treat SVT in children

Preparation	Dosage / frequency	Therapy. conc. in plasma	Side and arrhythmogenic effects
Class I			
Procainamid Novocainamid IA	40–100 mg/kg/day 4–6/d IV 7–15 mg/kg	4–10 mkg/ml	lupus-like syndrome, hypotension, QRS expanding by more than 50% of elongation, VT "pirouette"
Phlecainide (Tambocorum) IC	80–200 mg/m^2/day (1–8 mg/kg/day) 2–3 times	0,2–1 mkg/ml	Disorders of the gastrointestinal tract, impaired vision, VT
Propafenoni (Rhythmonorm) IC	150–200 mg/m^2/day (8–10 mg/kg/day) 3 times i/v 1–2 mg/kg	0,15–0,2 mkg/ml	Lupus-like syndrome, disorders of the gastrointestinal tract, central nervous system, visual impairment, ST
Class II			
Propranolol (Anapriline)	1–4 mg/kg/day – 4 times IV 0,01–0,15 mgr/kg	0,05–0,1 mkg/ml	Bronchospasm, hypotension, Raynaud's syndrome, CNS disorders, hypoglycemia, thrombocytopenia, bradycardia, AV blockade, HF
Nadolol	1–2 mg/kg/day		Hypotension, hypoglycemia, bradycardia, AV blockade, HF
Atenolol	0,5–2 mg/kg/ day – 2 times		Hypotension, hypoglycemia, bronchospasm, bradycardia, AV blockade, HF
Metoprolol	1–2 mg/kg/day – 3 times		Bronchospasm, hypotension, CNS disorders, hypoglycemia, vomiting, bradycardia, AV blockade, HF
Class III			
Amiodarone	10–15 mg/kg/day 1–2 times to 2 weeks, next 2,5–5 mg/kg/day i/v 5 mg/kg	1–2,5 mkg/ml	Thyroid dysfunction, microdeposition in the cornea, photosensitivity of the skin, chemical hepatitis, severe bradycardia, AV blockade, ST "pirouette"

Preparation	Dosage / frequency	Therapy. conc. in plasma	Side and arrhythmogenic effects
Sotalol	90–200 mg/m²/day (2–6 mg/kg/day) 2 times	0,2–1,05 mkg/ml	Hypotension, headache, bradycardia, AV blockade, ST "pirouette"
Класс IV			
Verapamil	4–17 mg/kg/day – 3 times i/v 0,1 mg/kg	0,05-0,2 mkg/ml	Hypotension, dizziness, AV blockade, negative inotropic effect

The effectiveness of antiarrhythmic and basic therapy is assessed by the clinical course of the paroxysmal period (decreased heart rate, no hemodynamic disturbances, rapid purchase of vibrational tests) and Holter ECG monitoring (change of paroxysms from night to daytime, reduction and disappearance of attacks). The effectiveness of standard treatment (drugs normalize neurovegetative and vascular interactions, bracing means) is estimated not earlier than 3–6 months in.

Risk of sudden cardiac death in children with supraventricular tachycardia.

The risk of sudden cardiac death in SVT is small, compared with ventricular tachycardia. However, a number of clinical situations that are considered life-threatening need to be considered:

1. WPW syndrome.
2. Chronic SVT with the development of arrhythmogenic cardiomyopathy.
3. SVT with hemodynamic disorders.
4. SVT with si nkope.
5. Combination of SVT with BBC and organic myocardial diseases.
6. SVT in athletes.
7. Arrhythmogenicity (proarrhythmic) action of antiarrhythmic drugs in the treatment of SVT.

In WPW syndrome, the risk criteria for sudden death syndrome are:

1. Clinical death in the anamnesis.
2. Spontaneous/induced steady-state AF with conduction pulses on AAVC with RR < 250 ms and/or RR min < 220 ms.
3. Combination of WPW phenomenon with CHD, myocarditis, hypertrophic cardiomyopathy.
4. High level of conductivity on AAVC (240 imp/min and more) and/or short ERP of AAVC (250 ms and less) at athletes with the WPW phenomenon.
5. Multiple AAVC.

Follow up

Patients receiving long-term therapy with antiarrhythmic drugs should register an ECG once every 3 months, Holter ECG monitoring is performed once every 6 months. Antriarrhythmic drugs are canceled at emergence of intraventricular and atrioventricular blockades. The appointment of a further antiarrhythmic drug is possible only 24 hours after the cancellation of the previous one and assessment of the heart rate profile during the day. At long appointment of amiodarone once in 6 months it is necessary to investigate

ultrasound and function of a thyroid gland. At the same time it is possible to prescribe no more than 2–3 drugs of basic therapy (correction of neurovegetative dysfunction, nootropic and vegetotropic drugs), changing them alternately courses up to 6 months. When the effect is achieved, a repeat course is prescribed after 3 months.

Antiarrhythmic drugs prescribed to children with permanent non-paroxysmal SVT type SVT oscillating type of heterotopic 80 % pace during the day, signs of cardiomyopathy arrythmogenic and inability to intervention treatment of arrhythmias. In the presence of arrhythmogenic dysfunction and signs of heart failure, digoxin is prescribed in a maintenance dose of 0.005 mg/kg/day in 2 doses or capoten at a dose of 0.5 mg/kg/day in 3 doses. At detection of diastolic dysfunction of disturbances of processes of repolarization on an ECG antiarrhythmic and antioxidant drugs, vitamins and microelements consistently on one drug a month for a period of 1 month are appointed.

At paroxysmal SVT at children long appointment of classical antiarrhythmic drugs is extremely limited and is applied only to acquisition of a paroxysm. The prognosis of paroxysmal tachycardia depends on the degree of neurovegetative dysfunction. Prolonged (more than a year) antiarrhythmic drugs (especially class I drugs and amiodarone) are prognostically unfavorable, because they block the arrhythmogenic substrate and changes in the frequency of the basal rhythm (decrease) further facilitate the onset of the attack, and the arrhythmia becomes continuously relapsed.

Basic therapy of SVT

Digoxin

Digoxin has for many years been considered a universal drug for SVT in children, especially in the first years of life, with an efficiency of 42 % to 61 %. Digoxin inhibits Na^+-K^+-ATPase, ie disrupts the operation of the Na^+ pump. As a result, the intracellular content of Na^+ ions increases and the intake of Ca^{2+} ions into the cell increases. This provides a positive ionic effect, but reduces the intracellular content of K^+, which can cause an arrhythmogenic effect. The antiarrhythmic effect of d and hoxin is associated with a slowing of heart rate and a decrease in the rate of conduction of excitation in the myocardium and AV node.

Currently, digoxin continues to be used in newborns and infants with various types of SVT. Appointment of digoxin children older than 1 year of chronic SVT possible to control the frequency of arrhythmias, but as a reserve drug.

It is necessary to dwell on the limitations of digoxin in WPW syndrome. Previously, it was often used in children of the first year of life with WPW syndrome. The electrophysiological effects of digoxin are manifested in the slowing of the conduction of the pulse in the AV node and the prolongation of its refractory period. However, the use of digoxin in the treatment of

tachycardia in children with WPW syndrome is questionable and of great concern. Digoxin reduces the anterograde effective refractory period of the additional conductive pathway, which in children with WPW syndrome potentially creates the possibility of AV conduction 1: 1 with atrial flutter and can lead to a high frequency of ventricular responses in atrial fibrillation. This can lead to ventricular fibrillation and sudden cardiac death. In the literature there are descriptions of isolated cases of ventricular fibrillation in children of the first years of life with WPW syndrome on the background of constant intake of digoxin. Thus, if we talk about WPW syndrome, prescribe digoxin, if necessary, can only be a hidden type of WPW syndrome.

Quite often digoxin is used in children of the first year of life with atrial tachycardia. The recommended dose of the drug is calculated based on the age and weight of the child. Measuring the concentration of digoxin in the blood helps maintain therapeutic concentrations and prevent overdose and toxic effects of the drug. Symptoms of intoxication: nausea, visual disturbances, drowsiness, AV blockade and ventricular tachycardia. Combination with other drugs (preferably with β-blockers) is possible, but usually requires a reduction in the dose of digoxin . Therapeutic plasma concentration is 0.7–2 ng/ml. signs of digoxin intoxication usually appear when its plasma concentration is more than 3 ng/ml.

Digoxin is available in tablets of 0.25 mg and 0.125 mg and in ampoules of 0.025 % solution in 1 ml (0.25 mg).

The therapeutic dose of saturation is 30–50 μg/kg/day. 50 % in the first reception, then 25 % in a 6-hour interval. Maintenance dose of digoxin 7–10 μg/kg/day. Perhaps the primary intravenous administration of digoxin at a dose of 75 % of the estimated. The effect of digoxin occurs 15–30 minutes after intravenous administration.

Contraindications to the appointment of digoxin are ventricular arrhythmia, AV blockade, subaortic stenosis, constrictive pericarditis. Renal function should be evaluated before prescribing digoxin . Conducting EIT on the background of digoxin may lead to ventricular fibrillation.

Ivabradine (Coraxan)

Ivabradine selectively blocks If-channels that control spontaneous diastolic depolarization, leading to suppression of sinus node automatism and decreased heart rate. The main use of this drug is sinus tachycardia, in particular chronic sinus tachycardia. It is possible that this drug will be effective and relatively accelerated ectopic rhythms.

Ivabradine is available in tablets of 5 mg. There are currently no recommendations for prescribing ivabradine to children. The recommended dose for adults is 10 mg/day 2 g/day with a possible increase to 15 mg/day.

Fenlipsin (carbamazepine)

Fenlipsin has antiarrhythmic effects in frequent extrasystoles and some

SVT. Fenlipsin has neurotropic, cardiotropic, membrane stabilizing and antiaffective action.

Available in tablets of 0.2 g. Appointed in the following doses: children under 1 year – 0.1–0.2 g/day in three doses, aged – 1–5 years – 0.1–0.4 g/day in three doses, at the age of 6–10 years – 0.2–0.6 g/day in three doses, older than 10 years – 0.2–1 g/day in three doses. The course of treatment is 1–4 months.

Other psychopharmacological agents, such as phenibut, grandaxin, may be useful in SVT, especially in children with autonomic dysfunction.

Phenibut has elements neuroprotective activity provides trankv ilizuyuchu effect, reduces stress, anxiety, improves sleep. Available in tablets of 0.25 g Designed after eating in the following doses: children up to 8 years – 0.45 g/day intake of three and the age – 8–14 years – 0.75 g/day in three receiving and, older than 14 years – 0.75–1.5 g/day in three doses. The course of treatment is 14–21 days.

"Pill in your pocket" - in the event of such tachycardia attacks, you can use the appointment of antiarrhythmic drugs per os only to buy attacks, without the appointment of tread therapy. For this purpose, *verapamil* (40 mg) or *propafenone* (150 mg) is used – in children older than 6 years, once in the form of a tablet, ground into a powder and dissolved in a tablespoon of warm water. At the same time take 2 tablets of *panangin*, also ground into a powder. In children under 6 years of age, drug doses are calculated depending on age and body weight.

In the event of a tachycardia attack, it is recommended that a single-dose dose of phenibut be given to calm the child. For the same purpose one of tinctures – tincture of a peony, valerian, motherwort or *valocardinum*, *corvalol* – in a single dose on 1 drop a year of life is appointed. Reception gives a psychotherapeutic effect, which is important for further measures to purchase a tachycardia attack.

In pediatric arrhythmology, basic therapy is traditionally used to correct and normalize cardiocerebral interactions, neurovegetative disorders and restore the regulatory function of the sympathoadrenal system. These regimens include nootropic drugs, membrane stabilizers and metabolic agents. Nootropic and nootropopodibni drugs (*nootropil, piracetam, Pantogam, lutsetam, aminolon, glutamic acid, Phenibut*) stimulate redox processes involved in the synthesis of ATP, increase tolerance CNS hypoxia, have vaholitychnyy effect involved in the activation serotonynerhichnoyi system (*Phenibut*). When prescribing basic therapy, the course of treatment should be from 4 to 6 weeks, 2 times a year. At the same time prescribed more than three drugs (representatives of different groups).

Arrhythmogenic effects of antiarrhythmic drugs are often associated with hypokalemia and hypomagnesemia. Therefore, in the complex medical treatment of SVT it is necessary to include drugs of potassium and magnesium,

which in addition to the prevention of arrhythmogenic effects of other drugs have their own antiarrhythmic effect. You should also recommend eating foods rich in potassium and magnesium salts: raisins, dried apricots, buckwheat, oats, barley, wheat, spinach, zucchini, soy, beans, peas, carrots, cranberries, apricots, etc.

In recent years, widely used magnesium preparations such as *Magnerot* , *Magne B6* , which affect metabolic processes and have antiarrhythmic effects.

Magne B6 is available in tablets and solution for drinking inside in ampoules. Doses: children aged 1–6 years (oral solution) – 10–30 mg/kg/day three times a day, aged 6–12 years – 2–6 tablets (1–3 ampoules) three times a day, over 12 years – 6–8 tablets (3–4 ampoules) three times a day. The course of treatment is 1 month.

Metabolic drugs are involved in all types of metabolism, restore normal electrical activity of the myocardium. Doses of the most frequently used drugs:

– *Coenzyme Q10* (capsules) – 1 capsule 1–3 times a day.

– *Kudesan* (coenzyme) (drops for oral administration) – age 1–3 years – 4–10 drops 1 time per day, 3–7 years – 10–16 drops 1 time per day, 7–12 years – 16–20 drops once a day, over 12 years – 20–60 drops once a day. The course of treatment is 2–3 months.

–*Mildronate* (capsules 250, 500 mg; syrup – in 5 ml 250 mg) – children under 12 years – 500 mg/day 2 times a day, over 12 years – 500–1000 mg/day 2 times a day. The course of treatment is 2–3 weeks.

– *Elkar* (levocarnitine) (20 % solution) – children under 3 years – 10 drops 2 times a day, 3–6 years – 14 2–3 times a day, 6–12 years – 28–42 drops 2–3 times a day. The course of treatment is 1 month.

Of great importance in the complex therapy of SVT is the normalization of the daily routine, nutrition and physical activity of children. Necessary walks in the fresh air, restrictions on watching TV, computer games, night sleep should be at least 8–10 hours. If there is no clear connection between SVT and physical activity, moderate physical exercises at the level of therapeutic physical training are necessary. Hypodynamia leads to further autonomic maladaptation, which is often manifested by worsening of the disease. You should avoid drinking coffee, strong tea, energy drinks. Absolutely unacceptable smoking and alcohol.

Parents of the child should be able to use vagal techniques, be informed about possible complications and their manifestations during antiarrhythmic therapy. It is desirable to keep a diary of tachycardia attacks with their clinical characteristics (duration of the attack, provoking factors, methods of purchase), which helps the doctor to properly assess the clinical course of the disease and timely adjust therapy.

But once again we emphasize that long-term antiarrhythmic therapy should be prescribed to children only if there are restrictions (usually age) or the impossibility of radical treatment – RCA electrophysiological substrate of tachycardia.

Vaccination of children should be carried out according to the general rules, taking into account the severity of the process of the underlying disease (UTI, carditis, cardiomyopathy). After radical correction of SVT (RCA) preventive vaccinations are carried out in a planned manner.

Ventricular tachyarrhythmias

Ventricular tachyarrhythmias (VT) are much less common in childhood than SVT. At a ventricle the heart rhythm leaves from ventricles, Purkinje fibers, legs of a bunch of His. VT is the leading cause of sudden cardiac death (SCD). VT, as a rule, arise at an organic, hereditary pathology of heart (a syndrome of the extended interval of QT, Brugada's syndrome, etc.), cardiomyopathies, myocarditises, an arrhythmogenic dysplasia of a right ventricle.

Classification of ventricular tachycardias

To date, there are several classifications that are based on etiological, pathogenetic, clinical characteristics. In addition to some differences in individual classifications, ventricular tachycardia can be divided into the following groups:

1. By clinical manifestations:
   1.1. Hemodynamically stable VT:
   - asymptomatic;
   - with minimal symptoms (eg, palpitations).

   1.2. Hemodynamically unstable VT:
   - presyncope;
   - syncope;
   - sudden cardiac arrest;
   - CHD.

2. During:
   2.1. Unstable VT (duration of attack < 30 sec.);
   2.2. Steady VT (duration of attack > 30 sec.).
3. Electrocardiographic classification:
   3.1. Monomorphic ventricular hiccardia (stable QRS morphology and constancy of RR intervals):
   - VT from the outflow tract of the right ventricle;
   - VT from the left ventricular outflow tract;
   - fascicular tachycardia (branching of the left leg of the bundle of His);
   - rare localization of the ventricle (ventricular tachycardia with MS, from the apex of the pancreas, valvular localization).

   3.2. Polymorphic ventricular tachycardia (constant changes in morphology and frequency of QRS complexes in any of the ECG leads):
   - unidirectional polymorphic VT;
   - bidirectional VT;
   - spindle-shaped VT type "pirouette";

• ventricular fibrillation.

Electrophysiological classification of VT includes topical localization fibrillation (plural, right ventricular, fascicular). By pathophysiological mechanisms – ectopia, trigger activity. By morphology – monomorphic, polymorphic, bidirectional. According to the classification of Laun VT belongs to IV B–V gradations of ventricular arrhythmias.

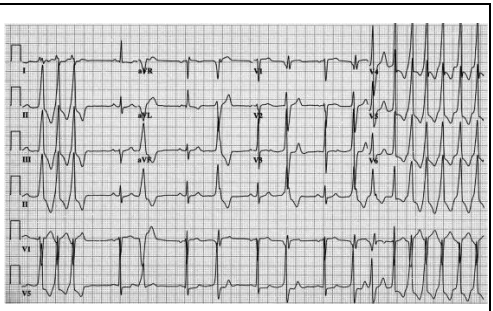
Clinical manifestations and ECG criteria of the ventricle

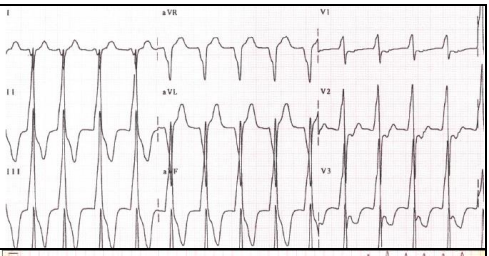
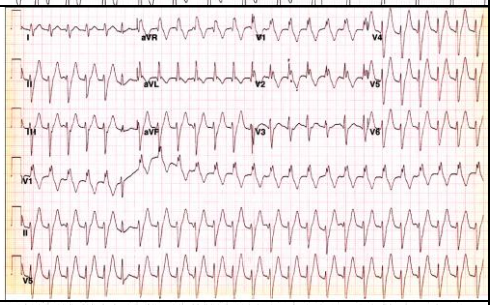
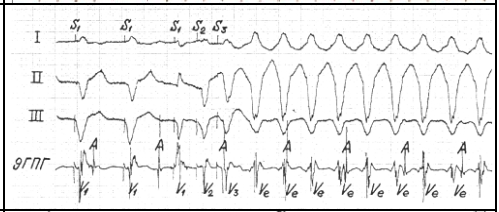
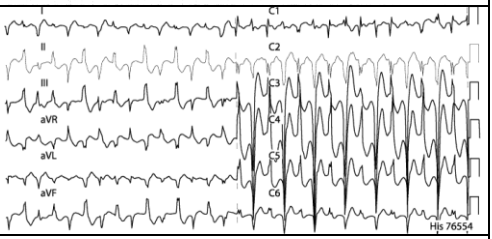
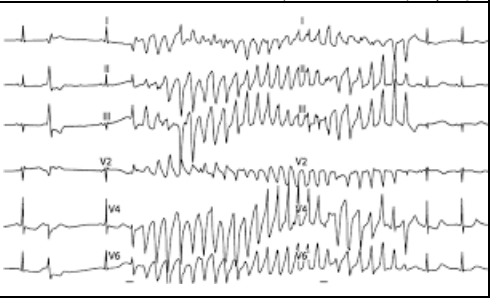
Clinical manifestations of CT are variable – from asymptomatic course (detected accidentally on the ECG during the examination) to serious frequent attacks with circulatory disorders (weakness, dizziness, shortness of breath, loss of consciousness).

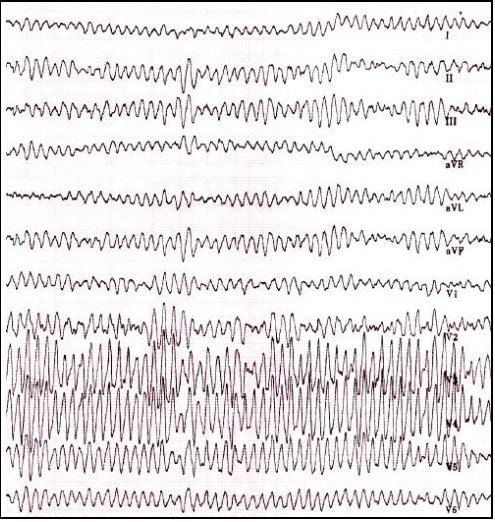
In children, myocarditis can be a common cause of atrial fibrillation, so you should pay attention to infections, changes in the ECG (ventricular hypertrophy, atria, repolarization, arrhythmias). To exclude (confirm) the acute course of myocarditis, troponins, LDH and its fractions, CPK are examined. One of the most sensitive laboratory tests for myocarditis is the reaction of inhibition of migration of lymphocytes (RIML) with antigen heart of the city, increasing the CD4, the ratio of CD4/CD8, increasing CD22, IgM, IgG, IgA and C and K. resorted to histological examination infarction, MRI heart with contrast, scintigraphy.

Tables 4

EKG-criterion VT

Ventricular tachycardia from the right ventricular outflow tract (right ventricular): 1. Expansion of the QRS complex. 2. Deviation of the electrical axis of the heart to the right (from +60° to +120°). 3. High tooth R in the lead aVF (R aVF > RI). 4. Orientation of the QRS complex in leads II and aVF upwards. 5. QS or rS - type in leads V1–V2. 6. Gradual increase in the amplitude of the tooth R in the chest leads. 7. RsR 'or M-shaped pattern of the QRS complex in lead V6.	

Ventricular tachycardia with outflow tract of left ventricle (VTOTLV): 1. Expansion of the QRS complex. 2. Deviation of the electrical axis of the heart to the right. 3. Changes in the morphology of the QRS complex in the right precordial leads of the type -rsr', rSr', Rsr', RSR', or "rabbit ears" (such as blockade of the right leg of the His bundle).	
Fascicular and tachycardia (FT): 1. Expansion (relative) of the QRS complex. 2. QRS complex morphology (during paroxysm) as a right bundle branch block (most often). 3. Deviation of the electrical axis of the heart to the left (alpha angle from -110° to -70°, the most common option).	
Polymorphic bidirectional VT: 1. Extensive QRS complexes with morphology of blockade of the right leg of the His bundle. 2. With and change of an electric axis in the frontal plane from a complex to a complex (from -70° to +120°).	
Catecholamine polymorphic VT: 1. Expansion of the QRS complex. 2. Changes in the electrical axis in the frontal plane from the complex to the complex (from -70° to +120°). 3. HR during attack 140–200 beats/min. 4. Normal QT interval.	
Spindle-shaped VT type of "pirouette": 1. Prolongation of the QT interval, and sometimes the U-wave on the surface ECG outside the attack. 2. Seizures induced ventricular extrasystole with different coupling intervals (often «R on T", at least – longer). 3. The total duration of the RR intervals ranges from 200 to 400 ms. 4. Widened QRS complex (more than 120 ms), a large second amplitude. 5. Over a short period of time, the height and	

polarity of the QRS changes, creating picture of sinusoidal rotation around the imaginary isoelectric lines. 6. During the transition positive QRS in negative normal individual systems can be register. 7. Tachycardia is unstable. 8. At visualization of a tooth P recognition of AV-dissociation is possible. 9. The attack usually stops spontaneously, sometimes with a gradual lengthening of the RR intervals. 10. The tendency to relapse, the attack can be repeated in a few seconds or minutes after its termination.	
Ventricular fibrillation : 1. QRS complex irregular, different shape and amplitude. 2. Heart rate greater than 220 beats/min. 3. There is always after any other ventricular arrhythmia and is a "terminal" fibrillation.	

Method and examination for VT

1. ECG.
2. Daily ECG monitoring.
3. EchoCG.
4. Test with dosed exercise (contraindicated in children with catecholaminergic polymorphic ventricular tachycardia).
5. Chest radiography.
6. Long cardiomonitoring ECG type REVEAL (in children with unexplained syncope).
7. Genetic examination.
8. Endocardial electrophysiological examination of the heart (endo-EPE) – the localization of the arrhythmogenic focus, induced tachycardia is determined and the possibility of its purchase is established .

9. Endomyocardial biopsy (to exclude myocarditis, CMP).

10. Research of blood electrolytes .

11. Study of thyroid hormones.

12. MRI of the heart (to exclude arrhythmogenic dysplasia of the right ventricle).

13. In the anamnesis it is necessary to specify whether there were attacks of loss of consciousness, circulatory disorders in tachycardia, familial cases of early death, convulsions, the presence of CHD, CMP, heart surgery, how exercise is transferred.

Treatment of VT.

Treatment of pulmonary embolism is divided into medical and non-medical (surgical and interventional). Drug therapy is the emergency purchase of a seizure and therapy to prevent seizures.

Buying an attack of monomorphic VT.

The first-line drug for the purchase of ventricular tachycardia with stable hemodynamics is lidocaine.

1. *Lidocaine* 2 %, 10 % solution. 1 ampoule – 2 ml (40 mg) and 1 ml (20 mg). It is administered intravenously, slowly in 5–10 ml of 5 % glucose solution (or 0.9 % NaCl solution, 5–10 ml) – a starting dose of 0.5–1 mg/kg. Dilution should be at least 1 : 1.

If rhythm is not restored: after 5–10 minutes additional administration of S dose (several injections are possible, to the total dose no more than 3 mg/kg).

After restoration of sinus rhythm – to prevent recurrence – maintenance infusion at a rate of 20–0 μg/kg/min (for several hours, until a lasting effect).

2. Synchronized cardioversion. If a child is unconscious at the time of the attack, if he develops cardiovascular collapse or acute heart failure with low cardiac output, the first stage of the purchase should be electrical cardioversion. Rules for applying electrodes for children: one electrode is placed to the right of the sternum under the clavicle, and the second – in the projection of the left mid-axillary line. If this arrangement of electrodes is not possible due to the small size of the chest, then in emergencies you can use the following positions: the first electrode above the projection of the heart, and the second - on the side wall of the chest at the level of the first electrode. The electrodes are lubricated with gel or gauze soaked in 0.9 % sodium chloride solution is placed under the electrodes. The initial discharge energy for children is 0.5–1 J/kg.

3. *Amiodarone* (cordarone) – III class. 1 ampoule of 5 % solution – 3 ml (150 mg). It is administered intravenously, slowly (diluted with only 5 % glucose solution) at a saturation dose of 5–10 mg/kg for 60 minutes. Then switch to a maintenance dose of 5–15 μg/kg/min

The use of amiodarone is justified in children in cases of ineffective

purchase of other antiarrhythmic drugs by tachycardia, in patients with organic heart disease and in cases where the onset of a ventricular attack develops circulatory failure with reduced ejection fraction.

4. *Verapamil* – (IV class) 0.25 % solution. 1 ampoule – 2 ml (5 mg). The estimated dose of 0.1 mg/kg is dissolved by nat. solution (20 ml of 0.9 % NaCl) and administered intravenously, slowly over 2 minutes. If there is no effect, it can be re-administered after 30 minutes (in the absence of hypotension).

This drug is used only for the relief of verapamil-sensitive, fascicular ventricular tachycardia. Verapamil is contraindicated for relief of other variants of ventricular tachycardias!!!

5. Correction of metabolic disorders (hip okaliyemiya , hypoxia me who can maintain persistence W T).

6. Drugs of the second series. *Obzidan* (*anaprilin*, *propranolol*, inderal). The estimated dose for intravenous administration is 0.01–0.02 mg/kg (maximum dose 0.2 mg/kg). Introduce e ovenno slowly. *Sotalol* (sotaleks) estimated dose for internal ovennoho input – 0.5–1.5 mg/kg to tively slowly for 10 minutes.

Tactics of ventricular fibrillation attack.

The success of resuscitation depends on the start time of these activities. Early defibrillation is important. The longer the FS lasts , the less likely it is to save the baby. The chances of success are reduced by 7–10 % every minute.

Treatment:

I. Resuscitation measures:

1. First of all , emergency defibrillation is performed (before intubation of the trachea and providing venous access). Defibrillate discharge 2 J/kg with no response – 4 J/kg, if necessary – 6 J/kg. In case of absence of independent breath or its inefficiency in parallel with defibrillation ALV with 100 % oxygen is carried out. Mandatory monitoring of ECG, non-invasive blood pressure, oxygen saturation.

2. With the preservation of ventricular fibrillation pre- or endotracheal administration of adrenaline at a dose of 0.1–0.2 mg/kg, if within 30–60 seconds after administration the situation does not change – re-defibrillation 4 J/kg, after 2–3 minutes – *adrenaline* (repeated defibrillation can be performed in 30–60 s after each administration of the drug).

3. In case of successful defibrillation is correction of metabolic disorders (acidosis, hypoxia, hyperkalemia, hypokalemia, and others.) Such as the availability of these changes can cause an unsuccessful resuscitation.

4. Prolonged infusion of anti-arrhythmic drugs, most often *amiodarone* 5 mg/kg iv.

II. All children after successful defibrillation are shown implantation of a cardioverter-defibrillator .

Buying a spindle-shaped VT.

The drug of choice for the purchase of ventricular tachycardia type "pirouette" is magnesium sulfate. *Magnesium sulfate* ($MgS0_4$) 25 % solution estimated dose of 25–50 mg/kg, administered intravenously, slowly for 1–3 minutes. If ineffective, you can repeat the introduction of the same dose in 5–10 minutes (maximum allowable dose – 2 g).

Treatment of the underlying disease (myocarditis, CHD, CMP) is mandatory.

Dispensary observation.

The nature of observation and continuation of antiarrhythmic and basic therapy depends on the frequency, duration and severity of attacks, organic heart pathology, age, localization of the arrhythmia substrate, the effectiveness of previously used drugs, the development of arrhythmogenic CMP.

There is no single treatment for antiarrhythmic therapy in children. In children, the ventricle is often refractory to antiarrhythmic drugs, rapidly developing refractoriness to them, the drugs have a proarrhythmogenic effect (except for β-blockers). Moreover, the risk of mortality in the background of drugs usually increases, although the overall risk of RCC without anti-arrhythmic drugs is lower than proarytmohenna their performance. The drug of choice for the need for treatment of PI is β-blockers.

β-blockers.

Propranolol, *atenolol*, *nadolol* and *esmolol* (for intravenous use). Of these drugs in our country is most often used propranolol, abroad – nadolol. β-blockers do not have a proarrhythmogenic effect.

Interaction of β-blockers with other drugs: the appointment of β-blocker together with aluminum salts, barbiturates, calcium salts, NSAIDs, penicillin, cholesterol, rifampicin may reduce its effectiveness. Haloperidol, hydralazine, loop diuretics may increase the toxic effect.

Contraindications: hypersensitivity to the drug, severe heart failure, bradycardia, cardiogenic shock, AV-blockade of II–III degree, severe ventricular dysfunction, severe asthma, insulin-dependent diabetes mellitus.

Use with caution during pregnancy (justified only if the benefit to the mother outweighs the potential risk to the fetus), in patients with bronchial asthma and bronchospasm, with grade I AV block, depression, bradycardia, hypoglycaemia (especially in newborns). β-blockers may reduce the symptoms of acute hypoglycaemia.

Gradual discontinuation of the drug and careful monitoring of the child during the withdrawal period is required.

• *Propranolol* (and nderal, anaprilin, obzidan). Non-selective β-blocker (tablets of 10, 40, 80 mg; ampoules of 0.1 % in 1 ml and 5 ml (1 ml – 1 mg). The half-life is 4–6 hours. The presence of the liquid form facilitates its use in

newborns and infants – 4 mg/ml or 8 mg/ml. For children under 8 months, the daily dose is divided into 4 doses. The daily dose is 1–4 mg/kg/day from 2 to 4 times a day. Intravenous use of this drug for the treatment of children is not recommended.

• *Atenolol*. Cardioselective β-blocker of prolonged action. Compared with propranolol, it has better tolerability, higher efficiency and fewer side effects (tablets of 25 mg, 50 mg, 100 mg; ampoules of 0.05 % solution of 10 ml (1 ml–0.5 g)). The half-life is 5–10 hours. Peak action – in 2–3 hours. The daily dose is 1–2 mg / kg / day in 1–2 doses.

• *Nadolol* (corgard). Nonselective β-blocker. A better drug than propranolol. Daily dose 1–2 mg/kg/day 1–2 times a day (tablets of 20 mg, 40 mg, 80 mg, 120 mg, 160 mg). The half-life is 12–24 hours. Peak action – in 2–3 hours.

ECG is recorded once every 3 months, Holter monitoring – once every 6 months. In the absence of recurrence of tachycardia, other arrhythmias and conduction, the patient is removed from the dispensary.

Vaccination individually, taking into account the underlying disease, and in idiopathic nature and arrhythmia (with unknown etiology), taking into account the effectiveness of antiarrhythmic therapy.

Expansion of physical activity (physical education and sports) is also decided individually.

At the initial detection of ventricular arrhythmia in a child and in the absence of a comprehensive examination and final diagnosis, the activity of patients should be limited to static and dynamic loads, ie the child does not exercise. It is also recommended to exclude mobile games for the period of the survey. Especially restrictions apply to children with organic heart disease, even with SH T with minimal symptoms.

If in patients, after a comprehensive examination, the data obtained indicate a safe VT (rare, unstable runs of VT without clinical manifestations, no organic heart disease, no factors of adverse disease), the international guidelines do not prohibit such children to exercise in general. group.

It is known that professional athletes, mostly males, may have athletic heart syndrome, which includes a moderate increase in left ventricular myocardial mass with normal left ventricular cavity size. And every third of them on this background has either ventricular arrhythmia, or other ectopias of high gradations, such as: verses and jogging unstable ventricles. At the end of a sports career, there is a leveling of changes in the heart and the disappearance of arrhythmias.

There are no policy guidelines for restricting exercise in children with ventricular tachycardia. However, there are a number of international documents that do not restrict professional sports among athletes who have rare, unstable run-ups without clinical manifestations.

Children with detected monophonic VT need regular supervision to assess the clinical course and possible progression of the disease, to assess the effectiveness of AAT.

It is recommended to conduct control ECG, CM, echocardiography at least once every 6 months.

Indications for radiofrequency ablation of arrhythmogenic focus (RFA).

RFA is the method of choice for treatment of idiopathic forms of VT. According to various clinics, its efficiency reaches 90 %. There are certain limitations of RFA in children under 10 years: to be carried out only in the event of life-threatening conditions (syncope, cardiogenic shock, ventricular fibrillation, etc.), ineffectiveness of antiarrhythmic therapy, including *cordarone*.

Older children, as well as adults, have the recommendations of WNOA (2009) for RFA:

Class I (absolute readings):

1. Patients with hemodynamically significant long-term monomorphic VT, refractory to AAT, or with intolerance to AAT, or do not want to receive long-term AAT.

2. Patients with VT on the system of the legs of the bundle of His (bundle branch re-entry).

3. Patients with implanted cardioverter-defibrillators, experiencing frequent discharges due to persistent monomorphic VT, which can not be prevented by reprogramming the device or changing the drug AAT. PI, resistant to AAT, or the patient's reluctance to take AAT for a long time.

Class II (relative indications):

Class II A:

1. The patient and who have a low risk of RCC and sim ptomatychnu unstable monomorfnu VT AAT resistant or intolerant AAT, or do not wish to receive long AAT.

2. Patients who have a low risk of RCC and frequent symptomatic monomorphic AE, resistant to AAT, or with intolerance to AAT, or do not want to receive long-term AAT.

Class II B:

Very common asymptomatic SE in order to prevent or treat tachycardia, cardiomyopathy.

Class III (no indications):

1. Asymptomatic , infrequent SH.

Complications RCHA can be divided into several categories: vascular (eh mate, thrombosis, pneumothorax) and due to radiofrequency exposure (aortic insufficiency in the performance RF effects in the left ventricle, the development of stenosis of the coronary arteries during RCHA in the outflow

tract Liv th ventricle (sinuses Valsalva) Possible damage to the AV connection (grade III AV blockade) Several cases of fatalities during RCA due to perforation of HTS have been described .

The question of the implantation of cardioverter dyfibrilyator and (IKI) in children remains an unresolved problem because indications for this procedure calc obleni adults only .

In 2009, the All-Russian Scientific Society of Clinical Electrophysiology, Arrhythmology and Pacemaking (VNTA) based on the Arrhythmic College of Cardiology (ACC) and the American Heart Association (ANA) in 2008 proposed indications for IDC in children (*Table 5*).

Tables 5

Indications for IDC implantation in children

Class	Indications
IA	ICD therapy is recommended for secondary prophylactic RCC patients, survivors of ventricular fibrillation or hemodynamically unstable VT or VT with syncope, with LVEF of 40 % or less if the patient receives optimal medical therapy and projected lifetime with a good functional status than 1 year.
IA	ICD implantation with β- blockers is indicated for patients with prolonged QT syndrome who have experienced a sudden cessation of blood circulation, with a predicted life expectancy with good functional status of more than 1 year.
IC	ICD implantation in pediatric practice indicated to people who survived after sudden cardiac arrest, in the event that the diligent examination excludes the possibility of removing the cause, if the patient receives optimal medical therapy and projected lifetime with a good functional status over 1 year.
IB	ICD implantation is indicated in patients with CHD survivors after sudden cardiac arrest in the case ruled her cause reverse; if the patient receives optimal drug therapy and the predicted life expectancy with good functional status exceeds 1 year.
IC	ICD implantation with β- blockers is indicated for patients with Brugada syndrome who have experienced sudden circulatory arrest and receive optimal drug therapy, with a predicted lifespan with good functional status exceeding 1 year.
IC	ICD therapy in conjunction with pharmacological therapy indicated for children at high risk of RCC (ion channel defects or cardiomyopathy) or persistent episodes of ventricular arrhythmias. The decision to implant an ICD is based on an assessment of the risk of RCC associated with a particular disease, the potential effectiveness of drug therapy, possible complications due to ICD implantation, if the life expectancy with good functional status exceeds 1 year.
IC	Patients with CHD and spontaneous persistent ventricular tachycardia should be subjected to invasive research hemodynamics and electrophysiological of the survey . Recommended method and treatment include catheter ablation or surgical resection to eliminate VT. In the absence of effect, ICD implantation is recommended.
IB	ICD therapy is recommended for primary prophylactic RCC, to reduce total mortality by reducing sudden cardiac death in patients with not ischemic heart disease, which LVEF 30-35% and less FC in NYHA II or III, receiving optimal drug therapy, when the predicted life expectancy with good functional status exceeds 1 year.
IB	ICD implantation for prophylactic indicated in patients with RCC arrythmogenic RV cardiomyopathy and documented episodes of VT \ VF receiving optimal drug therapy and projected duration of their life with a good functional status may exceed 1 year.
IB	ICD therapy should be used in patients with hypertrophic cardiomyopathy and persistent episodes of VT \ V III , receiving optimal medical therapy and projected duration of their life with a good functional status may exceed 1 year.

Class	Indications
IIa B	ICD therapy is appropriate for children with spontaneous persistent episodes of VT associated with decreased left ventricular function (LVEF <35%) that continuously receive optimal medical therapy, if the projected lifetime of more than 1 year.
IIa B	For patients with the CHD and with unexplained syncope reduced left ventricular function is recommended invasive examination of hemodynamic and electrophysiological studies. If no reversible cause of fainting is identified, ICD implantation is indicated for such patients if the patient is receiving optimal drug therapy and the predicted lifespan with good functional status exceeds 1 year.
IIa B	ICD implantation in combination with β- blockers is an effective method of reducing the risk of RCC in patients with prolonged QT interval syndrome who have fainted and / or episodes of VT on the background of β-blockers, with a life expectancy with good functional status of more than 1 year.
IIa C	ICD implantation can be effective treatment for termination century persistent episodes of VT in patients with preserved LV function and the absence of structural heart disease, are constantly receiving optimal drug therapy if the estimated period of their lives than 1 year.
IIa C	ICD implantation may be useful in patients with life-threatening ventricular arrhythmias who are not in the acute phase of myocarditis, constantly receiving optimal drug therapy and projected duration of their life with a good functional status than 1 year.

Tests

1. ECG of a 10-year-old child shows a sharp increase in the heart rate 180/min, P tooth is layered on tooth T deforming it. M moderate elongation of interval is present. QRS complex is not changed. Which if the following is present in the child?

A. Paroxysmal supraventricular tachycardia.
B. Paroxysmal ventricular tachycardia.
C. Atrial flutter.
D. Atrial fibrillation.
E. Extrasystole.

2. A 5-year-old child with II A heart failure is brought to the hospiital. The child is prescribed digoxin. According to which if the schemes is it necessary toreach the dose of saturation if the method of moderately fast digitization is chosen?

A. Within 2 days. *C. Within 3 days.* *E. Within 5–7 days.*
B. During the day. *D. Within 4 days.*

3. A 12-year-old girl with oligoanuric stage of acute renal failure on the ECG revealed waves of different shapes, widths, heights, with a chaotic rhythm and a frequency of more than 320 beats/min. What complication arose?

A. Ventricular fibrillation. *D. Atrioventricular block.*
B. Atrial fibrillation. *E. Atrial fibrillation.*
C. Sinoatrial blockade.

4. A 16-year-old boy went to the doctor with complaints of heart failure. When performing an ECG, the rhythm is incorrect, every second contraction is premature, the tooth P is flat, the QRS complex is undeformed. Which research method is most informative for diagnosis?

A. Echocardiography in M-mode. *D. Phonocardiography.*
B. Echocardiography in B-mode. *E. Bicycle ergometry.*
C. Holter ECG monitoring.

5. A 9-year-old child has been suffering from ARI for 3 days. Suddenly the condition worsened, there is shortness of breath, cough, cold sweat. On examination, the child is pale, over the lungs there are of various wet rales, hepatosplenomegaly. With a diagnosis of acute myocarditis, the child was taken to the cardiorheumatology department. In the waiting room, the child suddenly fainted. The rhythm on the ECG is incorrect, the atrial complexes are registered separately from the ventricular. What is the child's diagnosis?

A. Syndrome of weakness of the sinus node.
B. Vasovagal syncope.
C. Morgan-Adams-Stokes syndrome.
D. Paroxysm of supraventricular tachycardia.
E. Paroxysm of ventricular tachycardia.

6. A 15-year-old girl is observed by an endocrinologist with a diagnosis of autoimmune thyroiditis , hypertrophic phase. Recently complains of heart pain, palpitations, headache, tremor of the whole body. Emotionally labile, tearful. When performing an ECG on every third contraction, a premature QRS complex is registered, dilated, deformed, T wave is absent. What is the child's diagnosis?

A. Atrial extrasystole.
B. Ventricular arrhythmia.
C. Tachycardia type pirouette.
D. Atrial fibrillation.
E. Ventricular tachycardia.

7. A 12-year-old child underwent surgery for the CHD (a large ventricular defect) 3 years ago. During the follow-up examination at the cardiorheumatologist did not complain. The ECG revealed a sinus rhythm, in lead V1-2 registered dilated deformed ventricular complex in the form of rR, tooth T discordant, in lead V6 wide tooth S. What is the child's diagnosis?

A. Ventricular arrhythmia.
B. WPW syndrome.
C. Syndrome of weakness of the sinus node.
D. Complete blockade of the right leg of the His bundle.
E. Incomplete blockade of the right leg of the His bundle.

8. At the teenager of 15 years at carrying out auscultation of heart arrhythmia is revealed, at carrying out an ECG research revealed: fluctuations of an integral RR within 10 % without change of a tooth of P and an interval of PQ. What is the child's diagnosis?

A. Adolescent respiratory arrhythmia.
B. Syndrome of weakness of the sinus node.
C. Supraventricular tachycardia.
D. Wolff-Parkinson-White syndrome.
E. Sinoauricular block.

9. Select the path of the pulse in the norm:

A. CB-node – AV-node – His bundle – Purkinje fibers.

B. AV-node – CB-node – His bundle – Purkinje fibers.
C. His bundle – Purkinje fibers.
D. AV node – His bundle – Purkinje fibers.
E. C in -vuzol – AV node – bundle of his.

10. What is the pre-excitation of the ventricles in WPW syndrome?
A. Blockade of the right branch of the legs of the bundle of His.
B. Incomplete AV blockade.
C. History of myocarditis.
D. The presence of additional ways of conducting the pulse.
E. The absence of the Torel beam.

11. A 13-year-old boy, after exercise, began to experience prickly heart pain, a feeling of fainting, as well as irregular heartbeat. Which test should be ordered first to determine the cause of the arrhythmia?
A. Electrocardiogram. *D. Cardiointervalography.*
B. Echocardiogram. *E. X-ray examination of the thoracic cavity.*
C. Bicycle ergometry.

12. Which of the following drugs is a first-line tool for the relief of ventricular tachycardia with stable hemodynamics?
A. Lidocaine. *C. Magnesium sulfate ($MgSO_4$) 25 %.* *E. Adrenaline.*
B. Propranolol. *D. Amiodarone.*

13. What complaints can be a child of fibrillation of atrial ?:
A. Headache, fever, general weakness.
B. "Fear of death", palpitations at rest, loss of consciousness.
C. Heartbeat at rest, fever, loss of consciousness.
D. Palpitations during physical or emotional stress, cardialgia, headache, dizziness.
E. Fever, general weakness, shortness of breath, cough.

14. What type of arrhythmia is characterized by waves with a frequency of 400–600 per minute and irregular ventricular complexes of variable amplitude and width:
A. Ventricular fibrillation. *D. WPW syndrome.*
B. Atrial flutter. *E. Atrial fibrillation.*
C. Multifocal, chaotic SVT.

15. For which type of arrhythmia are characterized by waves with a frequency of 250–400 per minute, with normal or wide ventricular complexes:
A. Ventricular fibrillation. *D. WPW syndrome.*
B. Atrial flutter. *E. Atrial fibrillation.*
C. Multifocal, chaotic SVT.

16. Which of the following are the drugs of choice in the treatment of atrial fibrillation:
A. ATP. *C. Adrenaline.* *E. C magnesium sulfate ($MgSO_4$).*
B. Lidocaine. *D. Verapamil.*

17. Which of the following is a priority measure in diagnosing SVT with a stable state of hemodynamics ?:

A. Oscillating samples. *D. ICD installation.*
B. In the introduction of isoptin. *E. In the administration of digoxin.*
C. CSEFD.

18. Which of the following is a priority measure in diagnosing SVT with an unstable state of hemodynamics ?:

A. Oscillating samples. *D. Lidocaine.*
B. Polarizing mixture. *E. Inhalation of 100 % oxygen, EIT.*
C. Adrenaline.

19. Which of the following drugs can NOT be used to buy an attack of VT?

A. Digoxin. *C. Novocainamide.* *E. Verapamil.*
B. Lidocaine. *D. Amiodarone.*

20. Which of the following measures is a priority when buying a ventricular fibrillation attack?

A. Implantation of a cardioverter-defibrillator.
B. Defibrillation with a discharge of 2 J/kg, in the absence of reaction – 4 J/kg, if necessary – 6 J/kg. In case of absence of independent breath or its inefficiency in parallel with defibrillation IVL with 100 % oxygen is carried out.
C. Pre-or endotracheal administration of adrenaline at a dose of 0.1–0.2 mg/kg.
D. Correction of metabolic disorders (acidosis, hypoxia, hyperkalemia, hypokalemia, etc.).
E. Prolonged infusion of antiarrhythmic drugs, most often amiodarone 5 mg/kg iv.

Standards of answers to tests

1. A.	**3.** A.	**5.** C.	**7.** D.	**9.** A.	**11.** A.	**13.** D.	**15.** B.	**17.** A.	**19.** A.
2. C.	**4.** C.	**6.** B.	**8.** A.	**10.** D.	**12.** A.	**14.** E.	**16.** D.	**18.** E.	**20.** B.

Situational tasks.

1. Name the etiological factors of cardiac arrhythmias and conduction in children.

2. Name the possible causes and clinical symptoms of supraventricular paroxysmal tachycardia.

3. Name the possible causes and clinical symptoms of ventricular paroxysmal tachycardia.

4. Name the possible causes and clinical symptoms of atrial fibrillation.

5. Name the mechanisms of supraventricular tachycardia.

6. Which of the instrumental studies in paroxysmal tachycardia, atrial fibrillation will you use?

7. Tactics of the patient with paroxysmal tachycardia, atrial fibrillation in children.

8. Provision of emergency care for supraventricular VT.

9. Emergency care for ventricular fibrillation.

10. Emergency care for atrial fibrillation.

Standards of answers to situational tasks

1. Cardiac causes: PVS, carditis, CMP, heart tumors. Extracardiac: associated with impaired innervation of the heart with damage to the central and autonomic nervous system (IRR, TBI, hyper- and hypothyroidism, obesity, etc.)

2. The causes may be neuroses on the background of residual organic lesions of the CNS, hypertension-hydrocephalus syndrome, vegetative-vascular dysfunction with sympathetic-adrenal crises, WPW syndrome, organic heart disease (myocarditis, cardiomyopathy, heart disease). Clinical: characterized by an increase in heart rate to 140–200 beats per minute, sudden onset and sudden cessation of the attack. The duration of the attack is from a few seconds to several hours and even days. With an increase in heart rate of more than 220–250 per minute, develops a small cardiac output syndrome.

3. The causes are organic myocardial damage: carditis, cardiomyopathy, heart disease, postmyocardial cardiosclerosis; intoxication with digitalis drugs, quinidine; hypo- and hyperkalemia, syndrome of prolonged QT interval. Very rarely develops in vegetative-vascular dysfunction, thyrotoxicosis, psychophysical overload. Chronic (continuously recurrent) ventricular PT causes severe hemodynamic disorders and is a life-threatening condition (transition to ventricular fibrillation and progression of circulatory failure). Clinical: increase in heart rate to 120–250 beats per minute, sudden onset and sudden end of the attack. With an increase in heart rate of more than 120–140 beats per minute, may develop a syndrome of small cardiac output. Continuously recurrent form is characterized by frequent recurrences and chronic course.

4. Atrial flutter – a form of atrial fibrillation, in which there is accelerated rhythmic excitation and contraction of the atria with a frequency of 250–400 impulses. per minute. The frequency of ventricular contraction as a result of functional AV-blockade is less than the frequency of atrial contraction and is recorded in the ratio 1 : 2, 1 : 3, 1 : 4. Clinical: children complain of palpitations during physical or psycho-emotional stress, cardialgia, headache, dizziness.

5. The causes of SVT may be impaired pulse formation (increased automatism; residual or latent rhythm drivers, such as sinus tachycardia; pathological automatism of ectopic foci in the atrial myocardium – focal atrial tachycardia or tachycardia with AV connection; trigger activity -– the occurrence of depolarization, which are associated with the previous action potential) and impulse conduction (re-entry), reciprocal tachycardia, for example, in the presence of additional conduction pathways (ACP).

6. Supraventricular PT: the ECG registers a number of consecutive atrial extrasystoles (not less than 4-6 with a frequency of more than 160 per minute). The tooth P of various forms (+, -) or is not defined. The QRS complex is not changed. Transient incomplete AV-blockade of the I-II degree can be layered. The most informative for diagnosis is transesophageal electrophysiological study of the conduction system of the heart and daily HM. Ventricular PT: ECG registers "volleys" of successive ventricular extrasystoles (more than 5) with short periods of sinus rhythm. QRS complexes are wide (more than 0.1 sec), deformed, T-waves are discordant to the main tooth of the QRS complex. The P wave is rarely recognized by layering on other elements of the ECG. Ventricular PT can be mono- or polymorphic. Polymorphic or chaotic ventricular PT is a threat to the development of ventricular fibrillation. One of the variants of polymorphic ventricular PT is tachyarrhythmia type "pirouette". Atrial fibrillation: the ECG records waves with a frequency of 250–300 pulses. per minute. Waves are better expressed in leads II, III, avF, V1-2. No isoelectric line. Ventricular complexes are normal, wide or deformed in the presence of a violation of intraventricular conduction, are recorded with a frequency of 120–150 per minute, in the ratio of waves as 1 : 2, 1 : 3 and less.

7. Tactics of the patient depends on the type of arrhythmia. In case of extrasystole, NPT and FA, which are not accompanied by clinical symptoms and decrease in cardiac function, AAT is not prescribed. Preference is given to the treatment of the underlying disease that led to the development of arrhythmia. At full ABB use drugs that increase heart rate, improve microcirculation and antioxidant. The presence of syncopal states, as well as a decrease in heart rate < 40–45 beats/min is an indication for the pacemaker.

8. The child is placed in a horizontal position and provide access to fresh air. Perform a sequence of reflex measures that increase the tone of the vagus nerve: in children older than 3–4 years: Valsalva test (tension with the nose closed for 10 seconds); massage of the carotid sinus in the carotid artery for 5–10 seconds, first on the right, and in the absence of effect – on the left; additional techniques used in children older than 7 years: pressing a spatula on the root of the tongue, slow deep swallowing, wiping with cold water. In the absence of effect from reflex receptions enter ATP 1 % solution in/in jet, quickly.Aantiarrhythmic drugs: verapamil 0.25 % solution in / in slowly (no dissolution) under the control of blood pressure and heart rate. Then 5–15 mg/kg of cordarone is administered daily. Then, in case of inefficiency, CSEFD is performed.

9. a) First of all, emergency defibrillation is performed (before intubation of the trachea and providing venous access). Carry out defibrillation with a discharge of 2 J/kg, in the absence of reaction – 4 J/kg, if necessary – 6 J/kg. In case of absence of independent breath or its inefficiency in parallel with

defibrillation IVL with 100 % oxygen is carried out. Mandatory monitoring of ECG, non-invasive blood pressure, oxygen saturation.

b) While maintaining ventricular fibrillation, pre- or endotracheal administration of adrenaline at a dose of 0.1–0.2 mg/kg, if within 30–60 seconds after administration the situation does not change – repeated defibrillation 4 J/kg, after 2–3 minutes – adrenaline (repeated defibrillation can be performed in 30–0 s after each administration of the drug).

in) In the case of successful defibrillation, the correction of metabolic disorders (acidosis, hypoxia, hyperkalemia, hypokalemia, etc.) is performed, as the presence of these changes may be the cause of unsuccessful resuscitation.

d) Prolonged infusion of antiarrhythmic drugs, most often amiodarone 5 mg/kg iv.

10. Urgent antiarrhythmic therapy of paroxysmal SVT in an unstable state (hemodynamically ineffective tachyarrhythmia, collapse, syncopal states), which is due to atrial fibrillation, including with antegrade conduction of the pulse by DPS requires urgent electroimpulsive therapy. Of the antiarrhythmic drugs (taking into account the presumed type of arrhythmia), class I drugs procainamide (novocainamide), lidocaine (for polymorphic ventricular tachycardia or ventricular fibrillation) are used. If tachycardia persists, cordarone is indicated, with a possible dose increase from 5 to 15 mg/kg (in young children), as well as magnesium supplements. Given the instability of hemodynamics, severe myocardial hypoxia, electrolyte imbalance to maintain central hemodynamics, the introduction of adrenergic drugs, polarizing mixture and antioxidants (Mexidol, 2 mg/kg daily) is indicated.

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Навчальне видання

Порушення ритму та провідності серця у дітей

Методичні вказівки для студентів V–VI курсів вищих мед. закладів освіти III–IV рівнів акредитації, що навчаються англійською мовою

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