Arrhythmias (pediatrics)

Heart rhythm disorders (arrhythmias, dysrhythmias) is a collective term for disorders of heart rate, heart rhythm, heart rate, or a combination thereof. The most common arrhythmia in children is **supraventricular tachycardia**, which can occur in the form of well-clinically tolerated short paroxysms or can cause heart failure with ventricular dysfunction, AV valve regurgitation, metabolic acidosis, hypotension and impaired consciousness within a few hours .

Clinical picture of arrhythmias

- abnormally fast (due to age and current load) or slow or irregular heartbeat;
- change in general condition: feeling of irregular heartbeat, palpitations, vertigo, fatigue, eventually syncope (potentially life-threatening);
- in newborns and infants: fatigue, apathy, cyanosis, refusal to eat.[1]

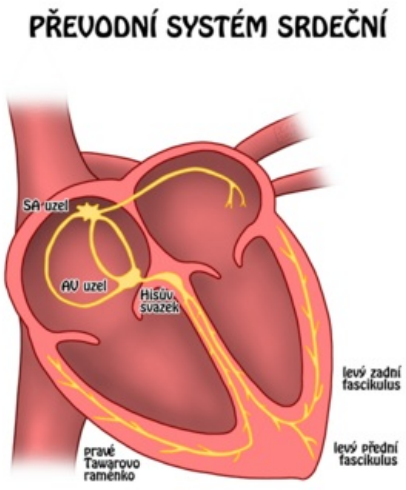
Diagnostics

- 12-lead ECG;
- 24-hour ECG monitoring (Holter);
- eventually stress examination (ergometry).[1]

Classification of arrhythmias

According to the **heart rate** that arrhythmias induce:

- bradyarrhythmia - reduced SA node automaticity, SA or AV blockade;
- tachyarrhythmias - reentry, abnormal automaticity, triggered activity.[1]



Heart conduction system

According to the **pathogenesis**: disorders of excitation, disorders of conduction, combined disorders.

Depending on **where the arrhythmias occur**: sinus, supraventricular, ventricular.

According to **clinical severity**: benign, malignant.[2]

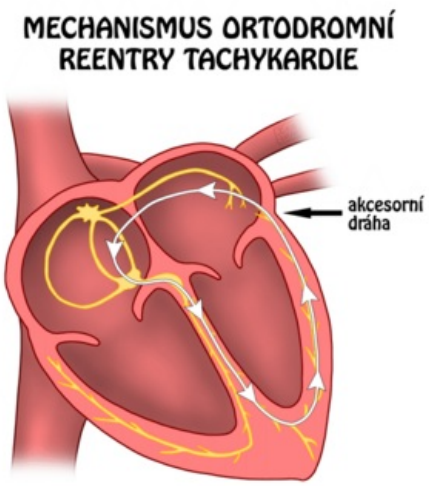
Basic heart rhythm disorders in children

Supraventricular tachycardia

- heart rate 200-300 / min. in newborns and infants; 170-240 / min. in older children;
- in newborns and infants most often AVRT and FAT, in older children most often AVRT and AVNRT.

Atrioventricular reentry tachycardia (AVRT)

- additional atrial connector - represents one part of the reentry circuit, the other part is the heart's own transmission system;
- most often orthodromic - the chambers are activated by their own transmission system (narrow QRS complex), the atria are activated retrogradely via an additional coupler (retrograde P wave just behind the end of the QRS);
- Wolff-Parkinson-White image - the additional conjunction is capable of antegrade conduction from the atria to the ventricles → preexcitation of the ventricles through the additional conjunction at the time of sinus rhythm;
- Wolff-Parkinson-White Syndrome - WPW image + AVRT episode;
- especially in newborns and infants (SVT paroxysms disappear spontaneously within 1 year of age) and children older than 5 years.



The principle of orthodromic AV reentry tachycardia: the signal reaches the chambers physiologically (via the AV node), from which, however, the accessory pathway in the septum between the left atrium and left ventricle (James's bundle) returns to the atrium, creating a reentry circuit leading to tachycardia.

Atrioventricular nodal reentry tachycardia (AVNRT)

- division of the AV node into 2 paths, each of which has a different line speed and length of the refractory period → forms a reentry circuit;
- rather in older children; very rare in newborns and infants.

Focal atrial tachycardia (FAT)

- abnormal automaticity of the ectopic atrial muscle deposit → via AV

node transmission to the ventricles;
- very common in newborns and infants;
- small children have a high transfer capacity of the AV node → transfer from the atrium to the ventricles 1: 1.

Therapy

- vagal maneuvers: immersion of the face in ice water (activation of n. X → slowing of conduction and prolongation of the refractory period in the AV node);
- adenosine 0.1-0.3 mg/kg in the form of a rapid bolus with ECG monitoring - interrupts the reentry circuit in the area of the AV node;
  - in WPW syndrome it is necessary to have the possibility of external cardioversion (risk of atrial fibrillation with transfer to the ventricles);
- inefficiency: propafenone i.v.
- in case of repeated early recurrences: continuous infusion of amiodarone, then orally sotalol;
- rapid stimulation of the atrium with an electrode inserted through the esophagus;
- synchronized cardioversion (0.5-1 J/kg);
- radiofrequency catheter ablation (from 5-6 years).[1]

Extrasystoles (ES)

- at a younger age there are more supraventricular ES, later more ventricular ES;
- monotopic ones come from one and polytopic ones from several deposits - these have several P-wave shapes (supraventricular ES) or QRS complexes (ventricular ES);
- if the ECG alternates between a normal sinus pulse and an ES, we are talking about **bigemia**;
- if the ES repeats after two sinus pulses, it is a **trigemina**;
- if they go after one sinusoidal ES pulse in a pair, we are talking about a **couplet**;
- when they go in trinity we talk about **triplet** → in three or more ES we talk about tachycardia.

- Ventricular ES: on the ECG we find an aberrant QRS complex in front of which the P wave is missing, the T wave is usually discordant (oriented opposite to the highest oscillation of the QRS complex).

- If we find the ES during rest, but it disappears during activity, it is a valuable indication of benignity

The potentially serious are:

- - **ES multifocal;**
  - **ES multiple;**
  - **ES emanating from the L chamber;**
  - **ES appearing during exertion and disappearing at rest;**
  - **ES with the phenomenon R on T: ventricular ES rests on the descending arm of the wave T → can lead to ventricular fibrillation!**

Etiology

- hypoglycemia;
- disorders of the internal environment;
- endocrinopathy;
- myocarditis;
- cardiomyopathy;
- ES can also occur in a healthy heart.[3]

Bradycardia

Bradycardia is often accompanied by hypoxic conditions, in which case a causal solution is necessary, i.e. adequate oxygen therapy.

Sinus node dysfunction / Sick sinus syndrome

- Heart rate creation disorder that may be functional/anatomical and transient/permanent.

Etiology

- increased vagotonia;
- endocrinopathy;
- medicaments;
- direct damage to the sinus node.

The clinical picture

- Most patients are asymptomatic, in young children there may be difficulty feeding, in larger ones fatigue, vertigo.
- However, some children are at risk of syncope or sudden death.

Diagnosis

- On the ECG we observe a slow or irregular sinus action, various surrogate rhythms or, conversely, paroxysms of tachycardia.
- The negative effect of the autonomic nervous system confirms the ability of the sinus node to increase HR during exercise (confirmed by ergometry)
- Holter ECG shows the variability of HR during the day and night, episodes of sinus pauses or the occurrence of other severe arrhythmias.

Therapy

- Asymptomatic patients do not require treatment.
- In bradycardia with a hemodynamic disorder, we indicate a pacemaker implantation.

AV blockade II. degree Mobitz type II

- It is characterized by intermittent blockade of AV transmission without prior prolongation of PQ, i.e. the interval of PQ is still constant, after several "normal" strokes the P wave is not followed by the QRS complex.

Etiology

- direct injury;
- inflammation;
- degenerative diseases.

Mobitz II requires very careful monitoring, because unlike Mobitz I there is a real threat of progression to III. degree of blockade, i.e. complete AV block → risk of syncope / sudden death.

Therapy

- Isoprenaline 0.02 mg/kg, ev. isoprenaline infusion 0.02 mg/kg (interestingly, Atropine has no effect here).
- Pacemaker implantation is a long-term solution.

AV blockade III. stage (complete AV block)

- There is a complete interruption in the transmission of impulses from the atrium to the ventricles.
- The atria are controlled by a rhythm from the sinus node, the ventricles by a substitute slower rhythm from the junction or ventricular area.

Etiology

- surgical damage;
- inflammation;
- cardiomyopathy;
- newborns of mothers with SLE.

Clinic

- Clinically, it manifests itself in the image of low cardiac output.

Diagnosis

- The activity of the atria and ventricles is independent of the ECG, the frequency of P waves is higher than the frequency of QRS, there is no constant time interval between P and QRS, QRS has an abnormal shape, but it is usually narrow.

Therapy

- Atropine: 0.02 mg/kg iv, i.o., e.t., i.e. 0.1 mg/5 kg (0.2 ml);
- alternatively Isoprenaline 0.02 mg/kg;
- pacing as the definitive solution.

In a child who has symptoms of heart failure and <60 / min., AV block III must be excluded according to the ECG![3]

Tachycardia (tachyarrhythmia)

Pathophysiology

Tachycardia is caused by 3 possible mechanisms:

1. the principle of abnormal automaticity → tachycardias are started by spontaneous depolarization of a cell that is not part of the sinus node, these tachycardias are very difficult to treat therapeutically, because they do not

have cardioversion or overdriving, digoxin and beta-blockers are used in treatment;
2. the principle of triggered activity → tachycardia arises on the basis of afterdepolarizations;
3. the principle of circuit reentry → is the mechanism of most arrhythmias, reentry is the phenomenon of the intact heart and means the circular conduction of signal between two different heart pathways, where the pathways must be functionally separate.

It should be remembered that even with a sinus rhythm, there are limits to the tolerance of tachycardia. One of the recommendations is based on the equation for determining the maximum allowable frequency for a patient's age:

HR_{max} = 220 - age in years.

Heart rate in excess of this value can already reduce cardiac output by the principle of reduced diastolic filling. Patients with low ventricular compliance are the most compromised.

Tachyarrhythmias are divided into **supraventricular (SVT)** → above the His-beam bifurcation and **ventricular (VT)** → below the His-beam bifurcation.

- If the QRS complex is "narrow" (<0.08 seconds), we hypothesize that one of the supraventricular mechanisms is the cause of tachycardia.
- If the QRS is "wide" (> 0.10 seconds), it may be a ventricular tachyarrhythmia or an atypical SVT.

⚠**In practice, it is quite difficult to distinguish VT from atypical SVT only on the basis of a standard ECG (an excellent diagnostic method here is the esophageal ECG) → tachycardia with "wide" QRS in an acute situation is always treated as VT until proven otherwise.**

Tachyarrhythmia with "narrow" QRS (SVT)

SVT rarely endangers the patient's life if it does not last too long, so the first rule of SVT treatment is calm and composure, with the current effort to find out the mechanism of the cause of tachycardia before we start treatment.

On the other hand, long-term SVTs or some SVTs in WPW require urgent therapeutic intervention!

Diagnosis

To determine the exact mechanism of the arrhythmia (.ie. the time connection between the P-wave and the QRS complex, determining the axis of the P-wave), a 12-lead record is much more useful than a single-lead record.

- Paroxysmal SVTs with a narrow QRS and reentry mechanism have an RP interval shorter than PR.

Diagnosis is based on:

- anamnesis;
- physical examination;
- 12-lead ECG;
- echocardiography;
- ergometry;
- Holter ECG;
- transtelephone ECG transmission;
- esophageal atrial stimulation;
- intracardiac electrophysiological studies.

Therapy

Acute therapy

- vagal maneuvers - the most commonly used diving reflex today → applying ice to the face;
- Adenosine (Adenocor) 0.1 mg/kg i.v., i.o. very quickly!;
- preferably with a subsequent FR bolus, as adenosine has an extremely short half-life, at a repeated dose of 0.2 mg/kg i.v.

Further according to experience

- Propafenone (Rytmonorm) 1-2 mg/kg i.v. in 5 minutes;
- esophageal atrial stimulation;
- ev. electrical cardioversion.

Follow-up therapy

- β-blockers;
- Digoxin (KI in WPW syndrome) ;
- Propafenone;
- Amiodarone;
- Sotalol;
- in older children with SVT reentry, we prefer radiofrequency catheter ablation.

Sinus tachycardia must be differentiated diagnostically. Here, unlike SVT, HR is usually <200 / min., accelerates and decelerates according to the sympathetic tone (reentry tachycardia occurs and recedes suddenly, has no frequency variation), the P-axis is always normal, the A:V ratio is typically 1:1. Therapeutically, it is always necessary to address the underlying disease!

The most common causes of sinus tachycardia are

- fever;
- anemia;
- shock;
- heart Failure;
- pain/stress;
- hyperthyroidism.

Tachyarrhythmia "wide" QRS (Atypical SVT and VT)

- VT forms three or more aberrant QRS complexes that are wider than during sinus rhythm;
- Diagnosis is based on anamnesis, physical examination - here the crucial question is whether the patient requires CPR, 12-lead ECG.
- Ventricular tachyarrhythmias are the most malignant arrhythmias, typically occurring in patients with impaired myocardial function.
- Most forms of VT also arise from the reentry mechanism;
- QRS complexes are wide and either have identical morphology (monomorphic) or have different QRS shapes (polymorphic) → more malignant forms.
- In many cases, the P wave is not visible at all.

Therapy

Acute treatment

- if the patient is hypotensive or unresponsive, we perform urgent electrical cardioversion;
- if cardioversion is not available, Amiodarone (Cordaron) 5 mg/kg iv will be given as the 1st choice drug within 30 minutes, it can be repeated in 15-20 minutes;
- alternatively, 1% Mesocaine or Lidocaine 1 mg/kg i.v. may be given as a bolus followed by 20-60 µg/kg/min in a continuous infusion;
- during ventricular fibrillation, we perform unsynchronized cardioversion.

Specific treatment alternative with knowledge of QRS morphology

- **VT monomorphic reentry:** prokainamide 10–15 mg/kg i.v. within 20 minutes;
- **torsade de pointes**: $MgSO_4$ 25–50 mg/kg i.v. within 15–30 minutes, max. 2 g per dose, treat ionic imbalances by supplementing missing ions;
- **any VT:** electrical kardioversion, initial discharge 1-2 J/kg, can be repeated 1-2 times with discharge up to 4 J/kg.

Follow-up therapy

- β-blockers are useful for the permanent securing of congenital forms with prolonged QT.

Torsade de pointes

A specific form of VT is the form known as torsade de pointes. It is a polymorphic VT in which the direction of the QRS complexes gradually revolves around the isoelectric ECG line. Torsades occurs classically in patients with abnormally prolonged QT interval.

- when connected with deafness we talk about sy. Jervell-Lang-Nielsen,
- if hearing is the norm we are talking about sy. Roman-Ward,

Another etiological group are patients with ionic imbalances such as hypokalemia, hypocalcemia and hypomagnesemia.

- Clinically torsades can cause hypotension, syncope, ev. eventually ventricular fibrillation and death.

Atrial fibrillation and flutter

- atrial fibrillation and flutter are caused by the reentry mechanism in the atrial muscle;
- flutter represents a single reentry circuit, fibrillation involves multiple small and constantly changing reentry circuits.

Atrial fibrillation and flutter are also the most serious forms of arrhythmia in WPW syndrome with the possibility of rapid antegrade transfer to the ventricles → catastrophically rapid ventricular response with the risk of cardiac arrest!

Etiology

- atrial dilatation;
- inflammation;
- hyperthyroidism;
- WPW syndrome.

Diagnosis

- atrial flutter has a classic ECG-like appearance of **saw teeth** (F waves);
- they are best seen in leads II, III and aVF and have a very irregular frequency around 300 / min (there may be an isoelectric line between the individual F waves);
- atrial fibrillation has the appearance of irregular, low-voltage, and rapid atrial waves (f-waves) on the ECG;
- the frequency of the ventricles in both conditions is determined by the ability of the transmission system to transfer excitations to the ventricles → risk of ventricular tachyarrhythmias.

Therapy

- because the mechanism of both arrhythmias is reentry, synchronized cardioversion is elegant and almost always successful;
- alternatively, Digoxin can be administered pharmacologically (increases the degree of AV block → decreases ventricular conduction);
- overdriving can also be tried with flutter (it is ineffective in fibrillation for multiple fast reentry circuits).[3]

NOTE: ***Synchronized cardioversion:***

- it is a discharge intended for the termination of SVT or organized VT;
- the discharge must therefore be synchronized with the QRS complex, coincidence with the T wave could induce ventricular fibrillation.

Defibrillation:

- it is a discharge using more energy;
- is indicated for polymorphic VT or ventricular fibrillation - there is no QRS with which the shock could be synchronized.

Overdrive pacing (overdriving):

- it is a stimulation of a part of the affected myocardial areas with a frequency slightly higher than the conductive ability of the reentry circuit.[3]

Links

Related articles

- Heart rhythm disorders
- Heart rhythm disorders (neonatology)

Reference

1. KUBUŠ, Peter. Poruchy srdečního rytmu u dětí. *Pediatrie pro praxi.* 2014, roč. 15, vol. 4, s. 216-221
2. PASTOR, J. *Langenbeck's medical web page* [online]. [cit. 2009]. <https://www.freewebs.com/langenbeck/>
3. HAVRÁNEK, Jiří: Arytmie.