
Thematic chapter: Differential diagnosis of the most common disease of the blood circulatory system in children. Emergency care in common emergency conditions

Academic hours: 6
Self-education: 4

1. SIGNIFICANCE

The significance of the topic is that the most prevalent form of pediatric arrhythmias, supraventricular tachycardia (SVT) is estimated to occur in one in 250–1000 children. It is the most common in infants younger than 4 months of age. Other common age groups include the years around puberty and 5–7 y of age.

SVT is associated with infection, fever, or drug (decongestant or sympathomimetic amines) exposure in 20–25%, WPW syndrome in 25%, congenital heart disease in 20%, concealed accessory pathways, and AV nodal reentry in the remainder. Atrial flutter occurs in 25%–40% of patients after atrial surgery, especially intra-atrial repair of complete transposition of the great arteries or the Fontan repair. The above dictates careful review of essential approaches to ECG reading by a student.

2. PREREQUISITES

The skills listed below will not be taught in this lesson but are necessary to perform physical examination of the patient with alterations in cardiac rhythm and conductivity in the intensive care unit during practical training.

Therefore, before beginning this lesson, student has to be sure of the ability to:
• Auscultate the heart;
• Examine perfusion of the skin.
• Measure blood pressure;
• Count pulse rate;
• Read ECG tracings.

3. EDUCATIONAL OBJECTIVES

Student should know:
- etiology, pathogenesis, classification, clinical manifestation, differential diagnosis and treatment principles of arrhythmia in children and infants.

Student should be able:
- to identify the child with alterations in cardiac rhythm and conductivity, make correct decisions during physical examination of the patient with listed conditions, take appropriate actions based on those decisions, demonstrate skills to develop treatment plan and follow-up.

4. INTERDISCIPLINARY INTEGRATION

<p>| Normal anatomy, Physiology | Anatomic and physiologic features of the cardio-vascular system in children of different age groups | Use knowledge of anatomic and physiologic features of the cardio-vascular system in children for evaluation of clinical findings |</p>
<table>
<thead>
<tr>
<th>Pathologic physiology</th>
<th>Pathophysiologic mechanisms of the cardiac arrhythmias</th>
<th>Recognize symptom and signs of cardiac arrhythmias</th>
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<tbody>
<tr>
<td>Propedeutics of pediatric diseases</td>
<td>Physical examination of the cardio-vascular system in children. Physiology of electrocardiogram.</td>
<td>Perform physical examination of the respiratory system (gross inspection, palpation, percussion, auscultation). Assess the results of electrocardiography</td>
</tr>
<tr>
<td>Imaging studies</td>
<td>Indications for the heart ultrasound</td>
<td>Assess radiologic examination of the chest</td>
</tr>
<tr>
<td>Intensive care</td>
<td>Symptoms and signs of different arrhythmias, its etiology, and principles of intensive care</td>
<td>Recognize different arrhythmias, assess its severity, provide emergency care</td>
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5. ABSTRACT FOR PRE-WORKSHOP SELF-EDUCATION

1. **Supraventricular tachycardia**
   Rapid heart rate originating in or involving structures located above ventricular tissue. Supraventricular tachycardia (SVT) includes the following:
   - Atrioventricular (AV) nodal reentrant supraventricular tachycardia. AV reentrant SVT (concealed accessory pathway with SVT and Wolff–Parkinson–White (WPW) syndrome with SVT);
   - Primary atrial tachycardia;
   - Junctional ectopic tachycardia;
   - Atrial flutter; atrial fibrillation.
   **Symptoms and signs:**
   - Palpitations: rapid regular heart rate with sudden onset and offset;
   - Throbbing in neck; sensation of fluttering in chest;
   - Chest or abdominal pain: discomfort in chest, often associated with nausea or abdominal discomfort;
   - Syncope or presyncope: loss of consciousness, dizziness or light-headedness at onset of rapid heart rate, due to hypotension.
   - Tachycardia: rapid heart rate of 150–320 bpm, greater than expected for age or activity level; tachypnea: secondary to pulmonary venous congestion from congestive heart failure (CHF); hypotension: associated with pallor and diaphoresis.
   - Congestive heart failure. Unlikely to occur unless structural or functional heart disease present or tachycardia persists for >24 hours.
   - Infants: poor feeding, irritability, lethargy, tachypnea, dyspnea, pallor, diaphoresis; children and adolescents: dyspnea, tachypnea, orthopnea, exercise intolerance, easy fatigability, pallor, diaphoresis.
   **Laboratory.**
   - ECG: Regular, rapid rhythm usually with narrow QRS complex. An ECG should be obtained immediately on conversion from SVT to sinus rhythm to look for the presence of delta waves confirming the WPW syndrome.
   - Chest radiography: usually normal unless CHF or associated congenital heart disease or cardiomyopathy is present.
- Echocardiography: to rule out the presence of associated congenital heart disease or functional heart disease: Ebstein’s anomaly of the tricuspid valve or corrected L-transposition of the great arteries are the most common congenital lesions.
- Electrophysiologic study: indicated in patients who present with syncope or cardiac arrest, those refractory to medication, or with medication side effects that cannot be tolerated. For risk assessment in WPW and to radiofrequency ablation.

  **Complications include:**
- Congestive heart failure.
- Cardiac arrest: associated with atrial fibrillation and WPW with rapid AV conduction.
- Differential diagnosis. various forms of SVT and VT.

**Treatment.**
- Vagal maneuvers:
  - Valsalva maneuver
  - Gagging,
  - Headstand. Ice.
- Radiofrequency ablation.
- Surgical ablation.
- Avoid caffeine, chocolate.
- Activity restriction generally not required.

  **Acute treatment.**
  - Adenosine, i.v. bolus, 50–100 mcg/kg followed by saline flush. Increase by 50 mcg/kg increments every 2 min to 400 mcg/kg or 12 mg maximal dose.
  - Digoxin, 30 mcg/kg i.v. total digitalizing dose (TDD); maximum dose, 1 mg; initial dose, one-half TDD; second dose, one-quarter TDD; third dose, one-quarter TDD.
  - Propranolol (beta-blocker), 0.05–0.1 mg/kg over 5 min every 6 h.
  - Esmolol (beta-blocker), i.v. load: 500 mcg/kg/min over 1 min followed by 50 mcg/kg/min over 4 min; repeat in 5 min with 500 mcg/kg/min over 1 min, 100 mcg/kg/min over 4 min; maintenance infusion: 50–200 mcg/kg/min.
  - Amiodarone, 5 mg/kg over 1 h i.v., followed by 5–10 mcg/kg/min infusion.
  - Procainamide (class I antiarrhythmic), 5 mg/kg i.v. over 5–10 min or 10–15 mg/kg over 30–45 min; infusion: 20–100 mcg/kg/min.

  **Longterm oral therapy.**
  - Digoxin: 40–60 mcg/kg TDD p.o. (i.v. dose is 75% of p.o. dose); maintenance: 10–15 mcg/kg/d divided every 12 h.
  - Propranolol: (beta-blocker), 0.25–1 mg/kg/oral dose every 6 h.
  - Nadolol (beta-blocker), 0.5–1 mg/kg/dose every 12 h.
  - Atenolol beta-blocker), 25–100 mg/d in single dose used for older children and adolescents.
  - Procainamide: 20–100 mg/kg/d every 4–12 h.
  - Flecainide (class I antiarrhythmic): 50–200 mg/m2/d every 12 h.
  - Propafenone: 200–300 mg/m2 in 3–4 doses or 8–10 mg/kg.
  - Amiodarone, loading dose: 10 mg/kg/dose twice daily for 7–14 d; maintenance: 5–10 mg/kg/dose daily.
  - Sotalol, 135 mg/m2/d.

**Prognosis.**
- Excellent for control and eventual cure;
- 75% controlled with first medication used;
- 10%–20% refractory.
- 1%–3% incidence of sudden death associated with WPW.
- With radiofrequency ablation, 80%–95% cure achievable.

**Follow-up and management.**
- Periodic ECGs and office visits.
• Holter or transtelephonic monitoring to assess symptoms.
• Aspirin after radiofrequency ablation for 4–6 wk.
• Follow-up ECGs after radiofrequency ablation.

2. **Differential diagnosis of other tachyarrhythmias**

<table>
<thead>
<tr>
<th><strong>ELECTROCARDIOGRAPHIC FINDINGS</strong></th>
<th><strong>HEART RATE (BEATS/MIN)</strong></th>
<th><strong>P WAVE</strong></th>
<th><strong>QRS DURATION</strong></th>
<th><strong>REGULARITY</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>Sinus tachycardia</td>
<td>&lt;225</td>
<td>Always present normal axis</td>
<td>Normal</td>
<td>Rate varies with respiration</td>
</tr>
<tr>
<td>Atrial tachycardia</td>
<td>180–320</td>
<td>Present—50%</td>
<td>Normal or prolonged (RBBB pattern)</td>
<td>Regular</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Superior axis common</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Atrial fibrillation</td>
<td>120–180</td>
<td>Fibrillatory waves</td>
<td>Normal or prolonged (RBBB pattern)</td>
<td>Irregularly irregular</td>
</tr>
<tr>
<td>Atrial flutter</td>
<td>Atrial: 250–400</td>
<td>Saw-toothed flutter waves</td>
<td>Normal or prolonged (RBBB pattern)</td>
<td>Regular ventricular response (e.g., 2:1, 3:1, 3:2, and so on)</td>
</tr>
<tr>
<td>Ventricular tachycardia</td>
<td>Ventricular response variable: 100–320</td>
<td>Absent or atrioventricular dissociation</td>
<td>Usually prolonged</td>
<td>Slightly irregular</td>
</tr>
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</table>

RBBB, right bundle branch block.

3. **Long Q-T Syndromes**

They are a cause of syncope and sudden death. May be associated with sudden infant death syndrome or drowning. About 50% of cases are familial: Romano-Ward syndrome (RWS) is a common form of LQTS that exhibits autosomal dominant transmission with low penetrance. Jervell and Lange-Nielsen syndrome (JLNS) is an uncommon form of LQTS, has autosomal recessive transmission, and is associated with congenital deafness. The remainder of cases are sporadic. Asymptomatic (presymptomatic) patients carrying the gene mutation may not all have a prolonged Q-T duration. This may become apparent with exercise or during catecholamine infusions.

Drugs may prolong the QT interval directly (terfenadine, astemizole, cisapride, droperidol), but more often do so when drugs such as erythromycin or ketoconazole inhibit their metabolism.

The clinical manifestation of LQTS in children is most often a syncopal episode brought on by exercise, fright, or a sudden startle; some events occur during sleep (LQT3). Patients can initially be seen with seizures, presyncope, or palpitations; about 10% are initially in cardiac arrest. Treatment of LQTS includes the use of β-blocking agents at doses that blunt the heart rate response to exercise. Some patients require a pacemaker because of drug-induced profound bradycardia. In patients with continued syncope despite treatment, an implantable cardiac

defibrillator is indicated for those who do not respond to β-blocking drugs and those who have experienced cardiac arrest.

**6. MATERIALS FOR METHODOLOGICAL BACKGROUND OF THE WORKSHOP**

**6.1. Quiz**

1. What is the differential diagnosis of supraventricular tachycardia?
2. What are the features of atrial flutter?
3. What are the features of atrial fibrillation?
4. What are the symptoms and signs of supraventricular tachycardia?
5. What are the complications of supraventricular tachycardia?
6. What are the symptoms and signs of supraventricular tachycardia in infants?
7. What are the features of SVT on ECG?
8. What is the WPW syndrome?
9. What are the findings on chest X-ray examination?
10. What is the use of ultrasonography in cardiac arrhythmias?
11. What is the differential diagnosis of syncope?
12. What are the treatment aims in SVT?
13. What vagal maneuvers do you know?
14. What are the diet restrictions in tachyarrhythmia?
15. What is acute treatment in SVT?
16. What is long-term therapy for SVT?
17. What is the prognosis in SVT?
18. What are the follow-up measures in SVT?
19. What are ECG findings in sinus tachycardia?
20. What are ECG findings in atrial fibrillation?
21. What are ECG findings in atrial flutter?
22. What are ECG findings in ventricular tachycardia?
23. What is clinical manifestation of long Q-T syndromes?

**6.2. Multi-choice questions**

What is this?

![Lead 2](image_url)

A. Premature ventricular contraction
B. Premature atrial contraction
C. Sinus arrhythmia with a junctional escape beat*
D. Paroxysmal supraventricular tachycardia
E. Wolff-Parkinson-White (WPW) syndrome
6.3. Sample case report
An 8-year old boy was playing in the school playground when he started vomiting and collapsed. His stepmother was called who took him to the emergency department where his heart rate was recorded 37 beats per minute. On further questioning the stepmother told that the boy had complained of uneasiness in chest which she had ignored. Capillary perfusion rate is increased upto 2 seconds. The child was administered atropine to which he remained unresponsive and a simultaneous ECG revealed: Upright P waves in 1, 2 and 3 leads. Occasionally a P wave is not followed by a QRS complex (2:1). QRS complex is 0.25 sec (widened).

1. What is the most probable cause of this condition?
2. What is the differential diagnosis of the condition?

Suggested reading

Additional reading
2. Caughey RW, Humphrey JM, Thomas PE. High-degree atroventricular block in a child with acute myocarditis.( Ochsner J. 2014 Summer;14(2):244-7) http://www.ncbi.nlm.nih.gov/pmc/articles/PMC4052592/