Acquired conduction disturbance in structurally normal heart in children

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Incidence

- Cardiac conduction disturbances detected in a pediatric population
  - 432,166 elementary and high school students
  - Prevalence of CCD increased with age: 0.48% → 0.97%
  - Higher in males
  - Incomplete RBBB (0.32%), complete RBBB (0.11%), VPC (0.11%), WPW syndrome (0.067%)
  - Sensitivity of IRBBB in screening for ASD was 34.67%

Classification

- First AV Block
- Second AV Block
- Complete AV Block
- Intraventricular block
- Drug-induced long QT syndrome
Causes of acquired heart block

- medications
- myocardial inflammation
- Myopathy
- infection (Lyme disease, viral myocarditis, endocarditis)
- Hypothyroidism
- surgical trauma
- high levels of vagal tone
- Anorexia nervosa
Drug–induced atrioventricular block

- AV block (diagnosed during therapy with antiarrhythmic medication) that resolved when the drug discontinued and never recurred during a follow-up period (≤ 3wks)
- Beta–blockers, nondihydropyridine calcium channel antagonists (verapamil, diltiazem)
Drug–induced atrioventricular block: prognosis: benign?

Zelster et al. JACC 2004;44:105-8
Infectious myocarditis – infectious agent

<table>
<thead>
<tr>
<th>Bacteria</th>
<th>Virus</th>
<th>Parasites</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diphtheria</td>
<td>Epstein-Barr virus</td>
<td>Candidosis</td>
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<tr>
<td>Cholera</td>
<td>Mumps</td>
<td>Aspergillosis</td>
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<tr>
<td>Leptospirosis</td>
<td>Cytomegalovirus</td>
<td>Trichinosis</td>
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<tr>
<td>Mycoplasma</td>
<td>Rubella</td>
<td>Hydatidiosis</td>
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<td>pneumoniae</td>
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<td>Rickettsia</td>
<td>Poliomyelitis</td>
<td>Toxoplasmosis</td>
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<tr>
<td>Streptococcus</td>
<td>Parainfluenza</td>
<td>Paludism</td>
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<td>Meningococcus</td>
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<tr>
<td>Listeriosis</td>
<td>Varicella</td>
<td>Bilharziosis</td>
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<tr>
<td>Staphylococcus</td>
<td>Herpes</td>
<td>Trypanosoma gambiense</td>
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<tr>
<td>Tuberculosis</td>
<td>measles</td>
<td>Cysticercosis</td>
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<td>Syphilis</td>
<td>Arbovirosis</td>
<td>Opisthorchiasis</td>
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<tr>
<td>Shigella</td>
<td>Hepatitis</td>
<td>Paragonimiasis</td>
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<tr>
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<td>Coxsackie B</td>
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<td></td>
<td>Adenovirus</td>
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<tr>
<td></td>
<td>Respiratory syncytial virus</td>
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<tr>
<td></td>
<td>Hantaan virus</td>
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<tr>
<td></td>
<td>Enterovirus</td>
<td></td>
</tr>
</tbody>
</table>

The underlined data are agents for which the implantation of a permanent pacemaker has been reported. (Complete references available on request.)

Maury et al., J of Electrophysiology 2008:41;665–667
Infectious myocarditis

- Acquired AV block in children (even without myocardial systolic dysfunction) → r/o myocarditis

- Biopsy
  - Acute phase: injury of the conduction system, with mitochondrial inclusions, lymphocytes, mononuclear cells infiltrates, areas of inflammation and necrosis
  - Delayed phase: scar fibrosis

  Batra et al. Pediatr Cardiol 2003;24:495–497
Infectious myocarditis

- Electrophysiologic investigations
  - Supra–hisian, intra–hisian, or infra–hisian block
  - Distal lesion: intraventricular block

- Treatment
  - Transient pacing (2/3; recovered at 1 week)
  - Permanent pacemaker
    - 20–30% of the cases

Batra et al. Pediatr Cardiol 2003;24:495-497
Infectious myocarditis

- Clinical course of CHB a/w acute myocarditis (40 patients)
  - Recovery: 67%
    - Average time for recovery: 3.3 ± 2.8 days
    - 1 week of presentation in nearly all cases
  - Temporary pacing: 95% of cases
  - Permanent pacemakers in 27% (after 1 week)
  - Immunosuppression?

Batra et al. Pediatr Cardiol 2003;24:495–497
Rheumatic fever

- Asymptomatic rhythm and conduction abnormalities in children with acute RF
  - 64 children, 24 hr electrocardiography
  - First-degree AVB: 21.9%
    - Not related to the presence of carditis
  - Mobitz type I AVB: 1 (1.56%)
  - Rheumatic affection of atrioventricular conduction is proximal to the trifascicular system?

Karacan et al. Cardiology in the Young 2010;20:620–630
Kawasaki disease

- T wave change, prolonged PR, QT interval, complete heart block
- Most prevalent in the first month of disease
- Do not predict the type of echocardiographic abnormalities

Duchenne’s muscular dystrophy

- High incidence of ECG abnormalities in young pts with Duchenne’s MD
  - 69 pts, aged ≤ 18 yrs
  - Deep Q waves, low RV5 + SV1 (91.3% of pts)
  - Initial and primary sites of myocardial dystrophy: posterobasal and contiguous left ventricular wall as
  - Dystrophin gene deficiency

Arrhythmia & obstructive sleep apnea
Arrhythmia & obstructive sleep apnea

- a/w the number of apneic episodes and the severity of hypoxemia
- Nocturnal arrhythmia: 50% of pts
- Nonsustained VT, sinus arrest, second-degree AVB, PVC
- Prolonged apnea and hypoxemia → diving reflex → cardiac vagal activation → bradycardia, AVB and PVC
## Cardiac Arrhythmia or Conduction Abnormality in 50 Patients With Sleep Apnea Syndrome Before and After Tracheostomy

<table>
<thead>
<tr>
<th>Cardiac Arrhythmia or Conduction Abnormality</th>
<th>Before Tracheostomy</th>
<th>After Tracheostomy</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Awake</td>
<td>Asleep</td>
</tr>
<tr>
<td>Sinus arrest 4 to 13 s</td>
<td>0</td>
<td>15</td>
</tr>
<tr>
<td>Second-degree atrioventricular block</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mobitz type I</td>
<td>0</td>
<td>5</td>
</tr>
<tr>
<td>Mobitz type II</td>
<td>0</td>
<td>10</td>
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<tr>
<td>Ventricular tachycardia</td>
<td>0</td>
<td>8</td>
</tr>
<tr>
<td>Atrial flutter</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>Atrial fibrillation</td>
<td>0</td>
<td>8</td>
</tr>
<tr>
<td>Extreme sinus bradycardia</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>Frequent premature ventricular contractions</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Obesity

- Cardiac conduction system involvement in sudden death of obese young people
  - 7 patients, 5 obese and 2 mild to moderately obese
  - 6~32 years of age
  - 3 patients: OSA
  - Pathology
Obesity

**Fig. 1.** Patient 1. Focal accumulation of mononuclear cells in approaches to SA node. AP, Approaches to the SA node. *Arrow* point to accumulation of mononuclear cells. (Hematoxylin-eosin stain—original magnification ×200.)

**Fig. 6.** Patient 3. Arteriolosclerosis of AV node with fatty infiltration in adjacent ventricular septum. N, AV node; C, central fibrous body; F, fat in central fibrous body and ventricular septum; V, ventricular septum. *Arrow* points to...
Obesity

Fig. 5. Patient 3. Remnant of SA node with fibrosis and fatty infiltration. SA, Sinoatrial node; F, fat; FI, fibrosis. (Hematoxylin-eosin stain; original magnification x40.)

Fig. 2. Patient 1. Right ventricular infundibular muscle pressing on branching bundle, with focal fibrosis of branching bundle, mid part of left bundle branch, and ventricular septum. I, Infundibular septal bulge pressing on the branching bundle; B, branching bundle; LBB, mid part of left bundle branch. Arrows point to fibrosis. Weigert–van Gieson stain; original magnification x24.)
Obesity

- Pathologic findings of conduction system were more marked in patients with obesity of long-standing duration and a history of obstructive sleep apnea.
- May produce arrhythmias that may be silent in nature but may form a milieu for an arrhythmic event that end fatality during an altered physiologic state.

Anorexia nervosa

- Bradycardia, hypotension, prolonged QTc
  - 12 yr old girl
  - Second-degree AV block (Mobitz type I)
  - Intrinsic or complication of AN?

QTc

- Predisposing factors to QT prolongation
  - Age, female gender, LVH, heart failure, myocardial ischemia, hypertension, DM, increased thyroid hormone concentration, elevated serum cholesterol, high BMI, slow HR, electrolyte abnormalities (hypokalemia, hypomagnesemia), drugs
Long QT syndrome – medications

- Class IA and III antiarrhythmics
- Antibiotics (macrolides and quinolones)
- Antidepressants (tricyclics and selective serotonin reuptake inhibitors)
- Antipsychotics (haloperidol and phenothiazines)
- Antiemetics (ondansetron and prochlorperazine)
LQTS – medication

- **Mechanism**
  - Inhibition of the KCNH2-encoded HERG potassium channel → prolongation of the action potential duration and a prolonged QT interval
  - 10%: quiescent LQTS-susceptibility mutations
  - Risk factors: advanced age, female gender, hypokalemia, bradycardia, hypomagnesemia

Ayad et al. Proc(Bayl Univ Med Cent) 2010;23(3):250–255