CARDIAC ARRHYTHMIAS
INTRODUCTION

• Disturbance in heart rate or rhythm.

• Result of abnormal impulse initiation or conduction

• Recognition is challenging.

• Precise diagnosis is important.
CLASSIFICATION

• According to site of origin -
  – Atria
  – AV junctional
  – Ventricle

• According to speed of conduction -
  – Abnormally fast
  – Abnormally slow
  – Delayed -Premature beats, Escape beats

• According to rate -
  – Brady arrhythmias
  – Tachyarrhythmia's
SYMPTOMS

- Chest pain
- Palpitation
- Syncope
- Shortness of breath
- Excessive sweating
- Neurologic change
HISTORY TAKING

- Duration of illness & triggering factor
- Relationship to exercise, meals, stress
- Medical history- H/O of tachycardia, cardiac problems
- Prior medications, toxins
- H/O allergies
- Sudden death in the family, cardiac disease
INVESTIGATIONS

• 12 lead ECG, cardiac rhythm strip, CBC
• X-ray chest, echocardiography
• Electrolyte- potassium, glucose, calcium, magnesium
• Toxicology screen
• Arterial blood gases
• 24 hrs ambulatory Holter monitoring
• Electro physiological studies
SINUS RHYTHMS

• Passage of a normal depolarization wave
• Heart rate in NB: 110-160
  2 yrs: 85-125
  4 yrs: 75-115
  Over 6 yrs: 60-100
• Rates faster or slower called-Tachy/ Bradycardia
SINUS TACHYCARDIA

• Benign and asymptomatic
• Increase in HR beyond the upper limit for that age
• Reduction in parasympathetic tone
• Increase in sympathetic stimulation
ETIOLOGY

• Normal physiological situations
• Increased catecholamine release during –stress, fright, flight, anger
• Fever, anxiety
• Hypoxia, dehydration, hypovolemia, hypotension, shock, heart failure, sepsis, myocarditis
DIAGNOSIS

• ECG – rhythm is regular- P waves are upright- PR interval 0.12 to 0.2 seconds, shortens with increase in HR

• More than 160 per minute- P wave may be difficult to distinguish from previous T wave

• Treat the underlying cause
SINUS BRADYCARDIA

- Rate less than 80bpm for infants, 60bpm in older

- Benign- often appears in sleep, players, athletes,

- Digitalis, beta blockers, raised ICT, hyperkalemia, hypercalcemia, hypoxia

- Treat underlying cause
SINUS RHYTHM

Regular Sinus Rhythm

Sinus Tachycardia

Sinus Bradycardia

Sinus Arrhythmia

Sinus Pause
SINUS ARRHYTHMIA

- Beat to beat variation in HR, irregular
- HR faster during inspiration & slow in expiration
- Normal in infants and young children
PREMATURE BEATS (ECTOPIC BEATS)

• A focus stimulates early contraction of myocardium

• Atrial ectopic: Normal ventricular complex

• Ventricular ectopic: Broad QRS
ATRIAL ECTOPICS

• In healthy neonates

• Digoxin toxicity, post cardiac surgery

• P wave may be upright in high atrial ectopic
ATRIAL ECTOPIC BEAT

DOUBLE ATRIAL ECTOPIC
TACHYARRHYTHMIA

• **Narrow complex tachycardia** (QRS <0.09sec)
  – Sinus tachycardia,
  – Supraventricular tachycardia (SVT)
  – Atrial flutter

• **Wide complex tachycardia** (QRS >0.09sec)
  – Ventricular tachycardia (VT)
  – SVT with aberrant intraventricular conduction
SUPRAVENTRICULAR TACHYCARDIA

• Commonest arrhythmia in children

• Narrow complex tachycardia at a rate of >220/min

• Accessory pathway is the most common mechanism of SVT in infants.
• Poor feeding, rapid breathing in young children
• Palpitation, chest discomfort in older children
• Most children have normal hearts
• May occur with Ebstein’s anomaly, corrected TGA etc.
SUPRAVENTRICULAR TACHYCARDIA
EVALUATION & INITIAL MANAGEMENT OF SVT

- History, physical exam, ECG, Echo (later)

- Child who looks ill with SVT assessed rapidly for shock, CCF
## DISTINGUISHING FEATURES BETWEEN SVT & ST

<table>
<thead>
<tr>
<th>Criterion</th>
<th>Supraventricular Tachycardia</th>
<th>Sinus Tachycardia</th>
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</thead>
<tbody>
<tr>
<td>Heart rate</td>
<td>&gt;220/minute</td>
<td>&lt;180/minute</td>
</tr>
<tr>
<td>Heart rate variability</td>
<td>No marked variation</td>
<td>Marked variation present</td>
</tr>
<tr>
<td>Surface ECG</td>
<td>P wave absent or abnormal if detected</td>
<td>P wave normal if detected</td>
</tr>
<tr>
<td>Identifiable cause</td>
<td>Not obvious</td>
<td>Obvious (e.g., sepsis, fever)</td>
</tr>
</tbody>
</table>
MANAGEMENT OF SVT

• **Asymptomatic children**
  – Vagal maneuvers such as cold ice placement over face

• **Symptomatic children**
  – Adenosine
  – 0.05- 0.1mg/kg rapid push; increase by 0.05mg/kg per dose;
  – Given by rapid two syringe flush technique
ATRIAL FLUTTER

- Atrial rate around 300/min with ventricular rate of 150/min

- 12 lead ECG of typical AF- (saw tooth) waves are seen in limb leads II, III and aVF
TREATMENT OF ATRIAL FLUTTER

• To control ventricular rate: verapamil, digoxin or diltiazem

• To restore sinus rhythm: Quinidine or procainamide

• To prevent recurrences: Radiofrequency Ablation
ATRIAL FIBRILLATION

• Irregularly irregular radial pulse with pulse deficit

• Paroxysmal or persistent

• Causes (markedly enlarged LA):
  – Rheumatic mitral valve disease,
  – Tricuspid atresia,
  – Ebstein’s anomaly,
  – Myocarditis
ATRIAL FIBRILLATION WITH FIBRILLATORY WAVES AND IRREGULARLY IRREGULAR QRS COMPLEX
TREATMENT OF ATRIAL FIBRILLATION

• **Ventricular rate control:**
  – AV nodal blocking agents: β-blocker, Ca channel blockers

  – AV junction ablation and pacemaker insertion: those who fail drugs

• **Ventricular rhythm control:**
  – Antiarrhythmic drugs
VENTRICULAR DYSRHYTHMIA

• Premature ventricular contraction

• Ventricular tachycardia
PREMATURE VENTRICULAR CONTRACTION

- Isolated PVC’s frequently observed in normal infants and adolescents (10-15% in infants: 20-25% adolescents)

- Benign PVC’s of single QRS morphology and easily suppressed by exercise: no treatment needed

- If frequent PVC’s: serum electrolytes, Echo, 24 hr Holter monitoring
VENTRICULAR TACHYCARDIA

• Can be life threatening

• Wide QRS complex

• Reentry of downward propagating re-polarization that causes the signal to repeat itself.
MANAGEMENT OF WIDE QRS TACHYCARDIA
VENTRICULAR FIBRILLATION

<table>
<thead>
<tr>
<th>Heart Rate</th>
<th>Rhythm</th>
<th>P wave</th>
<th>PR Interval</th>
<th>QRS Complexes</th>
</tr>
</thead>
<tbody>
<tr>
<td>300-600</td>
<td>Extremely irregular</td>
<td>Absent</td>
<td>NA</td>
<td>Fibrillatory Baseline</td>
</tr>
</tbody>
</table>
MANAGEMENT OF VF

• Immediate CPR and artificial ventilation

• DC electric defibrillation 2 J/kg upto 4 J/kg- if recurs +IV amiodarone

• Treat precipitating causes

• Refractory causes: permanent implantable automatic cardioverter- defibrillator
BRADYARYRTHMIAS

• Failure of impulse generation or impulse propagation

Causes:
• Drugs: - Digoxin, B-blockers , Ca channel blockers
  - tricyclic antidepressants, lithium
• Organophosphate pesticides
• Plant toxin- oleander
ATRIOVENTRICULAR BLOCK

- 1st degree block
- PR interval >0.20 seconds
- Normal sinus rhythm: normal QRS: no dropped beats
- May be seen normally: also in myocarditis (rheumatic and infective), cardiomegaly, ASD
IIIND DEGREE BLOCK (MOBITZ I & II)
TYPE I WENCKEBACH PHENOMENON

• Progressive increase in PR until a QRS dropped-
  Usually every 3-6 cycles
• Can be seen in myocaditis, CHD’s, digoxin toxicity,
  post op. (cardiac)
MOBITZ TYPE II (CONSTANT 2:1 OR 3:1) AV BLOCK

• All or none phenomenon: Either a normal conduction with normal PR or completely blocked conduction
IIIRD DEGREE (COMPLETE) AV BLOCK

• P- completely dissociated from QRS (AV dissociation)
• PP & RR interval regular
• Congenital: Newborn to mothers with SLE
• Acquired: myocarditis, rheumatic fever, Kawasaki, SLE, hypocalcemia
TREATMENT OF HEART BLOCK

• No treatment for I & II degree block: evaluate and treat underlying disorder

• Mobitz type II may progress to complete block—prophylactic pace maker

• Complete heart block: Atropine or isoproterenol till permanent pace maker implantation
Thank You