• Anatomy
• Electrocardiograms
• Conduction defects
• Arrhythmias
• Medical therapy
• Indications for pacing
Normal Sinus Rhythm

- SA node begins impulse that spreads, stimulating both atria (P Wave)
- Impulse reaches and pauses in AV node
- Impulse passes in HIS bundle and branches, then through the Purkinje fibers to the myocardial cells (QRS)
- Repolarization of the ventricular cells (T wave)
General Concepts

- **PR Interval**
  - Onset of P wave to beginning of QRS

- **QRS Duration**
  - Onset of Q wave to termination of the S wave

- **QT Interval**
  - Measured from onset of Q wave to end of T wave
Systematic Approach

- Heart Rate
- Rhythm (sinus or non-sinus)
- QRS Axis
- Intervals: PR, QRS, QT
- P Wave Amplitude and Duration
- QRS Amplitude
- ST Segments and T Wave Abnormalities
Heart Rate

- 25mm = 1.0s (5 large boxes)
- 5mm = 0.20s (1 large box)
- 1mm = 0.04s (1 small box)
- Easy to memorize 300, 150, 100, 75, 60... with each big box or
- 300/# of large boxes between RR interval; 1500/# small boxes between RR interval
- Normal rhythm is sinus rhythm
- SA node is pacemaker
- P wave in front of each QRS complex
- P axis range of 0-90° (upright in leads I and aVF)
- Abnormal or non-sinus rhythm is suggested if abnormal #, shape or axis of P waves
P Wave

- **P Amplitude**
  - Measure in Lead II
  - Usually 1.5mm, Max 3.0mm
  - >3.0 mm RAE

- **P Duration**
  - Time required for depolarization of atria
  - Normal 0.06 ± 0.02 sec, Max .10 sec
  - Prolongation seen in LAE
QRS Axis

- Represents the mean vector of the ventricular depolarization process in frontal plane
- NB usually has RAD
  - NB  +125° (up to 180°)
  - 1 month  +90°
  - 3 years  +60°
-ve QRS in aVF (superior axis) and -ve lead I with rS in aVL, severe RAD

qR pattern in leads I and aVL is LAD (think AV canal or tricuspid atresia)

RAD (normal in newborn)
RVH
RBBB

+ QRS in I / aVF Axis 0 to +90 (normal in adult)
QRS Duration

- Time required for ventricular depolarization
- Premature infants: 0.04 sec
- Term infants: 0.05 sec
- If Increased QRS duration: ventricular conduction disturbance
  - In infants, QRS > 0.08 is RBBB
  - QRS: prolonged in ventricular hypertrophy
  - Consider WPW
• Abnormally large deflections
  - Ventricular Hypertrophy
  - Ventricular conduction disturbances

• Low voltage QRS complexes (< 5mm)
  • Normal newborn
  • Pericardial effusion
  • Myocarditis
  • Hypothyroidism
Time for atrial depolarization and physiologic delay of impulse in AV node

- **PR prolongation (1° AV block)**
  - Myocarditis
  - CHD (AVSD, ASD, Ebstein’s)
  - Digoxin toxicity
  - Hyperkalemia
  - RAE
  - Normal

- **Short PR interval**
  - WPW
  - GSD

- **Variable**
  - Wandering atrial pacemaker
  - 2° AV block, Type I
• Represents the time of both ventricular depolarization and repolarization
• Varies with HR but not with age (except in infancy)
• QT is inversely proportional to HR
• \( QTc = \frac{QT}{\sqrt{RR}} \)
• QTc up to .49s may be normal in first 6m
Prolonged QTc

- Hypocalcemia
- Myocarditis
- Long QT syndrome
  - Romano-Ward syndrome
  - Jervell-Lange-Neilsen

Short QT interval
- Hypercalcemia
- Digoxin effect

- Head injury or CVA
- Diffuse myocardial disease
Q waves represent septal depolarization

Provided there is a ventricular septum, direction of depolarization is from embryonic L to R ventricle

Normally, direction of septal depolarization is to the right producing Q waves in leads I, V6, V7

If “left” ventricle is to the right of the RV, septal depolarization will be to the left
  - Q in V3R and V1
  - Q absent in leads I, V6, V7
Abnormal ST Segment

- Pericarditis
- Myocarditis
- Acute MI or Ischemia
- Hyperkalemia or hypokalemia
- Severe ventricular hypertrophy ("strain")
- Drug effect (Digitalis)
- Intracranial pathology
• T-waves represent ventricular repolarization
• In infants, usually less than 11 mm in V6
• Tall peaked T waves may be seen in
  - Hyperkalemia
  - LVH (volume overload)
  - CVA
• Flat or low T waves
  - Normal
  - Hypothyroid
  - Hypokalemic
  - Myocarditis or pericarditis
Atrial enlargement

• Changes with atrial enlargement are increases in amplitude and/or duration of the P wave
  - **RAE**
    • Tall P waves, > 3mm in any lead (lead II)
  - **LAE**
    • Prolonged P duration, > 0.10s in any lead
    • A broad notched P wave in the limb lead is characteristic but still requires prolongation
Ventricular hypertrophy

Should see an increase in the voltage of the QRS complex in the leads that reflect the respective ventricle

**RVH**
- RAD for age
- Increased right and anterior QRS vector
  - >R in V3R, V1, V2 or aVR
  - >S in I or V6
- Upright T in V1 > 3 days
- Q wave in V1

**LVH**
- LAD for age
- Increased ORS voltages in LV
  - >R in I, II, III, aVL, aVF, V6, V7
  - >S in V3R or V1
- Pressure Overload
- Volume Overload
- Deep Q in V6, V7
Electrolyte Abnormalities

**Potassium**
- $< 2.5 \text{ mEq/L}$: Depressed ST Segment, Diphasic T Wave, Prominent U Wave
- Normal
- $> 6.0 \text{ mEq/L}$: Tall T Wave
- $> 7.5 \text{ mEq/L}$: Long PR Interval, Wide QRS Duration, Tall T Wave
- $> 9.0 \text{ mEq/L}$: Absent P Wave, Sinusoidal Wave

**Calcium**
- Hypercalcemia
- Normal
- Hypocalcemia
Normal Newborn ECG

- PR interval $< 0.11$ Second
- QRS $+ 45$ to $+ 180$ degrees
- V3R and V1 Rs
  - R wave $< 15$ mm
  - Negative T $> 3$ days
- V6 and V7 qrS
Top Ten Neonatal Arrhythmias
Neonatal Arrhythmias

10. Junctional ectopic tachycardia
Junctional ectopic tachycardia

- Usually a postoperative arrhythmia, rarely congenital
  - high catecholamine state
  - increased sympathetic tone
- Rapid rate
- Regular rhythm
  - unless competing sinus rate
- AV dissociation
  - look for the “p”
9. Ventricular tachycardia
10. Junctional ectopic tachycardia
Ventricular Tachycardia

- Three or more “consecutive PVC’s”
- QRS morphology
  - Different from sinus
  - Not necessarily wide
    - May be 70-80 msec in infants
    - Usually has a LBBB pattern in infancy
  - May be monomorphic or polymorphic (e.g., “torsades de pointes”)
Ventricular tachycardia

Length 4 beats (227 bpm) - Sep-2007 10:40:35
Neonatal Causes: Metabolic

- Acidosis
- Hypoxemia
- Hypokalemia
- Hyperkalemia
- Hypercalcemia
- Hypoglycemia
- Hypothermia
Neonatal Causes: Drugs

- Digoxin
- Quinidine
- Flecainide
- Sotalol
- Sympathomimetics
- Cocaine
- Halothane
Neonatal Causes: Cardiac

- Post-operative ventriculotomy
- Myocarditis
- Cardiomyopathy
- Arrhythmogenic RV dysplasia
- Long QT syndrome
- Ventricular tumors
- Bacterial Endocarditis
- Kawasaki disease
8. Atrial ectopic tachycardia
9. Ventricular tachycardia
10. Junctional ectopic tachycardia
Atrial ectopic tachycardia

- May go unrecognized in neonates
  - unless multifocal or chaotic atrial tachycardia
- Abnormal firing of atrial cells other than sinus node
- Usually fast; may have hemodynamic instability
  - Clinically may be mistaken for sepsis
- Usually difficult to treat medically
3 week old infant male presents with irregular heart rate. Diagnosis: Multifocal atrial tachycardia with variable AV block.

Same infant develops bradycardia after antiarrhythmic therapy began. Diagnosis: Atrial flutter with 6:1 AV block.
Neonatal Arrhythmias

7. Atrial flutter
8. Atrial ectopic tachycardia
9. Ventricular tachycardia
10. Junctional ectopic tachycardia
Atrial flutter

- **Atrial rate/rhythm**
  - **Rapid**
  - **In neonates: 500/min**
  - **Regular**
    - Sustained
    - “sawtooth”

- **Ventricular rate/rhythm**
  - Regular or irregular
  - Variable AV conduction
Atrial Flutter

“The syphilis of cardiac arrhythmias”

- 1:1 conduction $\rightarrow$ ? SVT
- 1:1 with WPW $\rightarrow$ ? VT
- Flat flutter waves $\rightarrow$ ? AET or Afib
- 2:1 - 3:1 AV conduction $\rightarrow$ ? NSR or AET

MUST BE SUSPICIOUS !!!!

Don’t trust post-op CHD patients !!!!
Transesophageal overdrive pacing converts atrial flutter to NSR
Neonatal Arrhythmias

6. Complete AV block
7. Atrial flutter
8. Atrial ectopic tachycardia
9. Ventricular tachycardia
10. Junctional ectopic tachycardia
• Independent “p” and QRS
  - “p rate” > “QRS rate”
  - “p” wave that should conduct, doesn’t
  - Ventricular rate remains regular

AV Dissociation ≠ Complete AV Block
Complete AV Block

Congenital
• In utero diagnosis
• Prolonged QTc in 20%
• Associated with maternal lupus

Acquired
• Postoperative
• Autoimmune
• Infectious (rare)
  - Viral
  - Borrelia
  - Mycoplasma
Congenital Complete AV Block

- Occurs in 1/22,000 live births, 1/60 SLE mothers
  - Maternal collagen vascular disease (SLE, RA)
    - SS anti-Ro and anti-La antibodies (75%)
    - Cross placenta and enter fetal circulation as early as the 16th-23rd week gestation
  - Structural heart disease
    - Abnormal development of the AV node
      - Ventricular inversion with L-TGA
Complete AV Block

- **Clinically**
  - Asymptomatic
  - Symptomatic
    - Decreased C.O.
    - CHF
      - Hydropic
      - Anarsarca
      - Hepatomegaly
      - Acidotic

- **Therapy**
  - Treat symptoms
  - “Pace me now or pace me later”
  - More aggressive with symptoms or heart defects
Management:
- Temporary AV pacing
- Difficult in young children
- 50% resolve by the 8th post operative day

If AV conduction resumes, but still have 2:1 or Mobitz II, permanent pacemaker is indicated
5. Supraventricular tachycardia
6. Complete AV block
7. Atrial flutter
8. Atrial ectopic tachycardia
9. Ventricular tachycardia
10. Junctional ectopic tachycardia
• Many mechanisms with subtle EKG differences
• Look for the p wave
  - At fast rates “p” waves may not be visible
  - Provides clues to potential mechanisms
• Therapy
  - Generally not urgent
  - Hemodynamic compromise?
Wolff-Parkinson-White syndrome
1 month old with severe metabolic acidosis
SVT + Adenosine
Neonatal Arrhythmias

4. Premature ventricular contractions
5. Supraventricular tachycardia
6. Complete AV block
7. Atrial flutter
8. Atrial ectopic tachycardia
9. Ventricular tachycardia
10. Junctional ectopic tachycardia
Premature ventricular contractions

- May occur in 17% premies; 4% full-term neonates
  - All infants with PVCs on ECG, need Holter
- Usually disappear in the first 4-8 weeks
  - If persists, obtain an echocardiogram
- Must rule out: long QT and myocarditis
- Other causes: cardiomyopathy or ventricular tumors
Premature ventricular contractions

- Early beat with wide QRS without preceding P wave
- T wave axis is directly opposite QRS
- Followed by compensatory pause (P wave interval double sinus interval)
3. PAC’s / Wandering atrial pacemaker
4. Premature ventricular contractions
5. Supraventricular tachycardia
6. Complete AV block
7. Atrial flutter
8. Atrial ectopic tachycardia
9. Ventricular tachycardia
10. Junctional ectopic tachycardia
Premature atrial contractions

- Premature beats arising in the atrium
- May occur in 33% premies; 14% full-term
- Conduction may be normal, aberrant or blocked
- If frequent, may induce atrial flutter/fib
- Improves in weeks; usually resolves in 6-12 months
PACs - aberrant and blocked
Premature atrial contractions

Etiology in healthy infants

• Increased vagal tone
  - Retards conduction throughout atrium
  - Atrial cell irritability
  - Abnormal firing probably a result of incomplete atrial stimulation
  - Usually resolves at faster heart rates
• Decreased vagal tone
Premature atrial contractions

Acute causes

- Mechanical factors, e.g. central line
- Hypokalemia
- Hypercalcemia
- Hypoxia
- Hypoglycemia
- Drugs- Digoxin, sympathomimetics
Premature atrial contractions

Approach in healthy infants

• If \( \leq 2 \) (occasional) PACs on ECG, repeat at 2 month check

• If \( \geq 3 \) (frequent) PACs on ECG, obtain 24-hour Holter monitor
  - If frequent PACs, repeat Holter in 2 months
  - If occasional PACs, repeat Holter in 6 months
  - If SVT, consult Pediatric Cardiology
Wandering atrial pacemaker

- **Atrial-based rhythm**
  - Non-dominant sinus node
  - Varying p-wave morphologies
  - Varying atrial rate

- **All QRS’s preceded by p waves**

- **Occurs at slower heart rates; resolves at faster heart rates**

- **Related to increased vagal tone**
2. Sinus bradycardia/sinus arrhythmia
3. PAC’s / Wandering atrial pacemaker
4. Premature ventricular contractions
5. Supraventricular tachycardia
6. Complete AV block
7. Atrial flutter
8. Atrial ectopic tachycardia
9. Ventricular tachycardia
10. Junctional ectopic tachycardia
Sinus Bradycardia/Escape Rhythms

- **What is slow (sinus)?**
  - Infant - < 70 bpm
  - Child - < 50 bpm
  - Teen - < 40 bpm

- **Are “p”-waves present?**
  - Yes - sinus or atrial
  - No - “narrow” = junctional
    “wide” = ventricular
Sinus Bradycardia/Escape Rhythms

Neonatal Causes

- Digoxin
- Abdominal distension
- Hypoxia
- Acidosis
- Increased ICP
- Hypothyroidism
- Low glucose
- Low calcium
- Beta-blockers
- Increased vagal tone
- Most anti-arrhythmics
- Post-op atrial surgery
- Hypertension
Things To Remember

• Infants
  - Sinus pauses - 72% full term infants
  - Rule out “blocked” PACs

• All patients
  - Check corrected QT interval (QTc)
    • Long QT syndrome
    • QTc = QT/square root of RR interval
    • QTc < 0.46
Sinus Bradycardia

Wenckeback

Progressive PR prolongation
Dropped sinus beat
Short “recovery” PR
Neonatal Arrhythmias

1. **Sinus tachycardia**
2. Sinus bradycardia
3. PAC’s / Wandering atrial pacemaker
4. Premature ventricular contractions
5. Supraventricular tachycardia
6. Complete AV block
7. Atrial flutter
8. Atrial ectopic tachycardia
9. Ventricular tachycardia
10. Junctional ectopic tachycardia
Sinus tachycardia
Sinus Tachycardia

- Occurs in 99% full-term infants
- “PR” < “RP” on EKG
- Still “sinus” at highest rates
  - Infant: 220-230 bpm
  - Child: 200 bpm
  - Teen: 180 bpm
Sinus Tachycardia

Neonatal Causes

- Crying
- Fever
- Infection
- Hyperthyroidism
- CHF
- Anemia
- Myocarditis
- Hypovolemia
Top Ten Neonatal Arrhythmias

1. Sinus tachycardia
2. Sinus bradycardia
3. PAC’s / Wandering atrial pacemaker
4. Premature ventricular contractions
5. Supraventricular tachycardia
6. Complete AV block
7. Atrial flutter
8. Atrial ectopic tachycardia
9. Ventricular tachycardia
10. Junctional ectopic tachycardia
Common Neonatal Arrhythmias

Neonatal arrhythmias can begin as fetal arrhythmias.
Fetal Echocardiography

M-mode

Complete Heart block
SVT Requirements for Reentry

Zone of slow conduction

Unidirectional block

Heart beat
PAC conducts in slow pathway

PAC blocks in fast pathway due to shorter ERP
Fast pathway recovers.
PAC conducts retrograde and “reenters”, producing SVT.
Temporary pacing

- Supportive for bradycardia, CHB or AV dysynchrony
- Rapid overdrive pacing of re-entry tachycardia
- Wires: usually for up to 2 weeks
- Check thresholds daily
- Check battery and change every other day
- Keep fresh battery supply
- Check underlying rhythm daily
  - Decrease rate gradually
Indications for permanent pacing

- Persistent postoperative CHB
- Late onset postoperative CHB
- Sinus node dysfunction
  - HR<55
  - Minimum HR is 30/min
  - Pauses>3.5sec
- If need therapy for SVT/ VT and sinus node dysfunction
<table>
<thead>
<tr>
<th>Chamber paced</th>
<th>Chamber Sensed</th>
<th>Response to sensing</th>
</tr>
</thead>
<tbody>
<tr>
<td>O= none</td>
<td>O= none</td>
<td>O= none</td>
</tr>
<tr>
<td>A= Atrium</td>
<td>A= Atrium</td>
<td>I= Inhibited</td>
</tr>
<tr>
<td>V=Ventricle</td>
<td>V=Ventricle</td>
<td>T= Triggered</td>
</tr>
<tr>
<td>D= Dual</td>
<td>D= Dual</td>
<td>D= Dual</td>
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</tbody>
</table>
Pacemaker terminology

- AOO/ VOO: pace atrium or ventricle at set rate
- AAI: atrial pacemaker if normal AV conduction
- VVI: Ventricular back up
- DDD: Paces and senses A / V
<table>
<thead>
<tr>
<th>Vaughan-William classification</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>IA</strong> Marked inhibition of Na</td>
</tr>
<tr>
<td>Prolong repolarization</td>
</tr>
<tr>
<td><strong>IB</strong> Moderate inhibition of Na</td>
</tr>
<tr>
<td>Shorten repolarization</td>
</tr>
<tr>
<td><strong>IC</strong> Mild inhibition of Na</td>
</tr>
<tr>
<td>Variable repolarization</td>
</tr>
<tr>
<td><strong>II</strong> Sympathetic blockade</td>
</tr>
<tr>
<td><strong>III</strong> Prolonged repolarization</td>
</tr>
<tr>
<td><strong>IV</strong> Calcium blockade</td>
</tr>
<tr>
<td>Others</td>
</tr>
</tbody>
</table>
• Short acting, cardioselective B-blocker
• Effective in catecholamine sensitive state
• Slows sinus rate and AV conduction
• Little effect on accessory pathways
• Reduction in BP in 50% of patients
• Dose: 200 mcg/kg/min IV. Titrate by 50–100 mcg/kg/min every 5–10 minutes, until 10% reduction in HR or BP
Amiodarone

- Safety not established in children
- Prolongs monophasic action potential
- Slow oral absorption
- Dose: 10–15 mg/kg/day PO QD or BID for 10 days, then 300 mg/1.73 m2 PO QD.
- IV dose 5 mg/kg IV bolus over 1 hour
- S/E hypotension, bradycardia, thyroid, pulmonary fibrosis, hepatic, optic neuropathy. May need pacing.
Adenosine

- Endogenous purine nucleotide
- Transient AV nodal conduction block
- Rapid IV bolus
- T1/2 6 to 15 sec
- Therapeutic and diagnostic
- S/E apnea, bronchospasm and arrhythmia, prolonged asystole or VT
ECG and Rhythm Questions
What is the diagnosis?
What do you do next?
• Represents the time for both ventricular depolarization and ventricular repolarization

• Varies with HR but not with age (except in infancy)

• \( QTc = \frac{QT}{\sqrt{RR}} \)

• \( QTc \) should not exceed 0.44s, except in infants
Prolonged QTc

- Hypocalcemia
- Myocarditis
- Long QT syndrome
  - Jervell-Lange-Neilsen syndrome
  - Romano-Ward syndrome
- Head injury or CVA
- Diffuse myocardial

Short QT interval

- Hypercalcemia
- Digoxin effect
Q Wave

- Represent depolarization of the ventricular septum
- Present in II, III, aVF, V5, V6
- Absent in V3R and V1
- Usually < 5mm in aVF, V5, V6

- If no Q waves in V6, V7 think:
  - L-TGA
  - Single Ventricle
  - Mirror image dextrocardia
  - LBBB

- If Q waves present in V3R, V1 think:
  - Severe RVH
  - L-TGA, Single Vent
  - Occasional normal NB
• If deep Q waves in left leads think:
  - LVH volume overload
  - BVH
  - Myocardial disease
  - Restrictive CM

• If deep and wide Q waves think:
  - infarction
  - IHSS
• In adults and children more than 3 yo there is smooth progression in the precordial leads from rS in V3R and V1, through RS in V2, V4 and qRs in V6, V7
• In infants, there may be a complete reversal
• Deviation from the norm may be seen in:
  - Ventricular Hypertrophy
  - Ventricular conduction disturbances
  - Single ventricle
• After ventricular depolarization and before repolarization
• Normal ST segment is horizontal and isoelectric
• Limb leads: 1mm elevation or depression may be normal
• Left precordial leads: up to 2 mm maybe normal

Normal J-depression
Abnormal ST segment changes
Fetal Echocardiography
M-mode, PAC

Right Atrium
Left Ventricle

PAC