EKG Boot Camp

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Objectives

- The Basics
- Interpretation
- Clinical Pearls
- Practice Recognition
The Normal Conduction System
11 Step Method for Reading EKG’s

“Data Gathering” – steps 1-4

• 1. Standardization – make sure paper and paper speed is standardized
• 2. Heart Rate
• 3. Intervals – PR, QT, QRS width
• 4. Axis – normal vs. deviation
11 Step Method for Reading EKG's

“Diagnoses”
– 5. Rhythm
– 6. Atrioventricular (AV) Block Disturbances
– 7. BundleBranch Block or Hemiblock
– 8. Preexcitation Conduction
– 9. Enlargement and Hypertrophy
– 10. Coronary Artery Disease
– 11. Utter Confusion
Lead Placement
Rate

- Rule of 300-
  Divide 300 by the number of boxes between each QRS = rate

<table>
<thead>
<tr>
<th>Number of big boxes</th>
<th>Rate</th>
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<tbody>
<tr>
<td>1</td>
<td>300</td>
</tr>
<tr>
<td>2</td>
<td>150</td>
</tr>
<tr>
<td>3</td>
<td>100</td>
</tr>
<tr>
<td>4</td>
<td>75</td>
</tr>
<tr>
<td>5</td>
<td>60</td>
</tr>
<tr>
<td>6</td>
<td>50</td>
</tr>
</tbody>
</table>
What is the heart rate?

(300 / 6) = 50 bpm
Normal Intervals

- PR
  - 0.20 sec (less than one large box)
- QRS
  - 0.08 – 0.10 sec (1-2 small boxes)
- QT
  - 450 ms in men, 460 ms in women
  - Based on sex / heart rate
  - Half the R-R interval with normal HR
Prolonged QT

• Normal
  - Men 450ms
  - Women 460ms

• Corrected QT (QTc)
  - QTm/√(R-R)

• Causes
  - Drugs (Na channel blockers)
  - Hypocalcemia, hypomagnesemia, hypokalemia
  - Hypothermia
  - AMI
  - Congenital
  - Increased ICP
The QRS Axis

- Represents the overall direction of the heart’s activity
- Axis of –30 to +90 degrees is normal
The Quadrant Approach

- QRS up in I and up in aVF = Normal

<table>
<thead>
<tr>
<th>Lead I</th>
<th>Positive</th>
<th>Negative</th>
</tr>
</thead>
<tbody>
<tr>
<td>Positive</td>
<td>Normal Axis</td>
<td>LAD</td>
</tr>
<tr>
<td>Negative</td>
<td>RAD</td>
<td>Indeterminate Axis</td>
</tr>
</tbody>
</table>
What is the axis?

- Normal: QRS up in I and aVF
Rhythm

- **Sinus**
  - Originating from SA node
  - P wave before every QRS
  - P wave in same direction as QRS
What is this rhythm?

Normal sinus rhythm:
Supraventricular Arrhythmias

- PSVT- regular; P waves retrograde if visible; rate 150-250 bpm; carotid massage: slows or terminates

- Flutter – regular; saw-toothed pattern; 2:1, 3:1, 4:1, etc. block; atrial rate 250-350 bpm; ventricular rate ½, ⅓, ¼, etc. of atrial rate; carotid massage: increases block

- Fibrillation – irregular; undulating baseline; atrial rate 350 to 500 bpm; variable ventricular rate; carotid massage: may slow ventricular rate

- Multifocal atrial tachycardia (MAT) – irregular; at least 3 different P wave morphologies; rate –usually 100 to 200 bpm; sometimes < 100 bpm; carotid massage: no effect

- PAT – regular; 100 to 200 bpm; characteristic warm-up period in the automatic form; carotid massage: no effect, or mild slowing
Supraventricular Arrhythmias

**Atrial fibrillation**

**Atrial flutter**

**Multifocal atrial tachycardia**

### Table 4.3 Characteristics of Supraventricular Tachycardia (SVT)

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rate</td>
<td>150-250 beats/min</td>
</tr>
<tr>
<td>Rhythm</td>
<td>Regular</td>
</tr>
<tr>
<td>P waves</td>
<td>Atrial P waves may be observed that differ from sinus P waves</td>
</tr>
<tr>
<td>PR interval</td>
<td>If P waves are seen, the PRI will usually measure 0.12-0.20 sec</td>
</tr>
<tr>
<td>QRS duration</td>
<td>Less than 0.10 sec unless an intraventricular conduction delay exists</td>
</tr>
</tbody>
</table>
Ventricular Arrhythmias

VT: Ventricular Tachycardia
VF: Ventricular Fibrillation

Torsades de Pointes
Blocks

AV blocks

– First degree block
  - PR interval fixed and > 0.2 sec
– Second degree block, Mobitz type 1
  - PR gradually lengthened, then drop QRS
– Second degree block, Mobitz type 2
  - PR fixed, but drop QRS randomly
– Type 3 block
  - PR and QRS dissociated
What is this rhythm?

First degree AV block
PR is fixed and longer than 0.2 sec
What is this rhythm?

Type 1 second degree block (Wenckebach)
What is this rhythm?

Type 2 second degree AV block
What is this rhythm?

3rd degree heart block (complete)
Bundle Branch Blocks

RBBB criteria:
1. QRS complex > 0.12 seconds
2. RSR’ in leads V1 and V2 (rabbit ears) with ST segment depression and T wave inversion
3. Reciprocal changes in leads V5, V6, I, and aVL
LBBB criteria:
1. QRS complex > 0.12 seconds
2. Broad or notched R wave with prolonged upstroke in leads V5, V6, I, and aVL with ST segment depression and T wave inversion.
3. Reciprocal changes in leads V1 and V2.
4. Left axis deviation may be present.
Hemiblocks

Diagnosed by looking at right or left axis deviation:

• **Left Anterior Hemiblock**
  • 1. Normal QRS duration and no ST segment or T wave changes
  • 2. Left axis deviation greater than -30°
  • 3. No other cause of left axis deviation is present

• **Left Posterior Hemiblock**
  • 1. Normal QRS duration and no ST segment or T wave changes
  • 2. Right axis deviation
  • 3. No other cause of right axis deviation is present
Bifascicular Block

- **RBBB with LAH**
  - RBBB – QRS > 0.12 sec and RSR’ in V1 and V2 with LAH – left axis deviation

- **RBBB with LPH**
  - RBBB – RS > 0.12 sec and RSR’ in V1 and V2 with LPH – right axis deviation
Preexcitation

- **Wolff-Parkinson-White (WPW) Syndrome**
  - 1. PR interval < 0.12 sec
  - 2. Wide QRS complexes
  - 3. Delta waves seen in some leads

- **Lown-Ganong-Levine (LGL) Syndrome**
  - 1. PR interval < 0.12 sec
  - 2. Normal QRS width
  - 3. No delta wave
Preexcitation

WPW

LGL

Lown Ganong Levine Syndrome
Atrial Enlargement

Look at P waves in leads II and V1
• Right atrial enlargement (P pulmonale)
  • 1. Increased amplitude in first portion of P wave
  • 2. No change in duration of P wave
• Left atrial enlargement (p mitrale)
  • 1. Occasionally, increased amplitude of terminal part of P wave
  • 2. More consistently, increased P wave duration
Ventricular Hypertrophy

Look at the QRS complexes in all leads

• Right ventricular hypertrophy (RVH)
  - 1. RAD > 100°
  - 2. Ratio of R wave amplitude to S wave amplitude > 1 in V1 and < 1 in V6

• Left ventricular hypertrophy (LVH)

<table>
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<tr>
<th>Precordial Criteria</th>
<th>Limb Lead Criteria</th>
</tr>
</thead>
<tbody>
<tr>
<td>R wave in V5 or V6 + S wave in V1 or V2 &gt; 35 mm</td>
<td>R wave in aVL &gt; 13 mm</td>
</tr>
<tr>
<td>R wave in V5 &gt; 26 mm</td>
<td>R wave in aVF &gt; 21 mm</td>
</tr>
<tr>
<td>R wave in V6 &gt; 18 mm</td>
<td>R wave in I &gt; 14 mm</td>
</tr>
<tr>
<td>R wave in V6 &gt; R wave in V5</td>
<td>R wave in I + S wave in III &gt; 25 mm</td>
</tr>
</tbody>
</table>
Myocardial Infarction

- Dx – Hx, PE, serial cardiac enzymes, serial EKG’s
- 3 EKG stages of acute MI
  - 1. T wave peaks and then inverts
  - 2. ST segment elevates
  - 3. Q waves appear
Q Waves

- **Criteria for significant Q waves**
  - Q wave > 0.04 seconds in duration
  - Q wave depth > $\frac{1}{3}$ height of R wave in same QRS complex
  - Q waves are normal in lead III and aVR due to their rightward orientation.
Localizing MI on EKG

- **Inferior infarction** – leads II, III, aVF
  - Often caused by occlusion of right coronary artery or its descending branch
  - Reciprocal changes in anterior and left lateral leads

- **Lateral infarction** – leads I, aVL, V5, V6
  - Often caused by occlusion of left circumflex artery
  - Reciprocal changes in inferior leads

- **Anterior infarction** – any of the precordial leads (V1- V6)
  - Often caused by occlusion of left anterior descending artery
  - Reciprocal changes in inferior leads

- **Posterior infarction** – reciprocal changes in lead V1 (ST segment depression, tall R wave)
  - Often caused by occlusion of right coronary artery
Localizing MI on EKG
ST segment

• **Elevation**
  - Seen with evolving infarction, Prinzmetal’s angina
  - Other causes – J point elevation, apical ballooning syndrome, acute pericarditis, acute myocarditis, hyperkalemia, pulmonary embolism, Brugada syndrome, hypothermia

• **Depression**
  - Seen with typical exertional angina, non-Q wave MI
  - Indicator of + stress test
Electrolyte Abnormalities on EKG

- **Hyperkalemia** – peaked T waves, prolonged PR, flattened P waves, widened QRS, merging QRS with T waves into sine wave, AV block, VF
- **Hypokalemia** – ST depression, flattened T waves, U waves
- **Hypocalcemia** – prolonged QT interval
- **Hypercalcemia** – shortened QT interval
Drugs

• Digitalis
  - Therapeutic levels — ST segment and T wave changes in leads with tall R waves
  - Toxic levels — tachyarrhythmias and conduction blocks; PAT with block is most characteristic.

• Multiple drugs associated with prolonged QT interval, U waves
  - Sotalol, quinidine, procainamide, disopyramide, amiodarone, dofetilide, dronedarone, TCA’s, erythromycin, quinolones, phenothiazines, various antifungals, some antihistamines, citalopram (only prolonged QT interval – dose-dependent)
Digitalis

Dubin, 4th ed. 1989
EKG Δ’s in other Cardiac Conditions

• **Pericarditis:**
  Diffuse ST segment elevations and T wave inversions; large effusion may cause low voltage and electrical alternans (altering QRS amplitude or axis and wandering baseline)

• **Myocarditis:** conduction blocks

• **Hypertrophic Cardiomyopathy:**
  ventricular hypertrophy, left axis deviation, septal Q waves
EKG Δ’s in Pulmonary Disorders

• **COPD:**
  low voltage, right axis deviation, and poor R wave progression.

• **Chronic cor pulmonale:**
  P pulmonale with right ventricular hypertrophy and repolarization abnormalities

• **Acute pulmonary embolism:**
  right ventricular hypertrophy with strain, RBBB, and S1Q3T3 (with T wave inversion).
  Sinus tachycardia and atrial fibrillation are common.
EKG Δ’s in Other Conditions

• **Hypothermia:**
  Osborn waves, prolonged intervals, sinus bradycardia, slow atrial fibrillation, beware of muscle tremor artifact

• **CNS Disease:**
  diffuse T wave inversion with T waves wide and deep, U waves
Utter Confusion

- Verify lead placement
- Repeat EKG
- Repeat standardized process of EKG analysis - starting over from the beginning with basics – rate, intervals, axis, rhythm, etc. and proceed through entire stepwise analysis
Case

- 45 yo male presents with acute SOB s/p long vacation in Paris
- PMHx - asthma, Crohn’s disease, anxiety, GERD, tobacco abuse
- VS 37, 148/92, 130, 26
- Patient appears uncomfortable but otherwise unremarkable exam
Case

Acute PE with sinus tachycardia, a PVC, and S1Q3T3 pattern
Case

• 27 yo female presents to the ED with c/o chest discomfort and palpitations after studying all night for graduate school exams
• Appears nervous and “uneasy” with rapid pulse
• PMHx – unremarkable; no meds, admits to occasional alcohol, non-smoker, denies illicit drug use, used coffee to stay awake to study
Case

**SVT:** regular, narrow - QRS tachycardia, rate of 160 bpm
Case

• 46 yo male presents to ED with c/o severe HA persisting over 5 hours despite acetaminophen and NSAID attempts as abortive therapy
• PMHx: occasional left shoulder pain, non-smoker
• Construction worker
• VSS; unremarkable exam
Normal ECG
Case

- 28 yo male presents for commercial driver’s license (CDL) evaluation
- No complaints
- VSS; asymptomatic; exam without significant findings
Case

- Diagnosis? EKG findings?
Case

• Typical preexcitation (WPW) pattern
• Short PR interval and delta waves in many leads
• Tx is close observation unless patient has had SVT or atrial fibrillation which indicates tx with ablation of accessory pathway
Case

- 32 yo male presents to ED with c/o feeling sick for the last 6 days
- Symptoms include fevers, cough, and difficulty catching his breath
- PMHx: hyperlipidemia, obesity, metabolic syndrome
- VS 38.1, 105, 128/84, 22
Acute pericarditis: diffuse ST elevation with PR segment depression is diagnostic
Case

- 67 yo male presents to his cardiologist for out-patient 6 week post-hospital visit
- Previous hospitalization for non-cardiac chest pain
- Post-hospital cardiac meds: ACE inhibitor, beta blocker, aspirin, nitrate
- No current complaints
Case

- Atrial fibrillation: irregularly irregular without P waves
- RBBB: wide QRS with rsR’ pattern in V1, broad S waves in leads I and aVL
- Inferior infarct: non-acute (> 1 week) pathologic Q waves in inferior leads (II, III, and aVF)
Case

- 79 yo male brought to ED via EMS with chest pain, SOB, and near-syncope
- PMHx: unobtainable secondary to patient distress
- VS: 36.9, 140’s, 82/40, 28
Monomorphic sustained ventricular tachycardia (VT) – could rapidly deteriorate into VF, torsades de pointes, asystole, or sudden death.
What is the diagnosis?

Acute inferior MI with ST elevation in leads II, III, aVF
Junctional Rhythm

Rate 40-60, no p waves, narrow complex QRS

54-year-old woman reports severe lightheadedness with walking; she recently started a new medication for hypertension.
Hyperkalemia

Tall, narrow and symmetric T waves
Wellen’s Sign

ST elevation and biphasic T wave in V2 and V3
Sign of large proximal LAD lesion
Brugada Syndrome

Male 39 Years

RBBB or incomplete RBBB in V1-V3 with convex ST elevation
Brugada Syndrome

- Autosomal dominant genetic mutation of sodium channels
- Causes syncope, v-fib, self terminating VT, and sudden cardiac death
- Can be intermittent on EKG
- Most common in middle-aged males
- Can be induced in EP lab
- Need ICD
Second Degree Heart Block, Mobitz Type II

PR interval fixed, QRS dropped intermittently
Hypokalemia

U waves: Can also see PVCs, ST depression, small T waves
Bonus Case

• 18 yo male undergoing military physical exam and evaluation prior to boot camp
• No complaints
• PMHx – denies
• VSS; exam unremarkable
Bonus Case

- Diagnosis? EKG findings?
Bonus Case

- Reversed arm leads – inverted P waves in lead I with normal R wave progression in precordial leads