EKG Interpretations

Chad Link, DO FACC
Cardiologist
Chairman Cardiology Section
Sparrow TCI
Lansing, MI
Disclosures
Objectives

• Review general method for EKG interpretation
• Review specific points of “data gathering” and “diagnoses” on EKG
• Review treatment considerations
• Review clinical cases/EKG’s
EKG – 12 Leads

- Anterior Leads - V1, V2, V3, V4
- Inferior Leads – II, III, aVF
- Left Lateral Leads – I, aVL, V5, V6
- Right Leads – aVR, V1
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
  – 7. Bundle Branch Block or Hemiblock
  – 8. Preexcitation
  – 9. Enlargement and Hypertrophy
  – 10. Coronary Artery Disease
  – 11. Utter Confusion

» The Only EKG Book You’ll Ever Need
Malcolm S. Thaler, MD
Heart Rate

• Regular Rhythms

R to R Method

Example

Count total seconds between two R waves:
divide into 60:
Reminder: 1 small box = 0.04 sec

60
Total secs
19.5 x 0.04 s = .78 s

77 beats/min
Heart Rate

- Irregular Rhythms

- 5 big boxes = 1 sec
- 1 ECG = 10 sets of 5 big boxes
- Therefore one ECG = 10 secs

- Rate = BPM or R Waves/60secs
- 1 ECG x 6 = 1 min
- Count up R waves in one ECG x 6 = BPM
Intervals

- Measure length of PR interval, QT interval, width of P wave, QRS complex
QTc

- QTc = QT interval corrected for heart rate
  - Uses Bazett’s Formula or Fridericia’s Formula

- Long QT syndrome – inherited or acquired (>75 meds); torsades de pointes/VF; syncope, seizures, sudden death
Axis
Rhythm

• 4 Questions
  – 1. Are normal P waves present?
  – 2. Are QRS complexes narrow or wide (≤ or ≥ 0.12)?
  – 3. What is relationship between P waves and QRS complexes?
  – 4. Is rhythm regular or irregular?

• Sinus rhythm = normal P waves, narrow QRS complexes, 1 P wave to every 1 QRS complex, and regular rhythm
Types of Arrhythmias

- Arrhythmias of sinus origin
- Ectopic rhythms
- Conduction Blocks
- Preexcitation syndromes
AV Block

- Diagnosed by examining relationship of P waves to QRS complexes
- First Degree – PR interval > 0.2 seconds; all beats conducted through to the ventricles
- Second Degree – only some beats are conducted through to the ventricles
  - Mobitz Type I (Wenckebach) – progressive prolongation of PR interval until a QRS is dropped
  - Mobitz Type II – All-or-nothing conduction in which QRS complexes are dropped without PR interval prolongation
- Third Degree – No beats are conducted through to the ventricles; complete heart block with AV dissociation; atria and ventricles are driven by individual pacemakers
Heart Blocks

- Constant P-R interval
  - First Degree Heart Block
- Variable P-R Interval
  - Second Degree Heart Block Type I
  - Second Degree Heart Block Type II
  - Third Degree Heart Block
Bundle Branch Blocks

- Diagnosed by looking at width and configuration of QRS complexes

> 120 msec (3 small boxes)
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.
Bundle Branch Blocks

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**RBBB**

- **V1**: rSR'
- **V6**: qR

**LBBB**

- **V1**: rS
- **V6**: R

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Bundle Branch Block

**Left bundle branch block**

- **P**: 0.35 s
- **R**: 1 mV
- **T**: Deep S

**Right bundle branch block**

- **P**: Double R
- **T**: 0.14 s
- **R**: Broad S
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

• Common Arrhythmias
  – Paroxysmal Supraventricular Tachycardia (PSVT) – narrow QRS’s are more common than wide QRS’s
  – Atrial Fibrillation – can be rapid and lead to ventricular fibrillation
Preexcitation

WPW

LGL

Atrioventricular (AV) node
Sinus (SA) node
Accessory pathway

Normal electrical pathways
Abnormal electrical pathway in Wolff-Parkinson-White syndrome

Lown Ganong Levine Syndrome

ECG tracings
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 1/2, 1/3, 1/4, 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 PR interval will usually measure 0.12-0.20 sec</td>
</tr>
<tr>
<td>QRS duration</td>
<td>Less than 0.10 sec unless intraventricular conduction delay exists</td>
</tr>
</tbody>
</table>

**A fib with Rapid Ventricular Response (RVR)**

![Electrocardiogram](image)
# Rules of Aberrancy

<table>
<thead>
<tr>
<th>Clinical Clues</th>
<th>Ventricular Tachycardia</th>
<th>Paroxysmal supraventricular Tachycardia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clinical History</td>
<td>Diseased heart</td>
<td>Usually normal heart</td>
</tr>
<tr>
<td>Carotid Massage</td>
<td>No response</td>
<td>May terminate</td>
</tr>
<tr>
<td>Cannon A Waves</td>
<td>May be present</td>
<td>Not seen</td>
</tr>
<tr>
<td><strong>EKG Clues</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>AV Dissociation</td>
<td>May be seen</td>
<td>Not seen</td>
</tr>
<tr>
<td>Regularity</td>
<td>Slightly irregular</td>
<td>Very regular</td>
</tr>
<tr>
<td>Fusion Beats</td>
<td>May be seen</td>
<td>Not seen</td>
</tr>
<tr>
<td>Initial QRS deflection</td>
<td>May differ from normal QRS complex</td>
<td>Same as normal QRS complex</td>
</tr>
</tbody>
</table>
Ventricular Arrhythmias

VT - Ventricular Tachycardia
VF - Ventricular Fibrillation

PVC’s

Torsades de Pointes
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
  – 3. Possible right axis deviation 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
  – 3. No significant axis deviation
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>
<thead>
<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

• Criteria for Non-Q Wave MI
  – T wave inversion
  – ST segment depression persisting > 48 hours in appropriate clinical setting
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, 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)
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
- Athlete’s Heart – sinus bradycardia, nonspecific ST segment and T wave changes, RVH, LVH, incomplete RBBB, first degree or Wenckebach AV block, possible supraventricular arrhythmia
Other Important Points

• 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
Arrhythmia Indications to Consult Cardiology

- Diagnostic or management uncertainty
- Medications not controlling symptoms
- Patient is in high-risk occupation or participates in high-risk activities (pilot, scuba driving)
- Patients prefers intervention over long-term meds
- Preexcitation
- Underlying structural heart disease
- Associated syncope or other significant symptoms
- Wide QRS
Care Considerations Prior to Cardiology Consult

- Thorough Hx and PE
- Basic labs
- EKG and repeat EKG
- Holter monitor
- Echocardiogram
- Acuity of care required – consider risks, hemodynamic stability
Pacemaker Considerations

- Third-degree (complete) AV block
- Symptomatic lesser degree AV block or bradycardia
- Sudden onset of various combinations of AV block and BBB during acute MI
- Recurrent tachycardias that can be overdriven and terminated by pacemakers
Osteopathic Considerations

• Treatments –
  – Lymphatics – thoracic inlet, abdominal diaphragm, rib raising, lymphatic pumps
  – Sympathetics (T1-T6) – cervical ganglion, rib raising, T1-T6, Chapman’s reflexes, T10-L2 for adrenal/kidney
  – Parasympathetics – OA/AA/cranial – vagus nerve
Clinical Cases/EKG’s
Case 1

• A 59 year old male develops an acute onset tachycardia while watching the Super Bowl. He admits to “having a few beers” and has a heavy smoking history. His pulse rate is 150 beats per minute and quite regular.
Case 2

- A 19-year-old student athlete presents for a sports physical. He is on no medications. On physical exam: BP: 110/85 mm Hg; HR: 55 bpm; pulse rate is intermittently irregular.
EKG Differential Diagnosis

ST Segment Elevation

- Myocardial infarction (STEMI)
- Acute pericarditis
- Benign early repolarization
- Ventricular aneurysm
- Coronary vasospasm (Prinzmetal Angina)
Case 3

- A 74 year old female has been noticing a feeling of lightheadedness whenever she attempts any activity. She does not notice these symptoms at rest. Her symptoms have been gradually progressing over the past year and today she felt that she was going to “nearly pass out”. Her blood pressure monitor at home has been recording heart rates in the 40’s.
Case 4

- A 22 year old medical student presents to the Health Center complaining of palpitations. She has just taken her Cardiology Final Exam, and admits to considerable stress and lack of sleep over the past few days.
Case 4
65 year old caucasian female with 5 day hx of severe chest pain on exertion, previously alleviated with rest; now worsened over last 24 hours and sustained at rest

PMHx – DM2, HTN
Case 5

• Acute anterior ST-elevation MI with “tombstone” or “fireman’s hat” in V1-V4

• Tx? Localization?
Case 5

• PCI stenting of LAD

• Post-procedure = resolving ST elevation; loss of ominous tombstone effect; Q waves developing
Case 6

• 50 yo male presents with acute SOB s/p long vacation in Paris
• PMHx - GERD, tobacco abuse
• VS- 150/90, 140, 30
• Patient appears uncomfortable but otherwise unremarkable exam
Case 6

- Acute PE with sinus tachycardia, a PVC, and S1Q3T3 pattern
Case 7

• 75 yo male presents to the office for evaluation prior to colonoscopy
• No complaints
• PMHx –HTN, hyperlipidemia, and chronic low back pain
• VS- 160/80, 76, 16
Case 7
Case 7

• LVH – QRS voltage criteria in precordial leads and repolarization changes in V5, V6
Case 8

• 32 yo female presents to the ED with c/o chest discomfort and palpitations after studying all night for medical school exams
• Appears nervous and complains of palpitations
• PMHx – no significant PMHx
Case 8

- SVT – regular, narrow-QRS tachycardia, rate of 160 bpm
Supraventricular tachycardia

- **Etiology**
  - Ischemic heart disease
  - Digoxin toxicity
  - Excessive caffeine/amphetamine
  - Excessive ETOH
  - Atrial flutter with RVR

**What is your next step in management?**
Supraventricular Tachycardia

**Treatment**
- Maneuvers to increase vagal tone and delay AV conduction to block reentry
  - Valsalva maneuver
  - Carotid sinus massage
  - Breath holding
  - Head immersion into cold water
- Pharmacotherapy
  - IV adenosine = agent of choice
    - Decreases sinoatrial and AV nodal activity
  - IV verapamil, IV esmolol/propranolol/metoprolol, digoxin
- DC cardioversion if unstable or meds ineffective

**Prevention**
- Verapamil, Beta-blockers
- Radiofrequency ablation
Case 9

• 53 yo male presents to ED with c/o severe HA persisting over 6 hours despite acetaminophen and NSAID attempts as abortive therapy
• PMHx – no significant PMHx
• Janitor
• VSS- unremarkable exam
Case 9
Case 9

• Normal EKG
Case 10

• 49 yo female presents to primary care physician with c/o dizziness and occasional palpitations in her chest
• PMHx – anxiety, depression, obesity
• Works as secretary
• VSS normal
Case 10

• Second degree AV block – Mobitz Type I – Wenckebach (specifically 3:2 AV Wenckebach phenomenon where every 3rd P wave is blocked)
Case 11

• 35 yo male presents for commercial driver’s license (CDL) evaluation
• No complaints or PMHx
• VSS- normal
Case 11
Case 11

- 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 12

• 56 yo male presents to ED with c/o feeling sick for the last 6 days
• Symptoms include shortness of breath and chest pain
• PMHx – DM, hyperlipidemia
• VS - tachycardic
Case 12

• Acute pericarditis – diffuse ST elevation with PR segment depression is diagnostic
Case 13

- 75 yo male presents to the office for post hospital follow up
- No significant PMHx
- Medications– ACE inhibitor, beta blocker, aspirin and a statin
Case 13
Case 13

- 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 14

• 86 yo male brought to ED via EMS with chest pain, SOB and syncope
• PMHx – unobtainable
• VS – 150’s, 90/50, 30
Case 14
Case 14

- Monomorphic sustained ventricular tachycardia (VT) – could rapidly deteriorate into VF, torsades de pointes, asystole, or sudden death
85 yo female admitted the hospital for chest pain
PMHx – HTN, PAD, Hyperlipidemia, DM2, CHF, obesity, depression
Case 15
Case 15

- LBBB – wide QRS; broad, notched R wave in V5, V6 and I with ST depression and T wave inversion
Case 16

- 52 yo male presents to ED with CP and appears diaphoretic and in acute distress
- PMHx – PAD, Hyperlipidemia, HTN, ESRD, DM2, Left BKA
- VS – 120, 100/68, 22
Case 16
Case 16

- Hyperkalemia – tall peaked T waves present throughout; other progressive EKG changes may follow with increasing potassium levels – prolonged PR interval, flattened P waves, widening QRS, sine waves
- Sinus tachycardia also present
Case 17

- 18 yo male undergoing physical exam
- No complaints
- PMHx – denies
- VSS-unremarkable
Case 17
Case 17

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

• With permission - Cardiology/EKG Board Review - MJ Bradley, DO
• Sources and Suggested References
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  – Rapid Interpretation of EKG’s – Dale Dubin, M.D.
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