Introduction

- Why did I choose this topic?
  - Most of the EKGs we order are normal
  - We rely on **pattern recognition** when interpreting them
  - As a diagnostic test EKGs are sensitive, but not specific
  - You don’t want to miss a potentially **life threatening** finding in a healthy appearing patient
The focus of this talk…

- Pattern recognition of different EKG findings in these common clinical scenarios
  - Chest pain
  - Syncope
  - Toxicology
  - During resuscitation
Things I’m not discussing

- A thorough review of EKG interpretation
- Every type of stable tachycardia
- Congenital heart disease EKGs – including post-op patients and those with pacers
- All manner of toxicology EKGs
- In depth management
EKG Overview
To order or not to order

- When should you get an EKG?

- Worrisome history
  - Acute onset
  - Associated with exercise
  - Associated with syncope
  - History of heart disease, connective tissue disorder, rheumatologic disease, sickle cell
  - Cocaine use
  - Abnormal PE with Cardiac findings
The “normal” EKG

- **Rate** – I won’t insult your intelligence here…

- **Sinus rhythm** – a P for every QRS

- **Axis**
  - I and aVF have mainly positive QRS complexes = normal axis.
  - I is positive and aVF is negative = left axis deviation (LAD).
  - I is negative and aVF is positive = right axis deviation (RAD).
  - Both leads negative = extreme RAD or extreme LAD
The “normal” EKG

- Normal values vary by age so check out “The Pocket” page 13

- Criteria for chamber enlargement are found on page 12
More about T waves

- **Normally in adults…**
  - Always upright in leads I, II, V3-6, and always inverted in lead aVR
  - Amplitude < 5mm in limb leads, < 15mm in precordial leads

- **Inverted T waves**
  - Normal in children (especially V1-3) due to predominance of RV forces
  - Can persist into adulthood, most often in African American/Caribbean women - asymmetric, shallow (<3mm) and usually limited to leads V1-3

- **Otherwise inverted T waves indicate pathology**
  - MI, BBB, RVH/LVH, PE, increased ICP
T wave changes at 3 different ages

- By age 16 T waves *should* be upright in V2-6
- Some normal adults will have a downward V1
Normal inverted T waves (V1-V3) in a two year old male
Abnormal T wave morphology

Prior MI - Inverted T and Q wave

Hyperkalemia

Acute MI – hyperacute T waves
Events causing a sudden rise in ICP (e.g. subarachnoid hemorrhage) produce widespread deep T-wave inversions with a bizarre morphology.
Biphasic T waves

- Ischemia (up then down)
- Hypokalemia (down then up)
S1 QIII TIII pattern of acute PE
What is this J Point Elevation Stuff?

- Normal variant most commonly seen in young males.
- Early repolarization of some myocardial fibers may deviate the ST upwards in a concave pattern, especially when R waves high with tall peaked T waves.
- The “J point” is frequently elevated where the QRS joins the concave ST elevation.

Differentiation from MI – In J Point you’ll see:
- ST segment elevation is typically less pronounced (equal to or less than 0.05mV)
- Ventricular surface area is greater (more leads involved)
J Point Elevation

Acute Myocardial Infarction: Anterior Wall
There’s also J Point depression?

- Normal depression is 1mm in limb leads and 2mm in precordial leads
Chest Pain
Ischemia

- T-waves flat or downsloping
- The location of the abnormalities are much less clearly correlated with anatomical findings
- May indicate **acute coronary syndrome** (e.g. unstable angina)
<table>
<thead>
<tr>
<th>Location</th>
<th>Leads</th>
<th>Complications</th>
<th>Vessels</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anterior</td>
<td>V3-V4</td>
<td>LV dysfunction, CHF, heart block</td>
<td>LCA</td>
</tr>
<tr>
<td>Septal</td>
<td>V1-V2</td>
<td>Bundle Branch Blocks and other heart blocks</td>
<td>LAD-septal branch</td>
</tr>
<tr>
<td>Inferior</td>
<td>II, III, AVF</td>
<td>Hypotension, possible severe and adverse reactions to nitrates and morphine</td>
<td>RCA</td>
</tr>
<tr>
<td>Lateral</td>
<td>V5-V6</td>
<td>AV nodal block, LV dysfunction</td>
<td>LCA-circumflex</td>
</tr>
<tr>
<td>Posterior</td>
<td>II, III, AVF, rV4 or marked depression V1-V4</td>
<td>AV nodal block, Atrial fibrillation and flutter, Hypotension, possible severe and adverse reactions to nitrates and morphine</td>
<td>RCA (RV infarct)</td>
</tr>
</tbody>
</table>

**ECG Diagram**

- **A.** Normal
- **B.** ST elevation min-hours
- **C.** Q waves days
- **D.** Inverted T weeks
- **E.** T normalizes months
1. Parent with chest pain

Acute Myocardial Infarction: Anterior Wall
2. Parent with chest pain

Acute Myocardial Infarction: Inferior Wall
3. Teenage male with exertional chest pain

Hypertrophic Cardiomyopathy (HOCM)

- LVH
- Deep T wave inversions in all precordial leads
Example 3

- A 13 year old female presents with chest pain, tachypnea, and fever
- Not caused by the 15 year old male seen here
Pericarditis
Chest pain EKG findings summary

- **STEMI** - duh, ST segment elevation
- **Ischemia** - ST segment depression, flat or downsloping T waves
- **Pericarditis** - diffuse ST segment elevation
- **HOCM** - LVH, deep T wave inversions in precordial leads
Syncope
Cardiac causes of syncope

- **Conduction abnormalities** – Block, Sick sinus, long QTc
- **Arrhythmias** – SVT, V Tach, V fib
- **Obstructive lesions** – arterial stenosis, HOCM, TOF, Anomalous origin of the left coronary artery
- **Acquired lesions** – tumors, myocarditis, cardiomyopathy
Syncope

- EKG findings in life threatening causes of syncope
  - Prolonged QT
  - Brugada pattern
  - Epsilon waves (arrhythmogenic right ventricular dysplasia)
  - Preexcitation syndrome (Wolff Parkinson White)
  - Nonsinus rhythm
  - Short QT interval (≤0.30 sec)
  - Conduction delay or atrioventricular block
  - Signs of myocardial injury
  - Ventricular hypertrophy or strain patterns
5. Wolf Parkinson White

**Sinus Rhythm with Short PR interval and Ventricular Pre-excitation Possible Wolf-Parkinson-White**

- **Short PR Interval**
- **Delta Wave**
6. SVT

Supraventricular Tachycardia
Rate = 190

ST depression at arrows
SVT by originating focus

- **SA node**
  - Inappropriate sinus tachycardia
  - SA node reentrant tachycardia

- **Atrial**
  - Ectopic atrial tachycardia
  - Multifocal atrial tachycardia
  - A fib with rapid ventricular response
  - A flutter with rapid ventricular response

- **AV Node (Junctional)**
  - AV node reentrant tachycardia
  - AV reentrant tachycardia (includes WPW)
  - Junctional ectopic tachycardia

*Spoiler alert! The key finding of SVT is a Narrow QRS*
Atrial focus

- A fib with rapid ventricular response

Irregularly irregular. No p-waves. Rapid ventricular rate = 177

These beats are abarently conducted

There is some ST depression
V4 V5 V6
Atrial focus

- Multifocal atrial tachycardia

![ECG Image with labeled P-waves of at least 3 morphologies]
AV Node

- AVNRT – P is in ST segment, after QRS

- JET

  Atrial activity
  Ventricular activity

  Short PR waves, P waves fall before and after the narrow QRS
7. Atrial Flutter

- Sawtooth pattern best seen in V1, II and III
- Atrial rate can be as high as 300
A couple more examples of atrial flutter
8. Long QT

\[ QTc = \frac{QT}{\sqrt{RR \text{ (sec)}}} \]
9. First degree AV block with sinus bradycardia
AV Block

- Disturbance in conduction between sinus impulse and ventricular response
  - 1\textsuperscript{st} degree – PR > upper limit of normal for age
  - 2\textsuperscript{nd} degree
    - Mobitz Type I (Wenckebach) – Progressive lengthening of PR until a beat is dropped
    - Mobitz Type II – failure at bundle of His, either normal AV conduction or none – the ventricle will not contract without a P wave
  - 3\textsuperscript{rd} degree – complete AV dissociation
AV Block

First degree AV block

Second degree AV block
- Mobitz Type I (Wenckebach phenomenon)
- Mobitz Type II

2:1 AV block

Complete (Third degree) AV block
10. Right Bundle Branch Block
Triple B (Right)

- **RBBB**
  - Right axis deviation
  - QRS longer than normal for age
  - Terminal slurring of QRS to the right
    - Wide/slurred S in I, V5, V6
    - Terminal slurred R’ in V4R, V1, and V2
  - In adults ST depression and T wave inversion

Pro-Tip: You can’t reliably diagnose RVH on EKG when RBBB is present
Triple B (Left)

- **LBBB**
  - Left axis deviation
  - QRS longer than normal for age
  - Loss of Q waves in V5-6
  - Slurred QRS Complex to the left and posterior
    - Wide R waves in I, aVL, V5, and V6
    - Wide S waves in V1 and V2
  - ST depression and T wave inversion in V4-6

Pro-Tip: You can’t reliably diagnose LVH or ischemia on EKG when LBBB is present
- Idiopathic causes of pulmonary HTN can affect adolescents
- Older females 3x > males
- In young children M:F ~ 1:1

- Syncope is a common presenting symptom!
Right ventricular hypertrophy  Right ventricular hypertrophy due, in this case, to primary pulmonary hypertension. The characteristic features include marked right axis deviation (+210° which is equal to -150°), tall R wave in V1 (as part of a qR complex), delayed precordial transition zone with prominent S waves in leads V5 and V6, inverted T waves and ST depression in V1 to V3 consistent with right ventricular “strain”, and peaked P waves in lead II consistent with concomitant right atrial enlargement. Courtesy of Ary Goldberger, MD.
Right ventricular hypertrophy and right axis deviation in a 10-year-old male with primary pulmonary hypertension with tall R waves in V1 and V2 and deep S waves in V5 and V6
Syncope EKG findings summary

- **Supraventricular tachycardia** – narrow QRS
  - Atrial focus
    - **A fib with rapid ventricular response** – irregularly irregular, no P waves
    - **Atrial flutter** - very fast rate (300), sawtooth pattern
    - **Multifocal atrial tachycardia** – at least 3 P wave morphologies
  - AV Node (junctional) focus
    - **AVNRT** – P is in ST segment, after QRS
    - **JET** - Short PR waves, P waves fall before and after the narrow QRS

#1 cause of SVT in kids are the reentrant mechanisms
Syncope EKG findings summary

- **WPW** – delta wave, sinus rhythm, short PR
- **Long QT** – >440ms
- **Primary Pulmonary HTN** – RVH, R axis deviation, RA enlargement (peaked P in II), ST depression V1-3
Toxicology

- In general EKGs have low yield in overdoses and accidental ingestions
- Certain patterns may be seen on the boards however…
11. 4 year old with altered mental status, tachycardia, dilated pupils and dry mucous membranes

**TCA Overdose**
- QRS >100 msec predictive of seizures / >160msec predictive of arrhythmias
- R wave in aVR>3mm also predictive of seizure / arrhythmia.
- Sinus Tachycardia
- QT prolongation
12. A 7 year old cardiac patient has the following EKG. Grandma can’t remember what meds he is on.

**Digitalis effects**
- Shortened QT interval
- Characteristic down-sloping ST depression
- Dysrhythmias
- Ventricular / atrial premature beats
- Paroxysmal A-tac with variable AV block
- V-tac and V-fib

Salvador Dali’s moustache
**Table 5. Differences In Evaluating The ECG In Patients With Acute Digoxin, β-blocker, And CCB Toxicity.**

<table>
<thead>
<tr>
<th></th>
<th>Digoxin</th>
<th>β-Blocker</th>
<th>CCB</th>
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<tbody>
<tr>
<td>Atrial tachycardia</td>
<td>+</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>Wide QRS</td>
<td>+</td>
<td>+/-</td>
<td>–</td>
</tr>
<tr>
<td>High-degree AV block</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Biventricular tachycardia</td>
<td>+</td>
<td>–</td>
<td>–</td>
</tr>
</tbody>
</table>

– Indicates absent; and + indicates present

http://www.ebmedicine.net/topics.php?paction=showTopicSeg&topic_id=91&seg_id=1752
Abnormal rhythms originating in the ventricle (distal to the Bundle of His) are the kind that kill folk

In adults #1 cause is CAD

In children #1 reentrant mechanism
Ventricular tachycardia

1. Absence of RS complexes in the precordial ECG leads (V1-V6)
2. RS duration greater than 100 milliseconds in any precordial lead
3. Ventriculoatrial dissociation
14.

Ventricular fibrillation

Chaotic, random, asynchronous electrical activity of the ventricles resulting in rapid, irregular bizarre QRS complexes. Wavy chaotic ventricular depolarization resulting in fibrillatory waves.
Torsades de pointes

- Rapid, polymorphic ventricular tachycardia with a characteristic twist of the QRS complex around the isoelectric baseline
- Can degenerate into V fib
Factors associated with torsades
des des pointes

- Familial long QT syndrome
- Class IA antiarrhythmics
- Class III antiarrhythmics
- Hypomagnesemia
- Hypokalemia
- Hypocalcemia
- Hypoxia

- Acidosis
- Heart failure
- LVH
- Slow heart rate
- Female gender
- Hypothermia
- Subarachnoid hemorrhage
16. V tach | Torsades | V fib
SVT with aberrancy is SVT wide QRS complexes

**In V tach**
- No P wave association with QRS
- Preceded by PVC
- QRS > 0.16 sec

**In SVT with aberrancy**
- QRS 0.12-0.26 sec
- Preceded by PAC
- RBBB pattern - rsR’ in V1 or qRs in V6
Pro-Tip: SVT with aberrancy is very rare in kids – so a wide complex tachycardia without P waves is probably V tach
Brugada syndrome

- Seen in young men from South East Asia
- Pseudo RBBB with J point elevation and concave ST elevation
- Can lead to paroxysmal V fib and then death
- UCED attendings like to discuss it

- Normal QRS
- ST segment elevation and T wave inversion in the right precordial leads V1 and V2 (arrows)
- Wide S wave in left lateral leads that is characteristic of RBBB is absent
Ventricular dysrhythmias EKG findings summary

- **V tach** - >3 PVCs, rate 120-180

- **V fib** – rapid irregular rate, bizarre QRS pattern of varying size/morphology

- **Torsades** - Rapid, polymorphic ventricular tachycardia with a twist of the QRS complex around the isoelectric baseline

- **Brugada syndrome** - Pseudo RBBB with J point elevation and concave ST elevation
Other Examples
17. Pulled from a lake

18. 3 year old with Kawasaki disease

**Osborn waves**
Positive deflections between QRS and ST segment
- Hypothermia
- Hypercalcemia
- Head injury
- V fib

**Sinus tachycardia**
Kawasaki EKGs can show findings consistent with myocarditis such as arrhythmias, prolonged PR/QT intervals, and nonspecific ST changes. Or they can be normal…
Wrap up

- EKGs are usually normal when we order them.
- If they’re abnormal we have a pretty good idea that they’ll be abnormal before we get them (e.g. history).
- It takes practice and repetition to get good at picking out the abnormal findings.
- Don’t miss those findings in the well looking patient.
- Check out these resources:
  - How To Read Pediatric EKGs by Park
  - ECG Library
  - Emergency Medicine Education Online