12-Lead EKG Interpretation

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Goals for today’s ECG Review:

• Determine Right vs Left bundle branch block
• Diagnose Acute MI
• Diagnose old MI
• Location of the infarct
• Other Acute Coronary Syndromes
• Determine Axis
• Other ECG confounders
• Pfun!

“Ask questions any time—I like answering them”

What a 12-Lead EKG can help you do

• Diagnose ACS / AMI
• Interpret arrhythmias* (computer Dx)
• Identify life-threatening syndromes (WPW, LGL, Long QT synd., Wellens synd., etc)
• Infer electrolyte imbalances
• Infer hypertrophy of any chamber
• Infer COPD, pericarditis, drug effects, and more!

Disclosures:

• I work for Virginia Garcia Memorial Health Center, Beaverton, Oregon.
• And I am a medical editor for Jones & Bartlett Publishing.
For example: 73 y.o. male with nausea, syncope

Acute Inferior MI

What rhythm? (look at V1 for P waves)

Atrial flutter (w/septal MI?)
another example...

Wolff-Parkinson-White synd.

- short PR
- wide QRS
- delta wave

WPW with Atrial Fib

Same pt, converted to SR
Limitations of a 12-Lead ECG

- Truly useful only ~40% of the time
- Each ECG is only a 10 sec. snapshot
- Serial ECGs are necessary, especially for ACS
- Other labs help corroborate ECG findings (cardiac markers, Cx X-ray)
- Confounders must be ruled out (dissecting aneurysm, pericarditis, WPW, LBBB, digoxin, RVH)

The Problem with Bundle Branch Blocks

- Desynchronized contraction of the ventricles
- Reduced cardiac output
- Worsened heart failure
- **LBBB confounds the EKG interpretation and makes it harder to find ACS**

Confounder: Left Bundle Branch Block

Right BBB (V1, V2, MCL1: rsR' pattern)

Left BBB (L1, V5, V6: upright QRS with a notch)
Bundle Branch Blocks
Two QRSs

Healthy ventricle
Blocked bundle

RBBB
V1 & V2

LBBB
V5 V6
(& I, aVL)

Practice: Bundle Branch Block
Which Bundle Branch is Blocked?

1. Right Bundle Branch Block (Lead V1)

2. Left Bundle Branch Block (L I, V5, V6)
Where is the Pathology?

Right Bundle Branch Block

Left Bundle Branch Block
Limitations of a 12-Lead ECG

- They are occasionally wrong!

ECG Pearls

- Lead II is the easiest lead to read / most intuitive
- **But Lead V1 is our single best lead.**
- “A Q in III is free.” (isolated Q in L III)
- If you know where the + electrode is, you can read any ECG
- *Finding the “P” is ~80% of reading an ECG!*
ECG Lead Placement & Electrophysiology Review

Einthoven’s Triangle

Limb Leads

- I
- II
- III (standard leads)

Normal 12-Lead ECG

Leads I, II, III

AVR
V1
V4

II
AVL
V2
V6

III
AVF
V3
V6
Rapid Interpretation Tips

Dr. Willem Einthoven

• Invented the electrocardiograph
• Discovered atrial fibrillation
• Won Nobel Prize for Medicine 1924

Conduction System

SA Node → AV Node → His Bundle → BBs → Purkinje Fibers

Lead II

P wave axis

...upright in L II

R wave axis

...upright in L II
Intervals

PR Interval: 120 – 200 mSec (3 – 5 boxes)
QRS width: 60 – 120 mSec (1½ – 3 boxes)
QT/QTc interval: 400 mSec (10 boxes)

Heart Rate Calculations

Triplicate Method:
- 300, 150, 100,
- 75, 60, 50
- Quick, easy, sufficient

6-second strip:
- Count PQRST cycles in a 6-second strip & multiply x 10
- Easy, & more accurate

Horizontal axis is time (mS); vertical axis is electrical energy (mV)

Normal Sinus Rhythm

What is the heart rate?

EKG Leads

Limb (frontal plane) Leads
- I
- II (standard leads)
- III
- aVR
- aVL (augmented leads)
- aVF
Normal 12-Lead ECG

6 Frontal Plane Leads (limb leads)

Axis

Leads
I
II
III
aVR*
aVL
aVF

If you know where the “+” electrode is, you can read any EKG
EKG Leads

**Limb (frontal plane) Leads**
- I
- II
- III
- aVR
- aVL
- aVF
  - (augmented leads)

**Chest (precardial) Leads**
- V1
- V2
- V3
- V4
- V5
- V6
  - (anterior leads)
  - (lateral leads)
Normal 12-Lead ECG

New 12-Lead ECG Format

Axis Determination
Why We Care About Axis Deviations

The axis shifts towards hypertrophy & away from infarction.

Axis Deviation

- **Horizontal heart** (0°): obesity, 3rd trimester pregnancy, Ascites
- **Vertical heart** (90°): slender build
- **Left Axis Deviation**: LBBB, Anterior MI, Inferior MI, Left anterior hemiblock, LVH
- **Right Axis Deviation**: Anterior MI, Lateral MI, RBBB, COPD, RVH, Left posterior hemiblock
- **Extreme RAD**: Ectopic rhythm (VT), massive MI

QRS Morphology in Lead II

**How to calculate Axis**

- **Easiest**: the computer does it for you!
- **Easy**: find the tallest R wave (if tallest is Lead II = normal axis)
- **Most fun**: Thumbs up / Thumbs down
Calculating Axis: Thumbs Up / Down Method

Lead I — Your Left thumb
Lead aVF — Your Right thumb
Lots of ways to read EKGs…

- QRSs wide or narrow?
- Regular or irregular?
- Fast or slow?
- P waves?
- Sinus rhythm or not?
- If not, is it atrial fibrillation?
- BBB?
- MI?

Symptoms:
- Syncope is bradycardia, heart blocks, or VT
- Rapid heart beat is AF, SVT, or VT

Step-by-step method for reading a 12-Lead

Rapid Interpretation Tips

- Identify the rhythm. If supraventricular*,
- Rule out left bundle branch block. If no LBBB,
- Check for: ST elevation, or ST depression with T wave inversion, and/or pathologic Q waves. If present,
- Rule out other confounders: WPW, pericarditis, LVH, digoxin effect
- Identify location of infarct, and consider appropriate treatments: MONA, PCI [or fibrinolytic], nitrate infusion, heparin infusion, GP IIb, IIIa inhibitor, beta-blocker, clopidogrel, statin, etc.
Supraventricular rhythms

- Sinus rhythm
- Atrial fibrillation
- Junctional rhythm
- PSVT / AVNRT
- Atrial tachycardia
- Atrial flutter
- Wandering atrial pacemaker
- MAT

Normal 12-Lead ECG

Rapid Interpretation Tips

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Ischemia Injury Infarction

Normal Ischemia Injury Infarction

Normal ECG ST-segment depression ST-segment elevation Plus Q-waves

Unstable Angina (no ST-segment elevation)

Myocardial Infarction (ST-segment elevation)

Extent of myocardial damage

Serum cardiac marker release

CK-MB Troponin T Or l CRP, P-selectin

ST elevation, ST depression, T wave inversion, pathologic Q waves

STEMI

Percutaneous Coronary Intervention

Occluding Thrombus
Acute Regional Myocardial Infarction

Figure 1

Thrombus formation at the site of plaque rupture. Platelets adhere to the surface of the injured plaque and aggregate into a platelet-rich "white clot" portion. Erythrocytes and a few white blood cells become trapped in the more extensive "red clot" region by fibers that form as a result of activation of the clotting cascade. Fibroblasts also develop platelets and the protease to the proteins that mediate its degradation (plasminogen, not shown). Thrombus is incorporated into the clot structure.
**STEMI: ECG Changes**

A. Normal ECG  

B. **Hyperacute T wave changes** - increased T wave amplitude and width; may also see ST elevation  

C. **Marked ST elevation** with hyperacute T wave changes (transmural injury)  

D. **Pathologic Q waves**, less ST elevation, terminal T wave inversion (necrosis)  

E. Pathologic Q waves, **T wave inversion** (necrosis and fibrosis)  

F. Pathologic Q waves, **loss of R waves** (fibrosis)

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**Why Pathologic Q Waves Form**

- **Injury**
  - ST segments usually elevate within minutes of the onset of cardiac chest pain

- **Ischemia**
  - T waves invert fully by 24 hours

- **Acute Infarction**
  - Pathologic Q waves ≥0.03 sec or ≥1/6 height of QRS begin to form in 1 hour

- **Non-Q-Wave Infarction**
  - Flat, depressed ST segments in two or more contiguous leads; or may have inverted T waves

- **Old Infarction**
  - ST segments are normal; Q waves remain forever

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**Myocardial Infarction ECG Patterns**

- Early reperfusion is the definitive treatment for most AMI patients. The patient can lose 1% of salvageable myocardium for each minute of delay. Remember: “**Time is Muscle**”

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**RCA before and after stenting**

Before stenting  

After stenting
STEMI — Typical Progression

Acute Inferior MI

1. **Acute Inferior MI**
   - ST elevation
   - Qs
   - Axis is shifting leftward...

2. **Acute Inferior MI #2**
   - Same Patient ~2 hrs later
   - Worsened ST elevation

3. **Acute Inferior MI #3**
   - Same Patient 9 days later
   - Permanent Q waves (inferior wall scar)
   - Permanent left axis deviation
   - But NO anterior infarct (no Qs)
**Acute Anterior MI**

( ST segment elevation ≥1 mm, with or without Q waves in two or more contiguous Leads: V1-V4. Poor R wave progression* and inverted T waves may also be present. Reciprocal ST depression may be present: II, III, AVF.)

*Note: LVH also can cause poor R wave progression & Q waves in V1-V2. Rule it out first."

**Acute Inferior MI**

( ST segment elevation ≥1 mm in two or more contiguous Leads: II, III, AVF. Q waves and inverted T waves may also be present. Reciprocal ST depression may be present in Leads: I, AVL, V2-V4.)

The right (or left) coronary artery is occluded. May cause: left posterior hemiblock; left axis deviation; LBB, sinus bradycardia, 1st AV block, 2nd AV block Mobitz I (Wenckebach), 3rd AV block & AV nodal block."

**Acute Right Ventricle MI**

( ST segment elevation in Leads: V4-V6, (MC4,5,6,8). Q wave and inverted T wave may also be present). Accompanies inferior MI in 30% of cases.

RCA is occluded. May cause: AV block, A-Fib, A-Flutter, right heart failure, JVD with clear lungs, BP may drop if preload is reduced (be cautious with morphine, NTG, furosemide). Treat hypotension with IV fluids, pacing.

**Acute Lateral MI**

( ST segment elevation ≥1mm in Leads: I, AVL, V5, V6. Q waves and inverted T waves may also be present)

The circumflex branch of the left coronary artery is occluded. May cause: LV dysfunction, AV nodal block.  

Note: Lateral MI may be a component of a multiple site infarction, including anterior, inferior and/or posterior MI.
Acute Posterior MI

(St segment depression with or without large R waves in Leads: V1, V2, V3. Inverted T waves may also be present)

*Acute posterior MI is rarely seen alone. It is usually a component of a multiple site infarction, including inferior MI.

Note: RVH can also cause a large R wave in V1. Rule out RVH first.

Practice: Infarct Location

Where is the Pathology?

Acute Anterior MI
(ST elevation in V1 - V4)

What is the R wave axis?

ST Elevation
Acute Inferior MI
(ST elevation in II, III, F)

Note the axis has not shifted yet, because it is early in the AMI, and there are no loss of R waves yet.
**Where is the Pathology?**

**10**

**Acute Inferior MI & Right Ventricle MI**

**10**

**Where is the MI?**

**11**

**Acute Posterior MI**

**11**

- V1, V2, V3
- Large R Waves
- Depressed STs

Normal V1 – V3

Normal V1 – V3
Rapid Interpretation Tips

- Identify the rhythm. If supraventricular,
- Rule out left bundle branch block. If no LBBB,
- **Check for:** ST elevation, or ST depression with T wave inversion, and/or pathologic Q waves. *If present,*
- **Rule out other confounders:** WPW, pericarditis, LVH, digoxin effect
- Identify location of infarct, and consider appropriate treatments: MONA, PCI [or fibrinolytic], nitrate infusion, heparin infusion, GP IIb, IIIa inhibitor, beta-blocker, clopidogrel, statin, etc.

**Wolff-Parkinson-White synd.**

- Short PR, Wide QRS, “Delta” wave

**Wolff-Parkinson-White synd.**

- Short PR interval
- Normal QRS interval
- No delta wave

- Short PR interval
- Prolonged QRS interval
- Delta wave present
  (See Case 37)

- Short PR interval
- Slightly prolonged QRS interval
- Delta wave present
  (See Cases 34-36)
**WPW**

False Q waves mimic MI

**Other Confounders**

- **Benign Normal Variant**
  - ST Elevation
  - Mild upsloping ST segments

- **Pericarditis**
  - Elevated STs in multiple leads
  - Depressed PR segments

- COPD (small QRSes in limb leads)
  - RVH may also be present.
Other Confounders

Left Ventricular Hypertrophy

- Tall R waves in V5, V6; deep S waves in V1, V2

Other Confounders: Digoxin

(ST Depression)

Atrial fibrillation and digitalis effect

Depressed ST segments

ST Depression

(a diagnostic challenge)

Can be caused by:
- Ischemia
- Digoxin effect
- Taehycardia
- LVH, BBB
- Hypokalemia
- NSTEMI (Non Q wave MI)
Ischemic ST Depression (a positive exercise ECG)

Practice: Confounders

44 y.o. female with history of tachycardia

WPW (short PR, Wide QRS, Delta waves)

- short PR
- false Q waves
- delta waves
30 y.o. male with *positional* chest pain

**Pericarditis**
- Depressed PR segments
- Elevated ST segments in multiple leads

**Rhythm? Pathology?**
- Large Old Anterolateral MI
- Large Qs V1–V6
- Ventricular aneurysm

**Large Old Anterolateral MI**
- Ventricular aneurysm