Disclosures:

- Work for Virginia Garcia Memorial Health Center.
- And I am a medical editor for Jones & Bartlett Publishing.

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
- Other ECG confounders
- Determine Axis

What a 12-Lead EKG can help you do

- Diagnose ACS / AMI
- Interpret arrhythmias (computer Dx)
- Identify life-threatening syndromes (WPW, LQTS, etc)
- Infer electrolyte imbalances
- Infer hypertrophy of any chamber
- Infer COPD, pericarditis, drug effects, and more!

For example:

73 y.o. male with nausea, syncope

Acute Inferior MI

ST elevation
What rhythm? (look at V1 for P waves)

Atrial flutter (w/septal MI?)

The flutter waves are maximal in Lead III.

Another example...

WPW with Atrial Fib

Wolff-Parkinson-White synd.

- short PR
- wide QRS
- delta wave

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).

**Confounder: Left Bundle Branch Block**

- Impending AMI with normal ECG!

**Confounder: Wolff-Parkinson-White syndrome**

- Pt is a 4 y.o. child w/ one episode of tachycardia and shortness of breath.
- WPW mimicking MI *(false Q waves* in Lead II, III, AVF, V1, & V3). Also mimicking LBBB.

**“ECG Pearls”**

- Lead II is the easiest lead to read / most intuitive.
- But Lead V1 is our single best lead.
- Lead V3 is best for QT interval measurement.
- “A Q in III is free.” *(isolated Q in L III)*
- 80% of reading an ECG is finding the P wave!
- The other half is knowing where the + electrode is. 😊
ECG Lead Placement & Electrophysiology Review

Einthoven’s Triangle

Limb Leads

- I
- II
- III

(standard leads)

Normal 12-Lead ECG

The first EKG machine  ca 1903

Conduction System

Lead II
QRS Morphology in Lead II

Leads I, II, III

Intervals

PR Interval: 120 – 200 mSec (3 – 5 boxes)
QRS width: 60 – 120 mSec (1 ½ – 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 PQRS cycles in a 6-second strip & multiply x 10
- Easy, more accurate

Normal Sinus Rhythm

• What is the heart rate?

EKG Leads

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

Axis

6 Frontal Plane Leads (limb leads)

12 Lead Electrode Placement

Limb (frontal plane) Leads

Chest (precordial) Leads

EKG Leads

(augmented leads)

(anterior leads)

(lateral leads)

(standard leads)
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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    If present,
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**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

**Bundle Branch Blocks**
- Bundle Branch Blocks (QRS > 0.12 sec.)
  - Right BBB (V1, V2, MCL1: rsR' pattern)
  - Left BBB (L1, V5, V6: upright QRS with a notch)
Practice: Bundle Branch Block

RBBB (Right Bundle Branch Block)
- V₁ & V₂

LBBB (Left Bundle Branch Block)
- V₅, V₆
  (& I, aVL)

Which Bundle Branch is Blocked?

1. Right Bundle Branch Block (Lead V₁)

Which Bundle Branch is Blocked?

2.
Left Bundle Branch Block (L I, V5, V6)

Where is the Pathology?

Right Bundle Branch Block

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

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), MI

How to calculate Axis

Easiest: the computer does it for you!

Easy: find the tallest R wave (if tallest is Lead II = normal axis)

Even easier: if Lead II is upright = normal axis

Funnest: Thumbs up / Thumbs down

Calculating Axis: Thumbs Up / Down Method

Lead I — Your Left thumb

Lead aVF — Your Right thumb

Calculating Axis: Thumbs Up / Down Method

Practice: Axis

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Left Axis Deviation

Right Axis Deviation

Extreme Right Axis Deviation
New 12-Lead ECG Format

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.

Normal | Ischemia | Injury | Infarction
--------|---------|-------|---------
Normal ECG | ST segment depression | T wave inversion | Path Q wave
Excess myocardial damage | Unstable Angina: (ST segment elevation) | Myocardial Infarction: (ST segment elevation)
Serum cardiac marker release

STEMI

Acute Regional Myocardial Infarction

Figure 1

Thrombus formation in the lumen of plaque rupture. Platelets adhere to the surface of the ruptured plaque and aggregate into a platelet-rich “white clot” prone to thrombosis and atherothrombosis. This white clot may be composed of platelets, red blood cells, and fibrin. Failure to treat the platelet-secreted serotonin can lead to the formation of a thrombus that forms an occlusive or macroscopic of the underlying plaque. Post-surgery or major trauma, and the presence of the drugs that accelerate the degradation (plasminogen, tPA enzyme) fibrin clot results in reperfusion and the retraction of the clot.
Percutaneous Coronary Intervention

RCA before and after stenting

STEMI: ECG Changes

O. 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)

STEMI — Typical Progression

Myocardial Infarction: ECG Patterns

Why Pathologic Q Waves Form

(If signs of AMI are not present on the initial ECG — perform serial ECGs)

(STEMI — Typical Progression)
Acute Inferior MI

ST elevation
Qs
Axis is shifting leftward...

Same Patient ~2 hrs later
Worsened ST elevation
Qs

New ST elevation

Same Patient 9 days later
Permanent Q waves (inferior wall scar)
But NO anterior infarct (no Qs)
Permanent left axis deviation

45% of MIs
Acute Anterior MI

40% of MIs
Acute Inferior MI

1/3 of Inferior MIs
Acute Right Ventricle MI

*Note: LVH also can cause poor R wave progression & Q waves in V1-V3. Rule it out first.

The anterior descending branch of the left coronary artery is occluded. May cause: Left anterior hemiblock, Right bundle branch block, BBB, 2 AV block Mobitz II, 3 AV block 2:1 RII, CHF, pump failure.

The right (or left) coronary artery is occluded. May cause: left posterior hemiblock, left axis deviation, LBBB, sinus bradycardia, 1 AV block, 2 AV block Mobitz I (Wenckebach), 3 AV block 4:1 R II.

Right ventricle AMI accompanies Inferior AMI 30% of the time. Check Lead V2R for elevated ST segment & Q wave.

RCA is occluded. May cause: AV block, A-Fib, A-VLATER, right heart failure, JVD with clear lungs, BP may drop if preloaded is reduced (be cautious with morphine, NTG, furosemide). Treat hypotension with IV fluids, pacing.
15% of MIs

**Acute Lateral MI**
(ST segment elevation ≥ 1mm in Leads: I, AVL, 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.*
- The right coronary artery or the circumflex branch of the left coronary artery is occluded. May cause: Sinus arrest.

**Practice: Infarct Location**

Where is the Pathology? 7

**Acute Anterior MI**
(ST Elevation in V1 - V4)

What is the R wave axis?

Where is the Pathology? 8
Acute Inferior MI
(ST elevation in II, III, F)

Acute Inferolateral MI
(ST elevation in II, III, F, V5, V6)

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

Acute Inferior MI & Right Ventricle MI

- V1, V2, V3
- Large R Waves
- Depressed STs
Acute Posterior MI

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

Normal V1 – V3

Confounders

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

WPW
False Q waves mimic MI
ST Depression
(a diagnostic challenge)

Can be caused by:
- Ischemia
- Digoxin effect
- Tachycardia
- LVH, BBB
- Hypokalemia
- NSTEMI (Non Q wave MI)

Ischemic ST Depression
(a positive exercise ECG)

Exercise-induced ischaemic changes

Rest

Exercise

Practice: Confounders

44 y.o. female with history of tachycardia

WPW (short PR, Wide QRS, Delta waves)

30 y.o. male with positional chest pain
Pericarditis

Where is the Pathology?

Putting it all together

Right Bundle Branch Block

Left Bundle Branch Block
What rhythm is this?

Acute Anterior MI

Elevated ST segments

Anterior MI

Sinus Tachycardia

Acute Anteroseptal MI

Elevated ST segments

Acute Inferolateral MI

Elevated STs II, III, aVF, V5, V6
Anterolateral Ischemia

Junctional Rhythm

Rhythm? MI?

Junctional Rhythm; Acute Anterolateral Ischemia

MI?

Acute Inferolateral MI ECG

Rhythm? Pathology?

Large Old Anterolateral MI

Ventricular aneurysm
Case report:
58 y.o. male c/o chest "tightness" and shortness of breath x 20 minutes, which gradually subsided. Recurrent episodes over several months. Pt thought it was "acid reflux", but finally goes to ED. Pt is noncompliant with statin therapy, & admits to poor diet. Family Hx cardiac disease. Hx HTN. Meds: Plavix, ACE inhibitor.

EKG follows. What treatment?

Angiography reveals 90% occlusion in some coronary arteries.

Treatment: quadruple CABG (coronary artery bypass graft).

Excellent outcome:
Pt is active, healthy, has improved diet, is compliant with meds; and has inspired thousands of Americans to go to their physician for cardiac evaluations…

“The Bill Clinton Effect”