Objectives

1. Recognize the presence of anterior and posterior fascicular block, bifascicular blocks, and trifascicular blocks on the 12 lead ECG and discuss the clinical implications of these findings.

2. Evaluate ECG changes in the QT interval, ST segment, and T waves that indicate drug effect or electrolyte imbalances.

3. Identify ST elevation MI, non-ST elevation MI, and Wellen’s Warning on the 12 lead ECG and recognize ECG signs that indicate high risk in these patients.

I have no commercial interest or conflict of interest related to this presentation
Steps in Reading a 12 Lead ECG

- Rate
- Rhythm
- Axis
- P Waves
- PR Interval
- QRS Complex
- ST Segment
- T Waves
- U Waves
- QT Interval

In a patient having chest pain, the #1 priority is to look for ST elevation!

How Leads Record

- Positive electrode is the recording electrode or “camera lens”
- Negative electrode or reference point tells camera which way to shoot
- If positive electrode sees depolarization approaching it, it records an upright complex
- If positive electrode sees depolarization heading away from it, it records a negative complex.

If depolarization is proceeding perpendicular to a lead, no deflection is recorded.
Fascicular Blocks (Hemiblock)

Conduction System Anatomy

AV Node → Bundle of His → Right Bundle Branch → Main Left Bundle Branch

Anterior Fascicle → Posterior Fascicle

RV → LV
Blood Supply to Conduction System

- SA Node
  - RCA in 60%
  - Circumflex (LCx) in 40%
- AV Node
  - RCA in 90%
  - Circumflex in 10%
- His Bundle
  - RCA (AV nodal branch)
  - Minor supply from septal perforators from LAD
- Main LBB
  - Mostly LAD
  - Collateral from RCA, LCx
- Left Anterior Fascicle
  - Mostly LAD septal perforators
  - AV nodal artery contributes in some
- Left Posterior Fascicle
  - LAD septal perforators
  - Posterior descending
- Right Bundle Branch
  - LAD septal perforators
  - Some collateral from RCA or LCx (whichever is dominant)
Clinical Significance of Fascicular Block

- Represents conduction failure in one of the two main branches of the left bundle branch.
  - Especially significant when RBBB is also present
- May mimic or mask the ECG signs of infarction or ischemia
- May mimic or mask ventricular hypertrophy.

The best leads for recognizing hemiblock are:
- I and aVF for the QRS axis
- I and III for the pattern of hemiblock

Normal LV Depolarization

QRS = .06 - .10 sec wide
Left Anterior Fascicular Block (LAFB)

- Common
  - Smaller than posterior fascicle
  - Anterior fascicle has mostly one blood supply – from septal perforators of LAD (minor from AV node artery)
  - Located in LV outflow tract so subjected to high pressures
- Does not widen QRS but does cause axis deviation
  - QRS can be wider in presence of MI or hypertrophy

Causes of LAFB

- Anterior or anterolateral MI (same blood supply)
- Hypertension
- Cardiomyopathies
- Aortic valve disease
- Lev and Lenègre diseases
- Myocarditis
- Surgical closure of a VSD

Isolated LAFB is not a risk factor of cardiac morbidity or mortality, and in a healthy population it should be regarded as an incidental ECG finding.
**Left Anterior Fascicular Block**

- QRS = .06 - .11 sec wide

**Criteria for LAFB**

- **Left Axis Deviation:** -45° to -90°
- **qR in lead I** (and aVL)
- **rS in II, III, aVF** (deeper S in III than in II)
- **QRS width < .12 sec**
- Often increased QRS voltage due to unopposed forces in LV (can mimic LV hypertrophy)
What is the QRS axis? (-60⁰) Is there a pattern of hemiblock?

Tiny Q in I, aVL; rS in II, III, aVF (deeper S in III than in II)

LAH can mimic or mask infarction or ischemia

In every lead the 1st beat is normal conduction and the 2nd beat is LAH

Axis shifts from 0⁰ to -40⁰

In leads II, III, aVF during normal conduction – T wave inversion of ischemia. With LAH T waves are upright, masking ischemia.

Q wave in aVL mimics lateral MI

Causes of Left Axis Deviation

- Left anterior hemiblock
- Left ventricular hypertrophy
- Inferolateral MI
- CAD
- Ventricular beats, VT
- Ventricular pacing
- WPW syndrome - R sided accessory pathway
- Emphysema
- Horizontal heart position: pregnancy, short build

If the S wave in lead II is deeper than the S wave in lead III, LAH is very unlikely

Left Posterior Fascicular Block

- Rare – especially in isolation
  - Thicker than anterior fascicle
  - Located in LV inflow tract so low pressures
  - Has two blood supplies: LAD and posterior descending artery
- More often seen with RBBB: dangerous!
- Does not widen QRS but does cause axis deviation
Causes of LPFB

- Does not occur in healthy people without cardiac disease
- Associated with many myocardial diseases – Lenegre’s disease, Chagas disease, myocarditis
- When it occurs in acute MI it is usually associated with RBBB and carries a poor prognosis

Left Posterior Fascicular Block (LPFB)

QRS = < .12 sec wide
Criteria for LPFB

- Right Axis Deviation ($\geq +100^\circ$)
- rS in I and aVL
- qR in II, III and aVF
- Normal QRS width
- No RVH (right ventricular hypertrophy)

Right axis deviation, S1, QIII, no RVH

Whenever QRS in lead I is mostly negative:

- Most common cause is arm lead reversal!
- Re-do the ECG and make sure arm leads are on the correct arms!!
- If so, then there is right axis deviation
Limb Leads

What is the QRS axis? (RAD)
Is there a pattern of hemiblock? rS in lead I; Q in lead III

V Leads: evaluate R wave progression

Any signs of RVH? No. R wave progression is normal
What is the axis? **Right axis deviation**

Is there a pattern of hemiblock?

rS in I and aVL, and qR in III and aVF = typical of LPH

V leads show huge R wave in V1 and reversal of precordial pattern = RVH

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**Causes of Right Axis Deviation**

- Normal in children and tall thin adults
- **Left posterior hemiblock**
- Right ventricular hypertrophy
- Right bundle branch block
- Chronic lung disease
- Pulmonary hypertension
- Lateral myocardial infarction
- Pulmonary embolus
- WPW syndrome - L sided accessory pathway
- Dextrocardia

**Rightward shift of prior normal axis: think PE**
Hemiblock Practice #1

Hemiblock Practice #2

Limb Leads
V Leads

Hemiblock Practice #3
V Leads

Hemiblock Practice #4
Bifascicular Block: 
RBBB + LAFB

Because the RBB shares the same blood supply with the anterior fascicle, one or both together are likely to occur in anterior or anteroseptal infarction

- Left axis deviation
- Pattern of LAFB = QI, SIII or rS in II, III, aVF
- Pattern of RBBB and QRS 0.12 sec. or more

Axis

Q in I, S in III (deeper S in III than in II) = LAFB
V1 has wide QRS, rSR’ = RBBB
Q in I, S in III (deeper S in III than in II) = LAFB
V1 has wide QRS, rSR’ = RBBB

**Bifascicular Block:**
**RBBB + LPFB**

- Right axis deviation
- Pattern of LPH = SI, QIII
- Pattern of RBBB and QRS 0.12 sec or more
- No RVH

LPH plus RBBB in acute MI is associated with a high mortality rate (80% to 87%) and high incidence of complete heart block requiring permanent pacemaker.
S in I, Q in III (rS in I and aVL) = LPFB
V1 has wide QRS, qR pattern = RBBB

S in I, Q in III (rS in I and aVL) = LPFB
V1 has wide R wave = RBBB
Trifascicular Block

- Can be complete or incomplete
- What would complete trifascicular block look like?

Incomplete Trifascicular Block

- Bifascicular block with evidence of delayed conduction in the 3\textsuperscript{rd} fascicle
  - RBBB with either LAFB or LPFB and 1\textsuperscript{st} degree AVB
  - RBBB with either LAFB or LPFB and intermittent blocked P waves
- RBBB with alternating LAFB and LPFB
What is the rhythm?  PR = .32 sec

Axis

Q in I, S in III (deeper S in III than in II) = LAFB
V1 has wide QRS, rSR’ = RBBB

What is the rhythm?  PR = .24 sec

Axis

No Q in I, S in III = LAFB
V1 has wide QRS, rSR’ = RBBB
Drug and Electrolyte Effects

- ECG changes are nonspecific and many can occur with either drug effect or electrolyte imbalances
  - QRS width
  - ST segment
  - T waves
  - QT interval
- ECG is suggestive but not diagnostic
**Digitalis**

- **ST segment & T waves**
  - T wave flattening, inversion
  - “Scooped” or “sagging” ST segment (common indication of digitalis effect)
- **QT interval shortens**
- **U waves may develop or increase in size**
- **PR interval may prolong due to AV block**
- **Toxicity causes many arrhythmias**

- Typical ST segment sagging in many leads
- 1st degree AV block
Rhythm? Atrial fibrillation
ST sagging in several leads

Drug Effect on the QT Interval

- Long QT interval reflects abnormally prolonged ventricular repolarization time
  - QTc normally < 460 msec (.46 sec) in men and < 470 msec (.47 sec) in women
  - QTc ≥ 500 msec dangerously long and increases risk of Torsades
- Drugs that block K⁺ channels prolong QT
- Hypokalemia and hypomagnesemia can contribute to or cause long QT interval
What You Should Know About the QT Interval

- QT interval is heart rate dependent
  - Shortens at fast heart rates (short R-R interval)
  - Lengthens at slow heart rates (long R-R interval)
- Measurement must be corrected for heart rate
  - Bazett formula often used for correction
    \[
    QTc = \frac{QT}{\sqrt{R-R}}
    \]
    All measurements in seconds
- Guestimate: QT should be no more than half the preceding R-R interval

Drugs That Prolong the QT Interval

- Antiarrhythmics
  - Class IA: quinidine, procainamide, disopyramide
  - Class III: sotalol, ibutilide, dofetilide, amiodarone
- Antidepressants (tricyclics most common)
- Anti-psychotics (thorazine, haldol, many others)
- Antibiotics (erythromycin, pentamidine, levaquin, many others)
- Anticonvulsants (carbamazepine)
- Anti-fungals
Patient overdosed on amitriptyline (Elavil – antidepressant)

- Measured QT = .64 sec
- QTc = .66 sec

Antihistamine overdose

- QT = .74 sec (740 msec)
- QTc = 795 msec
• Sinus bradycardia (rate 43)
• QT prolongation (QTc = 560 msec)

Sotalol Toxicity

Drug Effect on QRS Complex

➢ Sodium channel blocking drugs widen the QRS complex
  • Tricyclic antidepressants (and other types of antidepressants)
  • Antipsychotics (phenothiazines)
  • Antiarrhythmics (Class IA & IC)
  • Antimalarials
  • Cocaine

➢ Intraventricular conduction delay – QRS >.10 sec

➢ Right axis deviation of the terminal part of QRS
  • S waves in I, aVL
  • Terminal R wave ≥3 mm in aVR (predicts arrhythmias in patients with tricyclic antidepressant poisoning)

**TCA Overdose** (doxepin)

- Wide QRS
- Right axis of terminal QRS
  - S waves I, aVL
  - R wave aVR

**Tricyclic antidepressant overdose** (dothiepin)

- Wide QRS
- Rightward axis of terminal QRS
  - S wave in I
  - R wave in aVR
Drug Effect on ST Segment, T waves, U waves

- Nonspecific ST-T wave changes
  - T wave flattening or inversion
  - ST segment sagging
  - These are primary ST-T changes due to changes in repolarization of the cardiac action potential (not secondary to abnormal depolarization)

- Enlarged U waves

Patient on digoxin and quinidine
Rollercoaster appearance of ST-T waves
Hypokalemia can cause large U waves too
ECG Signs of Electrolyte Imbalances

Potassium
Calcium
Magnesium

Electrolyte Effects

- Nonspecific ST-T changes
- ECG is suggestive, not diagnostic
- $K^+$ and $Ca^{++}$ imbalances can cause ECG changes
  - Heart is significantly affected by too much or too little potassium
- $Mg^{++}$ imbalances don’t affect ECG but can cause arrhythmias
Hypokalemia
Normal: 3.5 - 5.5mEq/L

- ST segment depression
- T wave flattening
- Large U waves
  - Sometimes merging with the T wave to create a “camel hump” appearance

- Increases risk of Torsades and other ventricular arrhythmias
- Increases risk of digitalis toxicity

K+ = 2.0
Hyperkalemia
Normal: 3.5 - 5.5mEq/L

- ECG changes progress as K⁺ rises, but can’t determine K⁺ level from ECG
- Mild elevation (around 5.5-6.5 mEq/L)
  - Peaked T waves, often narrow at the base and resembling a thorn.
  - Suggestive of hyperkalemia but can also be seen with ischemia or as a normal variant.
- Moderate elevation (6.5-8 mEq/L)
  - PR interval prolongs
  - P wave amplitude decreases and eventually P waves disappear
  - ST segment elevation
  - QRS begins to widen
- Severe elevation (> 8 mEq/L)
  - Further widening of QRS progressing to “sine” wave pattern
  - Ventricular fibrillation
$K^+ = 7.8 \text{ mEq/L}$

$K^+ = 9.6 \text{ mEq/L}$
K+ = 9.2 meq/L

**Hypocalcemia**

Normal serum Ca**++** = 8.6 – 10 mg/dl;
Ionized (not attached to proteins) = 4.64 – 5.28 mg/dl

- Prolongation of ST segment
- QT prolongs, but is due to long ST rather than repolarization abnormality
- Usually does not cause arrhythmias
Ca++ = 8.2 mg/dl

Hypocalcemia (level unknown)
Hypercalcemia

- Short ST segment
  - End of QRS takes off to peak of T wave
  - This can also occur with acute STEMI
- Short QT interval
- Osbourn waves (J waves) at end of QRS with severe hypercalcemia
- VF can occur with severe hypercalcemia
ECG of a 41-year old man with parathyroid cancer who presented to ED with a serum calcium of 24.4 mg/dl. He had a VF arrest not long after this ECG was taken.

The ECG in Acute Coronary Syndromes
Signs of Increased Risk for Complications
ECG Changes in ACS

ECG Criteria for STEMI:
ST elevation at the J point of 1mm or more in at least two contiguous leads

I (Circ)  aVR  V₁ (LAD)  V₄ (LAD)  
II (RCA)  aVL (Circ)  V₂ (LAD)  V₅ (Circ)  
III (RCA)  aVF (RCA)  V₃ (LAD)  V₆ (Circ)

Lateral Wall: I, aVL, V₅, V₆
Anterior Wall: V₁-V₄
Inferior Wall: II, III, aVF
Septum: V₁, V₂
Threshold Values for Abnormal ST Changes (J point elevation)

- **Men > 40 years old**
  - 2 mm in V2 and V3
  - 1 mm in all other leads
- **Men < 40 years old**
  - 2.5 mm in V2 and V3
- **Women**
  - 1.5 mm in V2 and V3
  - 1 mm in all other leads
- **Men and women**
  - 0.5 mm in V3R and V4R (right ventricle)
  - 0.5 mm in V7-V9 (posterior wall)


Primary versus Secondary ST Segment and T Wave Changes

- **Secondary ST-T wave abnormalities**
  - Repolarization abnormality due to depolarization abnormality (BBB, WPW, ventricular beats, ventricular hypertrophy)
  - ST segment and T waves directed opposite to terminal QRS (discordant)
- **Primary ST-T wave abnormalities**
  - Due to abnormalities unrelated to abnormal depolarization (ischemia, drug effect, electrolytes, pericarditis, PE, Wellens Syndrome)

Terminal T wave inversion

- Wellen’s Syndrome when seen with unstable angina in V2, V3 = ischemia due to critical proximal LAD stenosis
- Expected during evolution of STEMI as ST elevation resolves and T waves invert

Deep symmetrical T wave inversion

- Ischemia if no Q waves of infarction
- Commonly follows terminal T wave inversion in Wellen’s Syndrome
- Expected evolution of T waves following infarction

T wave inversion where it should be upright

- Ischemia
- Ventricular hypertrophy

Ischemia

ST Segment Depression

Digitalis effect

LVH
ST changes with acute coronary syndrome
- ST elevation is associated with transmural injury
- ST depression (that is not reciprocal) is associated with subendocardial ischemia – UA or NSTE-ACS

NSTE-ACS has lower in-hospital mortality but similar or worse long-term outcome than STEMI
- STEMI associated with larger infarctions (higher in-hospital mortality)
- NSTE-ACS leaves more myocardium at risk for future ischemic events or infarction (worse long-term outcome)

Resolution of ST segment elevation following PCI or fibrinolysis is a marker of reperfusion and coronary artery patency and is associated with smaller infarct size and better short term and long term outcomes.
- We expect to see ST down by 50% within 60-90 minutes after successful reperfusion.
- Failure of ST elevation to resolve by at least 50% within 60-90 minutes after fibrinolysis is indication for rescue PCI.


ECG signs indicating worse prognosis:
- Anterior worse outcomes than inferior infarcts
- Q waves on admission ECG
- A greater number of leads showing ST elevation
- Lack of ST elevation resolution at 90 to 180 minutes after reperfusion

Resolution of ST segment elevation following PCI or fibrinolysis is a marker of reperfusion and coronary artery patency and is associated with smaller infarct size and better short term and long term outcomes.
- We expect to see ST down by 50% within 60-90 minutes after successful reperfusion.
- Failure of ST elevation to resolve by at least 50% within 60-90 minutes after fibrinolysis is indication for rescue PCI.

**ST Elevation Recovery after PCI**

- **Single-lead ST elevation recovery**: percent reduction in ST-E from baseline ECG to post-PCI ECG in the lead with maximum baseline ST-E

  Post ECG ~ 30 min after PCI

<table>
<thead>
<tr>
<th>Death (%)</th>
<th>Shock (%)</th>
<th>CHF (%)</th>
<th>Death/Shock/CHF (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>≥ 50%</td>
<td>2.7</td>
<td>1.0</td>
<td>3.6</td>
</tr>
<tr>
<td>&lt; 50%</td>
<td>6.0</td>
<td>2.5</td>
<td>6.7</td>
</tr>
</tbody>
</table>

- **Worst-lead residual ST-E**: the absolute magnitude of residual ST-E in the most affected lead on post-PCI ECG

<table>
<thead>
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<th>Death (%)</th>
<th>Shock (%)</th>
<th>CHF (%)</th>
<th>Death/Shock/CHF (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;1 mm</td>
<td>1.7</td>
<td>0.6</td>
<td>1.7</td>
</tr>
<tr>
<td>1 to &lt;2 mm</td>
<td>2.5</td>
<td>0.6</td>
<td>3.5</td>
</tr>
<tr>
<td>≥ 2 mm</td>
<td>5.5</td>
<td>2.7</td>
<td>7.0</td>
</tr>
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**Acute Injury Patterns**

- **ST elevation of 1 mm or more in two contiguous leads** (baseline shown as dotted line)

- **ST segment pulled to peak of T wave**

- **Tall, wide-based T waves (hyperacute T waves)**

  T wave should be subordinate to the QRS
Progressive Changes of Infarction

- ST elevation usually first
  - Appears within minutes of pain and can last 3-4 days (early reperfusion drastically shortens time)
- Q waves can appear immediately but more commonly appear within hours of pain
  - Usually permanent but may disappear with early reperfusion
- T wave inversion occurs within hours and can last for months.
  - Can return upright or remain inverted
  - Early return to upright is NOT a good sign!

Serial ECGs from same patient
**Anterior/Anterolateral Wall MI**

- LAD supplies anterior wall & anterior 2/3 of septum
  - Most of the RBB and LBB
  - Main supply to left anterior fascicle
  - Anterior papillary muscle of mitral valve
  - Branches can supply part of lateral wall

- ECG Leads
  - Leads $V_1$ and $V_2$ look at septum
  - Leads $V_3$ and $V_4$ look at anterior wall
  - Anteroseptal MI = occlusion of proximal LAD - $V_1$-$V_4$

- Occlusion in distal LAD beyond 1st diagonal branch = low anterior wall
  - ST elevation in V2-V6 (often less in V2)
  - No ST elevation in V1, aVL, or aVR
  - No ST depression in II, III, aVF
- Occlusion between 1st septal and 1st diagonal = anterior wall (and lateral wall?)
- V1 not elevated
- ST elevation in aVL, V2-V4
- ST depression in III

- Occlusion of proximal LAD above 1st septal and 1st diagonal branch = base of LV, anterior and lateral walls, septum
- ST elevation in V1-V4, I, aVL (often aVR)
- ST depression in II, III, aVF (often V5)
Wellens Syndrome

- History of unstable angina
- ECG Changes (present without chest pain)
  - **Terminal T wave inversion** V1-V4 (especially V2,V3) (24%) OR **deep symmetrical T wave inversion** V1-V4 (76%)
  - No Q waves
  - No or minimal (<1 mm) ST elevation
  - Normal R wave progression
- Normal or minimally elevated cardiac biomarkers
- Indicates critical proximal LAD stenosis
- It not treated can result in large anterior MI
  - 75% of patients in original study infarcted within a mean of 8 days if not reperfused

Very slight ST elevation in V1-V3
Terminal T wave inversion V1-V3

Complications of Anterior/Anterolateral MI

- Heart Block (less common than with inferior MI)
  - Infranodal block: Type II second degree, 3rd degree
  - Often without warning, may be preceded by bifascicular block
  - Unstable ventricular escape rhythm
  - 80% mortality due to size of infarct, pump failure
- LV Free Wall Rupture (more common than inferior MI)
- Septal Rupture (more common in apical septum)
- Heart Failure
- Cardiogenic Shock – higher incidence and lower survival with LAD lesions than with RCA lesions
**Inferior Wall Blood Supply**

- RCA is the dominant artery in ~ 90% of people
- Circumflex is dominant in ~ 10% of people
- The dominant artery provides the posterior descending artery that supplies the inferior wall, posterior 1/3 of septum, and the AV node

**Inferior STEMI**

- ST elevation only in II, III, aVF can be due to RCA or Circumflex occlusion (whichever is dominant)
  - With RCA occlusion, ST elevation in III > II and there is reciprocal ST depression in aVL and lead I
  - With LCx occlusion, ST elevation in II often > III and there may be ST elevation in I and aVL (lateral wall involvement)
- ST depression in V1-V3 can be due to RCA or LCx occlusion and is indicative of posterior wall MI
  - Absence of ST depression in V1-V3 more common with RCA occlusion
- ST depression in V leads is associated with larger infarcts, higher incidence of 3-vessel disease, and more complications

- Occlusion of distal RCA – inferior MI
- ST elevation in II, III, aVF
- Often ST elevation in III > II
- Reciprocal ST depression in aVL

- Occlusion of proximal dominant RCA – inferior MI and often RVMI and/or posterior MI
- ST elevation in II, III, aVF, and V1 typical of RVMI
- Record right side leads V3R and V4R in all patients with ST elevation in II, III, aVF
- V4R is best lead for identifying proximal RCA occlusion and RV infarction
12 Lead Clues to RV Infarction

- ST elevation in II, III, AVF, V₁
- ST elevation in III > II or AVF
- ST discordance between V₁ and V₂

- ST elevation II, III, aVF, V₁
- ST III > II
- ST discordant between V₁ and V₂
- Reciprocal ST depression I, aVL
- Occlusion of circumflex – lateral MI if RCA is dominant artery (isolated lateral MI is rare)
  - ST elevation in I, aVL, V5, V6
- Occlusion of circumflex if it is dominant – lateral, posterior, and inferior MI
  - ST elevation in II, III, aVF, ST depression in V1-V3 = posterior MI

- ST elevation I, aVL
- Reciprocal ST depression in II, III, aVF
- Lateral MI due to non-dominant circumflex
- ST elevation II, III, aVF (inferior)
- ST elevation in V4-V6 (lateral)
- ST depression I, AVL (reciprocal to inferior wall)
- ST depression in V1-V3 (reciprocal to posterior wall)
- Inferior / lateral MI due to dominant circumflex artery

- ST elevation in I, V4-V6 = lateral wall
- ST elevation in II, III, aVF = inferior wall
- ST depression V1-V3 = posterior wall (reciprocal)
- Dominant circumflex artery
Posterior Wall MI

- None of the standard 12 leads looks at the posterior wall
- Reciprocal ST depression and large R waves in V1-V3
- Should record posterior leads V7, V8, V9

Normal $V_1$ – $V_3$

Posterior MI
- ST elevation II, III, aVF (inferior wall)
- ST depression I, aVL (reciprocal to inferior wall)
- ST depression and tall R waves in V1-V3 (reciprocal to posterior wall)

- Large R waves and 1mm ST depression V1-V3
- 1mm ST elevation in I, aVL
- Posterior wall MI
18 Lead ECG

- Record right side leads with all inferior wall STEMI (ST elevation in II, III, AVF)
- Record posterior leads (V₇-V₉) with ST depression in V₁-V₃ (with or without ST elevation in other leads)
- Might as well record 18 lead ECG in all patients except straight forward anterior wall MI
  - Adds 3 posterior leads and 3 right side leads to standard 12 lead ECG
Use the V Lead as a Rover

- Normal chest lead position for V1
- Chest lead at V4R for looking at RV
- Chest lead in posterior position for looking at posterior wall

Complications of Inferior MI

- Sinus bradycardia (40% of patients in first 2 hrs)
- Heart block (more common than with anterior MI)
  - Intranodal: 1st degree, Wenckebach
  - 3rd degree, progresses from 1st and 2nd degree, narrow QRS junctional escape rhythm
  - Usually transient, may need temporary pacing
- Ventricular or septal rupture (more common at base of septum)
- Papillary muscle rupture and acute mitral regurgitation
  - Posterior papillary muscle has one blood supply from RCA
  - Anterior papillary muscle has dual supply from LAD and LCx
Unstable Angina & NSTE-ACS

ECG looks the same: ST depression or T wave inversion
Cardiac biomarkers needed for differential diagnosis

Patterns of Ischemia

<table>
<thead>
<tr>
<th>ST depression and T wave inversion concordant with terminal portion of QRS</th>
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</thead>
<tbody>
<tr>
<td>Horizontal or downsloping ST-segment depression with upright or biphasic negative-positive T wave</td>
</tr>
<tr>
<td>Terminal T wave inversion (biphasic positive-negative T wave) in V₃-V₅ (also called &quot;Wellen's warning&quot;)</td>
</tr>
<tr>
<td>Deep symmetrical inverted T waves with isoelectric, slightly up-sloping, or horizontally depressed ST segment</td>
</tr>
<tr>
<td>Tall, wide T waves</td>
</tr>
<tr>
<td>U wave inversion</td>
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ECG in UA/NSTEMI

- Major ECG factors that predict risk are the # of leads and location of ST segment depression.
  - ST depression ≥ 2mm increases risk of death at 1 year
  - The more leads with ST depression the higher the 30 day mortality
- ST depression is associated with worse outcome than T wave inversion alone.
  - Patients with ST depression more likely to benefit from early invasive strategy than those with T wave inversion

- Two patterns of ST-segment depression reflect STEMI rather than NSTE-ACS
  - ST-segment depression that is reciprocal to a subtle and often overlooked ST-segment elevation
  - ST-segment depression that is maximal in leads V1-V3, suggesting true posterior infarction.
- T-wave inversion in the inferior and anterior precordial leads can be seen in acute PE
- Flattened T waves with prominent U waves and ST-segment depression may reflect hypokalemia or digitalis therapy.

- Patient eventually developed obvious ST elevation in II, III, aVF
- T wave inversion in V leads indicates more extensive disease

- In patients with angina at rest, ischemic changes in at least 8 leads with ST elevation in aVR &/or V1 indicates high likelihood of significant **left main** coronary artery stenosis or significant **triple vessel disease** (75% predictive accuracy)
- ST elevation higher in aVR than in V1 indicates left main disease
84 year old woman with chest pain and elevated troponin
A quick case study

Admission ECG

Rhythm?
Axis?
BBB?
Any signs of ischemia or infarction?

While waiting to go to cath lab a repeat ECG was done about 15 minutes later

Axis?
BBB?
What is the risk?
Recommendations for the Standardization and Interpretation of the Electrocardiogram:

Part I: The Electrocardiogram and Its Technology  
(Circulation 2007;115;1306-1324)

Part II: Electrocardiography Diagnostic Statement List  
(Circulation 2007;115;1325-1332)

Part III: Intraventricular Conduction Disturbances  
(Circulation. 2009;119:e235-e240.)

Part IV: The ST Segment, T and U Waves, and the QT Interval  
(Circulation. 2009;119:e241-e250.)

Part V: Electrocardiogram Changes Associated With Cardiac Chamber Hypertrophy  
(Circulation. 2009;119:e251-e261.)

Part VI: Acute Ischemia/Infarction  
(Circulation. 2009;119:e262-e270.)