Advanced 12 Lead ECG Interpretation

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Welcome to NTI 2014
We are going to have a great day and a wonderful week!
Advanced Concepts in 12 Lead ECG Interpretation
Part 1

Prerequisite Review
Hemiblocks
Chamber Enlargement
Electrolyte and Drug Effects

Karen Marzlin DNP, RN, CCNS, CCRN-CMC, CHFN
Key Concepts You Should Know

• Definitions of Q, R, S waves
• Anatomy of a 12 Lead ECG
  – Which leads look where
  – How leads record
• QRS Axis Calculation
• Right and Left Bundle Branch Block

QRS Complexes

• Q wave
  – Initial negative deflection from baseline

• R wave
  – Always an upright deflection
  – If 2 are present, second one is R’

• S wave
  – Negative deflection following an R wave
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.

Anatomy of a 12 Lead ECG

Limb Leads: I, II, III, aVR, aVL, aVF

Precordial (Chest) Leads: V1, V2, V3, V4, V5, V6

Frontal plane leads: up/down, right/left

Horizontal plane leads: anterior/posterior, right/left
6 Limb Leads

6 Precordial (Chest) Leads
Inferior Leads

Anterior Leads
Lateral Leads

Axis Quadrants:

Adults:
-30° to +90°

Normal Axis

Note: Normal axis is extended into the left quadrant by 30 degrees.
Axis Quadrants: Leftward Axis and Left Axis Deviation

**Causes:**
- Aging (changes in left anterior fascicular conduction)
- Left ventricular hypertrophy
- Acute MI (peri infarction block)
- Congenital heart disease
- Hyperkalemia
- **Marked:** Associated with left anterior fascicular block

Axis Quadrants: Right Axis Deviation

**Causes:**
- Infants / Children
- RV Hypertrophy
- Chronic Lung Disease
- Pulmonary Hypertension
- **Pulmonary Embolus**
- **Marked:** Often associated with left posterior fascicular block
Axis Quadrants:
Extreme (Right Superior) Axis Deviation

Causes:

• Ventricular Tachycardia
• Other significant conduction abnormalities

Calculating Degree of Axis

• Before you consider the degree of axis – always know the direction.
• Which quadrant do you expect?
Calculating Degree of Axis “ESPN” Method – Step 1

• **E=Equiphasic**
  - Find the QRS complex in the limb leads which is the most equiphasic
  - OR
  - Find the smallest QRS complex (height of R wave minus depth of S wave)

  • We are looking for the lead recording closest to baseline

“ESPN” Method – Step 2

• **S=Sister Lead**
  - Utilizing the “Criss-Cross” method Find the “Sister” lead to the lead with the most equiphasic QRS complex
“ESPN” Method – Step 3

- **P/N = Positive or Negative**
  - Is the “Sister” lead positive or negative?

“ESPN” Method – Step 4

- Go to the AXIS Wheel
- If positive: locate the positive pole of that lead
- If negative: locate the negative pole of that lead
- What degree of AXIS is assigned to the positive or negative pole of the sister lead
Right Bundle Branch Block

- **V1**
  - Triphasic complex rsR’ (rsr’, rSR’) pattern
  - Or an M shaped R wave with right peak taller
  - Or a qR pattern (in patients with septal infarct)

- **V6**
  - Triphasic complex
  - Large R is maintained
  - qRs with wide S waves
  - Note: Also a wide S wave in lead 1

QRS = .12 sec or more
Left Bundle Branch Block

- $V_1 = QS$
- $V_1 = rS$
- $V_6 = $ wide R
- QRS = .12 sec or more
Left Bundle Branch Block

• **V1**
  – Wide QS or rS complex - negative
  – Slick downstroke
  – Time to nadir <0.06 sec

• **V6**
  – Large R wave is maintained
  – Wide R wave with no initial septal q wave

Additional diagnostic criteria include:
Broad slurred or notched R waves in leads 1,aVL, V5, and V6.
Left Anterior Hemiblock (LAHB)

Causes
- Disease of left ventricular outflow tract
- Ischemia / injury of anterior septum or anterior lateral wall
- HTN
- Coronary artery disease
- Aortic valve disease
- Cardiomyopathy
- Closure of septal defect
- Surgical complication
- Lev and Lenègre diseases

Implications
- May be incidental finding in a healthy person
- Not benign in a hospitalized patient
  - Associated with increased mortality and morbidity in patients with coronary artery disease or myocardial infarction
  - In association with RBBB: very high risk
- May conceal the ECG signs of myocardial infarction and left ventricular hypertrophy

Anterior fascicle is vulnerable: All blood supply from septal perforator of left anterior descending artery.
Left Anterior Hemiblock Recognition

- **Lead 2, Lead 3 and aVF**
  - rs pattern
  - Small r waves
  - Slightly wide / deep S waves
  - Increased limb lead voltage
- **Lead 1 and aVL**
  - qR pattern

Note: Diagnostic criteria is qR in aVL with a R peak time of > 45 msec or >

Left Anterior Hemiblock: Additional Diagnostic Criteria

- Normal QRS duration
- Left axis deviation
  - -45° to -90°
  - Common at – 60°

Clinical Pearl:
Key for recognizing -60° Axis
aVR is most equiphasic limb lead in presence of leftward axis
Handy method for axis tells direction of depolarization: Left
ESPn method: II and aVR are most equiphasic. Degree of deviation about -45

Handy method for axis tells direction of depolarization: Left
ESPn method: aVR is most equiphasic. Degree of deviation about -60
Note lead aVR is most equiphasic in presence of leftward axis.
Also note the 1º degree AVB. This used to be called trifascicular block.

Note lead aVR is the most equiphasic in presence of leftward axis.

INTERESTING: Lead 1 is most equiphasic in presence of leftward axis.
Let’s Do the ESPN Method

- Lead 1 is most equiphasic
- Sister lead is lead aVF
- aVF is negative
Left anterior HemiBlock during acute MI

LAHB estimated to occur in approximately 7% to 15% of anterior / anteroseptal myocardial infarctions.

When combined with RBBB the stakes are higher.
Using the Bedside Monitor: V1 and Lead III
Recognition of Left Posterior Hemiblock

- rS in I and aVL

- qR in III and aVF

Left Posterior Hemiblock: Additional Diagnostic Criteria

- Right Axis Deviation (90° to 180°)

- Normal QRS

Let’s do the handy method.

Your right hand is UP.
Causes of Left Posterior Hemiblock

• Does not occur in healthy people without cardiac disease
• Associated with many myocardial diseases.
• When it occurs in acute MI it is usually associated with RBBB and carries a poor prognosis
Other Conduction Delays

- QRS duration > 110 msec (0.10 sec) in adults is considered abnormal
- Incomplete RBBB: QRS duration 110 to 120 msec with rsr’ in V1
- Incomplete LBBB: QRS duration 110 to 119 msec with presence of LV hypertrophy pattern
- QRS > 110 msec may also be a non specific conduction disturbance
- QRS width can change in response to injury or ischemia
  - Peri infarction block with a pathological Q wave
  - Peri ischemic block – transient with acute injury
Chamber Enlargement (Hypertrophy)

More on Chamber Enlargement

- Chambers can be enlarged for one of two reasons:
  - Increased pressure resulting in hypertrophy
    - Example: Aortic valve stenosis results in left ventricular hypertrophy
  - Increased volume resulting in dilatation
    - Example: Mitral valve regurgitation results in left atrial volume overload and subsequent enlargement

- The ECG is not the ideal tool for differentiating the cause of enlargement
Right Atrial Enlargement

• Right atrial enlargement is caused by conditions that increase volume or pressure in the right atrium
  – Tricuspid stenosis or regurgitation
  – High right ventricular pressure
  – Right ventricular hypertrophy
  – Pulmonary artery hypertension
  – Chronic lung disease
  – Pulmonic valve stenosis or regurgitation
  – Congenital heart disease

Left Atrial Enlargement

• Left atrial enlargement is caused by conditions that increase volume or pressure in the left atrium
  – Mitral stenosis or regurgitation
  – Systemic hypertension
  – Left ventricular failure
  – Left ventricular hypertrophy
P Waves

• P waves represent atrial depolarization and spread of electrical impulse through the atria
  – Upslope of P represents depolarization of right atrium
  – Downslope of P represents depolarization of left atrium

P Waves: Lead II

• Tall P waves could indicate RA hypertrophy

• A fat P wave could indicate LA hypertrophy
P Waves: V1

- A predominate 1\textsuperscript{st} half of biphasic P wave could indicate RA hypertrophy

- A predominate 2\textsuperscript{nd} half of a biphasic P wave could indicate LA hypertrophy

RA Hypertrophy
ECG in Ventricular Hypertrophy

- ECG criteria used to identify ventricular hypertrophy are not very reliable
  - Good specificity
    - When ECG changes of ventricular hypertrophy are seen there is usually hypertrophy present
  - Very poor sensitivity
    - ECG changes are not always seen even when there is hypertrophy present
- Patients who meet one set of criteria may not meet another
- QRS voltage is influenced by age, gender, race and body build
Left Ventricular Hypertrophy

Large S waves in $V_1$, $V_2$

Large R waves in $V_5$, $V_6$

Causes of LVH

- Chronic hypertension (most common)
- Aortic stenosis or insufficiency
- Hypertrophic cardiomyopathy
- Coarctation of aorta
LVH

• Common reason for false positive ST elevation

• Anatomic LVH may be present in absence of ECG criteria
  – To ascribe ST elevation to LVH the ECG must meet the voltage criteria

Left Ventricular Hypertrophy

• **V1 and V2 (V3)**
  – Deeper than normal S waves
  – Small r waves

• **V5 and V6 (V4)**
  – Taller than normal R waves
  – Small S waves
LVH Voltage Criteria

• One or more voltage criteria
• Only applicable if QRS is < 120 ms
• **Precordial lead voltage criteria**
  – R-wave in V5 or V6 > 26 mm
  – R-wave in V5 or V6 + S-wave in V1 > 35 mm
  – Largest R-wave + largest S-wave in precordial leads > 45 mm

ST – T Wave Changes Secondary to LVH

• ST elevation is generally discordant
  – ST elevation in V2-V3 (V1)
  – ST elevation in lead III
  – ST depression in V4-V6
    • Previously called strain pattern
    • Down sloping – not horizontal
• **Not due to LVH**
  – ST elevation in lateral leads
  – ST depression in V2-V3
Estes Scoring System for LVH

<table>
<thead>
<tr>
<th>Condition</th>
<th>Points</th>
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</thead>
<tbody>
<tr>
<td>R or S in limb lead: 20 mm or more</td>
<td>3</td>
</tr>
<tr>
<td>S in V1, V2, or V3: 25 mm or more</td>
<td></td>
</tr>
<tr>
<td>R in V4, V5, or V6: 25 mm or more</td>
<td></td>
</tr>
</tbody>
</table>

Sokolow and Lyon Criteria for LVH

- S wave in V1 + R wave in V5 or V6 (whichever is larger)
  - If greater than 35 mm = LVH

Total = 52 mm
LVH

<table>
<thead>
<tr>
<th>Condition</th>
<th>Score</th>
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<tr>
<td>R or S in limb lead: 20 mm or more</td>
<td></td>
</tr>
<tr>
<td>S in V1, V2, or V3: 25 mm or more</td>
<td>3</td>
</tr>
<tr>
<td>R in V4, V5, or V6: 25 mm or more</td>
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</tr>
<tr>
<td>Any ST shift (without digitalis)</td>
<td>1</td>
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<tr>
<td>Typical “strain” ST-T (with digitalis)</td>
<td></td>
</tr>
<tr>
<td>LAD: -15 degrees or more</td>
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<tr>
<td>QRS width = 0.09 sec or more</td>
<td></td>
</tr>
<tr>
<td>Intrinsicoid deflection V5 or V6 = 0.04 or more</td>
<td></td>
</tr>
<tr>
<td>P-terminal force in V1 &gt; .04</td>
<td></td>
</tr>
<tr>
<td>Total Points</td>
<td>9</td>
</tr>
</tbody>
</table>

5 = LVH; 4 = probable LVH
ECG #4

R-wave in V5 or V6 > 26 mm
R-wave in V5 or V6 + S-wave in V1 > 35 mm
Largest R-wave + largest S-wave in precordial leads > 45 mm

ECG #5

R-wave in V5 or V6 > 26 mm
R-wave in V5 or V6 + S-wave in V1 > 35 mm
Largest R-wave + largest S-wave in precordial leads > 45 mm
Right Ventricular Hypertrophy

- Tall R waves V1, V2
- Deep S waves V5, V6

Causes of RVH

- Mitral valve disease
- COPD
- Pulmonary hypertension
- Pulmonic stenosis
- Tricuspid insufficiency
RVH

• Large R waves in RV leads $V_1$ and $V_2$
• Deep S waves in the LV leads ($V_5$, $V_6$)
• Axis is often deviated to the right (> +90°)
• ST-T strain pattern in RV leads ($V_1$-$V_2$) and in leads II, III, and AVF
• Intrinsicoid deflection is often delayed to 0.05 sec in $V_1$-$V_2$

Right Ventricular Hypertrophy

• Right Axis deviation is one of earliest signs
• Reverse R wave progression
• Dominant R wave in V1 and V2 (QRS width is normal)
• Deep S wave in V5 and V6
ECG Clues to RVH

- RAD > + 90 degrees
- R in V₁ ≥ 7 mm
- R in V₁ + S in V₅ or V₆ ≥ 10 mm
- R/S ratio in V₁ = 1.0 or more (R ≥ S)
- S/R ratio in V₆ = 1.0 or more (S ≥ R)
- V₁ ID = .04 or more
- Incomplete RBBB
- Right atrial enlargement

RVH

Right axis deviation? + 150°
R in V₁ ≥ 7 mm? Yes: 17mm
R in V₁ + S in V₅ or V₆ ≥ 10 mm? Yes: 22mm
R/S ratio in V₁ ≥ 1 (R = or > S)? Yes
S/R ratio in V₆ ≥ 1 (S = or > R)? No
ID in V₁ ≥ .04 sec? Yes: .04
Incomplete RBBB pattern? No
ST-T strain in II, III, aVF? Yes
Right atrial enlargement? Yes
Electrolyte Effects

- Potassium and calcium are the two electrolytes with the most influence on the ECG
- Changes are non-specific
  - ECG cannot be considered diagnostic of an electrolyte abnormality
  - Electrolyte abnormalities can occur in the absence of ECG changes
- Magnesium abnormalities aren’t revealed by changes on the ECG
  - Can result in cardiac arrhythmias
  - Magnesium is treatment of choice in Torsades de Pointes
**Action Potential of Cardiac Cells**

- **Phase 0**: Rapid depolarization – Sodium Influx
  (beginning of QRS complex)
- **Phase 1**: Brief, rapid initiation of repolarization
- **Phase 2**: Slowing of the repolarization – Calcium Influx – correlates with ST segment

Hypocalcemia results in the prolonged opening of the calcium channels during Phase II of the Cardiac Action Potential: Thus extending the ST segment.

**The Electronics**

- **Phase 3**: Sudden acceleration in the rate of repolarization - Potassium Efflux – Correlates with T wave
- **Phase 4**: Resting membrane potential

Potassium abnormalities directly affect ventricular repolarization during Phase III and thus affect the T wave and QT interval.
Potassium

- 95% or > of potassium is intra cellular
- Majority of potassium contained in muscle
  - Declines with age due to decrease in muscle mass
- Dietary intake is the major source / kidneys responsible for excretion
- Ratio of extracellular to intracellular important for electrical membrane potentials
- Major body systems impacted by abnormalities:
  - GI
  - Neuromuscular
  - Cardiac

Nerve impulse and muscular function transmission dependent on potassium.

Hyperkalemia:
K+ greater than 5.0 mEq / L

✓ Rarely occurs in healthy people
✓ Impaired potassium management:
  - Renal Disease
  - Diabetics

- Decreased Excretion
  - Renal disease
    - Decreased renal perfusion
    - Sickle cell disease
  - Decreased aldosterone
    - Addison’s
    - Diabetes
    - Drugs inhibiting aldosterone (aldactone, ACE-I, ARBs, Non steroidal antinflammatories, Heparin)

- Increased Intake
  - Salt substitutes
  - Supplements
  - High dose penicillin with K+
  - Lactated ringers
  - Transfusion of banked blood
Hyperkalemia: Causes

- Cellular disruption with leak of intracellular K+
  - Crush injuries
  - Rhabdomyolysis
  - Hemolysis (blood transfusion reaction)
  - Early burns
  - Trauma
  - Large hematoma
  - Severe catabolic state
  - Lysis of tumor cells (chemotherapy)

- Intracellular to extracellular shift
  - Metabolic acidosis
  - Hypertonic glucose with insulin deficiency
  - Hyperosmolality
  - Digitalis toxicity
  - Depolarizing neuromuscular blocking agents
  - Beta blockers

Hyperkalemia: Signs and Symptoms

Symptoms when K+ > 6.0 mEq/L
- Skeletal muscle effects when K+ > 7.0 mEq/L
- Neuromuscular effects complicated by acidosis, low sodium, low calcium, high magnesium
  - *Parathesia*
- Lower extremity weakness
- Hypotension

EKG Changes
- Tall narrow peaked T waves
- Wide QRS
  - Prolonged PR and flattened to absent P wave
  - Dysrhythmias
    - Bradycardia / heart block
    - Sine wave pattern
    - Asystole
Hyperkalemia

[ECG diagram]

Hyperkalemia

[ECG diagram]
Hyperkalemia

Note: This is not a normal sinus rhythm.

Hyperkalemia

Potassium 8.8
BUN 240
Creatinine 24.4

Note: Prolonged PR interval and flattening of the P wave.
Hyperkalemia: Treatment

- Level > 6.0 mEq/L should be treated. Urgency based on clinical manifestations.

- Limit K+ intake
- Volume expansion
- K+ > 6.5 or dysrhythmias
  - Stabilize cardiac membrane with calcium chloride
    - Not if digitalis toxic
  - Shift potassium into cell
    - 50%Dextrose and insulin (50 ml and 10 units)
    - High dose inhaled beta agonists (synergistic)
    - Sodium bicarbonate to correct acidosis

Hyperkalemia: Treatment

- Kayexalate is an exchange resin
  - Exchange sodium for K+ and moves K+ out via the GI tract
  - Can be given orally or as retention enema
- Oral dose is administered in sorbital
  - Sorbitol orally acts as osmotic laxative
- Retention enema is administered in dextrose
  - Sorbitol can cause intestinal necrosis when given by enema

- Loop diuretics if functioning kidneys
- Dialysis if renal dysfunction
Hypokalemia: Causes

- K+ less than 3.5 mEq / L (total body deficit of 5-10%)

- Causes:
  - Poor K+ intake
  - Increased GI loss (not usually cause of symptomatic imbalance)
  - Increased renal loss
    - Renal tubular acidosis
    - Diuretics
    - Excess mineral or glucocorticoids (aldosterone)
    - Low magnesium
    - Certain antibiotics

- Extracellular to intracellular shifts
  - Alkalosis (potassium exchanged for hydrogen ions)
  - Insulin
  - Treatment of DKA or HHNK
    - Insulin
    - Beta adrenergic agonists
  - Note: Does not reflect total body potassium – Correct with caution
  - Caution with hypokalemia in presence of acidosis.

Urinary K+: High with renal loss; low with other causes

Hypokalemia: Signs and Symptoms

- Symptoms occur when K+ < 3.0 mEq/L
- Severity dependent on:
  - Rapidness of onset
  - Systemic pH
  - Calcium level
- S&S related to altered membrane potentials and impaired muscle contractility
  - Increase in resting membrane potential of neuronal and muscular cells
  - Reduces excitability
- GI
- Orthostatic hypotension
- Parasthesias, weakness, fatigue and muscle cramps
  - Lower extremities are typically impacted first
- Respiratory muscle weakness, dyspnea, paralysis and arrest (< 2.5 mEq/L)
- Enhanced digitalis effect
- Severe hypokalemia can result in rhabdomyolysis
Hypokalemia:
ECG Changes

• Mild hypokalemia: delays ventricular repolarization
  – ST depression, flattening of T wave, inverted T wave
  – Heightened U waves, prolonged QT interval
• Increases risk for Torsades de Pointes
• Lowered threshold for ventricular fibrillation and reentrant tachycardias
• Severe hypokalemia
  – Increased PR interval
  – Increased QRS interval

Clinical Pearls

Digoxin

• Hypokalemia increases risk of digoxin toxicity.

Class III Antiarrhythmics

• Hypokalemia increases the risk of Torsades de Pointes with potassium channel blocking medications
Hypokalemia: Severe

Etiology of Cardiac Arrest

Hypokalemia: Treatment

- Treat cause
- Correct alkalosis
- **Correct hypomagnesemia**
- Increased potassium intake (dietary or supplement) if potassium ≥ 3.0 mEq/L
  - Foods high in potassium: orange juice, bananas, raisins, milk, green vegetables
  - Oral supplements up to 40 mEq can be used safely several times per day.
- Add potassium to maintenance IV fluid

- IV potassium bolus for severe deficiency (less than 3.0 mEq /L if on digoxin, symptoms related to hypokalemia, or less than 2.5 mEq / L without symptoms)
  - Non glucose solution
  - Safe dosage: 10 mEq / 100 cc over 1 hour
  - May give 20 mEq over 1 hour if K+ is < 3.5 mEq / L (higher doses if life threatening)
  - Concentration should not exceed 10 mEq per 100 ml via peripheral line or 20 mEq per 100 ml if central line

Note: Replace cautiously in those with impaired ability to excrete.
Calcium

• Less than 50% of dietary intake is absorbed.
• The majority of the body’s calcium is in the bone.
• Serum level regulated by parathyroid levels and vitamin D.
  – Also influenced by serum phosphate levels (inverse relationship), albumin levels, and blood pH.
  – Calcium in bone can be exchanged to maintain extracellular levels.

There are 3 types of serum calcium:
  – > 40% of calcium is protein bound (mostly albumin)
  – 10% is chelated (non-ionized) with substances such as citrate or phosphate
  – 50% is ionized (free to leave the extracellular fluid and participate in intracellular function)

Important for several key processes:
✓ Muscle contraction
✓ Transmission of nervous system impulses
✓ Hormone secretion
✓ Blood clotting and wound healing
✓ Cellular function

Hypocalcemia

• Calcium < 8.8 mg / dL or ionized calcium < 4.65 mg / dL.

Common disorder in critical care.

Generally asymptomatic if development is slow or if ionized calcium remains normal.
Hypocalcemia: Causes

<table>
<thead>
<tr>
<th>Decreased calcium intake or absorption</th>
<th>Increased calcium excretion</th>
<th>Impaired ability to mobilize calcium from bone stores</th>
<th>Increased calcium binding; Increased calcium chelation (decreased ionized calcium)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low dietary intake</td>
<td>Diuretic therapy</td>
<td>Inadequate levels of parathyroid hormone</td>
<td>Alkalosis Acute Pancreatitis Drugs</td>
</tr>
<tr>
<td>Hypomagnesemia</td>
<td>Chronic Diarrhea</td>
<td>(Decreased magnesium inhibits parathyroid release)</td>
<td>Cimetidine</td>
</tr>
<tr>
<td>Renal failure</td>
<td>Hyperphosphatemia</td>
<td></td>
<td>Heparin</td>
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<tr>
<td>Vitamin D deficiency</td>
<td></td>
<td></td>
<td>Theophylline</td>
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<td>Liver disease</td>
<td></td>
<td></td>
<td>Aminoglycosides</td>
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<tr>
<td>Steroid therapy</td>
<td></td>
<td></td>
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<tr>
<td>Cushing's disease</td>
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</tbody>
</table>

Hypocalcemia: Signs and Symptoms

Most common symptoms due to neuromuscular irritability.

- **Parathesias (common)**
- Hyperreflexia
- Tetany (spasms of face, hands, and feet)
- Chvostek's sign
  - Tapping of face over facial nerve located below the temple
  - Positive sign results in spasm of lip, nose or face.
- Trousseau's sign
  - Inflate blood pressure above systolic BP and hold for 3 minutes
  - Positive sign results in contraction of fingers or hand.
- Stridor / wheezing / bronchospasm
- For severe deficit: laryngeal spasm, change in mental status, seizures
- Chronic: dry skin and hair and brittle nails; bone pain and risk of fracture

- **Cardiovascular effects:**
  - Decreased contractility
  - Hypotension
  - Prolonged QT
    - ST segment hugging baseline for extended period
    - QT prolongation is not due to delay in ventricular repolarization
  - Torsades de pointes
  - Bradycardia / heart block
  - **Digitalis insensitivity**
  - Heart failure
  - Cardiac arrest

Hypocalcemia results in the prolonged opening of the calcium channels during Phase II of the Cardiac Action Potential: Thus extending the ST segment.
Hypocalcemia

Note the hugging of the ST segment to baseline.

Hypocalcemia

Note the hugging of the ST segment to baseline.
Hypocalcemia: Treatment

• Goal: Low-normal range
• High calcium, low phosphorous diet
• Vitamin D supplements if deficiency
• Phosphate binding antacids *
• **Magnesium for hypomagnesemia**
• Correct alkalosis (increases ionized Ca++)
• Thiazide diuretics (increase tubular calcium reabsorption)
• IV calcium chloride or calcium gluconate

IV Calcium Administration

• Calcium gluconate
  • Give 10 ml
  • 10 ml contains 4.5 mEq of calcium

• Calcium chloride
  • Give 3-4 ml
  • 10 ml contains 13.6 mEq of calcium

*Administer no faster than 1 ml per minute
May cause sloughing or necrosis (central vein preferred)
Hypercalcemia

• Calcium > 10.4 mg / dL or ionized calcium > 5.26 mg / dL

• Causes:
  • Increased calcium intake (supplement or antacids)
  • Increased calcium absorption (hypophosphatemia, excessive vitamin D)
  • Increased mobilization of calcium from the bone (Vitamin D excess, immobility, hyperparathyroidism, thyroidtoxicosis, neoplasms)
  • Acidosis (increased ionized calcium)
  • Decreased calcium excretion (thiazide diuretics)

Hypercalcemia: Signs and Symptoms

- Hypophosphatemia
- Signs and symptoms related to dehydration
- Gastrointestinal symptoms (slowing of GI tract)
- Bone and flank pain / osteoporosis / pathological fractures
- Muscular symptoms: Hypotonicity / weakness / fatigue
- Neurological symptoms: Decreased mentation, agitation, comma, seizures.
- Calcium salts form at high levels
  - Pruritis from skin deposits.
  - Renal calculi and potential kidney injury
  - Deposits on the aorta, cardiac valves, and coronary arteries.
Hypercalcemia: Signs and Symptoms

- Cardiac symptoms:
  - hypertension (may be offset by co-existing dehydration)
  - cardiac ischemia
  - arrhythmias (conduction abnormalities)
  - digitalis toxicity.

- **ECG signs**
  - shortened QT segments (secondary to shortened ST segments)
  - Short ST segments can cause ST to merge with T wave (similar to what occurs with hyperacute T wave in a STEMI)

- Life threatening signs and symptoms are rare unless calcium levels reach > 14 mg/dL.

Hypercalcemia

Note lack of horizontal component of ST segment and abrupt take off of T wave after QRS.
Hypercalcemia

![ECG tracing](image)

Note lack of horizontal component of ST segment and abrupt take off of T wave after QRS.

Hypercalcemia: Treatment

- **Primary Treatment: Rehydration with 0.9 NS**
- Decrease calcium absorption
  - Low calcium, high phosphorous diet
  - Glucocorticoids
- Increase calcium excretion
  - Fluids (0.9NS)
  - Loop diuretics
  - Dialysis if renal failure or life threatening
  - Inhibit bone resorption (calcitonin, mithramycin, biphosphonates)
- Prevent cardiac effects
  - Calcium Channel Blockers
- Prevent renal calculi
  - Acidify urine
Clinical Pearl

Calcium imbalances can alter appearance of ECG but are less likely to cause cardiac arrhythmias than potassium imbalances.
Digoxin

- Inhibits the Na+ and K+ membrane pump
  - Increase in intracellular Na+
  - Enhances the Na+ and Ca++ exchange
  - Leads to ▲ in intracellular Ca++
  - ▲ inotropic activity

Digoxin also increases vagal activity and decreases conduction velocity through the AV node
(sympathetic stimulation easily overrides the inhibitory effects of digoxin on AV node conduction)

Digoxin decreases sympathetic outflow and decreases renin production (Beneficial in heart failure)

Digoxin

- Indications
  - HF (no mortality benefit)
  - Atrial arrhythmias (older indication)

- Contraindication / cautions
  - Myocardial infarction
  - Ventricular arrhythmias, HB, Sick Sinus Syndrome
  - Hypertrophic cardiomyopathy
  - Electrolyte abnormalities
Digoxin

• Narrow therapeutic range
• Toxicity may occur at therapeutic levels
• Lower doses routinely used
  – 0.125 mg daily
• Amiodorone increases serum digoxin concentration (digoxin doses must be reduced if starting amiodarone)
• Multiple other medication interactions
• Dialysis is not effective with digoxin toxicity because of high tissue binding

• EKG Changes with Toxicity
  – Increased automaticity with impaired conduction is common

• Other Signs and Symptoms of Toxicity
  – N & V, HA, Confusion
  – Visual disturbances: halos, change in color perception

Digoxin Toxicity Arrhythmias

• Sinus bradycardia, sinus exit block, sinus pause or arrest
• AV block of any degree
  – 3rd degree or complete block can occur
  • Due to physiological effect of block through AV node
  – Can occur in patients with atrial fibrillation
• Atrial tachyarrhythmias
  – Due to physiological effect of accelerated conduction through atrial tissue
• Junctional tachycardia
  – Otherwise not common in adult population
• Ventricular arrhythmias
  – Fascicular VT (from one of the fascicles of left bundle)
  – Bidirectional VT
  – V Flutter
  – V Fibrillation
Digitalis Effect

- Sagging depression of ST segment in leads with positive QRS

- Reduced T wave amplitude
- Possible T wave inversion
- Increased U wave amplitude

- Difficult to evaluate if hypertrophy or BBB

PR interval may also be prolonged.

Atrial Fibrillation with Complete Heart Block
A Final Thought:

We must not, in trying to think about how we can make a big difference, ignore the small daily differences we can make which, overtime, add up to big differences that we often cannot foresee.

- Marian Wright Edelman
Cardiac Action Potential

- Time-dependent change in electrical voltage across a cell membrane
  - Due to movement of ions across cell membrane during depolarization and repolarization
- The “ECG” of a single cardiac cell
- Records depolarization and repolarization of a single cardiac cell
- The ECG is the sum of all the cells’ action potentials recorded from body surface electrodes
**Anatomy of an Action Potential**

Phase 0: depolarization of cell membrane as Na⁺ enters cell; corresponds to QRS. Ca^{++} channels open at about -50mV and Ca^{++} enters cell.

Phase 1: early rapid repolarization as Na⁺ channels close.

Phase 2: plateau maintained mostly by Ca^{++} ions; corresponds to ST segment.

Phase 3: repolarization of cell membrane as K⁺ channels open and K⁺ leaves cell; corresponds to T wave.

Phase 4: resting state maintained partly by Na⁺-K⁺ pump.
Depolarization Abnormalities

- **Depolarization abnormalities** affect the QRS width and/or direction, and reflect interventricular or intraventricular conduction abnormalities
  - Wide QRS seen in bundle branch block or ventricular paced beats
  - Wide QRS of PVCs
  - Delta waves in WPW
  - Abnormal QRS axis in hemiblocks
  - Abnormal Q waves or late intrinsicoid deflection in ventricular hypertrophy

Repolarization Abnormalities

- Abnormalities in the ST segment, T wave, and QT interval duration reflect abnormalities in ventricular repolarization

![ECG waveforms](image)
• **Primary repolarization abnormalities** result from changes in the shape and/or duration of the repolarization phases (phase 2 and phase 3) of the cardiac action potential
  – Disease processes: ischemia, myocarditis
  – Drugs or toxins (digitalis, quinidine, QT interval prolonging drugs)
  – Electrolyte abnormalities – especially Ca\(^{++}\) & K\(^{+}\)
  – Abrupt change in heart rate, hyperventilation, changes in body position, catecholamines, sympathetic stimulation, temperature changes

• When the direction of the QRS is normal, an abnormal direction of the T-wave is generally an indication of a primary repolarization abnormality

• **Secondary repolarization abnormalities** occur as a result of changes in the sequence and/or duration of ventricular depolarization.

  “Whenever the ventricle depolarizes abnormally it also repolarizes abnormally”
Secondary Repolarization Abnormalities

- **RBBB** – ST and T directed opposite to terminal QRS direction
- **LBBB** – ST and T directed opposite to main QRS direction
- **Ventricular pacing** – same as LBBB
- **PVCs** – ST and T directed opposite to main QRS direction
- **WPW** – ST and T directed opposite delta wave

Common Causes of ST Depression

- **Ischemia**
  - Horizontal or downsloping

- **Strain pattern of hypertrophy**
  - Depressed but convex (bows upwards); asymmetrical T wave inversion

- **Dig effect**
2 Types of T Wave Inversion

Terminal T wave inversion

- “Wellens Warning” when seen in V₂ and V₃ of undiagnosed patient
- Indicates tight proximal LAD stenosis at risk for occlusion
- Is an **expected sign of successful reperfusion** in anterior STEMI

Symmetrical T wave inversion

- Sign of ischemia if troponin negative
- Indicates NSTEMI if troponin positive
- Is **expected sign in evolution** of STEMI and following reperfusion

Wellens’ Warning
Proximal LAD Stenosis
T Wave Inversion

• T wave in leads I, II, aVL, and $V_2$ to $V_6$ should be called:
  – Inverted when the T-wave is 1mm to 5mm negative
  – Deep negative when 5mm to 10mm negative
  – Giant negative when more than 10 mm negative

• Giant T wave inversion can be seen in hypertrophic cardiomyopathies, NSTEMI, and neurological events (especially intracranial hemorrhage).

Subarachnoid Hemorrhage
ECG Signs of Ischemia & Injury

Q waves = necrosis
ST elevation = injury
ST depression = ischemia or non-Q wave MI
T wave inversion = ischemia or non-Q wave MI
Tall wide T waves = early ischemia or injury
Patterns of Ischemia

<table>
<thead>
<tr>
<th>Pattern Description</th>
<th>Leads</th>
<th>Image</th>
</tr>
</thead>
<tbody>
<tr>
<td>ST depression and T wave inversion concordant with terminal portion of QRS</td>
<td><img src="image1.jpg" alt="Image" /></td>
<td></td>
</tr>
<tr>
<td>Horizontal or down-sloping ST-segment depression with upright or biphasic</td>
<td><img src="image2.jpg" alt="Image" /></td>
<td></td>
</tr>
<tr>
<td>negative-positive T wave</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Terminal T wave inversion (biphasic positive-negative T wave) in V₁-V₃ (also</td>
<td><img src="image3.jpg" alt="Image" /></td>
<td></td>
</tr>
<tr>
<td>called “Wellen’s warning”)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Deep symmetrical inverted T waves with isolectric, slightly up-sloping, or</td>
<td><img src="image4.jpg" alt="Image" /></td>
<td></td>
</tr>
<tr>
<td>horizontally depressed ST segment</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tall, wide T waves</td>
<td><img src="image5.jpg" alt="Image" /></td>
<td></td>
</tr>
<tr>
<td>U wave inversion</td>
<td><img src="image6.jpg" alt="Image" /></td>
<td></td>
</tr>
</tbody>
</table>

How many leads show ischemia?

In patients with angina at rest, ST-segment depression in 8 or more leads combined with ST elevation in aVR and/or V1 is associated with a 75% predictive accuracy of significant 3-vessel or left main stenosis.
### Patterns of Injury

#### ST elevation 1 mm or more in two contiguous leads

<table>
<thead>
<tr>
<th>Description</th>
<th>Example Images</th>
</tr>
</thead>
<tbody>
<tr>
<td>ST pulled up to peak of T wave with no J point</td>
<td><img src="image1" alt="Example Images" /></td>
</tr>
<tr>
<td>Tall, peaked T waves</td>
<td><img src="image2" alt="Example Images" /></td>
</tr>
<tr>
<td>Symmetrical T wave inversion</td>
<td><img src="image3" alt="Example Images" /></td>
</tr>
</tbody>
</table>

#### Threshold for abnormal J point elevation

<table>
<thead>
<tr>
<th>Group</th>
<th>Thresholds</th>
</tr>
</thead>
<tbody>
<tr>
<td>Men 40 years or older</td>
<td>2 mm in V2 and V3; 1 mm in all other leads</td>
</tr>
<tr>
<td>Men &lt; 40 years old</td>
<td>2.5 mm in V2 and V3</td>
</tr>
<tr>
<td>Women</td>
<td>1.5 mm in V2, V3; 1 mm in all other leads</td>
</tr>
<tr>
<td>Men &lt; 30 years old</td>
<td>1 mm in V3R and V4R</td>
</tr>
<tr>
<td>All other men &amp; women</td>
<td>0.5 mm in V3R and V4R (right ventricular MI)</td>
</tr>
<tr>
<td>Men &amp; women</td>
<td>0.5 mm in V7-V9 (posterior MI)</td>
</tr>
</tbody>
</table>

#### Threshold for abnormal J point depression

<table>
<thead>
<tr>
<th>Group</th>
<th>Thresholds</th>
</tr>
</thead>
<tbody>
<tr>
<td>Men &amp; women</td>
<td>0.5 mm in V2, V3; 1 mm in all other leads</td>
</tr>
</tbody>
</table>

Right Ventricular Infarction

• Occurs in about 40-45% of inferior MIs
• RV infarction results in decreased LV filling and signs of LV forwards failure
  – Signs of high RV pressure (JVD) and low LV pressure
    (normal lung sounds, hypotension, decreased peripheral perfusion)
• Treatment of RV infarction
  – Fluids
  – Avoid preload reduction (NTG)
  – Inotropes if necessary
  – Dual chamber pacing rather than VVI if possible

Right Ventricular Infarction

♥ None of the standard 12 leads look directly at the RV
♥ Lead V₁ is closest to RV
♥ 12 Lead Clues:
  ▪ ST elevation in II, III, AVF, V₁
  ▪ ST elevation in III > AVF
  ▪ ST discordance between V₁ and V₂

Don’t count on changes in the V leads

♥ Record RV leads with all inferior MIs
  ▪ Might as well record the posterior leads too!
Recommendation: record right side leads in all patients with ST elevation in II, III, aVF to detect RV infarction

Any signs of injury or infarction?

Should we do right side leads?
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
Any signs of injury?

What additional leads do we need?
18 Lead ECG

♥ Adds 3 posterior leads and 3 right side leads to standard 12 lead ECG

♥ Right side leads should be recorded as soon as possible in all patients with ST elevation in the inferior leads (II, III, aVF)

♥ Record 18 leads with:
  – All inferior wall STEMIs (ST elevation in II, III, AVF)
  – ST depression in $V_1$-$V_3$ (with or without ST elevation in other leads)

18 lead ECG is appropriate in all patients with chest pain unless they are a straightforward anterior wall MI

Right Ventricular Leads
V4 → V4R position
V5 → V5R position
V6 → V6R position

Posterior Leads
V1 → V7 position
V2 → V8 position
V3 → V9 position
Labeling the 18 Lead ECG

<table>
<thead>
<tr>
<th>Original V Leads</th>
<th>New V Leads</th>
</tr>
</thead>
<tbody>
<tr>
<td>V1</td>
<td>V7</td>
</tr>
<tr>
<td>V2</td>
<td>V8</td>
</tr>
<tr>
<td>V3</td>
<td>V9</td>
</tr>
<tr>
<td>V4</td>
<td>V4R</td>
</tr>
<tr>
<td>V5</td>
<td>V5R</td>
</tr>
<tr>
<td>V6</td>
<td>V6R</td>
</tr>
</tbody>
</table>

Can you diagnose an MI in the presence of bundle branch block?

- Right bundle branch block does not interfere with MI diagnosis
  - ST elevation in RBBB is usually due to injury
  - Does not interfere with initial septal depolarization
    - Does not cause abnormal Q waves

T waves should be opposite terminal QRS direction
Normal RBBB

RBBB with Acute ALMI
MI with LBBB

- LBBB is a bigger challenge
  - The normal secondary ST & T wave changes cause ST elevation in leads with a negative QRS
  - LBBB causes the septum to depolarize abnormally from right to left instead of left to right → loss of normal R waves in V1, V2

- Often results in QS complexes in V1-V3 which can mimic MI

Normal LBBB

- **Secondary ST-T wave changes:** ST and T wave should be directed opposite the QRS (when QRS is negative, ST segment and T wave are upright; when QRS upright, ST and T are negative: discordant)
- **LBBB** typically shows ST elevation in leads with a negative QRS in the absence of injury so assessment of STEMI is challenging
Sgarbossa Criteria

Concordant ST elevation > 1 mm in leads with a positive QRS complex

Concordant ST depression > 1 mm in V1-V3

ST segment elevation of ≥ 5 mm that is discordant with the QRS complex


<table>
<thead>
<tr>
<th>ECG Finding</th>
<th>Assigned Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>ST-segment elevation ≥1mm in lead with concordant QRS complex</td>
<td>5 points</td>
</tr>
<tr>
<td>ST-segment depression ≥1 mm in leads V1, V2, or V3</td>
<td>3 points</td>
</tr>
<tr>
<td>ST-segment elevation ≥5 mm in lead with discordant QRS complex</td>
<td>2 points</td>
</tr>
<tr>
<td>A score of ≥ 3 has 98% specificity for MI in the presence of LBBB</td>
<td></td>
</tr>
</tbody>
</table>

Be suspicious for AMI when:
- ST elevation ≥ 1 mm in leads with positive QRS (I, aVL, V5, V6)
- ST depression ≥ 1 mm in leads with negative QRS (V1 – V4)
- ST elevation ≥ 5 mm in leads with a negative QRS
- Concordant T waves (same direction as terminal QRS)

Admission ECG showing LBBB: Concordant T waves II, V4-V6

Next day with chest pain
Paced Rhythm

- Same secondary ST-T wave changes as with LBBB: ST and T wave should be directed opposite the QRS
- This can look like AMI with ST elevation
- Be suspicious for AMI when:
  - ST elevation > 1 mm in leads where QRS is predominantly positive (I, aVL, V5, V6)
  - ST depression > 1 mm in one or more leads in leads where QRS is predominantly negative (V1 – V4)
  - ST elevation > 5 mm and disproportionate with the QRS voltage

Fusion beats show ST elevation
ST elevation > 5mm
ST depression instead of ST elevation

Same patient in NSR one minute later
MI Mimics

➤ Other causes of ST elevation
  - Pericarditis
  - Early repolarization
  - Acute cor pulmonale
  - Hyperkalemia
  - Intracranial hemorrhage
  - Prinzmetal’s angina
  - Acute pancreatitis
  - Acute cholecystitis
  - Myocardial metastases

➤ Other causes of Q waves
  - Hypertrophic cardiomyopathy
  - WPW
  - COPD
  - Pulmonary embolism
  - LVH
  - Left anterior hemiblock
  - Myocardial infiltrative disease
  - Misplaced leads
  - Cardiac displacement (eg pneumothorax)

Pericarditis

• Inflammation of pericardium – with or without effusion

❤ Normal pericardium has 2 layers
  - Parietal pericardium – fibrous layer attached to sternum, great vessels and diaphragm
  - Visceral pericardium (epicardium) covers outside of heart
  - Normally 15-30 cc fluid between the layer
  - Functions to lubricate heart during movement and to protect from inflammation, stretch, etc
• Clinical presentation can mimic MI
  – Chest pain – sharp, pleuritic, can mimic MI pain
    • 95% of cases present with chest pain
    • Worse with inspiration or coughing, relieved by leaning forward
    • May radiate to shoulders
  – Pericardial friction rub
    • Scratchy, squeaky, grating sound
    • May have 3 components: atrial systole, ventricular systole, ventricular diastole
    • Heard best with diaphragm over left sternal border
    • Transient – comes and goes, only heard about 50% of cases
  – CK MB and Troponin I can be elevated

ECG Changes in Pericarditis
• Wide-spread concave ST elevation seen in multiple leads – “smiles” at you.
  – Rarely higher than 5mm
  – ST segment may rises obliquely in a straight line
  – Occurs in multiple leads – not in anatomical groups like MI ST elevation
• No reciprocal ST depression as would be seen in AMI.
  – ST depression often seen in AVR and may be seen in V₁.
• PR segment depression in limb leads and V₅-V₆; PR segment may be elevated in aVR.
• Diffuse T wave inversion follows – after ST segments return to baseline.
  – T waves may normalize or remain inverted in chronic pericarditis.
• No Q waves develop (unless associated with AMI).
Stages of Pericarditis

- **Stage 1**
  - ST elevation (“smiles”)
  - Lasts up to 2 weeks
- **Stage 2**
  - ST returns to baseline
  - T wave amplitude decreases
  - Lasts from days to weeks
- **Stage 3**
  - T wave inversion
  - Begins in 2\textsuperscript{nd} to 3\textsuperscript{rd} week
- **Stage 4**
  - Gradual resolution
  - T waves may stay inverted for up to 3 months or longer if chronic pericarditis

Pericarditis

- Oblique ST rise in lead I
- ST elevation in multiple leads:
  - II, III, aVF
  - All V leads
- T waves remain upright until ST stage complete
- No Q waves
Early Repolarization

- ER has been considered a marker of good health because it is more prevalent in athletes, younger persons, and at slower heart rates.
  - ER estimated to be present in 5-13% of people.
  - More common in:
    - Young
    - Males
    - Athletes
    - African Americans
  - ST elevation in $V_2$ (sometimes $V_1$-$V_3$) up to 2 mm in men and 1.5 mm in women can be a normal variant.

- More recent reports have suggested an association between ER and an increased risk for idiopathic VF and SCD.
• ECG pattern:
  – A sharp well-defined positive deflection or notch immediately following a positive QRS complex at the onset of the ST segment
    • J point elevation that drags the ST up with it
    • ST usually maintains normal shape
    • ST elevation and “fish hook” usually seen in V3-V6
  – Tall upright T waves concordant with QRS
    • T waves do not eventually invert as they do in MI
  – Absence of reciprocal changes helps differentiate from AMI

• Proposed classification of ER
  – Type 1: associated with ER in the lateral precordial leads. This form is common among healthy male athletes and is thought to be largely benign.
  – Type 2: associated with ER in the inferior or inferolateral leads and is associated with a moderate level of risk.
  – Type 3: associated with ER globally in the inferior, lateral, and right precordial leads, and appears to be associated with the highest relative risk (though the absolute risk of sudden death remains small).
Early Repolarization

ST elevation in II, III, aVF, V3-V6
“Fish hook” in V4, V5

Early Repolarization

J point elevation and concave ST elevation II, III, aVF, V_3 - V_5
Prominent T waves concordant with QRS.
Early Repolarization

ST elevation in I, II, V₂-V₆
Notch at end of QRS in II, III, aVF, V₄-V₆
Prominent T waves concordant with QRS

Pericarditis Vs. Early Repolarization

<table>
<thead>
<tr>
<th></th>
<th>Pericarditis</th>
<th>Early Repolarization</th>
</tr>
</thead>
<tbody>
<tr>
<td>ST segment</td>
<td>J-point elevation with resultant ST segment elevation. Usually concave ST</td>
<td>J-point elevation of ≥1 mm in two adjacent leads which pulls ST segment up. ST</td>
</tr>
<tr>
<td></td>
<td>elevation (smiley). Widespread, seen in multiple leads. No reciprocal ST</td>
<td>maintains normal shape. Usually in precordial leads but can be seen in inferior</td>
</tr>
<tr>
<td></td>
<td>depression. ST elevation resolves.</td>
<td>leads. ST elevation does not resolve.</td>
</tr>
<tr>
<td>QRS</td>
<td>No QRS changes.</td>
<td>Sharp, well-defined notch or slurring of terminal part of QRS (fish-hook).</td>
</tr>
<tr>
<td>T waves</td>
<td>Widespread T wave inversion after resolution of ST segment elevation.</td>
<td>Tall upright T waves in leads with positive QRS. T waves do not evolve.</td>
</tr>
<tr>
<td>Other</td>
<td>PR depression common. Pain can mimic MI.</td>
<td>PR depression not seen. Pain not typically present.</td>
</tr>
</tbody>
</table>
K⁺ = 7.8 mEq/L

Hyperkalemia can mimic MI by causing hyperacute looking T waves and ST elevation

47 year old man.
Any indication of infarction?
Same 47 year old man after dialysis

- Patient had rectal cancer with obstructed ureters.
  - Renal failure resulted in K⁺ 9.2

Cor Pulmonale

- Alteration in the structure and function of the right ventricle caused by a disease of the lungs or pulmonary blood vessels.
- ECG changes of right heart strain
  - S1, Q3, T3 Pattern
    - S wave in Lead 1 (often aVL)
    - Q in Lead III
    - Inverted T in Lead III
  - T wave inversion in both inferior and precordial leads
  - May see elevated ST segments in III, aVR and V₁-V₂
    - Usually not in II or aVF (differentiates from inferior MI)
  - RAD or incomplete or complete RBBB
  - Tall P waves in inferior leads (right atrial strain)
Acute Cor Pulmonale

When the limb leads suggest inferior MI and the precordial leads suggest anterior MI: THINK PULMONARY EMBOLISM!

New right axis deviation in patient with previously normal axis
Tako-tsubo Cardiomyopathy (TC)

• Also called
  – Stress-induced cardiomyopathy
  – Transient-left-ventricular ballooning syndrome
  – Apical ballooning syndrome
  – Broken heart syndrome

• 90% of cases occur in postmenopausal women (mean age 68 years) after a physical or emotional stressor (80% of cases)

• Chest pain and ECG changes that mimic acute MI
  – ST-segment elevation in the precordial leads in 70%-80% of cases mimicking anterior wall MI
  – Diffuse T-wave inversions are common
  – QT prolongation can occur
  – TC occurs in about 2% of patients presenting with symptoms of acute MI
Tako-tsubo Cardomopathy

T wave inversion in inferior and anterior leads
QT prolongation

V leads from 68 year old woman presenting with chest pain showing ST elevation V2-V4. Coronary arteries were normal but ventriculogram showed apical ballooning.
Tako-tsubo Cardiomyopathy

Widespread ST elevation
Q waves V1-V3
RBBB

Printzmetal angina

Printzmetal (Variant) Angina
• Angina due to coronary artery vasospasm
• Presents with ST elevation
• Resolves with treatment with nitroglycerin or other therapy that relieves the spasm
• Treatment with Ca++ channel blockers
ECG of a patient who had severe chest pain and huge ST elevations in the anterior and lateral leads with reciprocal depression in the inferior leads. Cardiac cath showed no significant stenosis.

Recorded during an episode of chest pain.
Any signs suggestive of MI?
Surprise!! A normal a few minutes later after receiving SL nitroglycerin.

Printzmetal’s angina due to coronary artery vasospasm. Patient had discontinued his verapamil due to constipation.
Ability is what you are capable of doing. Motivation determines what you do. Attitude determines how well you do it.
- Lou Holtz
DIFFERENTIATING SUPRAVENTRICULAR & VENTRICULAR TACHYARRHYTHMIAS

A: Normal
B: SVT with aberrant conduction
C: Pre-excited tachycardia
D: VT
Ectopy VS Aberrancy

<table>
<thead>
<tr>
<th>SVT with aberrancy</th>
<th>Ventricular tachycardia</th>
</tr>
</thead>
<tbody>
<tr>
<td>• SVT with RBBB Aberration</td>
<td>• VT with LBBB Pattern</td>
</tr>
<tr>
<td>• SVT with LBBB Aberration</td>
<td>– Right VT</td>
</tr>
<tr>
<td>• VT with LBBB Pattern</td>
<td>• VT with RBBB Pattern</td>
</tr>
<tr>
<td>– Right VT</td>
<td>– Left VT</td>
</tr>
</tbody>
</table>

Acute Management of Wide Complex Tachycardias

- Wide complex tachycardia presumed to be VT if diagnosis is unclear
- DC cardioversion with sedation if hemodynamically unstable
- Don’t assume VT cannot be well tolerated!
- The rate, size of the heart and presence of additional complications are often more important than the source of the tachycardia

✓ Check the patient (need to defib?)
✓ Check the blood pressure (need to cardiovert?)
✓ Check the ECG (determine the rhythm)
Criteria for Differentiating Ectopy from Aberrancy

- Patient history / assessment
- QRS Width
- Concordance
- AV Dissociation
- Axis
- Morphology

Note:
VT is much more common than supraventricular tachycardia with bundle branch aberration. In wide QRS tachycardias VT is the right answer up to 80% of the time. A wide complex tachycardia is always considered ventricular in origin if the diagnosis is uncertain.

Patient History
- Acute ischemia / injury (Abnormal automaticity)
- Post myocardial infarction / ischemic cardiomyopathy (Reentrant circuit within myocardium)
- Non ischemic dilated cardiomyopathy (Bundle branch reentrant VT)

QRS Width
- The wider the QRS – VT is favored – However:
- SVT with LBBB will have a wider QRS than SVT with RBBB
- Other causes of SVT with wider than expected QRS: antidromic tachycardia and patients on Class I antiarrhythmics or amiodarone
- Not all VT is significantly wide
  - VT originating from septum more narrow than VT from free wall
  - If QRS more narrow than sinus rhythm = VT
AV Dissociation

- Independent atrial and ventricular activity (AV dissociation) is diagnostic for ventricular ectopy

  **Only seen in 30% VTs**

- Ventricular tachycardia may also have retrograde P waves (retrograde P waves do not confirm VT)
AV Dissociation: Fusion or Capture Beats

Axis

- Extreme axis is strong indicator of ectopy
- Right axis deviation confirms ectopy with LBBB pattern
- Ventricular tachycardia rarely occurs with normal axis
Morphology (Shape)

Ventricular Ectopy compared to Aberrancy (BBB)

Morphology Challenges:
BBB Reentrant VT
Idiopathic RVOT
Antidromic tachycardia
### Comparison of Morphology in Lead V1

<table>
<thead>
<tr>
<th>RBBB</th>
<th>LBBB</th>
</tr>
</thead>
<tbody>
<tr>
<td>VT from Left Ventricle</td>
<td>VT from Right Ventricle</td>
</tr>
</tbody>
</table>

### Comparison of Morphology in Lead V1

<table>
<thead>
<tr>
<th>RBBB</th>
<th>LBBB</th>
</tr>
</thead>
<tbody>
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<td>VT from Left Ventricle</td>
<td>VT from Right Ventricle</td>
</tr>
</tbody>
</table>

- **Left ventricle first**
- **Right ventricle first**
Left Ventricular Ectopy
Lead V1

• Right Bundle Branch shaped
  – R wave with an early left peak (R'r')
  – R wave with a single peak
  – q wave followed by R wave

Can also be shape of RBBB

Right Ventricular Ectopy
Lead V1

• LBBB shaped
  – Primarily negative wide rS complex
    • delay to the nadir
      – > 0.06 sec
    • r wave broader than 0.03 sec
    • Slurring on the down stroke

Note: LBBB shaped VT can come from RV or septum.
VT from RV includes: Idiopathic VT,
BB Reentrant VT,
Arrhythmogenic right ventricular dysplasia, VT from Brugada Syndrome
Lead V1 (VT Patterns)

VT with RBBB pattern or LVT

VT with LBBB pattern or RVT

Comparison of Morphology in Lead V1

<table>
<thead>
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<th>RBBB</th>
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</table>

[Diagrams and waveforms for RBBB and LBBB patterns in Lead V1, showing typical morphology and intervals.]
Bundle Branch Block Morphology in Lead V6

- RBBB
- LBBB

Ventricular Ectopy Morphology in Lead V6

Left Ventricular VT
- QS complex
- r wave followed by S wave with R:S ratio < 1

Right Ventricular Like VT
- Any Q Wave
- QS wave
Methodology for Differentiation Using ECG / Bedside Monitoring

Nice to Knows: AV dissociation, Negative concordance V1-V6, V6 changed from upright to negative, axis changed to right superior

Evaluate QRS Morphology

Practice ECG 1

AV Dissociation or Negative Concordance Extreme Axis or V6 Negative
Practice ECG 2
AV Dissociation or Negative Concordance
Extreme Axis or V6 Negative

Practice ECG 3
AV Dissociation or Negative Concordance
Extreme Axis or V6 Negative
Practice ECG 4
AV Dissociation or Negative Concordance
Extreme Axis or V6 Negative

Practice ECG 5
AV Dissociation or Negative Concordance
Extreme Axis or V6 Negative
Practice ECG 6
AV Dissociation or Negative Concordance
Extreme Axis or V6 Negative

Practice ECG 7
AV Dissociation or Negative Concordance
Extreme Axis or V6 Negative
Practice ECG 8
AV Dissociation or Negative Concordance
Extreme Axis or V6 Negative

Vital Signs Stable
12 lead ECG Interpretation:
• Atrial Fibrillation
• RBBB with Left Anterior Hemiblock

Practice ECG 9
AV Dissociation or Negative Concordance
Extreme Axis or V6 Negative

Vital Signs Stable
12 lead ECG Interpretation: Non Specific Intraventricular Conduction Delay
Dissociation or Negative Concordance
Extreme Axis or V6 Negative
Linking to the Bedside Monitor Practice ECG 3

Dissociation or Negative Concordance
Extreme Axis or V6 Negative

Linking to the Bedside Monitor Practice ECG 4

Dissociation or Negative Concordance
Extreme Axis or V6 Negative
Dissociation or Negative Concordance
Extreme Axis or V6 Negative
Linking to the Bedside Monitor Practice ECG 7

Dissociation or Negative Concordance
Extreme Axis or V6 Negative

Linking to the Bedside Monitor Practice ECG 8

Dissociation or Negative Concordance
Extreme Axis or V6 Negative
Case Study

[Image of an electrocardiogram waveform with annotations: SVT, RBBB, SVT, LBBB, LVT, RVT]
Case Study

Morphology Challenges
The Not so Common: Congenital and Acquired Long QT, Brugada, WPW, Arrhythmogenic Cardiomyopathy

Be on the Lookout
Arrhythmogenic Cardiomyopathy

ECG Signs

- T Wave inversion in leads V1-V6
- Epsilon wave
- VT with LBBB pattern
- Conduction delays through right bundle
QT Interval Evaluation
Expected QTc Intervals

<table>
<thead>
<tr>
<th></th>
<th>1 to 15 Years</th>
<th>Adult Males</th>
<th>Adult Females</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>&lt; .44 seconds</td>
<td>&lt; .43 seconds</td>
<td>&lt; .45 seconds</td>
</tr>
<tr>
<td>Borderline</td>
<td>.44 to .46 seconds</td>
<td>.43 to .45 seconds</td>
<td>.45 to .47 seconds</td>
</tr>
<tr>
<td>Prolonged</td>
<td>&gt; .46 seconds</td>
<td>&gt; .45 seconds</td>
<td>&gt; .47 seconds</td>
</tr>
</tbody>
</table>


QTc .50 sec (500 msec or more is dangerous and should be considered an ominous sign of impending Torsade's de Pointes.

QT Interval

- Measured from beginning of QRS complex to the end of the T wave

- Reflects both ventricular depolarization (QRS) and ventricular repolarization (T wave)

- Used most specifically to reflect ventricular repolarization
Heart Rate Adjustment

- QT interval needs to be adjusted for HR
- QT does not adjust to HR on a beat to beat basis

- **Dynamic changes are most important**
- Abnormal findings are uncovered during abrupt changes in the R to R

- **Irregular heart rhythms (i.e. atrial fibrillation) remain a clinical challenge**
What are Early After Depolarizations?

• Right after repolarization (or during) there is a transient sub threshold depolarization
  — Can occur during Phase II or III of the cardiac action potential
  — If an early after depolarization reaches threshold a second upstroke occurs and a triggered beat follows
  — The triggered beat may have its own after depolarization that reaches threshold – thus causing another triggered beat
• Thought to be etiology of Torsade's de Pointes
  — Acquired
  — Congenital

More on Early After Depolarizations

• Precipitating Factors
  — Hypokalemia
  — Hypomagnesemia
  — Heightened sympathetic tone
  — Slow heart rate
  — Prolonged repolarization (QT interval)
Cardiac Ion Channel Abnormalities

- Long QT Syndrome (LQTS)
- Brugada disease
- Idiopathic short QT
  - < 300 to 340 msec
- Diagnosed by family history and ECG

Note: Patients with heart failure can develop channelopathies
LQTS

- QTc > 450 ms
- Genetic defect in either potassium (LQT1 or LQT2) or sodium (LQT3) channels
  - Delayed repolarization (1 and 2)
  - LQT1 and LQT2 = 95%
    - Beta blockers
  - LQT3 = 5%
    - Beta blockers may be harmful
- Autosomal dominant trait
- 1 in 2500
- QT prolongation important risk factor for SCD
  - QTc < 440 ms / < 5%
  - QTc 460 to 500 ms / 20%
  - QTc > 500 ms / 50%

Each Type of Congenital QT Looks Differently in Terms of T Wave Morphology

- Interestingly – some acquired Torsade's may be preceded by T wave morphology looking like congenital LQTS

- Long QT 1: wide, broad-based T waves
- Long QT 2: low amplitude, often notched T waves
- Long QT 3: long ST segment and tall, peaked T waves
wide, broad-based T waves
low amplitude, often notched T waves
long ST segment and tall, peaked T waves

11 year old male LQT1 patient ECG showing a normal T wave pattern and average QTc of about 480 msec.
Brugada Syndrome

- Inherited ion channelopathy.
  - Disorder of cardiac sodium channel (20%)
- Autosomal dominant
  - Most common in Southeast Asian countries
  - 90% of patients are male
- Predispose to Syncope or sudden cardiac death (SCD)
  - Impacts action potential
  - Events occur more commonly at rest or during sleep
  - Events occur in 3rd or 4th decade of life
  - Increased risk for SCD
    - Syncopal episode
    - Early repolarization pattern on ECG
    - Family history of SCD
    - Asymptomatic patients at low risk for SCD
- Treatment
  - ICD
  - Quinidine or isoproterenol for VT Storm
Diagnosis of Brugada Syndrome

- 3 characteristic ECG patterns identified
- If type 1, 2 or 3 ECG findings are present one of the following must also be present to consider a diagnosis of the BS:
  - Documented ventricular fibrillation
  - Self-terminating polymorphic ventricular tachycardia
  - Family history of sudden cardiac death at < 45 years
  - Type 1 ST-segment elevation in family members
  - Electrophysiologic inducibility of VT
  - Unexplained syncope suggestive of a tachyarrhythmia
  - Nocturnal agonal respiration

### ECG Findings With Brugada Syndrome

| Type 1  | Coved ST elevation  
|         | ST gradually descends to an inverted T wave  
<table>
<thead>
<tr>
<th></th>
<th>Present in more than one right precordial lead V1-V3.</th>
</tr>
</thead>
</table>
| Type 2  | T wave remains positive or biphasic  
|         | The terminal portion of the ST-segment is elevated ≥ 1 mm  
|         | Present in more than one right precordial lead V1-V3 |
| Type 3  | T Wave is positive  
|         | The terminal portion of the ST-segment is elevated < 1 mm  
|         | Present in more than one right precordial lead V1-V3 |

Source: Chaturvedi et al., 2011.
Torsade's De Pointes

• Recognition of this life-threatening arrhythmia is important because it is not treated like other VTs
• Two groups: Acquired and congenital
• Acquired
  • Drugs prolonging repolarization
    – Most often as a result of blocking the potassium channel
  • Electrolyte abnormalities
    – Low potassium
    – Low magnesium
  • Severe bradycardias / pauses

More on Drugs that Prolong Repolarization (blocking of potassium channel efflux)

  • www.QTdrugs.org
  • www.torsades.org

• Class Ia and Class III antiarrhythmics
• Antihistamines
• Antibiotics
• Antipsychotics
• Antidepressants
• Sedatives
• Gastric motility agents
• Anticancer agents
• Opiate agonists

✓ Risk
✓ Possible Risk
✓ Conditional
Other Risk Factors for Torsade's de Pointes

- Rapid (IV) administration of QT prolonging agent
- Renal or hepatic dysfunction
- Female gender (particularly for drug induced)
- Advanced age
- Anorexia
- Heart disease
- Poly pharmacy
Warning signs for Torsades de Pointes

- Increase QTc from predrug baseline of 60 ms,
- Marked QTc interval prolongation .500 ms
- T-U wave distortion that becomes more exaggerated in the beat after a pause
- Visible (macroscopic) T-wave alternans
- New-onset ventricular ectopy, couplets
- Nonsustained polymorphic ventricular tachycardia initiated in the beat after a pause.
Torsade's de Pointes

• Class I
  – Discontinue offending drugs
    • Note: Class IA drug induced TdP usually appears soon after the initial administration of the drug
  – Correct electrolytes
    • Magnesium
    • Potassium
  – Increase HR
    • Isoproterenol
      – 2 mcg/min then titrate to HR of 100 beats per minute
    • Temporary pacing at rate of 100 to 110
    • Permanent pacing if bradycardia or CHB cannot be resolved.

• Defibrillation if sustained
  – However, continue to assess for and treat cause

Magnesium is considered treatment of choice.
More on Magnesium in Torsade's de Pointes

- 2 Gm IV bolus over 1-2 minutes
  - Followed in 15 minutes by another bolus if necessary
  - May start continuous infusion at rate of 3-20 mg/min
- Benefit occurs without shortening of QT interval and in presence of normal Magnesium level

Case Example
QT Interval Monitoring Case Example

- Patient admitted for syncope after having motor vehicle crash while driving.
- Long standing history of paroxysmal atrial fibrillation – on dofetilide (Tykosin) for several years.
- Recent chemotherapy for breast CA resulting in a reduction of EF.
- Recent increase in carvedilol and lisinopril per general cardiology to improve EF.

- Next slide is admission ECG. Note the QTc interval.
1. Strip 1: QTc consistent with admission ECG.
2. Strip 2: Marked QTc prolongation when patient asleep.
3. Initial run of ventricular tachycardia initiated by PVC firing at end of T wave,

Same patient with sustained Torsades de Pointes. Treated effectively with 2 grams IV Magnesium (magnesium level was normal at baseline). Magnesium is the drug of choice to stabilize the cardiac membrane. Dofetilide (Tikosyn) was also discontinued.

Note: Although the patient had been on dofetilide (Tikosyn) for several years, the recent change in ejection fraction and increase in beta blocker therapy increased her risk for Torsades de Pointes.
Polymorphic VT with normal QT:

- Seen frequently in ischemic conditions
  - Think revascularization
  - Think beta blockers

Special Considerations: Polymorphic VT (normal QT)

- DC cardioversion with sedation when unstable
- IV beta-blockers if ischemia suspected
  - Improve mortality
- IV amiodarone in absence of abnormal repolarization
  - Amiodarone better than placebo
  - Magnesium not better than placebo
- Urgent angiography to exclude ischemia
- Lidocaine may be reasonable if ischemia suspected
- Check electrolytes
- Consider any other potential reversible cause
WOLFF-PARKINSON-WHITE (WPW) SYNDROME

Atrioventricular Reciprocating Tachycardias (AVRT)

- Requires the presence of a bypass tract or accessory pathway
- Most common: Kent bundles in “Wolf Parkinson White” Syndrome
- Left lateral free wall, right lateral free wall, and posterior septum
Concept of Pre-excitation

- Termed Pre-excitation because some conduction occurs via the Kent bundles in addition to the normal pathway; because conduction via the Kent bundles is faster than via the AV node the ventricles are pre-excited

- This produces a “delta wave” on the EKG
- Fusion beat
  - Short PR
  - Wider than normal QRS
Delta Wave of Pre-excitation Syndrome

- 60 to 70% of WPW shows evidence in SR

Left sided accessory pathway:
- Positive delta wave in V1

Right sided accessory pathway:
- Negative delta wave in V1
Arrhythmias of WPW (AVRT or CMT)
Atrioventricular Reentrant Tachycardia (AVRT)

- **Orthodromic**
  - Traveling down the AV junction and up an accessory pathway
  - Sequential depolarization
  - Narrow because travel via the AV node
  - More common than antidromic tachycardia

- **Antidromic**
  - Activation of the ventricles is initiated by impulses descending via an accessory pathway
  - Ventricular depolarization begins at an ectopic site in the myocardium and returns via the AV node

Presence of pre-excitation on 12 lead and paroxysmal palpitations.

Orthodromic Tachycardia

This WPW tachycardia can mimic AVNRT (most common form of SVT).

In AVNRT ‘p’ waves are usually buried in the QRS.
In orthodromic tachycardia the ‘p’ wave is typically distinct from the QRS.
62 year old male presenting to ED 4 hours after onset of palpitations. BP 110/72, pale and anxious.
Antidromic Tachycardia

- The less common form of atrioventricular reentrant tachycardia
- The path of tachycardia passes from the atrium to the ventricle via the accessory pathway (Kent bundles) and returns to the atrium via the AV node
- The QRS complex is wide because antegrade conduction bypasses the AV node
- Antidromic tachycardia is very difficult to distinguish from ventricular tachycardia because ventricular depolarization begins where the accessory pathway enters the ventricle
  - Negative concordance will not be antidromic tachycardia
WPW and Atrial Fibrillation

- Mechanism of Action
  - Development of Atrial Fibrillation in WPW
    - 10-32% of patients
  - Refractory period of accessory pathway is short
AF in WPW

Accessory Pathway
Treatment for WPW Tachycardias

• AV Reentrant (orthodromic)
• AV Reentrant (antidromic)
• Atrial Fib with antegrade conduction over accessory pathway

Slow conduction over accessory pathway:
• Procainamide
• Flecainide
• Sotalol
• Propafenone
• Ibutelide
Catheter Ablation of Accessory Pathway in Wolff-Parkinson-White Syndrome

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LET’S PULL IT ALL TOGETHER!!
Final Case Study: ECG #1

Final Case Study: ECG #2
Final Case Study: ECG #3

Final Case Study: ECG #4
Final Case Study: ECG #5

Final Case Study: ECG #6
Class Lesson Summary!

The 12 lead ECG is an inexpensive, noninvasive, risk-free diagnostic tool, that can provide you with an amazing amount of information about your patient’s heart.

However: Always treat the patient not the ECG or rhythm strip.

A Final Thought

Impact every patient and family on their journey and provide safe passage by meeting them where they are, connecting with them in a meaningful way, and delivering care with wisdom and intention.

- Karen, Carol and Cindy
THANK YOU!!!

Enjoy NTI!

BE THE BEST THAT YOU CAN BE EVERY DAY. YOUR PATIENTS ARE COUNTING ON IT!

cindy@cardionursing.com

Final slides will be available at www.cardionursing.com next week