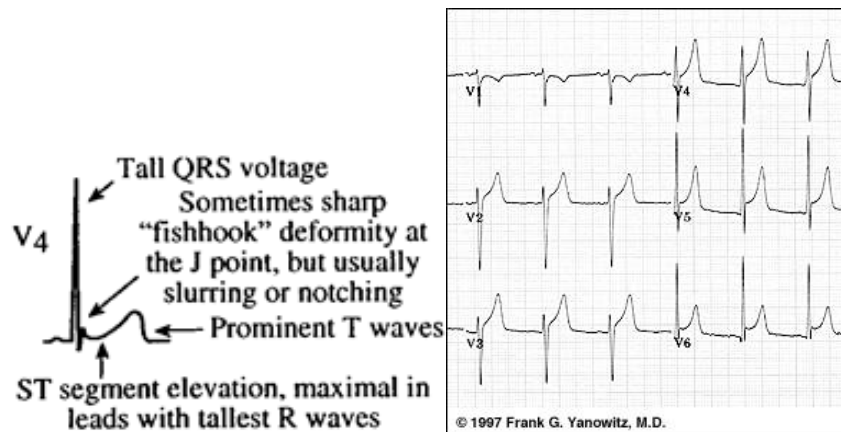


## EKG Abnormalities

### I. Early repolarization abnormality:

**A. A normal variant.** Early repolarization is most often seen in healthy young adults. Look for ST elevation, tall QRS voltage, "fishhook" deformity at the J point, and prominent T waves. ST segment elevation is maximal in leads with tallest R waves. Note high take off of the ST segment in leads V4-6; the ST elevation in V2-3 is generally seen in most normal ECG's; the ST elevation in V2-6 is concave upwards, another characteristic of this normal variant.



#### *Characteristics' of early repolarization*

- notching or slurring of the terminal portion of the QRS wave
- symmetric concordant T waves of large amplitude
- relative temporal stability
- most commonly presents in the precordial leads but often associated with it is less pronounced ST segment elevation in the limb leads

#### *To differentiate from anterior MI*

- the initial part of the ST segment is usually flat or convex upward in AMI
- reciprocal ST depression may be present in AMI but not in early repolarization
- ST segments in early repolarization are usually <2 mm (but have been reported up to 4 mm)

#### *To differentiate from pericarditis*

- the ST changes are more widespread in pericarditis
- the T wave is normal in pericarditis
- the ratio of the degree of ST elevation (measured using the PR segment as the baseline) to the height of the T wave is greater than 0.25 in V6 in pericarditis.

## **II. Acute Pericarditis:**

### **Stage 1 Pericarditis Changes**

- A. Timing
  - 1. Onset: Day 2-3
  - 2. Duration: Up to 2 weeks
- B. Findings
  - 1. Diffuse concave upward ST segment elevation
  - 2. ST segment depression in leads aVR or V1
  - 3. Concordant T Wave changes
  - 4. PR Segment depression in leads II, AVF, and V4-V6
- C. Stage I (with PR segment abnormalities):



- 1. Look for widespread ST segment elevation with concomitant PR depression in the same leads. The PR segment in aVR sticks above the baseline like a knuckle, reflecting atrial injury.

### **Stage 2 Pericarditis Changes**

- A. Timing
  - 1. Duration: Days to several weeks
- B. Findings
  - 1. ST segment returns to baseline
  - 2. T Wave flattens

### **Stage 3 Pericarditis Changes**

- A. Timing
  - 1. Onset: Week 2-3
  - 2. Duration: Several weeks
- B. Findings
  - 1. ST segment returns to baseline
  - 2. T Wave inverts in leads II, AVF, and V4-V6

### **Stage 4 Pericarditis Changes**

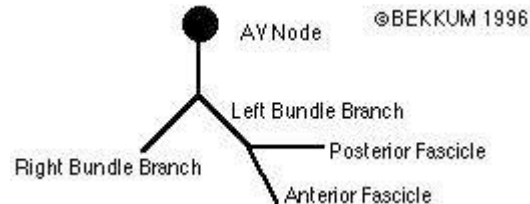
- A. Timing
  - 1. Duration: Up to 3 months

B. Findings

1. Gradual resolution of T Wave inversion

### III. Fascicular/Hemiblocks

**Fascicular/Hemiblocks:** Blocks of either the anterior or posterior division of the L bundle branch



Diagnosed by looking for (L) or (R) axis deviation

#### Left anterior hemiblock

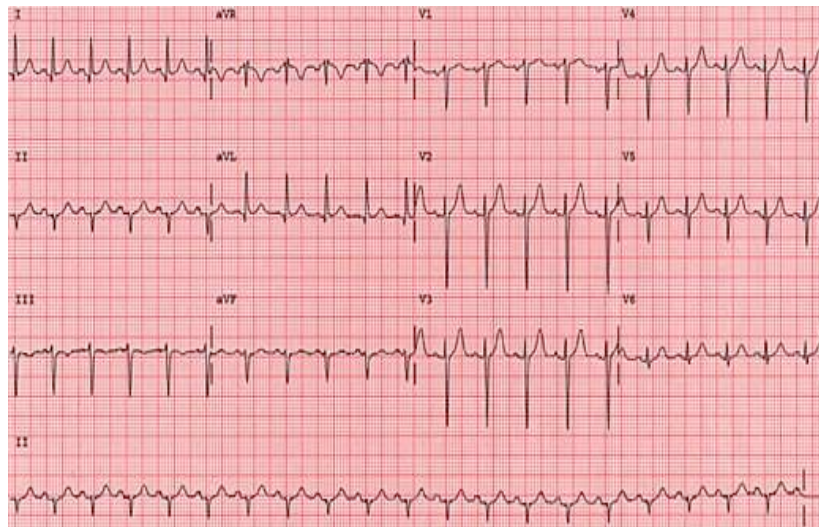
1. Normal QRS and no ST or T wave changes
2. LAD
3. No other cause or LAD is present
4. Q<sub>1</sub>, S<sub>3</sub>

#### Left Posterior Hemiblock

1. Slightly widened or normal QRS and no ST or T wave changes
2. RAD
3. No other cause of RAD present
4. S<sub>1</sub>, Q<sub>3</sub>

#### ***Anterior fascicular block - the most common.***

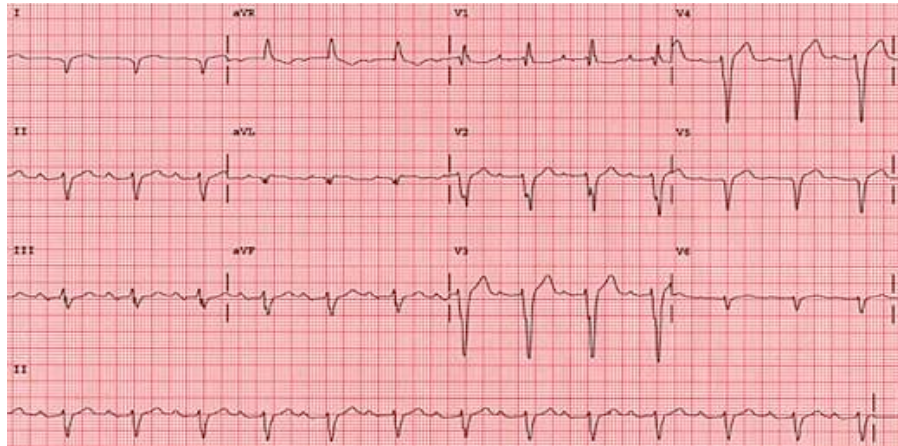
You will see left axis deviation (-30 to -90) and a small Q wave in lead I and an S in lead III (Q1S3). The QRS will be slightly prolonged (0.1 - 0.12 sec).



Anterior fascicular block

***Posterior fascicular block - less common.***

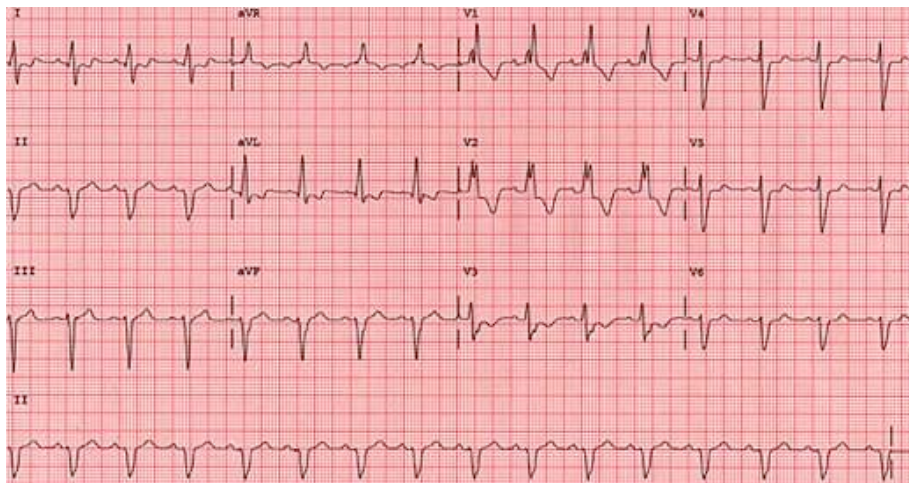
You will see right axis deviation, an S in lead I and an Q in lead III (S1Q3). The QRS will be slightly prolonged (0.1 - 0.12 sec).



Posterior fascicular block

***Bifascicular block.***

This means two (2) of the three (3) fascicles (in diagram) are blocked. The most important example is a right bundle branch block and a left anterior fascicular block. Watch out for this. Only one fascicle is left for conduction, and if that fascicle is intermittently blocked, the dangerous Mobitz 2 is set up!



Right bundle branch block and left anterior fascicular block

**"Fascicular Blocks" may seem a bit complicated - simply remember that axis deviation is the clue.** In your differential, consider posterior fascicular blocks with right axis deviation and consider anterior fascicular blocks with left axis deviation. Fascicular blocks cause axis deviations, like infarcts and hypertrophy. If you see a left or right axis deviation, first look for infarct or hypertrophy. If neither are present, the remaining diagnosis of fascicular block is usually correct.

## **Ventricular Hypertrophy**

Look at the QRS complexes in all leads.

### ***Left ventricular hypertrophy (LVH)***

***There are many different criteria for LVH.*** (Remember that the L ventricle wall is very thick)

1. S wave in V<sub>1</sub> or V<sub>2</sub> (in mm) + R wave in V<sub>5</sub> or V<sub>6</sub> (in mm) = 35mm or greater
2. Any precordial lead is  $\geq 45$  mm
3. The R wave in aVL is  $\geq 11$  mm
4. The R wave in lead I is  $\geq 12$  mm
5. The R wave in lead aVF is  $\geq 20$  mm
6. Also may be present
  - a. LAD with slightly widened QRS
  - b. Inverted T wave (in V<sub>5</sub> & V<sub>6</sub>) - slants downward slowly & up rapidly

Hypertrophy with “Strain”: The ST segment becomes depressed and humped with either of the above.

**Right V. Hypertrophy** is characterized by:

1. RAD  $> 100^{\circ}$
2. R wave  $>$  S in V<sub>1</sub>, but R wave gets progressively smaller in V<sub>1</sub> - V<sub>6</sub>
3. S wave persists in V<sub>5</sub> and V<sub>6</sub>

### ***Left atrial abnormality (dilatation or hypertrophy)***

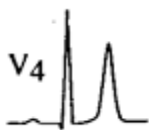
- M shaped P wave in lead II
- prominent terminal negative component to P wave in lead V1

## ***Hyperkalaemia***

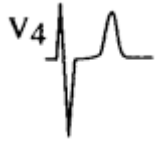
The following changes may be seen in hyperkalaemia

- small or absent P waves
- atrial fibrillation
- wide QRS
- shortened or absent ST segment
- wide, tall and tented T waves
- ventricular fibrillation
- Patterns are best seen in leads V4-V5.

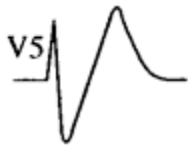
Common: Tall, peaked, narrow-based, symmetric T waves.



The most common pattern: R/S ratio < 1 in V4, with broad, prominent S waves and symmetric, not necessarily peaked, T waves.

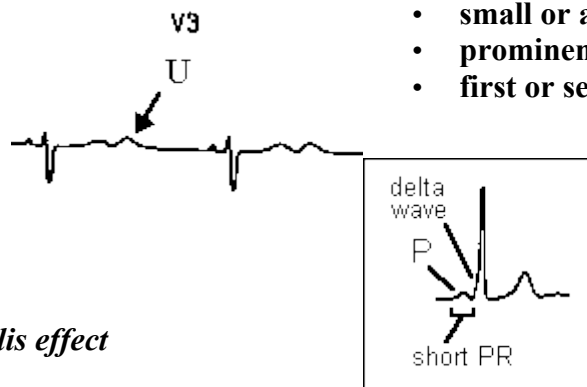


An "M" shaped QRS-ST-T morphology in lead V4-5.



### Hypokalaemia

The following changes may be seen in hypokalaemia.



- small or absent T waves
- prominent U waves (see diagram)
- first or second degree AV block
- slight depression of the ST segment

### Digitalis effect

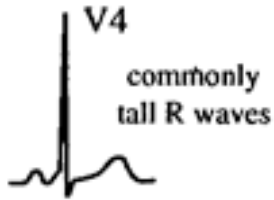
- shortened QT interval
- characteristic down-sloping ST depression, reverse tick appearance, (shown here in leads V5 and V6)
- dysrhythmias
  - ventricular / atrial premature beats
  - paroxysmal atrial tachycardia with variable AV block
  - ventricular tachycardia and fibrillation
  - many others

### Wolf-Parkinson-White syndrome

- short PR interval, less than 3 small squares (120 ms)
- slurred upstroke to the QRS indicating pre-excitation (delta wave)
- broad QRS
- secondary ST and T wave changes

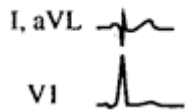
### The WPW pattern:

Most commonly seen as an absent PR segment and initial slurring of the QRS complex in any lead. The lead with the best sensitivity is V4.

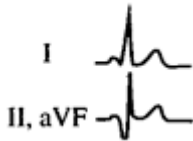


### Typical WPW patterns:

- Left lateral accessory pathway (mimics lateral or posterior MI):



- Posteroseptal accessory pathway (mimics infero-posterior MI):



### *Lown Ganong Levine Syndrome*



- short PR interval, less than 3 small squares (120 ms)
- no delta wave

### *Acute pulmonary embolus*

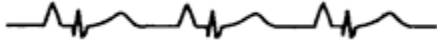
The following, often transient, changes may be seen in a large pulmonary embolus.

- an S1Q3T3 pattern
  - a prominent S wave in lead I
  - a Q wave and inverted T wave in lead III
- sinus tachycardia
- T wave inversion in leads V1 - V3
- Right Bundle Branch Block
- low amplitude deflections

### **COPD pattern:**

The P wave amplitude in the inferior leads is equal to that of the QRS complexes.

In lead II, look for prominent P waves with low QRS voltage



### **Atrial Enlargement**

Look at the P wave in leads II and V<sub>1</sub>,

Right Atrial Enlargement:

1. Increased amplitude of the first portion of the P wave
2. No change in the duration of the P wave
3. Possible RAD of the P wave

#### **Right atrial enlargement (RAE):**

RAE is synonymous with RVH. The best criterion is a positive component of the P wave in lead V<sub>1</sub> OR V<sub>2</sub>  $\geq 1.5$  mm, shown below.



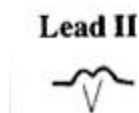
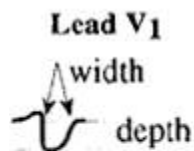
Another criterion is a P wave amplitude in lead II  $> 2.5$  mm. Caveat: A tall, peaked P in lead II may represent RAE, but is more commonly due to either COPD and/or increased sympathetic tone.

Left Atrial Enlargement:

1. Occasionally, increased amplitude of the terminal component of the P wave
2. More consistently, increased P wave duration
3. No significant axis deviation

#### **Left atrial enlargement (LAE):**

The most sensitive lead for the diagnosis of LAE is lead V<sub>1</sub>, but criteria for lead II are more specific. With LAE, consider: LVH, coronary artery disease, mitral valve disease or cardiomyopathy.



In lead V<sub>1</sub>, look for a terminal negative wave  $\geq 1$  mm deep and  $\geq 40$  msec wide (one



small box by one small box in area). In lead II, look for > 40 msec between the first (R) & second (L) atrial components.

### **Bundle Branch Blocks**

Look at the width and configuration of the QRS complexes

#### **RBBB:**

1. QRS > 0.12 sec
2. RSR' in leads V<sub>1</sub> & V<sub>2</sub> (rabbit ears) with ST segment depression and T wave inversion
3. Reciprocal changes in left lateral leads (V<sub>5</sub>, V<sub>6</sub>, I & AVL)

#### **LBBB**

1. QRS > 0.12 sec
2. Broad or notched R wave with prolonged upstroke in leads V<sub>5</sub>, V<sub>6</sub>, I and AVL with ST segment depression and T wave inversion
3. Reciprocal changes in V<sub>1</sub> & V<sub>2</sub>
4. LAD may be present

#### **Incomplete BBB**

1. R,R' and/in QRS of normal duration

**Preexcitation** the accessory conduction pathways act as short circuits, allowing the atrial wave of depolarization to bypass the AV node and activate the ventricles prematurely.

#### WPW-Bundle of Kent (bypass pathway)

1. PRI < 0.12 sec
2. Wide QRS
3. Delta wave seen in some leads

#### LGL (Lown-Ganong-Levine) Syndrome-James fibers pathway

1. PRI < 0.12 sec
2. Normal QRS width
3. No delta wave

### ***The ST Segment***

Elevation may be seen:

1. With an evolving infarction
2. In Prinzmetal's angina

Depression may be seen:

1. With typical exertional angina
2. In non-Q wave infarction

## Miscellaneous EKG Changes

### Electrolytes

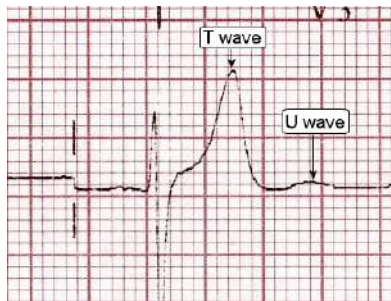
1. Hyperkalemia: evolution of peaked T waves, PR prolongation and P wave flattening and QRS widening. Ultimately, V-fib will develop if not corrected.
2. Hypokalemia: ST depression, T wave flattening, U waves
3. Hypocalcemia: Prolonged QT interval
4. Hypercalcemia: Shortened QT interval

### Drugs

1. Digitalis: Toxic level associated with tachyarrhythmias and conduction blocks; PAT with block is most characteristic.
2. Quinidine: Prolonged QT interval, U waves

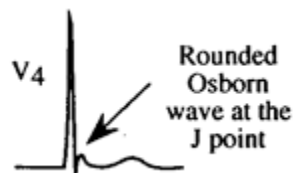
### U waves

The precise physiologic meaning is not fully understood. They are most characteristic in hypokalemia.



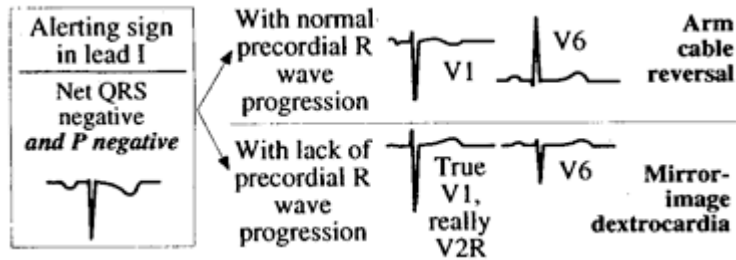
### Hypothermia:

Usually characterized by a slow rate, long QT interval, and muscle tremor artifact. An Osborn wave is typically present.



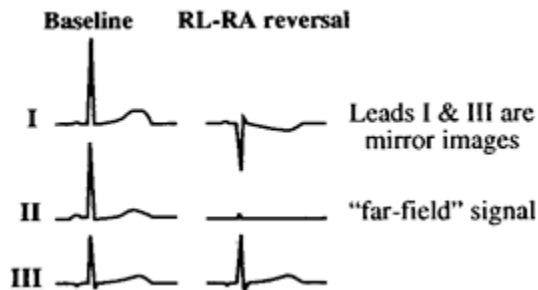
### Lead misplacement:

### Right-left arm lead reversal vs. mirror-image dextrocardia:



**Misplacement of the right leg cable:**

This error should not happen, but is common. It produces a "far-field" signal, which occurs when one of the bipolar leads (I, II or III) records the signal between the left and right legs. The lead appears to have no signal, save for a tiny deflection representing the QRS complex. There are usually no discernible P waves or T waves. RL-RA cable reversal is illustrated.



**TABLE 14-3 -- Clues to Improper Limb Lead Connections**

| Reversed Leads | Old ECG Necessary for Detection? | Key Findings   |
|----------------|----------------------------------|--|
| LA RA          | No                               | PQRST upside down in lead I  |
|                |                                  | Precordial leads normal (not dextrocardia)                         |
| LA LL          | Yes                              | III is upside down   |
|                |                                  | I ↔ II; aVL ↔ aVF; aVR no change                                   |
| LA RL          | No                               | III is straight line   |
| RA LL          | No                               | PQRST upside down in all leads except aVL                          |
| RA RL          | No                               | II is straight line  |
| LL RL          | Cannot detect change             | Looks like normal lead placement                                   |
| LA LL          | No                               | I is straight line   |
| RA RL          |                                  | aVL, aVR are same polarity and amplitude and II is upside down III |

## **Prolonged QT**

The QT interval represents repolarization, or "recharge," of a cardiac cell. After the heart's electrical impulse stimulates a heart cell (thus causing it to beat), recharging must occur in order for the cell to be ready for the next electrical impulse.

The QT interval is measured from the beginning of the QRS complex to the end of the T wave. In Long QT Syndrome, the QT interval is prolonged.

It represents the time between the start of ventricular depolarization and the end of ventricular repolarization. It is useful as a measure of the duration of repolarization.

The QT interval will vary depending on the heart rate, age and gender.

It increases with bradycardia and decreases with tachycardia. Men have shorter QT intervals (0.39 sec) than women (0.41 sec). The QT interval is influenced by electrolyte balance, drugs, and ischemia.

### Heart Rate determined QT

1. 115 - 84 bpm: QT 0.30 to 0.37 seconds
2. 83 - 72 bpm: QT 0.32 to 0.40 seconds
3. 71 - 63 bpm: QT 0.34 to 0.42 seconds
4. 62 - 56 bpm: QT 0.36 to 0.43 seconds
5. 55 - 45 bpm: QT 0.39 to 0.46 seconds

### **The four major causes of a prolonged QT interval:**

#### **1. Electrolyte abnormalities:**

Hypokalemia and hypocalcemia

#### **2. Drugs:** (also associated with torsades de pointes)

- Class Ia antiarrhythmic agents: quinidine, procainamide, disopyramide
- Class Ic agents: propafenone
- Class III agents: amiodarone, bretylium, dofetilide, n-acetylprocainamide, sotalol
- Psychotropic agents: tricyclic antidepressants, tetracyclic antidepressants, phenothiazines, haloperidol
- Antihistamines: astemizole, terfenadine
- Antibiotics: erythromycin, trimethoprim-sulfamethoxazole
- Antifungals: ketoconazole, itraconazole
- Serotonin antagonists: ketanserin, zimeldine
- Chemotherapeutics: pentamidine, possibly anthracyclines
- Miscellaneous: bepridil, cisapride, prednisone, prenylamine, probucol, chloral hydrate
- Toxins and poisons: organophosphate insecticides, anthopleurinn-A, liquid protein diets, some herbs

#### **3. Congenital long Q-T syndromes:**

While congenital long QT syndromes are rare, identification of a patient with this

problem may allow for life-saving therapy to be instituted. It should be searched for in any young patient who presents with syncope or presyncope.

4. **A miscellaneous group, including patients with:**
  - Third-degree and sometimes second-degree A-V block
  - At the cessation of ventricular pacing
  - Left ventricular hypertrophy (usually minor degrees of lengthening)
  - Myocardial infarction (in the evolutionary stages where there are marked repolarization abnormalities)
  - Significant active myocardial ischemia
  - Cerebrovascular accident (subarachnoid hemorrhage)
  - Hypothermia

**The four causes of a short QT interval:**

1. Hypercalcemia
2. Digitalis
3. Thyrotoxicosis
4. Increased sympathetic tone