INTRODUCTION

ECG terminology and diagnostic criteria often vary from book to book and from one teacher to another. In this tutorial an attempt has been made to conform to standardized terminology and criteria, although new diagnostic concepts derived from the recent ECG literature have been included in some of the sections. Finally, it is important to recognize that the mastery of ECG interpretation, one of the most useful clinical tools in medicine, can only occur if one acquires considerable experience in reading ECG's and correlating the specific ECG findings with the pathophysiology and clinical status of the patient.

The sections in this tutorial are organized in the same order as the recommended approach outlined in the "Method" of ECG interpretation (see Chapter 2, p7). Beginning students should first go through the sections in the order in which they are presented. Others may choose to explore topics of interest in any order they wish. It is hoped that all students will be left with some of the love of electrocardiography shared by Dr. Lindsay.

TABLE OF CONTENTS

<table>
<thead>
<tr>
<th>1. The Standard 12 Lead ECG (p. 4)</th>
<th>7. Atrial Enlargement (p. 52)</th>
</tr>
</thead>
<tbody>
<tr>
<td>2. A &quot;Method&quot; of ECG Interpretation (p. 7)</td>
<td>8. Ventricular Hypertrophy (p. 53)</td>
</tr>
<tr>
<td>3. Characteristics of the Normal ECG (p. 12)</td>
<td>9. Myocardial Infarction (p. 57)</td>
</tr>
<tr>
<td>4. ECG Measurement Abnormalities (p. 14)</td>
<td>10. ST Segment Abnormalities (p. 68)</td>
</tr>
<tr>
<td>5. ECG Rhythm Abnormalities (p. 17)</td>
<td>11. T Wave Abnormalities (p. 72)</td>
</tr>
<tr>
<td>6. ECG Conduction Abnormalities (p. 43)</td>
<td>12. U Wave Abnormalities (p. 74)</td>
</tr>
</tbody>
</table>

Basic Competency in Electrocardiography
(Modified from: ACC/AHA Clinical Competence Statement, JACC 2001;38:2091)

In 2001 a joint committee of the American Collage of Cardiology and the American Heart Association published a list of ECG diagnoses considered to be important for developing basic competency in ECG interpretation. This list is illustrated on the following page and is also illustrated on the website with links to examples or illustrations of the specific ECG diagnosis. Students of electrocardiography are encouraged to study this list and become familiar with the recognition of these ECG diagnoses. Most of the diagnoses are illustrated in this tutorial.
Basic Competency in Electrocardiography

NORMAL TRACING
- Normal ECG

TECHNICAL PROBLEM
- Lead misplaced
- Artifact

SINUS RHYTHMS/ARRHYTHMIAS
- Sinus rhythm (50-90 bpm)
- Sinus tachycardia (>90 bpm)
- Sinus bradycardia (<50 bpm)
- Sinus Arrhythmia
- Sinus arrest or pause
- Sino-atrial exit block

OTHER SV ARRHYTHMIAS
- PAC's (nonconducted)
- PAC's (conducted normally)
- PAC's (conducted with aberration)
- Ectopic atrial rhythm or tachycardia (unifocal)
- Multifocal atrial rhythm or tachycardia
- Atrial fibrillation
- Atrial flutter
- Junctional premature
- Junctional escapes or rhythms
- Accelerated Junctional rhythms
- Junctional tachycardia
- Paroxysmal supraventricular tachycardia

Ventricular Arrhythmias
- PVC's
- Ventricular escapes or rhythm
- Accelerated ventricular rhythm
- Ventricular tachycardia (uniform)
- Ventricular tachycardia (polymorphous or torsades)
- Ventricular fibrillation

AV CONDUCTION
- 1st degree AV block
- Type I 2nd degree AV block (Wenckebach)
- Type II 2nd degree AV block (Mobitz)
- AV block, advanced (high grade)
- 3rd degree AV block (junctional escape rhythm)
- 3rd degree AV block (ventricular escape rhythm)
- AV dissociation (default)
- AV dissociation (usurpation)
- AV dissociation (AV block)

INTRAVENTRICULAR CONDUCTION
- Complete LBBB, fixed or intermittent
- Incomplete LBBB
- Complete RBBB, fixed or intermittent
- Incomplete RBBB
- Left anterior fascicular block (LAFB)
- Left posterior fascicular block (LPFB)
- Nonspecific IVCD

QRS AXIS AND VOLTAGE
- WPW preexcitation pattern
- Right axis deviation (+90 to +180)
- Left axis deviation (-30 to -90)
- Bizarre axis (-90 to -180)
- Indeterminate axis
- Low voltage frontal plane (<0.5 mV)
- Low voltage precordial (<1.0 mV)

HYPERTROPHY/ENLARGEMENTS
- Left atrial enlargement
- Right atrial enlargement
- Left ventricular hypertrophy
- Right ventricular hypertrophy

ST-T, AND U ABNORMALITIES
- Early repolarization (normal variant)
- Nonspecific ST-T abnormalities
- ST elevation (transmural injury)
- ST elevation (pericarditis pattern)
- Symmetrical T wave inversion
- Hyperacute T waves
- Prominent upright U waves
- U wave inversion
- Prolonged QT interval

MI PATTERNS (acute, recent, old)
- Interior MI
- Inferoposterior MI
- Inferoposterolateral MI
- True posterior MI
- Anteroseptal MI
- Anterior MI
- Anterolateral MI
- High lateral MI
- Non Q-wave MI
- Right ventricular MI

CLINICAL DISORDERS
- Chronic pulmonary disease pattern
- Suggests hypokalemia
- Suggests hyperkalemia
- Suggests hypocalcemia
- Suggests hypercalcemia
- Suggests digoxin effect
- Suggests digoxin toxicity
- Suggests CNS disease

PACEMAKER ECG
- Atrial-paced rhythm
- Ventricular paced rhythm
- AV sequential paced rhythm
- Failure to capture (atrial or ventricular)
- Failure to inhibit (atrial or ventricular)
- Failure to pace (atrial or ventricular)
1. THE STANDARD 12 LEAD ECG

The standard 12-lead electrocardiogram is a representation of the heart's electrical activity recorded from electrodes on the body surface. This section describes the basic components of the ECG and the standard lead system used to record the ECG tracings.

The diagram illustrates ECG waves and intervals as well as standard time and voltage measures on the ECG paper.

ECG WAVES AND INTERVALS: What do they mean?

- **P wave**: *sequential* depolarization of the right and left atria
- **QRS complex**: right and left ventricular depolarization
- **ST-T wave**: ventricular repolarization
- **U wave**: origin of this wave is still being debated!
- **PR interval**: time interval from onset of atrial depolarization (P wave) to onset of ventricular muscle depolarization (QRS complex)
- **QRS duration**: duration of ventricular muscle depolarization (width of the QRS complex)
- **QT interval**: duration of ventricular depolarization and repolarization
- **PP interval**: rate of atrial or sinus cycle
- **RR interval**: rate of ventricular cycle
ORIENTATION OF THE 12-LEAD ECG:

It is important to remember that the 12-lead ECG provides spatial information about the heart's electrical activity in 3 *approximately* orthogonal directions (think: X,Y,Z):
- Right – Left (X)
- Superior – Inferior (Y)
- Anterior – Posterior (Z)

Each of the 12 leads represents a particular orientation in space, as indicated below (RA = right arm; LA = left arm, LL = left leg):
- **Bipolar limb leads (frontal plane):**
  - Lead I: RA (- pole) to LA (+ pole) (Right -to- Left direction)
  - Lead II: RA (-) to LL (+) (mostly Superior -to- Inferior direction)
  - Lead III: LA (-) to LL (+) (mostly Superior -to- Inferior direction)
- **Augmented limb leads (frontal plane):**
  - Lead aVR: RA (+) to [LA & LL] (-) (Rightward direction)
  - Lead aVL: LA (+) to [RA & LL] (-) (Leftward direction)
  - Lead aVF: LL (+) to [RA & LA] (-) (Inferior direction)
- **"Unipolar" (+) chest leads (horizontal plane):**
  - Leads V1, V2, V3: (Posterior -to- Anterior direction)
  - Leads V4, V5, V6: (Right -to- Left direction)

Behold: Einthoven's Triangle! Each of the 6 frontal plane or "limb" leads has a negative and positive pole (as indicated by the '+' and '-' signs). It is important to recognize that lead I (and to a lesser extent aVL) are right -to- left in direction. Also, lead aVf (and to a lesser extent leads II and III) are superior -to- inferior in direction. The diagram below further illustrates the frontal plane hookup.
Note: the actual ECG waveform in each of the 6 limb leads varies from person to person depending on age, body size, gender, frontal plane QRS axis, presence or absence of heart disease, and many other variables. The precordial leads are illustrated below.

**Precordial lead placement**

V1: 4th intercostal space (IS) adjacent to right sternal border

V2: 4th IS adjacent to left sternal border

V3: Halfway between V2 and V4

V4: 5th IS, midclavicular line

V5: horizontal to V4; anterior axillary line

V5: horizontal to V4-5; midaxillary line

(Note: in women, the precordial leads should be placed on the breast surface not under the breast to insure proper lead placement)
2. A "METHOD" OF ECG INTERPRETATION

This "method" is recommended when reading 12-lead ECG's. Like the approach to doing a physical exam, it is important to follow a standardized sequence of steps in order to avoid missing subtle abnormalities in the ECG tracing, some of which may have clinical importance. The 6 major sections in the "method" should be considered in the following order:

1. **Measurements**
2. **Rhythm analysis**
3. **Conduction analysis**
4. **Waveform description**
5. **ECG interpretation**
6. **Comparison with previous ECG (if any)**

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1. **MEASUREMENTS (usually made in frontal plane leads):**
   - Heart rate (state both atrial and ventricular rates, if different)
   - PR interval (from beginning of P to beginning of QRS complex)
   - QRS duration (width of most representative QRS)
   - QT interval (from beginning of QRS to end of T)
   - QRS axis in frontal plane (see "How to Measure QRS Axis" on p8)

2. **RHYTHM ANALYSIS:**
   - State basic rhythm (e.g., "normal sinus rhythm", "atrial fibrillation", etc.)
   - Identify additional rhythm events if present (e.g., "PVC's", "PAC's", etc)
   - Remember that arrhythmias may originate from atria, AV junction, and ventricles

3. **CONDUCTION ANALYSIS:**
   - "Normal" conduction implies normal sino-atrial (SA), atrio-ventricular (AV), and intraventricular (IV) conduction.
   - The following conduction abnormalities are to be identified if present:
     - 2nd degree SA block (type I vs. type II)
     - 1st, 2nd (type I vs. type II), and 3rd degree AV block
     - IV blocks: bundle branch, fascicular, and nonspecific blocks
     - Exit blocks: blocks just distal to ectopic pacemaker site

4. **WAVEFORM DESCRIPTION:**
   - Carefully analyze each of the 12-leads for abnormalities of the waveforms in the order in which they appear: P-waves, QRS complexes, ST segments, T waves, and.... Don't forget the U waves.
     - P waves: are they too wide, too tall, look funny (i.e., are they ectopic), etc.?
     - QRS complexes: look for pathologic Q waves, abnormal voltage, etc.
     - ST segments: look for abnormal ST elevation and/or depression.
     - T waves: look for abnormally inverted T waves.
     - U waves: look for prominent or inverted U waves.

5. **ECG INTERPRETATION:**
   - This is the conclusion of the above analyses. Interpret the ECG as "Normal", or "Abnormal". Occasionally the term "borderline" is used if unsure about the significance of certain findings or for minor changes. List all abnormalities. Examples of "abnormal" statements are:
- Inferior MI, probably acute
- Old anteroseptal MI
- Left anterior fascicular block (LAFB)
- Left ventricular hypertrophy (LVH)
- Right atrial enlargement (RAE)
- Nonspecific ST-T wave abnormalities
- Specific rhythm abnormalities

**Example of a 12-lead ECG interpretation:**

![ECG Image]

- HR=72 bpm; PR=0.16 s; QRS=0.09 s; QT=0.36 s; QRS axis = -70° (left axis deviation)
- Normal sinus rhythm; normal SA, AV, and IV conduction; rS waves in leads II, III, aVF
- **Interpretation:** Abnormal ECG: 1) Left anterior fascicular block

**6. COMPARISON WITH PREVIOUS ECG:**
- If there is a previous ECG in the patient’s file, the current ECG should be compared with it to see if any significant changes have occurred. These changes may have important implications for clinical management decisions.

**HOW TO MEASURE THE QRS AXIS:**

**INTRODUCTION:** The frontal plane QRS axis represents the average direction of ventricular depolarization forces in the frontal plane. As such this measure can inform the ECG reader of changes in the sequence of ventricular activation (e.g., left anterior fascicular block), or it can be an indicator of myocardial damage (e.g., inferior myocardial infarction). Determination of the QRS axis requires knowledge of the direction of the individual frontal plain ECG leads. Einthoven’s triangle enables us to visualize this.
In the diagram below the normal range is shaded (-30° to +90°). In the adult left axis deviation (i.e., superior and leftward) is defined from -30° to -90°, and right axis deviation (i.e., inferior and rightward) is defined from +90° to +180°. From -90 to -180 degrees is very unusual and may indicate lead misplacement.

QRS Axis Determination:

- First find the isoelectric lead if there is one; i.e., the lead with equal forces in the positive and negative direction (i.e., above and below the baseline). Often this is the lead with the smallest QRS.
- The QRS axis is perpendicular (i.e., right angle or 90 degrees) to that lead's orientation (see above diagram).
- Since there are two possible perpendiculars to each isoelectric lead, chose the one that best fits the direction of the QRS in other ECG leads.

<table>
<thead>
<tr>
<th>Isoelectric Lead</th>
<th>More likely axis</th>
<th>less likely axis</th>
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</thead>
<tbody>
<tr>
<td>I</td>
<td>+90</td>
<td>-90</td>
</tr>
<tr>
<td>II</td>
<td>-30</td>
<td>+150</td>
</tr>
<tr>
<td>III</td>
<td>+30</td>
<td>-150</td>
</tr>
<tr>
<td>aVR</td>
<td>-60</td>
<td>+120</td>
</tr>
<tr>
<td>aVL</td>
<td>+60</td>
<td>-120</td>
</tr>
<tr>
<td>aVF</td>
<td>0</td>
<td>+/-180</td>
</tr>
</tbody>
</table>

- If there is no isoelectric lead, there are usually two leads that are nearly isoelectric, and these are always 30° apart. Find the perpendiculars for each lead and chose an approximate QRS axis within the 30° range.
- Occasionally each of the 6 frontal plane leads is small and/or isoelectric. The axis cannot be determined and is called indeterminate. This is often a normal variant.
Examples of QRS Axis Determination:

- **Axis in the normal range:**

1. Lead aVF is the isoelectric lead (equal forces positive and negative).
2. The two perpendiculcals to aVF are 0° and 180°.
3. Lead I is positive (i.e., oriented to the left).
4. Therefore, of the two choices the axis has to be 0°.
- **Left Axis deviation (LAD):**

  1. Lead aVR is the smallest and isoelectric lead.
  2. The two perpendiculars to aVR are -60° and +120°.
  3. Leads II and III are mostly negative
     (i.e., moving away from the + left leg)
  4. The axis, therefore, is -60° (LAD).
Right Axis Deviation (RAD):

1. Lead aVR is closest to being isoelectric (slightly more positive than negative)
2. The two perpendiculars are -60° and +120°.
3. Lead I is mostly negative; lead III is mostly positive.
4. Therefore the axis is close to +120°. Because aVR is slightly more positive, the axis is slightly beyond +120° (i.e., closer to the positive right arm for aVR).
3. CHARACTERISTICS OF THE NORMAL ECG

It is important to remember that there is a wide range of normal variation in the 12 lead ECG. The following "normal" ECG characteristics, therefore, are not absolute. It takes considerable ECG reading experience to discover all the normal variants. Only by following a structured “Method of ECG Interpretation” and correlating the various ECG findings with the particular patient’s clinical status will the ECG become a valuable clinical tool.

I. Normal MEASUREMENTS

- **Heart Rate:** 50 - 90 bpm
- **PR Interval:** 0.12 - 0.20s
- **QRS Duration:** 0.06 - 0.10s
- **QT Interval:** $\text{QT} \leq 0.44s$  
  
  **Poor Man's Guide** to the upper limit of QT: @ 70 bpm, QT ≤ 0.40s; for every 10 bpm increase above 70 bpm subtract 0.02s, and for every 10 bpm decrease below 70 bpm add 0.02s. For example:
  - QT ≤ 0.38 @ 80 bpm
  - QT ≤ 0.42 @ 60 bpm
- **Frontal Plane QRS Axis:** +90° to -30° (in the adult)

II. Normal RHYTHM: Normal sinus rhythm

III. Normal CONDUCTION: Normal Sino-Atrial (SA), Atrio-Ventricular (AV), and Intraventricular (IV) conduction

IV. Normal WAVEFORM DESCRIPTION:

**P Wave:** It is important to remember that the P wave represents the sequential activation of the right and left atria, and it is common to see notched or biphasic P waves of right and left atrial activation.
- P duration < 0.12s
- P amplitude < 2.5 mm
- Frontal plane P wave axis: 0° to +75° (must be up in I and II)
- May see notched P waves in frontal plane, and biphasic (+/-) in V1

**QRS Complex:** The normal QRS represents the simultaneous activation of the right and left ventricles, although most of the QRS waveform is derived from the larger left ventricular musculature.
- QRS duration ≤ 0.10s
- QRS amplitude is quite variable from lead to lead and from person to person. Two determinants of QRS voltages are:
  - Size of the ventricular chambers (i.e., the larger the chamber, the larger the voltage)
  - Proximity of chest electrodes to ventricular chamber (the closer, the larger the voltage)
- Frontal plane leads:
  - The normal QRS axis range (+90° to -30°) implies that the QRS direction must always be positive (up going) in leads I and II.
  - Small "septal" q-waves are often seen in leads I and aVL when the QRS axis is to the left of +60°, and in leads II, III, aVF when the QRS axis is to the right of +60°.
- Precordial leads:
  - Small r-waves begin in V1 or V2 and increase in size to V5. The R-V6 is usually smaller than R-V5.
  - In reverse, the s-waves begin in V6 or V5 and increase in size to V2. S-V1 is usually smaller than S-V2.
The usual transition from S>R in the right precordial leads to R>S in the left precordial leads is V3 or V4.
Small "septal" q-waves may be seen in leads V5 and V6.

**ST Segment:** In a sense, the term "ST segment" is a misnomer, because a discrete ST segment distinct from the T wave is often not seen. More frequently the ST-T wave is a smooth, continuous waveform beginning with the J-point (end of QRS), slowly rising to the peak of the T and followed by a rapid descent to the isoelectric baseline or the onset of the U wave. This gives rise to asymmetrical T waves in most leads. The ST segment occurs during Phase 2 (the plateau) of the myocardial action potentials. In some normal individuals, particularly women, the T wave is more symmetrical and a distinct horizontal ST segment is present.

The ST segment is often elevated above baseline in leads with large S waves (e.g., V2-3), and the normal configuration is concave upward. ST segment elevation with concave upward appearance may also be seen in other leads; this is often called early repolarization, although it's a term with little physiologic meaning (see example of "early repolarization" in leads V4-6 in the ECG below). J-point elevation is often accompanied by a small J-wave in the lateral precordial leads. The physiologic basis for the J-wave is related to transient outward K+ movement during phase I of the epicardial and mid-myocardial cells, not present in the subendocardial cells. Prominent J waves are often seen in hypothermia (also called Osborn waves).
4. ABNORMALITIES IN THE ECG MEASUREMENTS

1. PR Interval (measured from beginning of P to beginning of QRS in the frontal plane)
   - Normal: 0.12 - 0.20s
   - Differential Diagnosis of Short PR: < 0.12s
     - Preexcitation syndromes:
       - WPW (Wolff-Parkinson-White) Syndrome: An accessory pathway (called the "Kent" bundle) connects the right atrium to the right ventricle (see diagram below) or the left atrium to the left ventricle, and this permits early activation of the ventricles (*delta* wave) and a short PR interval.

![Diagram of the heart showing the accessory pathway](image)

- LGL (Lown-Ganong-Levine) Syndrome: An AV nodal bypass track into the His bundle exists, and this permits early activation of the ventricles without a *delta* wave because the ventricular activation sequence is normal.

- AV Junctional Rhythms with retrograde atrial activation (*inverted P waves in II, III, aVF*): Retrograde P waves may occur *before* the QRS complex (usually with a short PR interval), *in* the QRS complex (i.e., hidden from view), or *after* the QRS complex (i.e., in the ST segment). It all depends upon the relative timing from the junctional focus antegrade into the ventricles and retrograde back to the atria.

- Ectopic atrial rhythms originating near the AV node (the PR interval is short because atrial activation originates close to the AV node; the P wave morphology is different from the sinus P and may appear inverted in some leads)

- Normal variant (PR 0.10 - 0.12s)
Differential Diagnosis of Prolonged PR: >0.20s

First degree AV block (PR interval usually constant from beat to beat); possible locations for the conduction delay include:
- Intra-atrial conduction delay (uncommon)
- Slowed conduction in AV node (most common site of prolonged PR)
- Slowed conduction in His bundle (rare)
- Slowed conduction in a bundle branch (when contralateral bundle is totally blocked; i.e., 1st degree bundle branch block)

Second degree AV block (PR interval may be normal or prolonged; some P waves do not conduct to ventricles and are not followed by a QRS)
- Type I (Wenckebach): Increasing PR until nonconducted P wave occurs
- Type II (Mobitz): Fixed PR intervals plus nonconducted P waves
- AV dissociation: Some PR's may appear prolonged, but the P waves and QRS complexes are dissociated (i.e., not married, but strangers passing in the night).

2. QRS Duration (duration of QRS complex in frontal plane):
- Normal: 0.06 - 0.10s
- Differential Diagnosis of Prolonged QRS Duration (>0.10s):
  - QRS duration 0.10 - 0.12s
    - Incomplete right or left bundle branch block
    - Nonspecific intraventricular conduction delay (IVCD)
    - Some cases of left anterior or posterior fascicular block
  - QRS duration ≥ 0.12s
    - Complete RBBB or LBBB
    - Nonspecific IVCD
    - Ectopic rhythms originating in the ventricles (e.g., ventricular tachycardia, accelerated ventricular rhythm, pacemaker rhythm)

3. QT Interval (measured from beginning of QRS to end of T wave in the frontal plane; corrected QT = QTc = measured QT - sq-root RR in seconds; Bazet's formula)
- Normal QT is heart rate dependent (upper limit for QTc = 0.44 sec)
- Long QT Syndrome - LQTS (based on upper limits for heart rate; QTc ≥ 0.47 sec for males and ≥0.48 sec in females is diagnostic for hereditary LQTS in absence of other causes of long QT):
  - This abnormality may have important clinical implications since it usually indicates a state of increased vulnerability to malignant ventricular arrhythmias, syncope, and sudden death. The prototype arrhythmia of the Long QT Interval Syndromes (LQTS) is Torsades-de-pointes, a polymorphic ventricular tachycardia characterized by varying QRS morphology and amplitude around the isoelectric baseline. Causes of LQTS include the following:
    - Drugs (many antiarrhythmics, tricyclics, phenothiazines, and others)
    - Electrolyte abnormalities (↓ K+, ↓ Ca++, ↓ Mg++)
    - CNS disease (especially subarachnoid hemorrhage, stroke, trauma)
    - Hereditary LQTS (at least 7 genotypes are now known)
    - Coronary Heart Disease (some post-MI patients)
    - Cardiomyopathy
• **Short QT Syndrome (QT<sub>c</sub> < 0.32 sec):** Newly described hereditary disorder with increased risk of sudden arrhythmic death. The QT criteria are subject to change.

4. **Frontal Plane QRS Axis**

• **Normal:** -30 degrees to +90 degrees

• **Abnormalities in the QRS Axis:**

  • **Left Axis Deviation (LAD):** > -30° (i.e., lead II is mostly ‘negative’)
    - Left Anterior Fascicular Block (LAFB): rS complex (i.e., small r, big S) in leads II, III, aVF, small q in leads I and/or aVL, and -45 to -90° (see ECG on p 8)
    - Some cases of inferior MI with Qr complex in lead II (making lead II ‘negative’)
    - Inferior MI + LAFB in same patient (QS or qRS complex in II)
    - Some cases of LVH
    - Some cases of LBBB
    - Ostium primum ASD and other endocardial cushion defects
    - Some cases of WPW syndrome (large negative delta wave in lead II)

  • **Right Axis Deviation (RAD):** > +90° (i.e., lead I is mostly ‘negative’)
    - Left Posterior Fascicular Block (LPFB): rS complex in lead I, qR in leads II, III, aVF (however, must first exclude, on clinical basis, causes of right heart overload; these will also give same ECG picture of LPFB)
    - Many causes of right heart overload and pulmonary hypertension
    - High lateral wall MI with Qr or QS complex in leads I and aVL
    - Some cases of RBBB
    - Some cases of WPW syndrome
    - Children, teenagers, and some young adults

  • **Bizarre QRS axis:** +150° to -90° (i.e., lead I and lead II are both negative)
    - Consider limb lead error (usually right and left arm reversal)
    - Dextrocardia
    - Some cases of complex congenital heart disease (e.g., transposition)
    - Some cases of ventricular tachycardia
5. ECG RHYTHM ABNORMALITIES

INTRODUCTION TO ECG RHYTHM ANALYSIS:

THINGS TO CONSIDER WHEN ANALYZING ARRHYTHMIAS:

Arrhythmias may be seen on 12-lead ECGs or on rhythm strips of one or more leads. Some arrhythmias are obvious at first glance and don't require intense analysis. Others, however, are more challenging (and fun)! They require detective work, i.e., logical thinking. Rhythm analysis should begin with identifying characteristics of impulse formation (if known) as well as impulse conduction. Here are some things to think about:

- Descriptors of impulse formation (i.e., the pacemaker or region of impulse formation)

  - Site of origin - i.e., where is the abnormal rhythm coming from?
    - Sinus Node (e.g., sinus tachycardia; P waves may be hidden in the T waves)
    - Atria (e.g., PAC)
    - AV junction (e.g., junctional escape rhythm)
    - Ventriles (e.g., PVC)
  - Rate (i.e., relative to the expected rate for that pacemaker location)
    - Accelerated - faster than expected for that pacemaker site (e.g., accelerated junctional rhythm)
    - Slower than expected (e.g., marked sinus bradycardia)
    - Normal (or expected) (e.g., junctional escape rhythm)
  - Regularity of ventricular or atrial response
    - Regular (e.g., paroxysmal supraventricular tachycardia - PSVT)
    - Regular irregularity (e.g., ventricular bigeminy)
    - Irregular irregularity (e.g., atrial fibrillation or MAT)
    - Irregular (e.g., multifocal PVCs)
  - Onset (i.e., how does arrhythmia begin?)
    - Active onset (e.g., PAC or PVC)
    - Passive onset (e.g., ventricular escape beat or rhythm)

- Descriptors of impulse conduction (i.e., how does the abnormal rhythm move through the heart chambers?)

  - Antegrade (forward) vs. retrograde (backward) conduction
  - Conduction delays or blocks: i.e., 1st, 2nd (type I or II), 3rd degree blocks
  - Sites of potential conduction delay
    - Sino-Atrial (SA) block (only 2nd degree SA block on ECG is recognized as an unexpected failure of a sinus P-wave to appear resulting in an unexpected pause)
    - Intra-atrial delay (usually not recognized)
    - AV conduction delays (common)
    - IV blocks (e.g., bundle branch or fascicular blocks)
Now let's continue with some real rhythms…………..

I. Supraventricular Arrhythmias

- **Premature Atrial Complexes (PAC's)**
  - Occur as single or repetitive events and have unifocal or multifocal origins.
  - The ectopic P wave (often called P') is often hidden in the ST-T wave of the preceding beat. (Dr. Marriott, master ECG teacher and author, likes to say: "Cherchez le P sur le T" which in French means: "Search for the P on the T wave", and it's clearly sexier to search in French!)
  - The P'R interval is normal or prolonged if the AV junction is partially refractory at the time the premature impulse enters it.
  - PAC's can have **three different outcomes** depending on the degree of prematurity (i.e., coupling interval from previous P wave), and the preceding cycle length (or RR interval). This is illustrated in the "ladder" diagram where normal sinus beats are followed by three possible PACs (a,b,c):

![Diagram showing the three fates of PACS](image)

Outcome #1. Nonconducted (or blocked) PAC; i.e., no QRS complex because the PAC finds the AV node still refractory to conduction. (see PAC 'a' in the ladder diagram labeled 1, and a nonconducted PACs in ECG shown below – after 3\textsuperscript{rd} QRS)
Outcome #2. Conducted with aberration; i.e., PAC makes it into the ventricles but finds a bundle branch or fascicle asleep. The resulting QRS is usually wide, and is sometimes called an **Ashman beat** (see PAC 'b' in the ladder diagram labeled 1, and ECG below showing a PAC with RBBB aberration)

![ECG showing PAC with RBBB aberration](image)

Outcome #3. Normal conduction; i.e., similar to other QRS complexes in that ECG lead. (See PAC 'c' in the ladder diagram labeled 1)

- In the ladder diagram (p19), labeled '2', the cycle length has increased (slower heart rate). This results in increased refractoriness of all the structures in the conduction system. PAC 'b' now can't get through the AV node and is nonconducted; PAC 'c' is now blocked in the right bundle branch and results in a RBBB QRS complex (aberrant conduction); PAC 'd' occurs later and conducts normally. **Therefore, the fate of a PAC depends on 1) the coupling interval from the last P wave, and 2) the preceding cycle length or heart rate.**

- The pause after a PAC is usually **incomplete**; i.e., the PAC usually enters the sinus node and resets its timing, causing the next sinus P to appear earlier than expected. (PVCs, on the other hand, are usually followed by a **complete** pause because the PVC does not usually perturb the sinus node; see ECG below and the diagram on page 26.)

![Lead V1 diagram with PAC and sinus P](image)

**Complete vs Incomplete Pause**
• **Premature Junctional Complexes (PJC's)**
  - Similar to PAC's in clinical implications, but less frequent.
  - The PJC focus in the AV junction captures the atria (retrograde) and the ventricles (antegrade). The retrograde P wave may appear before, during, or after the QRS complex; if before, the PR interval is usually short (i.e., <0.12 s). The ECG tracing and ladder diagram shown below illustrates a classic PJC with retrograde P waves following the QRS.

![ECG tracing and ladder diagram](image)

- **Atrial Fibrillation (A-fib)**

<table>
<thead>
<tr>
<th>Heart Rate</th>
<th>Rhythm</th>
<th>P Wave</th>
<th>PR interval (in seconds)</th>
<th>QRS (in seconds)</th>
</tr>
</thead>
<tbody>
<tr>
<td>A: 350-650 bpm</td>
<td>Irregular</td>
<td>Fibrillatory (fine to course)</td>
<td>N/A</td>
<td>&lt;.12</td>
</tr>
<tr>
<td>V: Slow to rapid</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

- Atrial activity is poorly defined; may see course or fine baseline undulations (wiggles) or no atrial activity at all. If atrial activity is seen, it resembles the teeth on an **old saw** (when compared to atrial flutter that often resembles a clean **saw-tooth pattern** especially in leads II, III, aVF).
- Ventricular response (RR intervals) is **irregularly irregular** and may be **fast** (HR >100 bpm, indicates inadequate rate control), **moderate** (HR = 60-100 bpm), or **slow** (HR <60 bpm, indicates excessive rate control medication, AV node disease, or drug toxicity).
• A regular ventricular response with A-fib usually indicates complete or 3rd degree AV block with an escape or accelerated ectopic pacemaker originating in the AV junction or ventricles (i.e., consider digoxin toxicity or AV node disease). In the ECG shown below the last 2 QRS complexes are junctional escapes indicating high-grade AV block due (note constant RR intervals).

![ECG Image]

• The differential diagnosis includes atrial flutter with an irregular ventricular response and multifocal atrial tachycardia (MAT), which is usually irregularly irregular. The differential diagnosis is often hard to make from a single rhythm strip; the 12-lead ECG is best for differentiating these three arrhythmias.

**Atrial Flutter (A-flutter)**

<table>
<thead>
<tr>
<th>Heart Rate</th>
<th>Rhythm</th>
<th>P Wave</th>
<th>PR interval (in seconds)</th>
<th>QRS (in seconds)</th>
</tr>
</thead>
<tbody>
<tr>
<td>A: 220-430 bpm</td>
<td>Regular or variable</td>
<td>Sawtoothed appearance</td>
<td>N/A</td>
<td>&lt;.12</td>
</tr>
<tr>
<td>V: &lt;300 bpm</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

• Regular atrial activity with a clean saw-tooth appearance in leads II, III, aVF, and usually discrete 'P' waves in lead V1. The atrial rate is usually about 300/min, but may be as slow as 150-200/min or as fast as 400-450/min.

• Untreated A-flutter often presents with a 2:1 A-V conduction ratio. This is the most commonly missed arrhythmia diagnosis because the flutter waves are often difficult to find. Therefore, always think "atrial flutter with 2:1 block" whenever there is a regular supraventricular tachycardia @ approximately 150 bpm! (You aren’t likely to miss it if you look for it.)
- The ventricular response may be 2:1, 3:1 (rare), 4:1, or irregular depending upon AV conduction properties. A-flutter with 2:1 block is illustrated below:

  ![ECG Image](image)

  - **Ectopic Atrial Tachycardia and Rhythm**
    - Ectopic, discrete looking, unifocal P' waves with atrial rate <250/min (not to be confused with slow atrial flutter).
    - Ectopic P' waves usually precede QRS complexes with P'R interval < RP' interval (i.e., not to be confused with paroxysmal supraventricular tachycardia with retrograde P waves shortly after the QRS complexes).

    ![ECG Image](image)

    - The above ECG shows sinus rhythm, a PVC, and the onset of ectopic atrial tachycardia (note different P wave morphology)
    - Ventricular response may be 1:1 (as above ECG) or with varying degrees of AV block (especially in the setting of digitalis toxicity).
    - Ectopic atrial rhythm is similar to ectopic atrial tachycardia, but with HR < 100 bpm

  - **Multifocal Atrial Tachycardia (MAT) and rhythm**
    - Discrete, multifocal P' waves occurring at rates of 100-250/min and with varying P'R intervals (should see at least 3 different P wave morphologies in a given lead).
    - Ventricular response is irregularly irregular (i.e., often confused with A-fib).
    - May be intermittent, alternating with periods of normal sinus rhythm.
    - Seen most often in elderly patients with chronic or acute medical problems such as exacerbation of chronic obstructive pulmonary disease.
    - If atrial rate is <100 bpm, call it *multifocal atrial rhythm*.

  - **Paroxysmal Supraventricular Tachycardia (PSVT)**
    - **Basic Considerations:** These arrhythmias are *circus movement* tachycardias that utilize the mechanism of *reentry*, they are also called *reciprocating* tachycardias. The onset is sudden, usually initiated by a premature beat, and the arrhythmia also stops abruptly - which is why they are called *paroxysmal*. They are usually narrow-QRS tachycardias unless there is preexisting bundle branch block (BBB) or aberrant ventricular conduction (i.e., rate related BBB). There are several types of PSVT depending on the location of the reentry circuit.
AV Nodal Reentrant Tachycardia (AVNRT): This is the most common form of PSVT accounting for approximately 50% of all symptomatic PSVTs. The diagram below illustrates the probable mechanism involving dual AV nodal pathways, alpha and beta, with different electrical properties. In the diagram alpha is a fast pathway but with a long refractory period (RP), and beta is the slow pathway with a short RP. During sinus rhythm alpha is always used because it is faster and there is plenty of time between sinus beats for recovery to occur. An early PAC, however, finds alpha still refractory and must use the slower beta pathway to reach the ventricles. By the time it traverses beta, however, alpha has recovered allowing retrograde conduction back to the atria. The retrograde P wave (called an atrial echo) is often simultaneous with the QRS and not seen on the ECG, but it can reenter the AV junction because of beta's short RP.

In the above ECG 2 sinus beats are followed by PAC (in the ST segment) and onset of PSVT. Retrograde P waves immediately follow each QRS (little dip at onset of ST segment)

If an early PAC is properly timed, AVNRT results as seen in the diagram below. Rarely, an atypical form of AVNRT occurs with the retrograde P wave appearing in front of the next QRS (i.e., RP' interval > 1/2 the RR interval), implying antegrade conduction down the faster alpha, and retrograde conduction up the slower beta pathway.
AV Reciprocating Tachycardia (Extranodal bypass pathway): This is the second most common form of PSVT and is seen in patients with the WPW syndrome. The WPW ECG, seen in the diagram on p. 14, shows a short PR, delta wave, and somewhat widened QRS.

This type of PSVT can also occur in the absence of the WPW ECG if the accessory pathway only allows conduction in the retrograde direction (i.e., concealed WPW). Like AVNRT, the onset of PSVT is usually initiated by a PAC that finds the bypass track temporarily refractory, conducts down the AV junction to the ventricles, and reenters the atria through the bypass track. In this type of PSVT retrograde P waves appear shortly after the QRS in the ST segment (i.e., RP'< 1/2 RR interval). Rarely the antegrade limb for PSVT uses the bypass track and the retrograde limb uses the AV junction; the PSVT then resembles a wide QRS tachycardia and must be differentiated from ventricular tachycardia.

Sino-Atrial Reentrant Tachycardia: This is a rare form of PSVT where the reentrant circuit is between the sinus node and the right atria. The ECG looks like sinus tachycardia, but the tachycardia is paroxysmal; i.e., it starts and ends abruptly.

Junctional Rhythms and Tachycardias

Junctional Escape Beats: These are passive, protective beats originating from subsidiary pacemaker cells in the AV junction. The pacemaker's basic firing rate is 40-60 bpm; junctional escapes are programmed to occur whenever the primary pacemaker (i.e., sinus node) defaults or the AV node blocks the atrial impulse from reaching the ventricles. The ECG strip below shows sinus arrhythmia with two junctional escapes (arrows). Incomplete AV dissociation is also seen during the junctional escapes.

Junctional Escape Rhythm: This is a sequence of 3 or more junctional escapes occurring by default at a rate of 40-60 bpm. There may be AV dissociation or the atria may be captured retrogradely by the junctional pacemaker.

Accelerated Junctional Rhythm: This is an active junctional pacemaker rhythm caused by events that perturb pacemaker cells (e.g., ischemia, drugs, and electrolyte abnormalities). The rate is 60-100 bpm.

Nonparoxysmal Junctional Tachycardia: This usually begins as an accelerated junctional rhythm but the heart rate gradually increases to >100 bpm. There may be AV dissociation, or retrograde atrial capture may occur. Ischemia (usually from right coronary artery occlusion) and digitalis intoxication are the two most common causes.
II. Ventricular Arrhythmias

- **Premature Ventricular Complexes (PVCs)**

PVCs may be unifocal (see above), multifocal (see below) or multiformed. Multifocal PVCs have different sites of origin, which means their coupling intervals (from previous QRS complexes) are usually different. Multiformed PVCs usually have the same coupling intervals (because they originate in the same ectopic site but their conduction through the ventricles differs. Multiformed PVCs are common in digitalis intoxication.

![Multifocal PVC's: more than one shape](image)

PVCs may occur as isolated single events or as couplets, triplets, and salvos (4-6 PVCs in a row) also called brief ventricular tachycardias.

![Triplet PVC's: occur in groups of three](image)

PVCs may occur early in the cycle (R-on-T phenomenon), after the T wave (as seen above), or late in the cycle - often fusing with the next QRS (fusion beat). R-on-T PVCs may be especially dangerous in an acute ischemic situation, because the ventricles are more vulnerable to ventricular tachycardia or fibrillation.
In the example below, late (end-diastolic) PVCs are illustrated with varying degrees of fusion. For fusion to occur the sinus P wave must have made it to the ventricles to start the activation sequence. Before ventricular activation is completed, however, the "late" PVC occurs, and the resultant QRS looks a bit like the normal QRS, and a bit like the PVC; i.e., a fusion QRS (arrows).

The events following a PVC are of interest. Usually a PVC is followed by a complete compensatory pause, because the sinus node timing is not interrupted; one sinus P wave near the PVC isn't able to reach the ventricles because they are still refractory from the PVC; the following P wave occurs on time based on the sinus rate. In contrast, PACs are usually followed by an incomplete pause because the PAC can reset the sinus node timing; this enables the next sinus P wave to appear earlier than expected. These concepts are illustrated below.

Not all PVCs are followed by a pause. If a PVC occurs early enough (especially if the heart rate is slow), it may appear sandwiched in between two normal beats. This is called an interpolated PVC. The sinus P wave following the PVC usually has a longer PR interval because of retrograde concealed conduction by the PVC into the AV junction, slowing subsequent conduction of the sinus impulse.
Finally a PVC may retrogradely capture the atrium, reset the sinus node, and be followed by an incomplete pause. Often the retrograde P wave can be seen on the ECG, hiding in the ST-T wave of the PVC.

A most unusual post-PVC event occurs when retrograde activation of the AV junction (or atria) re-enters (or comes back to) the ventricles as a ventricular echo. This is illustrated below. The "ladder" diagram under the ECG helps us understand the mechanism. The P wave following the PVC is the sinus P wave, but the PR interval is too short for it to have caused the next QRS. (Remember, the PR interval following an interpolated PVC is usually longer than normal, not shorter!). Isn't that cool?

PVCs usually stick out like "sore thumbs" or funny-looking-beats (FLB's), because they are bizarre in appearance compared to the normal complexes. However, not all premature sore thumbs are PVCs. In the example below 2 PACs are seen, #1 with a normal QRS, and #2 with RBBB aberrancy - which looks like a sore thumb. The challenge, therefore, is to recognize sore thumbs for what they are, and that's the next topic for discussion!
**Aberrant Ventricular Conduction**

**INTRODUCTION**

Aberrant ventricular conduction (AVC) is a very common source of confusion in interpreting 12-lead ECGs and rhythm strips. A thorough understanding of its mechanism and recognition is essential to all persons who read ECGs.

Before we can understand aberrant ventricular conduction we must first review how normal conduction of the electrical impulse occurs in the heart. **What a magnificent design!** Impulses from the fastest center of automaticity (SA node) are transmitted through the atria and over specialized fibers (Bachmann’s bundle to the left atrium and three internodal tracts) to the AV node. The AV node provides sufficient conduction delay to allow atrial contraction to contribute to ventricular filling. Following slow AV node conduction high velocity conduction tracts deliver the electrical impulse to the right and left ventricles (through the His bundle, bundle branches and fascicles, and Purkinje network). Simultaneous activation of the two ventricles results in a **NARROW NORMAL QRS COMPLEX** (0.06-0.1 sec QRS duration). Should a conduction delay in one or the other of the bundle branches occur then an **ABNORMAL WIDE QRS COMPLEX** will result. (A delay in a fascicle of the left bundle branch will result in an abnormal QRS that is not necessarily wide but of a different morphology (i.e., a change in frontal plane QRS axis) from the person’s normal QRS morphology).

![Bachmann’s Bundle Diagram](image)

**Figure 1**

Figure 2 below illustrates a basic principle of AVC. AVC is a *temporary alteration of QRS morphology when you would have expected a normal QRS complex*. Permanent bundle branch block (BBB) is **NOT AVC**.

![ECG Lead V1](image)

**Figure 2**
In this discussion let us concentrate on AVC through normal bundle branch and fascicular pathways and not consider conduction through accessory pathways (e.g., as in WPW syndrome). The ECG illustrated in Figure 2 from lead V1 shows two normal sinus beats followed by a premature atrial complex (PAC, first arrow). The QRS complex of the PAC is narrow. Following the usual incomplete pause, another sinus beat is followed by a slightly earlier PAC. Now, because of this slightly increased prematurity (and longer preceding RR cycle), the QRS complex is abnormal (rsR' morphology of RBBB). If you were not careful you might mistake this wide premature beat as a PVC and attach a different clinical significance (and therapy). The key features to recognizing AVC in this tracing are:

1. Identifying the premature P-wave (P')
2. Recognizing the typical RBBB QRS morphology (rsR' in lead V1)

**ABERRANT VENTRICULAR CONDUCTION**

A term that describes temporary alteration of QRS morphology under conditions where a normal QRS might be expected. The common types are:

1. Through normal conduction pathways:
   - Cycle-length dependent (Ashman phenomenon)
   - Rate-dependent tachycardia or bradycardia
2. Through accessory pathways (e.g., Kent bundle)

As seen below five features or clues help identify AVC of the right bundle branch block variety. It should be emphasized that although RBBB morphology is the commonest form of AVC, LBBB or interruption of one of its fascicles may also occur, particularly in persons with more advanced left heart disease or those taking cardiovascular drugs. In healthy people the right bundle branch has a slightly longer refractory period than the left bundle at normal heart rates and, therefore, is more likely to be “sleeping” when an early PAC enters the ventricles. The “second-in-a-row” phenomenon will be illustrated later in this section.

**FEATURES FAVORING RBBB ABERRANT CONDUCTION**

1. Preceding atrial activity (premature P wave)
2. rSR' or rsR' morphology in lead V1
3. qRs morphology in lead V6
4. Same initial r wave as normal QRS complex (in lead V1)
5. “Second-in-a-row” phenomenon

The Ashman Phenomenon is named after the late Dr. Richard Ashman who described, in 1947, AVC of the RBBB variety in patients with atrial fibrillation. Ashman reasoned from observing ECG rhythms in these patients that the refractory period (during which conducting tissue is recovering and cannot be stimulated) was directly proportional to cycle length. The longer the cycle length (or slower the heart rate) the longer the refractory period is. In Figure 3 a premature stimulus (PS) can be conducted if the preceding cycle length is of short or medium duration but will be blocked if the preceding cycle length is long. Ashman observed this in atrial fibrillation when long RR cycles were followed by short RR cycles and the QRS terminating the short RR cycle was wide in duration.
Look at the ECG rhythm strips in Figure 3. Simultaneous Lead II and Lead V1 are recorded. The first PAC (arrow in V1) conducts to the ventricles with a normal QRS because the preceding cycle was of normal or medium length. The second PAC (next arrow) conducts with RBBB (rsR’ in V1) because the preceding cycle was LONGER. Both PACs have identical coupling intervals. Thus, a long cycle-short cycle sequence often leads to AVC. Unfortunately this sequence helps us UNDERSTAND AVC but is not DIAGNOSTIC OF AVC. PVCs are often observed in a long cycle-short cycle sequence. It is important, therefore, to have other clues to the presence of AVC such as a preceding ectopic P wave.

**Figure 3**

Years ago Dr. Henry Marriott, a master teacher of electrocardiography and author of many outstanding ECG textbooks offered valuable guidelines regarding QRS morphology (especially in lead V1). These morphologies contrasted with the QRS complexes often seen with PVCs and enhanced our ability to diagnose AVC. For example, if the QRS in lead V1 is predominately up-going or positive (Figure 4) the differential diagnosis is between RBBB aberrancy and ventricular ectopy from the left ventricle. A careful look at each of the 5 QRS complexes in Figure 4 will identify “Las Vegas” type betting odds of making the right diagnosis.

**Figure 4**

1. 2. 3. 4. 5.
QRS #1 and #2 are “classic” RBBB morphologies with rsR’ or rSR’ triphasic QRS shapes. When either of these is seen as premature beats in lead V1 we can be at least 90% certain that they are aberrant RBBB conduction and not ventricular ectopy. Examples #3 and #4 are notched or slurred R wave QRS complexes. Where’s the notch or slur? Think of rabbit ears. If the notch or slur is on the downstroke of the QRS (little right rabbit ear in Example #4), then the odds are almost 100-to-1 that the beat is a ventricular ectopic beat (or PVC). If, on the other hand, the notch or slur is on the upstroke of the QRS (little rabbit ear on the left in Example #3), than the odds are 50:50 and not helpful in the differential Dx. Finally if the QRS complex has just a qR configuration (Example #5) than the odds are reasonably high that the beat in question is a ventricular ectopic beat and not AVC. Two exceptions to this last rule (Example #5) need to be remembered. Some people with normal ECG’s do not have an initial little r-wave in the QRS of lead V1. If RBBB occurs in such a person the QRS morphology in V1 will be a qR instead of an rsR’. Secondly, in a person with a previous anterior or anteroseptal infarction the V1 QRS often has a QS morphology, and RBBB in such a person will also have a qR pattern.

Now consider mostly down-going or negative QRS morphologies in lead V1 (Figure 5).

Here the differential diagnosis is between LBBB aberration (Example #1) and right ventricular ectopy (Example #2). Typical LBBB in lead V1 may or may not have a “thin” initial r-wave, but will always have a rapid S-wave downstroke as seen in #1. On the other hand any one of three features illustrated in #2 is great betting odds that the beat in question is ventricular ectopy and not AVC. These three features are: 1) fat little initial r-wave, 2) notch or slur in the downstroke of the S wave, and 3) a 0.06 sec or more delay from the beginning of the QRS to the nadir of the S-wave.

![Figure 5](image-url)
Now, let’s look at some real ECG examples of the preceding QRS morphology rules. We will focus on the V1 lead for now since it is the best lead for differentiating RBBB from LBBB and right from left ventricular ectopy.

**Figure 6**

Figure 6 (above) illustrates two premature funny-looking beats (FLBs) for your consideration. FLB ‘A’ has a small notch on the upstroke of the QRS complex resembling Example #3 in Figure 4. Remember, that’s only a 50:50 odds for AVC and therefore not helpful in the differentiating it from a PVC. However, if you look carefully at the preceding T wave, you will see that it is more pointed than the other T wave in this V1 rhythm strip. There is very likely a hidden premature P-wave in the T before ‘A’, making the FLB a PAC with RBBB aberrancy. Dr. Marriott likes to say: "Cherchez le P" which is a sexy way to say in French "Search for the P" before the FLB to determine if the FLB is a PAC with AVC. FLB ‘B’, on the other hand, has a small notch or slur on the downstroke of the QRS resembling QRS #4 in Figure 4. That’s almost certainly a PVC.

Alas, into each life some rain must fall! Remember life is not always 100% perfect. In Figure 7 after 2 sinus beats a bigeminal rhythm develops. The 3 premature FLBs have 'TYPICAL RBBB MORPHOLOGY (rSR') and yet they are PVCs! How can we tell? They are not preceded by premature P-waves, but are actually followed (look in the ST segment) by the next normal sinus P-wave which cannot conduct because the ventricles are refractory at that time. The next P wave comes on time (complete pause). Well, you can’t win them all.

**Figure 7**
The ECG in Figure 8 was read as "Ventricular bigeminy" in our ECG lab by a tired physician reading late at night. Try to see if you can do better. The first thing to notice is that all the early premature FLBs have RBBB morphology...already a 10:1 odds favoring AVC. Note also that the T waves of the sinus beats look "funny" – particularly in Leads 1, 2, and V2. They are small, short, and peak too early, a very suspicious signal that they are indeed hidden premature P-waves (Cherchez-le-P).

The clincher, however, is that the premature beats are followed by **INCOMPLETE COMPENSATORY PAUSES**. How can you tell? One lead (aVF) has no premature FLBs, so you can measure the exact sinus rate. Taking 2 sinus cycles from this lead (with your calipers), you can now tell in the other leads that the P wave following the FLBs comes earlier than expected suggesting that the sinus cycle was reset by the premature P waves (a common feature of PACs, but not PVCs). The correct diagnosis, therefore, is atrial bigeminy with RBBB aberration of the PACs.

![Figure 8](image-url)
The diagram illustrated in Figure 9 helps us understand the difference between a “complete” compensatory pause (characteristic of most PVCs) and an “incomplete” pause (typical of most PACs). The top half of Figure 9 shows (in “ladder” diagram form) three sinus beats and a PAC. The sinus P wave after the PAC comes earlier than expected because the PAC entered the sinus node and reset its timing. In the bottom half of Figure 9 three sinus beats are followed by a PVC. As you can see the sinus cycle is not interrupted, but one sinus beat cannot conduct to the ventricles because the ventricles are refractory due to the PVC. The next P wave comes on time making the pause a complete compensatory pause.

![Diagram of compensatory pauses]

**Figure 9**
The top ECG strip in Figure 10 illustrates 2 PACs conducted with AVC. Note how the premature ectopic P-wave peaks and distorts the preceding T-wave (Cherchez-le-P). The first PAC conducts with LBBB aberrancy and the second with RBBB. In the second strip atrial fibrillation is initiated by a PAC with RBBB aberration (note the long preceding RR interval followed by a short coupling interval to the PAC). The aberrantly conducted beat that initiates atrial fibrillation is an example of the "second-in-a-row" phenomenon which is frequently seen in atrial tachyarrhythmias with AVC. It’s the second beat in a row of fast beats that is most often conducted with AVC because of the long-short rule (Ashman phenomenon).

In Figure 11 you can see Ashman beats at their finest. RBBB beats in lead V1 follow the long cycle-short cycle sequence. Since the atria are fibrillating, you can’t identify “preceding atrial activity” so you have to presume that all beats are conducted. Note that the 2nd Ashman beat in the top strip is followed by a quicker but narrow QRS beat – the right bundle is now responding to a short cycle-short cycle sequence and behaves normally. Dr. Ashman noticed this in 1947!
If you’re ready for some fun, consider the next example illustrated in Figure 12. This unfortunate man suffered from palpitation and dizziness when he swallowed. What you see is an ectopic atrial tachycardia with intermittent RBBB aberrant conduction. The arrows point to ectopic P-waves going at nearly 200 bpm. Note how the PR interval gradually gets longer until the 4th P-wave in the tachycardia fails to conduct (Wenckebach phenomenon). This initiates a pause, and when 1:1 conduction resumes the second and subsequent QRS complexes exhibit upright QRS complexes in the form of atypical RBBB. This has to be a truly cool ECG rhythm strip!

Figure 12

Bundle branch block aberration can occur during a “critical rate” change which means that AVC comes with gradual changes in heart rate and not necessarily with abrupt changes in cycle length as in the Ashman phenomenon. Think of a “tired” but not “dead” bundle branch. This is illustrated in Figure 13, an example of rate-dependent or acceleration-dependent AVC. When the sinus cycle, in this instance 71 bpm, is shorter than the refractory period of the left bundle then LBBB ensues. It is almost always the case that as the heart rate slows it takes a slower rate for the LBBB to disappear, as seen in the lower strip.

Figure 13
Figure 14 shows another example of acceleration-dependent RBBB, this time in the setting of atrial fibrillation. Even the “normal” beats have a minor degree of incomplete RBBB (rsr’). At critically short cycles, however, complete RBBB ensues and remains until the rate slows again. You can tell that these are not PVCs and runs of ventricular tachycardia because of the typical RBBB morphology (rsR’ in lead V1) and the irregular RR cycles of atrial fib.

**Figure 14**

Things can really get scary in the coronary care unit in the setting of acute myocardial infarction. Consider the case illustrated in Figure 15 (lead V1) with intermittent runs of what looks like ventricular tachycardia. Note that the basic rhythm is irregularly irregular indicating atrial fibrillation. The wide QRS complexes are examples of tachycardia-dependent LBBB aberration and not runs of ventricular tachycardia. Note the morphology of the wide beats. Although there is no initial “thin” r-wave, the downstroke of the S wave is very rapid (see Example 1 in Figure 5).

**Figure 15**

Finally we have an example in Figure 16 of the rarest and most perplexing form of AVC --- deceleration or bradycardia-dependent aberration. Note that the QRS duration is normal at rates above 65 bpm, but all longer RR cycles are terminated by beats with LBBB. What a paradox! You have to be careful not to classify the late beats ventricular escapes, but in this case the QRS morphology of the late beats is classic for LBBB (see Example 1 in Figure 5) as evidenced by the “thin” r-wave and rapid downstroke of the S-wave. This type of AVC is sometimes called “Phase 4” AVC because it’s during Phase 4 of the action potential that latent pacemakers (in this case located in the left bundle) begin to depolarize. Sinus beats entering the partially depolarized left bundle conduct more slowly and sometimes are nonconducted (resulting in LBBB).

**Figure 16**

The rhythm in Figure 16 may be difficult to determine because sinus P-waves are hard to see in lead V1. P-waves were more easily seen in other leads from this patient. The rhythm was sinus arrhythmia with intermittent 2nd degree AV block.
The ECG strips in Figure 17 summarize the important points made in this lesson. In strip '1' intermittent RBBB is seen with atrial fibrillation. The first two RBBB beats result from an accelerating rate (tachycardia-dependent RBBB) while the later triplet of RBBB beats are a consequence of the Ashman phenomenon (long cycle-short cycle sequence). Strip '2' from the same patient when in sinus rhythm shows two premature FLB’s. The first FLB has a QR configuration similar to Example 5 in Figure 4 and is most certainly a PVC as the pause following it is a complete compensatory one. The 2nd FLB has the classic triphasic rsR’ morphology of RBBB AVC (see Example 1 in Figure 4). The pause following this beat is incomplete which is expected for PACs.

![ECG strips](image)

**Figure 17**

**SUMMARY**

The differential diagnosis of FLBs is intellectually challenging and has important clinical implications. This section provides clues that help distinguish wide QRS complexes that are supraventricular in origin with AVC from ectopic beats of ventricular origin (PVCs and ventricular tachycardia). When looking at single premature FLBs always search for hidden premature P-waves in the ST-T wave of the preceding beat (Cherchez-le-P). Measure with calipers the pause after the FLB to determine if it’s compensatory or not. Remember the lead V1 morphology clues offered in Figures 4 and 5 that provide the betting odds that a particular beat in question is supraventricular or ventricular in origin. These morphology clues may be the only way to correctly diagnose wide QRS-complex tachycardias.

Don’t be fooled by first impressions. **Not all FLBs are ventricular in origin!**

The next section focuses on ECG aspects of ventricular tachycardia and the differential diagnosis of wide QRS tachycardias. Other ventricular rhythms are also discussed.
**Ventricular Tachycardia**

- Descriptors to consider when considering ventricular tachycardia:
  - Sustained (lasting >30 s) vs. nonsustained
  - Monomorphic (uniform morphology) vs. polymorphic vs. Torsades-de-pointes
    (Torsades-de-pointes: a polymorphic ventricular tachycardia associated with the long-QT syndromes characterized by phasic variations in the polarity of the QRS complexes around the baseline. Ventricular rate is often >200bpm and ventricular fibrillation is a consequence.)
  - Presence of AV dissociation (independent atrial activity) vs. retrograde atrial capture
  - Presence of fusion QRS complexes (also called Dressler beats) which occur when supraventricular beats (usually sinus) get into the ventricles during the ectopic activation sequence.

- **Differential Diagnosis:** just as for single premature funny-looking beats, **not all wide QRS tachycardias are ventricular in origin (they may be supraventricular tachycardias with bundle branch block or WPW preexcitation)!**

**Differential Diagnosis of Wide QRS Tachycardias**

Although this is an ECG tutorial, let's not forget some simple bedside clues to ventricular tachycardia:

- Advanced heart disease (e.g., coronary heart disease) favors ventricular tachycardia
- Cannon 'a' waves in the jugular venous pulse suggests ventricular tachycardia with AV dissociation. Under these circumstances ventricular contractions may occur when the tricuspid valve is still open which leads to the giant pulsations seen in the JV pulse. With AV dissociation these giant waves occur irregularly.
- Variable intensity of the S1 heart sound at the apex (mitral closure); again this is seen when there is AV dissociation resulting in varying position of the mitral valve leaflets depending on the timing of atrial and ventricular systole.
- If the patient is hemodynamically unstable, think ventricular tachycardia and act accordingly!

**ECG Clues:**

- **Regularity of the rhythm:** If the wide QRS tachycardia is sustained and monomorphic, then the rhythm is usually regular (i.e., RR intervals equal); an irregularly-irregular rhythm suggests atrial fibrillation with aberration or WPW preexcititation.
- **A-V Dissociation** strongly suggests ventricular tachycardia! Unfortunately AV dissociation only occurs in approximately 50% of ventricular tachycardias (the other 50% have retrograde atrial capture or "V-A association"). Of the patients with AV dissociation, it is only easily recognized if the rate of tachycardia is <150 bpm. Faster heart rates make it difficult to visualize dissociated P waves.
- **Fusion beats or captures** often occur when there is AV dissociation and this also strongly suggests a ventricular origin for the wide QRS tachycardia.
- QRS morphology in lead V1 as described on p. 31 for single premature funny looking beats is often the **best clue** to the origin, so go back and check out the clues!
Also consider a few additional morphology clues:

- Bizarre frontal-plane QRS axis (i.e. from +150 degrees to -90 degrees or NW quadrant) suggests ventricular tachycardia
- QRS morphology similar to previously seen PVCs suggests ventricular tachycardia
- If all the QRS complexes from V1 to V6 are in the same direction (positive or negative), ventricular tachycardia is likely
- Mostly or all negative QRS in V6 suggests ventricular tachycardia
- Especially wide QRS complexes (>0.16s) suggests ventricular tachycardia
- Also consider the following Four-step Algorithm reported by Brugada et al, Circulation 1991;83:1649:
  
  - **Step 1:** Absence of RS complex in all leads V1-V6? Yes: Dx is ventricular tachycardia!
  
  - **Step 2:** No: Is interval from beginning of R wave to nadir of S wave >0.1s in any RS lead? Yes: Dx is ventricular tachycardia!
  
  - **Step 3:** No: Are AV dissociation, fusions, or captures seen? Yes: Dx is ventricular tachycardia!
  
  - **Step 4:** No: Are there morphology criteria for VT present both in leads V1 and V6? Yes: Dx is ventricular tachycardia!
  
  - **NO:** Diagnosis is supraventricular tachycardia with aberration!

The ECG shown below illustrates several features of typical VT: 1) QRS morphology in lead V1 looks like QRS #4 on page 31; 2) QRS is mostly negative in lead V6; 3) bizarre frontal plane QRS axis of -180 degrees. This is most likely from the left ventricle (note QRS direction is rightward and anterior).
The next ECG illustrated below shows another typical VT, but this time coming from the right ventricle. Note the V1 QRS morphology has all the features of left ventricular VT origin including 1) fat, little R wave; 2) notch on the downstroke or the S-wave; and 3) >0.06 s delay from QRS onset to the nadir (bottom) of the S-wave.

---

**Accelerated Ventricular Rhythms (see ECG below)**

- An "active" ventricular rhythm due to enhanced automaticity of a ventricular pacemaker (reperfusion after thrombolytic therapy is a common causal factor).
- Ventricular rate 60-110 bpm (anything faster would be ventricular tachycardia)
- Sometimes called *isochronic ventricular rhythm* when the ventricular rate is close to underlying sinus rate
- May begin and end with fusion beats (ventricular activation partly due to the normal sinus activation of the ventricles and partly from the ectopic focus)
- Usually benign, short lasting, and not requiring of therapy.

**Lead MCL₁**

[Image of ECG showing isochronic ventricular rhythm with fusion beats and arrows pointing to dissociated P waves]
• **Idioventricular Rhythm**
  
  • A "passive" escape rhythm that occurs by default whenever higher-lever pacemakers in AV junction or sinus node fail to control ventricular activation.
  
  • Escape rate is usually 30-50 bpm.
  
  • Seen most often in complete AV block with AV dissociation or in other bradycardic conditions.

• **Ventricular Parasystole**
  
  • Non-fixed coupled PVCs where the inter-ectopic intervals (i.e., timing between PVCs) are some multiple (i.e., 1x, 2x, 3x, ...etc) of the basic rate of the parasystolic focus
  
  • PVCs have uniform morphology unless fusion beats occur
  
  • Usually entrance block is present around the ectopic focus, which means that the primary rhythm (e.g., sinus rhythm) is unable to enter the ectopic site and reset its timing.
  
  • May also see exit block; i.e., the output from the ectopic site may occasionally be blocked (i.e., no PVC when one is expected).
  
  • Fusion beats are common when ectopic site fires while ventricles are already being activated from primary pacemaker
  
  • Parasystolic rhythms may also originate in the atria (i.e., with non-fixed coupled PAC’s) and within the AV junction

![Lead V1 with Parasystolic PVCs and Fusion Beats](image-url)
**Pacemaker Rhythms**

Pacemakers come in a wide variety of programmable features. The following ECG rhythm strips illustrate the common types of pacing functions.

- **Atrial pacing:** note small pacemaker spikes before every P wave followed by normal QRS complexes.

  ![Atrial Pacing](image)

- **A-V sequential pacing with ventricular pacing** (note tiny spike before each QRS) and atrial sensing of normal sinus rhythm (note: pacemaker spikes are sometimes difficult to see):

  ![A-V Sequential Pacing](image)

- **A-V sequential pacing with both atrial and ventricular pacing** (note pacing spikes before each P wave and each QRS)

  ![A-V Dual-Chamber Pacing](image)

- **Normal functioning ventricular demand pacemaker.** Small pacing spikes are seen before QRS #1, #3, #4, and #6 representing the paced beats. There is marked sinus bradycardia (that’s the reason for the pacemaker), but when P waves are able to conduct they do (see QRS #2 and #5). This is a nice example of incomplete AV dissociation due to sinus slowing where the artificial pacemaker takes over by default.

  ![Ventricular Demand Pacing](image)
6. ECG CONDUCTION ABNORMALITIES

**INTRODUCTION:** This section considers all disorders of impulse conduction that may occur within the cardiac conduction system (see diagram page 28). Heart block can occur anywhere in the specialized conduction system beginning with the sino-atrial connections, the AV junction, the bundle branches and fascicles, and ending in the distal ventricular Purkinje fibers. Disorders of conduction may manifest as slowed conduction (1st degree), intermittent conduction failure (2nd degree), or complete conduction failure (3rd degree). In addition, there are two varieties of 2nd degree heart block: Type I (or Wenckebach) and Type II (Mobitz II). In Type I block there is *decremental conduction* that means that conduction velocity progressively slows beat by beat until failure of conduction occurs. This is the form of conduction block in the AV node. Type II block is *all or none* and is more likely found in the His bundle or below the His bifurcation (i.e., in the bundle branches). The term *exit block* is used to identify conduction delay or failure immediately distal to a pacemaker site. Sino-atrial (SA) block, for example, is an exit block. This section considers conduction disorders in the anatomical sequence that defines the cardiac conduction system; *so lets begin.........*

I. **SINO-ATRIAL EXIT BLOCK (SA Block):**

- **2nd Degree SA Block:** this is the only degree of SA block that can be recognized on the surface ECG (i.e., intermittent conduction failure between the sinus node and the right atrium). There are two types, although because of sinus arrhythmia they may be difficult to differentiate.
  - Type I (SA Wenckebach): the following 3 rules represent the classic rules of Wenckebach which were originally used to describe Type I AV block. The rules are the result of decremental conduction where the *increment* in conduction delay for each subsequent impulse gets smaller until conduction failure finally occurs.
    1. PP intervals gradually shorten until a pause occurs (i.e., the blocked sinus impulse fails to reach the atria)
    2. The pause duration is *less than* the two preceding PP intervals
    3. The PP interval following the pause is *greater than* the PP interval just before the pause

  **Differential Diagnosis:** sinus arrhythmia without SA block. The following rhythm strip illustrates SA Wenckebach with a ladder diagram to show the progressive conduction delay between SA node and the atria. Note the similarity of this rhythm to marked sinus arrhythmia.

---

**Lead II**

![ECG Sino-Atrial Exit Block (type I)](image-url)
Type II SA Block:
- PP intervals fairly constant (unless sinus arrhythmia present) until conduction failure occurs.
- The pause is approximately \textit{twice} the basic PP interval

\begin{figure}[h]
\centering
\includegraphics[width=\textwidth]{sino-atrial退出block.png}
\caption{Sino-Atrial Exit Block (Type II)}
\end{figure}

\section*{II. ATRIO-VENTRICULAR (AV Block):

Possible sites of AV block:
- AV node (most common)
- His bundle (uncommon)
- Bundle branch and fascicular divisions (in presence of already existing bundle branch block)

- \textbf{1st Degree AV Block}: PR interval > 0.20 sec; all P waves conduct to the ventricles.

\begin{figure}[h]
\centering
\includegraphics[width=\textwidth]{first_degree_av_block.png}
\caption{1st degree AV block (PR = 280 ms)}
\end{figure}

- \textbf{2nd Degree AV Block}: The ladder diagram below illustrates the difference between Type I (Wenckebach) and Type II AV block.

\begin{figure}[h]
\centering
\includegraphics[width=\textwidth]{type_i_and_type_ii_av_blocks.png}
\caption{Diagram comparing Type I and Type II AV blocks}
\end{figure}
In "classic" Type I (Wenckebach) AV block the PR interval gets longer (by shorter increments) until a nonconducted P wave occurs. The RR interval of the pause is less than the two preceding RR intervals, and the RR interval after the pause is greater than the RR interval before the pause. These are the 3 classic rules of Wenckebach (described above for SA block). In Type II (Mobitz) AV block the PR intervals are constant (for at least 2 consecutive PR intervals) until a nonconducted P wave occurs. The RR interval of the pause is equal to the two preceding RR intervals.

2nd Degree Type I (Wenckebach) AV block (note the RR intervals in ms duration):

![Lead V1](image1)

"Classic Wenckebach"

| 680 | 640 | 1180 | 1680 |

NOTE: Type I AV block is almost always in the AV node itself, which means that the QRS duration is usually narrow, unless there is preexisting bundle branch disease.

2nd Degree Type II (Mobitz) AV block (note PR is constant for two consecutive PR's)

![Lead V1](image2)

2nd degree AV block (type II) with LBBB

Type II AV block is almost always a bundle branch problem, which means that the QRS duration is wide indicating complete block of one bundle, and the nonconducted P waves are blocked in the other bundle. In Type II block several consecutive P waves may be blocked as illustrated below:

![Lead V1](image3)
Complete (3rd Degree) AV Block:
- Usually see complete AV dissociation because the atria and ventricles are under control of separate pacemaker bosses.
- Narrow QRS rhythm suggests a junctional escape focus for the ventricles with block above the focus, usually in AV node.
- Wide QRS rhythm suggests a ventricular escape focus (i.e., idioventricular rhythm). This is seen in ECG 'A' below; ECG 'B' shows the treatment for 3rd degree AV block; i.e., a ventricular pacemaker. The location of the block may be in the AV junction or bilaterally in the bundle branches.

AV Dissociation (independent rhythms in atria and ventricles):
- Not synonymous with 3rd degree AV block, although AV block is one of the causes.
- May be complete or incomplete. In complete AV dissociation the atria and ventricles are always independent of each other as in complete AV block. In incomplete AV dissociation there is either intermittent retrograde atrial capture from the ventricular focus or antegrade ventricular capture from the atrial focus.
- There are three categories of AV dissociation (categories 1 & 2 are always incomplete AV dissociation):
  Slowing of the primary pacemaker (i.e., SA node); subsidiary escape pacemaker takes over by default:
1. Acceleration of a subsidiary pacemaker faster than sinus rhythm; i.e., takeover by *usurpation*:

![Lead V1](image1)

**Incomplete AV dissociation (usurpation)**

due to accelerated ventricular rhythm

F = fusion beat

2. 2\textsuperscript{nd} or 3\textsuperscript{rd} degree AV block with escape rhythm from junctional focus or ventricular focus:
   - In the example (below) of complete AV dissociation (3\textsuperscript{rd} degree AV block with a junctional escape pacemaker) the PP intervals are alternating because of *ventriculophasic sinus arrhythmia* (phasic variation of vagal tone on the sinus rate depending on the timing of ventricular contractions).

![Lead II](image2)

1. What is the diagnosis?
2. Why are the PP intervals alternating?
III. **INTRAVENTRICULAR BLOCKS**

- **Right Bundle Branch Block (RBBB):**
  - "Complete" RBBB has a QRS duration ≥0.12s (120 ms)
  - Close examination of QRS complex in various leads reveals that the terminal forces (i.e., 2nd half of QRS) are oriented rightward and anteriorly because the right ventricle is depolarized after the left ventricle.
    - Terminal R' wave in lead V1 (usually see rSR' complex) indicating late anterior forces
    - Terminal S waves in leads I, aVL, V6 indicating late rightward forces
    - Terminal R wave in lead aVR indicating late rightward forces
  - The frontal plane QRS axis in RBBB should be in the normal range (i.e., -30 to +90 degrees). If left axis deviation is present, must also consider left anterior fascicular block, and if right axis deviation is present, must consider left posterior fascicular block in addition to the RBBB.
  - "Incomplete" RBBB has a QRS duration of 0.10 - 0.12s with the same terminal QRS features. This is often a normal variant, but could be seen in RVH.
  - The "normal" ST-T waves in RBBB should be oriented opposite to the direction of the terminal QRS forces or last half of the QRS; i.e., in leads with terminal R or R' forces (e.g., V1) the ST-T should be downwards (negative); in leads with terminal S forces (e.g., I, V6) the ST-T should be positive. If the ST-T waves are in the same direction as the terminal QRS forces, they should be labeled primary ST-T wave abnormalities because they may be related to other conditions affecting ST-T wave morphology (e.g., ischemia, drug effects, electrolyte abnormalities)

- **Left Bundle Branch Block (LBBB)**
  - "Complete" LBBB has a QRS duration ≥0.12s
  - Close examination of QRS complex (see ECG below) in various leads reveals that the terminal forces (i.e., 2nd half of QRS) are oriented leftward and posteriorly because the left ventricle is depolarized after the right ventricle.
    - Terminal S waves in lead V1 indicating late posterior forces
    - Terminal R waves in lead I, aVL, V6 indicating late leftward forces; usually broad, monophasic R waves are seen in these leads as illustrated in the ECG below; in addition, poor R progression from V1 to V3 is common.
The "normal" ST-T waves in LBBB should be oriented opposite to the direction of the terminal QRS forces; i.e., in leads with terminal R or R' forces the ST-T should be downwards (negative); in leads with terminal S forces the ST-T should be upwards (positive). If the ST-T waves are in the same direction as the terminal QRS forces, they should be labeled primary ST-T wave abnormalities. In the above ECG the ST-T waves are "normal" for LBBB; i.e., they are secondary to the change in the ventricular depolarization sequence.

"Incomplete" LBBB looks like LBBB but QRS duration = 0.10 - 0.12s, with less ST-T change. This is often a progression of LVH.

**Left Anterior Fascicular Block (LAFB)...... the most common intraventricular conduction defect**
- Left axis deviation in frontal plane, usually -45 to -90 degrees
- rS complexes in leads II, III, aVF (i.e., small initial r, large S)
- Small q-wave in leads I and/or aVL
- S in III > S in II; R in aVL > R in aVR
- R-peak time in lead aVL >0.04s, often with slurred R wave downstroke
- QRS duration usually <0.12s unless coexisting RBBB
- Usually see poor R progression in leads V1-V3 and deeper S waves in leads V5 and V6
- May mimic LVH voltage in lead aVL, and mask LVH voltage in leads V5, V6

In the above ECG, note -45 degree QRS axis, rS complexes in II, III, aVF, tiny q-wave in I, aVL, S in III > S in II, R in aVL > R in aVR, and late S waves in leads V5-6. QRS duration is normal, and there is a slight slur to the R wave downstroke in lead aVL.
• **Left Posterior Fascicular Block (LPFB).** Very rare intraventricular defect!
  
  - Right axis deviation in the frontal plane (usually > +100 degrees)
  - rS complex in lead I
  - qR complexes in leads II, III, aVF, with R in lead III > R in lead II
  - QRS duration usually <0.12s unless coexisting RBBB
  - **Must first exclude (on clinical or other grounds) other causes of right axis deviation such as cor pulmonale, pulmonary heart disease, pulmonary hypertension, etc., because these conditions can result in the identical ECG picture!**

• **Bifascicular Blocks**
  
  - RBBB plus either LAFB (common) or LPFB (uncommon)
  - Features of RBBB plus frontal plane features of the fascicular block (axis deviation, etc.)
  - The above ECG shows classic RBBB (note rSR' in V1) plus LAFB (QRS axis = - 60°, rS in II, aVF; and small q in I and aVL).

![ECG Example](image)

• **Nonspecific Intraventricular Conduction Defects (IVCD)**
  
  - QRS duration >0.10s indicating slowed conduction in the ventricles
  - Criteria for specific bundle branch or fascicular blocks not met
  - Causes of nonspecific IVCD's include:
    - Ventricular hypertrophy (especially LVH)
    - Myocardial infarction (so called **periinfarction blocks**)
    - Drugs, especially class IA and IC antiarrhythmics (e.g., quinidine, flecainide)
    - Hyperkalemia
- **Wolff-Parkinson-White Preexcitation**

- Although not a true IVCD, this condition causes widening of QRS complex and, therefore, deserves to be considered here.
- QRS complex represents a fusion between two ventricular activation fronts:
  - Early ventricular activation in region of the accessory AV pathway (Bundle of Kent). This is illustrated in the diagram on p 14.
  - Ventricular activation through the normal AV junction, bundle branch system.
- ECG criteria include all of the following:
  - Short PR interval (<0.12s)
  - Initial slurring of QRS complex (delta wave) representing early ventricular activation into ventricular muscle in the region of the accessory pathway.
    - Delta waves, if negative in polarity (see lead III and V1 below), may mimic infarct Q waves and result in false positive diagnosis of myocardial infarction.
  - Prolonged QRS duration (usually >0.10s)
  - Secondary ST-T changes due to the altered ventricular activation sequence.
  - QRS morphology, including polarity of delta wave depends on the particular location of the accessory pathway as well as on the relative proportion of the QRS complex that is due to early ventricular activation (i.e., degree of fusion).
7. ATRIAL ENLARGEMENT

- **Right Atrial Enlargement (RAE, P-pulmonale, "Viagra P-waves")**
  - P wave amplitude $>2.5$ mm in II and/or $>1.5$ mm in V1 (these criteria are not very specific or sensitive)
  - Better criteria can be derived from the QRS complex; these QRS changes are due to both the high incidence of RVH when RAE is present, and the RV displacement by an enlarged right atrium.
  - QR, Qr, qR, or qRs morphology in lead V1 (in absence of coronary heart disease)
  - QRS voltage in V1 is $<5$ mm and V2/V1 voltage ratio is $>6$ (Sensitivity = 50%; Specificity = 90%)

- **Left Atrial Enlargement (LAE. P-mitrale)**
  - P wave duration $\geq 0.12$s in frontal plane (usually lead II)
  - Notched P wave in limb leads with interpeak duration $\geq 0.04$s
  - Terminal P negativity in lead V1 (i.e., "P-terminal force") duration $\geq 0.04$s, depth $\geq 1$ mm.
  - Sensitivity = 50%; Specificity = 90%

- **Bi-Atrial Enlargement (BAE)**
  - Features of both RAE and LAE in same ECG
  - P wave in lead II $>2.5$ mm tall and $\geq 0.12$s in duration
  - Initial positive component of P wave in V1 $>1.5$ mm tall and prominent P-terminal force
8. VENTRICULAR HYPERTROPHY

Introductory Information:
- The ECG criteria for diagnosing right or left ventricular hypertrophy are very insensitive (i.e., sensitivity ~50%, which means that ~50% of patients with ventricular hypertrophy cannot be recognized by ECG criteria). When in doubt...Get an ECHO! However, the criteria are very specific (i.e., specificity >90%, which means if the criteria are met, it is very likely that ventricular hypertrophy is present).

I. Left Ventricular Hypertrophy (LVH)
- General ECG features include:
  - QRS amplitude (voltage criteria; i.e., tall R-waves in LV leads, deep S-waves in RV leads)
  - Delayed Intrinsicoid deflection in V6 (i.e., time from QRS onset to peak R is ≥0.05 sec)
  - Widened QRS/T angle (i.e., left ventricular strain pattern, or ST-T oriented opposite to QRS direction). This pattern is more common with LVH due to pressure overload (e.g., aortic stenosis, systemic hypertension) rather than volume overload.
  - Leftward shift in frontal plane QRS axis
  - Evidence for left atrial enlargement (LAE)

ESTES Criteria for LVH ("diagnostic", ≥5 points; "probable", 4 points)

<table>
<thead>
<tr>
<th>+ECG Criteria</th>
<th>Points</th>
</tr>
</thead>
<tbody>
<tr>
<td>Voltage Criteria (any of):</td>
<td></td>
</tr>
<tr>
<td>a. R or S in limb leads ≥ 20 mm</td>
<td>3 points</td>
</tr>
<tr>
<td>b. S in V1 or V2 ≥ 30 mm</td>
<td></td>
</tr>
<tr>
<td>c. R in V5 or V6 ≥ 30 mm</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>ST-T Abnormalities:</th>
<th></th>
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</thead>
<tbody>
<tr>
<td>Without digitalis</td>
<td>3 points</td>
</tr>
<tr>
<td>With digitalis</td>
<td>1 point</td>
</tr>
</tbody>
</table>

| Left Atrial Enlargement in V1 | 3 points |
| Left axis deviation | 2 points |
| QRS duration 0.09 sec | 1 point |
| Delayed intrinsicoid deflection in V5 or V6 (≥0.05 sec) | 1 point |

- CORNELL Voltage Criteria for LVH (sensitivity = 22%, specificity = 95%)
  - S in V3 + R in aVL > 24 mm (men)
  - S in V3 + R in aVL > 20 mm (women)

- Other Voltage Criteria for LVH
  - Limb-lead voltage criteria:
    - R in aVL ≥11 mm or, if left axis deviation, R in aVL ≥13 mm plus S in III ≥15 mm
    - R in I + S in III >25 mm
  - Chest-lead voltage criteria:
    - S in V1 + R in V5 or V6 ≥ 35 mm
**Example 1**: (Limb-lead Voltage Criteria; e.g., R in aVL >11 mm; note wide QRS/T angle)

![ECG Example 1](image1)

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**Example 2**: (ESTES Criteria: 3 points for voltage in V5, 3 points for ST-T changes; also LAE and LAD of -40 degrees; note also the PVC)

![ECG Example 2](image2)

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II. **Right Ventricular Hypertrophy (RVH)**

- **General ECG features include:**
  - Right axis deviation (>90 degrees)
  - Tall R-waves in RV leads; deep S-waves in LV leads
  - Slight increase in QRS duration
  - ST-T changes directed opposite to QRS direction (i.e., wide QRS/T angle)
  - May see incomplete RBBB pattern or qR pattern in V1
  - Evidence of right atrial enlargement (RAE)

- **Specific ECG features (assumes normal calibration of 1 mV = 10 mm):**
  - Any one or more of the following (if QRS duration <0.12 sec):
    - Right axis deviation (>90 degrees) in presence of disease capable of causing RVH
    - R in aVR > 5 mm, or
    - R in aVR > Q in aVR
  - Any one of the following in lead V1:
    - R/S ratio > 1 and negative T wave
    - qR pattern
    - R > 6 mm, or S < 2mm, or rSR' with R' >10 mm
  - Other chest lead criteria:
    - R in V1 + S in V5 (or V6) 10 mm
    - R/S ratio in V5 or V6 < 1
    - R in V5 or V6 < 5 mm
    - S in V5 or V6 > 7 mm
  - ST segment depression and T wave inversion in right precordial leads is usually seen in severe RVH such as in pulmonary stenosis and pulmonary hypertension.

**Example #1:** (note RAD +30 degrees; RAE; R in V1 > 6 mm; R in aVR > 5 mm)
Example #2: (more subtle RVH: note RAD +100 degrees; RAE; Qr complex in V1 rather than qR is atypical)

Example #3: (note: RAD +130 degrees, qRs in V1; R/S ratio in V6 <1)

III. Biventricular Hypertrophy (difficult ECG diagnosis to make)
- In the presence of LAE any one of the following suggests this diagnosis:
  - R/S ratio in V5 or V6 < 1
  - S in V5 or V6 > 6 mm
  - RAD (>90 degrees)
- Other suggestive ECG findings:
  - Criteria for LVH and RVH both met
  - LVH criteria met and RAD or RAE present
9. MYOCARDIAL INFARCTION

Introduction to ECG Recognition of Acute Coronary Syndrome (ACS)

- The ECG changes of ACS are the result of a sudden reduction of coronary blood flow to a region of ventricular myocardium supplied by a coronary artery with a ruptured atherosclerotic plaque and intracoronary thrombus formation. Depending on how quickly the patient gets to the hospital for definitive treatment (usually percutaneous revascularization or thrombolytic Rx) myocardial necrosis (infarction) may or may not occur. The diagram below shows four possible ECG outcomes of myocardial ischemia in the setting of an acute coronary syndrome. On the left no myocardial infarction occurs but there is either subendocardial ischemia manifested by reversible ST segment depression or transmural ischemia manifested by reversible ST segment elevation. On the right are two kinds of myocardial infarction, one manifested by ST segment elevation (STEMI) and one manifested by no ST segment elevation (NSTEMI). Previously these two MI types were called Q-wave MI and non-Q-wave MI respectively. Because Q waves may not appear initially, early treatment decisions are based on the presence or absence of ST segment elevation, and if revascularization is accomplished quickly Q-waves may never appear (“time is muscle” says the interventional cardiologist).

No-MI
Subendocardial Ischemia
Transient ST ↓↓
New onset angina

Non-Q MI
Non-ST elevation MI
ST depression or T-wave changes or normal ECG

No-MI
Transmural Ischemia
Transient ST ↑↑
Variant Angina

Q-wave MI
ST elevation MI (STEMI)
Typical evolution of ST-T changes
The following discussion will focus on ECG changes during the evolution of a STEMI

- Most MI's are located in the **left ventricle**. In the setting of a proximal right coronary artery occlusion, however, there may also be a component of **right ventricular** infarction as well. Right sided chest leads are usually needed to recognize RV MI.

- In general, the more leads of the 12-lead ECG with MI changes (Q waves and ST elevation), the larger the infarct size and the worse the prognosis.

- The left anterior descending coronary artery (LAD) and its branches usually supply the anterior and anterolateral walls of the left ventricle and the anterior two-thirds of the septum. The left circumflex coronary artery (LCx) and its branches usually supply the posterolateral wall of the left ventricle. The right coronary artery (RCA) supplies the right ventricle, the inferior (diaphragmatic) and true posterior walls of the left ventricle, and the posterior third of the septum. The RCA also gives off the AV nodal coronary artery in 85-90% of individuals; in the remaining 10-15%, this artery is a branch of the LCX.

- The usual ECG evolution of a STEMI with Q-waves is illustrated in the diagram below. Not all of the patterns may be seen; the time from onset of MI to the final pattern is quite variable and related to the size of MI, the rapidity of reperfusion (if any), and the location of the MI.

  A. Normal ECG prior to MI
  B. Hyperacute T wave changes - increased T wave amplitude and width; QT prolongs; may also see ST elevation
  C. Marked ST elevation with hyperacute T wave changes (transmural injury)
  D. Pathologic Q waves, ST elevation decreases, terminal T wave inversion (necrosis); this is also called the "fully evolved" phase.
  E. Pathologic Q waves, T wave inversion (necrosis and fibrosis)
  F. Pathologic Q waves, upright T waves (fibrosis)

Evolution of Acute MI
I. Inferior MI Family of STEMI's (Q-wave MI's); includes inferior, true posterior, and right ventricular MI's

- Inferior MI
  - Pathologic Q waves and evolving ST-T changes in leads II, III, aVF
  - Q waves usually largest in lead III, next largest in lead aVF, and smallest in lead II. Q wave ≥30ms in aVF is diagnostic.

Example #1: Acute inferior MI injury pattern. Note hyperacute T waves with ST elevation in II, III, aVF; reciprocal ST depression in I, and aVL. ST depression in V1-3 represents true posterior injury pattern and not a reciprocal change (see true posterior MI patterns below). The V4 and V5 electrodes are interchanged (technical error).

Example #2: Old inferior MI (note largest Q in lead III, next largest in aVF, and smallest in lead II). Axis = -50° (LAD)
• **True posterior MI**
  - ECG changes are seen in anterior precordial leads V1-3, but are the **mirror image** of an anteroseptal MI (because the posterior wall is behind the anterior wall):
    - Increased R wave amplitude and/or duration ≥40 ms in V1-2 (i.e., a "pathologic R wave" is the mirror image of a pathologic Q on the posterior wall)
    - R/S ratio in V1 or V2 >1 (i.e., prominent anterior forces)
    - Hyperacute ST-T wave changes: i.e., ST depression and large, inverted T waves in V1-3
    - Late normalization of ST-T with symmetrical upright T waves in V1 to V3
  - Often seen with inferior MI (i.e., "infero-posterior MI")

Example #3: Acute infero-posterior MI (note tall R waves V1-3, marked ST depression V1-3, ST elevation in II, III, aVF)

Example #4: Old inferoposterior MI: Note tall pathologic R in V1-3 (Q wave equivalent), upright T waves and inferior Q waves)
Right Ventricular MI (only seen with proximal right coronary occlusion; i.e., with inferior family MI's)
- ECG findings usually require additional leads on right chest (V1R to V6R, analogous to the left chest lead locations, but in opposite direction)
- Criteria: ST elevation, ≥1mm, in right chest leads, especially V4R (see below)

Example #5: Acute inferior MI with right-sided ECG leads showing marked ST segment elevation in V3R, V4R, V5R, V6R.

II. Anterior Family of STEMI's; includes anteroseptal, anterior, anterolateral, and high lateral
- Anteroseptal MI
  - Q, QS, or qrS complexes in leads V1-V3 (V4)
  - Evolving ST-T changes

Example #6: Hyperacute anteroseptal MI; marked ST elevation in V1-3 before Q waves developed
Example #7: Fully evolved anteroseptal MI (note QS waves in V1-2, qrS complex in V3, plus ST-T wave changes)

Anterior MI (similar changes, but usually V1 is spared; if V4-6 involved call it "anterolateral"; if changes also in leads I and aVL it’s a "high-lateral" MI.

Example 8: Acute Anterolateral injury; note ST elevation V3-6. Possible inferior MI also present of uncertain age.
Example #9: Anterolateral MI with high lateral changes as well. Note Q's V2-6 plus Q's in leads I and aVL. Axis = +120° (RAD)

**Comment:** The precise naming of MI locations on the ECG is evolving as new heart imaging (e.g., MRI) better defines the ventricular anatomy. New terminology has been suggested (see *Circulation 2006;114:1755*). While not universally accepted, the following “new” Q-wave MI patterns have been defined for left ventricular segments:

- **Septal MI:** Q (or QS) waves in V1-2
- **Mid-Anterior MI:** Q waves in aVL, sometimes in lead I, V2, V3, but not in V5-6.
- **Apical-Anterior MI:** Q waves in V3, V4, and sometimes in V5-6. No Q waves in I, aVL
- **Extensive Anterior MI:** Combination of above 3 locations.
- **Lateral MI:** Prominent R waves in V1-2 (this replaces the true posterior MI location; MRI imaging of the left ventricle shows no posterior wall). Q waves may also be present in I, aVL, V5-6.
- **Inferior MI:** Q waves in II, III, aVF, but without prominent R waves in V1-2

**III. MI with Bundle Branch Block**

- MI + Right Bundle Branch Block
  - Usually easy to recognize because Q waves and ST-T changes are not altered by the RBBB

**Example #10:** Inferior MI + RBBB (note Q's in II, III, aVF and rSR' in lead V1)
Example #11: Extensive anterior MI with RBBB + LAFB; note Q's in leads V1-V5, terminal fat R wave in V1-4, fat S wave in V6). Axis = -80° (rS in II, III, aVF: left anterior fascicular block or LAFB.)

- MI + Left Bundle Branch Block
  - Often a difficult ECG diagnosis because in LBBB the right ventricle is activated first and left ventricular infarct Q waves may not appear at the beginning of the QRS complex (unless the septum is involved).
  - Suggested ECG features, not all of which are specific for MI include:
    - Q waves of any size in two or more of leads I, aVL, V5, or V6 (See ECG #13 below: one of the most reliable signs and probably indicates septal infarction, because the septum is activated early from the right ventricular side in LBBB)
    - Reversal of the usual R wave progression in precordial leads (see above)
- Notching of the downstroke of the S wave in precordial leads to the right of the transition zone (i.e., before QRS changes from a predominate S wave complex to a predominate R wave complex); this may be a Q-wave equivalent.
- Notching of the upstroke of the S wave in precordial leads to the right of the transition zone (another Q-wave equivalent).
- rSR' complex in leads I, V5 or V6 (the S is a Q-wave equivalent occurring in the middle of the QRS complex)
- RS complex in V5-6 rather than the usual monophasic R waves seen in uncomplicated LBBB; (the S is a Q-wave equivalent).
- "Primary" ST-T wave changes (i.e., ST-T changes in the same direction as the QRS complex rather than the usual "secondary" ST-T changes seen in uncomplicated LBBB); these changes may reflect an acute, evolving MI.
- Exaggerated ST deviation in same direction as the usual LBBB ST changes in LBBB (see Example #12)

Example #12: Acute anterior MI with LBBB. Note convex-upwards ST elevation in V1-3 with exaggerated ST depression in V-6.

Example #13: Old MI (probably septal location) with LBBB. Remember LBBB without MI should have monophasic R waves in I, aVL, V6). This ECG has small q waves in I, aVL, V5-6 which suggests septal MI location. Note also the notching on upslope of S wave in V4 and the single PVC.
IV. Non-ST elevation MI (NSTEMI)
- ECG changes may be minimal, may show only T wave changes (inversion), or may show ST segment depression with or without T wave inversion.
- Although it is tempting to localize the non-Q MI by the particular leads showing ST-T changes, this is probably only valid for the ST segment elevation pattern.
- Evolving ST-T changes may include any of the following patterns:
  - Convex downward ST segment depression only
  - Convex upwards or straight ST segment elevation only
  - Symmetrical T wave inversion only
  - Combinations of above changes

V. The Pseudoinfarcts
- These are ECG conditions that mimic myocardial infarction either by simulating pathologic Q or QS waves or mimicking the typical ST-T changes of acute MI.
  - WPW preexcitation (*negative* delta wave may mimic pathologic Q waves; see ECG on p52)
  - IHSS (septal hypertrophy may make normal septal Q waves "fatter" thereby mimicking pathologic Q waves)
  - LVH (may have QS pattern or poor R wave progression in leads V1-3)
  - RVH (tall R waves in V1 or V2 may mimic true posterior MI)
  - Complete or incomplete LBBB (QS waves or poor R wave progression in leads V1-3)
  - Pneumothorax (loss of right precordial R waves)
  - Pulmonary emphysema and cor pulmonale (loss of R waves V1-3 and/or inferior Q waves with right axis deviation)
  - Left anterior fascicular block (may see small q-waves in anterior chest leads)
  - Acute pericarditis (the ST segment elevation may mimic acute transmural injury)
  - Central nervous system disease (may mimic non-Q wave MI by causing diffuse ST-T wave changes)

VI. Abnormalities of the QRS Complex: Miscellaneous Abnormalities
- **Poor R Wave Progression** – arbitrarily defined as small, or no R waves in leads V1-3 (R <2mm, plus R/S ration V4 <1). Differential diagnosis includes:
  - Normal variant (if the rest of the ECG is normal)
  - LVH (look for voltage criteria and ST-T changes of LV "strain")
  - Complete or incomplete LBBB (- QRS duration)
• Left anterior fascicular block (should see LAD in frontal plane)
• Anterior or anteroseptal MI
• Emphysema and COPD (look for R/S ratio in V5-6 <1)
• Diffuse infiltrative or myopathic processes
• WPW preexcitation (look for delta waves, short PR)

• **Prominent Anterior Forces** - defined as R/S ratio >1 in V1 or V2. Differential diagnosis includes:
  • Normal variant (if rest of the ECG is normal)
  • True posterior MI (look for evidence of inferior MI)
  • RVH (should see RAD in frontal plane and/or P-pulmonale)
  • Complete or incomplete RBBB (look for rSR' in V1)
  • WPW preexcitation (look for delta waves, short PR)

10. **ST Segment Abnormalities**

*General Introduction to ST, T, and U wave abnormalities*

• **Basic Concept**: the **specificity** of ST-T and U wave abnormalities is provided more by the clinical circumstances in which the ECG changes are found than by the particular changes themselves. Thus the term, **nonspecific ST-T wave abnormalities**, is frequently used for ST depression and T wave inversion when the clinical data are not available to correlate with the ECG findings. This does not mean that the ECG changes are unimportant! **It is the responsibility of the clinician providing care for the patient to ascertain the importance of the ECG findings.**

• Factors affecting the ST-T and U wave configuration include:
  • Intrinsic myocardial disease (e.g., myocarditis, ischemia, infarction, infiltrative or myopathic processes)
  • Drugs (e.g., digoxin, antiarrhythmics, tricyclics, and many others)
  • Electrolyte abnormalities of potassium, magnesium, calcium
  • Neurogenic factors (e.g., stroke, hemorrhage, trauma, tumor, etc.)
  • Metabolic factors (e.g., hypoglycemia, hyperventilation)
  • Atrial repolarization (e.g., at fast heart rates the atrial T wave may pull down the beginning of the ST segment; this is not a true ST change)
  • Genetic abnormalities of channel membrane proteins or **channelopathies**. Examples include hereditary long QT syndromes, and Brugada Syndrome.

• ST-T changes may be called **secondary** if they are due to changes resulting from alterations in the sequence of ventricular depolarization (e.g., bundle branch block, WPW, and ventricular ectopic beats); they are called **primary** if they are independent of changes in the sequence of ventricular depolarization (e.g., ischemic ST changes, electrolyte abnormalities, drug effects, etc.)

I. **Differential Diagnosis of ST Segment Elevation**

• Normal Variant "Early Repolarization" (usually concave upwards, ending with symmetrical, large, upright T waves)
  • "Early Repolarization": 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.
- Ischemic Heart Disease (usually convex upwards, or straightened ST segment)
  - Example: Acute transmural injury - as in this acute anterior MI

- Note: Persistent ST elevation after an acute MI suggests failure to reperfuse, a ventricular aneurysm, or an akinetic scar

- Reversible ST elevation may also be seen as a manifestation of Prinzmetal's (or "variant") angina which is caused by transient coronary artery spasm.
- ST elevation during exercise testing suggests an extremely tight coronary artery stenosis or transient spasm (transmural ischemia).

- Acute Pericarditis (see ECG below)
  - Concave upwards ST elevation in most leads except aVR
- No reciprocal ST segment depression (except in lead aVR)
- Unlike "early repolarization", T waves are usually low amplitude, and heart rate is usually increased.
- May see PR segment depression, a manifestation of atrial injury

Other Causes or ST segment elevation:
- Left ventricular hypertrophy (in right precordial leads with large S-waves)
- Left bundle branch block (in right precordial leads with large S-waves)
- Advanced hyperkalemia
- Hypothermia (prominent J-waves or Osborne waves)

II. Differential Diagnosis of ST Segment Depression
- Normal variants or artifacts:
- Pseudo-ST-depression (wandering baseline due to poor skin-electrode contact)
- Physiologic J-junctional depression with sinus tachycardia (most likely due to atrial repolarization and not a true ST change)
- Hyperventilation-induced ST segment depression (seen with anxiety)
- Subendocardial ischemia or infarction (e.g., positive exercise ECG, angina pectoris, acute coronary syndrome)
- Reciprocal ST depression in STEMI (e.g., ST depression in I, aVL during an acute inferior STEMI)
- True posterior MI (ST depression in V1-3)
- "Strain" pattern of RVH (right precordial leads) and LVH (left precordial leads)
- Drugs (e.g., digoxin)
- Electrolyte abnormalities (e.g., hypokalemia)
- Neurogenic effects (CNS disease)
11. T Wave Abnormalities

**INTRODUCTION:** The T wave is the most labile wave in the ECG. T wave changes including low-amplitude T waves and abnormally inverted T waves may be the result of many cardiac and non-cardiac conditions. The normal T wave is usually in the same direction as the QRS except in the right precordial leads (V1-3 below). Also, the normal T wave is asymmetric with the first half moving more slowly than the second half. In the normal ECG (see below) the T wave is always upright in leads I, II, V3-6, and always inverted in lead aVR. The other leads are variable depending on the QRS axis and the age of the patient.
I. Differential Diagnosis of T Wave Inversion

- Q wave and non-Q wave MI (e.g., evolving anteroseptal MI; see below):

![ECG example 1](image)

- Myocardial ischemia
- Subacute or healed pericarditis
- Myocarditis
- Myocardial contusion (from trauma; e.g., steering wheel accident)
- CNS disease causing long QT interval (especially subarachnoid hemorrhage; see ECG below with giant negative T waves):

![ECG example 2](image)

- Idiopathic apical hypertrophy (a rare form of hypertrophic cardiomyopathy with giant negative T waves)
- Mitral valve prolapse
- Hereditary long QT syndromes
- Digoxin effect
  - RVH and LVH with "strain" pattern (pressure overload)
**Miscellaneous ST-T Changes**

- Electrolyte abnormalities
  - Hypercalcemia (abbreviated ST segment with short QT interval)
  - Hypocalcemia (long ST segment with prolonged QT interval)
  - Hyperkalemia (peaked T waves, prolonged QRS duration; see ECG below)

- Hypokalemia (ST depression, low T waves, large U waves)

- Brugada type ECG (seen in the hereditary Brugada syndrome); this is an unusual pattern of ST segment elevation with or without T wave inversion in the right precordial leads. An example is seen in the ECG below. Like the long QT syndrome, there is increased incidence of malignant arrhythmias in this condition)

12. **U Wave Abnormalities**
**INTRODUCTION:** The U wave is the only remaining enigma of the ECG, and probably not for long. The origin of the normal U wave is still in question, although many authorities correlate abnormal U wave with electrophysiologic events called "afterdepolarizations" in the ventricles. These afterdepolarizations can be the source of arrhythmias caused by "triggered automaticity" including *torsade de pointes*. The normal U wave has the same polarity as the T wave and is usually less than one-third the amplitude of the T wave. U waves are usually best seen in the right precordial leads especially V2 and V3. The normal U wave is asymmetric with the ascending limb moving more rapidly than the descending limb (just the opposite of the normal T wave).

- Normal U waves are illustrated in the precordial leads below. Look closely after the T waves in V2, V3, V4 and note the small upward deflections. That's looking at 'U'!!

![ECG Illustration](image)

**Differential Diagnosis of U Wave Abnormalities**
- Prominent upright U waves
  - Sinus bradycardia accentuates normal U waves
  - Hypokalemia (remember the triad of ST segment depression, low amplitude T waves, and prominent upright U waves)
  - Various drugs including antiarrhythmics
  - LVH (may see prominent upright or inverted U waves in left V leads)
  - CNS disease and other causes of long QT (T-U fusion waves); see ECG below.
- Negative or "inverted" U waves
  - Ischemic heart disease (often indicating left main or LAD disease)
    - Myocardial infarction (in leads with pathologic Q waves)
    - During episode of acute ischemia (angina or exercise-induced ischemia)
    - Postextrasystolic in patients with coronary heart disease
    - During coronary artery spasm (Prinzmetal's angina)

- Nonischemic causes: some cases of LVH or RVH (usually in leads with prominent R waves)
  - Some patients with LQTS (see below: Lead V6 shows giant negative TU fusion wave in patient with LQTS; a prominent upright U wave is seen in Lead V1)
Cardiac Conduction System

Ventricular Conduction

Right Ventricular PVC

Left Ventricular PVC
Electrical and Mechanical Events

Ventricular Pacing
The pacing lead is inserted into the ventricle to cause ventricular depolarization.

Atrial Pacing
The pacing lead is inserted into the atrium to cause atrial depolarization.

A-V Sequential Pacing
The pacing leads are inserted into both the atrium and ventricle stimulating at set intervals.

Pacemaker Lead Wire Replacement
All About Premature Beats

1. PAC with incomplete compensatory pause
2. PVC with complete compensatory pause
3. Interpolated PVC: the following sinus impulse has an increased PR interval due to retrograde concealed conduction of the PVC
4. PVC with retrograde atrial capture; the pause is incomplete because the retrograde P wave resets the sinus node.
5. PVC with retrograde atrial capture and then return to ventricles (using dual AV pathways) to cause a ventricular echo beat (rare!)
6. Nonconducted PAC: the most common cause of an unexpected pause in an otherwise regular rhythm.
The J point occurs at the end of the QRS complex. The ST segment begins at the J point and extends to a user defined interval.

**ST Segment Depression**

**Normal Sinus Rhythm**

<table>
<thead>
<tr>
<th>Heart Rate</th>
<th>Rhythm</th>
<th>P Wave</th>
<th>PR interval (in seconds)</th>
<th>QRS (in seconds)</th>
</tr>
</thead>
<tbody>
<tr>
<td>60-100 bpm</td>
<td>Regular</td>
<td>Before each QRS, identical</td>
<td>.12 to .20</td>
<td>&lt; .12</td>
</tr>
</tbody>
</table>
### Sinus Tachycardia

<table>
<thead>
<tr>
<th>Heart Rate</th>
<th>Rhythm</th>
<th>P Wave</th>
<th>PR interval (in seconds)</th>
<th>QRS (in seconds)</th>
</tr>
</thead>
<tbody>
<tr>
<td>&gt; 100 bpm</td>
<td>Regular</td>
<td>Before each QRS, identical</td>
<td>.12 to .20</td>
<td>&lt;.12</td>
</tr>
</tbody>
</table>

### Sinus Bradycardia

<table>
<thead>
<tr>
<th>Heart Rate</th>
<th>Rhythm</th>
<th>P Wave</th>
<th>PR interval (in seconds)</th>
<th>QRS (in seconds)</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;60 bpm</td>
<td>Regular</td>
<td>Before each QRS, identical</td>
<td>.12 to .20</td>
<td>&lt;.12</td>
</tr>
</tbody>
</table>
### Wandering Pacemaker

<table>
<thead>
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<th>Heart Rate</th>
<th>Rhythm</th>
<th>P Wave</th>
<th>PR interval (in seconds)</th>
<th>QRS (in seconds)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Usually &lt;60 bpm</td>
<td>Irregular</td>
<td>Multiple forms</td>
<td>Variable</td>
<td>&lt;.12</td>
</tr>
</tbody>
</table>