Basic ECG Interpretation
ECG

• The ECG records the electrical signal of the heart as the muscle cells depolarize (contract) and repolarize.

• Normally, the SA Node generates the initial electrical impulse and begins the cascade of events that results in a heart beat.

• Recall that cells resting have a negative charge with respect to exterior and depolarization consists of positive ions rushing into the cell.
Cell Depolarization

- Flow of sodium ions into cell during activation

![Diagram of cell depolarization and ionic balance](image)
Propagating Activation Wavefront

• When the cells are at rest, they have a negative transmembrane voltage – surrounding media is positive

• When the cells depolarize, they switch to a positive transmembrane voltage – surrounding media becomes negative

• This leads to a propagating electric vector (pointing from negative to positive)
Propagating Activation Wavefront

**DEPOLARIZATION**
Positive ions (Na⁺) flowing into the depolarizing cells make \( \Phi_0 \) (outside the cells) more negative.

**REPOLARIZATION**
Positive ions (K⁺) flowing out from the repolarizing cells make \( \Phi_0 \) (outside the cells) more positive.

Resting | Depolarizing | Activated (Depolarized) | Repolarizing | Resting
--- | --- | --- | --- | ---
- | + | + | + | -
- | - | + | + | -
- | + | + | + | -
- | - | + | + | -

(Action pulse)  | Direction of wavefront movement
ECG Leads

- In 1908, Willem Einthoven developed a system capable of recording these small signals and recorded the first ECG.
- The leads were based on the Einthoven triangle associated with the limb leads.
- Leads put heart in the middle of a triangle
Rules of ECG

• Wave of depolarization traveling towards a positive electrode causes an upward deflection on the ECG

• Wave of depolarization traveling away from a positive electrode causes a downward deflection on the ECG
Propagating Activation Wavefront

Depol. toward positive electrode  
Positive Signal

A  
Resting  Activated

Depol. away from positive electrode  
Negative Signal

B  
Resting  Activated

Rerpol. toward positive electrode  
Negative Signal

C  
Still activated  Resting again

Rerpol. away from positive electrode  
Positive Signal

D  
Still activated  Resting again
The Normal Conduction System
ECG Signal

• The excitation begins at the sinus (SA) node and spreads along the atrial walls
• The resultant electric vector is shown in yellow
• Cannot propagate across the boundary between atria and ventricle
• The projections on Leads I, II and III are all positive
ECG Signal

• Atrioventricular (AV) node located on atria/ventricle boundary and provides conducting path

• Pathway provides a delay to allow ventricles to fill

• Excitation begins with the septum
ECG Signal

- Depolarization continues to propagate toward the apex of the heart as the signal moves down the bundle branches.

- Overall electric vector points toward apex as both left and right ventricles depolarize and begin to contract.
ECG Signal

- Depolarization of the right ventricle reaches the epicardial surface
- Left ventricle wall is thicker and continues to depolarize
- As there is no compensating electric forces on the right, the electric vector reaches maximum size and points left
- Note the atria have repolarized, but signal is not seen
ECG Signal

- Depolarization front continues to propagate to the back of the left ventricular wall
- Electric vector decreases in size as there is less tissue depolarizing
ECG Signal

- Depolarization of the ventricles is complete and the electric vector has returned to zero
ECG Signal

- Ventricular repolarization begins from the outer side of the ventricles with the left being slightly dominant.
- Note that this produces an electric vector that is in the same direction as the depolarization traveling in the opposite direction.
- Repolarization is diffuse and generates a smaller and longer signal than depolarization.
ECG Signal

- Upon complete repolarization, the heart is ready to go again and we have recorded an ECG trace
ORIENTATION OF THE 12 LEAD ECG
ECG Information

• The 12 leads allow tracing of electric vector in all three planes of interest

• Not all the leads are independent, but are recorded for redundant information
Orientation of the 12 Lead ECG

Heart's electrical activity in 3 approximately orthogonal directions:

• Right Left
• Superior Inferior
• Anterior Posterior
EKG Leads

The standard EKG has 12 leads:

3 Standard Limb Leads
3 Augmented Limb Leads
6 Precordial Leads

The axis of a particular lead represents the viewpoint from which it looks at the heart.
Each of the 12 leads represents a particular orientation in space, as indicated below:

- **Bipolar limb leads (frontal plane):**
  - Lead I: RA (-) to LA (+) (lateral)
  - Lead II: RA (-) to LF (+) (Inferior)
  - Lead III: LA (-) to LF (+) (Inferior)

- **Augmented unipolar limb leads (frontal plane):**
  - Lead aVR: RA (+) to [LA & LF] (-) (Rightward) cavity
  - Lead aVL: LA (+) to [RA & LF] (-) (Lateral)
  - Lead aVF: LF (+) to [RA & LA] (-) (Inferior)

- **Unipolar (+) chest leads (horizontal plane):**
  - Leads V1, V2, V3: (Posterior Anterior)
  - Leads V4, V5, V6: (lateral)
Standard Limb Leads
Standard Limb Leads
Augmented Leads

- Three additional limb leads are also used: $aV_R$, $aV_L$, and $aV_F$
- These are unipolar leads
Augmented Limb Leads
All Limb Leads
Precordial Leads

- Unipolar leads

V1 – 4th intercostal space to rt of sternum
V2 – 4th intercostal space to lt of the sternum
V3 – between V2 and V4
V4 – 5th intercostal space midclavicular line
V5 – anterior axillary line, in line with V4
V6 – midaxillary line, in line with V4
Lead Orientation

Anterior, Posterior, Lateral, Inferior Views

- Anterior – V1 – V4
- Left Lateral – I, avL, V5 and V6
- Inferior – II, III, and avF
- Posterior – avR, reciprocal changes in V1
## Summary of Leads

<table>
<thead>
<tr>
<th></th>
<th>Limb Leads</th>
<th>Precordial Leads</th>
</tr>
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<tbody>
<tr>
<td><strong>Bipolar</strong></td>
<td>I, II, III (standard limb leads)</td>
<td>-</td>
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<tr>
<td><strong>Unipolar</strong></td>
<td>aVR, aVL, aVF (augmented limb leads)</td>
<td>V₁-V₆</td>
</tr>
</tbody>
</table>

- **Limb Leads**
- **Precordial Leads**
Arrangement of Leads on the EKG

<table>
<thead>
<tr>
<th></th>
<th>I</th>
<th>aVR</th>
<th>V₁</th>
<th>V₄</th>
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<tbody>
<tr>
<td>Ⅰ</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Ⅱ</td>
<td></td>
<td></td>
<td>V₂</td>
<td>V₅</td>
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<tr>
<td>Ⅲ</td>
<td></td>
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## Anatomic Groups
*(Septum)*

<table>
<thead>
<tr>
<th>I</th>
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<th>$V_4$</th>
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Anatomic Groups
(Anterior Wall)

<table>
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<tr>
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Anatomic Groups (Lateral Wall)

<table>
<thead>
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## Anatomic Groups

**(Inferior Wall)**

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<th>V(_4)</th>
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<tr>
<td>II</td>
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<td>V(_2)</td>
<td>V(_5)</td>
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### Anatomic Groups

(Summary)

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ECG Diagnosis

- The trajectory of the electric vector resulting from the propagating activation wavefront can be traced by the ECG and used to diagnose cardiac problems.
NORMAL
ECG
TRACINGS
ECG

- Right and left ventricular depolarization
- Depolarization of the right and left atria
- Septal depolarization
- Ventricular repolarization
- "after depolarizations" in the ventricles
Wave definition

- P wave
- Q wave – first downward deflection after P wave
- Rwave – first upward deflection after Q wave
- R` wave – any second upward deflection
- S wave – first downward deflection after the R wave
ECG Waves and Intervals:

P wave : the *sequential* activation (depolarization) of the right and left atria

QRS complex : right and left ventricular depolarization (normally the ventricles are activated *simultaneously*).

ST-T wave : ventricular repolarization

U wave : origin for this wave is not clear - but probably represents "after depolarizations" in the ventricles.

PR interval : time interval from onset of atrial depolarization (P wave) to onset of ventricular depolarization (QRS complex).

QRS duration : duration of ventricular muscle depolarization.

QT interval : duration of ventricular depolarization and repolarization.

RR interval : duration of ventricular cardiac cycle (an indicator of ventricular rate).

PP interval : duration of atrial cycle (an indicator of atrial rate).
3. Conduction:

Normal Sino-atrial (SA), Atrio-ventricular (AV), and Intraventricular (IV) conduction

Both the PR interval and QRS duration should be within the limits specified above.
4. 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 < 0.12 sec

   P amplitude < 2.5 mm

   Frontal plane P wave axis: 0° to +75°

   May see notched P waves in frontal plane
QRS Complex

The 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 $\leq 0.10$ sec

QRS amplitude is quite variable from lead to lead and from person to person. Two determinates 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)
The normal QRS axis range (+90° to -30°); this implies that the QRS be mostly positive (upright) in leads II and I.

Normal q-waves reflect normal septal activation (beginning on the LV septum); they are narrow (<0.04s duration) and small (<25% the amplitude of the R wave). They 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°.

Septal q waves should not be confused with the pathologic Q waves of myocardial infarction.
QRS Complex

Small r-waves begin in V1 or V2 and progress in size to V5. The R-V6 is usually smaller than R-V5.

In reverse, the s-waves begin in V6 or V5 and progress 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 and T wave

ST-T wave is a smooth, continuous waveform beginning with the J-point (end of QRS), slowly rising to the peak of the T wave. Normal ECG the T wave is always upright in leads I, II, V3-6, and always inverted in lead aVR.

Normal ST segment configuration is concave upward. Convex or straight upward ST segment elevation is abnormal and suggests transmural injury or infarction.

ST segment depression characterized as "upsloping", "horizontal", or "downsloping” is always an abnormal finding.
U Wave

The normal U Wave: (the most neglected of the ECG waveforms) U wave amplitude is usually < 1/3 T wave amplitude in same lead

U wave direction is the same as T wave direction in that lead

U waves are more prominent at slow heart rates and usually best seen in the right precordial leads.

Origin of the U wave is thought to be related to *afterdepolarizations* which interrupt or follow repolarization.
METHOD
OF
ECG
INTERPRETATION
Method of ECG interpretation

1. **Measurements (usually made in frontal plane leads)**
   - Heart rate (state atrial and ventricular, if different)
   - PR interval (from beginning of P to beginning of QRS)
   - QRS duration (width of most representative QRS)
   - QT interval (from beginning of QRS to end of T)
   - QRS axis in frontal plane

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)
   - Consider all rhythm events from atria, AV junction, and ventricles

3. **Conduction Analysis**
   - Normal" conduction implies normal sino-atrial (SA), atrio-ventricular (AV), and intraventricular (IV) conduction
   - SA block , 2nd degree (type I vs. type II)
   - AV block - 1st, 2nd (type I vs. type II), and 3rd degree
   - IV blocks - bundle branch, fascicular, and nonspecific blocks
   - Exit blocks: blocks just distal to ectopic pacemaker site
4. Waveform Description

- **P waves**: Are they too wide, too tall, look funny (i.e., are they ectopic), etc.?
- **QRS complexes**: Look for pathological 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

Interpret the ECG as "Normal", or "Abnormal".

Example: Inferior MI, probably acute

- Old anteroseptal MI
- Left anterior fascicular block (LAFB)
- Left ventricular hypertrophy (LVH)
- Nonspecific ST-T wave abnormalities
- Any rhythm abnormalities
ECG- Heart rate

- ECG paper moves at a standardized 25mm/sec
- Each large square is 5 mm
- Each large square is 0.2 sec
- 300 large squares per minute / 1500 small squares per minute
- 300 divided by number of large squares between R-R
- 1500 divided by number of small squares between R-R
1. Measurements (Normal)

Heart Rate: 60 - 90 bpm
PR Interval: 0.12 - 0.20 sec
QRS Duration: 0.06 - 0.10 sec
QT Interval (QT<sub>c</sub> ≤ 0.40 sec)

*Poor Man's Guide* to upper limits of QT:
For HR = 70 bpm, QT<sub>c</sub>≤0.40 sec;
for every 10 bpm increase above 70 subtract 0.02 sec, and
for every 10 bpm decrease below 70 add 0.02 sec.

For example:
QT < 0.38 @ 80 bpm
QT ≤ 0.42 @ 60 bpm

Frontal Plane QRS Axis:
+90 ° to -30 ° (in the adult)
ECG Rhythm

Interpretation

How to Analyze a Rhythm
Normal Sinus Rhythm (NSR)

- Etiology: the electrical impulse is formed in the SA node and conducted normally.

- This is the normal rhythm of the heart; other rhythms that do not conduct via the typical pathway are called arrhythmias.
Step 1: Calculate Rate

- Count the # of R waves in a 6 second rhythm strip, then multiply by 10.
- Reminder: all rhythm strips in the Modules are 6 seconds in length.

Interpretation?

\[ 9 \times 10 = 90 \text{ bpm} \]
Step 1: Calculate Rate

R wave

- Option 2
  - Find a R wave that lands on a bold line.
  - Count the # of large boxes to the next R wave. If the second R wave is 1 large box away the rate is 300, 2 boxes - 150, 3 boxes - 100, 4 boxes - 75, etc.

(cont)
Step 2: Determine regularity

- Look at the R-R distances (using a caliper or markings on a pen or paper).
- Regular (are they equidistant apart)? Occasionally irregular? Regularly irregular? Irregularly irregular?

Interpretation?

Regular
Step 3: Assess the P waves

- Are there P waves?
- Do the P waves all look alike?
- Do the P waves occur at a regular rate?
- Is there one P wave before each QRS?

Interpretation?

Normal P waves with 1 P wave for every QRS
Step 4: Determine PR interval

- Normal: 0.12 - 0.20 seconds.
  (3 - 5 boxes)

Interpretation?

0.12 seconds
Step 5: QRS duration

• Normal: 0.04 - 0.12 seconds.
  (1 - 3 boxes)

Interpretation?

0.08 seconds
Rhythm Summary

- Rate: 90-95 bpm
- Regularity: regular
- P waves: normal
- PR interval: 0.12 s
- QRS duration: 0.08 s

Interpretation?

Normal Sinus Rhythm
RHYTHM DISTURBANCES
Normal Sinus Rhythm (NSR)

• This is the normal rhythm of the heart; other rhythms that do not conduct via the typical pathway are called arrhythmias.
**NSR Parameters**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
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<tbody>
<tr>
<td>Rate</td>
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<tr>
<td>Regularity</td>
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<tr>
<td>P waves</td>
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<tr>
<td>PR interval</td>
<td>0.12 - 0.20 s</td>
</tr>
<tr>
<td>QRS duration</td>
<td>0.04 - 0.12 s</td>
</tr>
</tbody>
</table>

Any deviation from above is sinus tachycardia, sinus bradycardia or an arrhythmia
Arrhythmia Formation

Arrhythmias can arise from problems in the:

- Sinus node
- Atrial cells
- AV junction
- Ventricular cells
SA Node Problems

The SA Node can:

- fire too slow
  - Sinus Bradycardia

- fire too fast
  - Sinus Tachycardia
Atrial Cell Problems

Atrial cells can:
• fire occasionally from a focus

• fire continuously due to a looping re-entrant circuit

Premature Atrial Contractions (PACs)

Atrial Flutter
Teaching Moment

- A re-entrant pathway occurs when an impulse loops and results in self-perpetuating impulse formation.
Atrial Cell Problems

Atrial cells can also:

• fire continuously from multiple foci
or
• fire continuously due to multiple micro re-entrant “wavelets”

Atrial Fibrillation
Multiple micro re-entrant “wavelets” refers to wandering small areas of activation which generate fine chaotic impulses. Colliding wavelets can, in turn, generate new foci of activation.
AV Junctional Problems

The AV junction can:

• fire continuously due to a looping re-entrant circuit

• block impulses coming from the SA Node

Paroxysmal Supraventricular Tachycardia
AV Junctional Blocks
Ventricular Cell Problems

Ventricular cells can:

• fire occasionally from 1 or more foci
• fire continuously from multiple foci
• fire continuously due to a looping re-entrant circuit

Premature Ventricular Contractions (PVCs)
Ventricular Fibrillation
Ventricular Tachycardia
Arrhythmias

- Sinus Rhythms
- Premature Beats
- Supraventricular Arrhythmias
- Ventricular Arrhythmias
- AV Junctional Blocks
Sinus Rhythms

- *Sinus Bradycardia*
- *Sinus Tachycardia*
Sinus Bradycardia

- Rate? 30 bpm
- Regularity? regular
- P waves? normal
- PR interval? 0.12 s
- QRS duration? 0.10 s

Interpretation? Sinus Bradycardia
Sinus Bradycardia

- **Etiology:** SA node is depolarizing slower than normal, impulse is conducted normally (i.e. normal PR and QRS interval).
Sinus Tachycardia

• Rate? 130 bpm
• Regularity? regular
• P waves? normal
• PR interval? 0.16 s
• QRS duration? 0.08 s

Interpretation? Sinus Tachycardia
Sinus Tachycardia

- Etiology: SA node is depolarizing faster than normal, impulse is conducted normally.
- Remember: sinus tachycardia is a response to physical or psychological stress, not a primary arrhythmia.
Premature Beats

- *Premature Atrial Contractions (PACs)*
- *Premature Ventricular Contractions (PVCs)*
Premature Atrial Contractions

- Rate? 70 bpm
- Regularity? occasionally irreg.
- P waves? 2/7 different contour
- PR interval? 0.14 s (except 2/7)
- QRS duration? 0.08 s

Interpretation? NSR with Premature Atrial Contractions
Premature Atrial Contractions

- Deviation from NSR

- These ectopic beats originate in the atria (but not in the SA node), therefore the contour of the P wave, the PR interval, and the timing are different than a normally generated pulse from the SA node.
Premature Atrial Contractions

• **Etiology:** Excitation of an atrial cell forms an impulse that is then conducted normally through the AV node and ventricles.
Teaching Moment

- When an impulse originates anywhere in the atria (SA node, atrial cells, AV node, Bundle of His) and then is conducted normally through the ventricles, the QRS will be narrow (0.04 - 0.12 s).
Sinus Rhythm with 1 PVC

- Rate? 60 bpm
- Regularity? occasionally irreg.
- P waves? none for 7th QRS
- PR interval? 0.14 s
- QRS duration? 0.08 s (7th wide)

Interpretation? Sinus Rhythm with 1 PVC
• Deviation from NSR
  • Ectopic beats originate in the ventricles resulting in wide and bizarre QRS complexes.
  • When there are more than 1 premature beats and look alike, they are called “uniform”. When they look different, they are called “multiform”.
PVCs

- **Etiology:** One or more ventricular cells are depolarizing and the impulses are abnormally conducting through the ventricles.
Teaching Moment

• When an impulse originates in a ventricle, conduction through the ventricles will be inefficient and the QRS will be wide and bizarre.
Ventricular Conduction

Normal
Signal moves rapidly through the ventricles

Abnormal
Signal moves slowly through the ventricles
The QRS Axis

The QRS axis represents the net overall direction of the heart’s electrical activity.

Abnormalities of axis can hint at:
- Ventricular enlargement
- Conduction blocks (i.e. hemiblocks)
The QRS Axis

By near-consensus, the normal QRS axis is defined as ranging from -30° to +90°.

-30° to -90° is referred to as a left axis deviation (LAD).

+90° to +180° is referred to as a right axis deviation (RAD).
Determining the Axis

• The Quadrant Approach

• The Equiphasic Approach
The Quadrant Approach

1. Examine the QRS complex in leads I and aVF to determine if they are predominantly positive or predominantly negative. The combination should place the axis into one of the 4 quadrants below.
2. In the event that LAD is present, examine lead II to determine if this deviation is pathologic. If the QRS in II is predominantly positive, the LAD is non-pathologic (in other words, the axis is normal). If it is predominantly negative, it is pathologic.
Quadrant Approach: Example 1

Negative in I, positive in aVF \( \rightarrow \) RAD

The Alan E. Lindsay ECG Learning Center
http://medstat.med.utah.edu/kw/ecg/
Quadrant Approach: Example 2

Positive in I, negative in aVF  $\rightarrow$  Predominantly positive in II  $\rightarrow$

Normal Axis (non-pathologic LAD)
The Equiphasic Approach

1. Determine which lead contains the most equiphasic QRS complex. The fact that the QRS complex in this lead is equally positive and negative indicates that the net electrical vector (i.e. overall QRS axis) is perpendicular to the axis of this particular lead.

2. Examine the QRS complex in whichever lead lies 90° away from the lead identified in step 1. If the QRS complex in this second lead is predominantly positive, than the axis of this lead is approximately the same as the net QRS axis. If the QRS complex is predominantly negative, than the net QRS axis lies 180° from the axis of this lead.
Determining the Axis

- Predominantly Positive
- Predominantly Negative
- Equiphasic
Equiphasic Approach: Example 1

Equiphasic in aVF $\rightarrow$ Predominantly positive in I $\rightarrow$ QRS axis $\approx 0^\circ$
Equiphasic Approach: Example 2

Equiphasic in II → Predominantly negative in aVL → QRS axis ≈ +150°
QRS Axis Determination

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<td>+120</td>
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CONDUCTION

DISTURBANCES
Measurement abnormality
PR Interval

PR Interval
Normal: 0.12 - 0.20s

**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 or the left atrium to the left ventricle, and this permits early activation of the ventricles (delta wave) and a short PR interval.

**LGL (Lown-Ganong-Levine):** An AV nodal bypass track into the His bundle permits early activation of the ventricles without a delta-wave because the ventricles activate normally.
Short PR Interval

**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).

**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)

**Normal variant**
Prolonged PR: >0.20s

**First degree AV block**
(PR interval usually constant) Intra-atrial conduction delay (uncommon)
Slowed conduction in AV node (most common site)
Slowed conduction in His bundle (rare)
Slowed conduction in bundle branch (when contralateral bundle is blocked)

**Second degree AV block** (PR interval may be normal or prolonged; some P waves do not conduct)
- 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
1st Degree AV Block

- **Etiology:** Prolonged conduction delay in the AV node or Bundle of His.
First degree AV block

1st degree AV block is defined by PR intervals greater than 200 ms caused by
- drugs, such as digoxin;
- excessive vagal tone;
- ischemia; or
- intrinsic disease in the AV junction or bundle branch system
First degree AV block

- Rate? 60 bpm
- Regularity? regular
- P waves? normal
- PR interval? 0.36 s
- QRS duration? 0.08 s

Interpretation? 1st Degree AV Block
2nd Degree AV Block, Type I

- Deviation from NSR
  - PR interval progressively lengthens, then the impulse is completely blocked (P wave not followed by QRS).
2nd Degree AV Block, Type I

- **Etiology**: Each successive atrial impulse encounters a longer and longer delay in the AV node until one impulse (usually the 3rd or 4th) fails to make it through the AV node.
2nd Degree AV Block, Type I

- Rate? 50 bpm
- Regularity? regularly irregular
- P waves? nl, but 4th no QRS
- PR interval? lengthens
- QRS duration? 0.08 s

Interpretation? 2nd Degree AV Block, Type I
Type I (Mobitz)- 2nd degree AV block
Type I vs. Type II 2nd Degree AV Block

In **type I 2nd degree AV block** the PR progressively lengthens until a nonconducted P wave occurs. The PR gets longer by smaller and smaller increments; this results in gradual shortening of the RR intervals. RR interval after the pause is longer.

In **type II AV block**, the PR is constant until the nonconducted P wave occurs. The RR interval of the pause is usually 2x the basic RR interval.
2nd Degree AV Block, Type II (Mobitz)

• Deviation from NSR
  • Occasional P waves are completely blocked (P wave not followed by QRS).
2nd Degree AV Block, Type II (Mobitz)

- **Etiology:** Conduction is all or nothing (no prolongation of PR interval); typically block occurs in the Bundle of His.
Type II - AV block (Mobitz)

In **type II AV block**, the PR is constant until the nonconducted P wave occurs. The RR interval of the pause is usually 2x the basic RR interval. Block may be 2:1 or 3:1.
Type II - AV block (Mobitz)
3rd Degree AV Block

• Deviation from NSR
  • The P waves are completely blocked in the AV junction; QRS complexes originate independently from below the junction.
3rd Degree AV Block

- **Etiology:** There is complete block of conduction in the AV junction, so the atria and ventricles form impulses independently of each other. Without impulses from the atria, the ventricles own intrinsic pacemaker kicks in at around 30 - 45 beats/minute.
Remember

• When an impulse originates in a ventricle, conduction through the ventricles will be inefficient and the QRS will be wide and bizarre.
Complete AV Block (3rd Degree) with Junctional Rhythm
**Junctional Rhythm**

- Rate: 40–60 bpm
- Rhythm: Regular
- P Waves: Absent, inverted, buried, or retrograde
- PR Interval: None, short, or retrograde
- QRS: Normal (0.06–0.10 sec)
Idioventricular Rhythm

Rate: 20–40 bpm
Rhythm: Regular
P Waves: None
PR Interval: None
QRS: Wide (0.10 sec), bizarre appearance

Idioventricular rhythm may also be called agonal rhythm
Asystole

Electrical activity in the ventricles is completely absent

Rate: None
Rhythm: None
P Waves: None
PR Interval: None
QRS: None
Bundle Branch Blocks
Bundle Branch Blocks

Turning our attention to bundle branch blocks...

Remember normal impulse conduction is:
- SA node →
- AV node →
- Bundle of His →
- Bundle Branches →
- Purkinje fibers
Normal Impulse Conduction

- Sinoatrial node
- AV node
- Bundle of His
- Bundle Branches
- Purkinje fibers
Bundle Branch Blocks

So, depolarization of the Bundle Branches and Purkinje fibers are seen as the QRS complex on the ECG.

Therefore, a conduction block of the Bundle Branches would be reflected as a change in the QRS complex.
Bundle Branch Blocks

With Bundle Branch Blocks you will see two changes on the ECG.

1. QRS complex widens
2. QRS morphology changes
Why does the QRS complex widen?

When the conduction pathway is blocked it will take longer for the electrical signal to pass throughout the ventricles.
Bundle-branch Block

RIGHT BUNDLE-BRANCH BLOCK
QRS duration greater than 0.12 s
Wide S wave in leads I, V_5 and V_6
Right Bundle Branch Blocks

What QRS morphology is characteristic?

For RBBB the wide QRS complex assumes a unique, virtually diagnostic shape in those leads overlying the right ventricle (V₁ and V₂).

“Rabbit Ears”
Left Bundle Branch Blocks

What QRS morphology is characteristic?

For **LBBB** the wide QRS complex assumes a characteristic change in shape in those leads opposite the left ventricle (right ventricular leads - $V_1$ and $V_2$).

Normal

Broad, deep S waves
Left Anterior Fascicular Block (LAFB)

LAFB is the most common of the intraventricular conduction defects. It is recognized by:
1) left axis deviation;
2) rS complexes in II, III, aVF; and
3) small q in I and/or aVL.
Bifascicular Block: RBBB + LAFB

This is the most common of the bifascicular blocks.

**RBBB** is most easily recognized in the precordial leads by the rSR' in V1 and the wide S wave in V6

**LAFB** is best seen in the frontal plane leads as evidenced by left axis deviation (-50 degrees), rS complexes in II, III, aVF, and the small q in leads I and/or aVL.
Supraventricular and Ventricular Arrhythmias
Arrhythmias

• Sinus Rhythms
• Premature Beats
• Supraventricular Arrhythmias
• Ventricular Arrhythmias
• AV Junctional Blocks
PREMATURE VENTRICULAR CONTRACTION (PVC)

- A single impulse originates at right ventricle

Time interval between normal R peaks is a multiple of R-R intervals
Supraventricular Arrhythmias

• Atrial Fibrillation

• Atrial Flutter

• Paroxysmal Supraventricular Tachycardia
Atrial Fibrillation

- **Deviation from NSR**
  - No organized atrial depolarization, so no normal P waves (impulses are not originating from the sinus node).
  - Atrial activity is chaotic (resulting in an irregularly irregular rate).
  - Common, affects 2-4%, up to 5-10% if > 80 years old
Atrial Fibrillation

• **Etiology:** Recent theories suggest that it is due to multiple re-entrant wavelets conducted between the R & L atria. Either way, impulses are formed in a totally unpredictable fashion. The AV node allows some of the impulses to pass through at variable intervals (so rhythm is irregularly irregular).

**ATRIAL FIBRILLATION**
Impulses have chaotic, random pathways in atria
Atrial Fibrillation

- Rate? 100 bpm
- Regularity? irregularly irregular
- P waves? none
- PR interval? none
- QRS duration? 0.06 s

Interpretation? Atrial Fibrillation
Atrial Flutter

• Deviation from NSR
  • No P waves. Instead flutter waves (note “sawtooth” pattern) are formed at a rate of 250 - 350 bpm.
  • Only some impulses conduct through the AV node (usually every other impulse).
Atrial Flutter

or 4th impulse generating a QRS (others are blocked in the AV node as the node repolarizes).

ATRIAL FLUTTER
Impulses travel in circular course in atria –
Atrial Flutter

- Rate? 70 bpm
- Regularity? regular
- P waves? flutter waves
- PR interval? none
- QRS duration? 0.06 s

Interpretation? Atrial Flutter
PSVT - *Paroxysmal Supraventricular Tachycardia*

- Deviation from NSR
  - The heart rate suddenly speeds up, often triggered by a PAC (not seen here) and the P waves are lost.
• **Etiology:** There are several types of PSVT but all originate above the ventricles (therefore the QRS is narrow).

• Most common: abnormal conduction in the AV node (reentrant circuit looping in the AV node).
Paroxysmal Supraventricular Tachycardia (PSVT)

- Rate? 74 → 148 bpm
- Regularity? Regular → regular
- P waves? Normal → none
- PR interval? 0.16 s → none
- QRS duration? 0.08 s

Interpretation? Paroxysmal Supraventricular Tachycardia (PSVT)
Ventricular Arrhythmias

- Ventricular Tachycardia
- Ventricular Fibrillation
Ventricular Tachycardia

- Deviation from NSR
  - Impulse is originating in the ventricles (no P waves, wide QRS).
Ventricular Tachycardia

- **Etiology:** There is a re-entrant pathway looping in a ventricle (most common cause).

- Ventricular tachycardia can sometimes generate enough cardiac output to produce a pulse; at other times no pulse can be felt.
Ventricular Tachycardia

- Rate? 160 bpm
- Regularity? regular
- P waves? none
- PR interval? none
- QRS duration? wide (> 0.12 sec)

Interpretation? Ventricular Tachycardia
Ventricular Fibrillation

- Deviation from NSR
  - Completely abnormal.
Ventricular Fibrillation

- **Etiology**: The ventricular cells are excitable and depolarizing randomly.

- Rapid drop in cardiac output and death occurs if not quickly reversed
Ventricular Fibrillation

- Rate? none
- Regularity? irregularly irreg.
- P waves? none
- PR interval? none
- QRS duration? wide, if recognizable

Interpretation? Ventricular Fibrillation
ST Elevation and non-ST Elevation MIs
Myocardial Ischemia and Infarction

• Oxygen depletion to heart can cause an oxygen debt in the muscle (**ischemia**)
• If oxygen supply stops, the heart muscle dies (**infarction**)
• The infarct area is electrically silent and represents an inward facing electric vector...can locate with ECG
ECG Changes

Ways the ECG can change include:

- Appearance of pathologic Q-waves
- T-waves: peaked, flattened, inverted
- ST elevation & depression
ECG Changes & the Evolving MI

There are two distinct patterns of ECG change depending if the infarction is:

- ST Elevation
- Non-ST Elevation
ST Elevation Infarction

The ECG changes seen with a ST elevation infarction are:

Before injury  Normal ECG

Ischemia  ST depression, peaked T-waves, then T-wave inversion

Infarction  ST elevation & appearance of Q-waves

Fibrosis  ST segments and T-waves return to normal, but Q-waves persist
ST Elevation Infarction

Here’s a diagram depicting an evolving infarction:

A. Normal ECG prior to MI

B. Ischemia from coronary artery occlusion results in ST depression (not shown) and peaked T-waves

C. Infarction from ongoing ischemia results in marked ST elevation

D/E. Ongoing infarction with appearance of pathologic Q-waves and T-wave inversion

F. Fibrosis (months later) with persistent Q-waves, but normal ST segment and T-waves
ST Elevation Infarction

Here’s an ECG of an inferior MI:

Look at the inferior leads (II, III, aVF).

Question: What ECG changes do you see?

ST elevation and Q-waves

What is the rhythm? Atrial fibrillation (irregularly irregular with narrow QRS)!
Non-ST Elevation Infarction

Here’s an ECG of an inferior MI later in time:

Now what do you see in the inferior leads?

ST elevation, Q-waves and T-wave inversion
Non-ST Elevation Infarction

The ECG changes seen with a non-ST elevation infarction are:

Before injury  Normal ECG

Ischemia   ST depression & T-wave inversion

Infarction   ST depression & T-wave inversion

Fibrosis   ST returns to baseline, but T-wave inversion persists
Non-ST Elevation Infarction

Here’s an ECG of an evolving non-ST elevation MI:

Note the ST depression and T-wave inversion in leads V₂-V₆.

Question:
What area of the heart is infarcting?

Anterolateral
Atrial & Ventricular Hypertrophy
Atrial Hypertrophy: Enlarged Atria

**RIGHT ATRIAL HYPERTROPHY**
- Tall, peaked P wave in leads I and II

**LEFT ATRIAL HYPERTROPHY**
- Wide, notched P wave in lead II
- Diphasic P wave in V₁
Left Ventricular Hypertrophy

Compare these two 12-lead ECGs. What stands out as different with the second one?

Normal

Left Ventricular Hypertrophy

Answer: The QRS complexes are very tall (increased voltage)
Left Ventricular Hypertrophy

Why is left ventricular hypertrophy characterized by tall QRS complexes?

As the heart muscle wall thickens there is an increase in electrical forces moving through the myocardium resulting in increased QRS voltage.
Ventricular Hypertrophy: Enlarged Ventricle

**LEFT VENTRICULAR HYPERTROPHY**
Large S wave in leads $V_1$ and $V_2$
Large R wave in leads $V_6$ and $V_6$
Left Ventricular Hypertrophy

• Criteria exists to diagnose LVH using a 12-lead ECG.

• However, for now, all you need to know is that the QRS voltage increases with LVH.
Right Ventricular Hypertrophy

1. Any one of the following in lead V1:
   - R/S ratio > 1 and negative T wave
   - R > 6 mm, or S < 2mm,

2. Right axis deviation (>90 degrees) in presence of disease capable of causing RVH.

3. 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.
Electrolyte disturbances and ECG changes
Hyperkalaemia: ECG changes

1. Appearance of tall, pointed, narrow T waves.

2. Decreased P wave amplitude, decreased R wave height, widening of QRS complexes, ST segment changes (elevation/depression), hemiblock (esp. left anterior) and 1st degree heart block.

3. Advanced intraventricular block (very wide QRS with RBBB, LBBB, bi- or tri-fascicular blocks) and ventricular ectopics.

4. Absent P waves, very broad, bizarre QRS complexes, AV block, VT, VF or ventricular asystole.

5. Marked widening of the QRS duration combined with tall, peaked T waves are suggestive of advanced hyperkalaemia.
Hyperkalaemia: ECG changes

- Tall, pointed, narrow T waves
- Widening of QRS complexes
- LBBB
Hypokalaemia

- ST segment depression,
  decreased T wave amplitude,
  increased U wave height.(common)

- Cardiac arrhythmias

- Prolongation of the QRS duration, increased P wave amplitude and duration
• **Hypokalaemia**
  Reduction in the Q-T interval

• **Hypocalcaemia**
  Prolongation of the Q-T interval.

• **Magnesium**
  In hypomagnesaemia, there is flattening of the T waves,
  ST segment depression, prominent U waves and, occasionally, a prolonged P-R interval occurs.

  In hypermagnesaemia, there may be a prolonged P-R interval and widened QRS complexes.
• ECG changes of hypomagnesaemia resemble that of hypokalaemia

ECG changes of hypermagnesaemia resemble that of hyperkalaemia

Hypokalaemia, hypomagnesaemia and hypercalcaemia aggravate digitalis toxicity