12-Lead ECG Interpretation: A primary care perspective

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Disclosure
• No real or potential conflict of interest to disclose.
• No off-label, experimental or investigational use of drugs or devices will be presented.

Objectives
• At the conclusion of this session the participant will:
  – Discuss 5-step approach to 12-lead ECG interpretation.
  – Analyze acute and chronic morphologic changes.
  – Determine axis with the hexaxial plot.
  – Apply 5-step analysis to case study presentation.

Outline
• Propagation of the AP
  – Normal conduction
  – Axis deviation
• 5-step approach
  – Rate, rhythm, intervals, axis, morphology
• Case study

Vector Analysis and Axis Determination
• Initiation and propagation – Sequence of cardiac activation
  – The SA node depolarizes spontaneously.
  – Atrial muscle depolarizes rapidly.
  – The wave of depolarization funnels to AV node where it is delayed.
  – Current travels to the bundle of HIS.

Vector Analysis and Axis Determination (continued)
• Initiation and propagation – Sequence of cardiac activation (cont.)
  – Current divides into right and left bundles.
  – Depolarization of interventricular septum is left to right.
  – Current moves simultaneously through the right and left bundle branches.
  – Ventricles repolarize.
Initiation and Propagation

Limb Leads

- Vector is a voltage force that has direction as well as amplitude.
- Electrical events in the heart occur in three dimensions.
- ECG paper converts those dimensions to a one dimension picture – hence 12 leads.
- Using 12 leads allows us to visualize events from the anterior, inferior, and lateral perspective.

Limb Leads (continued)

- The leads
  - Offer a lateral and inferior view
  - Axis is plotted based on the hexaxial system.
  - Find the limb lead with the voltage closest to 0.
  - Identify its right angle lead.
  - On the ECG, see if that lead is positive (+) or negative (-).

The Hexaxial Plot
Axis Determination

- Determine the corresponding direction on the hexaxial plot.
- Because the net vector is normally down and to the left, the normal axis should be in the vicinity of 60° – a range of −30° to +110° is normal.

Axis Determination (continued)

- If the axis deviates to the left of −30°, this represents a left axis deviation.
- If the axis deviates to the right of +110°, this represents a right axis deviation.

The Hexaxial Plot
The System of ECG Interpretation

- Rate
- Rhythm
- Intervals
- Axis
- Morphology

Rate

- Determine the R-R interval.
- Each large square is 0.2 seconds.
- Divide the number of large squares between R waves into 300 to determine rate.
- Normal rate is 60 to 100 bpm.

Rhythm

- Rhythm interpretation is presumed as a prereq to this program!
- The second step in 12-lead ECG assessment is identification of the rhythm, e.g., NSR, SB, ST, A-V block, atrial dysrhythmia, ventricular dysrhythmia, etc.

Intervals

- P-R interval represents A-V conduction
  - Should be 0.12 to 0.22 seconds
  - Prolonged P-R interval indicates a first degree block.
  - Shortened P-R interval indicates a junctional rhythm with retrograde conduction.

Intervals (continued)

- QRS duration represents ventricular depolarization.
  - Should be <0.12 seconds
  - Prolonged duration indicates a block in the bundle branches or a ventricular ectopic foci.
Intervals (continued)

- Q-T interval represents repolarization of the ventricle.
  - Q-T interval should be \(< \frac{1}{2} \) the R-R interval.
  - Long Q-T interval increases the risk of ventricular dysrhythmia and sudden death.

QRS Axis

- Identify the lead where the net voltage of the QRS is closest to 0.
- Look for the perpendicular lead.
- If the deflection of the perpendicular lead is +, then the axis is at the positive end of the pole.
- If the deflection of the perpendicular lead is -, then the axis is toward the negative end of the pole.

Abnormalities Caused by Drugs and Metabolic Conditions

- Sinus bradycardia
  - Beta adrenergic antagonists
  - Calcium channel antagonists
  - Digitalis
  - Adenosine
  - Hypoxemia
  - Hypothyroidism
  - Hypothermia
  - Hyperkalemia

Sinus Tachycardia

- Catecholamines
- Caffeine
- Amphetamines
- Hyperthyroidism
- Anemia
- Fever

Heart Block

- Digitalis
- Beta-adrenergic blockers
- Calcium channel blockers
- Adenosine
- Hyperkalemia
Second-degree AV Block

Atrial Flutter/Fibrillation
- Flutter
  - Hypoxemia
- Fibrillation
  - Thyroid hormone
  - Hyperthyroidism

Atrial Flutter

Atrial Fibrillation

Ventricular Fibrillation
- Most antidysrhythmic drugs
- Digoxin
- Tricyclic overdose
- Hypokalemia
- Hypomagnesemia
- Hypocalcemia

Ventricular Fibrillation (continued)
Torsade de pointe

- Class I antidysrhythmics
- Amiodarone
- Phenothiazine derivatives
- Tricyclic overdose
- Long QT syndrome

Analysis of the 12-lead ECG
Part 2

Morphologic Changes

- The V leads (V₁ to V₆), aka precordial leads, represent the anterior wall of the heart.
  - V leads may be referred to as “anterior” leads.
  - The limb leads represent the inferior and lateral walls of the heart.

<table>
<thead>
<tr>
<th>Inferior Wall</th>
<th>Lateral Wall</th>
<th>Anterior Wall</th>
</tr>
</thead>
<tbody>
<tr>
<td>II, III, aVF</td>
<td>I, aVL, (V₆)</td>
<td>V leads</td>
</tr>
</tbody>
</table>

P Wave Abnormalities

- The P wave represents atrial depolarization; an abnormal P wave would logically suggest an atrial abnormality.
- Left atrial abnormalities
  - Biphasic P wave in V₁ is most common.
    - Must be 1 x 1 mm to be significant
  - Biphasic P waves occur in conditions that increase LVEDP.
  - CHF, LVH, hypertensive heart disease can all cause this abnormality.

P Wave Abnormalities (continued)

- Broad, notched P waves in limb leads suggest left atrial dilation.
- These occur in conditions such as mitral stenosis and regurgitation.
Right Atrial Abnormalities

- P wave > 2.5 mm in any lead
- Occurs in conditions such as lung disease and pulmonary artery hypertension

QRS Abnormalities

- Right bundle branch block (RBBB)
  - QRS > 0.12 seconds
  - Remember that current normally moves left to right in the interventricular septum
    - ECG will record normal left to right activation in V1.
    - This is followed by normal LV activation.
    - Late current LV to RV results in second upward deflection in V1.
    - After RV activation, return to baseline.

QRS Abnormalities (continued)

- Remember the normal flow of current and how it reflects on an ECG.
  - ECG will record normal left to right activation in lead I – initial deflection is negative.
  - LV depolarization produces an upward deflection.
  - Late LV to RV current produces a negative deflection.
  - After RV activation, return to baseline.
Incomplete RBBB

- Usually a normal variant
- Can reflect RV hypertrophy or dilation
- Very common with atrial septal defect
- RSR pattern in V₁
- QRS is <0.12 seconds.

Left Bundle Branch Block

- Sequence is opposite RBBB
- Loss of initial normal left to right activation
- Interventricular septum is activated from right to left, causing an abnormal upward deflection in the left lateral leads.
- QRS is >0.12 seconds.
- Septum is activated from right to left, but the blocked left bundle limits the impulse.

LBBB (continued)

- Right side depolarizes first. It is thin walled, so it produces a small current.
- After RV depolarization, the current travels around to left ventricle.
- Late left depolarization produces terminal QRS force.
Fascicular Blocks

- The left bundle branch divides into two fascicles; the anterior and posterior.
- LBBB is when both fascicles are blocked; QRS is wider than 0.12 seconds.
- When only one of the fascicles is blocked, the diagnosis is either “left anterior fascicular block” or “left posterior fascicular block.”

Fascicular Blocks (continued)

- Diagnosis of fascicular block is made when there is a shift in axis.
- The QRS is not necessarily wider than normal.
- LAFB is extreme left axis deviation, at least -45° and not caused by IWMI.
- LPFB is diagnosed by right axis deviation, at least >90°, usually >110 to 120°.

The Hexaxial Plot

LAFB

LPFB
The Hexaxial Plot

Bifascicular Block
- A right bundle branch block
  - RSR pattern in V1
  - QRS>0.12 seconds
- A coincident block of either the left anterior or posterior fascicle
- AKA – a RBBB with either left or right axis deviation

Left Ventricular Hypertrophy
- When you have hypertrophy of muscle a variety of changes occur
  - The larger muscle mass produces more voltage.
  - The increased size changes axis of electrical conduction.
  - Resultant high pressure in left atria can change character of voltage movement through left atria.

Left Ventricular Hypertrophy (continued)
- Sokolow + Lyon (Am Heart J, 1949;37:161)
  - \( SV_1 + R V_5 \) or \( V_6 \) >35 mm
- Cornell criteria (Circulation, 1987;3:565-72)
  - \( SV_3 + R aVL \) >28 mm in men
  - \( SV_3 + R aVL \) >20 mm in women
Left Ventricular Hypertrophy
(continued)
• Framingham criteria
  *(Circulation, 1990; 81:815-820)*
  – R aVL >11 mm
  – R V4-6 >25 mm
  – S V1-3 >25 mm
  – S V1 or V2 + R V5 or V6 >35 mm
  – R I + S III >25 mm

Romhilt + Estes
Point Score System
(continued)
• Amplitude – any of the following=3 points
  – Largest R or S wave in any limb lead ≥20 mm
  – S wave in V1 or V2 ≥30 mm
  – R wave in V5 or V6 ≥30 mm
• ST-T strain (change in lateral leads)
  – On digitalis=1 point
  – Not on digitalis=3 points

• Left atrial abnormality= 3 points
• LAD>-30°= 2 points
• QRS duration ≥0.09 sec= 1 point
• Intrinsicoid deflection in V5 or V6 ≥0.05 sec= 1 point
5 or more points=LVH
4 points=probable LVH

Right Ventricular Hypertrophy
• Most voltage in the QRS generated by LV
• Normally QRS in right precordial leads (V1-2) is negative.
• Normally QRS in left precordial (V5-6) leads is positive.
• Transition occurs in V3-4.
RVH

• Increased RV voltage over right leads causes QRS shift to the right.
• RV strain pattern (tall R in V1 deep S in V6, ST-T changes in right precordial leads).

RVH (continued)

• Diagnostic criteria
  – R/S in V1 ≥1 or
  – R in V1 + S in V6 >10.5 mm

• Supportive criteria
  – Right axis deviation ≥110°
  – Right atrial abnormality
  – ST depression + T wave inversion in V1 or V2

Poor R Wave Progression

• In the normal ECG, the transition from negative V1-2 to positive V5-6 deflection occurs during V3-4.
• A delay or absence of this transition on ECG just means that anatomically the transition point has moved.
Causes of PRWP

- COPD
- LV dilation
- Anterior wall MI
- Misplaced precordial leads

Low QRS Voltage

- QRS amplitude <5 mm in all limb leads
- QRS amplitude in V leads usually <10 mm, but not necessary for diagnosis.

Causes of Low QRS Voltage

- Effusion
- Cardiomyopathy
- Hypothyroidism
- Obesity
- Emphysema
- Normal variant

ST-T Wave Abnormalities

- Ischemia and infarction tend to be regional events.
- Depending upon anatomy, there may be some overlap.
ST-T Wave Abnormalities (continued)

- An event in a large RCA that loops around the lateral wall might cause inferolateral ECG changes.
- An event in a large anterior descending artery that has branches to the lateral wall can cause an anterolateral event.

Arteries and Corresponding Leads

ST Segment Depression

- Stenosed artery with some retrograde flow
- \( O_2 \) demand exceeds supply
- Subendocardial ischemia
- Region of myocardium furthest from the stenosed artery is occluded
- If ischemia persists and myocardial injury occurs, a subendocardial MI occurs.
  - Later changes will show T wave inversion

Subendocardial Injury

ST Segment Elevation

- Most common cause is transmural MI.
- Affected artery is totally occluded.
- Is the primary ECG indication for thrombolytic therapy
- Prinzmetal’s angina (acute vasospasm) usually produces complete vessel occlusion.
  - Will produce ST segment elevation if ECG recorded during event
ST Segment Elevation
(continued)

• The size of the inferior and lateral MI is proportional to the sum of the elevation in the appropriate leads.
• The size of the anterior wall MI is proportionate to the number of anterior leads with elevation.
Other Causes of ST Elevation

- There are causes of ST elevation that are not specific to myocardial damage.
  - Pericarditis
  - Early repolarization

Nonspecific ST changes

- A label typically applied to ST depression that is not placed in a clinical context
- Specific ST changes
  - During exercise ECG
  - During chest pain

T Wave Inversion

- Reflects altered repolarization of ventricular muscle during ischemia/injury event
- Can reflect permanent injury with scar formation and loss of muscle; permanent atypical path of repolarization

Q Waves

- Initial negative deflection of the QRS complex
- Must be 1 mm deep and 1 mm wide to be significant
- May be normal in leads III and V₆
- A Q wave indicates transmural injury.
Atypical Situations

Lateral Wall MI
- Lateral wall sometimes called the “electrocardiographically silent” region
- Can have transmural injury of the lateral wall with few or no ST-T changes and no Q waves
- Patients with typical chest pain and enzyme elevations, but normal ECG, should be admitted.

Silent MI
- Some patients can have significant Q waves and corresponding regions of akinesis on echocardiogram.
- Most common in patients with DM and diabetic neuropathy

Pseudo MI
- Infrequently other conditions produce Q waves.
  - LVH
  - Conditions that cause PRWP
  - Hypertrophic cardiomyopathy
  - When unexplained Q waves occur, evaluate for wall motion abnormality.
WPW Syndrome

- Activation of accessory pathway results in preexcitation of the ventricle.
- Delta wave can appear to be a Q wave.
- No history of MI
- Normal echocardiogram
- Short P-R interval

The Hexaxial Plot
**Morphology**
- P wave abnormality
- Bundle branch block
  - Right or left
  - Incomplete RBBB
  - LAFB or LPFB
  - Bifascicular block

- LVH
- RVH
- PRWP
- Low QRS voltage
- ST-T abnormality
- Q wave

**Interpretation**

End of Presentation
Thank you for your time and attention.
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**References**

**References (continued)**
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Abnormalities of Rate

• Sinus bradycardia
  – Beta adrenergic antagonists
  – Calcium channel antagonists
  – Digitalis
  – Adenosine
  – Hypoxemia
  – Hypothyroidism
  – Hypothermia
  – Hyperkalemia
Sinus Tachycardia
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