12-Lead ECG Interpretation: A primary care perspective
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Disclosure
• No real or potential conflict of interest
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• 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.
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 $60^\circ$ – a range of $-30^\circ$ to $+110^\circ$ is normal.

Axis Determination (continued)

• If the axis deviates to the left of $-30^\circ$, this represents a left axis deviation.
• If the axis deviates to the right of $+110^\circ$, this represents a right axis deviation.
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.

(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 < ½ 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

Abnormalities of Rate

• 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
Atrial Flutter/Fibrillation

- Flutter
  - Hypoxemia
- Fibrillation
  - Thyroid hormone
  - Hyperthyroidism

Ventricular Fibrillation

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

Torsade de pointe

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

Analysis of the 12-lead ECG
Part 2
Morphologic Changes

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, (V₄)</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.
  - 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°.
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)
  – S V1 + R V5 or V6 > 35 mm
- Cornell criteria (Circulation, 1987;3: 565-72)
  – SV3 + R aVL > 28 mm in men
  – SV3 + 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

- 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

Romhilt + Estes
Point Score System
(continued)

- Left atrial abnormality = 3 points
- LAD > -30° = 2 points
- QRS duration ≥ 0.09 sec = 1 point
- Intrinsicsoid 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.

ST-T Wave Abnormalities (continued)

- An event in the left main artery can cause an anterolateral event.
- Global ST-T changes are more typically caused by pericarditis.

Arteries and Corresponding Leads

ST Segment Depression

- Stenosed artery with some retrograde flow
- O₂ 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

Normal conduction

Preechitation

Rate

Rhythm

Intervals
  - P-R
  - QRS
  - Q-T

Axis

Deviation?
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

References
### References (continued)


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