The Fundamentals of 12 Lead EKG

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Reviewing the Cardiac Conductive System

- SA Node
- Intranodal Pathways
- AV Junction
- AV Fibers
- Bundle of His
- Septum
- Bundle Branches
- Purkinje System

ECG Recording

J-Point

- The point marking the end of the QRS complex and the beginning of the following part that merges into the T wave in an electrocardiogram
- Generally, where the waveform begins to move more horizontal than vertical
**ECG Leads**

- ECG Leads
  - Bipolar
    - Leads I, II, and II
  - Unipolar
    - Leads aVR, aVL, and aVF
  - Precordial
    - V₁, V₂, V₃, V₄, V₅, V₆
Frontal Plane

- Leads I, II, III, aVR, aVL, and aVF
- Records electrical activity of the heart in the frontal plane of the body through the extremity leads
**Horizontal Plane Leads**

- Leads V1, V2, V3, V4, V5, and V6
- Looks at the heart on a horizontal plane

**Bipolar Leads**

- Bipolar Limb Leads
  - Lead I
  - Lead II
  - Lead III
  - Einthoven’s Triangle

**Unipolar Leads**

- Unipolar or Augmented Limb Leads
  - Lead aVR
  - Lead aVL
  - Lead aVF
Precordial Leads

- Precordial Leads
  - Lead V_1
  - Lead V_2
  - Lead V_3
  - Lead V_4
  - Lead V_5
  - Lead V_6

Electrical Axis of the Heart

- **Lead Axis**: An imaginary line from the positive electrode to the negative electrode for each lead, depicted by an arrow (vector)
- **Mean QRS Axis**: The axis of the heart as a whole; the aggregate of all the electrical vectors in the heart
- Measured in degrees
- Most 12 leads determine axis of P, QRS, and T waves

Mean QRS Axis

- To determine exact axis manually, the height of the QRS complexes can be measured and plotted on a triaxial reference system.
  - Is little use in the pre-hospital setting
- A more practical system can be used by looking at Leads I, II, and III....
Rapid Axis Determination Utilizing Leads I, II, and III

<table>
<thead>
<tr>
<th>Axis (Quick and Easy)</th>
<th>Lead 1</th>
<th>Lead 2</th>
<th>Lead 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LAD</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>RAD</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Indeterminate</td>
<td></td>
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</table>

EKG Deflections

An equiphase deflection (equally positive and negative) is produced when the lead axis is perpendicular to the heart's mean QRS axis.

The deepest negative deflection is created when the lead axis is parallel to the mean QRS axis and current is flowing away from that lead's positive electrode.

The tallest positive deflection is created when the lead axis is parallel to the mean QRS axis and current is flowing toward that lead's positive electrode.

Wave Definitions

The Normal 12-Lead (1 of 2)

- The Normal 12-Lead
  - Views the same series of electrical events from 12 perspectives.
Interpretation of 12 Lead EKGs

- 5 + 3 Approach
- Five basic steps
  - Rate
  - Rhythm
  - P-wave
  - PR Interval
  - QRS complex

Interpretation of 12 Lead EKGs

- +3
  - ST Depression
    - Present?
    - In which leads?
    - Reciprocal?
  - ST Elevation
    - Present?
    - In which leads?
    - Is there reciprocal ST depression present?
  - Q Waves
    - Present?
    - In which leads?
    - Are they pathologic or nonpathologic?
Multi-Lead Heart Assessment

12 Lead ECG Basics

Limb Leads

<table>
<thead>
<tr>
<th>Lead</th>
<th>P wave</th>
<th>QRS</th>
<th>T wave</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>upright</td>
<td>upright</td>
<td>upright</td>
</tr>
<tr>
<td>II</td>
<td>upright</td>
<td>upright</td>
<td>upright</td>
</tr>
<tr>
<td>III</td>
<td>upright</td>
<td>upright</td>
<td>upright</td>
</tr>
</tbody>
</table>

Augmented Limb Leads

<table>
<thead>
<tr>
<th>Lead</th>
<th>P wave</th>
<th>QRS</th>
<th>T wave</th>
</tr>
</thead>
<tbody>
<tr>
<td>aVR</td>
<td>negative</td>
<td>negative</td>
<td>negative /upright</td>
</tr>
<tr>
<td>aVL</td>
<td>upright</td>
<td>upright</td>
<td>upright</td>
</tr>
<tr>
<td>aVF</td>
<td>upright</td>
<td>upright</td>
<td>upright</td>
</tr>
</tbody>
</table>

Chest Leads

<table>
<thead>
<tr>
<th>Lead</th>
<th>P wave</th>
<th>QRS</th>
<th>T wave</th>
</tr>
</thead>
<tbody>
<tr>
<td>V1</td>
<td>upright/ biphasic</td>
<td>small R wave / QS</td>
<td>upright</td>
</tr>
<tr>
<td>V2</td>
<td>upright/ biphasic</td>
<td>small R wave / QS</td>
<td>upright</td>
</tr>
<tr>
<td>V3</td>
<td>upright</td>
<td>equiphase QRS - upright</td>
<td>upright</td>
</tr>
</tbody>
</table>
**ST Elevation**
- ST elevation of one millimeter or more, in at least two anatomically contiguous leads is considered presumptive evidence of an AMI
- Lateral Leads: I, aVL, V5, and V6
- Inferior Leads II, III, and aVF
- Septal Leads V1 and V2
- Anterior Leads V3 and V4

**Leads and Artery Correlation**
- Leads I, aVL, V5, and V6
  - Circumflex artery
  - Left anterior descending artery (LAD)
- Leads II, III, and aVF
  - Right coronary artery (RAC)
- Leads V1 and V2
  - Left anterior descending artery (LAD)
- Leads V5 and V6
  - Circumflex artery
  - Left anterior descending artery (LAD)

**Coronary Perfusion**

**Pathologic Q Waves**
- Also referred to as “significant” Q waves
- Defined as a width greater than or equal to 1 small box (0.04msec) or a depth greater than 1/3 of the R wave in the same lead
- Indicates irreversible tissue damage
Physiologic Q Waves
• AKA: Non-pathologic Q Waves
• Less than 0.04msec (one small box)
• Considered “Normal”

Acute Coronary Syndromes
Definition:
• Sudden ischemic disorders of the heart
• Includes unstable angina and acute myocardial infarction
• Represents a continuum of a similar disease process

Acute Coronary Syndromes
ACS refers to 3 levels of progressing cardiac disease findings:
• Myocardial Ischemia:
• Myocardial Injury:
• Myocardial Infarction

Myocardial Ischemia
• Lack of oxygen normally causing abnormalities in repolarization
• Can cause depression of the ST segment and inversion of the T wave
• If corrected, permanent damage can be avoided
Myocardial Injury

- Injury to the myocardium, typically following myocardial ischemia that results from loss of blood and oxygen supply to the tissue.
- The injured area tends to be partially or totally depolarized
- Sometimes causes ST elevation

Myocardial Infarction

- Death of the heart muscle
- Due to lack of blood and oxygen
- Location of the MI affects corresponding lead changes
- Can cause ST elevation

Acute Coronary Syndromes

- All have sudden ischemia
- Cannot be differentiated in the first hours of episode
- All have the same initiating events
  - Plaque Rupture
  - Thrombus Formation
  - Vasoconstriction

Risk Factors for ACS

- Diabetes
- Smoking
- Hypertension
- Age
- Hyperlipidemia
- Family history of CAD
- Obesity
- Stress
- Sedentary
- Non-estrogenized females
Atypical Presentations of ACS

- Pain that is sharp or intermittent
- Pain that is in the teeth, neck, shoulder, arm, or abdomen
- Mostly affects females, diabetics, and the elderly

Anginal Equivalents

- Dyspnea
- Palpitations
- Syncope or near syncope
- Generalized weakness with no history of a GI bleed or recent fever
- DKA
- May be the only signs/symptoms of ACS

Recognizing ACS

Story + Risk Factors + EKG = Treatment

STEMI and Non-STEMI

- STEMI: ST elevation MI
- Non-STEMI: non ST elevation MI
Disease Findings

- **Ischemia**
  - ST segment depression with or without T wave inversion
- **Injury**
  - ST elevation >1mm in 2 congruent leads
    - With or without loss of R wave
    - >2mm in septal leads (V1, V2)
- **Infarction**
  - Pathological Q waves
    - >.04 sec wide or 1/3 of R, with ST elevation
    - STEMI
    - Non-STEMI

Evolution of STEMI

- **STEMI**
  - Transmural infarction
    - Before coronary occlusion
    - Onset and first several hours
    - First day

  - Heart muscle normal
  - Subendocardial injury and myocardial ischemia. No cell death (infarct) yet
  - Infarction extends to epicardial surface. Subendocardial muscle dying in area of most severe injury

  - Normal ECG
  - ST segment normal or nearly normal
  - T wave peaked
  - R wave amplitude diminishing
  - ST elevation more marked

Evolution of STEMI

- **First and second days**
  - Transmural infarction nearly complete. Some ischemic and injury may be present at borders
  - Infarcted tissue replaced by fibrous scar, sometimes bulging (ventricular aneurysm)

- **After 2 or 3 days**
  - Transmural infarction complete
  - Some R wave may persist

- **After several weeks or months**
  - Transmural infarction may persist if aneurysm develops
Localization of an AMI

<table>
<thead>
<tr>
<th>Region of ST Elevation</th>
<th>Region of ST Depression</th>
<th>Leads</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anterior (V1-V4)</td>
<td>Inferior (true posterior)</td>
<td>I, V2, V3, and V4</td>
</tr>
<tr>
<td>Inferior (II, III, aVF)</td>
<td>Anterior (V1-V3 or aVL)</td>
<td>I, aVL, V5, and V6</td>
</tr>
<tr>
<td>Lateral (I, aVL, V5, V6)</td>
<td>Inferior (II, III, aVF)</td>
<td>V7 and V8</td>
</tr>
<tr>
<td>True Posterior</td>
<td>Anterior (V1-V3)</td>
<td>I and aVL (often with V6, V8)</td>
</tr>
</tbody>
</table>

Reciprocal Changes

- ST elevation may show as ST Depression in reciprocal leads and vice-versa
- **Not** necessary to presume infarction
- Strong confirming evidence when present

3 Subsets of 12 Lead EKG

- Nondiagnostic
- Suspicious for Ischemia
- Suspicious for Injury
Nondiagnostic EKG

- No ST or T wave abnormalities
- Does NOT rule out MI
- Not a candidate for reperfusion therapy

Suspicious for Ischemia EKG

- ST depression or T wave inversion
- Does NOT rule out MI
- Not a candidate for reperfusion therapy

Suspicious for Injury EKG

- Evidence of an MI
- ST elevation
- Candidate for reperfusion therapy

Evidence of an MI

- Persistent chest pain or anginal equivalents
- Risk Factors
- ST elevation of 1mm or more in two anatomically contiguous leads
Anterior Infarction

Anterior Infarction

Anterior infarct

Oclusion of proximal left anterior descending coronary artery

Anterior Leads

Anterolateral Infarct

Anterolateral Infarct

Conclusion of
left circumflex coronary artery,
marginal branch of left circumflex artery, or
diagonal branch of left anterior descending artery

Anterolateral Leads

Lateral Leads
Inferior Infarct

Inferior Infarct

Inferior Leads

Inferior Leads

Lateral Leads

Lateral Leads

True Posterior Infarct

True Posterior Infarct

Since no ECG lead reflects posterior electrical forces, changes are reciprocal of those in anterior leads. Lead V4 shows unusually large R wave (reciprocal of posterior Q wave) and upright T wave (reciprocal of posterior T wave inversion).
True Posterior Leads

- Mirror Test

Acute Anterior MI
S-T elevation, and T wave inversion in leads I, V2, V3, and V4
Reciprocal ST depression in leads III and aVF

Bundle Branch Blocks

- The Turn-Signal Rule
  - QRS > 0.12 seconds throughout the ECG.
  - Look at the QRS in V1.
  - Identify the J point.
  - Draw a horizontal line.
  - Triangle pointing up indicates RBBB.
  - Triangle pointing down indicates LBBB.

Bundle Branch Blocks

- STEMI cannot be determined in the presence of a bundle branch block
- STEMI cannot be determined if QRS > 0.012 seconds (120mSec)
- A new LBBB is just as important as a STEMI
  - Must have old tracing to diagnose a new LBBB
Right Bundle Branch Blocks

Left Bundle Branch Block

Right Bundle Branch Block

Right Ventricular Infarct

• Rare but must be managed differently
• RVI is most commonly associated with an inferior wall infarct
  – Studies range from 10% to 50%
• Right ventricle is considered to be a low-pressure volume pump
  – Contractility is dependant on diastolic pressure
Assessment of an RVI

- Distended neck veins
- Clear lung fields
- Hypotension
- ST elevation in Leads II, III, and aVF
- ST elevation in lead V4R
- These patients are very sensitive to preload reducing agents such as nitro and MS

Right Ventricular Infarct

- ST elevation in leads II, III, and aVF
- Note that the elevation is greater in lead III than lead II
  - Typical for inferior MI with RVI

Right Ventricular Infarct

- Right sided V4R showing ST elevation along with ST elevation in Leads II, III, and aVF
- When using R sided EKG or just V4R, label the EKG as “Right-Sided” or “V4R” and disregard machine’s interpretation

Management of an RVI

- Extreme caution must be used with nitro and MS
  - Use small incremental doses of MS
  - NTG best given by drip
- Fluid therapy if hypotensive
- Vasopressors if fluid is ineffective
  - Dopamine
  - Dobutamine
Practice 1

- ST Elevation in Leads I, V2 and V4 (Anterior)
- ST Elevation in Leads I and V5 (Anteriolateral)
- Reciprocal ST Depression in III and aVF
- What is the interpretation?
- ________________________________ Infarct

Practice 2

- ST Elevation in Leads V2, V3 and V4 (Anterior)
- ST Elevation in Leads V5, V6 (Lateral)
- Reciprocal ST Depression in III and aVF
- What is the interpretation?
- ________________________________ Infarct
Practice 3

- ST Elevation in leads II, III, and aVF (Inferior)
- Reciprocal ST Depression in V2 and aVL
- What is the interpretation?
- **Acute Infarct**

Key Points

- A normal 12-lead ECG does NOT rule out an MI
- You cannot see ST elevation without a 12-lead ECG
- ST elevation is presumptive evidence of an MI until proven otherwise
- Other conditions may cause ST elevation

Key Points

- Being able to pinpoint the exact location of an infarct is less important than being able to recognize that an MI is occurring
- Always maintain a high index of suspicion
- LISTEN to your patient
- Get history and risk factors
- Practice, Practice, Practice
Most Importantly......

TREAT THE PATIENT, NOT THE MONITOR