Basic 12-Lead EKG Interpretation

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OBJECTIVES

- Upon the successful completion of this course, the participant will be able to:
  - Relate the significance of the anatomy and physiology of the heart to the identification and management of myocardial infarction
  - Identify the components of a cardiac waveform and the correlation to cardiac activity
  - Demonstrate measurement of waveform duration
  - Identify different types of waveform morphology and identify the components of a 12 lead EKG and discuss the advantages over a traditional 3 lead
  - Discuss critical concepts in ACS
  - Identify the differences between a diagnostic and monitoring 12 lead

Objectives Continued

- Recognize the presence and significance of the following conditions upon obtaining and interpreting the 12 lead EKG:
  - Myocardial Infarction
  - Bundle branch blocks
  - Axis deviation
- 12 Lead EKG Mimics
  - Identify the common mimics and describe their prevalence.
  - Describe process and interpretation techniques for managing mimics.
- 12 Lead EJG Case Study
Critical Concepts in ACS

• ST elevation - the key to the acute reperfusion therapy subset
• You can’t confirm ST elevation without a 12-lead ECG
  ◦ Perform on every patient suspected of ACS
  ◦ Obtain early
  ◦ Repeat frequently

Background and Prevalence

• The Center for Disease control reported in its national vital statistics report that in 2011 there were 2,512,873 deaths in the United States. Of those 778,503 were from major cardiovascular diseases. (Hoyert & Xu, 2012) That means 32% of the deaths that occurred in 2011 were from a heart diseases related event.
• The American College of Cardiology Foundation/American Heart Association (ACCF/AHA) 2013 Guideline for the Management of ST-Elevation Myocardial Infarction states, “At present, STEMI comprises approximately 25% to 40% of MI presentations (O’gara et al., 2013, p. 6).”

Etiology

• Cardiac cells can only survive about 20 minutes without oxygen before they are completely dead. However EKG changes will occur within 30-60 seconds of hypoxia.
• After a few seconds all of the oxygen reserves in the myocardial cells are used up. The cardiac cells then turn to glycogen stores and anaerobic metabolism.

(McCance, Huether, Braskers, & Rose, 2010)
Etiology

- Risk factors that increase the risk of an acute myocardial infarction:
  - Age: anyone over 55 years old
  - Tobacco use
  - Diabetes
  - Hypertension
  - Hyperlipidemia
  - Family history of AMI
  - Obesity
  - Stress
  - Illegal drug use.


Anatomy and physiology

- Chambers
  - Atria
  - Ventricles

- Valves
  - Tricuspid
  - Pulmonary
  - Mitral
  - Aortic

- Septum

Anatomy and Physiology

- Epicardium
  - Very thin outer layer
  - Blood supply to the heart begins in this layer.

- Myocardium
  - Middle layer; majority of the blood supply for the heart is in this layer.

- Endocardium
  - The inner layer, has the least circulation because the vessels are smaller and fewer as they branch inward.
Anatomy and Physiology

- **RCA**
  - Right ventricle
  - Inferior wall of LV
  - Posterior wall of LV (75%)
  - SA Node (60%)
  - AV Node (>80%)

- **LCA**
  - Septal wall of LV
  - Anterior wall of LV
  - Lateral wall of LV
  - Posterior wall of LV (10%)

(McCance, Huether, Brashers, & Rote, 2010), picture retrieved from texasheartinstitute.org

Laboratory and Diagnostic tests

- **Laboratory**
  - Myoglobin: onset as early as 1-2 hours, peaks in 4-8 hours. Low specificity! Best to r/o AMI. Normal 0 to 85 ng/mL.
  - Troponin I: onset in 2-4 hours, peaks in 10-24 hours. Not affected by cardioversion. Also a clinical indicator of reperfusion. Normal <0.4 ng/ml.
  - CK-MB: onset in 3-4 hours, peaks in 10-24 hours. Normal 0-3 ng/ml

- Troponin is by far the most cardiac specific enzyme. "Cardiac troponin is the preferred biomarker and is more sensitive than creatine kinase isoenzyme (CK-MB). Cardiac troponins are useful in diagnosis, risk stratification, and determination of prognosis. An elevated level of troponin correlates with an increased risk of death, and greater elevations predict greater risk of adverse outcome. (O’Conner et al., 2010, p. S791)."

Laboratory and Diagnostic tests

- **Diagnostic**
  - Diagnostic cardiac catherization.
  - 12- Lead EKG
    - The gold standard in AMI diagnosis is to perform a 12-lead EKG. A 12-Lead EKG allows for the following:
      - Rapid Identification of Infarction/Injury
        - diagnosis made sooner in many cases
      - Decreased Time to Reperfusion Treatment
        - speeds preparation of & time to reperfusion therapies
      - Modification to Therapies

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Conduction Basics

HEART ANATOMY
Conduction System

- Sinus Node
  - Bachmann's Bundle
  - Internodal Pathways
- AV Junction
  - AV node
  - Bundle of His
- Ventricular System
  - Bundle Branches
  - Purkinje Fibers

EKG Leads

- EKG leads detect depolarization and repolarization as they spread through the atria and the ventricles.
- The flat line on an EKG readout is called the isoelectric line.
- All EKG leads are set up so that a depolarizing current moving towards a lead produces a deflection on the ECG paper above the isoelectric line. This is known as a positive deflection.
- A depolarizing current moving away from a lead produces a deflection below the isoelectric line. This is also known as a negative deflection.
- For repolarization, the exact opposite occurs.
The P Wave

- The P wave is the first wave of the cardiac cycle.
- It denotes the depolarization of the right and left atria. Also known as the firing of the SA node with the resulting contraction of the atria.
- So the P wave represents Atrial Systole.
- The P wave should be smooth and rounded.
- The PR interval should be less than 0.20 seconds. This is measured from the start of the P wave to the start of the QRS.
The QRS complex

- The QRS complex represents ventricular depolarization and contraction. Also known as ventricular systole.
- The Q wave is the first negative (or downward) deflection following the P wave. It represents depolarization of the interventricular septum.
- The R and S wave represent depolarization of the right and left ventricles.
- The beginning of the QRS is measured from the point where the first wave of the complex begins to deviate from baseline.
- The end of the QRS is marked at the point in which the last wave of the complex begins to level out in the isoelectric line. This is also called the J point.
- The normal QRS interval is less than .12 seconds.
- During the QRS complex atrial repolarization occurs but the atrial activity is hidden by the QRS complex.

The T Wave and ST segment

- The T wave represents ventricular repolarization.
- The ST segment is the portion between the QRS complex (J Point) and the T wave. The ST segment should be even with the isoelectric line or elevated 1mm (one small box). A deviation is bad and usually indicates infarct/ischemia.
The Basics of 12-Lead EKG principles

How to Obtain a 12-Lead EKG

- V1: fourth intercostal space to right of sternum
- V2: fourth intercostal space to left of sternum
- V3: directly between leads V2 and V4
- V4: fifth intercostal space at left midclavicular line
- V5: level with V4 at left anterior axillary line
- V6: level with V5 at left midaxillary line
Chest Lead Placement

Why does placement matter?

- As little as 2cm off can give false readings.
  - ST depression
  - ST elevation
  - Normal EKG

What do these leads represent?
Lead “Views”

EKG Leads
Limb Leads

Limb Leads
Precordial Leads

Monitoring vs Diagnostic ECGs

- Monitoring Quality ECG
  - Designed to provide information needed to determine rate and underlying rhythm
  - Designed to "filter out" artifact
    - Reduces the amount and degree of electrical activity seen by the ECG monitor

- Diagnostic Quality ECG
  - Designed to accurately reproduce QRS, ST and T waveforms
  - Designed to look more broadly at the cardiac electrical activity
  - Unfortunately, may result in greater artifact being visible
Monitoring vs Diagnostic ECGs

- Frequency Response
  - Term used to describe the breadth of the electrical spectrum viewed by the ECG monitor
  - Diagnostic quality is usually 0.05 Hz to 150 Hz
  - Monitor quality is usually 0.5 Hz to 20-50 Hz
  - Usually printed on the ECG recording strip

ECG Accuracy

- Calibration
  - Voltage measured vertically
  - Each 1 mm box = 0.1 mV
  - 1 mV = 10 mm
  - calibration standard
- Confirm calibration
  - calibration impulse should be 10 mm (2 big boxes tall)
  - stated calibration should be “x 1.0”
Calibration

ECG Accuracy

- Paper Speed
  - Standard is 25 mm/sec
    - Faster paper speed means the rhythm will appear slower and the QRS wider
    - Slower paper speed means the rhythm will appear faster and the QRS narrower

Paper Speed
How to Obtain a 12-Lead

- Check for calibration and paper speed. Calibration should be 10 mm (2 big boxes) and scaled at 1/0, and the paper speed should be 25 mm/sec.

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12-lead ECG

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12-Lead ECG

80 milliseconds = 0.08 seconds
ECG Accuracy

Look for:
- Negative aVR
  - if aVR upright, look for reversed leads
- One complete cardiac cycle in each lead
- Diagnostic frequency response
- Proper calibration
- Appropriate speed

Evolving Pattern of Myocardial Infarction

- Tall T wave
- ↑ ST segment
- ↓ T wave
- Pathologic Q wave
- EKG normalizes, Q wave remains as evidence of MI!

Pathophysiology of AMI

- Zones of Infarction, Injury, and Ischemia ("Bulls-Eye" – inner to outer circle)

  Infarction: Necrosis; Q waves on ECG
  Injury: Cells cannot fully recover; ST elevation on ECG
  Ischemia: Viable cells; T wave inversion on ECG
Waveform Components: Practice

- Find J-points and ST segments

How to Read a 12-Lead EKG

Lead Views

<table>
<thead>
<tr>
<th>LEADS</th>
<th>VIEW</th>
</tr>
</thead>
<tbody>
<tr>
<td>II, III, AVF</td>
<td>Inferior</td>
</tr>
<tr>
<td>V1, V2</td>
<td>Septal</td>
</tr>
<tr>
<td>V3, V4</td>
<td>Anterior</td>
</tr>
<tr>
<td>V5, V6, I, AVL</td>
<td>Lateral</td>
</tr>
</tbody>
</table>
How to read a 12-Lead EKG

- This is a normal 12-Lead.

Some 12 leads will have a “running” lead at the bottom.
How to read a 12-Lead EKG

- To make it easy on yourself remember this saying: “I See All Leads”. I = Inferior, S = Septal, A = anterior, and L = lateral

- Continuous Leads
How to read a 12-Lead EKG

Practice

- It is important to note that EKG changes must be present in two or more anatomically contiguous leads to be indicative of MI!

Pathophysiology of AMI

- Zones of Infarction, Injury, and Ischemia ("Bulls-Eye" – inner to outer circle)

Infarction: Necrosis; Q waves on ECG

Injury: Cells cannot fully recover; ST elevation on ECG

Ischemia: Viable cells; T wave inversion on ECG
What does that 12-lead mean to us?

- First and foremost this patient is having an active myocardial infarction and needs to get to a cardiac cath lab.
- But we also now know a lot more thanks to the 12-Lead. Let's review our anatomy.

- We know the inferior wall is being affected.
- We also know that the inferior wall is supplied by the RCA.
- So we now know that we have an occlusion in our RCA.
- Since the RCA also supplies the right ventricle, we want to be very cautious with nitroglycerin administration in an inferior wall MI. This is because the right ventricle and right side of the heart is preload dependent. Since nitroglycerin is a potent vasodilator it will cause severe hypotension if given in an inferior wall MI.

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**12 LEAD EKG INTERPRETATION**

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**Right Side ECG**

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One more practice.

SO! Did you see all of this elevation? Did this throw you off? Remember, there needs to be elevation in two anatomical continuous leads! But we see no elevation here. So no lateral wall involvement.

What does that 12-lead show us?

- This patient is obviously having an AMI.
- And this 12-lead shows us it is a very bad one and we need to hurry! But why?
  - We know we have an Anteroseptal Infarct with lateral extension.
  - That tells us that the infarct is occluding both the LAD and the circumflex, so the occlusion is likely in the LCA.
  - Knowing our anatomy that means the whole left side of the heart is ischemic and is not receiving oxygenated blood.
  - These patients are often called “widow makers” because they never make it to the hospital.
  - If you see this 12-lead this patient needs a cath lab as soon as possible!!

[McCance, Huether, Brashers, & Ross, 2010]
NOTE 1: Inferior wall supplied by either the right (85% to 90% of people) or left coronary artery.

NOTE 2: If there is acute injury in inferior leads (II, III, aVF), unknown whether left or right coronary artery is blocked.

NOTE 3: KEY — you must obtain a RIGHT-SIDED ECG at once.
Extent of Infarct

Inferior Lateral Infarction
NOTE 1: Inferior wall supplied by either the right (85% to 90% of people) or left coronary artery.

NOTE 2: If there is acute injury in inferior leads (II, III, aVF), unknown whether left or right coronary artery is blocked.

NOTE 3: KEY — you must obtain a RIGHT-SIDED ECG at once.

Posterior View of the Heart

Extends of Infarct

12 LEAD EKG INTERPRETATION
Acute Reciprocal Changes
I2 LEAD EKG INTERPRETATION
Acute Reciprocal Changes

So, what do you see here?
How about here?

And here?
Bundle Branch Blocks

Objectives
- Identify criteria for determining Left and Right Bundle Branch Blocks
- Identify criteria for determining Axis deviation
- Discuss implications for patients with BBB or Axis deviation

Why do we care?
- Delay in conduction through either right or left bundle branches
- Causes ventricular activation and depolarization to occur more slowly
- Delays in conduction exacerbate CHF and acute infarctions
- Knowing this can effect your plan of care
Right BBB
- Late depolarization of the right ventricle.
- QRS is widened greater than 0.12
- Complex will appear mainly positive with R wave appearing late
- Viewed best in lead V1
- Possible causes include CAD, heart defects, acute heart failure, MI, drug effects or electrolyte imbalances

Significance of RBBB
- May be an aid in diagnosing certain conditions
- May occur in normal hearts

RBBB
Left BBB

- Blockage of the left BB disrupts normal left to right ventricular activation
- Depolarizing impulse travels from right to left through interventricular septum
- QRS widened greater than 0.12

Significance of LBBB

- LBBB has a more serious prognosis than RBBB because of its close correlation with organic heart disease
- Indicates underlying CAD, valvular disease, HTN or MI
- Does not occur in healthy people
RBBB vs LBBB

Bundle Branch Blocks

Right bundle branch block

Left bundle branch block
RECOGNITION OF AXIS DEVIATION

Axis Deviation
- Shifting of the heart's electric axis beyond the normal range of 0 to 90 degrees
- Develops when an abnormality of the heart occurs such as hypertrophy, MI, LBBB, WPW, cardiomyopathy
- May develop in non-cardiac conditions such as emphysema, ascites, abdominal tumors, pregnancy and PE

Left Axis Deviation
- May occur in
  - Anterior Lateral MI's
  - WPW
  - Hyperkalemia
  - Emphysema
  - Blocks
  - Hypertrophy
Right Axis Deviation

- May occur with
  - Inferior MI's
  - RBBB
  - Right Ventricular hypertrophy
  - PE
  - Right axis deviation is a normal finding in children

Extreme Axis Deviation

- Caused by a more severe presentation of typical causes
- As an example a small inferior MI would produce right axis deviation, while a massive inferior MI would shift axis even further to produce extreme right axis deviation

Another Method

<table>
<thead>
<tr>
<th>Axis</th>
<th>Lead I</th>
<th>Lead II</th>
<th>Lead III</th>
<th>aVF</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal 0 to 90</td>
<td>Positive</td>
<td>Positive</td>
<td>Positive</td>
<td>Positive</td>
<td></td>
</tr>
<tr>
<td>Physiologic Left 0 to -40</td>
<td>Positive</td>
<td>Positive or split</td>
<td>Negative</td>
<td>Negative</td>
<td></td>
</tr>
<tr>
<td>Pathologic Left -40 to -90</td>
<td>Positive</td>
<td>Negative</td>
<td>Negative</td>
<td>Negative</td>
<td>Anterior hemiblock</td>
</tr>
<tr>
<td>Right 90-180</td>
<td>Negative</td>
<td>Positive, split or negative</td>
<td>Positive</td>
<td>Positive</td>
<td>Posterior hemiblock</td>
</tr>
<tr>
<td>Extreme right</td>
<td>Negative</td>
<td>Negative</td>
<td>Negative</td>
<td>Negative</td>
<td>Ventricular origin</td>
</tr>
</tbody>
</table>
What kind of block is it?

Imposters

Pericarditis

- Represents 5% of the patients that are seen in the hospital each year for chest pain.
- Causes include: Viral, bacterial, parasites, fungal, neoplastic, immune disorders, trauma, and idiopathic.
- Pericarditis should be identified by a sound assessment before it is noted on the 12-Lead.
Pericarditis Signs and Symptoms

- The classic sign of pericarditis is a pericardial friction rub that is heard over the left lower sternal border.
- Sharp, severe retrosternal chest pain that is worse in the supine position and with inspiration.
- Fever, chills, tachycardia.
- These patients will often present like an AMI patient.
- Pain is not relieved by NTG.

EKG changes in Pericarditis

- Diffuse ST Segment elevation in all leads except AVR.
- ST Segment will have a concave appearance.
- PR Depression will be present in the acute phase.

Pericarditis 12-Lead
Pericarditis 12-Lead

Pericarditis Treatment
- In most patients treatment is supportive and with NSAIDS.
- If the underlying cause has been identified and is not viral, a specific treatment regimen can be ordered.

Left Ventricular hypertrophy (LVH)
- Increase in mass of the left ventricle.
- Can be a common finding in patients with hypertension.
- Diagnosed by 12-Lead EKG or echocardiography.
- Sensitivity of ECG to diagnose LVH is 7 to 35 percent in mild LVH and only 10 to 50 percent with moderate to severe disease.
- A patient with LVH has a 40% increased risk for a major myocardial event.
Sokolow-Lyon indices

- Sum of S wave in V1 and R wave in V5 or V6 ≥ 3.5 mV (35 mm)
  and/or
- R wave in aVL ≥ 1.1 mV (11 mm)

Cornell voltage criteria

- This criteria is based off of EKG studies that are designed to detect LVH in patients.
- The only gender specific criteria.
- For men: S in V3 plus R in aVL > 2.8 mV (28 mm)
- For women: S in V3 + R in aVL > 2.0 mV (20 mm)
Cornell Voltage Criteria for 56 yo male

\[ 16 + 15 = 31 \]

Romhilt-Estes Point Score System

- This system is a points based system for specific EKG findings.
- A score of 5 or more is indicative of LVH.
- A score of 4 is a probable finding for LVH.
Left Ventricular hypertrophy (LVH)
Treatment
- Treating the patients hypertension will decrease the cardiac mass through:
  - Antihypertensive agents
  - Weight loss
  - Sodium restrictions

Hyperkalemia
- High potassium levels in the body can be caused by inadequate excretion (urinary), or excessive release of potassium from the cells.
- Clinical manifestations of hyperkalemia occur when serum potassium levels reach ≥7.0 meq/L in chronic patients or possibly at lower levels with an acute rise in serum potassium.

Hyperkalemia Clinical Manifestations
- Muscle weakness and paralysis: Ascending in nature usually starting at the feet and progressing to the trunk and arms. Respiratory weakness is rare.
- Cardiac Abnormalities/Arrhythmias: RBB, LBB, Sinus Bradycardia, Sinus Arrest, Ventricular Tachycardia, Ventricular Fibrillation, and Asystole.
Hyperkalemia Clinical Manifestations

- 12-lead EKG Changes Include:
  - Tall peaked T waves.
  - Shortened QT interval.
  - As serum potassium levels rise to dangerous levels the PR interval will lengthen, QRS will widen, and the P wave may disappear.

Hyperkalemia on a 12-lead

Hyperkalemia
Case Study

- A 47 Y/O male with no prior history of CAD developed onset of Left-sided chest pain intermittently over the last 2 months. It occurred with exertion and at rest with some bilateral hand tingling and arm pain. No nausea or vomiting, or shortness of breath. Symptoms lasted several minutes, and then self-resolved.

Case Scenario

- His symptoms worsened over the last 3 nights occurring at rest. 3 days ago he called his local EMS and had an ECG and blood pressure checked. His BP was slightly elevated and his ECG was found to be WNL. He waited until the next day to call his PCP. He was seen 2 days later.

Case Scenario

- His PCP repeated his EKG, which was normal, and started him on HCTZ and Nexium. That evening, he developed 7/10 chest pain with bilateral hand tingling and pain. He called 911 and was taken to his local ED. His ECG was repeated and looked like this…
Case Scenario

- He had received ASA 325mg and NTG SL X3 per EMS. The ED gave him Retavase, PO Plavix, NTG infusion, Zofran, Fentanyl, and Heparin bolus and infusion. He was accepted at a PPCI and flown. Upon arrival to the tertiary care facility, his ECG showed some resolution of the ST elevation, but he still had persistent chest pain. He was taken semi-urgently to the Cath Lab.
Case Scenario

- Once in the Cath Lab, it was discovered he had a Proximal LAD with 99% occlusion and the 1st diagonal branch off the LAD was 80% occluded. He received 2 stents and had his occlusions reduced to 0% and 20%, respectively. Our gentleman was discharged home from the hospital 2 days later.

What would you do?

Case Study #1

- Paramedics arrive to your ED with a 68 y/o male who c/o SSCP that awoke him from sleep 1 hour ago. The pain is still 7/10 despite 3 SL NTG and 6 mg of MS. Vital signs are: BP 88/56, HR 52, RR 28, SPO2 96% on 4 lpm NC. Skin signs are pale, cool, and clammy. He is anxious. The cardiac monitor show a SB w/o ectopy. Lungs are clear. He has 1 PIV of NS and has had 100cc PTA.
- What is your initial plan of care?
Case Study #1

- His 12-lead EKG shows this:

Case Study #1

- This is what you see on the Right Heart 12-lead:

Case Study #1

- After 500cc of NS, vitals are: BP 100/62, HR 52 SB without ectopy, RR 24, SPO2 98% on 15 lpm by NRB. CP is still 7/10.
- What is your plan of care now, given the EKG?
References

- AHA Mission Lifeline STEMI protocol.