Multi-Lead Medics™ Enhanced 12 Lead ECG Interpretation Course

Purpose of This Course
The enhanced course is designed to show acute care providers how to get extra information from a 12 or 15 lead ECG allowing greater insight into the pathophysiology behind the patient with cardiac or other problems. The course is designed for those who have a strong working knowledge and high level of comfort with the basic concepts taught in the Multi-Lead Medics™ Course. Participants should also be experienced in caring for cardiac patients in an emergency or critical care environment.

Objectives:
By the end of the course, the participant should be able to:
- Demonstrate the ability to rapidly assess a 12 lead ECG for the following information: rhythm, axis and hemiblock bundle branch block, and AMI location.
- Using a table, formula and morphology clues, determine which patients with a LBBB could be having and MI.
- Describe different criteria for determining the presence of Left and Right Ventricular Hypertrophy and the presence of strain.
- Using various criteria, identify cases of LVH and RVH on 12 lead ECG.
- Describe the clinical significance in LVH.
- Identify criteria suggestive of right and left atrial enlargement.
- Describe ECG changes associated with drug and electrolyte disturbances such as the digitalis effect, hyper and Hypokalemia, hyper and hypocalcemia, and the clinical implications of these conditions.
- Describe the potential implications of a prolonged QT interval.
- Identify ECG changes that could indicate conditions such as pericarditis, acute pulmonary embolism and early repolarization variant.
- Identify ECG changes that could indicate acute coronary syndromes such as subendocardial (non-q wave) infarction.

REVIEW of BASIC MLM CONCEPTS
1. First name the rhythm; In wide complex tachycardia, use the VT criteria.
2. Once you are certain the rhythm is not ventricular, determine the presence of axis deviation and hemiblock.
3. Next, determine the presence of bundle branch block.
4. Finally, using the ISAL method, determine the infarct location and reciprocal changes. Use the 15 lead to cover the right ventricle and posterior walls.

These 4 steps are designed to provide you with important information for the acute management of the cardiac patient. The enhanced segment of this course will present the not-so-acute and not-so-obvious aspects of 12-lead ECG interpretation.
Enhanced Participant Workbook

LBBB and Acute MI: An Enigma inside a Dilemma
In the basic MLM class you learned that an AMI is difficult to diagnose in the setting of a left bundle branch block. This meant that although a person is presenting with the signs and symptoms of an AMI, the ECG evidence is inconclusive. Therefore, treatment may be delayed until enzyme tests or an old 12 lead is found.

Facts about a Left Bundle Branch Block:
- Higher mortality than RBBB or no BBB
- Most often seen in large anterior MI’s
- They have lower ejection fractions (< 50% when QRS is >170ms)
- They have higher left ventricular end diastolic pressures (LVEDP)
- They have poorer global LV function
- ISIS II showed AMI with LBBB had higher mortality when not treated with a thrombolytic agent.
- FTT trial showed mortality within 6 hours of symptom onset significantly reduced in patients receiving thrombolytic.

Remember How You Found the LBBB?

- Lead V1 (MCL-1)
- QRS is > 120ms
- Circle the J point
- Draw a line into the complex
- Go down with the terminal deflection
- Arrow points down, turn LEFT (LBBB)

LBBB had ST elevation as a normal finding because of late repolarization of the left ventricle that did not allow the return to isoelectric.

For this reason, the AMI could hide behind the Left Bundle Branch Block. Many researchers have been looking into this “Enigma” Several criteria have been mentioned as an aid in determining the presence of a MI in the presence of a left bundle branch block. It should be noted again that no ECG criterion is foolproof, ECG criteria are not a substitute for clinical assessment and history taking skills.

Easiest of Criteria for AMI in LBBB

Perhaps the easiest criterion for this condition is a new onset of left bundle branch block along with signs and symptoms of an AMI. This will generally point to the conclusion that it is an AMI. Of course, one would have to have an old 12 lead that demonstrated that there was not a LBBB before this episode. In the hospital, medical records may have the chart available for this. However, in the out of hospital setting, that is not usually the case. At any rate, the prehospital
provider should supply the receiving hospital with the information necessary for a rapid search of the records to find the file. In some cases, paramedics have received a return call to the ambulance by an ER physician, who found the records, and diagnosed the MI based on new onset. This heads up action will speed up the time to intervention with aspirin and heparin, and reduce door to drug or door to balloon time.

**ECG Criteria for AMI in LBBB**

**Changes in QRS configuration:** Think: **Q-R-S**

- **Q** waves seen in at least two lateral leads (I, aVL, V5, V6)

![ECG waveforms](image)

- **R** wave Regression seen from leads V1-V4:

![ECG waveforms](image)
Another Way to Determine the Likelihood AMI in LBBB and Chest Pain

Sgarbossa, Pinski, Barbagetata, et al developed this simple table: The evidence came about during the GUSTO-1 trial.

Basically, The interpreter is to determine 3 things looking at an ECG with a LBBB.

1. **Is there ST segment elevation >= 1mm and is concordant with QRS axis?** This means that the elevation is in the same direction as the QRS deflection.

If this criterion is met, it is worth 5 points. This is the most heavily weighted criterion of the three that are listed.
2. \textit{Is there ST Segment Depression} $\geq 1$ \textit{mm in V1, V2, or V3?} This simple criterion can be seen in either of these leads. Remember to find the J point when looking for the point of ST depression.

If this criterion is met, it is worth 3 points.

3. \textit{Is there ST Segment elevation} $\geq 5$ \textit{mm discordant from the QRS axis?} This means that the ST elevation goes up and the QRS deflection is down.

This criterion is worth 2 points if present.

There are various combinations that can be present. It is important that you use the chart to help you determine the likelihood of an AMI.

Furthermore, the chart has relatively high reliability as ECG goes. As long as the score total is at least 3, the sensitivity is 78%. This means that 22% would be missed or would not meet the criteria, but still be having an AMI. The specificity is 90%. In other words, if the score totals at least 3, then 90 out of 100 would have accuracy that the table suggests.

This is a very promising method that is easy to use. In light of current practice that renders an ECG with a LBBB non-diagnostic, this new development brings new hope.
Chamber Enlargement

Objectives: By the end of this section you will be able to:
1. Identify by criteria, evidence of right and left atrial enlargement.
2. Identify by criteria, evidence of right and left ventricular hypertrophy and the presence of strain pattern.
3. Describe the clinical implications of atrial and ventricular enlargement.

Atrial Abnormalities

We all learned in basic ECG classes that sinus “p” waves were upright, round and uniform in shape in lead II. In fact, this is the governing criteria for the title “Sinus” rhythm, or “Sinus” tachycardia, etc. However, the more ECG’s we look at, the more we realize that not all “p” waves look nice and rounded, or even “normal” for that matter. What is the meaning of all this? Is there an easy way of determining why a “p” wave is shaped the way it is? This section will present some simple criteria for the determination of Right and Left atrial enlargement.

Normal “P” Waves

- ECG Recognition: P wave is rounded and less than 3mm in height and less than 120ms wide. It is upright in lead II, III and AVF, and Upright, negative, or biphasic in Lead V1 (MCL-1)

Right Atrial Abnormality (Enlargement) RAE

- ECG Recognition: Tall, Pointed “P” waves in the inferior leads II, III, or AVF. The P wave will be more than 2.5mm (.25mV)

Since the right side of the heart is the pulmonary side, the 3 P’s have been used to describe RAE: P pointed, P prominent, P pulmonary or “P-Pulmonale”.

Causes of RAE:
- Congenital heart disease
- Tricuspid or pulmonary valve disease
- Pulmonary hypertension (any cause)
Clinical Implications:
- Generally not an acute problem.
- Frequently seen with Right Ventricular Hypertrophy
- Can be seen with other criteria pointing to other more severe problems, i.e. pulmonary embolism

Left Atrial Abnormality (Enlargement) LAE

- ECG Recognition:
  - Lead II: Widened (>120ms or 3mm) P wave with a notched or “m” shaped appearance.
  - Lead V1 or MCL-1: A broad, terminal negative “p’ deflection of more than 1mm or one small square.

It has been suggested that the criteria demonstrate a conduction abnormality and not necessarily atrial enlargement. It could also mean left atrial dilation due to increased pressure or volume overload.

Causes of LAA:
- Hypertension
- Pulmonary edema
- Mitral or aortic valve stenosis
- LVH
- AMI

Clinical Implications:
- See causes
- No treatment of the specific problem
- Can give clues as to overall patient hemodynamics

Ventricular Hypertrophy

Right Ventricular Hypertrophy

Increased pressure or volume in the right ventricle causes RVH. Generally occurring in similar circumstances as mentioned earlier in RAE. It has large forces that go away from the lateral leads and towards lead V1 (MCL-1).
ECG Criteria:
- RAE
- Narrow QRS
- Right Axis Deviation
- R wave height in V1 (MCL-1) is ≥ 7mm
- Asymmetrical downsloping ST segment (strain) in inferior leads.

Clinical Concerns with RVH:
- Not an acute problem (no specific treatment).
- Can be confused with a posterior hemiblock.
- Remember the hemodynamics that caused it.

Left Ventricular Hypertrophy (LVH)

Increased pressure or volume in the left ventricle usually causes Left Ventricular Hypertrophy. It is often found in mitral or aortic stenosis, hypertension, AMI, cardiomyopathy or ischemic heart disease.

Experts disagree as to the accuracy of ECG criteria for diagnosing LVH. Some say that ECG criteria cannot be used with accuracy, while some point out that if the criteria show it is present, then it is. This appears to be the classic “Specificity vs Sensitivity” issue. This being said, we will explore the measurement criteria.

ECG Criteria:
- LAE (This plus any other QRS voltage criteria is diagnostic)
- QRS is generally narrow or slightly widened with “strain”
- Axis is usually normal, although can be physiologic left as it progresses. Some sources say LVH is the cause for an axis that is -15 degrees or more.

Voltage Criteria: (several)

- “Rule of 35"
  - Measure the deepest S wave (in mm from the isoelectric line to the nadir) from either lead V1 or V2; add this number to the tallest r wave of lead V5 or V6. If this number is greater than 35, and the patient is at least 35 years old, then Voltage criteria is met.
Voltage Criteria (con't)

- R wave in Lead aVL is > 11mm
- R wave > 20mm in any inferior lead (II, III, aVF)
- R wave > 20mm in lead V6
- R wave > 25mm in V5
- S waves > 25 mm in leads V1 or V2

With so many measurements staring me in the face, do we need to measure all to determine if LVH exists? The answer is no. If the complexes look large, assume hypertrophy. Apply whichever criteria you need to make the call. To confirm LVH, the clinician should look for evidence of strain, the hallmark of hypertrophy. A strain pattern to hypertrophy is like reciprocal changes are to ST elevation; they both clinch the diagnosis.

Defining Strain:

If you look at many machine generated analysis, a common message printed is “voltage criteria for LVH met” means that at least one of the above (or other) criteria has been met. When the analysis reads LVH with “repolarization abnormality” this is another way of indicating “strain”.

Strain is best seen on the ECG in the lateral or inferior leads. (II, III, AVF, V5 or V6). Strain is evidenced by asymmetrical ST depression and T wave inversion that almost looks biphasic.

Clinical Concerns with LVH

- Patients with LVH have a higher incidence of sudden death and ischemic arrhythmias
- It can mimic the ST depression or elevation seen with myocardial ischemia.
- It may be caused by an AMI
- In the presence of a LBBB, LVH criteria are not determined.
- Maybe a useful clue as to hemodynamic condition.

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Drug and Electrolyte Disturbances

Electrolytes are extremely important to the body's electrical system. It is electrolytes that are responsible for the changes in polarity in your heart that are picked up by the ECG machine. Most criteria for interpretation is based on the assumption of homeostasis, that is, the electrolytes are in their normal ranges for that person.

Certain electrolytes, namely potassium and calcium, have strong influences over the cardiac cycle. Abnormalities sometimes maybe seen on the ECG. It is important to note that an ECG is not a substitute for blood studies to determine the serum level. These criteria are designed as guidelines to aid in discovering possibly covert conditions.

Potassium (K+)

Major benefits of Potassium
- Prevents action potential from being too short (QT interval),
- Allows for organized fast heart rates,
- Protects from excitability,
- Slows the heart rate in vagal conditions

Hypokalemia (General Information)
- Serum levels below 3.5-5 mEq/L
- Most common causes include vomiting, diarrhea, diuretics, gastric suctioning
- Hypomagnesemia (*low magnesium is a cause with the same ECG characteristics)
- Muscles weakness and polyuria are common signs and symptoms
- Digitalis can take advantage of hypokalemia, causing serious dysrhythmias (Torsades de pointes)
- Atrial Flutter, heart blocks and bradycardia are common as well.
Hypokalemia (ECG Changes)
- ST segment depression
- T waves flatten or join with “U” waves
- U waves get larger than the T as K+ falls
- QT interval appears to lengthen as T combines with U
- PR Interval increases

What to do about Hypokalemia
- Monitor ECG
- Increase dietary intake of potassium – bananas?
- In severe cases, IV KCL 20 mEq/hour

Hyperkalemia (General Information)
- Serum levels above the normal range
- Most common cause is renal failure
- Sinus node can quit at 7.5 mEq/L
- VF or asystole at 10 - 12 mEq/L

Hyperkalemia (ECG Clues)
- Mild cases (<6.5 mEq/L)
  - Tall tented “peaked” T waves with a narrow base (QTc is still normal)
  - Best seen in leads II, III, V2 and V4
  - Normal P waves
- Moderate cases (less than 8 mEq/L)
  - QRS widens
  - Broad S wave in V leads
  - Left axis deviation
  - ST segment is gone, contiguous with the “peaked” T wave
  - P wave changes, starts to go
- Severe cases (more than 8mEq/L)
  - P waves disappear
  - Sine waves
What to do about Hyperkalemia

- Consider IV Calcium Gluconate 10% 10-30ml IV over 1-5 minutes
- Consider Glucose 10% 200-500ml in 30 min and 500 – 1000ml over the next several hours
- Sodium Bicarb 100-150 mEq added to 1 liter bag of NS

**Calcium**

Hypercalcemia: ECG Clue

- Shortened QT interval (QTc for heart rate)

Hypocalcemia: ECG Clue

- Prolonged QT Interval (QTc for heart rate)

**The Effects of Drugs**

**The QT Interval**

The QT interval represents the time from the start of depolarization of the ventricles to the end of repolarization. This is in effect, the time of the refractory period.
Measurement

The QT interval is measured from the start of the QRS complex to the end of the T wave. This distance is done for the clinician on the “vital signs” part of the 12 lead. Note on the 12 lead the QT/QTc interval. The QT is the actual measurement. The QTc represents the “corrected” QT interval for the current heart rate. To determine whether a QT is prolonged or not, refer to the QTc chart below.

Another “Rule of thumb” is sometimes used to determine if the QT is prolonged: If the QT is more than half the R-R, then it is considered to be prolonged. This concept works only if the heart rate is between 60 & 100.

The QT interval does not include the U wave (if present). If there is a U wave, the measurement ends before the U wave starts. Also, if the QRS complex is more than 80 ms, this excess must be subtracted from the QT measurement. QTc takes this into consideration.

Causes of Prolonged QT Interval
- Hypokalemia, Hypocalcemia
- Drugs: quinidine, amiodarone, procainamide, tricyclics, disopyramide, phenothiazines
- Liquid protein diets, myocarditis, AMI, LVH, hypothermia

Causes of Shortened QT Interval
- Hypercalcemia, Digitalis therapy

Where the QT interval matters
- Prolonged QT could lead to Torsades de Pointes, an ominous polymorphic, potentially lethal form of Ventricular Tachycardia

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The Digitalis Effect
About 60% of those on Digitalis will present with the "Dig" effect. On the ECG, this manifests itself as ST segment depression with a “scooped out” appearance to the ST segment. Best seen in the inferior and lateral leads.

Other Conditions with ECG criteria

Pericarditis
Pericarditis is inflammation of the pericardium. Pericarditis usually occurs in younger patients without cardiac risk factors.

Signs and Symptoms of Pericarditis
- Chest pain, dyspnea, tachycardia, FEVER, weakness, chills
- Chest Pain can be sharp and severe, and radiate to back, neck, jaw
- Made worse by LYNIG FLAT. Better by SITTING UP.
- Often pleuritic pain, made worse by breathing
- Pain will last for hours and days
- Pericardial friction rub, heard along lower left sternal border

ECG Clues for Pericarditis: (90% have ECG evidence)
- ST segment elevation
- Concave (curved up) in almost all leads (except aVR, V1)
- T wave elevation (starts above the isoelectric line)
- ST segment depression or T wave inversion
- Almost all leads down (later stage)
Pericarditis Diagnosis
1. Physical Criteria (Chest pain, pleuritic, relieving factors)
2. No response to nitroglycerin
3. Pericardial rub
4. ECG changes that do not localize an artery (everything up!)

Early Repolarization Variant
This condition can occur in young healthy individuals. It is characterized by a notched “J” point and concave upward ST Elevation. This condition is best seen in the inferior and lateral leads (II, III, aVF, V4, V5, V6).

Acute Pulmonary Embolism
An acute pulmonary embolism is a diagnosis of exception. There are no unique signs and symptoms to the condition. The best clues for finding an acute pulmonary embolism include history of recent surgery or anticoagulant drugs in a patient with chronic atrial fibrillation.

There is also ECG changes that can occur with a large embolism. ECG evidence must be considered only with a complete history and physical of the patient. Alone, these criteria are inconclusive.

- Sinus Tachycardia
- Right atrial enlargement
- Right axis deviation
- Right bundle branch block (possible)
- Deep S wave in lead I
- Abnormal Q waves in lead III
  “S1, Q3, T3” pattern
Finally, the Non Specific ST-T Wave Changes
Over the course of two courses, we have looked at many 12 leads and many forms and shapes of ECG complexes. We have come close to identifying and explaining nearly every abnormality. However, there still remains some that defy explanation or identification. These are known as non-specific ST-T wave changes.

Flattened T waves, biphasic T waves, and unrelated ST depression are just a few of those abnormalities that are classified as non-specific changes. It is important to note that thankfully, not many of these have cardiac causes.
Acute Coronary Syndromes

Every now and then, a new “buzz phrase” enters our world. The new “buzz phrase” that is going around now is Acute Coronary Syndromes or ACS. This phrase is to remind us that not all patients with chest pain and suggestive histories are having a transmural infarction.

Traditionally, we have referred to these as the 3 I’s of infarction. The three I’s were ischemia, Injury and Infarction. Based on ECG evidence, the evidence starts with the inverted T wave ischemia and ends with Q waves. The technical truth is not all MI’s will develop Q waves, but will end with damaged tissue. These infarctions that occur without Q waves have been called “non-q wave” infarctions.

Similarly, we have looked to ST elevation as the hallmark for treatment of the MI. ST elevation is translated into transmural (“across all of the wall”) injury, that is, full thickness injury. These types can and usually do end up with Q waves.

A smaller subset of infarctions can occur to the subendocardial wall. Since only one layer of the heart is involved, ECG signals are often not strong enough to produce the forces of ST elevation. Instead, they produce ST segment depression. Subendocardial infarcts have been discovered after enzyme tests for years. As we have waited for ST segment elevation to occur. In our race to discover the larger, more deadly, and more salvageable, transmural infarcts, the subendocardial variety was given less attention. Granted, the transmural does pose greater mortality and morbidity, yet if recognized early, subendocardial infarcts can also benefit from early treatment.
ECG Findings in Non-Q wave (subendocardial) infarctions

- ST segment depression in the indicative leads
- ST segment elevation in Lead aVR ONLY.

In the event of a transmural infarction with ST elevation, it is virtually impossible to see a subendocardial on the same ECG. This is because the forces of the transmural event are much stronger than the forces of subendocardial injury. This is similar to why you do not see the atria repolarization during the ventricular depolarization (QRS) on a 12 lead.
PUTTING THIS ALL TOGETHER (Everything)

You have learned a great deal about 12 Lead ECG Interpretation in a short amount of time. It will be very difficult to memorize everything and be able to do this unaided without much, much practice. The basic 12-lead course was designed to allow you to rapidly assess a 12 lead for vital information needed to treat the patient now.

The information from this Enhanced course will be helpful to look for when you have time, or the information is critical to treatment. In other words, this is nice to know stuff but not vitally critical to treat the acute patient.

Steps to use

<table>
<thead>
<tr>
<th>STEP</th>
<th>RATIONALE</th>
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<tbody>
<tr>
<td>1. Analyze Rhythm</td>
<td>Decide on VT or Life Threats Use the VT algorithm</td>
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<tr>
<td>2. Determine Axis and hemiblocks</td>
<td>Risk for CHB, drug contraindications</td>
</tr>
<tr>
<td>3. Determine Bundle Branch Block</td>
<td>Risk for CHB, drug contraindications</td>
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<tr>
<td>4. ISAL – Look for ST Elevation</td>
<td>Locate the AMI Use the Acute MI locator</td>
</tr>
<tr>
<td>5. 15 lead ECG – V4R, V8, V9</td>
<td>If 12 lead normal/inferior MI Or ST depression in V1 – V4</td>
</tr>
<tr>
<td>6. LBBB and AMI</td>
<td>Early recognition is good Early intervention is better</td>
</tr>
<tr>
<td>7. Subendocardial Injury?</td>
<td>ST depression only with ST elevation in lead aVR only</td>
</tr>
<tr>
<td>8. Chamber enlargement</td>
<td>If large complexes seen use “35” If not measure aVL or other criteria</td>
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<tr>
<td>9. QTc interval</td>
<td>If you are going to give antiarrhythmics, diuretics, stimulants</td>
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<tr>
<td>10. PRN: Pericarditis, Early Repolarization</td>
<td>If needed, you can apply these criteria when needed.</td>
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Handy Cutouts 2

### Acute MI Locator ST↑elevation

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<th>Location</th>
<th>Leads</th>
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<tr>
<td>Inferior (RCA)</td>
<td>II,III,F</td>
<td>I, AVL</td>
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<tr>
<td>Septal (LAD)</td>
<td>V1,V2</td>
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<tr>
<td>Anterior (LAD)</td>
<td>V3,V4</td>
<td>II,III,F</td>
</tr>
<tr>
<td>Lateral (CIRC)</td>
<td>V5,V6,II,III,F</td>
<td>I, L</td>
</tr>
<tr>
<td>Posterior (RCA)</td>
<td>V8,V9</td>
<td>ST↓</td>
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<tr>
<td>Right Vent. (RCA)</td>
<td>V4R</td>
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### QTc Interval Table

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### Key 3 Criteria for VT

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<tr>
<td>“BIG mountain, little mountain “steeple” or “fireman’s hat”</td>
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<tr>
<td>Upright V1 (MCL-1)</td>
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<td>Fat “R” wave</td>
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<td>Notch or slur to downstroke</td>
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<tr>
<td>Negative V1 (MCL-1)</td>
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<td>Fat “R” wave</td>
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<tr>
<td>Notch or slur to downstroke</td>
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<tr>
<td>V-6 (MCL-6)</td>
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<td>Any negative complex</td>
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Criteria for Left Ventricular Hypertrophy

| Rule of 35’s deepest of lead V1 or lead V2 plus the tallest of leads V5 or V6. | If > 35 mm and the patient is older than 35, Then LVH voltage met |
| Lead aVL r wave is > 11mm tall | Voltage Criteria for LVH present |
| R wave > 20mm in any inferior lead | II, III, or AVF |
| R wave > 20mm in lead V6 | |
| R wave > 25mm in V5 | |
| S wave > 25mm in V1 or V2 | |

Axis

| Normal Axis | 0 - 90 |
| Physiologic Left Axis | 0 to -40 |
| Pathological Left Axis | -40 to -90 |
| Right Axis | 90 - 180 |
| Extreme Right Axis | no man's land |

| Lead I | Lead II | Lead III | Comments |
| -- | -- | -- | |
| -- | -- | -- | Anterior Hemiblock |
| -- | -- | -- | Posterior Hemiblock |
| -- | -- | -- | Ventricular in origin |
Analyze rhythm

Wide complex tachycardia?

Identify rhythm

Axis and Hemiblock

QRS > 120ms?

Bundle Branch Block

LBBB

RBBB

Acute MI Locator

"I SEE ALL LEADS"

LBBB and AMI Criteria

15 Lead ECG Criteria

Begin Clinical Interventions for Acute Myocardial Infarction if present.

Comprehensive Assessment

Subendocardial Injury?

Chamber Enlargement

QT/QTc Interval

PRN Conditions

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