

Foundations EKG I - Unit 1 Summary

The accurate diagnosis of ST elevation myocardial infarction (STEMI) is one of the most time critical duties in the practice of EM. Diagnosis is not always easy so guidelines are very helpful. The most recent consensus on STEMI criteria are the following:

> 1mm of new STE above the J-point in two contiguous leads (except V2-3)

V2-3 Specifics

Men over 40 years old: > 2mm of new STE above the J-point

Men under 40 years old: > 2.5mm of new STE above the J-point

Women: > 1.5mm of new STE above the J-point

Posterior Lead (V7-9) Specifics

Men over 40 and Women: > 0.5mm of new STE above the J-point

Men under 40: > 1mm of new STE above the J-point

Right Sided Lead (V3R & V4R) Specifics

Men over 30 and Women: > 0.5mm of new STE above the J-point

Men under 30: > 1mm of new STE above the J-point

Left Bundle Branch Block

The presence of a LBBB complicates the interpretation of STEMI and requires application of the Sgarbossa and Modified Sgarbossa criteria that are well described by this CORE EM [blog post](https://coreem.net/core/stemi-lbbb/) (https://coreem.net/core/stemi-lbbb/)

Identifying the J-Point & Measuring ST Elevation

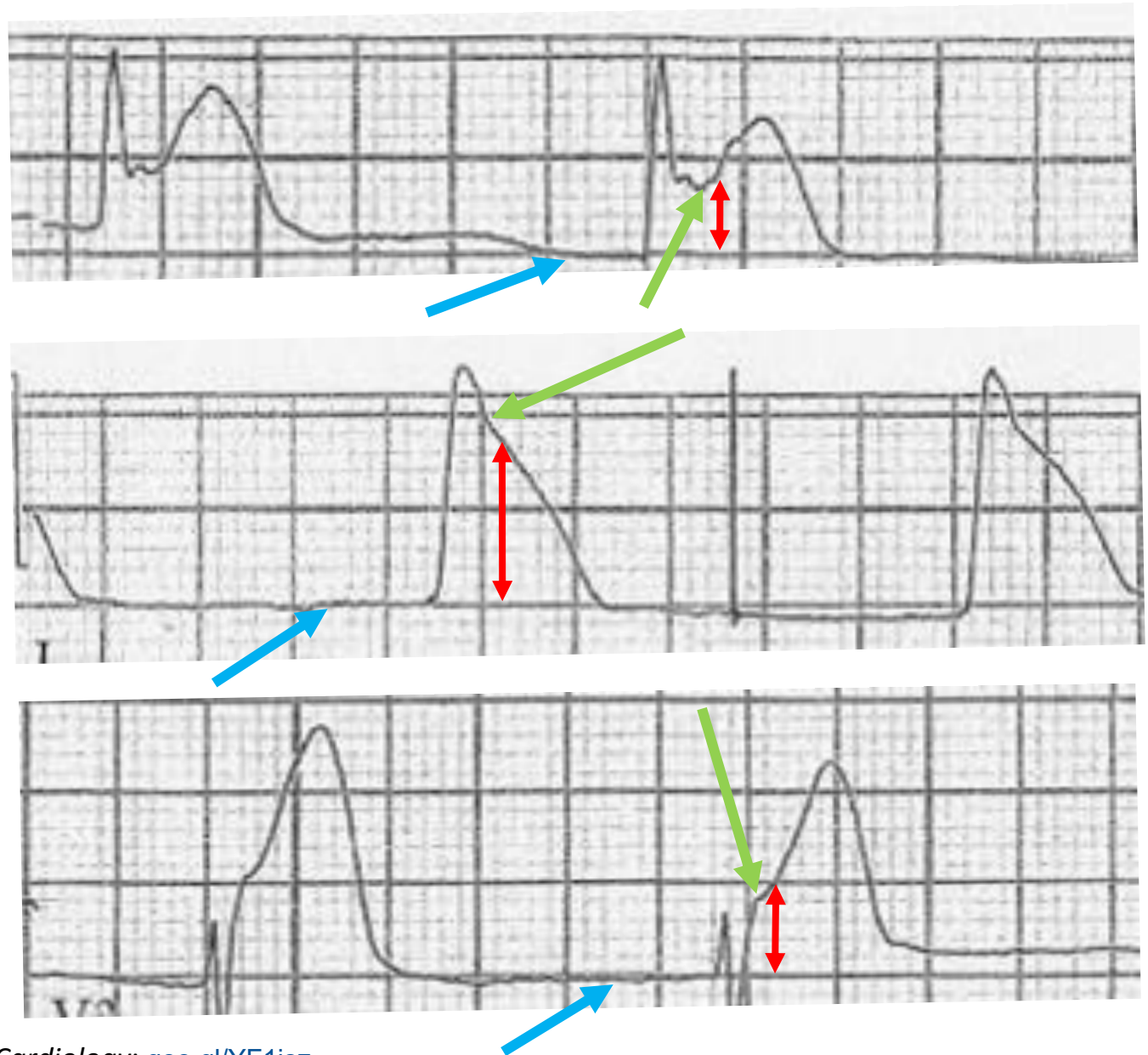
The **J point** marks the start of ventricular repolarization at the end of the QRS complex and the beginning of the ST Segment.

It is the landmark that guides ST elevation (STE) measurement.

The morphologic abnormalities associated with STEMI can make it difficult to identify the J-point. On the right are several examples with the **J-point (Green →)** highlighted.

Measure STE from isoelectric line to the point 0.04s (1 small box) after the J point. The **isoelectric line** is best defined as the segment between the T and P waves (aka TP segment) as the heart is electrically silent. The **isoelectric line (Blue →)** and **STE (Red →)** are highlighted.

A great resource for this topic is Dr. Venkatesan's blog *Expressions in Cardiology*: goo.gl/YF1isz



Localization of Myocardial Infarction

When the STEMI guidelines refer to contiguous leads they are referring to the territorial regions of the heart that the leads generally represent. For example, any combination of pathologic STE in at least 2 leads that are right next to each other (i.e. contiguous) in the septal/anterior/lateral leads V1/V2/V3/V4/V5/V6 would rule in STEMI but STE in V1 & V6 would not because they are not contiguous. Along the same lines, STE in II & III would rule in STEMI but I and aVR would not. Finally, any combination of II, III, and aVF with pathologic STE would rule in STEMI. For a deeper review of contiguous and reciprocal leads please see this post by Tom Boughillet of EMS 12 Lead—goo.gl/3zgZGu.

I—Lateral/High Lateral	aVR—only Right Facing Lead	V1—Septal	V4—Anterior
II—Inferior	aVL—Lateral/High Lateral	V2—Septal	V5—Lateral
III—Inferior	aVF—Inferior	V3—Anterior	V6—Lateral

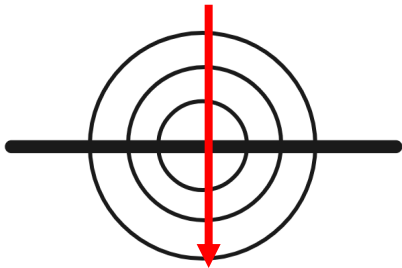
Localization of Myocardial Infarction Continued

The 12 lead EKG is a great tool however it has some blind spots. In particular, because there are normally no posterior leads placed so a **posterior STEMI will not cause any STE on a standard EKG but should cause ST depression in V-3**. That STD is commonly referred to as a **reciprocal change**. The causes of reciprocal change beyond the scope of this summary but you can imagine that they are the mirror image of the STEMI on the opposite side of the heart. This table shows typical reciprocal change patterns. It is important to realize that reciprocal changes are **not always present in STEMI** and also that reciprocal changes **may be earliest sign of STEMI!**

Infarction Distribution	ST Elevation	ST Depression (Reciprocal Change)
Anterior Wall	V1—V4, I, aVL	Not always present but II, III, & aVF m
Lateral Wall	I, aVL, V5, V6	V1 Also Right sided leads
Inferior Wall	II, III, aVF	V1—V4, I, aVL
Right Ventricle	III > II and usually V1 > V2 (V2R, V3R, V4R)	V2
Posterior Wall	Posterior Leads V7, V8, V9	V1-V3 (often tall R waves)

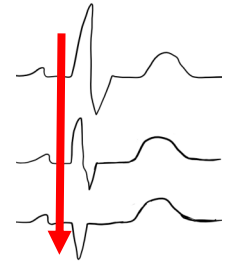
Other EKG Changes Suggestive of Ischemia

Although ST Elevation is likely the best known EKG change associated with ischemia there are many overt and subtle changes:

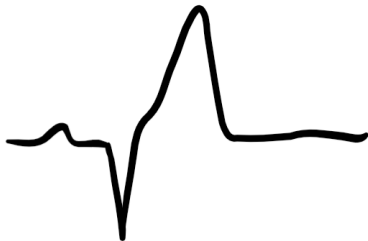


T Wave Inversion (TWI)—The best way to think about TWI is a loss of T wave amplitude (height above the isoelectric line) because before a T Wave becomes inverted it first loses amplitude until it is flat and then finally inverts. Loss of amplitude and flattening can represent early ischemia. Finally, don't fall into the trap of attributing localization to T waves because, unlike ST elevation, T wave changes do not localize.

R Wave Amplitude—Loss of R wave amplitude should be concerning for active ischemia or prior myocardial infarction in the right clinical scenario. In fact, a complete loss of R wave amplitude results in a Q wave. This finding can certainly be confounded by lead placement, effusion, or a change in habitus however so it is always reasonable to repeat the EKG.



ST Depression—Like ST elevation ST depression (STD) is best measured from the isoelectric line. Occasionally, STD is very significant and easy to measure but much more frequently is very minimal and sometimes is even difficult to measure. Avoid the temptation to ignore subtle ST depression. Finally, like T wave changes don't forget that STD doesn't localize.



Hyperacute T Waves—T waves that are **much** larger than their respective R waves (particularly when they are preceded by Q wave or near Q waves) should be very concerning for ischemia. Early repolarization can be difficult to distinguish from hyperacute T waves so a good history and comparison to old EKGs if available is critical.

Ischemia is usually associated with a **prolonged QT interval** and patient's are more likely to be **bradycardic** than tachycardic. These changes should only trigger suspicion and are neither sensitive or specific for ischemia.