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/)
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
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](https://goo.gl/3zgZGu).

### Localization of Myocardial Infarction

<table>
<thead>
<tr>
<th>Lead</th>
<th>Location</th>
<th>Corresponding V Leads</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Lateral/High Lateral</td>
<td>aVR—only Right Facing Lead</td>
</tr>
<tr>
<td>II</td>
<td>Inferior</td>
<td>aVL—Lateral/High Lateral</td>
</tr>
<tr>
<td>III</td>
<td>Inferior</td>
<td>aVF—Inferior</td>
</tr>
<tr>
<td>V1</td>
<td>Septal</td>
<td></td>
</tr>
<tr>
<td>V2</td>
<td>Septal</td>
<td></td>
</tr>
<tr>
<td>V3</td>
<td>Anterior</td>
<td></td>
</tr>
<tr>
<td>V4</td>
<td>Anterior</td>
<td></td>
</tr>
<tr>
<td>V5</td>
<td>Lateral</td>
<td></td>
</tr>
<tr>
<td>V6</td>
<td>Lateral</td>
<td></td>
</tr>
</tbody>
</table>
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!

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

Created by William Burns, MD    
Edited by Nick Hartman, MD & Kristen Grabow Moore, MD MEd
EKG interpretation is a difficult skill that requires deliberate practice to gain mastery. A formulaic method for interpretation minimizes missed diagnoses and provides a strategy for dealing with EKGs when the diagnosis is not immediately apparent. Below are two example interpretation strategies

**Rule of Fours**

*Four Initial Features:*
- History/Clinical Picture
- Rate
- Rhythm
- Axis

*Four Waves:*
- P Waves
- Q/R/S Waves
- T Waves
- U Waves

*Four Intervals/Segments:*
- PR Interval
- QRS Width
- ST Segment
- QT Interval

**“Standard”**

- Rate
- Rhythm
- Axis
- Intervals
- Hypertrophy
- Ischemia

Rule of Fours adapted from Gerard Fennessy (@doctorgerard) and *Life in the Fast Lane*

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Example Case

39yF with no PMHx with chest pain.

HR: 95   BP: 160/110   RR: 18   O2 Sat: 96%
Four Initial Features

**History/Clinical Picture**—39 year old woman with no known comorbidities or risk factors

**Rate**

Option 1: Count each QRS complex in rhythm strip and multiply by 6 (EKG is 10 sec. long). Ex: 12 x 6 = 72.

Option 2: “Rule of 300.” For regular (and only regular) rhythms count large boxes between QRS complexes and estimate.

1 Box = 300/1 = 300 bpm
2 Boxes = 300/2 = 150 bpm
3 Boxes = 300/3 = 100 bpm
4 Boxes = 300/4 = 75 bpm
5 Boxes = 300/5 = 60 bpm
6 Boxes = 300/6 = 50 bpm
7 Boxes = 300/7 = 43 bpm
8 Boxes = 300/8 = 37 bpm

**Rhythm**—Normal Sinus Rhythm is defined by morphologically identical P waves with a constant PR interval before every QRS.
Axis—Modern EKG machines are generally quite good at determining the axis value but it is still important to know how axis deviation is defined. Machine read was 50 on this EKG.

Axis can be often be manually determined by evaluating whether leads I & aVF are positive, equiphasic or negative. However when the axis is unclear, like the difference between pathologic and physiologic left axis deviation the tie breaker is lead II.

<table>
<thead>
<tr>
<th>LEAD I</th>
<th>LEAD II</th>
<th>LEAD III or aVF</th>
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<tbody>
<tr>
<td>POSITIVE</td>
<td>EQUIPHASIC</td>
<td>POSITIVE</td>
</tr>
<tr>
<td>POSITIVE</td>
<td>EQUIPHASIC</td>
<td>NEGATIVE</td>
</tr>
<tr>
<td>POSITIVE</td>
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<td>POSITIVE</td>
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Images courtesy of Life in the Fast Lane Creative Commons License
Four Waves (or complexes)

P Waves

Are P waves present? Yes, P waves are present.

Morphology—do all P waves look the same or do they vary? All look the same.

Are the P waves normal—do they look enlarged (>1.5mm tall in V1-6 or >2.5mm in any other lead) or are they peaked? Yes, they look normal.

Q, R, S Waves

Low voltage R waves? (V1-6 R waves are all less than 10mm tall or I/II/III R waves are all less than 5mm tall)? No

High voltage R waves (are the R waves in V1-6 excessively tall)? No

T Waves

Inversion (normally inverted in V1 and aVR, can be inverted in III if QRS complexes is negative)? No.

Peaked/Hyperacute? No

Flattened? No

U Waves (small deflection after the T wave)

Present? No
Four Intervals (or segments)

**PR Interval**

Machine read was 178 on this EKG.

Normal PR? Yes, normal range is 120-200ms.

**QRS Complexes**

Normal width (70-100ms)? Yes, the QRS complexes are 94 ms

Critical diagnoses that affect QRS morphology (Brugada, WPW, new bundle branch block, TCA Overdose)? No.

**ST Segments**

Any ST elevation? No.

Any ST depression? No.

**QT Interval**

Machine read was 424 on this EKG.

Normal QTc? Yes, normal upper limit is 440ms for men and 460ms for women.
57 y/o M with PMH of HL, DM2 c/o palpitations and SOB for the past 72 hours. Sent to the ED from PMD’s office.

<table>
<thead>
<tr>
<th>History/Clinical Picture</th>
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<tbody>
<tr>
<td>Rate</td>
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<tr>
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What is your interpretation of the EKG?

<table>
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<tr>
<th>What are the critical actions for this patient once the EKG diagnosis has been made and your clinical assessment is completed?</th>
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<table>
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<tr>
<th>What EKG finding is most sensitive for ongoing ischemia?</th>
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</table>
What is your interpretation of the EKG?

History/Clinical Picture

Rate

Rhythm

Axis

P Waves

Q/R/S Waves

T Waves

U Waves

PR Interval

QRS Width

ST Segment

QT Interval

What 2 conditions are frequently associated with this EKG pattern and how frequently do they occur? What EKG pattern is frequently associated? How can you confirm?

What medication is relatively contraindicated with this EKG pattern? Why?
Triage EKG—Unit 1, Case 2
53 y/o F with history of smoking and hyperlipidemia with chest pain for the past 2 hours.

HR: 95        BP: 130/110
RR: 18        O2 Sat: 96%

What is your interpretation of the EKG?

History/Clinical Picture
Rate
Rhythm
Axis

P Waves
Q/R/S Waves
T Waves
U Waves

PR Interval
QRS Width
ST Segment
QT Interval

What changes on the standard EKG are suggestive of this diagnosis?

Where should you place leads to get the 2nd EKG?

Adapted from http://www.wikilectures.eu/index.php/ECG_Leads

Resources Links:  Life in the Fast Lane

Dr. Steve Smith’s Blog
Repeat EKG—Unit 1, Case 3

Courtesy of Edward Burns of *Life in the Fast Lane*

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67yM with history of CAD, DM2, HTN c/o crushing CP & diaphoresis for 45 minutes. No old EKG but he states his doctor told him to tell others MDs that he has a LBBB.

**What is your interpretation of the EKG?**

- History/Clinical Picture
- Rate
- Rhythm
- Axis
- P Waves
- Q/R/S Waves
- T Waves
- U Waves
- PR Interval
- QRS Width
- ST Segment
- QT Interval

**How relevant is a prior LBBB to the diagnosis of STEMI?**

- What criteria should be used when evaluating this EKG for signs of ischemia?