7.5: Diagnosis of a STEMI

The diagnosis of STEMI is made predominantly using the 12-lead ECG and cardiac enzymes. There is significant myocardial necrosis occurring in the setting of STEMI, resulting in elevation of the cardiac enzymes.

Cardiac Enzymes

Also known as cardiac biomarkers these include myoglobin, troponin and creatine kinase (Figure 7.4). Historically, lactate dehydrogenase, or LDH, was also used but is nonspecific. Cardiac enzymes are released into the circulation when myocardial necrosis occurs, as seen in MI.

Figure 7.4
Myoglobin

Myoglobin is released into circulation with any damage to muscle tissue, including myocardial necrosis. Because skeletal muscle contains myoglobin, this measurement is quite nonspecific for MIs. The benefit is in the fact that a detectable increase is seen only 30 minutes after injury occurs, unlike troponin and creatine kinase, which can take 3 to 4 hours.

Troponin

The enzymes troponin I and troponin T are normal proteins important in the contractile apparatus of the cardiac myocyte. They are released into the circulation about 3 to 4 hours after MI and are still detectable for 10 days afterwards. The long half-life allows for the late diagnosis of MI but makes it difficult to detect re-infarction as can occur in acute stent thrombosis after PCI. Although, there are a number causes for troponin elevation unrelated to MI, troponin elevation is much more sensitive and specific than myoglobin and even CK.

Creatine Kinase

Creatine kinase — also known as creatine phosphokinase, or CPK — is a muscle enzyme that exists as isoenzymes. The MB type is specific to myocardial cells, whereas MM and BB are specific to skeletal muscle and brain tissue, respectively. The CK level will increase approximately 3 to 4 hours after a MI and stays elevated for 3 to 4 days. This makes it useful for the detection of re-infarction in the 4- to 10-day window of time after the initial insult compared with troponin, which remains elevated for 10 days and is less useful for this purpose.

Changes in EKG

ST segment elevation can take many forms during STEMI. The first change ECG change during STEMI is “hyperacute T waves” (Figure 7.5) that appear peaked and are related to localized hyperkalemia that arises as myocytes lyse. These changes are rarely seen as they are transient and frequently occur prior to hospital arrival. ST segment elevation, unlike depression, will localize to the ECG lead of the affected myocardium.

![Figure 7.5](https://med.libretexts.org/Under_Construction/VirginiaTech/Book%3A_Cardiovascular_Pathophysiology_(Binks)/07%3A_Isch...)

Obviously, which leads show the ST elevation will demonstrate the location of the infarcted tissue and provide insight into which coronary vessel is affected. How the leads of a 12-lead EKG relate to the coronary vessels is summarized in
Figure 7.5. The following looks at the characteristic EKG changes in relation to location in a bit more detail (tip: relate back to Figure 7.5 as you read the next sections).

![EKG diagram showing lead locations and changes](https://med.libretexts.org/Under_Construction/VirginiaTech/Book%3A_Cardiovascular_Pathophysiology_(Binks)/07%3A_Ischemia%20%26%20Arrhythmia/7.05 антиперентальная%20исхемия.jpg)

Figure 7.6 Which leads look at which coronary vessels? LCx = left circumflex, LAD = left anterior descending, RCA = right coronary.

### Anterior Wall Myocardial Infarctions (AWMI)

An anterior wall myocardial infarction occurs when anterior myocardial tissue usually supplied by the left anterior descending coronary artery suffers injury due to lack of blood supply. When an AWMI extends to the septal and lateral regions as well, the culprit lesion is usually more proximal in the LAD or even in the left main coronary artery. This large anterior myocardial infarction is termed an extensive anterior.

The ECG findings of an acute AWMI include:

1. **ST segment elevation** in the anterior leads (V3 and V4) at the J point and sometimes in the septal or lateral leads, depending on the extent of the MI. This ST segment elevation is concave downward and frequently overwhelms the T wave. This is called “tombstoning” for obvious reasons (Figure 7.7); the shape is similar to that of a tombstone.

2. **Reciprocal ST segment depression** in the inferior leads (II, III and aVF). See the full 12-lead ECG in Figure 7.8 for an example.

![ST elevation and J point](https://med.libretexts.org/Under_Construction/VirginiaTech/Book%3A_Cardiovascular_Pathophysiology_(Binks)/07%3A_Ischemia%20%26%20Arrhythmia/7.05 антиперентальная%20исхемия.jpg)

Figure 7.7
Inferior Wall Myocardial Infarction (IWMI)

An inferior wall myocardial infarction occurs when inferior myocardial tissue supplied by the right coronary artery, or RCA, is injured due to thrombosis of that vessel. When an inferior MI extends to posterior regions as well, an associated posterior wall MI may occur. The ECG findings of an acute inferior myocardial infarction (Figure 7.9) include the following:

1. ST segment elevation in the inferior leads (II, III and aVF)
2. Reciprocal ST segment depression in the lateral and/or high lateral leads (I, aVL, V5 and V6)

If the reciprocal ST segment depressions are not present, alternative causes of ST segment elevation, such as pericarditis should be considered.

An inferior MI can have multiple potential complications including cardiogenic shock, atrioventricular block or ventricular fibrillation and can be fatal.

Posterior Wall Myocardial Infarction

The ECG findings of a posterior wall myocardial infarction are different than the typical ST segment elevation seen in other myocardial infarctions. A posterior wall MI occurs when posterior myocardial tissue (now termed inferobasilar), usually supplied by the posterior descending artery —a branch of the right coronary artery in 80% of individuals — acutely loses blood supply due to intracoronary thrombosis in that vessel. This frequently coincides with an inferior wall MI due to the shared blood supply.
The ECG findings of an acute posterior wall MI (Figure 7.11) include the following:

1. ST segment depression (not elevation) in the septal and anterior precordial leads (V1-V4). This occurs because these ECG leads will see the MI backwards; the leads are placed anteriorly, but the myocardial injury is posterior.
2. A R/S wave ratio greater than 1 in leads V1 or V2 (Figure 7.10).
3. ST elevation in the posterior leads of a posterior ECG (leads V7-V9). Suspicion for a posterior MI must remain high, especially if inferior ST segment elevation is also present.
4. ST segment elevation in the inferior leads (II, III and aVF) if an inferior MI is also