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# Myocardial infarction on ECG

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Last month, we discussed the topic of myocardial ischemia. *Acute myocardial infarction* (MI) may occur either as a progression or end-point of MI or coronary spasm or, most commonly, as a result of sudden rupture of a thrombotic plaque. All causes of MI result in complete occlusion of one or more coronary arteries supplying certain areas of the myocardium. As an example, the *left anterior descending artery* (LAD) typically supplies the anterior wall and septum.<sup>1</sup>

Acute MI has the same symptoms as myocardial ischemia; however, the angina is usually more prolonged and is often not relieved by rest in acute MI. Initial treatment consists of oxygen, nitroglycerin, morphine, aspirin, and beta-blocker therapy.<sup>2</sup> Definitive treatment following these interventions requires a strategy for reperfusion of the blocked coronary artery. This is most commonly done with angioplasty in centers with a cardiac catheterization laboratory. In centers without these capabilities, pharmacologic reperfusion is an option.

There are two types of acute myocardial infarctions: *ST elevation MI (STEMI)* and *non-ST elevation MI (NSTEMI)*. STEMI means that the ECG shows ST elevation in two contiguous leads. This ST elevation is all that is needed to diagnose STEMI, although cardiac markers including troponin are usually also elevated. In NSTEMI, there is elevation of the cardiac markers without ST elevation.

Another type of MI is the *old MI* or *MI of undetermined age*. This is diagnosed by identifying pathologic Q waves on ECG. These Q waves signify a completed MI at some point in the past. Criteria for pathologic Q waves include a duration of longer than 4 milliseconds (1 small box) and a Q wave depth of at least 1/3 the height of the corresponding R wave.<sup>1</sup>

Using the ECG, we can classify the MI in terms of location by determining which leads the ST elevation appears in. The leads along with their locations are shown below.

Combinations of these are also possible; for example, an *inferolateral MI* involves leads II, III, AVF, V5, and V6.

Anterior Wall	Leads V1, V2, V3, V4, V5, and V6 (V1 and V2 also known as septal leads)
Inferior Wall	Leads II, III, and AVF
Lateral Wall	Leads I, AVL, V5, and V6
Posterior Wall	There are no leads overlying the posterior wall on a 12 lead ECG. Look for ST depression and T wave inversion especially in leads V1 and V2-V6.

## ECG Challenge

A 57-year-old, 100 pack-year male smoker with hypertension presents to the ED with a 2-hour history of midsternal chest pain. About an hour ago, the pain was relieved with rest. Now, it has been constant for 30 minutes despite rest. The following ECG is obtained from the patient:



### Stepwise approach:

1. Is the ECG **regular**? Yes. The QRS complexes march out and are consistent.
2. What is the **rate**? Find a QRS complex on or near a dark line. A) Counting the large boxes, we see that there are a little more than 4 large boxes, which suggests a heart rate of just less than 75 or about 70 bpm. B) There are about 7 QRS complexes in six seconds (30 large boxes), which estimates the rate at  $7 \times 10 = 70$  bpm.
3. There is a **P wave** for every QRS complex.
4. The **PR interval** is normal.
5. The **QRS complex** is normal.
6. There is **ST segment** elevation present in leads II, III, and aVF, suggesting an acute inferior wall myocardial infarction.
7. There are **no U waves**.

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### REFERENCES

1. Thaler M. *The Only EKG Book You'll Ever Need*. 6th ed. New York, NY: Lippincott Williams & Wilkins; 2010:216,227.
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