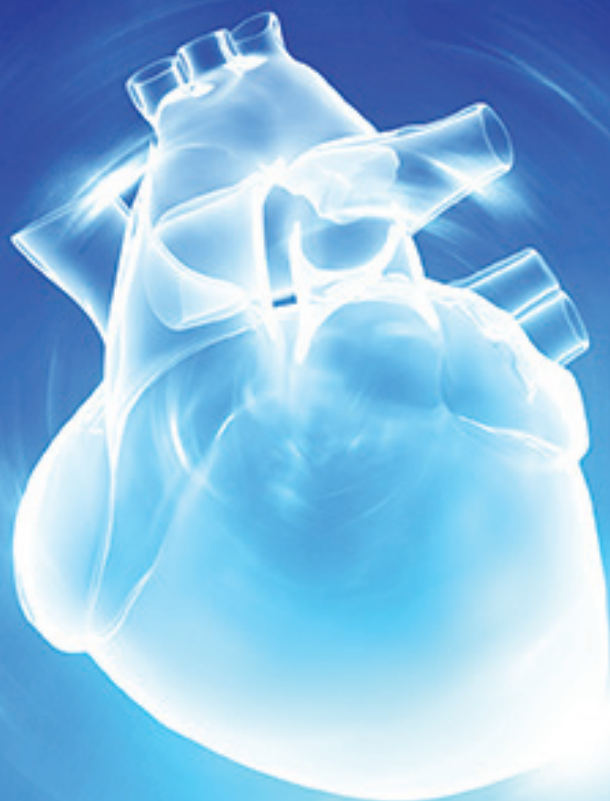


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# Introduction

By Michael Crawford, MD, Editor

The accurate diagnosis and appropriate treatment of acute myocardial infarction continues to be of critical importance to health professionals who care for such patients in the emergency department (ED), outpatient clinics, and hospital wards. Timely diagnosis and treatment of these acute coronary syndromes (ACS) may save patients' lives, preserve left ventricular function, return them to an active lifestyle, and prevent future events. Also, alleged mismanagement of such patients is a frequent cause of litigation. The cornerstones of the diagnosis of ACS are the history, physical examination, electrocardiogram (ECG), and serum troponin. However, these basic steps do not always provide sufficient diagnostic clarity to determine a management strategy. Therefore, increasingly, cardiac imaging is being used, often in the ED, to clarify the picture. In 2015, a multi-society document was published that discussed the appropriate use of imaging in ED patients with chest pain. In *STEMI Watch*, we provide the two key tables that outline their recommendations for patients with suspected ACS (see *Tables 1*

and 2). When appropriate cardiac imaging is added to the basic diagnostic approach, a definitive diagnosis can be made in almost all such cases.

Although there are several guidelines published by various societies on the diagnosis and management of suspected ACS patients, their recommendations can be out of date or controversial based on new knowledge collected since they were published. This book is a compilation of the latest literature on this topic. It includes summaries and discussions of key scientific papers and new educational material. Because of the important initial triage function of the ECG, we include several examples of challenging ECGs with a discussion of their interpretation. The book is divided into five sections: diagnosis, ED management, hospital management, complications, and post-hospital management. The relevant scientific studies are referenced, so further reading on a topic can be easily accomplished. We hope this book sharpens your diagnostic and therapeutic skills in this area, and enhances the care of your ACS patients. ■

**Table 1. Suspected Non–ST-Segment Elevation ACS: Early Assessment Pathway Based on Initial ECG, Biomarker Analysis, and Symptoms**

Indication	Echocardiography Rest	CMR Rest	SPECT Rest	CCTA	CCath
Positive initial diagnosis of NSTEMI/ACS 3. Initial ECG and/or biomarker analysis unequivocally positive for ischemia	R	R	R	R	A
Equivocal initial diagnosis of NSTEMI/ACS 4. Equivocal initial troponin or single troponin elevation without additional evidence of ACS 5. Ischemic symptoms resolved hours before testing	M*	M*	A	A	R
Low/intermediate likelihood initial diagnosis of NSTEMI/ACS 6. TIMI risk score = 0, early hsTrop negative	R	M	M*	A	R
7. Normal or nonischemic on initial ECG, normal initial troponin	R	R	R	A	R
<p>Appropriate use key: A = appropriate; M = may be appropriate with rating panel consensus; M* = may be appropriate as determined by lack of consensus by rating panel; R = rarely appropriate.</p> <p>ACS, acute coronary syndrome; CCath, catheter-based coronary angiography; CCTA, coronary CT angiography; CMR, cardiovascular MR; ECG, electrocardiography; hsTrop, high-sensitivity troponin T; NSTEMI, non–ST-segment elevation myocardial infarction; SPECT, single-photon emission computed tomography; TIMI, Thrombolysis in Myocardial Infarction.</p> <p>Reprinted with permission from: 2015 ACR/ACC/AHA/AATS/ACEP/ASNC/NASCI/SAEM/SCCT/SCMR/SCPC/SNMMI/STR/STS Appropriate Utilization of Cardiovascular Imaging in Emergency Department Patients With Chest Pain : A Joint Document of the American College of Radiology Appropriateness Criteria Committee and the American College of Cardiology Appropriate Use Criteria Task Force. Table 2.1: Suspected Non–ST-Segment Elevation ACS: Early Assessment Pathway Based on Initial ECG, Biomarker Analysis, and Symptoms.</p>					

**Table 2. Suspected Non–ST-Segment Elevation ACS: Observational Pathway—After Assessment of Serial Cardiac Troponin**

Indication	Exercise ECG	Echocardiography		CMR		SPECT/PET		CCTA	CCath
		Rest	Stress/Rest	Rest	Stress/Rest	Rest	Stress/Rest		
8. Diagnosis unequivocally positive for NSTEMI/A	M*	M*	M*	M*	M*	M*	M*	M*	A
Serial troponins or ECG not positive for NSTEMI/ACS 9. Serial ECG and troponins negative for NSTEMI/ACS	A	R	A	R	A	R	A	A	R
10. Serial ECG or troponins borderline for NSTEMI/ACS	M*	M*	A	R	A	R	A	A	M*
<p>Appropriate use key: A = appropriate; M* = may be appropriate as determined by lack of consensus by rating panel; R = rarely appropriate.</p> <p>ACS, acute coronary syndrome; CCath, catheter-based coronary angiography; CCTA, coronary CT angiography; CMR, cardiovascular MR; ECG, electrocardiography; NSTEMI, non–ST-segment elevation myocardial infarction; SPECT, single-photon emission computed tomography.</p> <p>Reprinted with permission from: 2015 ACR/ACC/AHA/AATS/ACEP/ASNC/NASCI/SAEM/SCCT/SCMR/SCPC/SNMMI/STR/STS Appropriate Utilization of Cardiovascular Imaging in Emergency Department Patients With Chest Pain : A Joint Document of the American College of Radiology Appropriateness Criteria Committee and the American College of Cardiology Appropriate Use Criteria Task Force. Table 2.2: Suspected Non–ST-Segment Elevation ACS: Observational Pathway—After Assessment of Serial Cardiac Troponin.</p>									



# Module 1: Diagnosis

(2.0 credit hours)

# Accuracy of ECG Localization of the Culprit Artery in STEMI

By Michael H. Crawford, MD, Editor

**SOURCE:** Noriega FJ, et al. Influence of the extent of coronary atherosclerotic disease on ST-segment changes induced by ST elevation myocardial infarction. *Am J Cardiol* 2014;113:757-764.

Early ECGs are the mainstay of predicting the culprit coronary artery in ST-segment elevation myocardial infarction (STEMI). However, in patients with multivessel coronary artery disease (CAD), other significant lesions may affect the accuracy of culprit artery prediction. Thus, these investigators from Spain reviewed the clinical records, ECGs, and acute angiograms of 289 patients with STEMI. Patients with left bundle branch block or ventricular-paced rhythm were excluded. On angiography, single vessel disease occurred in 149 patients (51%) and multivessel disease in 140. The patients were divided into three groups based on their culprit artery: 140 left anterior descending (LAD), 118 right coronary artery (RCA), and 31 left circumflex (LCx). With LAD occlusion, the overall pattern of ST segment changes was the same for single vessel and multivessel disease. However, only proximal LAD occlusion exhibited reciprocal ST depression in the inferior leads. With RCA occlusion, the ST segment pattern was similar for single and multivessel disease and included reciprocal ST depression in leads I, aVL, and V2, but in those with multivessel disease, ST depression often extended to leads V3-4. LCx occlusion resulted in ST elevation in the inferior leads and often V6 and reciprocal changes in V2-3 or even V4 in multivessel disease. In fact, ST elevation in V6 was highly predictive of LCx infarction. The authors concluded that patients with either single vessel or multivessel CAD have similar coronary artery-related ST changes on the admission ECG and that reciprocal changes in LAD occlusion patients predict a proximal culprit lesion.

## ■ COMMENTARY

Controversy has surrounded the interpretation of reciprocal ST segment changes on admission ECGs in patients with clear STEMI. Three major hypotheses have been advanced to explain them. The first is mirror ECG changes in opposing leads. This electrical phenomenon has been confirmed in animal studies where a single coronary is ligated and is supported by their study since reciprocal changes were present in both single vessel and multivessel disease patients in equal numbers in all three territories. Second is that reciprocal changes represent the ischemic zone around a large infarct that extends into adjacent leads. This study supports this hypothesis, especially in the LAD territory where proximal lesions exhibited reciprocal changes and more distal lesions did not. Presumably the proximal lesions subtended a larger infarct. Finally, there is the hypothesis of ischemia at a distance. This theory supposes that the occlusion of an artery may render an adjacent area with a significant coronary lesion ischemic when collaterals from the culprit vessel are lost. The data in this study did not support this theory as a major mechanism, but cannot totally exclude it either, since the authors did not perform this level of analysis. However, the investigators concluded that reciprocal changes do not require the presence of multivessel disease.

Also of interest is that this study has added another criterion for distinguishing RCA from LCx occlusion as a cause of inferior lead ST elevation. ST elevation in V6 was 71% sensitive and 83% specific for LCx occlusion. Three other criteria have been proposed in previous studies that demonstrated similar accuracy: 1) ST elevation in lead III > II suggests RCA occlusion; 2) ST depression in lead I supports RCA occlusion; and 3) ST depression in leads V1, V2, and V3 summed and divided by the ST elevation summed in leads II, III, and aVF is < 1, or the simpler ST depression in lead V3 divided by the ST elevation in lead III < 1 supports an RCA lesion. ■

# Chest Pain and an Anterior Culprit

By Ken Grauer, MD

Professor Emeritus in Family Medicine, College of Medicine, University of Florida, Gainesville

*Dr. Grauer is the sole proprietor of KG-EKG Press, and publisher of an ECG pocket brain book.*

## SCENARIO

The ECG in Figure 1 was obtained from a patient with new-onset chest pain. What is the likely “culprit” artery? Is this patient a good candidate for acute reperfusion?

## INTERPRETATION

The rhythm is sinus at about 60/minute. There is an obvious acute ST-elevation myocardial infarction (STEMI) in progress — with marked ST elevation across the precordial leads. The shape of this ST elevation is coved (convex-down), in association with “hyperacute” (broad and peaked) T waves. Factors suggesting that this patient is an ideal candidate for acute reperfusion include: 1) large extent of anterior involvement with marked ST elevation in multiple leads; 2) new-onset chest pain by history, in association with reciprocal inferior ST depression — both suggesting an early stage in evolution; and 3) no precordial lead Q waves have yet formed.

STEMIs are almost always associated with acute occlusion of a major coronary artery — called the “culprit” artery. Evaluation of the initial ECG obtained at the onset

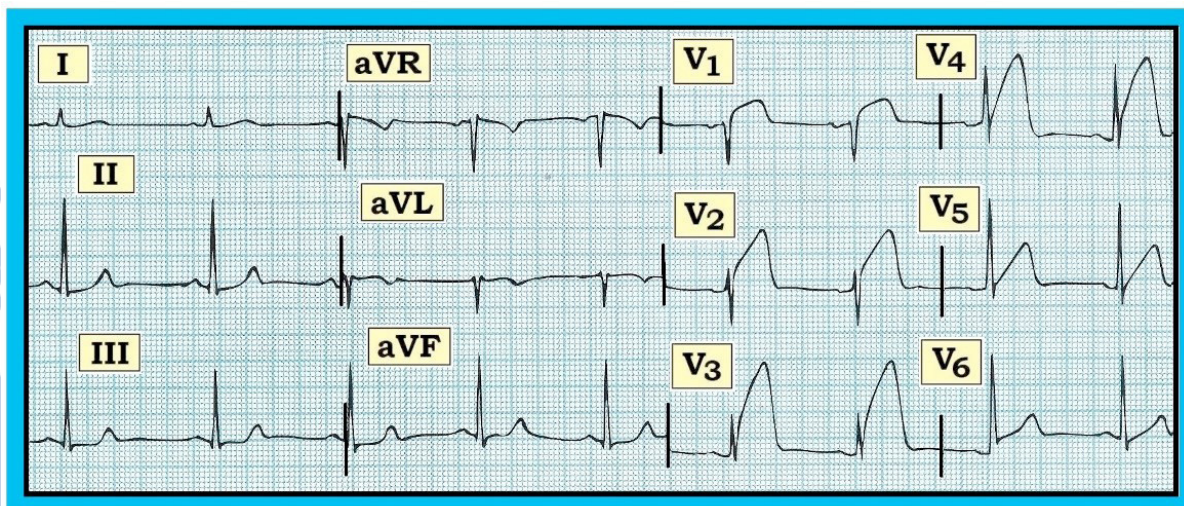
of chest pain may provide insight to the probable site of acute occlusion. Awareness of where to look facilitates the angiographer’s task during cardiac catheterization, which may expedite angioplasty and stent placement if acute reperfusion is attempted. Exclusive ST elevation in an anterolateral lead distribution (leads V1-through-V5 in Figure 1) localizes the occlusion to the left coronary system.

There are several reasons why the “culprit” lesion in this case is unlikely to be the left main coronary artery. First, acute occlusion of the left main is rarely seen in practice, because this lesion most often leads to rapid demise of the patient. In addition, ST elevation is usually prominent in lead aVR with left main occlusion, whereas no more than minimal ST elevation is seen in lead aVR in Figure 1.

Instead, the “culprit” lesion in this case is most likely to be at a proximal site in the left anterior descending (LAD) coronary artery. Factors in favor of a proximal LAD location include: 1) ST elevation that is most marked in leads V2 through V4; 2) prominent ST elevation already in lead V1 compared to no more than minimal ST elevation in lead aVR; and 3) reciprocal ST depression in the inferior leads.

A more distal site of occlusion in the LAD is less likely in this case because: 1) more distal LAD occlusions rarely manifest such prominent ST elevation in lead V1 and V2, and 2) reciprocal ST depression may be absent in the inferior leads with more distal LAD occlusion, and some inferior ST depression appears here. ■

Figure 1. ECG from a patient with new-onset chest pain.





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## MODULE 1: DIAGNOSIS

### CME/CE OBJECTIVES

At the conclusion of this activity, participants should be able to:

1. Discuss current scientific research and data regarding the diagnosis of ST segment elevation myocardial infarction (STEMI).
2. Discuss the pathogenesis and treatment of STEMI.
3. Cite new information regarding new drugs for STEMI and new uses for traditional drugs.
4. Identify ancillary issues of importance for health-care providers who treat STEMI patients.
5. Discuss advances in STEMI treatment.

### CME/CE INSTRUCTIONS

To earn credit for this activity, follow these instructions:

1. Read and study the activity, using the provided references for further research.
2. Log on to AHCMedia.com and click on My Account. First-time users will have to register on the site using the 8-digit subscriber number printed on the mailing label or invoice.
3. Once logged in, click on the “Tests Available to Take” tab and locate the *STEMI Watch* tests. Test questions for each chapter are included in separate tests.
4. Pass each online test with a score of 100%; you will be allowed to answer the questions as many times as needed to achieve a score of 100%.
5. After successfully completing the last section of the test, your browser will be automatically directed to the activity evaluation form, which you will submit online.
6. Once the completed evaluation is received, a credit letter will be emailed to you instantly. You can also navigate to “Tests Taken + Credit Letters” and download the credit letter.

1. **Which of the following supports a proximal LAD STEMI?**
  - a. ST depression in leads II, III, aVF
  - b. ST depression in I and aVL
  - c. ST elevation in V6
  - d. ST elevation in aVR
2. **According to Simon Mahler, MD, MS, FACEP, the United States spends somewhere between how much per year on workups for chest pain patients?**
  - a. \$10 million to \$13 million
  - b. \$13 million to \$20 million
  - c. \$5 billion to \$7 billion
  - d. \$10 billion to \$13 billion
3. **Mahler also explains that to improve the sensitivity of the HEART score, investigators added what to the HEART Pathway protocol?**
  - a. A second blood test to measure troponin levels
  - b. The provision that no patient is low risk with any positive troponin result
  - c. Both A and B
  - d. None of the above
4. **Troponin T blood levels are highest at:**
  - a. midnight.
  - b. 8 a.m.
  - c. noon.
  - d. 8 p.m.
5. **Which is true regarding malpractice claims alleging missed myocardial infarction, says Jeanie Taylor, RN, BSN, MS?**
  - a. Evidence clearly shows it is safe to have patients with chest pain wait for an evaluation once a single ECG is performed.
  - b. It is always inappropriate to have a stable patient with chest pain and a normal ECG remain in the waiting room while an evaluation is taking place.
  - c. It is not necessary for EDs to arrange for same- or next-day testing for intermediate risk patients if the ED does not employ CT imaging to evaluate coronary arteries.
  - d. EDs must arrange for same- or next-day testing for intermediate or high-risk patients if the ED does not employ CT imaging to evaluate coronary arteries.

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To earn credit for Module 1, log on to <https://med-ed.ahcmmedia.com/a/B193> to take the post-test.

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