Clinical Decision-Making In Adult Chest Pain Patients With Electrocardiographic ST-Segment Elevation: STEMI vs Non-AMI Causes Of ST-Segment Abnormality

It is 6:00 in the evening of an early winter weekday, and the ED is packed. You had agreed to cover this shift as a favor for a colleague, and now you wonder — Why did I make this change? You are seeing an elderly male (Patient #1) with a history of past myocardial infarction, hypertension, and diabetes mellitus. He is complaining of left shoulder pain and nausea, and his examination is significant for mild diaphoresis. The 12-lead electrocardiogram reveals a left bundle-branch block (LBBB) pattern that was first noted 2 years ago. (Figure 1A) You administer oxygen, aspirin, and nitroglycerin; laboratory studies, including a serum troponin and chest radiograph, are ordered.

Next you are called to see another patient with chest pain who has just been transported by EMS. You enter room 15 and observe a middle-aged male (Patient #2) holding his chest. He has no medical history, but notes a crushing substernal chest pain for the past 2 hours that is associated with weakness, dyspnea, and diaphoresis. His examination demonstrates an anxious appearance with a pale, ashen color and marked diaphoresis. The ECG reveals significant ST-segment elevation. (Figure 2) As you order the appropriate medications, you are asked to see another patient with dyspnea and chest pressure. This woman (Patient #3) is 63 years old and describes dyspnea with minimal exertion associated with chest tightness. Her medical history is significant for hypertension. She appears anxious; otherwise, her examination is unremarkable. The technician hands you the ECG (Figure 3), which at first glance is significant only for ST-segment depression in leads I and aVL; however, further review of the ECG demonstrates minimal ST-segment elevation in leads III and aVF.

CME Objectives
Upon completing this article, you should be able to:

1. Provide a systematic approach to ECG interpretation in patients with ST-segment elevation myocardial infarction (STEMI);
2. Identify distinguishing elements of STEMI vs other causes of ST-segment elevation; and

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The nurse then informs you that Patient #1 has increasing chest pain. You request a repeat ECG (Figure 1B) on your way into room 26, where you encounter a 39-year-old male with chest pain who denies other complaints. (Patient #4) His examination is normal. The ECG (Figure 4) demonstrates ST-segment elevation in the limb (II, III, and aVF) and precordial (V2-V6) leads. You give him an aspirin. Then it is back to Patient #1 for a review of the serial ECG.

Again, you wonder — Why did I make this schedule change?

The electrocardiogram (ECG) is a valuable and often-used tool for evaluating numerous patient complaints and scenarios in the ED. Perhaps the most frequent application of the ECG is the evaluation of the adult chest pain patient. In these patients, the ECG is used to establish the diagnosis of an acute coronary syndrome (ACS) or alternative cardiorespiratory ailment. ACS is a term referring to patients with clinical evidence of acute myocardial ischemia: unstable angina (UA), ST-segment elevation myocardial infarction (STEMI), and non-ST-segment elevation myocardial infarction (NSTEMI). For more on ACS pathophysiology, clinical diagnosis, risk stratification, and therapies, see Emergency Medicine Practice, Volume 6, Number 4, Evidence-Based Risk Stratification Of Patients With Suspected UA/NSTEMI, April 2004.) Furthermore, the ECG provides other clinically useful information in the ACS patient. The ECG is analyzed for signs of ST-segment elevation AMI (acute myocardial infarction), evidence of cardiac ischemia, determination of cardiac rhythm, and possible evidence of a noncardiac cause of the chief complaint (eg, pulmonary embolism, pericarditis). In patients with ACS, the ECG is used to assess the evolution of the syndrome, to determine the response to ED-delivered treatment, to assist in hospital disposition, and to predict the risk of cardiac complications, including mortality.

A primary goal of the emergency physician caring for the chest pain patient with an ECG that demonstrates ST-segment elevation (STE) is to differentiate STEMI from all other causes of ST-segment elevation. The American College of Cardiology/American Heart Association (ACC/AHA) currently recommends that fibrinolytic therapy be initiated (ideally) within 3 hours of presentation, and balloon angioplasty within 90 minutes of the onset of chest pain. There is substantial evidence that prompt opening of affected coronary arteries lowers rates of death, left ventricular dysfunction, and stroke.

The widely recognized benefits of early diagnosis and rapid revascularization treatment of AMI have emphasized the importance of the ECG interpretation in the ED. In the case of the chest pain patient demonstrating STE resulting from a noninfarction syndrome, the correct diagnosis must be made — not only to offer appropriate management for that particular illness, but also to avoid the incorrect application of potentially dangerous therapies, such as fibrinolysis.

The following discussion focuses on an algorithmic approach to the evaluation of the chest pain patient with electrocardiographic ST-segment elevation. While we urge the clinician to consider this approach in their medical decision-making in the setting of chest pain patients with electrocardiographic STE, strict adherence to this clinical pathway at the bedside is not recommended. Rather, our algorithm provides a systematic method for interpretation of the ECG with ST-segment elevation; its best use is illustrating the importance of a systematic approach to the interpretation of the ECG with ST-segment elevation. Throughout this article, please refer to the Critical Appraisal Of The Literature on page 11 for the algorithm.

Critical Appraisal Of The Literature

The medical literature used in the construction of this clinical pathway as a decision tool for the emergency physician was obtained with MEDLINE® and Cochrane database searches using key phrases, such as acute myocardial infarction (AMI), ST-segment elevation AMI (STEMI), acute coronary syndrome (ACS), diagnosis, and electrocardiogram (ECG). Furthermore, references cited in these papers were also reviewed. Lastly, the authors used their knowledge of the literature as a final resource for reference selection.

This clinical pathway was constructed using decision points with high specificity, which means that the clinician is able to rule in ST-segment elevation acute myocardial infarction with a high degree of confidence. Unfortunately, this clinical decision pathway cannot by used to rule out MI, in that the sensitivities of each decision point are not robust. In other words, sensitive tests are of value for ruling in a diagnosis; conversely, the rather low sensitivities of various ECG features do not assist the clinician in excluding STEMI.

Because we developed this clinical pathway as a decision tool only recently, it has yet to be tested, either retrospectively or prospectively. The clinical decision points in our algorithm represent fundamental questions the clinician must answer in their review of the ECG at the bedside of the adult chest pain patient. While each consideration has been in standard medical practice for extended time periods, these decision points have not been incorporated into a single tool — until now. Experience may demonstrate that this clinical pathway is best used in an educational setting (whether via personal learning or in-classroom training) to underscore the appropriate stepwise interpretation of the ECG with ST-segment elevation. On the other hand, this clinical pathway could become a valuable decision tool deployed at the bedside for review of the ECG in adult chest pain patients.

The ACC/AHA Guidelines for the Management for Patients with Acute Myocardial Infarction consider the presence of electrocardiographic ST-segment elevation of greater than 0.1 mV in 2 anatomically contiguous leads a strong indication for urgent reperfusion therapy in the patient with presumed AMI. Interestingly, these guidelines do not address the various syndromes that are potentially responsible for electrocardiographic ST-segment elevation in the chest pain patient. Rather, they simply mandate...
urgent reperfusion therapy in the presumed AMI patient with 2 anatomically oriented leads demonstrating greater than 0.1 mV of elevation.1 Strict adherence to such a mandate could clearly result in many unnecessary, potentially dangerous applications of primary reperfusion therapy. Of course, many ST-segment elevation, non-AMI syndromes can be recognized as non-STEMI presentations by the clinician using clinical, historical, and electrocardiographic clues, thereby leading to the most appropriate management.

Another policy statement addressing treatment considerations in the ED chest pain patient — the American College of Emergency Physicians (ACEP) Clinical Policy: Critical Issues in the Evaluation and Management of Adult Patients Presenting with Suspected Acute Myocardial Infarction or Unstable Angina2 — speaks to this electrocardiographic diagnostic dilemma. The policy states that patients with “ST-segment elevations greater than 0.1 mV in 2 or more contiguous leads that are not characteristic of early repolarization or pericarditis, nor of a repolarization abnormality from LVH or BBB” are candidates for fibrinolytic therapy consideration in the ED.3 Such a statement is much more comprehensive, addressing the reality of the situation in the ED better than the ACC/AHA publication.1

Definitions
In the chest pain patient, the ECG is the initial diagnostic investigation that can provide objective evidence of STEMI. ST-segment elevation is widely accepted as the most important indication of an AMI on the ECG; several different definitions of ST-segment elevation have been suggested. The ACC/AHA guideline defines ST-segment elevation as at least 0.2 mV of elevation in any 1 precordial lead, or greater than 0.1 mV of elevation in at least 2 anatomically contiguous precordial leads, or in at least 2 adjacent limb leads.1 By convention, the height of the ST segment is measured at a point 0.04 msec or 1 mm after or to the right of the J point at the end of the QRS complex. In this pathway, we chose to use the more sensitive criteria of at least 0.1 mV of STE in at least 2 anatomically contiguous leads.

Figure 1A. Initial ECG Tracing — Patient #1.

Normal sinus rhythm with left bundle-branch block and appropriate ST-segment and T-wave changes.

Figure 1B. Serial ECG Tracing — Patient #1.

Progressive ST-segment and T-wave changes are an indication of AMI in the LBBB patient, including concordant ST-segment elevation in leads I, V5, and V6; excessive discordant ST-segment elevation in leads V1 to V4; and flattening of the ST segments and T waves in the inferior leads.

Figure 2. ECG Tracing — Patient #2.

Extensive STEMI involving the inferior, lateral, and anterior walls.

Figure 3. ECG Tracing — Patient #3.

Inferior wall STEMI with subtle ST-segment elevation in the inferior leads; also, note the presence of reciprocal ST-segment depression in leads I and aVL.

Figure 4. ECG Tracing — Patient #4.

Diffuse ST-segment elevation with prominent T waves in leads II, III, aVF, and V4 to V6. The contour of the elevated ST segment is concave, consistent with benign early repolarization.
**Epidemiology And Electrophysiology**

**Epidemiology**
An estimated 5% of patients visit the ED for the chief complaint of chest pain, and 20% of those brought in by EMS will be experiencing an AMI. The rapid and accurate diagnosis of AMI is a formidable challenge for the emergency physician. In the ACS patient, various electrocardiographic findings not only suggest the diagnosis, but also provide indications for urgent management strategies. Perhaps the most ominous electrocardiographic finding is STE. ST-segment elevation may indicate AMI and remains an important indication for fibrinolytic therapy or percutaneous coronary intervention.

**Electrophysiology**
In the simplest terms, the ECG records the net flow of electrical current from negatively to positively charged myocytes from leads that are placed on the surface of the chest wall. In the normal heart, the flow of electrical current proceeds along efficient conducting fibers in the ventricular wall and is transmitted relatively equally in all layers; this rapid and efficient conduction of the impulse throughout the ventricular myocardium produces near-simultaneous depolarization, manifested by the QRS complex. Repolarization is manifested on the ECG as the ST segment. Since the repolarization normally occurs almost simultaneously and equally throughout the ventricular myocardium, there is no net electrical gradient, and the ECG records an isoelectric — or flat — ST segment.

In acute coronary syndromes, however, the injury resulting from AMI preferentially affects the endocardial cells, causing a negative charge on the inner surface of the heart, while the epicardial cells on the outside remain relatively positively charged. This charge distribution results in a net positive vector that points from the interior to the exterior of the heart and is manifested by an elevation of the ST segment on the ECG.

**Differential Diagnosis**
ST-segment elevation is not a specific marker of AMI, since numerous noninfarction syndromes that occur in the chest pain patient will manifest STE on the ECG. Certain patterns, such as left bundle-branch block (LBBB), left ventricular hypertrophy (LVH), and left ventricular aneurysm (LVA), occur with increased frequency in patients with known coronary artery disease; these patterns may confound the ED evaluation by mimicking STEMI. Other patterns, such as benign early repolarization (BER) and acute pericarditis (AP), are rarely associated with ischemic heart disease, though they may resemble acute infarction ST-segment waveforms. One prehospital study of adult chest pain patients demonstrated that the majority manifesting STE on the 12-lead ECG did not have AMI as a final hospital diagnosis; LVH and LBBB, followed by other syndromes, such as BER, represented the majority of the cases. Among adult ED chest pain patients, STE was encountered in approximately 20% of cases. AMI was the cause of this STE and was the final hospital diagnosis in only 15-30% of this population. (See Table 1 for a review of the various causes of ST-segment elevation in adult chest pain patients.)

Miller et al have demonstrated that, in patients admitted to the coronary intensive care unit with presumed AMI, STE was diagnostic for acute infarction in only half of patients; LVA and other STE, non-AMI syndromes were responsible for the ST-segment abnormalities resembling acute infarction in this patient group. Patients with non-AMI syndromes are not infrequently misdiagnosed as acute infarction, which may then subject them to unnecessary and potentially dangerous interventions. For example, a report by Sharkey et al noted that 11% of patients receiving a fibrinolytic agent were not experiencing AMI. The electrocardiographic syndromes producing this pseudoinfarct STE included BER (30%), LVH (30%), and various intraventricular conduction abnormalities (30%).

Ischemia is not the only process that can alter the electrical conduction within the heart. Other processes, such as a left bundle-branch block, left ventricular hypertrophy, ventricular paced rhythms (VPR), BER, left ventricular aneurysm (LVA), and acute pericarditis, can also present with electrocardiographic abnormalities, including ST-segment elevation. Many of these syndromes have characteristic electrocardiographic features that can assist in the identification of the underlying event.

**Prehospital Care**

**ECG Monitoring**
Single-lead electrocardiographic rhythm monitoring.

**Table 1. Causes Of Electrocardiographic ST-Segment Elevation In Adult Chest Pain Patients.**

<table>
<thead>
<tr>
<th>AMI</th>
<th>LVH</th>
<th>LBBB</th>
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<tr>
<td>Paced</td>
<td>BER</td>
<td>NSIVCD</td>
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which is most appropriate for rhythm identification, is the most frequently used ECG technique in the prehospital setting. Rhythm monitoring, however, does not reliably detect the electrocardiographic changes of ACS. On the other hand, the 12-lead ECG is easily obtained by prehospital providers and transmitted to hospital-based clinicians; furthermore, the quality of the transmitted ECG is excellent — not unlike the ECG performed in the ED.

The prehospital diagnosis of STEMI patients using the 12-lead ECG markedly reduces the time to hospital-based administration of fibrinolytic therapy. For instance, the Field Ambulance Study of Thrombolysis in Myocardial Infarction (FAST-MI) trial\(^{16}\) demonstrated that prehospital AMI patients with a diagnostic ECG performed prior to ED arrival experienced fewer hospital delays to the delivery of fibrinolysis. In this important early study, Karagounis et al investigated the ED and hospital courses in a total of 71 patients who were evaluated in the field and randomized to receive either prehospital or hospital-based 12-lead ECGs. The time from hospital arrival to initiation of fibrinolytic therapy in 6 AMI patients who had no prehospital ECG was 68 minutes, whereas the time from hospital arrival to initiation of thrombolysis for the 5 AMI patients who had prehospital ECGs was 48 minutes — demonstrating an average decrease of 20 minutes to treatment in patients for whom ECGs were performed in the field. In both groups, the delays to treatment were significantly shorter than the delay of 103 minutes previously demonstrated in a control group of 51 patients who were entered into various thrombolytic therapy studies over a 3-year period before the onset of the FAST-MI Trial.\(^{16}\) Several additional investigations have demonstrated similar benefits.\(^{17-19}\) In these studies,\(^{16-19}\) the administration of hospital-based fibrinolysis occurred much earlier than in patients who did not undergo prehospital ECGs. For example, hospital-based time to treatment with fibrinolysis in the Seattle trial was reduced from 102 minutes to 46 minutes, with the performance of the prehospital ECG.\(^{19}\) The Cincinnati Heart Project demonstrated a reduction in time to fibrinolytic therapy from approximately 89 minutes to approximately 63 minutes over a 1-year period. The greatest reduction in time to treatment was found in prehospital 12-lead ECG patients.\(^{17,19}\) These studies demonstrate that the prehospital ECG, when diagnostic of a STEMI, greatly reduces the time interval to hospital administration of a fibrinolytic agent; though unproven, the same statement likely applies to a PCI-based reperfusion strategy. One minor drawback of these studies is that they were all conducted over 15 years ago. The delay to treatment with an ED ECG is likely less today than it was then, and this factor may reduce the relative time benefit of the prehospital ECG.

**ED Management**

As with any patient in the ED, initial stabilization is the primary focus for the emergency physician. Attention to the airway with adequate oxygenation and ventilation is the first priority. Following correction of airway issues, circulation is next evaluated, with consideration of the blood pressure, pulse, cardiac rhythm, and end-organ perfusion. If a problem is found with these fundamentals, then the matter is addressed while focused evaluation and management continues. Many critical care situations would still allow ECG performance, even during active resuscitations. Clearly, the clinician directing the resuscitation is the most appropriate person to determine the correct time for the ECG; nonetheless, the clinician should be encouraged to obtain the ECG as soon as possible after presentation. In the stable chest pain patient suspected of ACS, the ECG should be performed rapidly (ie, in under 10 minutes). Furthermore, the ECG should be evaluated by a clinician who is well versed in the interpretation of the 12-lead electrocardiogram.\(^{20}\)

Initial electrocardiographic interpretations in the ACS patient range from normal to abnormal, including those patients with ST-segment elevation consistent with STEMI. In basic terms, the ECG may be described as nondiagnostic if it does not reveal a STEMI on presentation. In this application of the term, “nondiagnostic” covers normal ECGs, as well as electrocardiograms demonstrating nonspecific ST-segment/T-wave (NSSTTW) abnormality, ST-segment depression, or T-wave change. The “normal” ECG is exactly that — absolutely normal, with an absence of NSSTTW abnormalities, intraventricular conduction delay, repolarization changes, and rhythms other than sinus rhythm. “Minimal, nonspecific abnormality” is the next category of nondiagnostic electrocardiographic change. NSSTTW changes are defined as ST-segment changes (depression or elevation) less than 1 millimeter in deviation, or T waves that are blunted, flattened, or biphasic, without obvious inversion or hyperacuity. In a classic study of adult chest pain patients managed in the ED, Lee et al\(^{21}\) found that approximately 20% of these had an absolutely normal 12-lead electrocardiogram. In this study, the description “absolutely normal” translates to the absence of NSSTTW changes, atrioventricular block, intraventricular conduction delay, repolarization changes, and rhythms other than sinus rhythm. A significant minority of chest pain patients with an absolutely normal ECG were ultimately found to have an ACS. Considering the nonspecific ECG, Lee et al\(^{21}\) noted that adult chest pain patients with NSSTTW changes had a relatively low risk of AMI — ranging from 3 to 4% — but a significant risk of non-AMI ACS, which occurred in approximately 20% of cases. As these researchers demonstrated in this classic study, the clinical history must be relied upon heavily in patients with either normal or nonspecifically abnormal ECGs and with a convincing description of ischemic chest discomfort. Management and disposition decisions must be made based on the history — not on the nondiagnostic study.

The next category of nondiagnostic ECGs is far less problematic — those cases where the ECG is clearly abnormal, yet not diagnostic of a STEMI; this subgroup has demonstrated ST-segment depression or T-wave inversion — worrisome indicators of ACS. The last, and perhaps most troubling, group of nondiagnostic ECGs includes the
electrocardiogram with left bundle-branch block, left ventricular hypertrophy, or ventricular paced rhythm; in this setting, the ability of the ECG to detect ACS is limited by the repolarization abnormalities seen in these patterns of abnormal intraventricular conduction. A diagnostic ECG demonstrates ST-segment elevation of at least 1 mm in no fewer than 2 anatomically oriented leads.

The initial ECG may be a helpful guide for determination of cardiovascular risk. In this approach, the risk is a function of the electrocardiographic abnormality, when interpreted within the context of the clinical presentation. Brush et al have classified initial ECGs into 2 risk groups. The low-risk electrocardiographic group was composed of patients with normal, minimally abnormal (minimal nonspecific change), or unchanged (when compared with previous ECGs) electrocardiograms. The high-risk ECG group had a significant abnormality or confounding pattern — such as pathologic Q waves, ST-segment or T-wave changes, left ventricular hypertrophy, left bundle-branch block, or ventricular paced rhythm. Patients with initial ECGs classified as low-risk had a 14% incidence of AMI, a 0.6% incidence of life-threatening complications, and a 0% mortality rate; patients with initial ECGs classified as high-risk had a 42% incidence of AMI, a 14% incidence of life-threatening complications, and a 10% mortality rate.

If the clinician suspects ACS in the patient with a non-diagnostic ECG, serial monitoring for progressive alterations in the electrocardiogram should be performed. The frequency of monitoring is best determined by the treating clinician. Nearly 50% of patients with AMI come to the ED with a nondiagnostic 12-lead ECG, yet early in the course of hospitalization, up to 20% of these patients will develop changes consistent with STEMI. In the patient with continuous or recurrent chest pain and an initially nondiagnostic ECG, serial acquisition of a standard 12-lead electrocardiogram or the use of ST-segment trend monitoring may improve diagnostic accuracy. In particular, serial ECGs/ST-segment trend monitoring can identify the evolution of STEMI, identifying a candidate for reperfusion treatment.

History
Certainly, patient history is the most important feature of the diagnostic evaluation in chest pain presentations. The history guides the clinician, along with the physical examination and the acquisition of diagnostic investigations, such as the 12-lead ECG, serum markers, chest radiography, etc. Not only does the history and accompanying clinical presentation provide the indication for the ECG, but it also provides the framework around which the electrocardiogram and other diagnostic studies are interpreted — the approach to a 29-year-old female with pleuritic chest discomfort is markedly different from the approach to a 59-year-old male with hypertension and diabetes mellitus who notes chest pressure with associated dyspnea and diaphoresis. Not only is the diagnostic evaluation different in these 2 scenarios, but so is the interpretation of the investigations performed by the clinician.

Chest pain resulting from ACS is frequently described as “pressure,” “squeezing,” “fullness,” or “heaviness.” The classic location for ACS chest discomfort is substernal, with radiation to the left shoulder and jaw. Many patients experience associated symptoms, such as dyspnea, diaphoresis, nausea, vomiting, dizziness, and anxiety.

Physical Examination
The physical examination, although crucial to the ED evaluation, is often unrevealing in patients with STEMI. If the examination is abnormal, it most often demonstrates nonspecific findings, such as anxiety, diaphoresis, and poor color (eg, ashen appearance). Less commonly, the examination will provide evidence of acute complications of ACS, including pulmonary edema, active dysrhythmia, and/or cardiogenic shock. Certainly, the patient with acute congestive heart failure, ventricular dysrhythmia, or shock, once stabilized, can go on to demonstrate significant electrocardiographic abnormalities, such as STEMI.

Serum Marker Analysis
In the setting of a STEMI, the use of serum marker analysis — either as a single test or serial monitoring — is often unrevealing, particularly if the patient presents early in the course of the AMI. For STEMI patients, clinical decisions regarding reperfusion therapy should be made, in most cases, based upon an analysis of the clinical presentation and the ECG. Using the initial serum marker as a diagnostic branch point in the evaluation can lead to considerable delays to reperfusion therapy. Furthermore, early in the course of STEMI, the marker is more likely to be normal; both creatinine kinase MB (CK-MB) fraction and the troponins (TN) are unlikely to be abnormal within 3 to 5 hours of AMI onset. In the uncertain STEMI presenta-

Figure 5. ST-Segment Elevation — Concave Morphology.

A. Benign early repolarization with a concave form of ST-segment elevation. A line is “drawn” from the J point to the apex of the T wave. If the ECG waveform is below the drawn line (concave morphology), then a non-AMI cause of ST-segment elevation is suggested. B. & C. If the line is either superimposed on (B) or below (C) the ECG waveform, then AMI is suggested; in these scenarios, the waveform has a nonconcave morphology. D. & E. STEMI presentations with a concave morphology, illustrating the fallibility of this technique in all presentations.
associated with non-AMI causes, while localized changes occur in ACS presentations; however, this difference is not significant in most situations. STEMI demonstrates ST-segment elevation in an average of 3.4 leads; non-AMI patterns display ST-segment elevation in 4.1 leads on a typical ECG. Isolated ST-segment elevation in the inferior and lateral leads, however, frequently suggests STEMI, while anterior elevation is most often found in non-AMI syndromes.25 Regarding ST-segment depression, a specific anatomic distribution is not helpful in the diagnosis of ACS. An anatomic distribution, however, is significant in the LVH, bundle-branch block, and ventricular paced patterns — with these syndromes, ST-segment depression is seen in leads with predominantly positive QRS complexes.

**ST-segment Contour**

An analysis of the contour of the abnormal ST segment may be of assistance; this is true for both ST-segment elevation and ST-segment depression. With ST-segment elevation, a concave morphology of the elevated segment is more often associated with a non-AMI cause of the electrocardiographic abnormality, while a nonconcave shape is seen in AMI patients. The initial, upsloping portion of the ST segment is usually either convex or flat; if the ST segment is flat, it may be either horizontally or obliquely so. This technique uses the morphology of the initial portion of the ST-segment/T-wave — defined as beginning at the J point and ending at the apex of the T wave. (Figure 5 and Figure 6) Patients with noninfarctional ST-segment elevation (ie, those who demonstrate early repolarization or LVH-related changes) tend to have a concave morphology of the waveform. (Figure 7) Conversely, patients with ST-segment elevation due to AMI have either obliquely flat or convex waveforms (grouped together as nonconcave). (Figure 5 and Figure 6). The use of this ST-segment elevation waveform analysis (Figure 5A and Figure 5B) in ED chest pain patients is a very specific clinical tool — meaning it should be employed in the second tier of medical decision-making with respect to the ECG in a patient suspected of a STEMI.

This medical decision-making occurs as follows:

**Figure 7. NSTEMI vs STEMI with nonconcave ST-segment morphology.**

A. Non-AMI cause of ST-segment elevation, due to benign early repolarization. B. STEMI with a nonconcave ST-segment morphology.

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**The ECG With ST-Segment Elevation**

Electrocardiographic features to consider in the differential diagnosis of ST-segment elevation include the magnitude, morphology, and distribution of the ST-segment abnormality, and the width and size of the accompanying QRS complex.11,25,26 Overreliance on any single feature of the ECG, however, is not encouraged. Each piece of the diagnostic puzzle must be viewed as part of a whole that includes the history, examination, ECG (and its particular findings), and other investigations.

**Magnitude of ST-segment Changes**

Considering the magnitude of ST-segment changes, the total amount of ST-segment elevation is greater in the AMI patient than the noninfarction patient,11 with the total amount of ST-segment elevation being defined as the millivolt summation of elevation in all involved electrocardiographic leads. This has been observed from numerous perspectives, including the single-lead as well as the 12-lead ECG. For instance, the magnitude of individual-lead ST-segment elevation in AMI patterns averages 4.4 mV, while in non-AMI syndromes, 1.8 mV is seen. With the 12-lead ECG, the total summation of ST-segment elevation remains greater in AMI (15.3 mV) versus non-AMI (7.4 mV) presentations (p=.0004).25 Furthermore, in the STEMI patient, the total amount of ST-segment deviation — the sum of elevation and depression — is significantly greater in the AMI patient (17.8 mV versus 10.5 mV); this latter observation results from the presence of reciprocal ST-segment depression in many inferior and anterior STEMI presentations.10,25,26

**Anatomic Distribution**

The anatomic distribution of the ST-segment abnormality can be considered in the evaluation, though this information is less helpful in distinguishing ACS from non-ACS syndromes. More widespread ST-segment changes are associated with non-AMI causes, while localized changes occur in ACS presentations; however, this difference is not significant in most situations. STEMI demonstrates ST-segment elevation in an average of 3.4 leads; non-AMI patterns display ST-segment elevation in 4.1 leads on a typical ECG. Isolated ST-segment elevation in the inferior and lateral leads, however, frequently suggests STEMI, while anterior elevation is most often found in non-AMI syndromes.25 Regarding ST-segment depression, a specific anatomic distribution is not helpful in the diagnosis of ACS. An anatomic distribution, however, is significant in the LVH, bundle-branch block, and ventricular paced patterns — with these syndromes, ST-segment depression is seen in leads with predominantly positive QRS complexes.

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**Figure 6. STEMI with reciprocal ST-segment depression.**

Inferolateroposterior STEMI with reciprocal ST-segment depression in leads I and aVL. The ECG waveform in the leads with STE is nonconcave (ie, convex).
The precordial leads, characterized by either a QS or rS configuration complex. (The typical axis of the QRS complex is a function of typical placement of the distal pacing catheter in the RV apex.) A large monophasic R wave is noted in leads I and aVL — also, on occasion, in leads V3 and V6. QS complexes may also be encountered in leads II, III, and aVF. The T wave, especially in the right to mid-precordial and inferior leads, has a convex upward shape or a tall, vaulting appearance, similar to the hyperacute T wave of early myocardial infarction. The T waves in leads with the monophasic R wave are frequently inverted.

In patients with either bundle-branch block or ventricular-paced electrocardiographic patterns, the anticipated or expected ST-segment/T-wave configurations are discordant, directed opposite from the terminal portion of the QRS complex. This QRS complex-T wave axes discordance is termed the rule of appropriate discordance. According to this rule, leads with either QS or rS complexes (ie, complexes which are partially or entirely negative in deflection) may have markedly elevated ST segments, while leads with large monophasic R waves demonstrate ST-segment depression. The T wave, in leads with a negative or primarily negative QRS complex, has a convex upward shape or a tall, vaulting appearance, similar to the hyperacute T wave of early AMI. Conversely, the T waves in leads with the monophasic R wave are frequently inverted. Loss of this normal QRS complex-T wave axes discordance in patients can imply an acute process, such as AMI.

Large amplitude QRS complexes are a possible indication of electrocardiographic LVH, which, if present, may cause ST-segment/
T-wave changes; in fact, ST-segment/T-wave changes resulting from altered repolarization of the hypertrophied myocardium — the so-called “strain” pattern — are noted in approximately 70% of patients with LVH. The electrocardiographic LVH pattern is identified on the ECG by voltage criteria, including cumulative voltage of the S wave in lead V_1 and the R wave in lead V_5 or V_6 totaling more than 35 millimeters. LVH is associated with poor R wave progression and loss of the septal R wave in the right to mid-precordial leads, most commonly producing a QS pattern. In general, these QS complexes are located in leads V_1 and V_2, rarely extending beyond lead V_6. ST-segment elevation is encountered in this distribution, along with prominent T waves. The ST-segment elevation seen in this distribution is usually 2 to 4 millimeters in height. The initial, upsloping portion of the ST-segment/T-wave complex is frequently concave in LVH, compared to the either flattened or convex pattern observed in the AMI patient. ST-segment depression, characterized by downsloping ST-segment depression with asymmetric, biphasic, or inverted T waves in leads with prominent R waves, is seen in leads I, aVL, V_5, and V_6.

Medical Decision-Making And The Diagnostic Clinical Pathway

In the approach to the chest pain patient with electrocardiographic STE, the ST-segment elevation diagnostic algorithm represents one potential clinical pathway for diagnostic consideration. (See Clinical Pathway on page 11.) In step-wise fashion, the clinician evaluates the adult chest pain patient and obtains an ECG. If the ECG reveals ST-segment elevation, the ST-segment elevation clinical pathway can be employed as one possible tool in the continued interpretation of the ECG. Electrocardiographic features of significance, listed in order of consideration in the pathway, include the QRS complex width, QRS complex size (ie, amplitude), morphology of the elevated ST segment, presence of reciprocal ST-segment depression, and serial examinations with the ECG.

Widened QRS Complex

The diagnosis of an AMI in the presence of either a LBBB or a ventricular paced rhythm is extremely challenging, due to the confounding ECG pattern produced by the abnormal intraventricular conduction. The isolation of this electrocardiographic subset, the initial consideration in the pathway, is an important first step in determining the underlying etiology of the STE.

The QRS complex duration represents the total time of ventricular depolarization — the time from the initiation of ventricular depolarization after the electrical impulse has traversed the AV node and His-Purkinje system, to the end of ventricular depolarization, when the impulse ultimately arrives at the ventricular myocardium. An AMI itself does not cause an increase in the width of the QRS complex. Rather, the QRS complex width is increased by processes that interfere with specialized conduction pathways; for example, a left bundle-branch block pattern or ventricular paced rhythm represent electrocardiographic scenarios in which the QRS complex is widened due to abnormal intraventricular conduction, producing different patterns of depolarization. Importantly, the alteration in the ventricular depolarization also changes the repolarization process, which is manifested by variations in the ST

Figure 9C. The rule of appropriate discordance in patients with LBBB and VPR.


Figure 10. Ventricular paced rhythm pattern with ST-segment and T-wave configurations.

A. Discordant ST-segment depression — a normal electrocardiographic finding. B. Discordant ST-segment elevation — a normal electrocardiographic finding. C. Excessive discordant ST-segment elevation — an abnormal electrocardiographic finding, potentially consistent with AMI. D. Concordant ST-segment elevation — an abnormal electrocardiographic finding, potentially consistent with AMI. E. Discordant ST-segment depression — an abnormal electrocardiographic finding, potentially consistent with AMI.
segment and T wave, producing noninfarction STE. This STE is different from that of an AMI, in hat it is temporally static on serial ECGs; nonetheless, it still poses a unique challenge for the clinician, who must make clinical decisions on the basis of a single ECG.

Research done by Sgarbossa et al using patients enrolled in the GUSTO-1 (Global Utilization of Streptokinase and Tissue Plasminogen Activator for Occluded Coronary Arteries) trial investigated the ECGs of patients with either LBBB or ventricular paced rhythms and acute myocardial infarction. These investigators developed a clinical decision tool for use in these 2 electrocardiographic scenarios, to aid in the diagnosis of AMI based on a single ECG. Considering that patients with LBBB and VPR already have abnormal hearts, they are at a higher risk for infarction, and the prompt diagnosis of an AMI is extremely important.

Ventricular Paced Rhythms
A ventricular paced rhythm refers to the rhythm that results from an implanted pacemaker. Most commonly, the electrical impulse is generated at the apex of the right ventricle, travels across the intraventricular septum, and then in a retrograde direction travels towards the atria to depolarize the left ventricle. This impulse propagation manifests as a wide QRS complex, with a primarily negative QS or rS complex in the precordial leads. A monophasic R wave is seen in the lateral leads (I and aVL) and a negative QS complex in the inferior leads (II, III, aVF), resembling common changes seen in an acute infarction. Importantly for the clinician, the disruption in the normal depolarization of the heart also alters the repolarization process, which results in a baseline noninfarction STE. Understandably, these changes that mimic those of an AMI make the diagnosis of an actual infarct extremely difficult. Furthermore, the ST-segment and T-wave changes seen in VPR confound the ability of the ECG to detect ACS.

There is a large and growing population of patients with pacemakers. Due to the inherent poor health of this population and their higher risk for coronary events, it is important for the emergency physician to rapidly diagnose an acute infarction. Sgarbossa et al used data from 17 patients enrolled in the GUSTO-1 trial — those with a VPR only and those with a VPR and an enzyme-verified AMI — to develop a clinical decision-making tool for effectively “ruling in” an AMI, based on a single ECG. After an analysis of 9 different ECG characteristics, these 3 criteria were the only ones found to be significantly associated with an AMI (see page 9 for figures):

1. Discordant ST-segment elevation (Figure 9C(5)) — ST-segment elevation greater than or equal to 5 mm associated with a negative QRS complex (sensitivity of 53% and specificity of 88% for AMI);
2. Concordant ST-segment elevation (Figure 9C(3)) — ST-segment elevation greater than or equal to 1 mm associated with a positive QRS complex (sensitivity of 18% and specificity of 94% for AMI); and
3. Concordant ST-segment depression (STD) (Figure 9C(4)) — ST-segment depression greater than or equal to 1 mm associated with a negative QRS complex. (sensitivity of 29% and specificity of 82% for AMI).

This clinical decision rule proposed by Sgarbossa et al is best summarized by the “rule of appropriate discordance.” The relatively low sensitivity and specificity of the Sgarbossa criteria have led to criticisms questioning their usefulness. A case report by Madias pointed out that some patients with a VPR have extremely large depolarizations manifested by large QRS complexes. Since the rule of appropriate discordance predicts that the degree of
Clinical Pathway: ST-Segment Elevation Diagnostic Algorithm

1. Chest Pain Patient
   - Electrocardiogram
     - ST-segment elevation ≥1 mm in at least 2 anatomically contiguous leads?
       - Yes (Y)
         - QRS width > 0.12 sec?
           - No (N)
             - Left ventricular hypertrophy
           - Yes (Y)
             - Paced rhythm?
               - Yes (Y)
                 - High suspicion for acute myocardial infarction
               - No (N)
                 - Ventricular paced rhythm with no ECG evidence for acute infarction
             - No (N)
               - High suspicion for acute myocardial infarction
         - No (N)
           - Negative QRS and ST-segment elevation ≥5 mm?
             - Yes (Y)
               - High suspicion for acute myocardial infarction
             - No (N)
               - Positive QRS and ST-segment elevation ≥1 mm?
                 - Yes (Y)
                   - High suspicion for acute myocardial infarction
                 - No (N)
                   - Positive QRS and ST-segment elevation > 1 mm?
                     - Yes (Y)
                       - High suspicion for acute myocardial infarction
                     - No (N)
                       - Negative QRS and ST-segment elevation > 5 mm?
                         - Yes (Y)
                           - Bundle-branch block with high suspicion for acute myocardial infarction
                         - No (N)
                           - Positive QRS and ST-segment elevation ≥1 mm in V1, V2, or V3?
                             - Yes (Y)
                               - Bundle-branch block with high suspicion for acute myocardial infarction
                             - No (N)
                               - Bundle-branch block with high suspicion for acute myocardial infarction
           - No (N)
             - Bundle-branch block with high suspicion for acute myocardial infarction
         - Yes (Y)
           - PR-segment depression or ST height at J point in V6 to T-wave apex height in V6 ≥ 0.25?
             - Yes (Y)
               - Acute pericarditis
             - No (N)
               - Acute pericarditis
         - No (N)
           - Sum of T-wave apex heights in V1-V6 to sum of QRS apex heights in V1-V6 > 0.22?
             - Yes (Y)
               - High suspicion for acute myocardial infarction
             - No (N)
               - Initial part of ST segment concave?
                 - Yes (Y)
                   - Left ventricular hypertrophy with high suspicion for acute myocardial infarction
                 - No (N)
                   - Serial ECGs
                     - Yes (Y)
                       - ≥0.05-mm absolute change in ST-segment elevation or depression, Q-wave development, or T-wave inversion in ≥2 anatomically contiguous leads on repeat ECG at 3 to 4 hours?
                         - Yes (Y)
                           - High suspicion for acute myocardial infarction
                         - No (N)
                           - No ECG evidence for acute myocardial infarction
                     - No (N)
                       - Concavity lost?
                         - Yes (Y)
                           - High suspicion for acute myocardial infarction
                         - No (N)
                           - No ECG evidence for acute myocardial infarction
           - No (N)
             - Bundle-branch block without ECG evidence for acute infarction
         - No (N)
           - Bundle-branch block without ECG evidence for acute infarction
   - No (N)
     - Reciprocal ST-segment depression ≥1 mm in at least 1 lead?
       - Yes (Y)
         - High suspicion for acute myocardial infarction
       - No (N)
         - J point notching?
           - Yes (Y)
             - Benign early repolarization
           - No (N)
             - Benign early repolarization

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This clinical pathway is intended to supplement, rather than substitute for, professional judgment and may be changed depending upon a patient’s individual needs. Failure to comply with this pathway does not represent a breach of the standard of care.
repolarization is proportional to the degree of the proceeding depolarization; these patients also have a large amount of STE that would satisfy the criteria, even in the absence of AMI. However, Ufberg et al\(^8\) provided support for the practical application of these criteria in another case report that was later supported by angiographic evidence.

The key point to make, and one that was reinforced by Sgarbossa in the original paper, is that these criteria should be used to rule in an AMI, rather than to exclude it, which is how the criteria are presented in the clinical pathway. In support of their position, the authors cited a survey showing that patients with a “nondiagnostic” ECG are less likely to receive a fibrinolytic agent.\(^9\) These criteria play an important role in providing some objective evidence for use of the ECG to support initiation of treatment in a patient with a VPR and a possible AMI. To further address the low sensitivity of these criteria, the clinical pathway recommends serial ECGs for those patients who do not meet them. The aim is to look for the dynamic changes characteristic of an AMI and to help differentiate AMI patients from those with an uncomplicated VPR.

**Left Bundle-branch Block**

The diagnosis of AMI in a patient with a left bundle-branch block (Figure 9A on page 8) based on the ECG is yet another challenging task for the emergency physician. Considering that LBBB often occurs in patients with AMI, this population has the highest mortality rate of any group. \(^28\) Considering that LBBB often occurs in patients with AMI, this population has the highest mortality rate of any group. \(^28\)

A left bundle-branch block occurs when there is a deficit of the Purkinje system beyond the His bundle, which disrupts impulse conduction from the atria to the ventricles. When this pathway is interrupted, flow continues unimpeded down the right bundle to depolarize the right ventricle, and then across the interventricular septum in a much less efficient manner to depolarize the left ventricle. As with the ventricular paced rhythm scenario, the altered depolarization in LBBB produces a different pattern of repolarization; this altered repolarization is manifested on the ECG with ST-segment and T-wave abnormalities.

The challenge of differentiating these abnormalities from ACS is significant. Obviously, this distinction is vital, in that early treatment decisions are based upon the ECG and its interpretation within the context of the patient’s presentation. Currently, management guidelines suggest that patients with a new LBBB pattern and presentation consistent with AMI should be considered for fibrinolytic therapy. Despite this statement, this subset of the AMI population is actually 78\% less likely to receive fibrinolytic therapy than those without a LBBB.\(^8\) In an effort to more effectively direct therapy, Sgarbossa et al\(^8\) analyzed those patients in the GUSTO-I trial with uncomplicated LBBB, comparing them to patients with LBBB and AMI, to determine which ECG criteria can most appropriately discriminate between the 2 electrocardiographic presentations. The rule of appropriate discordance, as discussed in the ventricular paced rhythm section, underlies this electrocardiographic concept. The investigators noted the following criteria, ranging from potentially suggestive to completely diagnostic of AMI in LBBB presentations (see page 9 for figures):

1. Concordant ST-segment elevation (Figure 9C[3]) — ST-segment elevation greater than or equal to 1 mm associated with a positive QRS complex (sensitivity of 73\% and specificity of 92\% for AMI);
2. ST-segment depression greater than or equal to 1 mm in lead V\(1\), V\(2\), or V\(3\) (sensitivity of 25\% and specificity of 96\% for AMI); and
3. Discordant ST-segment elevation (Figure 9C[5]) — ST-segment elevation greater than or equal to 5 mm associated with a negative QRS complex (sensitivity of 31\% and specificity of 92\% for AMI).

Of these 3 criteria, the researchers found that the first 2 were highly predictive of an AMI, and that the third was very suggestive and warranted further evaluation. When a previous or serial ECG\(^45\) or cardiac marker levels\(^46\) are added to the analysis, the sensitivity and specificity of this decision-making tool are further increased.

These criteria have come under significant criticism, with critics stressing the low sensitivity and the relatively small number of patients that actually meet these criteria. A study conducted by Shlipak et al\(^34\) found that these criteria indicated a diagnosis of an AMI in only 3\% of cases and had a sensitivity of only 10\%. Shapiro et al\(^36\) also found a low sensitivity for the application of these criteria, as well as a low degree of interobserver reliability. However, as with the criteria for an AMI with a ventricular paced rhythm, Sgarbossa\(^45\) stresses that the application of these rules to the presenting ECG should primarily be used to rule in an AMI in this population, where the diagnosis of AMI would otherwise be extremely difficult. While the low sensitivity of these criteria means that they might be infrequently met, when they are satisfied, the emergency physician can be reassured and direct therapy accordingly.

To specifically address the low sensitivity of the criteria further, as we did with VPR patients, the clinical pathway presented recommends the use of serial ECGs to help rule in patients with a LBBB AMI based on dynamic changes who do not meet the criteria on the initial ECG.

Given the relatively low sensitivity of the criteria developed by Sgarbossa et al, the investigators recommended\(^46\) the use of serial electrocardiography in these patients. In the non-ACS patient, one can assume that the ECG tracing of a patient with a LBBB or a ventricular paced rhythm should be static on serial recordings, due to the relative permanence of the disruption of the electrical flow in these conditions, when compared to the ischemic-induced changes of an evolving AMI. Consequently, there is significant value in comparing the presenting ECG to a previous tracing or to an ECG that is obtained while serial marker levels are obtained. Unfortunately, while serial ECG monitoring is a common practice, there has been rela-
tively sparse investigation as to the specific criteria useful in such a diagnosis. In a well designed study, Fesmire\textsuperscript{31} showed that continuous ECG monitoring of a patient with a LBBB could be used to discern an AMI using a cutoff of at least a 0.2-mm change in STE magnitude in a single lead, or greater than or equal to a 0.1-mm change in any 2 leads.\textsuperscript{46} There have been no studies assessing the specificity and sensitivity of these criteria, but anecdotal they have been shown to strongly suggest an AMI. There have also been no studies specifically examining the use of serial ECGs in a patient with a ventricular paced rhythm and a questionable AMI. We have chosen to use the same criteria for both a VPR and a LBBB in this clinical pathway, given the somewhat similar characteristics in their baseline tracings. This assumption, however, is not based on direct evidence. Also, because of the unique baseline differences of this population compared to the remainder of chest pain patients, and given the fact that the only study to assess this population used these criteria, the clinical pathway uses separate criteria for serial ECGs for these patients than for those without a LBBB or a ventricular paced rhythm. The clinical pathway advises looping back to serial ECGs, to ensure that no changes are missed that might manifest on a subsequent ECG.

Large Amplitude QRS Complex (ie, The LVH Electrocardiographic Pattern)

The electrocardiographic LVH pattern (Figure 11A and Figure 11B on page 10) is yet another process that alters the conduction within the heart and can result in ST-segment and T-wave changes; as with the ventricular paced rhythms and LBBB scenarios, these LVH-related changes can also confound the ECG diagnosis of AMI. True anatomic LVH occurs with an increase in the mass of the left ventricle of the heart. This increased mass results in a greater flow of current in the left side of the heart, causing an extremely large and negative QRS complex, as seen in the right-sided precordial leads (V\textsubscript{1} to V\textsubscript{5}) as the current flows away from the right side of the chest. The lateral leads, I, aVL, V\textsubscript{6} and V\textsubscript{a}, demonstrate similarly large, yet positive QRS complexes.\textsuperscript{47} As predicted by the rule of appropriate discordance, the large QRS complex results in ST-segment/T-wave complex changes that are directed opposite from the QRS complex; such changes are seen in 70\% of cases.\textsuperscript{49} The T wave is inverted with a gradual, downsloping initial limb and a more abrupt return to the baseline. ST-segment and T-wave abnormalities as seen in these LVH patients are termed the “strain pattern.”\textsuperscript{48} This rule applies less in the LVH presentation, compared to the LBBB and VPR scenarios.

Numerous electrocardiographic criteria have been suggested as reasonable screens for true anatomic LVH. The Sokolow-Lyon criteria — \(S\textsubscript{v1} + R\textsubscript{v5} \geq 35\) mm — and the Cornell voltage criteria — \(R\textsubscript{v1} + S\textsubscript{v3} \geq 28\) mm in males, and \(R\textsubscript{v1} + S\textsubscript{v5} \geq 20\) mm in females — are used as the diagnostic criteria in our clinical pathway.\textsuperscript{49} It is important to realize, however, that the LVH pattern and related electrocardiographic abnormalities, when present, are the focus of scrutiny; the identification of anatomic LVH is not crucial to the electrocardiographic pattern.

To date, no diagnostic electrocardiographic tools exist that aid in the identification of AMI in the setting of LVH, but there are subtle differences in the ECG that have been anecdotally associated with an AMI. The branch point that is used in this clinical pathway is based on the evaluation of the initial morphology of the STE.\textsuperscript{48} In a patient with uncomplicated LVH, this segment is usually concave with respect to a line drawn between the J point at the end of the QRS complex to the apex of the STE. When an AMI is present, the STE is usually either obliquely flat or convex in morphology. This morphological criterion, however, is imperfect, because the ST segment might initially be concave early on in an AMI, particularly in the anterior distribution. Serial ECGs can detect AMI as the electrocardiographic changes become more apparent. Since the ECG changes that are due to LVH would be expected to remain static over time, the use of serial recordings would also be expected to detect changes that are due to AMI.

ST-segment Contour

Excluding patients with confounding ECG patterns of ventricular paced rhythms, LBBB, and LVH from the population that has reached this point in the clinical pathway presumably increases the predictive power of all branch points from here.

One electrocardiographic feature that has a rather powerful association with AMI is the morphology of the STE itself. (Figure 5 on page 6 and Figure 6 on page 7) While the presence of a convex or “tombstone” appearance of the STE has been clinically associated with AMI based on anecdotal evidence,\textsuperscript{48,50} Brady et al\textsuperscript{18} were the first to analyze this feature in a study, to demonstrate the actual predictive value of this criterion. Based on a study of 171 chest pain patients with STE, the researchers analyzed the morphology of the ST segment and showed that a convex or obliquely straight waveform was highly predictive of an AMI (sensitivity of 77\%, specificity of 97\%, and positive predictive value (PPV) of 94\%). Due to the relatively low sensitivity, they suggested that meeting this criterion should be taken as strong ECG evidence for effectively ruling in an AMI, but the failure to meet this criterion should not be used to exclude the diagnosis of an acute infarction. This study was limited to only the single presenting ECG, not serial ECGs or the use of previous ECGs for comparison. As a result of this limitation, the authors concede this criterion might miss an early and evolving AMI, before a convex STE becomes apparent. In this clinical pathway, we attempt to address this problem with the use of serial ECGs to identify those patients who progress to nonconcave (convex or obliquely straight) STE after the initial presentation.

With respect to the morphology of the elevated ST segment, Kosuge et al\textsuperscript{2} showed that the morphology of the ST segment is predictive of outcome following reperfusion therapy. After dividing the population into 3 categories, a concave morphology was shown to be associated
with an excellent prognosis, a straight ST segment was predictive of an intermediate prognosis, and a convex morphology was indicative of a poor prognosis and worsened left ventricular function at the time of hospital discharge. Furthermore, they showed that a concave ST-segment morphology was poorly associated with an angiographically proven anterior AMI, while a straight or convex shape was more strongly correlated. A recent study by Smith demonstrated that, in an anterior AMI patient population, a concave morphology of the ST segment is associated with a shorter duration of symptoms; however, he also noted that this criterion lacks the sensitivity to effectively rule out an AMI. The very high specificity of a convex or obliquely straight ST segment remains an effective tool to rule in an acute infarction, as it is used in this clinical pathway.

Reciprocal ST-segment Depression
Another ECG feature that has been associated with an AMI is the presence of reciprocal ST-segment depression (RSTD), or “reciprocal change.” Reciprocal change refers to ST-segment depression in leads that are opposite to those that are exhibiting STE. (Figure 12 and Figure 13) Also, reciprocal change does not describe ST-segment depression related to altered depolarization as seen in patients with VPR, LBBB, or LVH. Therefore, the definition of reciprocal ST-segment depression includes the following criteria:

1. ST-segment depression;
2. Presence of ST-segment elevation in distant leads; and
3. Absence of confounding ECG patterns (LVH, BBB, or VPR).

The majority of inferior wall AMIs and approximately one third of anterior wall AMIs show RSTD. A large, prehospital study by Otto and Aufderheide showed that RSTD had a sensitivity and a PPV of over 90% for AMI. Brady et al showed that, when all patient populations are included in the analysis, the predictive value of RSTD (sensitivity of 63% and PPV of 30%) is unacceptably low for it to be used to effectively predict an AMI. Yet, when patients with LBBB, VPR, and LVH are excluded from analysis, as they are at this point in this clinical pathway, both the specificity and PPV of RSTD rise to 93%. The sensitivity of RSTD for predicting an AMI in this population, however, is only 69%. Therefore, as with almost all other ECG criteria in this algorithm, the presence of RSTD is highly suggestive of AMI, while its absence cannot be used to rule out an acute infarction — in other words, use the presence of RSTD to rule in AMI in appropriate patients.

Serial Electrocardiography
A powerful electrocardiographic method used to differentiate an AMI from other processes is serial ECG tracings. (Figure 14) Compared to static processes, such as a non-ACS LBBB, VPR, or LVH, where one would expect few changes in the ECG over a short period, an AMI is a dynamic and evolving process with a predictable progression of changes. The normal progression of an AMI on the ECG begins with hyperacute T waves that appear within minutes of the interruption of blood flow to the myocardium. A giant R wave is the next feature that transiently appears, and it may not be present in all patients. Then the ST-segment elevation appears. As previously noted, the STE may be initially concave in an early AMI, but will progress to a nonconcave waveform. The STE gradually returns to baseline over 12 to 24 hours and is followed by T-wave inversion. Eventually, the R wave returns to baseline, and a Q wave arises after 12 hours. While these changes are highly specific for an AMI, they require a relatively long time to manifest, which reinforces the importance of this clinical pathway in providing evidence to initiate treatment based on the characteristics of the single presenting ECG.

Serial ECGs, however, can still provide important information in those patients with nondiagnostic electrocardiograms at initial presentation. Although the characteristic progression is well known, based on anecdotal evidence, there has been very little evidence-based research into specific criteria used to analyze serial ECGs. A study by Hedges et al that was designed to compare the effectiveness of serial ECGs to serial CK-MB levels showed that specific changes in serial ECGs can be used to diagnose an AMI, even though they are less specific than serial marker levels. The criteria that Hedges et al used, and that are adopted in the clinical pathway, were a greater than 0.5-mm change in STE or STD, Q-wave development, or T-wave inversion in 2 or more contiguous leads at 3 to 4

Figure 12. Reciprocal ST-segment depression.

Figure 13. Subtle inferior wall STEMI with reciprocal ST-segment depression in the lateral leads.
The researchers found that the satisfaction of one of these criteria indicates an AMI with a sensitivity of 88%, which is further increased with the addition of serial markers in the evaluation. Furthermore, a lack of change in serial ECGs was also found to be a strong predictor of the absence of an acute infarction. Consequently, these serial changes play an important role in identifying patients with an AMI that would otherwise be missed, not only by this clinical pathway, but also in clinical practice.

The other characteristic that has been anecdotally associated with an AMI on serial ECGs is the development of a nonconcave STE waveform — that is, the loss of ST-segment elevation concavity.\textsuperscript{11} It has been noted that a patient who presents soon after the onset of chest pain may not have the characteristic convex or obliquely straight STE on their presenting ECG. As the AMI progresses, however, the typical ECG changes are likely to become apparent. There have been no investigations looking specifically at this criterion, but based on the high specificity and PPV of a nonconcave STE, we elected to include this criterion in the clinical pathway.

The pathway ends with a loop back to serial ECGs. We believe there is value in the serial monitoring of cardiac activity while cardiac marker levels are measured. It is possible that the full changes of the ECG will not become apparent for several hours and could be detected by a repeat ECG. Consequently, any changes that become apparent will greatly aid the emergency physician in the diagnosis of AMI.

\textbf{Controversies/Cutting Edge}

\textbf{ST-segment Elevation in Additional ECG Leads}
Right ventricular myocardial infarction presents with hypotension, elevated jugular venous pressure, and clear lung fields; most often the ECG demonstrates ST-segment elevation in the inferior and right ventricular leads. Right ventricular myocardial infarction occurs in approximately one third of inferior wall AMIs.\textsuperscript{53-55} The standard 12-lead ECG will demonstrate ST-segment elevation in the inferior leads, with the greatest magnitude of elevation (compared to the other leads) in lead III; furthermore, lead \( V_1 \) may also demonstrate ST-segment elevation, in that this lead, of all the standard 12, most closely images the right ventricle.

The use of additional leads has greatly increased the ability to diagnose right ventricular infarction. The addition of lead \( V_{4R} \) (Figure 15A) provides further objective evidence of RV involvement, beyond the standard 12-lead ECG. RV infarction can be diagnosed with 80-100% sensitivity by ST-segment elevation greater than 1 mm in lead \( V_{4R} \).\textsuperscript{53-56} Alternatively, the clinician can use an entire reversal of the precordial leads, namely \( V_1R \) to \( V_6R \); compared to the use of single-lead \( V_{4R} \), the entire array did not increase the diagnostic ability of the additional lead approach. Regardless of the number of right ventricular leads used, the magnitude of the ST-segment elevation is less pronounced than is usually seen in the standard 12 leads of the ECG, due to the fact that the right ventricle is composed of considerably less muscle than the left ventricle.

Posterior wall myocardial infarction refers to AMI of the posterior wall of the left ventricle. This region is usually perfused by the left circumflex artery or by a dominant right coronary artery with prominent posterolateral or posterior descending branches. The 12-lead ECG will most often demonstrate an inferior or lateral wall STEMI, as well as reciprocal ST-segment depression, prominent R wave, and upright T wave in the right precordial leads. Myocardial infarction involving the posterior wall usually occurs in conjunction with inferior or lateral AMIs; isolated posterior wall myocardial infarction occurs rarely. The use of posterior electrocardiographic leads is fruitful in the evaluation of possible posterior wall AMI, when compared to the standard 12 leads. ST-segment elevation greater than 1 mm in leads \( V_8 \) and \( V_9 \) (Figure 15B) confirms the diagnosis of posterior myocardial infarction. In fact, the presence of ST-segment elevation in the posterior ele-

\textbf{Figure 15. ST-segment elevation in additional ECG leads.}\n
\begin{figure}[h]
\centering
\includegraphics[width=\textwidth]{figure15.png}
\caption{A. Right ventricular leads \( V_{4R} \) to \( V_{6R} \) with minimal ST-segment elevation consistent with right ventricular STEMI. B. Posterior wall STEMI with ST-segment elevation in the posterior leads \( V_8 \) and \( V_9 \).
\end{figure}
trocardiographic leads is more indicative of posterior AMI than the findings observed in leads \( V_1 \) to \( V_3 \).

The reported sensitivity of the posterior electrocardiographic leads may be as high as 90% for identifying posterior AMI, with a predictive accuracy up to 93.8%. Also, false-positive ST-segment elevation in leads \( V_4 \) and \( V_5 \) is unusual, occurring at a frequency similar to that encountered in the standard ECG. Finally, as with the right ventricular leads and the magnitude of ST-segment elevation, less pronounced elevation is usually encountered in the posterior electrocardiographic leads, but for a different reason — the posterior leads are further from the myocardium, allowing for more resistance to current flow and less pronounced ST-segment elevation.

**Electrocardiographic Body Surface Mapping**

Body surface mapping (BSM) is an electrocardiographic technique that uses numerous leads, enabling a more complete visualization of cardiac electrical activity and the status of the myocardium in the ACS patient. Output from BSM is displayed in a color contour or body map (Figure 16A and Figure 16B), 12-lead ECG (Figure 16C), and an 80-lead ECG (Figure 16D), all of which appear here in grayscale. The color contour maps can be displayed on a torso image or as a flat map. The value of BSM lies in greater spatial representation of cardiac electrical activity than the 12-lead ECG, thus allowing a more complete imaging of the heart and greater sensitivity for detecting AMI. Although many studies compare BSM to 12-lead ECG, BSM is not designed to replace 12-lead ECG, but to augment traditional diagnostic practices. The future of BSM in the ED is unclear; more research is necessary to define its benefits and limitations. BSM, however, does have the potential to enhance the emergency physician's ability to rapidly assess for AMI in difficult clinical settings, including right ventricular or posterior wall involvement.

**Summary**

The clinical pathway presented in this issue of *Emergency Medicine Practice* is designed to aid the physician in the diagnosis of an AMI. The integration of the ECG criteria for an acute myocardial infarction into a coherent algorithm will hopefully provide a means of analyzing the data to assist with early diagnostic and treatment decisions, such as the administration of a fibrinolytic agent. All decision points have been chosen with criteria that have a high specificity and positive predictive value for an AMI and are designed to rule in the diagnosis. Ultimately, though, the best use of this clinical pathway will likely be in conjunction with serum markers and clinical suspicion. The ECG, whether interpreted alone or within the direction of this pathway, must be reviewed within the context of the clinical presentation. The history, physical examination, and diagnostic studies, as interpreted by the treating physician, will guide the clinician to additional investigations and treatment options — the ECG and this electrocardiographic clinical decision-making pathway must be used with these caveats and recommendations in mind.

**Isolated posterior wall STEMI.** A. Body map of the anterior thorax with prominent red color (ST-segment elevation) in the superior portion of the heart (represented by darker shading at the top left) and prominent blue color (ST-segment depression) in the inferolateral region of the heart (represented by darker shading at the bottom right). B. Body map of the posterior thorax with prominent red color (ST-segment elevation) in the posterior portion of the heart (represented by darker shading at the top center). C. 12-lead ECG without obvious STEMI; minimal ST-segment depression is seen in leads \( V_1 \) to \( V_3 \). D. 80-lead ECG with ST-segment elevation in the posterior leads (leads 65 to 77).
Case Conclusions
(See page 3 for all figures referring to the case presentations.) Patient #1 was not improving, despite adequate therapy. His examination revealed an ahen appearance with marked diaphoresis. Given his increasing discomfort, you correctly ordered a repeat ECG (Figure 1B) that demonstrated significant abnormality, including progressive discordant ST-segment elevation in leads V₁ to V₄, new concordant ST-segment elevation in leads I, aVL, V₆, and V₃, and isoelectric ST segments in leads III and aVF. These serial electrocardiographic findings in a LBBB patient with an appropriate clinical presentation for ACS strongly supported a diagnosis of AMI. The on-call cardiologist is contacted regarding possible PCI; she readily agrees with you and requests that you transfer the patient immediately to the catheterization laboratory for PCI.

Patient #2 is experiencing a rather extensive STEMI with involvement of the anterior, lateral, and inferior regions. (Figure 2) Importantly, the simultaneous presence of Q waves and ST-segment elevation in leads V₂ to V₄ does not dissuade you from aggressive reperfusion therapy; you correctly recall that Q waves can appear as early as 2 hours into a STEMI, which correlates exactly with this patient’s history. He is referred urgently to the second-call cardiologist, who agrees to see the patient in the ED within the next 10 minutes. Approximately 20 minutes later, you note that the cardiologist has started intravenous TNK.

Patient #3, with continued chest pain, is suffering from an inferior wall STEMI; the ECG demonstrates minimal ST-segment elevation in leads III and aVF, with an oblique contour and reciprocal ST-segment depression in leads I and aVL. (Figure 3) This patient receives asprin, nitrates, heparin, beta-blockade, and glycoprotein inhibition; she is urgently transferred to the catheterization laboratory, now that Patient #1 is in the coronary care unit.

Patient #4, thankfully, remains clinically stable while you manage 3 STEMI cases over the course of 2 hours. His ECG (Figure 4) reveals diffuse ST-segment elevation in leads II, III, aVF, and V₁ to V₄; the contour of the elevated ST segment is concave, suggestive of a non-STEMI cause of the ST-segment abnormality. Serial ECGs show no evolution of the abnormalities, while a repeat serum troponin obtained 4 hours later is within normal limits. The patient is discharged from the ED with a chest wall pain syndrome and a non-STEMI cause of the ST-segment elevation — likely benign early repolarization. He is well at follow-up 4 weeks later.

References
Evidence-based medicine requires a critical appraisal of the literature based upon study methodology and number of subjects. Not all references are equally robust. The findings of a large, prospective, randomized, and blinded trial should carry more weight than a case report.

To help the reader judge the strength of each reference, pertinent information about the study, such as the type of study and the number of patients in the study, will be included in bold type following the reference, where available.

25. Brady WJ, Perron AD, Ullman EA, et al. Electrocardiographic ST segment elevation...


56. Molendez LJ, Jones DT, Salcedo JR. Usefulness of three additional electrocardiographic chest leads (V7, V8 and V9) in the diagnosis of acute myocardial infarction. Can Med Assoc J 1978;119:745-748. (Prospective cohort; 131 patients with chest pain admitted to a CCU)


Physician CME Questions

1. ST-segment elevation in the ED chest pain patient is:
   a. most often caused by AMI.
   b. most often related to a life-threatening event.
   c. usually due to patterns such as left bundle-branch block and left ventricular hypertrophy.
   d. most often due to benign early repolarization.

2. The prehospital 12-lead ECG has been:
   a. shown to reduce the time to hospital-based reperfusion therapy.
   b. demonstrated to be equivalent to single-lead ECG monitoring for the diagnosis of AMI.
   c. noted to be of poor quality.
   d. None of the above.
3. Choose the most correct statement:
   a. The amount of ST-segment deviation is greater in non-AMI patterns compared to AMI.
   b. The magnitude of ST-segment elevation is greatest in AMI patterns.
   c. Left ventricular hypertrophy usually produces greater magnitude ST-segment elevation than AMI.
   d. The magnitude of the reciprocal ST-segment depression is subtracted from the ST-segment elevation for the total magnitude of deviation.

4. ST-segment elevation in AMI patients:
   a. is usually noted in numerous leads simultaneously.
   b. is usually localized to a single anatomic distribution.
   c. is never seen with coexistent ST-segment depression.
   d. is usually widespread in distribution.

5. The contour of the elevated ST segment:
   a. reliably identifies AMI.
   b. does not separate AMI from non-AMI presentations.
   c. is a reliable tool to rule in AMI in patients with chest pain and ST-segment elevation.
   d. provides little information of value in AMI diagnosis.

6. Reciprocal ST-segment depression:
   a. is a useful diagnostic aid in the evaluation of STEMI.
   b. is a masquerading pattern in chest pain patients.
   c. identifies patients at lower risk.
   d. results from a clear-cut mechanism.

7. The QRS complex in the chest pain patient with ST-segment elevation:
   a. has no relation to ST-segment considerations.
   b. is always of normal amplitude and width.
   c. is always normal in bundle-branch block presentations.
   d. must be considered in the evaluation of ST-segment changes.

8. The clinical pathway:
   a. is composed of highly sensitive considerations.
   b. can be used to rule out AMI.
   c. involves electrocardiographic features which are specific for AMI.
   d. has been extensively tested in the chest pain population.

9. ECG findings suggestive of AMI in the left bundle-branch block presentation include all of the following, EXCEPT:
   a. concordant ST-segment elevation.
   b. discordant ST-segment elevation of any magnitude.
   c. concordant ST-segment depression.
   d. discordant ST-segment elevation of greater than 5 mm.

10. The ECG in ventricular paced rhythms:
   a. is of considerable value in making the ECG diagnosis of AMI.
   b. is suggestive of AMI with concordant ST-segment elevation.
   c. is most often diagnostic of AMI in such presentations.
   d. cannot be used to exclude AMI using the rule of appropriate discordance.

11. Which of the following is FALSE regarding serial ECGs in the chest pain patient?
   a. They do not increase the sensitivity and specificity of the single ECG in the diagnosis of AMI.
   b. They can assist in the identification of non-AMI causes of ST-segment elevation.
   c. They provide the clinician with a tool to rule in AMI with progressive change.
   d. They can be used in the patient with bundle-branch block and progressive chest pain.

12. The “rule of appropriate discordance”:
   a. has near 100% sensitivity diagnosing AMI in left bundle-branch block.
   b. should be considered only in patients with reciprocal ST-segment depression.
   c. can only be applied in bundle-branch block presentations.
   d. is valid in ECG interpretations in bundle-branch block and ventricular paced rhythms.

13. Regarding the additional-lead ECG, which of the following is FALSE?
   a. Patients with right precordial (V1 to V3) ST-segment depression may be experiencing a posterior wall infarction.
   b. Patients with inferior wall STEMI may be experiencing a right ventricular infarction.
   c. The use of additional-lead ECGs may increase the sensitivity and specificity of the standard 12-lead ECG.
   d. A normal 15-lead ECG reliably excludes AMI in chest pain patients.

14. Body surface mapping:
   a. involves cartography of the heart and great vessels.
   b. expands upon the concept of additional ECG imaging of the heart.
   c. has markedly improved the rate of anterior wall AMI diagnosis.
   d. should be used only in those with proven STEMI.
15. With respect to the confounding ECG pattern, which of the following is FALSE?
   a. The confounding ECG pattern includes left ventricular hypertrophy.
   b. The confounding ECG pattern entirely invalidates the ECG in the evaluation of potential AMI.
   c. The confounding ECG pattern includes left bundle-branch block.
   d. The confounding ECG pattern reduces the ability of the ECG to diagnose AMI.

16. Electrocardiographic criteria for fibrinolysis in AMI includes all of the following, EXCEPT:
   a. new left bundle-branch block pattern.
   b. ST-segment elevation of 0.4 mV in leads II and III.
   c. presumably new left ventricular hypertrophy pattern.
   d. simultaneous ST-segment depression in leads II, III, and aVF, and ST-segment elevation in leads V₁ to V₆.

Class Of Evidence Definitions

Each action in the clinical pathways section of Emergency Medicine Practice receives a score based on the following definitions.

Class I
- Always acceptable, safe
- Definitely useful
- Proven in both efficacy and effectiveness
- Levels of evidence: Case series, animal studies, consensus panels
- Occasionally positive results

Level of Evidence:
- One or more large prospective studies are present (with rare exceptions)
- High-quality meta-analyses
- Study results consistently positive and compelling

Class II
- Safe, acceptable
- Probably useful
- Levels of evidence: High-quality meta-analyses
- Indeterminate: Continuing area of research
- No recommendations until further research

Level of Evidence:
- Evidence not available
- Higher studies in progress
- Results inconsistent, contradictory
- Results not compelling

Class III
- May be acceptable
- Possibly useful
- Considered optional or alternative treatments
- Results consistently positive

Level of Evidence:
- Generally lower or intermediate

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Needs Assessment: The need for this educational activity was determined by a survey of medical staff, including the editorial board of this publication; review of morbidity and mortality data from the CDC, AHA, NCHS, and ACEP; and evaluation of prior activities for emergency physicians.

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