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## Electrocardiographic Diagnosis of Acute Coronary Syndromes (ACS): A Clinical Decision Support Tool for Optimizing Patient Management

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*Perhaps no diagnostic procedure performed in emergency medicine or cardiology is as useful, predictive, complex, or as essential for guiding life-saving therapy as the electrocardiogram (ECG). The well-documented benefits of early diagnosis and rapid revascularization for acutely ischemic coronary arteries—whether it be with pharmacological or procedural interventions—in the setting of acute myocardial infarction (AMI) have served to emphasize the critical importance of competency among emergency physicians in electrocardiographic interpretation. Because the emergency physician usually is the first physician to evaluate patients with chest pain, he or she is charged with the responsibility of rapid, accurate diagnosis and appropriate, timely therapy.*

*Although the ECG has remained a time-honored, diagnostic modality in the practice of emergency medicine for decades, from the perspective of false-positive and false-negative results, the value of the ECG is potentially limited by a number of shortcomings. These include the myriad causes of "normal" and "nondiagnostic" interpretations, the variable manifestations of evolving AMI patterns, the non-ST-T wave elevation AMI, confounding and mimicking patterns, and the isolated acute posterior wall AMI. The presence of left bundle-branch block (LBBB), ventricular paced rhythms (VPRs), and left ventricular hypertrophy (LVH) present additional interpretative challenges.*

*Complicating these atypical ECG characteristics is the fact that nearly 50% of patients with AMI present to the emergency department (ED) with a normal or nondiagnostic standard 12-lead ECG, although early in the course of hospitalization, up to 20% of these patients will develop changes consistent with transmural injury. Hence, the nondiagnostic, initial ECG may inappropriately reassure the clinician who is evaluating a low-risk patient with an atypical presentation of chest pain; this may lead to discharge from the ED. Accordingly, it should be stressed that in patients with continuous chest pain and an initially unremarkable ECG, serial acqui-*

*sition of a standard 12-lead ECG at frequent intervals may improve diagnostic accuracy.*

*With these clinical issues in focus, the authors of this landmark review present a systematic approach for ECG interpretation in the setting of acute ischemic oronary syndromes. To enhance the accuracy of diagnosis, a glossary of characteristic ECGs is included as a clinical decision support tool. Finally, because confirmation of AICS is an indication for prompt therapeutic intervention, a management supplement is included that outlines options for pharmacotherapeutic and procedural interventions for the entire range of AICS, including ST-elevation MI, non-ST elevation MI, and unstable angina.*

*— The Editor*

## Uses of the Electrocardiogram

In the chest pain patient, the electrocardiogram (ECG) is used to help establish the diagnosis of acute ischemic coronary syndrome or, alternatively, some other non-coronary ailment; to select appropriate therapy; to determine the response to ED-delivered treatments; to establish the correct inpatient disposition location; and to predict risk of both cardiovascular complication and death. In the case of AMI with ST segment elevation (STE), reperfusion treatments, such as thrombolysis or angioplasty and other therapies, urgently must be considered. Conversely, in the instance of the chest pain patient demonstrating STE resulting from a non-infarction 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 thrombolysis. The emergency physician also may use the ECG for monitoring response to treatment in the patient with an acute coronary syndrome. For example, thrombolytic therapy has been reported to cause more rapid resolution of STE.

The initial 12-lead ECG obtained in the ED can be a helpful guide for determination of cardiovascular risk and, as such, the choice of in-hospital admission location. Brush and colleagues have classified initial ECGs into high- and low-risk groups.[1] The low-risk electrocardiographic group had absolutely normal ECGs, non-specific ST-T wave changes (NSSTTW), or no change when compared with a previous ECG. High-risk ECGs had significant abnormality or confounding pattern, such as pathologic Q waves, ischemic ST segment or T wave changes, LVH, LBBB, or VPR. Patients who have initial ECGs that are classified as low risk have a 14% incidence of AMI, 0.6% incidence of life-threatening complications, and a mortality rate of 0%.[1] Patients who have initial ECGs that are classified as high risk have a 42% incidence of AMI, 14% incidence of life-threatening complications, and a mortality rate of 10%.[1] Another approach to risk prediction involves a simple calculation of the number of electrocardiographic leads with ST segment deviation (elevation or depression), with an increasing number of leads being associated with higher risk.[1] Along similar lines, the clinician also is able to qualitatively predict risk by a summation of the total millivolts of ST segment deviation; once again, higher totals are associated with greater risk.[1]

**Limitations of the Electrocardiogram.** From the perspective of the electrocardiographic diagnosis of AMI, the ECG has numerous shortcomings, including the "normal" and "nondiagnostic" interpretations, evolving AMI patterns, the non-STE AMI, confounding and mimicking patterns, and the isolated acute posterior wall AMI. The ECG that provides clear evidence of acute ischemia without obvious infarct—clearly not a diagnostic dilemma—is encountered in approximately 50% of patients found to have myocardial infarction. The remaining patients represent the potential electrocardiographic diagnostic dilemma group. Within this segment of patients, the ECG may be entirely normal, nonspecifically abnormal, or clearly abnormal yet without definitive pathologic STE indicative of AMI. Refer to Table 1 for a review of the electrocardiographic findings in the chest pain patient and the association with ultimate diagnosis.

In a classic study of adult chest pain patients managed in the ED, Lee and colleagues found that approximately 20% of such patients had an absolutely normal 12-lead ECG.[2] The description "absolutely normal" translates into the absence of NSSTTW changes, atrioventricular block, intraventricular conduction delay, repolarization changes, and rhythms other than sinus rhythm. The

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**Table 1. Electrocardiographic Findings in the ED Chest Pain Patient with Respect to the Final Hospital Diagnosis[2]**

| INTERPRETATION | AMI | USAP* | NONCORONARY DIAGNOSES |
|---|---|---|---|
| Normal | 1% | 4% | 95% |
| Nonspecific | 3% | 23% | 75% |
| Abnormal (not % diagnostic of ACS) | 4% | 21% | 75% |
| Abnormal (changes documented previously) | 7% | 48% | 45% |
| Abnormal (changes not documented previously) | 25% | 43% | 32% |
| Infarction | 73% | 13% | 14% |

* USAP = Unstable angina pectoris

final hospital diagnoses of this group of chest pain patients with a normal 12-lead ECG in the ED included numerous gastrointestinal, musculoskeletal, and pulmonary syndromes, as expected; this group also was composed of a minority of patients with the final hospital diagnoses of unstable angina pectoris (USAP) (4%) and AMI (1%). This highlights the fact that a small minority of chest pain patients with an absolutely normal ECG ultimately will be found to have acute coronary ischemic syndromes. Similar issues have been reported by Rouan and associates, who demonstrated that patients with a normal ECG and classic symptoms of angina are at risk of an acute coronary ischemic syndrome, with 3% of patients with final hospital diagnosis of AMI.[3] The clinical history must be heavily relied upon in patients with either normal or nonspecifically abnormal ECGs and a convincing description of ischemic chest discomfort. As with so many diagnoses made in the ED, management and disposition decisions primarily must be made based upon the history and not on the nondiagnostic study.

The ECG may be described as "nondiagnostic" if NSSTTW changes are noted. These nonspecific changes are defined as less than 1 millimeter (mm) ST segment depression (STD), or STE with or without abnormal morphology and blunted, flattened, or biphasic T waves without obvious inversion or hyperacuity. Other electrocardiographic issues that may produce nondiagnostic changes are sinus tachycardia and bradycardia or artifactual issues such as a wandering or irregular baseline. Lee and coworkers noted that adult chest pain patients with nonspecific or other nondiagnostic electrocardiographic features had a relatively low risk of AMI ranging from 3% to 4% but a significant risk of unstable angina, which occurred in approximately one-fifth of all such cases.[2] Other investigators have found that approximately 6% of patients with AMI demonstrate a "nonspecifically abnormal" ECG on presentation.

Additionally, it has been shown that over-reliance on a normal or nonspecifically abnormal ECG in a patient with anginal chest pain who currently is sensation-free—either due to spontaneous resolution or medical therapy—should be avoided. Patients with an initially nondiagnostic ECG who later develop AMI during that hospitalization more often are sensation-free or minimally uncomfortable on presentation; these patients also frequently lack a history of ischemic heart disease. Further, the total elapsed time from chest pain onset in patients with normal ECGs does not assist in "ruling out" the possibility of AMI in chest pain patients with a single electrocardiographic observation. Although the negative predictive value of the ECG is quite high, it is not 100%, even up to 12 hours after the onset of the patient's chest symptoms.[4] Once again, the patient's history of the event is more helpful for determining ED dispostion, particularly so if they are pain-free when the ECG is obtained.

Alternatively, in a somewhat different application of the term, the nondiagnostic ECG initially is encountered in approximately 50% of patients who ultimately are found to have experienced a myocardial infarction. With this use of the descriptor "nondiagnostic," the clinician is referring to the lack of pathologic STE noted on the ECG. Significant STD and/or T wave changes may be seen in these situations—findings certainly suggestive of an active coronary ischemic event.

The electrocardiographic abnormalities associated with AMI may be masked by the altered patterns of ventricular conduction encountered in patients with confounding patterns; classically, these patterns include LBBB, VPR, and LVH. These patterns can obscure and/or mimic the typical electrocardiographic findings of AMI. Common medical opinion holds that the electrocardiographic diagnosis of AMI is at least difficult if not impossible in the presence of such findings when, in fact, this diagnosis is at times straightforward when the physician is aware of the characteristics of these patterns.

## Electrocardiographic Abnormalities of Acute Ischemic Coronary Syndromes

The earliest electrocardiographic finding resulting from AMI is the hyperacute T wave (see Figure 1 in ECG supplement), which may appear minutes after the interruption of blood flow; the R wave also increases in amplitude at this stage. The hyperacute T wave, a short-lived structure that evolves rapidly into STE, often is asymmetric, with a broad base; these T waves also are not infrequently associated with reciprocal STD in other electrocardiographic leads. Such a finding on the ECG is transient in the AMI patient; progressive STE usually is the typical pattern encountered. The differential diagnosis of the hyperacute T wave includes transmural AMI, hyperkalemia, benign early repolarization (BER), acute pericarditis, and LVH. *(See Figure 2 in the ECG supplement.)*

As the infarction progresses, the hyperacute T wave evolves further into the giant R wave (see Figure 3 in ECG supplement), yet another transient structure. The giant R wave is best described as an intermediate electrocardiographic structure, occurring after the development of the hyperacute T wave and before the appearance of typical STE. The giant R wave is formed when the ST segment elevates and combines with the prominent R wave, particularly in the anterior distribution; the giant R wave also has been referred to as a "tombstone" on the ECG. In many instances, this structure is not seen; it usually is very transient in nature and may be missed entirely.

With continued infarction, the ST segment assumes a more typical morphology. *(See Figure 4 in the ECG supplement.)* The initial upsloping portion of the ST segment usually is either convex or flat; if the STE is flat, it may be either horizontally or obliquely so.

**Table 2. Electrocardiographic Differential Diagnosis of ST Segment Elevation**

- Acute myocardial infarction (AMI)
- Vasospastic angina
- Benign early repolarization (BER)
- Acute pericarditis
- Left ventricular hypertrophy (LVH)
- Left ventricular aneurysm
- Bundle-branch block
- Myocarditis
- Hyperkalemia
- Cardiomyopathies
- Ventricular paced rhythms (VPRs)

**Table 3. Electrocardiographic Differential Diagnosis of ST Segment Depression**

- Myocardial ischemia
- Non-ST segment elevation AMI
- Posterior wall AMI (leads $V_1$, $V_2$, and/or $V_3$)
- Digoxin effect
- Rate-related event
- Left ventricular hypertrophy (LVH)
- Bundle-branch block
- Ventricular paced rhythms (VPRs)
- Pericarditis

An analysis of the ST segment waveform may be particularly helpful with distinguishing among the various causes of STE and identifying the AMI case. This technique (see Figure 5 in the ECG supplement) 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). Patients with non-infarctional STE (i.e., with early repolarization or LVH-related changes) tend to have a concave waveform morphology. Conversely, patients with STE due to AMI have either obliquely flat or convex waveforms.[5] The use of this STE waveform analysis in ED chest pain patients markedly may increase the sensitivity for the correct electrocardiographic diagnosis of AMI. This morphologic observation should be used only as a guideline. As with most guidelines, it is not perfect. The electrocardiographic differential diagnosis of STE is broad (see Figure 6 in the ECG supplement and Table 2), including AMI, bundle-branch block, LVH, VPRs, pericarditis, and left ventricular aneurysm, among many other entities.

STD (see Figure 7 in the ECG supplement) generally is considered to represent subendocardial, noninfarctional ischemia, though STD may be the presenting electrocardiographic finding in non-STE AMI. The morphology of subendocardial ischemic STD is classically horizontal or down sloping; upsloping STD also can be seen, yet less often is associated with an acute ischemic event. With subendocardial ischemia, the STD often is diffuse and can be located in both the anterior and inferior leads. STD also occurs with non-STE AMI and transmural AMI that is associated with primary STE and reciprocal STD; also, STD in the right precordial leads may represent posterior wall AMI. Ischemic and non-ischemic causes of STD (see Figure 8 in the ECG supplement and Table 3) include myocardial ischemia, non-STE AMI, posterior wall AMI (in leads $V_1$, $V_2$, and/or $V_3$), bundle-branch block, LVH with strain, digitalis effect, hyperventilation, and hypokalemia.

Reciprocal STD, also known as reciprocal change, is defined as STD in leads that are separate and distinct from leads reflecting STE. The STD is either horizontal or down-sloping. *(See Figure 9 in the ECG supplement.)* Reciprocal change in the setting of transmural AMI identifies a patient with an increased chance of poor outcome and, therefore, an individual who may benefit from a more aggressive approach in the ED. Furthermore, its presence on the ECG supports the diagnosis of AMI with very high sensitivity and positive predictive values of greater than 90%.[6] The use of reciprocal change in both prehospital and ED chest pain patients retrospectively increased the diagnostic accuracy in the electrocardiographic recognition of AMI. Reciprocal change is seen in approximately 75% of inferior wall AMIs and much less often in anterior wall myocardial infarctions (30%).

Inverted T waves (see Figure 10 in the ECG supplement) produced by myocardial ischemia classically are narrow and symmetric. T wave inversion associated with acute ischemic coronary syndrome is morphologically characterized by an isoelectric ST segment that usually is bowed upward and followed by a sharp symmetric downstroke. The terms coronary T wave and coved T wave have been used to describe these T wave inversions. Prominent, deeply inverted, and widely splayed T waves are more characteristic of noninfarctional, nonischemic conditions such as cerebrovascular accident. *(See Figure 11 in the ECG supplement.)* Noninfarctional causes of T wave inversion include juvenile T wave patterns, LVH, acute myocarditis, Wolff-Parkinson-White (WPW) syndrome, acute pulmonary embolism, and cerebrovascular accident.

An important subgroup of patients with unstable angina often have deep T wave inversions (see Figure 12 in the ECG supplement, upper panel) in the precordial leads ($V_1$ through $V_4$); the T wave also may be biphasic in this same distribution (see Figure 12 in the ECG supplement, lower panel). The syndrome is important to recognize because it is highly specific for stenosis of the left anterior descending coronary artery with anterior wall AMI as the natural history. This has been termed the left anterior descending T wave, or Wellen's, syndrome. T wave inversion also may be caused by non-STE infarction and evolving states of STE AMI.

In general, Q waves represent established myocardial necrosis. Q waves alone rarely are the sole manifestation of AMI. Pathologic Q waves may be caused by a previously unrecognized prior infarction, or conversely, a prior myocardial infarction may mask ischemic extension in the same anatomic location. Approximately 10% of healthy young men have Q waves in the inferior leads.[5] Q waves usually develop within 8-12 hours after a transmural AMI yet they may be noted as early as 1-2 hours after the onset of complete coronary occlusion. As such, the simultaneous presence of Q waves and STE (see Figure 13 in the ECG supplement) does not preclude consideration of thrombolytic therapy.

**Anatomic Location of AMI.** It is important to be able to identify the anatomic location of an AMI to estimate the amount of jeopardized myocardium and to determine the relative risk of morbidity and mortality. The extent of myocardial necrosis varies

## Table 4. Electrocardiographic Leads, ST Segment Finding, and Involved Anatomic Segment of the Infarcting Heart

| ANATOMIC SEGMENT INVOLVED IN AMI | ELECTROCARDIOGRAPHIC LEADS | ST SEGMENT FINDING |
|---|---|---|
| Anterior Wall of LV* | $V_1$-$V_4$ | Elevation |
| Anterolateral Walls of LV | $V_1$-$V_6$ (± I & AVL) | Elevation |
| Lateral Wall of LV | I & AVL and/or $V_5$ & $V_6$ | Elevation |
| Inferior Wall of LV | II, III, & AVF | Elevation |
| Posterior Wall of LV | $V_1$-$V_3$ // $V_8$ & $V_9$ | Depression // Elevation |
| Right Ventricle | $RV_4$ (or $RV_1$-$RV_6$) | Elevation |

* LV = Left ventricle

## Table 5. Electrocardiographic Findings of Acute Posterior Wall AMI*

- Horizontal ST segment depression
- Tall, upright T wave
- Tall, wide R wave
- R/S wave ratio ≥ 1.0
- ST segment elevation (in leads $V_8$ & $V_9$)

* In leads $V_1$, $V_2$, or $V_3$ with the exception of the posterior leads $V_8$ and $V_9$

greatly with the site of occlusion and presence of collateral circulation. As an obvious example, a proximal occlusion of the left anterior descending (LAD) artery usually results in much more extensive anterior wall myocardial necrosis than mid-artery occlusion. See Table 4 for a listing of electrocardiographic leads, ST segment finding, and involved anatomic segment of the infarcting heart. Electrocardiographic identification of anterior wall AMI (see Figure 14 in the ECG supplement) is made using the standard precordial leads $V_1$ to $V_4$. If STE occurs in one or more of leads $V_1$ to $V_4$ only, it is termed either anterior or anteroseptal. Extension to the lateral wall (anterolateral AMI, see Figure 15 in the ECG supplement) results in additional STE located in leads I, aVL, $V_5$, or $V_6$.

The lateral wall of the left ventricle variably is supplied by the left circumflex coronary artery, the LAD coronary artery, or a branch of the right coronary artery. Isolated lateral wall infarctions (see Figure 16 in the ECG supplement) usually involve occlusion of the circumflex artery. More commonly, the lateral wall is involved with proximal occlusion of the LAD artery (anterolateral AMI) or a branch of the right coronary artery (inferolateral AMI).

The right coronary artery supplies the AV node and inferior wall of the left ventricle in 90% of patients. The left circumflex artery performs this function in the 10% of patients who have a dominant left coronary artery. Inferior AMIs (see Figure 17 in the ECG supplement) are characterized by STE in at least two of the inferior leads (II, III, and aVF). Patients with inferior wall AMI with electrocardiographic evidence of more extensive infarction, such as reciprocal changes in the anterior precordium, lateral extension with STE in leads $V_5$ and $V_6$, or right ventricular infarction identified by STE in the right precordial lead $V_4R$ are at risk of in-hospital mortality.

The term posterior myocardial infarction (PMI) refers to infarction involving the posterior wall of the left ventricle, resulting from occlusion of either the right coronary artery, its posterior descending branch, or less commonly, the circumflex artery. Posterior wall involvement occurs in 15-21% of all AMIs, usually in conjunction with inferior or lateral infarction. *(See Figure 18 in the ECG supplement.)* PMI can occur as an isolated phenomenon (see Figure 19 in the ECG supplement), but the incidence probably is low. Patients experiencing acute inferior wall AMI with either right precordial (leads $V_1$, $V_2$, and/or $V_3$) STD or posterior lead (leads $V_8$ and $V_9$) STE (see Figure 20 in the ECG supplement), in general have larger-sized myocardial infarctions with lower resultant ejection fractions and higher rates of acute cardiovascular complication and death compared to patients with inferior AMIs without such changes.[7]

As the standard 12-lead ECG does not include posterior leads, changes associated with necrosis in this region are reflected in the anterior chest leads. These electrodes are opposite rather than adjacent to the site of damage and the changes seen are the reverse of what one would normally expect. Thus, the STE that occurs with PMI becomes STD in the right precordial leads: $V_1$ through $V_3$ (see Figure 20 in the ECG supplement). Abnormalities most frequently are detected in leads $V_1$ and $V_2$, and to a lesser extent in lead $V_3$. Electrocardiographic abnormalities (see Figure 20 in the ECG supplement and Table 5) noted on the standard 12-lead ECG that are suggestive of acute PMI include the following (in leads $V_1$, $V_2$, or $V_3$): 1) horizontal STD; 2) a tall, upright T wave; 3) a tall, wide R wave; and 4) an R/S wave ratio of 1.0 or greater. Further, the combination of horizontal STD with an upright T wave increases the diagnostic accuracy of these two separate electrocardiographic findings. It must be remembered that a dominant R wave, which is in fact an evolving Q wave, takes a number of hours to develop and, therefore, is not frequently seen on the initial ECG. The use of the additional posterior leads, $V_8$ and $V_9$ (see Figure 20 in the ECG supplement), may well confirm the presence of a PMI and are felt to be superior to the findings noted in leads $V_1$ through to $V_3$.

Right ventricular infarction occurs in 25-40% of inferior wall AMIs. *(See Figure 21 in the ECG supplement.)* Right ventricular involvement usually occurs as a result of occlusion of the right coronary artery proximal to the right ventricular branch—with associated acute inferior wall infarction; less commonly, right ventricular infarction results from occlusion of a dominant circumflex artery in the setting of acute lateral wall myocardial infarction. In the setting of inferior AMI, the clinical findings of hypotension and raised jugular venous pressure are highly suggestive of right ventricular infarction. It also has been suggested that nitrate-induced hypotension is suggestive of right ventricular infarction. It is important to diagnose right ventricular infarction, since the associated hypotension will likely respond to intravenous fluid administration, whereas diuretic agents, morphine, and nitrates may further compound the situation. Patients with inferior wall AMI with coexistent right ventricular infarction have larger-sized infarcts and more often experience in-hospital complications and higher cardiac mortality rates.[8]

The standard 12-lead electrocardiographic findings for right ventricular infarction include STE in the inferior distribution as well

as in the right precordial chest leads, particularly lead $V_1$ (perhaps the only lead on the standard ECG that reflects changes in the right ventricle). In the setting of inferior wall AMI, if the degree of STE is disproportionally greater in lead II relative to the other inferior leads, RV infarction also is suggested. At times, co-existing acute posterior wall AMI may obscure the STE resulting from right ventricular infarction in lead $V_1$ as seen in the patient with the acute inferoposterior myocardial infarction with right ventricular involvement. Recordings from leads placed on the right side of the chest are much more sensitive and specific for detecting the changes of right ventricular infarction. The right-sided precordial electrodes are placed across the right side of the chest in a mirror image of the standard, left-sided leads and are labeled $V_1R$ to $V_6R$; $RV_1$ to $RV_6$ is another commonly used nomenclature for this lead distribution. The clinician may use either the entire right-sided leads $V_1R$ to $V_6R$ or the single lead $V_4R$ (see Figure 21 in the ECG supplement). Lead $V_4R$ (right 5th intercostal space mid-clavicular line), is the most useful lead for detecting STE associated with right ventricular infarction and may be used solely for the evaluation of the possible right ventricular infarction. The STE that occurs in association with right ventricular infarction frequently is quite subtle, reflecting the relatively small muscle mass of the right ventricle; at other times, the STE is quite prominent, similar in appearance to the ST segment changes seen in the standard 12-lead ECG.

## Serial Electrocardiography

Nearly 50% of patients with AMI come to the ED with a normal or nondiagnostic standard 12-lead ECG, yet early in the course of hospitalization, up to 20% of these patients will develop changes that are consistent with transmural injury. *(See Figure 22 in the ECG supplement.)* The nondiagnostic ECG can falsely reassure the emergency physician who is evaluating the low-risk patient with an atypical presentation of chest pain, leading to the release of this patient from the ED. In the patient with continuous chest pain and an initially unremarkable ECG, serial acquisition of a standard 12-lead ECG at frequent intervals may improve diagnostic accuracy. *(See Figure 22 in the ECG supplement.)* In particular, serial ECGs can identify the diagnostic evolution of STE, identifying a candidate for reperfusion treatment. Serial ECG also may assist the emergency physician with the patient who has both confounding and mimicking patterns (see Figures 23 [LVH], 24 [BER], and 25 [LBBB] in the ECG supplement). For example, the chest pain patient with electrocardiographic LVH will manifest STE; initial diagnostic uncertainly can be addressed using serial ECG—the patient with STE AMI will likely demonstrate progression of the elevation while the non-ischemic individual will reveal a static pattern over the short term in the ED. The serial ECG technique may be accomplished using either frequent applications of the standard 12-lead ECG or computer-assisted ST segment trend monitoring.

Potentially, serial ECGs may provide surveillance of patients coming to the ED with chest pain and a nondiagnostic ECG on presentation.[9,10] In the cardiac care unit (CCU) setting, applying ST segment monitoring in the early stages of admission reportedly provided important, additional information not noted on the initial ED ECG.[11] In fact, 17% of such patients demonstrated dynamic electrocardiographic change during the initial six hours of CCU monitoring. Such ST segment trend monitoring may provide the earliest evidence of coronary occlusion in patients with preinfarction angina. In addition, such evaluation may provide evidence of painless or silent ischemia. Scientific support for ST segment trend monitoring in the ED is sparse. Fesmire and colleagues applied this technique to 1000 ED chest pain patients;[12] serial ST segment monitoring identified an additional 16.2% of patients with myocardial injury who were not identified on the initial ECG. The patient with the confounding ECG also may benefit from this diagnostic technique. The LBBB pattern, a classic confounder to the electrocardiographic diagnosis of AMI, may be further investigated with serial ECG. Fesmire and colleagues encountered five patients with final diagnosis of AMI in the presence of LBBB who demonstrated significant electrocardiographic change during the early phase of ED care and surveillance with frequent serial ECGs.[13]

Serial 12-lead ECG tracings obtained every 20 seconds, with computer interpretation and comparison, have been developed for the continuous monitoring of the ST segment in patients with AMI receiving thrombolytic therapy. Three-dimensional computer representations allow graphic images of initial occlusion in patients with AMI and subsequent reperfusion. In one series, Krucoff and associates note that angiographically proven reperfusion is detected with a sensitivity of 89% using serial ST segment trend monitoring, with a corresponding specificity of 82%.[14] ST segment trend monitoring in multiple investigations has proved to be an effective method for noninvasive evaluation of reperfusion after delivery of thrombolytic therapy.

**Additional Lead Electrocardiograms.** It has been suggested that the sensitivity of the 12-lead ECG may be improved if three additional body surface leads are employed in selected individuals. Acute posterior and right ventricular myocardial infarctions are likely to be under diagnosed, as the standard lead placement of the 12-lead ECG does not allow these areas to be assessed directly. Additional leads frequently used include $V_8$ and $V_9$ which image the posterior wall of the left ventricle, and lead $V_4R$, which reflects the status of the right ventricle. The standard ECG, coupled with these additional leads, constitutes the 15-lead ECG, the most frequently employed extra-lead ECG in clinical practice. Without doubt, a more detailed description of the extent of the myocardial injury may be obtained if additional leads are used to augment the standard 12-lead ECG in selected patients. The use of the additional leads may not only confirm the presence of AMI but also alter treatment decisions. See Figure 26 A in the ECG supplement for a representative example of a 15-lead ECG in a patient with AMI of the inferior, posterior, and lateral walls, as well as of the right ventricle.

In a study of all ED chest pain patients, one group reported that the 15-lead ECG provided a more accurate description of myocardial injury in those patients with AMI yet failed to alter rates of diagnoses, the use of reperfusion therapies, or disposition locations.[15] Looking at a more select population of ED patients, Zalenski and colleageus investigated the use of the 15-lead ECG in chest pain patients with a moderate to high pretest probability of AMI who were already identified as candidates for hospital admission.[16] In this 15-lead ECG study, the authors reported an 11.7%

increase in sensitivity with no loss of specificity (i.e., no increase in false-positive findings for the diagnosis of STE AMI).[16] They concluded that "the findings of ST elevation by use of these extra leads can strengthen the ED diagnosis of AMI on the initial tracing and may provide an indication for thrombolytic treatment."[16] They further suggest that leads $V_8$ and $V_9$ are superior for the diagnosis of PMI to the reciprocal STD seen in leads $V_1$ to $V_3$.[16]

Additional-lead ECGs may produce valuable information about injury, necrosis, and ischemia in carefully selected cases.[15,16] Indications to obtain 15-lead ECGs in patients with suspected acute ischemic heart disease include: 1) STD in leads $V_1$ through $V_3$ or suspicious isoelectric ST segments in leads $V_1$ through $V_3$; 2) borderline STE in leads $V_5$ and $V_6$, or borderline STE in leads II, III, and aVf; 3) all STE inferior AMIs (STE in leads II, III, and aVF); or 4) isolated STE in lead $V_1$ or STE in leads $V_1$ and $V_2$. The physician must realize that these indications, despite their apparent clinical utility, remain unproven. Placement of the additional leads comprising the 15-lead ECG are demonstrated in Figure 26 B in the ECG supplement.

## Confounding Electrocardiographic Patterns

**Left Ventricular Hypertrophy.** Electrocardiographic LVH and the related repolarization changes are not uncommonly encountered in the ED chest pain patient. Their presence on the ECG, particularly the repolarization changes that alter the morphology of the ST segment and/or the T wave, may confound the early ED evaluation of the chest pain patient. In patients with LVH, ST segment/T wave changes (see Figure 27 in ECG supplement) are encountered in approximately 70% of cases; these changes result from altered repolarization of the ventricular myocardium[17] and represent the new normal for the patient with electrocardiographic LVH. These changes may mask and/or mimic the early findings consistent with acute coronary ischemia (see Figure 28 in ECG supplement); this effect, however, occurs to a lesser extent than is encountered with the LBBB and VPR situations. 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 $V_3$. As predicted by the concept of appropriate discordance, STE is encountered in this distribution along with prominent, "hyperacute" T waves. The STE seen in this distribution may be greater than 5 mm in height and is difficult to distinguish from that associated with AMI. The initial, up-sloping portion of the ST segment/T wave complex frequently is concave in LVH compared to the either flattened or convex pattern observed in the AMI patient. This morphologic feature is imperfect; early AMI may reveal such a concave feature.

**Left Bundle-Branch Block.** LBBB markedly reduces the diagnostic power of the ECG. The associated—and expected—ST segment and T wave abnormalities of LBBB may mimic both acute and chronic ischemic change, potentially leading the uninformed physician to make an incorrect diagnosis and to institute potentially dangerous therapy. Further, the electrocardiographic abnormalities associated with AMI may be masked by the altered patterns of ventricular conduction encountered in patients with LBBB. Many emergency physicians are of the opinion that the electrocardiographic diagnosis of AMI is virtually impossible in the presence of LBBB. Others believe this diagnosis can be straightforward using established criteria.

In the patient with LBBB, the anticipated or expected ST segment/T wave configurations are discordant, directed opposite from the terminal portion of the QRS complex and called QRS complex-T wave axes discordance.[5] *(See Figures 29 and 30 in ECG supplement.)* As such, inferior and right precordial leads with either QS or rS complexes may have markedly elevated ST segments, which mimic AMI. The lateral leads, with the large monophasic R wave, demonstrate STD. The T wave, especially in the right to mid precordial 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 frequently are inverted. Loss of this normal QRS complex/T wave axes discordance in patients with LBBB may imply an acute process, such as AMI. An inspection of the ECG in patients with LBBB must be performed, looking for a loss of this QRS complex vs. ST segment/T wave axes discordance.

Sgarbossa and colleagues have developed a clinical prediction rule to assist with the ECG diagnosis of AMI in the setting of LBBB using specific electrocardiographic findings.[18] They analyzed the numerous electrocardiographic abnormalities previously reported as suspicious or diagnostic for AMI in patients with LBBB and identified three criteria that are suggestive of acute infarction. This rule, developed from 131 patients with LBBB and enzymatically proven AMI who were enrolled in the Global Utilization of Streptokinase and Tissue Plasminogen Activator for Occluded Coronary Arteries (GUSTO-I) trial,[18] reported that three specific ECG criteria were independent predictors of myocardial infarction. The ECG criteria (see Figure 31 A in the ECG supplement) suggesting a diagnosis of AMI, ranked with a scoring system based on the probability of such a diagnosis, include: 1) STE greater than 1 mm that was concordant with the QRS complex (score of 5); 2) STD greater than 1 mm in leads $V_1$, $V_2$, or $V_3$ (score of 3); and 3) STE greater than 5 mm that is discordant with the QRS complex (score of 2). A total score of 3 or more suggests that the patient likely is experiencing an AMI based on the electrocardiographic criteria. With a score of less than 3, the electrocardiographic diagnosis is less assured, requiring additional evaluation. Refer to Figure 31 B in the ECG supplement for an example AMI noted in the setting of LBBB.

Subsequent literature has suggested that the Sgarbossa et al clinical prediction rules are less useful than reported.[18-20] The first such investigation,[19] which applied the Sgarbossa et al[18] criteria to patients with chest pain and LBBB in the ED of a North American hospital, found much less promising results—a very low sensitivity coupled with poor interobserver reliability. A second study investigated the diagnostic and therapeutic effect of this criteria; none effectively distinguished the patients who had AMI from those patients with noncoronary diagnoses.[20] The authors concluded that electrocardiographic criteria are poor predictors of AMI in LBBB situations and suggested that all patients suspected of AMI with LBBB should be considered for thrombolysis. A third investigation

followed just this recommendation—using a thrombolytic agent in all patients with LBBB presumed to have AMI;[21] they also reported an alarmingly high rate of inappropriate thrombolysis in chest pain patients with LBBB and presumed AMI (49%). The authors also retrospectively investigated the effect of the Sgarbossa et al criteria on the diagnosis and management.[18,21] These investigators, in contrast to the previously noted reports, found significant accuracy using the Sgarbossa et al criteria[18]—noting approximately an 80% rate of correct diagnosis using the prediction rule. Had the clinical prediction rule been employed, the authors suggest that inappropriate thrombolysis would have been avoided in many instances.

Traditional criteria for administration of thrombolytic agents in the AMI patient most often involve electrocardiographic STE situated in an anatomic distribution; the presence of a new LBBB pattern represents another electrocardiographic criterion for such therapy. This second criterion suggests that appropriate patients with LBBB pattern and a history suggestive of AMI receive a thrombolytic agent if it is not otherwise contraindicated. This approach is perhaps reasonable if the physician has a high suspicion of AMI and is comfortable initiating thrombolysis based solely on clinical information—in other words, an analysis of the patient's history and physical examination. Physicians, however, may be uncomfortable administering a thrombolytic agent under such circumstances; in fact, patients with electrocardiographic LBBB and AMI less often receive thrombolysis despite an increased risk of poor outcome[18,22] and the potential for significant benefit.[23] The clinician must realize that of all patients with chest pain, electrocardiographic LBBB pattern without obvious infarction, and clinically presumed AMI, only a minority actually will be experiencing AMI.[18] Treating all such patients with LBBB and presumed AMI will subject a number of non-infarction patients to the significant risks and expense of thrombolysis.[21]

Nonetheless, even if the Sgarbossa et al clinical prediction rule is found to be less useful in the objective evaluation of the ECG in the patient with LBBB,[18] the report has merit. It has forced the clinician to review the ECG in detail and cast some degree of doubt on the widely taught belief that the ECG is not useful for diagnosis of AMI in patients with LBBB.

**Ventricular Paced Rhythms.** As with the LBBB pattern, the right ventricular paced rhythm (VPR) pattern may both mimic and mask the manifestations of acute ischemic coronary syndrome. In VPR, the ventricular depolarization pattern is abnormal, with activation of the ventricles occurring from the right to the left, and resembling a LBBB pattern in part. In the patient with VPR, the ECG records the altered ventricular activation as it moves from right to left, producing a broad, mainly negative QS or rS complex in leads $V_1$ to $V_6$ with either poor R wave progression or QS complexes. A large monophasic R wave is encountered in leads I and aVL and, on occasion, in leads $V_5$ and $V_6$. QS complexes also may be encountered in leads II, III, and aVf. The expected ST segment/T wave configurations are discordant (see Figure 32 in the ECG supplement), directed opposite from the terminal portion of the QRS complex and are similar to the electrocardiographic principles applied in the setting of LBBB. As such, leads with QS complexes may have marked STE, mimicking AMI. Leads with large monophasic R waves demonstrate STD. 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 frequently are inverted. An inspection of the ECG in patients with VPR must be performed, looking for a loss of this QRS complex/T wave axes discordance. Loss of this normal QRS complex/T wave axes discordance in patients with VPR may imply an acute process.[24]

Sgarbossa et al published a report detailing the electrocardiographic changes encountered in patients with VPR experiencing AMI[25]—a report similar to their work in patients with LBBB and AMI. Three electrocardiographic criteria were found to be useful in the early diagnosis of AMI, including: 1) discordant STE of 5 mm or greater; 2) concordant STE of 1 mm or greater; and 3) STD of 1 mm or greater in leads $V_1$, $V_2$, or $V_3$.[25] The physician must realize, however, that these ST segment changes are only suggestions of AMI in patients with complicated ECGs; clinical decisions must be based upon an analysis of the clinical history, electrocardiographic results, and in most cases, the results of other investigations such as serum markers, nuclear scans, and echocardiography. Conversely, their absence does not rule out the possibility of AMI. Therapeutic decisions must be made with these caveats in mind.

Figure 33 in the ECG supplement illustrates AMI in the setting of a VPR. This ECG is the second study performed in serial fashion in a chest pain patient with a pacemaker. Note the progressive change noted in the inferior leads with the development of STE.

**Non-ST Segment Elevation AMI.** It has been reported that total occlusion of the infarct-related artery is uncommon in the early hours after non-STE myocardial infarction (formerly the non-Q wave infarction). When total occlusion is present, perfusion is maintained by collateral vessels. These anatomic findings support the contention that the non-STE infarction often indicates an incomplete ischemic event, with additional myocardium at risk. Patients with non-STE infarction may have transient and nonspecific findings, such as STD or T wave abnormalities in any of the anatomic leads of the 12-lead ECG. Symmetric, convex, downward STD or inverted or biphasic T waves characteristically are seen. Differentiating non-STE anterior AMI from posterior AMI can be difficult. The additional leads $V_8$ and $V_9$ may further assist this differentiation.

**Patterns that Resemble Acute Ischemic Coronary Syndromes.** Unfortunately, it is not uncommon to find STE on the ECG of the ED chest pain patient; its cause rarely involves AMI.[26] The occurrence of numerous other noninfarctional STE syndromes only reinforces the point that STE is an insensitive marker of AMI.[27] One prehospital study of adult chest pain patients demonstrated that the majority of patients manifesting STE on the ECG did not have AMI as a final hospital diagnosis; rather, LVH and LBBB accounted for the majority of the cases.[6] Further, in a review of adult ED chest pain patients with STE on the ECG, STE resulted from AMI in only 15% of this population; LVH, seen in 30% of adult chest pain patients, was the most frequent cause of this STE.[27] These non-infarct STE syndromes are not infrequently misdiagnosed as acute infarction, which then may subject the patient to unnecessary and potentially dangerous therapies and

procedures. For example, a report by Sharkey and coworkers noted that 11% of patients receiving a thrombolytic agent were not experiencing AMI.[28] The electrocardiographic syndromes producing this pseudo-infarct STE included BER (30%), LVH (30%), and various intraventricular conduction abnormalities (30%).[28]

LVH, LBBB, and VPR, which also must be considered in the electrocardiographic differential diagnosis of STE, have been discussed in the section concerning confounding patterns. Other such mimicking patterns include BER, acute pericarditis, and left ventricular aneurysm.

The syndrome of BER is felt to be a normal variant, not indicative of underlying cardiac disease. The electrocardiographic definition (see Figure 34 in the ECG supplement) of BER includes the following characteristics: 1) STE; 2) upward concavity of the initial portion of the ST segment; 3) notching or slurring of the terminal QRS complex; 4) symmetric, concordant T waves of large amplitude; 5) widespread or diffuse distribution of STE on the ECG; and 6) relative temporal stability.[5]

The STE begins at the "J" (or junction) point—the portion of the electrocardiographic cycle where the QRS complex ends and the ST segment begins. The degree of J-point elevation usually is less than 3.5 mm. This STE morphologically appears as if the ST segment has been evenly lifted upward from the isoelectric baseline at the J point. This elevation results in a preservation of the normal concavity of the initial, up-sloping portion of the ST segment/T wave complex, a very important electrocardiographic feature used to distinguish BER-related STE from STE associated with AMI. The STE elevation encountered in BER usually is less than 2 mm but may approach 5 mm in certain individuals. Eighty to ninety percent of individuals demonstrate STE of less than 2 mm in the precordial leads and less than 0.5 mm in the limb leads; only 2% of cases of BER manifest STE of greater than 5 mm. The degree of STE related to BER usually is greatest in the mid- to left precordial leads (leads $V_2$ to $V_5$). The ST segments of the remaining electrocardiographic leads less often are elevated to the extent observed in leads $V_2$ through $V_5$. The limb leads (I, II, III, aVl, and aVf) less often are observed to demonstrate STE; one large series reported that the limb leads revealed STE in only 45% of cases of BER. "Isolated" BER in the limb leads (i.e., no precordial STE) is a very rare finding. Such "isolated" STE in the inferior (II, III, and aVf) or lateral (I and aVl) leads should prompt consideration of another explanation for the observed ST segment abnormality.

Acute pericarditis produces diffuse inflammation of the superficial epicardium, resulting in a current of injury that is manifested by electrocardiographic STE (see Figure 35 in the ECG supplement). STE usually is less than 5 mm in height, observed in numerous leads, and is characterized by a concavity to its initial upsloping portion. In some instances, the STE actually may be obliquely flat; convexity of the STE, however, strongly suggests AMI. The STE due to acute pericarditis usually is noted in the following electrocardiographic leads: I, II, III, aVl, aVf, and $V_2$ to $V_6$ (essentially, all leads except $V_1$); reciprocal STD also is seen in lead aVr and occasionally in lead $V_1$. The STE most often is seen in many leads simultaneously, though it may be limited to a specific anatomic segment. If the process is focal, the inferior wall often is involved with leads II, II and aVf commonly affected. PR-segment depression (see Figure 35 in the ECG supplement, leads II, III, and aVF) associated with pericarditis is the most helpful feature for arriving at the correct electrocardiographic diagnosis; such a finding has been described as "almost diagnostic" for acute pericarditis and is best seen in lead $V_6$ and the inferior leads. Reciprocal PR segment elevation can be seen in lead aVr. *(See Figure 35 in the ECG supplement.)*

Left ventricular aneurysm is defined as a localized area of infarcted myocardium that bulges outward during both systole and diastole. Left ventricular aneurysms most often are noted after large anterior wall events but also may be encountered after post inferior and posterior wall myocardial infarctions. In most cases, the left ventricular aneurysm is manifested electrocardiographically by varying degrees of STE (see Figures 36A and B in the ECG supplement) which may be difficult to distinguish from ST segment changes due to AMI, particularly in the chest pain patient with known past myocardial infarction.[5] Left ventricular aneurysm is characterized electrocardiographically by persistent STE seen several weeks after AMI. Because of the frequent, anterior location of LVA, STE most often is observed in leads I, aVl, and $V_1$ to $V_6$. Of course, the inferior wall LVA would be manifested on the surface ECG by STE in the inferior leads; such STE, however, usually is less pronounced than the ST segment changes seen in the anterior leads. The actual ST segment abnormality due to the LVA may present with varying morphologies, ranging from obvious, convex STE to minimal, concave elevations. The distinction from STE in the AMI patient may be difficult.

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## Physician CME Questions

65. In adult chest pain patients, the ECG can be safely used in all of the the following fashions *except:*
    A. the use of thrombolytic therapy in AMI.
    B. the prediction of cardiovascular risk.
    C. the choice of inpatient unit location for admission.
    D. All of the above are correct

66. In the adult chest pain patient, the use of the 12-lead ECG is confounded by all of the following patterns *except:*
    A. benign early repolarization (BER).
    B. left ventricular hypertrophy (LVH).
    C. left bundle-branch block (LBBB).
    D. All of the above are correct

67. According to the Lee et al article,[2] which of the following electrocardiographic findings and rate (%) of abnormality among chest pain patients ultimately diagnosed with AMI is *incorrect*?
    A. Normal, 1%
    B. Nonspecific, 0%
    C. Abnormal with apparently new changes, 25%
    D. Acute infarction, 50-70%

68. The earliest electrocardiographic finding suggestive of AMI is:
    A. T wave inversion.
    B. flattening of the T wave.
    C. loss of the T wave concordance.
    D. hyperacute T wave.

69. The electrocardiographic differential diagnosis of ST segment elevation includes all of the following ECG syndromes *except*:
    A. benign early repolarization (BER).
    B. left ventricular hypertrophy (LVH).
    C. digoxin effect.
    D. AMI.

70. Electrocardiographic criteria suggestive of posterior wall AMI include which of the following?
    A. Right precordial ST segment depression
    B. Inferior wall ST segment elevation
    C. Large R wave in the right precordial leads
    D. ST segment elevation in leads $V_1$ and $V_2$.

71. In the chest pain patient with LBBB pattern and suspected AMI, all of the following statements are correct *except:*
    A. The ECG is of reduced diagnostic value.
    B. New LBBB pattern is an indication for thrombolysis.
    C. LBBB always represents AMI.
    D. LBBB identifies a high-risk patient.

72. Adjuncts to the 12-lead ECG in the diagnosis of AMI in adult chest pain patients include all of the following *except*:
    A. additional lead ECG.
    B. ST segment trend monitoring.
    C. stress testing.
    D. serial ECGs.

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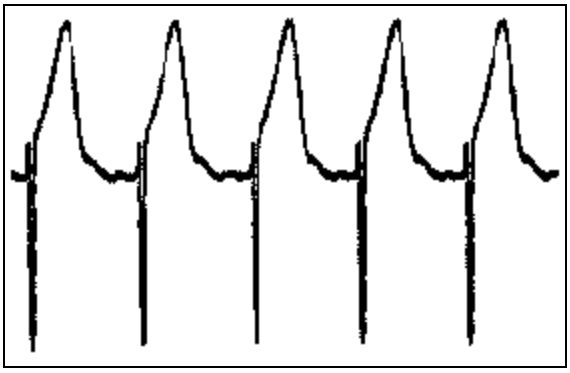
# Radiographic Diagnosis of Acute Coronary Syndromes

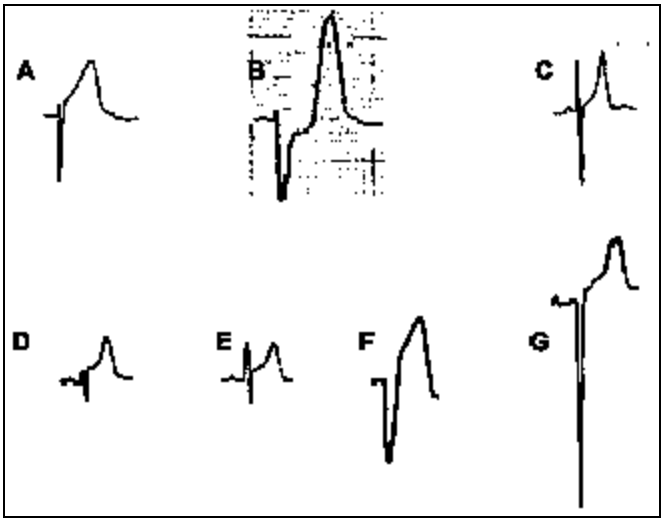
*(Enclosed for use with the April 23, 2001, issue)*

**Figure 1**



**Hyperacute T wave:** The hyperacute T wave is the earliest electrocardiographic finding encountered in the STE-AMI patient. These T waves are broad-based, asymmetric structures that rapidly evolve to more typical STE.

**Figure 2**




**Electrocardiographic differential diagnosis of the hyperacute T wave:** A, AMI. B, AMI. C, Hyperkalemia. D, Benign early repolarization (BER). E, Acute pericarditis. F, Left bundle-branch block (LBBB). G, Left ventricular hypertrophy (LVH).

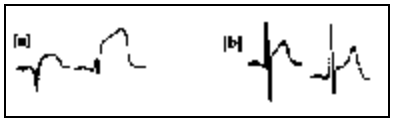
**Figure 3**

**Giant R wave:** The Giant R wave is an intermediate structure between the hyperacute T wave and the typical ST segment elevation.

**Figure 4**

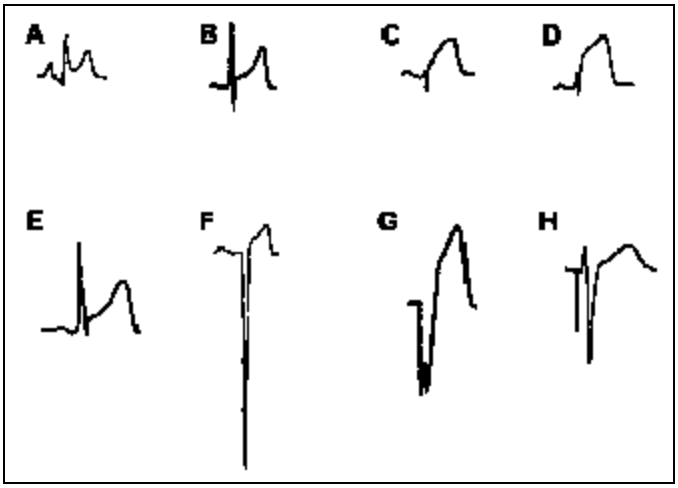
**ST segment elevation:** The morphology of the ST segment, when it is elevated in the setting of AMI, most often involves either an obliquely flat or a convex configuration.

**Figure 5**



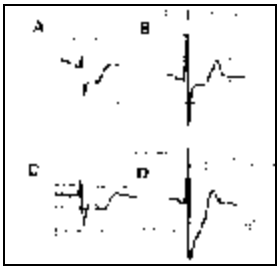

**Morphology of ST segment elevation:** The initial, upsloping portion of the ST segment usually is either flat or convex in the AMI patient (see A). This morphologic observation, however, should only be used as a guideline—it is not infallible. Patients with ST segment elevation due to non-AMI syndromes may demonstrate concavity of this portion of the waveform (see B)—pericarditis and benign early repolarization, respectively.

**Figure 6**



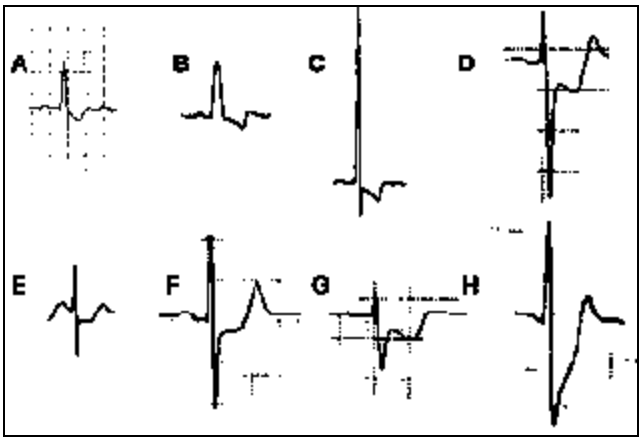

**Electrocardiographic differential diagnosis of ST segment elevation:** A, Acute pericarditis. B, Benign early repolarization (BER). C, AMI. D, AMI. E, Atypical morphology of AMI. F, Left ventricular hypertrophy (LVH). G, Left bundle-branch block (LBBB). H, Ventricular paced rhythm (VPR).

**Figure 7**



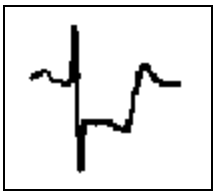

**ST segment depression as seen in acute coronary syndromes (ACS) of varying morphologies:** A, Horizontal. B, Horizontal. C, Downsloping. D, Upsloping.

**Figure 8**



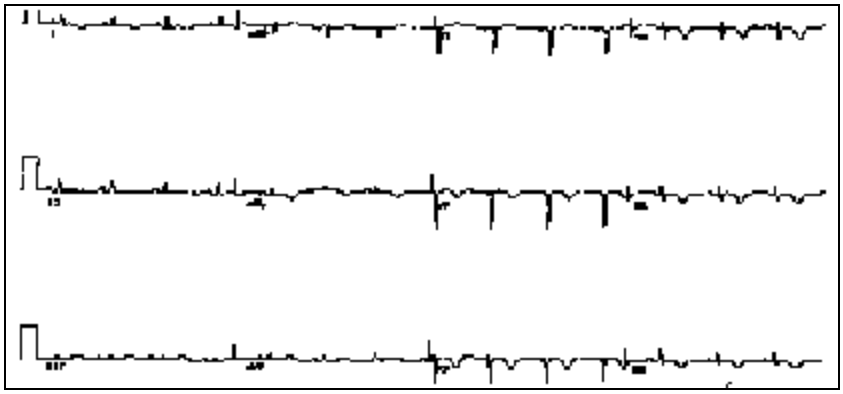

**Electrocardiographic differential diagnosis of ST segment depression:** A, Digoxin effect. B, Left bundle-branch block (LBBB). C, Left ventricular hypertrophy (LVH). D, AMI of the posterior wall (as seen in lead $V_2$). E, ST segment depression of acute coronary syndrome. F, ST segment depression of acute coronary syndrome. G, ST segment depression of acute coronary syndrome. H, ST segment depression of acute coronary syndrome.

**Figure 9**



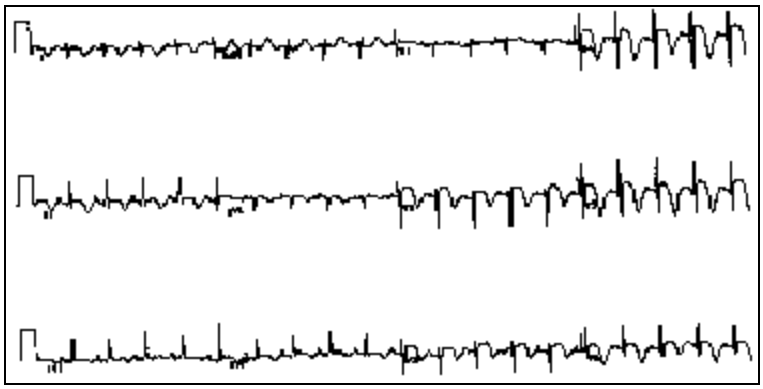
**Reciprocal ST segment depression:** In the setting of STE AMI, ST segment depression located in leads distant from the infarction is termed reciprocal change or reciprocal ST segment depression. Reciprocal change is useful diagnostically—its presence strongly suggests AMI—and prognostically—patients with such a finding have larger infarcts, lower resultant ejection fractions, and higher rates of death.

**Figure 10**



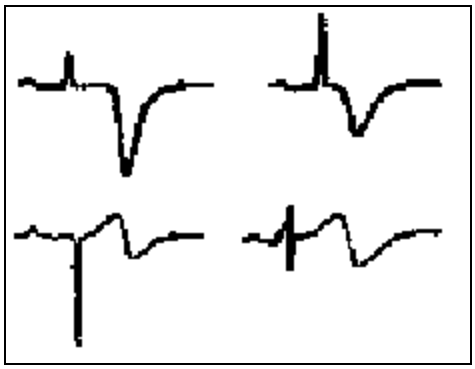
**T wave inversion associated with acute coronary syndrome (ACS) in a chest pain patient.**

**Figure 11**



**CNS T wave inversions seen in the anterolateral area.** This patient presented with severe headache and was found to have extensive subarachnoid hemorrhage by CT scan.

**Figure 12**

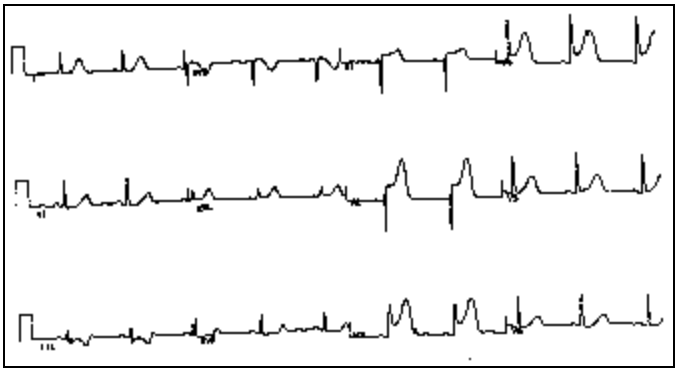


**Wellen's T waves:** Wellen's T waves occur in two basic forms. The upper examples depict the more common pattern—deeply inverted T waves. The less commonly encountered morphology, biphasic T wave, is shown in the lower panel.

**Figure 13**

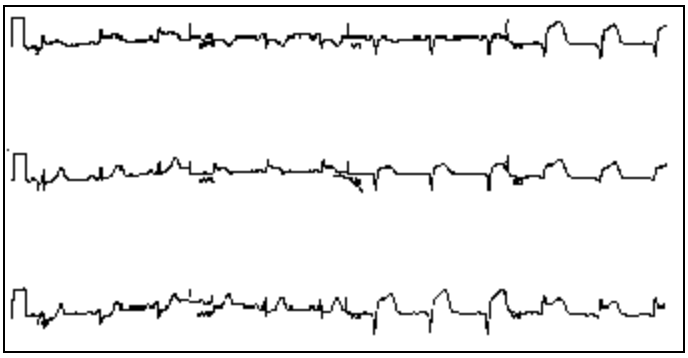
**Q wave with ST segment elevation:** Q waves most often indicate completed infarction and usually appear 9-12 hours after AMI that is not aborted. Q waves may appear as early as 1-2 hours after the onset of AMI. Such patients will present with ST segment elevation and pathologic Q waves.

**Figure 14**



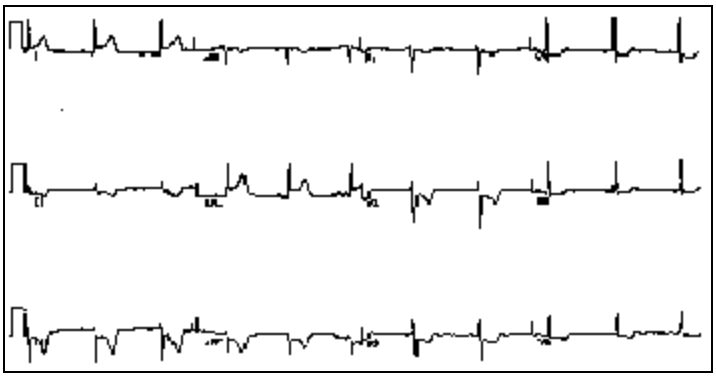
**Anterior AMI:** STE is seen in the leads $V_1$ to $V_4$, consistent with anterior wall AMI. This pattern may be described as either anterior or anteroseptal.

**Figure 15**



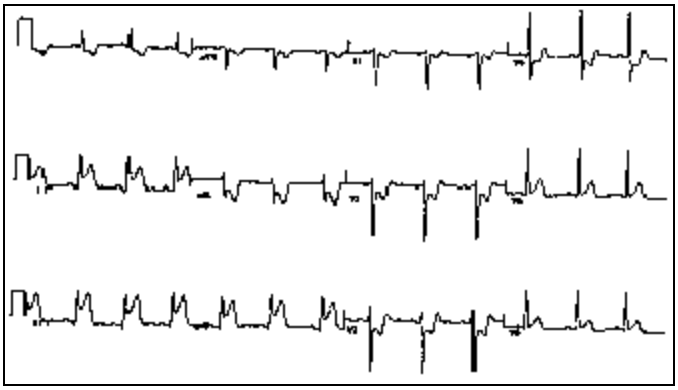
**Anterolateral AMI:** Extensive infarction is seen here with STE in leads $V_2$ to $V_4$ (anterior) and leads I, aVL, $V_5$, and $V_6$ (lateral), consistent with an anterolateral AMI.

**Figure 16**



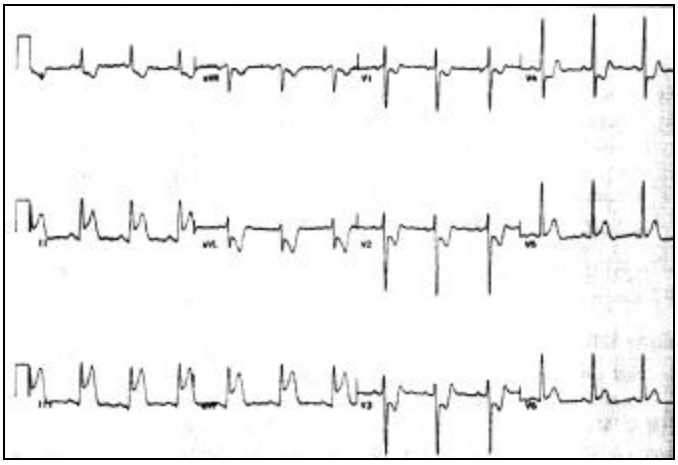
**Lateral AMI:** Isolated lateral wall AMI is seen with STE in leads I and aVL. Note the STD seen in the inferior and right precordial leads, consistent with reciprocal change. The STD in leads $V_1$ to $V_3$ also may represent posterior wall AMI.

**Figure 17**



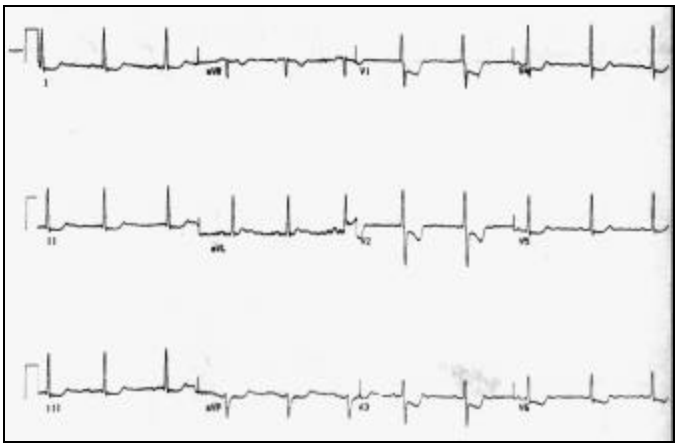
**Inferior AMI:** Inferior wall AMI is seen with STE in leads II, III, and aVF. Note the STD seen in the lateral and right precordial leads, consistent with reciprocal change. The STD in leads $V_1$ to $V_4$ also may represent posterior wall AMI.

**Figure 18**



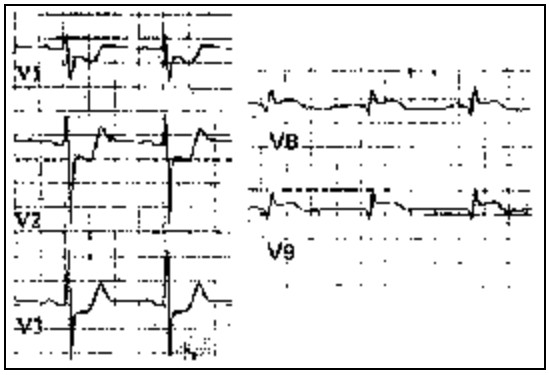
**Inferoposterior AMI:** 12-lead ECG of an acute myocardial infarction of the inferior and posterior walls of the left ventricle. In addition to the STE seen in the inferior leads, STD is encountered in the right precordial distribution. This STD is associated with a prominent R wave. These findings are suggestive of posterior wall AMI in addition to the inferior AMI. STD also is seen in leads I and aVL, consistent with reciprocal change.

**Figure 19**



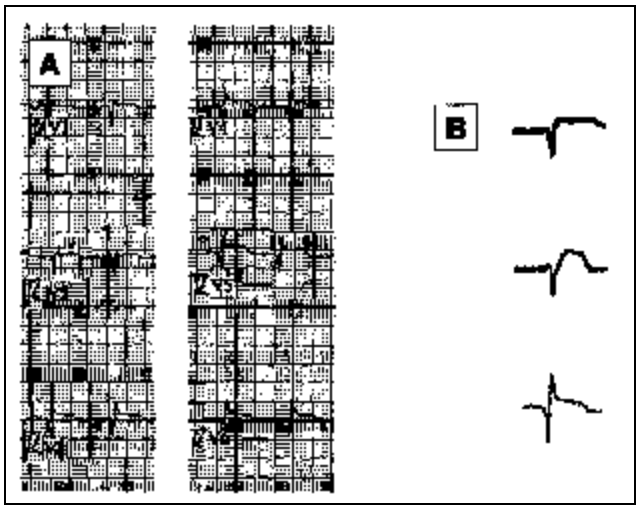
**Isolated posterior AMI:** As in Figure 18, STD is noted in the right precordial leads, consistent with an isolated posterior wall AMI.

**Figure 20**



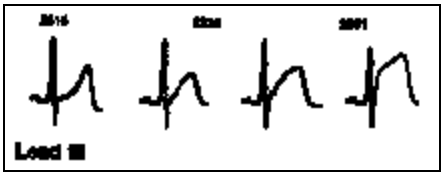

**Posterior wall AMI:** Right precordial (leads $V_1$ to $V_3$) ST segment depression and posterior thoracic leads with STE consistent with posterior wall AMI.

**Figure 21**



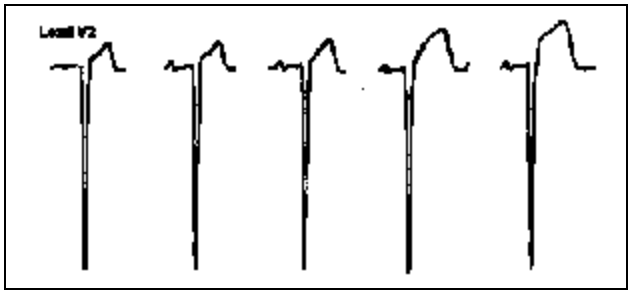

**Right ventricular infarction:** A, Right-sided anterior thoracic leads in right ventricular AMI. B, Right ventricular AMI: single lead $RV_4$.

**Figure 22**



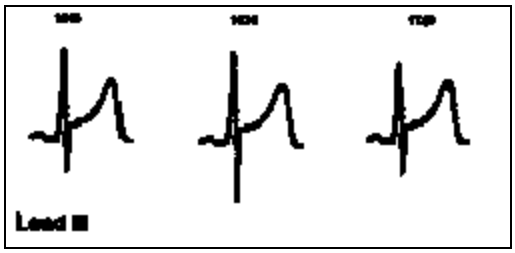

**AMI noted with serial ECGs:** Adult patient presents with chest pain and an initially normal ECG. With continued pain, serial ECGs are performed that quickly detect change, ultimately diagnostic of AMI.

**Figure 23**



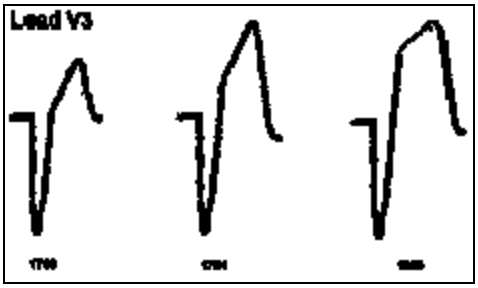

**ECGs in the AMI LVH patient:** Serial ECG demonstrating interval change consistent with AMI in the LVH pattern.

**Figure 24**



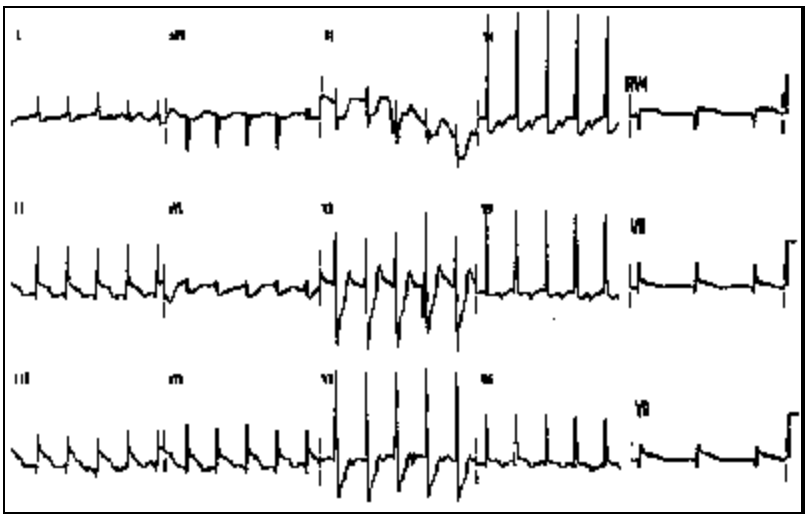

**Benign early repolarization (BER):** Serial ECG demonstrating lack of interval change in the BER pattern—confirming a non-infarction cause of the STE.

**Figure 25**



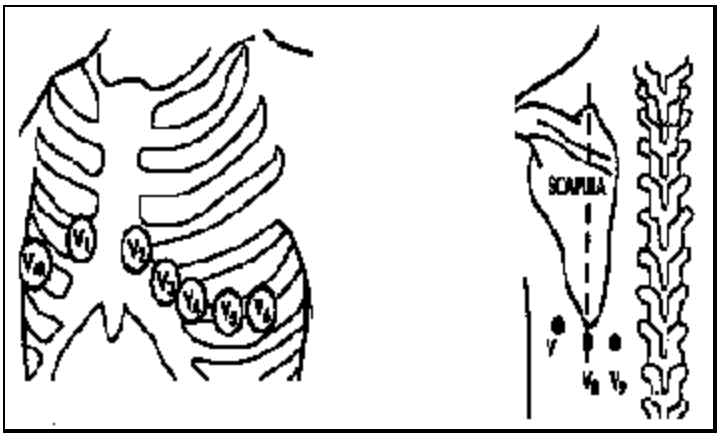

**Left bundle-branch block with electrocardiographic AMI:** Serial ECG demonstrating interval change in the LBBB pattern complicated by AMI.

Figure 26a



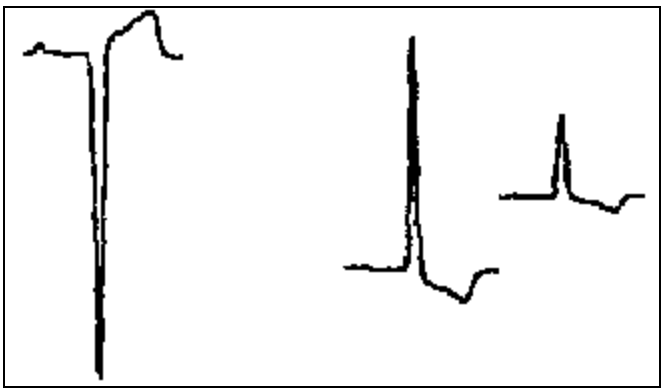
**A 15-lead ECG** with AMI of inferior, posterior, and right ventricular segments.

Figure 26b



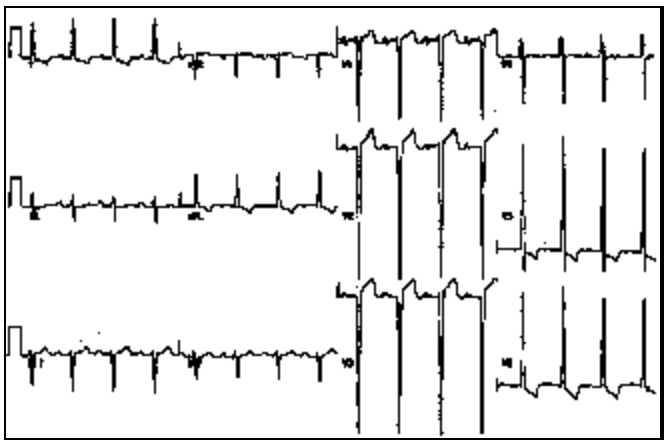

**Placement of the additional electrocardiographic leads of the 15-lead ECG:** Lead $RV_4$ is placed in a similar position to lead $V_4$ yet on the right thorax. The posterior leads $V_8$ and $V_9$ are placed on the patient's left back—$V_8$ at the tip of the scapula and $V_9$ in an intermediate position between lead $V_8$ and the left paraspinal muscles. The additional "V" notation located lateral to $V_8$ also may be used and is termed $V_7$.

Figure 27



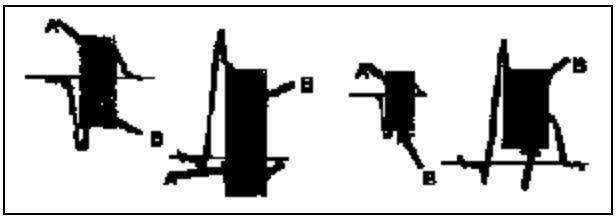
**Left ventricular hypertrophy:** Electrocardiographic changes associated with the LVH pattern.

Figure 28



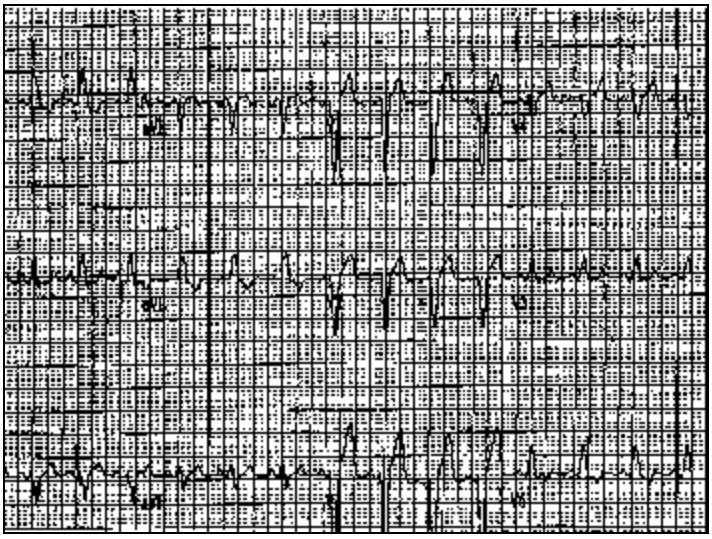
**12-lead ECG with LVH:** No AMI was found in this patient; the ST/T changes represent repolarization change associated with LVH.

Figure 29



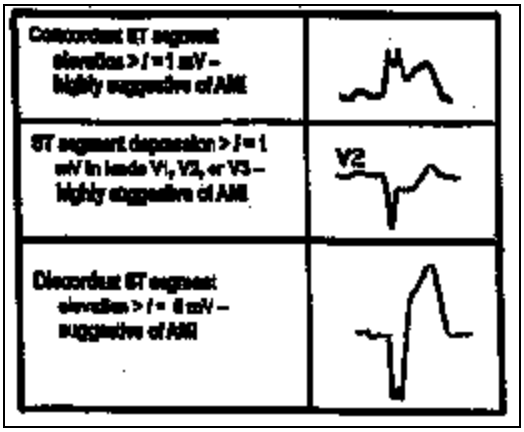
**The concept of appropriate discordance in the LBBB pattern:** The shaded areas note the portions of the waveform which must be evaluated in the LBBB pattern. In all examples listed, "A" depicts the initial portion of the ST segment/T wave complex while "B" refers to the major terminal segment of the QRS complex. The appropriate relationship of the ST segment to the T wave in the LBBB pattern is one of discordance ( i.e., the major terminal portion of the QRS complex and the ST segment/T wave complex must be on opposite sides of the isoelectric baseline). This "normal" relationship is seen in the two examples on the left. Abnormal relationships are seen in the two examples on the right—near right, concordant ST segment depression and, far right, concordant ST segment elevation. In both of these cases, such findings suggest acute coronary ischemia.

**Figure 30**



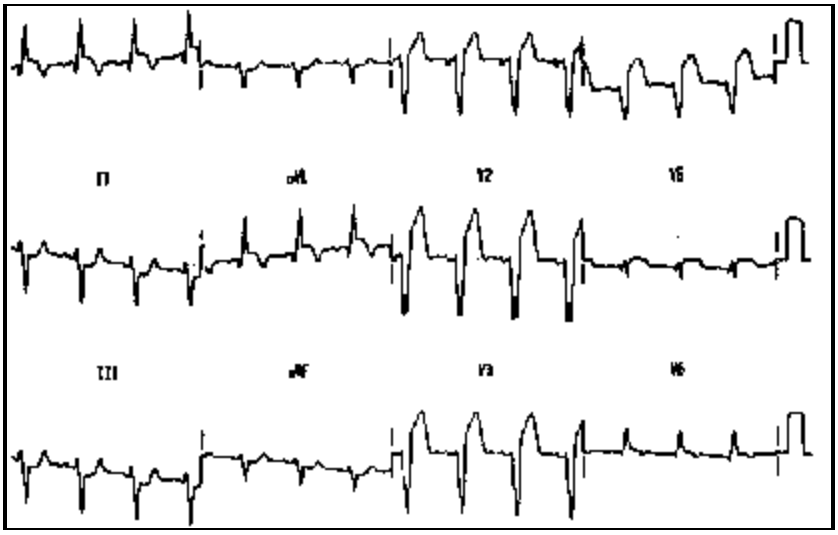
**LBBB pattern without electrocardiographic AMI:** In the patient with LBBB, the anticipated or expected ST segment-T wave configurations are discordant, directed opposite from the terminal portion of the QRS complex, and called QRS complex-T wave axes discordance. As such, leads with either QS or rS complexes may have markedly elevated ST segments, mimicking AMI. Leads with a large monophasic R wave demonstrate ST segment depression. The T wave, especially in the right to mid precordial 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 frequently are inverted.

**Figure 31a**



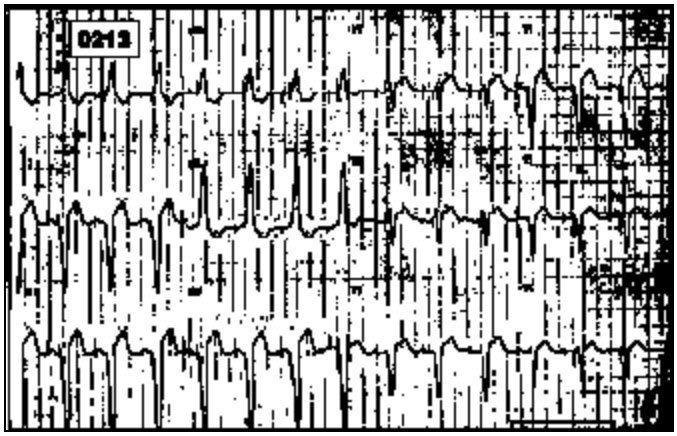

**Electrocardiographic criteria of the diagnosis of AMI in LBBB:** The electrocardiographic criteria suggesting a diagnosis of AMI according to Sgarbossa et al[18] include on the left (1) ST segment elevation greater than one millimeter which is concordant with the QRS complex (score of 5); in the middle (2) ST segment depression greater than 1 mm in leads $V_1$, $V_2$, or $V_3$ (score of 3); and on the right (3) ST segment elevation greater than 5 mm that is discordant with the QRS complex (score of 2). A total score of 3 or more suggests that the patient is likely experiencing an acute infarction based on the electrocardiographic criteria. With a score of less than 3, the electrocardiographic diagnosis is less assured, requiring additional evaluation.
Source: Brady WJ. Mastering the Electrocardiogram: State-of-the-art techniques for evaluating ST segment elevation in acute myocardiol infarction and other clinicl syndromes. *Emergency Medicine Reports* 1998;19:78-85.

**Figure 31b**



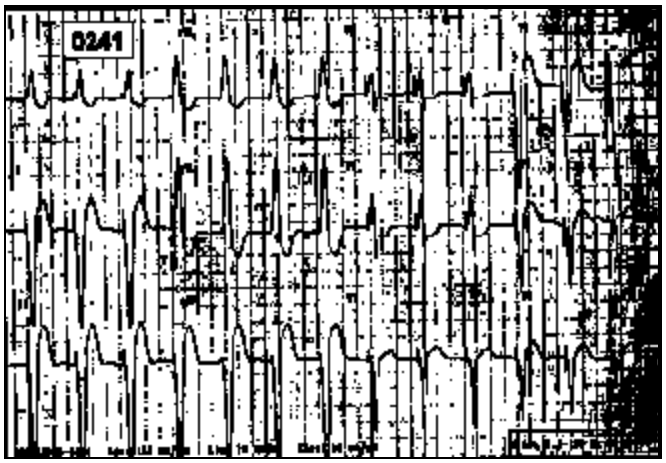
**12-lead ECG with AMI in the setting of LBBB:** Anterolateral AMI in a patient with preexisting LBBB. The rule of appropriate discordance is violated in a number of leads in this example. The lateral leads (I, aVl, $V_5$, and $V_6$) reveal concordant STE while the inferior leads (III and aVf) demonstrate concordant ST segment depression—both features that suggest acute coronary ischemia. Further, excessive discordant STE is seen in leads $V_2$ through $V_4$, another worrisome feature for AMI.

**Figure 32**




**Ventricular paced rhythm with appropriate relationships:** The concept of appropriate discordance also may be applied in this instance as it is in the LBBB patient.

**Figure 33**

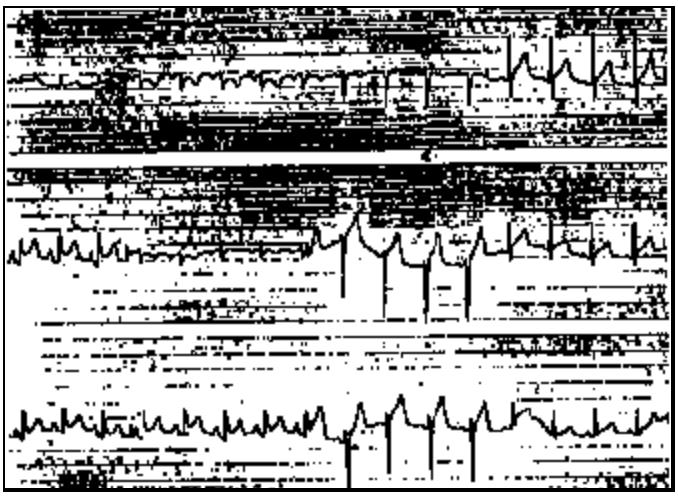



**AMI in the setting of ventricular paced rhythm:** This is a serial ECG performed in the patient seen in Figure 32. Note the progression of STE in the inferior leads and STD in the right precordial leads. While these changes are not diagnostic of AMI, the change noted over approximately 30 minutes in the appropriate patient (i.e., chest pain worrisome for ACS) is highly suggestive of AMI.

**Figure 34**

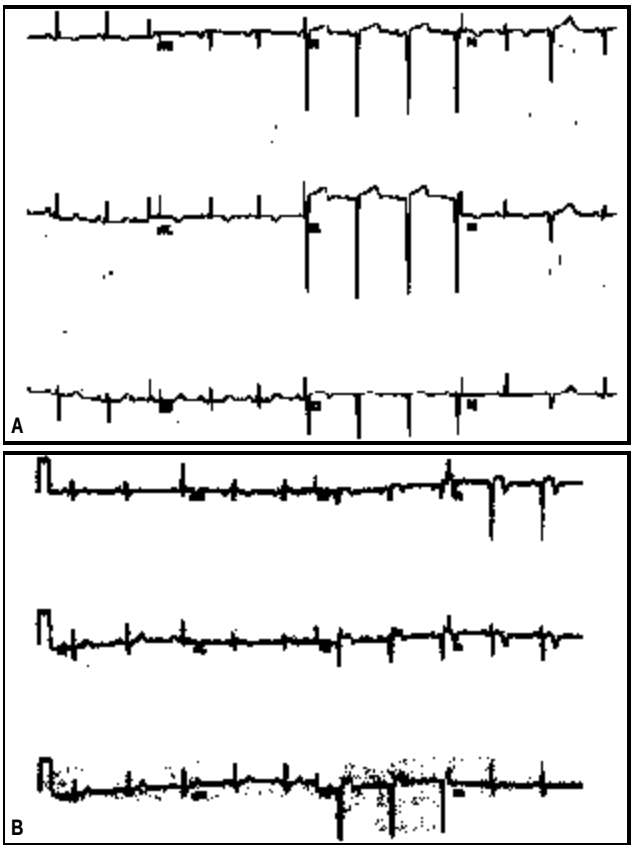
**Benign early repolarization (BER):** Note the STE in all leads except leads I, aVR, aVL, and $V_1$, as well as the prominent T waves in similar distribution.

**Figure 35**



**Acute pericarditis:** Diffuse STE is seen accompanied by PR segment depression in the inferior leads and PR segment elevation in lead aVR.

**Figure 36a and 36b**




**Left ventricular aneurysm:** A, Minimal ST segment elevation. B, More pronounced ST segment elevation.

Emergency Medicine Reports

The Practical Journal for Emergency Physicians

# Management Protocols for Acute Coronary Syndromes

Perhaps no aspect of emergency and cardiovascular medicine is evolving more rapidly than the pharmacological and procedural landscape devoted to the management of patients with acute coronary syndromes (ACS). As every emergency physician and cardiologist understands, making the right choice—whether it is drug therapy, a procedural coronary intervention (PCI), or some combination of both strategies—can make the difference between a favorable and unfavorable outcome.

The current issue of *Emergency Medicine Reports* provides a detailed, comprehensive analysis of the role of electrocardiography in the diagnosis of acute mycoardial infarction. The enclosed supplement provides evidence-based treatment pathways and pharmacotherapeutic strategies that will optimize outcomes of patients diagnosed with AMI or unstable angina. — The Editor

## Low Molecular Weight Heparin Trials in Acute Coronary Syndromes

| TRIALS | ESSENCE | TIMI IIB | FRIC | FRISC | FRISC II | FRAXIS |
|---|---|---|---|---|---|---|
| LMWH | Enoxaparin | Enoxaparin | Dalteparin | Dalteparin | Dalteparin | Nadroparin |
| **Patients:** # | 3171 | 3910 (3-8 d)<br>2346 (8-43 d) | 1482 (1-6 d)<br>1133 (6-45 d) | 1506 | (1) 2105<br>(2) 2457 | 3468 |
| CP w/i | 24 hr | 24 hr | 72 hr | 72 hr | 72 hr | 48 hr |
| EKG Δ | 57%<br>(ST or T ↓ or other changes) | 83%<br>(ST or T ↓ or other changes) | 100%<br>(ST or T ↓) | 100%<br>(ST or T ↓) | (100%)<br>(ST or T ↓) | (100%)<br>(ST or T ↓) |
| NQMI | 21% | 34% | 16% | 38% | — | ~ 16% |
| **Primary end point** | Death, MI, or RA at 14 d | Death, MI, urgent revasc at 8 and 43 d | Death, MI, or RA during days 6-45 | Death or MI at 6 d | Death or MI at 30 d and at 6 months | CV death, MI, or RA at 14 d |
| **Groups** | LMWH<br>UFH | LMWH<br>UFH | LMWH<br>UFH | LMWH<br>Placebo | (1) LMWH<br>Placebo<br>(2) PCI<br>No-PCI | LMWH × 6d<br>LMWH × 14d<br>IV UFH × 6d |
| **Dose** | 1 mg/kg SC bid × 2-8 d | Up to 8 d: 30 mg bolus + 1 mg/kg bid; 8-43 d: 40 mg (< 65 kg) or 60 mg (≥ 65 kg) SC bid | Up to 6 d: 120 Anti-Xa U/kg SC bid; 6-45 d; 7500 anti-Xa SC qd | 120 IU/kg SC bid× 6 d then 7500 IU qd X 42 d | 120 IU/kg SC bid × 1-5 d + 7500 IU SC bid × 5-90 d | 86 IU/kg IV bolus then 86 IU/kg SC bid |
| **UFH dose** | 5000 U IV bolus + infusion | 70 U/kg IV bolus + 15 U/kg IV infusion | 5000 U IV bolus + 1000 U/hr infusion | UFH only used as a rescue drug[3] | UFH only used as a rescue drug[3] | 5000 U IV bolus + 1250 U infusion |
| Death LMWH | — | — | — | 6 d: 1.8*<br>40 d: 8.0 | 30 d: 3.1<br>90 d: 6.7* | — |
| MI (%) Placebo | | | | 6 d: 4.8<br>40 d: 10.7 | 30 d: 5.9<br>90d: 8.0 | |
| Death LMWH MI | 14 d: 16.6*[1]<br>30 d: 19.8*[1] | 8 d: 12.4*<br>43 d: 17.3*[1] | 6 d: 9.3<br>6-45 d: 12.3 | — | — | 14 d: (6 d $R_x$ - 17.8)<br>(14 d $R_x$ - 20.0) |
| RA UFH (%) | 14 d: 19.8<br>30 d: 23.3 | 8 d: 14.5<br>43 d: 19.7 | 6 d: 7.6<br>6-45 d: 12.3 | — | — | 14 d: 18.1 |
| LMWH **Major Bleed[2] (%)** | 30 d: 7.0 | 8 d: 1.5<br>43 d: 2.9* | 6 d: 1.1<br>6-45 d: 0.5 | 6 d: 0.8<br>40 d: 0.3 | 90 d: 3.3 | 6 d & 14 d nadro at 6 d: 1.0<br>6 d nadro at 14 d: 1.5<br>14 d nadro at 14 d: 3.5* |
| UFH | 30 d: 6.5 | 8 d: 1.0<br>43 d: 1.5 | 6 d: 1.0<br>6 d-45 d: 0.4 | — | — | 6 d UFH at 6 d: 1.0<br>6 d UFH at 14 d: 1.6 |
| Placebo | — | — | — | 6 d: 0.5<br>40 d: 0.3 | 90 d: 1.5 | — |

d, day(s); RA, recurrent angina; UFH, unfractionated heparin; NQMI, non-Q wave MI; Revasc, revascularization (PTCA, CABG); CV, cardiovascular; w/i, within; NA, not applicable; SC, subcutaneous; nadro, nadroparin; LMWH, low molecular weight heparin; CP, chest pain.

* $P < 0.05$

[1] = Difference primarily due to need for fewer revascularization procedures.

[2] = Major hemorrhage defined: FRISC: ↓ hemoglobin of 20 g/L, required transfusion, was intracranial, or caused death or cessation of study treatment. In ESSENCE & TIMI IIB: bleeding resulting in death, transfusion of ≥ 2 units of blood, a ↓ hemoglobin of 30 g/L, or a retroperitoneal, intracranial, or intraocular hemorrhage. In FRAXIS: symptomatic bleeding associated with a ↓ hemoglobin > 2g/dL, retroperitoneal or intracranial hemorrhage, or if transfusion required or death caused.

[3] = UFH also was used, but the trial was not designed to compare UFH with a LMWH.

# Trials Using GP IIb/IIIa Inhibitors in Non-ST-Segment Elevation Acute Coronary Syndromes with Mandated PCI‡

| TRIAL | EPIC | EPILOG | EPISTENT | CAPTURE | IMPACT II | RESTORE |
|---|---|---|---|---|---|---|
| **Agent** | Abciximab | Abciximab | Abciximab | Abciximab | Eptifibatide | Tirofiban |
| **Entry Criteria** | Elective to emergent: MI w/i 12 hrs requiring rescue, early post-MI angina, UA w/i 24 hrs, or vessels at high risk for closure | Elective or urgent PCI pts w/ a stenosis of ≥ 60% (Not pts with acute ischemia) | Elective or urgent PCI pts w/ a stenosis of ≥ 60% (Not pts with acute ischemia) | Refractory UA defined as: CP + EKG Δ on admission, then more CP *or* EKG Δ despite medical Rx | Elective, urgent, or emergent PCI pts | Pts undergoing PCI w/i 72 hrs of presentation w/ UA, NQMI, or MI with ST ↑ |
| **Patient Number** | 2099 | 2792 | 2399 | 1265 | 4010 | 2141 |
| **Primary End Point** | Death, MI, CABG, repeat emergent PCI, *or* stenting at 30 d | Death, MI, *or* urgent revasc (CABG or PCI) at 30 d | Death, MI, *or* urgent revasc (CABG or PCI) at 30 d | Death, MI, *or* urgent revasc (CABG or PCI) at 30 d | Death, MI, *or* urgent revasc (CABG or PCI) at 30 d | Death, MI, *or* any revasc (CABG or PCI) at 30 d |
| **Drug Dosing** | Abcix bolus (0.25 mg/kg) and inf (10 mcg/min) | Abcix bolus (0.25 mg/kg) and inf (0.125 mcg/kg/min to max of 10 mcg/min) | Abcix bolus (0.25 mg/kg) and inf (0.125 mcg/kg/min to max of 10 mcg/min) | Abcix bolus (0.25 mg/kg) and inf (10 mcg/min) | Eptif 135 mcg/kg bolus, then inf at: LD: 0.5 mcg/kg/min HD: 0.75 mcg/kg/min | Tirofiban bolus (10 mcg/kg) and inf (0.15 mcg/kg/min) |
| **Drug Duration** | 12 hrs (started w/i 1 hr of PCI) | 12 hrs (started w/i 1 hr of PCI) | 12 hrs (started w/i 1 hr of PCI) | 18-24 hrs *before* PCI then 1 hr after PCI | 20-24 hrs beginning after access established | 36 hrs after angioplasty guidewire was across the lesion |
| **Vase Sheaths** | Removed 6 hrs after end of inf | Early removal and meticulous wound care | Early removal and meticulous wound care | Removed 4-6 hrs after end of inf. Meticulous site care. | Removed 4-6 hrs after end of PCI | Early removal |
| **Randomized Groups** | Three Arms:<br>Abcix bolus + abcix inf<br>Abcix bolus + placebo inf<br>Placebo bolus + placebo inf | Three Arms:<br>Placebo + stand UFH<br>Abcix + stand UFH<br>Abcix + LD UFH | Three Arms:<br>ST + placebo<br>ST + abcix<br>Angio + abcix | All with early angiography and had culprit lesions. Then, two arms:<br>Abcix bolus + abcix inf<br>Placebo bolus + placebo inf<br>Then, PCI performed | Three Arms:<br>LD Ept infusion<br>HD Ept infusion<br>Placebo bolus + placebo inf | Two Arms:<br>Tiro bolus + tiro inf<br>Placebo bolus + placebo inf |
| **1° End Point (30 d)** | | | | | | |
| IIb/IIIa | 8.3*[3] | 5.3* (Both abcix groups) | | 11.3* | LD: 9.2, HD: 9.9 | 10.3, 8.0[3] |
| Placebo | 12.8 | 11.7 | | 15.9 | 11.4 | 12.2, 10.5[5] |
| **2° End Point (6 m)**[2] | | | | | | |
| IIb/IIIa | 27.0* | 22.8 (stand); 22.3* (LD) | | 31 | LD: 10.5, HD: 10.1 | |
| Placebo | 35.1 | 25.8 | | 30.8 | 11.6 | |
| **1° End Point (30 d)**[1] | | | | | | |
| ST + Placebo | | | 10.8 | | | |
| ST + IIb/IIIa | | | 5.3* | | | |
| Angio + IIb/IIIa | | | 6.9* | | | |
| **Major/Intermediate Bleeding**[4] | | | | | | |
| IIb/IIIa | 14* | 3.5 (stand); 2.0 (LD) | 1.5 (ST + angio groups) | 3.8* | LD: 5.1, HD: 5.2 | 2.4 |
| Placebo | 7 | 3.1 | 2.2 | 1.9 | 4.8 | 2.1 |

**Key:** PCI (percutaneous coronary intervention) includes angioplasty, directional atherectomy, and/or stenting; CABG, coronary artery bypass grafting; MI, myocardial infarction; Abcix, abciximab; Ept, eptifibatide; Tiro, tirofiban; inf, infusion; LD, low-dose; HD, high-dose; pts, patients; Angio, angioplasty; ST, stent; Vasc, vascular; Rx, treatment; UFH, unfractionated heparin; d, day(s); m, months; w, with; w/i, within; hrs, hours.

‡ All patients in the trials received aspirin and heparin.

* $P < 0.05$

[1] Death, MI, or urgent revascularization.

[2] Death, MI, or any revascularization (except IMPACT II which was only death or MI).

[3] Data for abciximab bolus plus infusion group. The abciximab bolus only group was not different from placebo.

[4] Major bleeding defined by TIMI criteria for all reported trial results.

[5] These numbers reflect the combined end point when only emergent or urgent PTCA was considered ($P = 0.052$).

Supplement to *Emergency Medicine Reports*, April 23, 2001: "Electrocardiographic Diagnosis of Acute Coronary Syndromes in the Emergency Department." *Authors:* **William J. Brady, MD, FACEP,** Associate Professor of Emergency Medicine, Department of Emergency Medicine; Associate Professor of Internal Medicine, Department of Internal Medicine; Program Director, Emergency Medicine Residency, Department of Internal Medicine; Vice Chairman of Emergency Medicine, Department of Emergency Medicine, University of Virginia School of Medicine, Charlottesville, VA; **Andrew D. Perron, MD,** Assistant Professor of Emergency Medicine, Department of Emergency Medicine; Medical Director, First Med, Department of Emergency Medicine, University of Virginia Health System, Charlottesville, VA; **Edward Ullman, MD,** Department of Emergency Medicine, University of Virginia School of Medicine; Charlottesville, VA.

## Trials Using GP IIb/IIIa Inhibitors in Non-ST-Segment Elevation Acute Coronary Syndromes (PCI Not Mandated)‡

| TRIAL | PARAGON | PURSUIT | PRISM | PRISM-PLUS |
|---|---|---|---|---|
| **Agent** | Lamifiban | Eptifibatide | Tirofiban | Tirofiban |
| **Entry criteria** | CP w/i 12 hrs + EKG Δ (ST temp ↑ or ↓ or T ↓) | CP w/i 24 hrs + [EKG Δ (ST temp ↑ or ↓ or T ↓) *or* enzyme ↑] | CP w/i 24 hrs + [EKG Δ (ST temp ↑ or ↓ or T ↓) *or* enzyme ↑ *or* evidence prior CAD] | CP w/i 12 hrs + [EKG Δ (ST or T ↓) or enzyme ↑] |
| **Patients** | | | | |
| Number | 2282 | 10948 | 3232 | 1915 |
| EKG Δ (%) | 100 | 92 | 75 | 90 |
| Enzyme ↑ (%) | 36 | 45 | 25 | 45 |
| Revascularized | 25 | 38 | 38 | 54 |
| **Primary end point** | Death *or* nonfatal MI at 30 d | Death *or* nonfatal MI at 30 d | Death, MI, *or* refractory ischemia at 48 hrs | Death, MI, *or* refractory ischemia at 7 d[4] |
| **Drug therapy** | 3-5 d[2] | ≤ 72 hrs[2] | 48 hrs | 48 hrs[2] |
| **Randomized groups** | Five arms:<br>Placebo<br>Lam<br>(LD or HD)<br>(w/ or w/o UFH) | Three arms:<br>Placebo<br>HD or LD<br>Ept<br>(UFH)[3] | Two arms:<br>Tiro<br>UFH | Three arms:<br>Tiro[5]<br>UFH<br>Tiro + UFH |
| **Invasive procedures**[1] | Discourages × 48 hours | Physician discretion | Discouraged during 48 hour infusion | Discouraged during 48 hour infusion;<br>Encouraged 48-96 hours |
| **Outcome (primary end point) (%)** | 30 d:<br>No difference<br>6 m:<br>LD Lam ± UFH 13.7*<br>Placebo 17.9 | 30 d:<br>HD Ept 14.2*<br>Placebo 15.7<br>(No difference between groups in those with only medical Rx) | 2 d:<br>Tiro 3.8<br>UFH 5.6<br>30 d:<br>Tiro 15.9<br>UFH 17.1 | 7 d:<br>Tiro + UFH 12.9<br>UFH 17.9<br>30 d:<br>Tiro + UFH 18.5*<br>UFH 22.3<br>6m:<br>Tiro + UFH 27.7*<br>UFH 32.1 |
| **Major/intermediate bleeding (%)** | UFH 5.9*<br>Lam 7.8<br>UFH + Lam 10.5 | Ept 10.6<br>Placebo 9.1* | Tiro 0.4<br>Heparin 0.4 | Tiro + UFH 4<br>UFH 3 |

**Key:** PCI (percutaneous coronary intervention) includes angioplasty, directional atherectomy, and/or stenting; CP, chest pain; MI, myocardial infarction; CAD, coronary artery disease; LD, low-dose; HD, high-dose; w/, with; w/o, without; w/i, within; UFH, unfractionated heparin; Ept, eptifibatide; Lam, Lamifiban; Tiro, Tirofiban; hrs, hours; d, day(s); m, months; temp, temporary; Rx, treatment.

‡ All trials included aspirin for all patients and all contained patients with non-Q-MI. Some trials permitted patients who had temporary ST-segment elevation. PCI (percutaneous coronary intervention) includes angioplasty, directional atherectomy, and/or stenting.

* $P < 0.05$

[1] Includes diagnostic catheterization, PCI, CABG.

[2] If intervention was performed at end of drug therapy, the study drug could be infused for an additional 24 hours (PURSUIT), 48 hours (PRISM PLUS), or 12-24 hours (PARAGON) after the procedure.

[3] Heparin was optional.

[4] The 30-day and 6-month end points also included rehospitalization.

[5] Tirofiban alone arm dropped early in study because of increased adverse effects.

## Common Markers Used to Identify Acute Myocardial Infarction

| MARKER | INITIAL ELEVATION AFTER AMI | MEAN TIME TO PEAK ELEVATIONS | TIME TO RETURN TO BASELINE |
|---|---|---|---|
| Myoglobin | 1-4 h | 6-7 h | 18-24 h |
| CTnI | 3-12 h | 10-24 h | 3-10 d |
| CTnT | 3-12 h | 12-48 h | 5-14 d |
| CKMB | 4-12 h | 10-24 h | 48-72 h |
| CKMBiso | 2-6 h | 12 h | 38 h |
| LD | 8-12 h | 24-48 h | 10-14 d |

CTnI, CTnT = troponins of cardiac myofibrils; CPK-MB, MM = tissue isoforms of creatine kinase; LD = lactate dehydrogenase.

**Adapted from:** Adams JE III, Abendschein DR, Jaffee S. Biochemical markers of myocardial injury: Is MB creatine kinase the choice for the 1990s? *Circulation* 1993;88:750-763.

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*Please note:* Tables printed in this supplement have appeared in one of the following *Emergency Medicine Reports'* issues: Bosker G, Robinson DJ, Jerrard DA, et al. Acute Myocardial Infarction: Current Clinical Guidelines for Patient Evaluation, Thrombolysis, and Mortality Reduction. 1999;14:143-152; Kleinschmidt K. Acute Coronary Syndromes (ACS): Pharmacotherapeutic Interventions—Treatment Guidelines for Patients with and without Procedural Coronary Intervention (PCI), Parts I and II. 2000;23:257-272, 273-284.

# ACUTE CORONARY SYNDROMES — PRACTICAL, EVIDENCE-BASED GUIDELINES FOR OUTCOME-EFFECTIVE MANAGEMENT

**Patients With Unstable Angina, Non ST-Segment Evaluation Myocardial Infarction (NSTEMI), and ST-Segment Elevation MI - With and Without PCI.**
**Adapted, updated, and based upon ACC/AHA Recommendations (September, 2000) for UA/NSTEMI and ACC/AHA 1999 MI Guidelines.**

## CHEST PAIN TRIAGE

Developed by Kurt Kleinschmidt, MD, FACEP, for *Emergency Medicine Reports* (November, 2000)

Acute Coronary Syndrome (ACS): Pharmacotherapeutic Interventions For UA/NSTEMI— An Evidence-Based Review And Outcome-Optimizing Guidelines For ACS Patients With And Without Procedural Coronary Intervention (PCI)

**TRIAGE ASSESSMENT**
- Pain description • Age • Sex • CAD Hx
- Cocaine • Risk factors for CAD

Non-Ischemic / Non-Cardiac → Evaluate and treat suspected etiology

**Possible or definite ACS**

**12-LEAD ECG WITHIN 10 MINUTES**
- Intravenous access
- Oxygen
- Continuous ECG monitor
- Aspirin
- Consider
  - — Cardiac markers
  - — Beta-blockers
  - — Nitroglycerin
  - — Morphine sulfate

### ST-segment elevation or new or presumably new bundle branch block

**Treat with:**
- Beta-blockers
- Unfractionated heparin (preferred) or enoxaparin
- Nitroglycerin
- Morphine sulfate

≤12 hours / >12 hours chest pain

Institution capable of performing PCI → YES / NO

Persistent symptoms → NO / YES

- NO (PCI): Institutional policy is to routinely perform catheterization and then a procedural coronary intervention[2] or CABG → YES / NO
  - YES: Cardiac catheterization → Procedural coronary intervention or CABG
  - NO: Contraindication to thrombolysis or cardiogenic shock → YES / NO
    - YES: Cardiac catheterization → Procedural coronary intervention or CABG
    - NO: Thrombolysis
      - Alteplase or
      - Reteplase or
      - Streptokinase or
      - Tenecteplase

      (Door → needle time < 30 minutes) → Clinical evidence of reperfusion (Chest pain and EKG with resolution) → NO / YES
      - NO: Cardiac catheterization
      - YES: Continued medical mgmt.
- NO (Persistent symptoms): Medical management
- YES (Persistent symptoms):
  - Consider reperfusion
  - Aggressive medical therapy

### ST-segment depression or T-wave inversion

**Treat with:**
- Beta-blockers
- Enoxaparin (preferred) or unfractionated heparin[1]
- Tirofiban[2]
- Nitroglycerin
- Morphine sulfate

Chest pain is refractory or recurrent or patient has prior CABG or MI → Early cardiac catheterization → Abnormal / Normal
- Abnormal: Procedural coronary intervention or CABG
- Normal: Discharge and follow-up as needed

Good clinical response → Early conservative medical management → Hospital Course Becomes Complicated
- Recurrent chest pain
- Hemo instability
- New EKG change
- CHF starts
- Dysrhythmias

→ YES / NO
- YES: Assess LV function → <40% / ≥40%
  - <40%: Abnormal
  - ≥40%: Stress test → Abnormal / Normal
    - Abnormal: Assess LV function
    - Normal: Medical therapy discharge
- NO: Abnormal

**No EKG change or normal EKG**

**RISK STRATIFY**
- Complete H & P
- Consider
  - — Serial EKGs or continuous segment monitoring
  - — Second set cardiac markers (at ≥ 6 hours after chest pain onset)
  - — If first troponin obtained at < 6 hours, obtain second set between 6-12 hours
  - — 2-D echocardiogram
- Observation 4-12 hours
  - — Emergency department
  - — Chest pain unit
  - — 24 hours observation
  - — Admission
- Pain relief (initiate or intensify)
  - — Beta-blocker
  - — Nitroglycerin
  - — Morphine sulfate

**IF ANY ONE OF THE FOLLOWING:**

| | |
|---|---|
| HX — | • Prior coronary, peripheral, or cardiovascular disease<br>• Rest angina > 20 minutes in patient with moderate likelihood<br>• Accelerating tempo of angina within preceding 48 hours |
| PE — | • Age > 70 years old<br>• Congestive heart failure<br>• Hypotension, bradycardia, or tachycardia |
| ECG — | • ST-segment depression (> 0.05 mv) or transient elevation with pain<br>• T-wave inversion of > 0.2 mv with pain<br>• Pathological Q waves<br>• Sustained ventricular tachycardia |
| MARKERS— | • TnT or TnI elevated |

YES → **Enoxaparin (preferred)** or unfractionated heparin[3]

NO → LOW RISK
- Treat suspected etiology
- Consider stress testing to provoke ischemia (prior to discharge or as an outpatient)
- Follow-up as needed

**EKG change or marker increase**

YES → **Tirofiban[4] (preferred) if PCI not anticipated. Abciximab (preferred) if PCI anticipated.**

NO → Medical management (includes enoxaparin or unfractionated heparin)

**IF ANY ONE OF THE FOLLOWING:**
- Recurrent angina • CHF • Hemodynamic instability
- Sustained V-tach • PCI within 6 months • Prior CABG

YES → Early PCI

NO → Continued medical therapy

[1] Dalteparin or nadroparin are alternative options.
[2] Abciximab can be considered if patient is going to have a procedural coronary intervention. Eptifibatide is also an alternative.
[3] Dalteparin or nadroparin are alternative options.
[4] Eptifibatide is an alternative option.