Curriculum in Cardiology

Electrocardiographic diagnosis of acute myocardial infarction: Current concepts for the clinician

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Background Over the past two decades, the 12-lead electrocardiogram has attained special significance for the diagnosis and triage of patients with chest pain because timely detection of myocardial injury and a rapid assessment of myocardium at risk proved pivotal to implementing effective reperfusion therapies during acute myocardial infarction. However, this wealth of information could still be underutilized by clinicians who may restrict their diagnostic quest in patients with chest pain to the more classic electrocardiographic signs.

Methods The medical literature on electrocardiographic manifestations of acute myocardial infarction was extensively reviewed.

Results The widespread utilization of both coronary angiography and methods to determine myocardial function and metabolism in patients with acute myocardial infarction over the last 10 years has provided the means for rigorous comparisons with electrocardiographic information. We summarize these electrocardiographic signs and patterns in terms of their relevance to the clinician to help reduce the incidence of “nondiagnostic electrocardiograms” and improve timely decision-making.

Conclusions The electrocardiogram continues to be an invaluable tool in the initial evaluation of patients with chest pain. The plethora of data currently available on electrocardiographic changes correlating with myocardial injury allows clinicians to make faster and better decisions than ever before. (Am Heart J 2001;141:507-17.)

In comparison with other cardiac noninvasive diagnostic techniques, electrocardiography has evolved rather slowly over the past 40 to 50 years. Perhaps the most significant advance has been the introduction of computerized electrocardiographic interpretation. Although health professionals have increasingly relied on this feature, automated reports are not optimal and should always be overseen by an experienced, well-informed physician with electrocardiography training.1,2 This is crucial when expeditious diagnoses may be life-saving, as in acute coronary syndromes. Henry Marriott3 wrote that the electrocardiogram is “the single most often used, most cost-effective, and most diagnostic test in cardiology” and also “the most frequently misinterpreted.”

Many observations made over the past decade from both multicenter and small, prospective studies have systematically correlated the electrocardiographic changes of myocardial injury to biochemical markers, early coronary angiographic findings, ventricular contractility, and myocardial metabolic state. Such contributions have considerably improved our ability to diagnose acute myocardial infarction (AMI) and are summarized in this article in terms of their relevance to the clinician reviewing electrocardiograms.

Normal anatomic references

Several anatomic caveats are pertinent to the assessment of new fascicular blocks or axis changes and to the assessment of infarct location.

The heart lies horizontally with the atria at its base and the ventricles at its apex.4 Because the heart is rotated over its long axis, the right atrium and ventricle are more anterior than the left chambers, and the right and left sides of the heart are not aligned with the same sides of the body. Thus the interventricular septum is almost parallel with the frontal—not the sagittal—plane, and the left ventricular free wall (usually considered a lateral structure) includes nearly 300 degrees of the left ventricular circumference and faces superiorly, posteriorly, and inferiorly.2,4 In addition, the heart position relative to the electrocardiographic electrodes is highly variable; this results in a wide range of normalcy for the cardiac long axis.5

The conventional 12-lead system is unfortunately suboptimal. Whereas leads V1 through V6 adequately capture most electrical cardiac phenomena taking place in
the horizontal plane (and this anatomic area can be further extended by adding leads V7 through V9 and V3R through V5R), the cardiac activity that takes place in the frontal plane is not ideally represented. The reasons are 2-fold. One is that whereas 3 of the frontal lead pairs are separated between them by 30 degree angles, a 60 degree gap exists between leads I and II. This leaves an absent recording point where the lead “–aVR” should be placed. The conventional lead aVR, separated from lead III by a 90 degree angle, provides an inconvenient view from the right shoulder (Figure 1). This results in underdetection of injury currents at the left ventricular inferior and lateral walls and in a virtual neglect of lead aVR by physicians during electrocardiographic interpretation.6 The second limitation of the frontal electrocardiographic display is that it presents the electrical phenomena in the two groupings I, II, III, and aVR, aVL, aVF. This sequence is unlike the true spatial sequence; ventricular activation starts from the base of the left ventricle with lead aVL and continues with leads I, II, aVF, III, and aVR7 (Figure 1). Thus the electrocardiogram interpreter needs to make a “mental rearrangement” of the spatial lead distribution.

The diagnosis of AMI is compounded by the fact that the electrocardiographic signal integrates the balance of many competing forces across the myocardium and that coronary artery occlusions will result in widely different electrocardiographic manifestations, depending on the artery size, length, direction, and occlusion level; on the presence of collateral circulation, previous myocardial necrosis, and intraventricular conduction disorders; and on the position of the heart in the chest. Thus the 12-lead electrocardiogram is only moderately accurate to determine the anatomic location of AMI, and the correspondence of some electrocardiographic terms with the pertinent site of infarction is rather poor.8 The infarction descriptors “anterior,” “inferior,” and “lateral” have classically been attributed to occlusions of the left anterior descending artery (LAD), right coronary artery (RCA), and left circumflex artery (LCX), respectively. Other terms such as “apical,” “septal,” “high lateral,” and “posterior” are also in use. However, a detailed review of the anatomic, echocardiographic, and angiographic correlations has suggested that a categorization of infarction into anterior, lateral, and posterior (for the LAD, LCX, and RCA territories, respectively) would be more appropriate.9 Other authors maintain that because the left ventricle has a marked conal shape, the electrocardiogram can only differentiate between “anterior” and “posterior” and that the name “inferior” should be included with the latter category.8 Yet other investigators believe that the name “posterior” is misleading, given that this term applies better to the posterior thoracic wall facing the left ventricle than to the left ventricle itself.10

**Value of ST-segment elevation**

The electrocardiograms that give us the most concern in emergency departments are those with a large amount of ST-segment deviation. The initial ST-segment sum is the main variable influencing “door to thrombolysis” time; the largest ST deviations result in the shortest times to treatment.11 Although this is usually justified, many factors such as myocardial mass, distance between the electrodes and the ischemic zone, and reciprocal “cancellation” changes may affect the magnitude of ST elevation. This should be considered particularly in scenarios of less conspicuous ST shifts because the choice of electrocardiographic criteria will determine both the diagnosis and the therapeutic decisions. A recent controlled study analyzed the initial electrocardiogram of patients with and those without chest pain. The optimum electrocardiographic variables for the detection of AMI were ST elevation ≥1 mm in at least 1 lead, either inferior (II, III, aVF) or lateral (V5, V6, I, aVL), and ST elevation ≥2 mm in at least 1 anterior lead. Such a model (internally validated) correctly classified 83% of subjects with 56% sensitivity and 94% specificity. Changing the degree of ST elevation greatly modified both sensitivity (45.4% to 68.6%) and specificity (81.2% to 98.1%). The addition of multiple QRST variables increased specificity but improved overall classification only marginally.12

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**Figure 1**

Panoramic display of frontal-plane electrocardiographic leads showing both conventional aVR and a “–aVR” at 30 degrees (from Reference 34, with permission).
**Electrocardiographic-angiographic correlations revisited**

The most frequent infarct-related artery among patients admitted with chest pain or discharged from the hospital after AMI is the LAD (44% to 56% of cases), followed by the RCA (27% to 39%) and the LCX (17%).

**Anterior/anteroseptal/anterolateral infarction (LAD occlusion)**

After occlusion of the LAD, ST-segment elevation ≥1 mm is most frequently observed in lead V2 (sensitivity, 91% to 99%) and then—in decreasing order of frequency—in V3, V4, aVL, V1, and V6. The maximum ST elevation is recorded in V2 or V3.

Additional signs (concomitant with precordial ST elevation) were identified over the last decade by Birnbaum et al.15 and others16 as powerful predictors of proximal LAD occlusion. These include ST elevation in aVL and ST depression in inferior leads. ST elevation that extends to leads I and aVL often coexists with inferior ST depression; the magnitude of ST depression in inferior leads correlates better with that of ST elevation in leads I and aVL than with the ST elevation of precordial leads.16

Four electrocardiographic signs that had previously received little attention as indicators of anterior infarction were recently found to be specific for occlusions at the level of the first septal perforator. These include ST elevation in aVR (sensitivity, 43%; specificity, 95%), disappearance of preexistent septal Q waves in lateral leads (sensitivity, 30%; specificity, 84%), ST depression in V5 (sensitivity, 17%; specificity, 98%), and right bundle branch block (RBBB) (sensitivity, 14%; specificity, 100%).

Distal LAD occlusions, on the other hand, usually have ST elevation of ≤3.2 mm in V2 and slight to moderate ST elevation in V3.19 Also common are new Q waves in V1 through V6 and augmented R-wave amplitude in V2.19,20 This pattern of acute right septal conduction delay results from an ischemic disruption of the right septal activation pathway and increased septal anterior vectors and may be indistinguishable from the changes observed in recent or old posterior infarction.20 Concomitant ST depression in inferior leads, if present, is of a lesser magnitude than that seen in proximal LAD occlusions (0.9 mm vs 1.9 mm in lead III, respectively).

The significance of ST elevation in lead V1 merits discussion. Lead V1 captures electrical phenomena from the right paraseptal area, which is supplied by the septal branches of the LAD. In some patients, the septum is additionally protected by a blood supply from a conal branch of the RCA (double circulation). This explains why approximately two thirds of patients with anterior AMI have no ST elevation in V1.19 The presence of ST elevation in V1 correlates strongly with ST elevation in V3R and predicts the less common anatomic scenario in which a small conal branch of the RCA does not reach the interventricular septum.21 In 7% of the patients with ST elevation in leads V1 through V4 who undergo coronary angiography, this ST elevation is secondary to RCA occlusion.22

**Anterior/“high lateral” infarction (occlusion of first diagonal)**

The electrical activity of the anterolateral wall of the left ventricle (supplied by both the first diagonal and the first obtuse marginal branches) is well captured by the contiguous leads aVL and I. During acute anterior injury, a culprit lesion at the level of the first diagonal can be suspected when ST elevation is present in both leads I and aVL (a highly sensitive combination also for diagonal occlusion) and when precordial ST elevation is associated with ST elevation in lead aVL (a highly specific sign).15,23 Occlusion of the first diagonal may produce unique electrocardiographic changes characterized by ST elevation in the noncontiguous leads aVL and V2, plus ST depression in leads III and aVF or in V4. This pattern has been termed “mid-anterior” infarction because the akinetic region in the left ventricle excludes the apex and the septum.24 When ST elevation in leads I and aVL is accompanied by ST depression in lead V3, the culprit artery is usually the first marginal branch of the LCX.25

**Inferior infarction (right coronary or circumflex occlusion)**

The typical electrocardiographic pattern of inferior infarction consists of ST-segment elevation in leads II, III, and aVF. The occlusion is in the RCA in 80% to 90% of cases and is in the LCX in the remaining patients.26 Higher ST elevation in lead III than in lead II strongly suggests compromise of the RCA (Figure 2).27,28

A bedside differential diagnosis between culprit arteries can also be attempted by examining additional electrocardiographic leads. Because the only lead that faces the superior part of the left ventricle and directly opposes the inferior wall is aVL, ST depression in lead aVL is almost always determined by RCA occlusion (sensitivity, 94%; specificity, 71%), without indicating concomitant involvement of the posterior wall or the right ventricle (Figure 2). Injury in leads II, III, and aVF without ST depression in aVL indicates proximal LCX occlusion.29

Several studies in the 1980s concluded that ST elevation in leads V4 through V6 during inferior injury signaled LCX occlusion. However, because most inferior infarctions are caused by RCA occlusion, the positive predictive value of this sign is poor. The arteries that supply the posterolateral region of the left ventricle are
the obtuse marginal branch of the LCX, the posterolateral, and the LAD branches. Thus ST changes in leads V_{5} and V_{6} indicate rather posterolateral ischemia triggered by either RCA or LCX occlusion. When this ST elevation is significant (>2 mm), it is probably a sign of “mega-artery-related” (either the RCA or LCX) infarction with a large ischemic burden.

Precordial ST depression accompanying inferior injury is more likely to develop from LCX than RCA occlusion. Horizontal ST depression with initially negative, then upright T waves in leads V_{1} through V_{3}/V_{4} is associated with posterior wall motion abnormalities. The degree of ST depression in lead V_{3} compared with the degree of ST elevation in lead III ("V_{3}/III ratio") is highest (1.2 mm) when the occlusion is in the LCX and lowest (<0.5 mm) when it is in the proximal RCA (Figure 2). Occlusions of the mid RCA produce V_{3}/III intermediate ratios. The absence of ST depression in leads V_{1} to V_{2} rules out LCX occlusion, with a predictive value of >90%.

Some electrocardiographic leads underutilized in clinical practice can be very helpful in discriminating infarct-related arteries during inferior injury. ST elevation in leads V_{7} through V_{9} and ST depression in V_{5}R probably are related to LCX occlusion. ST elevation in leads I to aVL or V_{5} to V_{6} is frequently accompanied by ST depression in lead aVR (or ST elevation in “–aVR” at 30 degrees) (Figure 3). This sign is independent of ST depression in V_{1}, and it indicates a larger infarct size. However, during routine electrocardiographic interpretation, most physicians—even electrocardiographic experts—seem to ignore lead aVR; medical teaching should probably emphasize its value.

Lateral and posterior infarctions (LCX occlusion)

The vascular beds of the LCX have broad anatomic variability and supply a rather small ventricular area. This is why the standard 12-lead electrocardiogram shows ST elevation in less than half of cases of LCX occlusion. When present, ST elevation is more often seen in leads II, III, and aVF, followed by leads V_{5}, V_{6}, and aVL.

One third of patients with chest pain secondary to
spontaneous LCX occlusion have isolated ST depression in the electrocardiogram; ST depression in leads V1 to V2 is a sensitive sign. Another third of patients will not have any changes in the 12-lead electrocardiogram. In a study of 33 such consecutive patients, however, ST elevation in V7 through V9 was always detected and was associated with posterior wall motion abnormalities. Leads V7 through V9 are more specific than precordial leads for posterior infarction (84% vs 57%), with similar sensitivity (approximately 80%).

**Special electrocardiographic injury patterns**

**Anterior plus inferior injury**

The combination of anterior and inferior ST elevation in the electrocardiogram may give the impression of a critical mass of myocardial injury. However, it often results from distal occlusion of a long LAD, which “wraps around” the cardiac apex and results in wall motion abnormalities circumscribed to the cardiac apex. When injury in leads II, III, and aVF is accompanied by ST elevation in V1 but ST depression in V2, right ventricular—rather than apical—infarction is likely.

**Septal infarction**

The ST elevation in leads V1 through V4 encountered in most patients with LAD occlusion is usually assumed to represent septal infarction; however, it is significantly associated with apical wall motion abnormalities. Instead, the most frequent electrocardiographic correlate of echocardiographic septal hypokinesia is ST elevation in leads V4 to V6. Septal asynergy is also present in 58% of patients with both transmural inferior injury and precordial ST depression.

**Left main disease**

The electrocardiograms of patients with rest angina from occlusion or subocclusion of the left main coronary artery frequently show a combination of ST elevation in aVR and ST depression in leads I, II, and V4 through V6. A sum of ST changes ≥18 mm is 90% sensitive for left main disease (Figure 4).

**Right ventricular infarction**

Timely recognition of right ventricular injury is important because of both the high risk of complete atrioventricular block and the need to provide intravascular volume expansion. Transmural injury of the right ventricle translates into ST elevation ≥1
mm in precordial leads; ST elevation in V₁ is highly specific for proximal RCA occlusion. In approximately 7% of patients, ST elevation extends to lead V₅, suggesting anterior infarction. However, this ST elevation decreases toward V₄, whereas in anterior injury the ST segment is more elevated in V₂ to V₃ than in V₁.

Right ventricular infarction is usually concurrent with infarcts of the inferior wall. Fifty-four percent of patients with inferior injury have ST elevation in lead V₄R (sensitivity and predictive accuracy for right ventricular infarction are both 93%). Isolated right ventricular infarction is rare and occurs mainly in patients with right ventricular hypertrophy.

**ST-segment depression**

Many patients with acute chest pain have “reciprocal” ST-segment depression, that is, ST-segment depression concomitant with ST-segment elevation in a lead group different than the one showing ST elevation. The mechanism underlying this ST depression is usually mirroring, a phenomenon of electrical reflection of the transmural injury onto the opposite ventricular wall. The ST depression is captured by a lead placed at 180 degrees of the lead recording the ST elevation, although the terms “reciprocal” and “mirror” are loosely applied to recording points in the complementary electrocardiographic spatial plane as well. Another possible mechanism for ST depression is regional subendocardial ischemia or infarction. Although, strictly speaking, mirroring is also involved in the ST depression of subendocardial ischemia because the ST elevation in the subendocardial layer is reflected onto the epicardial layer, most clinicians consider this ST depression “nonmirror” because it is a primary manifestation of artery occlusion not associated with ST elevation in a different territory.

**ST-segment depression concomitant with ST elevation**

In patients with chest pain and predominant ST depression in any lead except aVR, ST depression of ≥4 mm is 97% specific (and 20% sensitive) for AMI. More than 85% of patients with ST depression in lead aVF have a culprit lesion in the LAD proximal to the first diagonal branch. Through assessments by perfusion imaging, several investigators have found that inferior ST depression during anterior injury does not represent inferior injury, suggesting that mirroring—rather than inferior subendocardial ischemia—is the cause of ST depression.

The significance of anterior ST depression accompanying inferior transmural injury, on the other hand, may depend on the leads involved. ST depression in leads V₁ through V₃ or I to aVL appears to correspond to mere mirroring, often from LCX occlusion (Figure 2). ST depression circumscribed to V₁ through V₃ during inferior injury is not accompanied by septal or anterior wall motion abnormalities. In a study of more than 1000 patients with inferior infarction, the presence of maximum ST depression in leads V₁ through V₃ indicated a much lower probability of proximal RCA occlusion than its absence or than a maximum ST depression in leads V₄ through V₆. On the other hand, a maximum ST depression in leads V₄ through V₆ is associated with septal asynergy and most likely corresponds to anterolateral or septal subendocardial injury from a severe lesion in the LAD or in the left main coronary artery, or it is associated with triple-vessel disease.

The fact that patients with ST depression in leads V₁ through V₃ often have a greater magnitude of ST elevation in inferior leads than patients with ST depression in leads V₄ through V₆ may be due to a “canceling effect” from the concomitant LAD lesion in the latter group.

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**Figure 5**

Anterior wall AMI in patient with RBBB. Precordial and lateral ST elevation as well as inferior ST depression are not obscured by conduction defect.
Isolated ST depression

Isolated ST depression ≥1 mm measured at 80 ms of the J point in ≥6 leads is 96.5% specific for AMI. A maximum ST depression in leads V2 to V3 indicates LCX occlusion (specificity, 96%; sensitivity, 70%). This is important because patients with isolated ST depression from LCX occlusion benefit from thrombolysis.

Diffuse precordial (and often inferior or lateral) ST depression with a peak in leads V4 through V6, on the other hand, may be caused by subendocardial ischemia from a subtotal obstruction of the LAD—particularly if the ST depression is accompanied by upright T waves not preceded by negative T waves. The maximum ST depression is observed in the lead with the highest R wave, either V4 or V5. Patients with counterclockwise rotation of the heart may have a maximum ST depression in V3, whereas patients with clockwise rotation will show maximum ST depression in V6. The subsequent evolution of subendocardial infarction may include left bundle branch block (LBBB) secondary to increased ventricular diastolic pressure; Q waves; and a decrease in R-wave amplitude. As discussed above, diffuse ST depression often signals left main or triple-vessel disease (Figure 4), and in autopsy series, it has been associated with large subendocardial infarctions. ST depression in leads V4 through V6 accompanied by negative T waves manifests either the subacute stage of posterior injury from LCX occlusion or an increased myocardial oxygen demand (eg, tachycardia). As discussed above, diffuse ST depression often signals left main or triple-vessel disease (Figure 4), and in autopsy series, it has been associated with large subendocardial infarctions. ST depression in leads V4 through V6 accompanied by negative T waves manifests either the subacute stage of posterior injury from LCX occlusion or an increased myocardial oxygen demand (eg, tachycardia).

An autopsy study found that ST-segment depression ≥1 mm in the initial electrocardiogram was a sensitive sign for infarction of a papillary muscle; for half of the patients, ST depression was not accompanied by ST elevation at admission. Inferior ST depression was seen exclusively in infarctions of the anterolateral papillary muscle, whereas ST depression in leads I, aVL, or both occurred only after infarction of the posteromedial papillary muscle.

Several prospective studies have shown that patients with chest pain and isolated ST depression did not have a survival benefit from timely thrombolysis. A subgroup analysis in the LATE study (which compared tissue plasminogen activator with placebo administered 6 to 24 hours after admission) did find a survival benefit in patients who had ST depression ≥2 mm. This finding is difficult to interpret. It is possible that the post hoc nature of the analysis introduced some bias. Or, if patients with ST depression in leads V1 through V3 were included, perhaps posterior AMI caused by LCX occlusion accounted for the survival benefit.

Nonischemic ST depression

The most important differential diagnosis in patients with chest pain and ST depression is aortic dissection. As many as 50% of patients with dissection of the thoracic aorta (some with abnormal levels of creatine kinase) will have electrocardiographic abnormalities, mainly ST-segment depression. True ischemia may also develop if the origin of a coronary artery is involved in the dissection.

Confounding factors

Bundle branch block/ventricular pacing

Inclusion criteria for patients with chest pain in many clinical studies and registries require grouping patients with either RBBB or LBBB into a common category of “difficult” or “impossible” early diagnoses. However, the diagnosis of AMI in patients with RBBB is rarely obscured by the conduction defect. Myocardial injury should not be missed more often in these patients than in those with normal conduction because ST-segment elevation in both anterior and inferior injury (as well as reciprocal ST depression) are all recognizable (Figure 5). Anterior injury can also be suspected when secondary T waves (ie, with opposite polarity to that of the QRS complex) in leads V1 through V4 are replaced by T waves of concordant polarity with the QRS (“pseudonormalization”).

In patients with LBBB, electrocardiographic interpretation is indeed more difficult because the normal sequence of ventricular activation and recovery can be similarly altered by both LBBB and acute myocardial injury. However, it has been shown that further distinct ST-segment elevation does occur in LBBB during coronary artery occlusion. Yet physicians do not always inspect the electrocardiogram in search of this ST elevation; one consequence is that patients with cardiac chest pain and LBBB are as unlikely to receive reperfusion as those without chest pain and less likely to receive thrombolysis than patients with previous stroke or with cardiac arrest at arrival. This is in part explained because these patients are elderly; the median age of patients with chest pain and LBBB in the United States is 74 years. On the other hand, the approach “thrombolysis to all [patients with chest pain and LBBB]” would seem an overcompensation in light of the recently reported high mortality rates among patients with LBBB receiving thrombolysis.

Thus, identifying patients with LBBB and specific electrocardiographic signs of infarction may allow for more effective, individualized therapies, taking into consideration the patient’s age. In a large population of patients with chest pain and LBBB undergoing thrombolysis (compared with patients without chest pain), independent signs of AMI during LBBB were identified: ST elevation ≥1 mm in leads with a positive QRS, ST depression ≥1 mm in leads V1 through V3, and ST eleva-
tion ≥5 mm in leads with a negative QRS. As a clinical prediction rule, the score value of each sign was 5, 3, and 2, respectively. A score ≥5 made a diagnosis of AMI with a 90% specificity and a score of 2 with >80%.65,66 These criteria have shown high interobserver agreement among both cardiologists and emergency physicians67 and have recently been validated in two independent populations.68,69

In persons with permanent ventricular pacing, the presence of ST elevation ≥0.5 mV in leads with negative QRS complexes in the admission electrocardiogram had a specificity of 88% and a sensitivity of 53% for the diagnosis of AMI (Figure 6).70

Wolff-Parkinson-White syndrome

From the few cases reported in the literature of patients with both Wolff-Parkinson-White syndrome (WPW) and AMI, it appears that the diagnosis of AMI can be made with reasonable specificity. Among 6 patients with WPW enrolled in the GUSTO-I study (3 with left posteroseptal accessory pathways and 3 with accessory pathways in other locations), ST elevation (∑ST, 7 to 18 mm) was recognizable in all. Five patients had abnormal creatine kinase-MB; one patient with a left posteroseptal accessory pathway was misdiagnosed with AMI from the presence of a 3-mm ST elevation in leads V₂ and V₃. In the predischarge electrocardiogram of 4 of the 5 patients with confirmed AMI (all of whom had inferior wall AMIs), preexcitation was still present but less evident, suggesting ischemic damage of the accessory pathway. The delta wave disappeared in a patient with a left posteroseptal accessory pathway and extensive anterior wall AMI.71 Because radiofrequency ablation of accessory pathways is now a widespread curative treatment for WPW, fewer patients are expected to have both WPW and AMI.

Pseudoinfarction patterns

Several conditions mimic AMI and may pose a diagnostic challenge.

Normal variants

Some persons have baseline ST-segment elevation in precordial leads; normal young men may show ST-segment elevation of up to 4 mm in leads V₁ through V₃.72 This pattern appears to result from the onset of ventricular repolarization in a nonhomogeneous fashion, and it is known as early repolarization.73 Early repolarization has been reported in 1% to 2% of the general population and in as many as 48% of patients seen in the emergency department with chest pain; these patients are at risk of receiving erroneous treatment for acute infarction.

Acute pericarditis

Pericarditis may complicate both AMI and myocarditis. Early electrocardiographic abnormalities during acute pericarditis include diffuse ST-segment elevation and upright T waves. The myocardial area underneath the inflammatory process depolarizes only partially. Newly developed ST vectors point toward the apical epicardium. Thus ST-segment elevation may be seen in all leads except aVR, which shows ST depression as well as lead V₁ (which faces the right atrium). Although the P wave is usually not affected, PR (STₐ) segment depression may develop, except in lead aVR or lead V₁.74 Acute pericarditis persists for 3 or 4 weeks. After the ST segment normalizes, the T-wave changes become conspicuous. In postinfarction pericarditis, these changes may consist of persistently positive deflections after 48 to 72 hours or of gradual reversal of initially inverted T waves. Premature restoration of the concordance of the ST segment with the T wave after infarction is deceptive because it renders the electrocardiographic appearance more “normal”; yet it is a piv-
otal manifestation of pericarditis (sensitivity, 100%; specificity, 77%).

Miscellaneous

Other conditions that may mimic AMI are severe hyperkalemia, primary and secondary cardiac tumors, acute pulmonary embolism, ventricular aneurysm, left ventricular hypertrophy, hypothermia with J waves, and exercise-induced ST elevation in patients with previous AMI. In vagotonic persons, marked negative T waves—like those observed after AMI—may be normally present in leads V6R through V2.

Nondiagnostic electrocardiograms

Fifteen percent to 18% of patients with AMI do not show changes in the initial electrocardiogram, and an additional 25% show nonspecific changes. Although nondiagnostic electrocardiograms in patients with chest pain are often associated with lesions in branch vessels, the probability of detecting AMI does increase by recording serial electrocardiograms. However, because reperfusion therapies are more effective when administered early, it is ideal to maximize the information provided by the admission electrocardiogram. Nondiagnostic electrocardiograms are usually 12-lead recordings; approximately 8% of patients with cardiac chest pain will display ST elevation only in posterior (V7 through V9) or right precordial (V3R through V6R) leads. These patients may not be offered reperfusion if only the 12-lead electrocardiogram is used for decision-making. Systematically recording leads V4R, V8, and V9 (ie, a 15-lead electrocardiogram) increases the probability of detecting ST elevation from 47% to 59%, with no decrease in specificity. It is also reasonable to assume that a systematic examination of lead aVR may increase sensitivity for acute infarction.

Conclusions and recommendations

Relative to other diagnostic methods in cardiology, electrocardiographic technology has lagged behind. Experts in electrocardiography have called for a concerted effort to incorporate modern features to the bedside diagnosis such as high-resolution, additional leads and 3D vectorcardiography imaging. These additions would improve our ability to diagnose AMI.

In the meantime, however, the plethora of data currently available on electrocardiographic changes accompanying chest pain should allow clinicians to make faster and better decisions than ever before. For example, it is now clear that isolated ST depression in leads V1 through V3 may indicate LCX occlusion and potential benefit from thrombolysis. Entirely nondiagnostic electrocardiograms may become diagnostic when serial or previous electrocardiograms are obtained or when posterior and right precordial leads are recorded. A few hospitals around the world are already using the 15- or 16-lead electrocardiogram for routine admission workups. Cardiologists and emergency physicians in the United States should make an effort to incorporate these leads in both teaching and clinical practice and should request electrocardiographic machine vendors that electrocardiographers be set to provide a panoramic display of the frontal plane leads including a “-aVR.” Finally, electrocardiographic technology would be more helpful if automated diagnoses were provided along with their probabilities in each case as well as with the clinical value of the electrocardiographic signs incorporated in them.

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