

AHA/ACCF/HRS Recommendations for the Standardization and Interpretation of the Electrocardiogram

Part VI: Acute Ischemia/Infarction

A Scientific Statement From the American Heart Association Electrocardiography and Arrhythmias Committee, Council on Clinical Cardiology; the American College of Cardiology Foundation; and the Heart Rhythm Society

Endorsed by the International Society for Computerized Electrocardiology

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This is the sixth and final section of the project to update electrocardiography (ECG) standards and interpretation. The project was initiated by the Council on Clinical Cardiology of the American Heart Association (AHA). The rationale for the project and the process for its implementation were described in a previous publication.¹

The ECG is considered the single most important initial clinical test for diagnosing myocardial ischemia and infarction. Its correct interpretation, particularly in the emergency department, is usually the basis for immediate therapeutic interventions and/or subsequent diagnostic tests. The ECG changes that occur

in association with acute ischemia and infarction include peaking of the T waves, referred to as hyperacute T-wave changes, ST-segment elevation and/or depression, changes in the QRS complex, and inverted T waves.

The ST-segment changes are produced by the flow of currents, referred to as “injury currents,” that are generated by the voltage gradients across the boundary between the ischemic and nonischemic myocardium during the resting and plateau phases of the ventricular action potential, which correspond to the TQ and ST segments of the ECG.^{2,3} Current guidelines suggest that when these ST-segment shifts reach

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The American Heart Association, the American College of Cardiology, and the Heart Rhythm Society make every effort to avoid any actual or potential conflicts of interest that may arise as a result of an outside relationship or a personal, professional, or business interest of a member of the writing panel. Specifically, all members of the writing group are required to complete and submit a Disclosure Questionnaire showing all such relationships that might be perceived as real or potential conflicts of interest.

Parts I and II of this series, “Recommendations for the Standardization and Interpretation of the Electrocardiogram,” were published in the March 13, 2007, issue of *Circulation* (*Circulation*. 2007;115:1306–1324 and 1325–1332). They are available online at <http://circ.ahajournals.org/content/vol115/issue10/>

Parts III, IV, V, and VI of this series are available online at <http://circ.ahajournals.org/content/vol119/issue10/> (*Circulation*. 2009;119:e235–e240; e241–e250; e251–e261; and e262–e270). They also published ahead of print February 19, 2009.

This statement was approved by the American Heart Association Science Advisory and Coordinating Committee on August 7, 2008, by the American College of Cardiology Board of Trustees on May 16, 2008, and by the Heart Rhythm Society on June 18, 2008.

The American Heart Association requests that this document be cited as follows: Wagner GS, Macfarlane P, Wellens H, Josephson M, Gorgels A, Mirvis DM, Pahlm O, Surawicz B, Kligfield P, Childers R, Gettes LS. AHA/ACCF/HRS recommendations for the standardization and interpretation of the electrocardiogram: part VI: acute ischemia/infarction: a scientific statement from the American Heart Association Electrocardiography and Arrhythmias Committee, Council on Clinical Cardiology; the American College of Cardiology Foundation; and the Heart Rhythm Society. *Circulation*. 2009;119:e262–e270.

This article has been copublished in the *Journal of the American College of Cardiology*.

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(*Circulation*. 2009;119:e262–e270.)

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Circulation is available at <http://circ.ahajournals.org>

DOI: 10.1161/CIRCULATIONAHA.108.191098

predetermined threshold values in 2 or more anatomically contiguous body surface ECG leads, a diagnosis of acute ischemia/infarction is indicated.⁴ When the ST segment is elevated, the term ST-segment–elevation myocardial infarction (STEMI) is used to describe the changes and to determine eligibility for acute reperfusion therapy. The STEMI designation is contrasted with that of NSTEMI (or non-STEMI), which includes all others, that is, those with lesser amounts of ST-segment elevation, abnormal ST-segment elevation in fewer than 2 contiguous leads, ST-segment depression, T-wave inversion, or no abnormalities at all.

The changes in the QRS complex reflect changes in electrical activation within the severely ischemic or infarcted region.⁵ The magnitude and extent of these ECG changes depend on the size and location of the ischemic/infarcted region and the relationship of this region to the spatial orientation of the particular ECG lead. The size and location of the affected region depend, in turn, on the coronary artery involved, the site of occlusion within the artery, and the presence or absence of collateral circulation.

With the advent of automated recording systems and the performance of large-scale, multicenter clinical trials, a large experience has accumulated that permits the redefinition of normal ECG characteristics and the correlation of ischemia-induced ECG abnormalities to angiographic findings obtained in temporal proximity to the recording of the ECG. As a result, the standard 12-lead body-surface ECG is now capable of providing a more accurate correlation of the waveform changes to the involved vessel and to the site of occlusion within that vessel. In addition, magnetic resonance imaging studies that correlate the ECG changes that result from ischemia/infarction to the extent and location of the involved myocardial region are beginning to appear⁶ and are expected to result in more anatomically valid nomenclature of the ischemic/infarcted region, particularly in what is now referred to as the posterior region of the left ventricle.

The purpose of this section of the standards document is to reexamine the existing ECG criteria for ischemia/infarction. Our recommendations are focused primarily on the changes in the ST segment that occur during the early acute phase of acute coronary syndromes; however, some of the T-wave changes in the postreperfusion phase and the QRS changes in the chronic phase are also considered. The following topics are discussed: (1) The meaning and importance of both ST-segment elevation and depression; (2) the concept of anatomically contiguous leads; (3) the threshold values for ST-segment changes; (4) the use of the ST-segment spatial vector to determine the region involved and the occluded coronary artery; (5) the importance of postischemic T-wave changes; (6) the diagnosis of ischemia/infarction in the presence of intraventricular conduction disturbances; and (7) quantitative QRS changes for estimation of chronic infarct size.

Meaning and Importance of ST-Segment Elevation and Depression

An understanding of the relationship between ST-segment elevation and depression requires consideration of certain bioelectric principles. One of these is that all leads are bipolar. However, only 3 leads (leads I, II, and III) use 2

dedicated electrodes, 1 connected to the positive input of the ECG machine and the other to the negative input. The remaining 9 leads use only 1 dedicated positive electrode. The negative electrode for the 6 chest leads (also referred to as the *precordial* or *V* leads) is provided by Wilson's central terminal, which is composed of the averaged inputs from the right arm, left arm, and left leg electrodes. The negative electrode for the 3 aV (or *augmented*) limb leads (aVR, aVL, and aVF) is provided by the averaged inputs from the 2 limb electrodes that do not serve as the positive electrode for a specified lead; for example, for aVR, the averaged potential of the left arm and left leg electrodes is used.

A second principle is that the ST-segment elevation in any lead will usually be associated with reciprocal ST-segment depression in leads whose positive pole is directed opposite to (ie, approximately 180° away from) the leads that show the ST-segment elevation and vice versa. If no body surface lead fulfills this condition, then only ST-segment elevation or depression will be displayed on the routine 12-lead ECG. In addition, reciprocal ST-segment change may be absent in leads in which it would be expected if the voltage transmitted to the body surface is inadequate to meet diagnostic criteria. This might occur if, in addition to ischemia/infarction, there is associated left ventricular hypertrophy with ST- and T-wave changes, an intraventricular conduction disturbance with secondary ST- and T-wave changes, or pericarditis.⁷ For this reason, the injury currents associated with acute ischemia/infarction may cause ST-segment elevation, ST-segment depression, both, or neither in any body surface lead depending on the relationship between the location of the positive and negative poles that determine the spatial orientation of that lead, the location of the ischemic region, the magnitude of the voltage transmitted to the body surface, and the presence of confounding ECG abnormalities. For instance, ST-segment depression in a lead with its positive pole to the left and superiorly placed, such as lead aVL, is the reciprocal of and similar in meaning to ST-segment elevation in a lead with its positive pole located to the right and inferiorly placed, such as lead III. Conversely, ST-segment depression in lead III is the reciprocal of and similar in meaning to ST-segment elevation in lead aVL. In the same way, ST-segment depression in leads V₁ and V₂, in which the positive pole is located anteriorly, is the reciprocal of and similar in meaning to the ST-segment elevation that would be recorded if the positive electrode were placed on the posterior chest wall, as in the V₈ and V₉ positions.

It is important to recognize that the magnitude of ST-segment elevation and reciprocal ST-segment depression (or vice versa) may not be identical because of differences in the distance of the leads recording these changes from the ischemic region and the deviation of the leads from being 180° opposite to each other. This is particularly relevant to the ST-segment changes that occur in lead aVL, because this lead frequently has a spatial orientation that is approximately perpendicular to the mean QRS vector.

It is also important to stress that factors other than acute ischemia may cause elevation or depression of the ST segment. Factors that may cause ST-segment depression include but are not limited to hypertrophy, cardioactive drugs, and lowered serum potassium. Factors that may cause ST-

segment elevation include but are not limited to pericarditis, elevated serum potassium, Osborne waves, acute myocarditis, certain cardiac tumors, and the normal variant referred to as *early repolarization*. Criteria have been published to differentiate these abnormalities in the ST segment from those associated with acute ischemia.⁷

Acute ischemia that typically results from coronary artery occlusion is associated with ST-segment elevation in leads whose positive poles are located over the ischemic region and with reciprocal ST depression in leads whose positive poles are oriented in the opposite direction. It is anatomically reasonable but conceptually limiting to refer to leads as *anterior*, *inferior*, or *lateral*, because this nomenclature refers only to the location on the body surface of the electrode that determines the positive pole of that lead. These lead descriptions, although frequently used, may mislead clinicians to consider that all ST-segment abnormalities in a particular lead, for example, an *anterior lead* such as V₁ or V₂, are located in that anatomic region and could not be the reciprocal of ischemic ST-segment changes occurring in the opposite anatomic region. Indeed, depression of the ST segment in leads V₁ and V₂ is a common manifestation of a posterior or lateral ST-segment elevation infarction. Note that the concept of subendocardial ischemia causing depression of the ST segment in multiple leads is still tenable and, as discussed below, may be thought of as the reciprocal of endocardial ST-segment elevation caused by currents of injury directed toward the ventricular chamber.

Recommendation

1. Labeling specific leads as anterior, inferior, and lateral should be avoided. Leads should be identified according to their original nomenclature, that is, leads I, II, III, aVR, aVL, aVF, and V₁ through V₆. This recommendation refers only to the labeling of the leads. Reference to the anatomic location of the ischemic/infarcted region, such as anterior or inferior, is still recommended and is based on the leads that show the ST-segment alterations. This is discussed below.

Concept of Anatomically Contiguous Leads

The current ECG standards for diagnosing acute ischemia/infarction require that ST-segment elevation be present in 2 or more contiguous leads and that the elevation of the ST segment at the J point be greater than 0.2 mV (2 mm with standard calibration) in leads V₁, V₂, and V₃ and greater than 0.1 mV in all other leads.⁴ In the classic ECG display, the chest leads V₁ through V₆ are displayed on the ECG paper in their anatomically contiguous order from right anterior (V₁) to left lateral (V₆); however, the limb leads are not. Rather, they are displayed in 2 groups of 3: leads I, II, and III and leads aVR, aVL, and aVF, which reflects their historical development. For these leads to be displayed in their anatomically contiguous manner, that is, from left superior-basal to right inferior, the display should be aVL, I, -aVR (ie, lead aVR with reversed polarity), II, aVF, and III. With this configuration, -aVR exists at 30° in the hexaxial frontal plane, ie, midway between limb lead I (at 0°) and limb lead II (at 60°). This display, known as the Cabrera format, has been the existing standard in Sweden for 25 years and was

recommended in the 2000 European Society of Cardiology/American College of Cardiology guidelines⁴ for universal adoption in 12-lead electrocardiography.

Recommendation

1. ECG machines should be equipped with switching systems that will allow the limb leads to be displayed and labeled appropriately in their anatomically contiguous sequence.

Threshold Values for ST-Segment Changes

The threshold values of ST-segment elevation of 0.2 mV (2 mm) in some leads and 0.1 mV (1 mm) in others results from recognition that some elevation of the junction of the QRS complex and the ST segment (the J point) in most chest leads is normal. Recent studies have revealed that the threshold values are dependent on gender, age, and ECG lead.⁸⁻¹² In healthy individuals, the amplitude of the ST junction is generally highest in leads V₂ and V₃ and is greater in men than in women.

Recommendations

1. For men 40 years of age and older, the threshold value for abnormal J-point elevation should be 0.2 mV (2 mm) in leads V₂ and V₃ and 0.1 mV (1 mm) in all other leads.
2. For men less than 40 years of age, the threshold values for abnormal J-point elevation in leads V₂ and V₃ should be 0.25 mV (2.5 mm).
3. For women, the threshold value for abnormal J-point elevation should be 0.15 mV (1.5 mm) in leads V₂ and V₃ and greater than 0.1 mV (1 mm) in all other leads.
4. For men and women, the threshold for abnormal J-point elevation in V₃R and V₄R should be 0.05 mV (0.5 mm), except for males less than 30 years of age, for whom 0.1 mV (1 mm) is more appropriate.
5. For men and women, the threshold value for abnormal J-point elevation in V₇ through V₉ should be 0.05 mV (0.5 mm).
6. For men and women of all ages, the threshold value for abnormal J-point depression should be -0.05 mV (-0.5 mm) in leads V₂ and V₃ and -0.1 mV (-1 mm) in all other leads.

Correlation of ST-Segment Elevation and Depression to the Region Involved and to the Occluded Vessel

It is currently proposed that the diagnosis of acute ischemia/infarction be based on the presence of ST elevation, as measured at the J point,⁴ equaling or exceeding the thresholds defined above in 2 or more anatomically contiguous leads. The affected region is identified by the position on the body surface of the leads that reveal the ST-segment elevation. The more precise the localization of the spatial vector of the ST-segment shift, as determined from analysis of leads with ST-segment depression and ST-segment elevation, the more robust will be the localization of the ischemic/infarcted region. The correlation of these ST-segment changes to the results of coronary angiography performed in temporal proximity has permitted the more accurate identification of the obstructed coronary artery and the proximal or distal location of the obstruction within that artery. These correlations have

been detailed in several publications^{7,13–15} and are reviewed below.

Anterior wall ischemia/infarction is invariably due to occlusion of the left anterior descending coronary artery and results in the spatial vector of the ST segment being directed to the left and laterally. This will be expressed as ST elevation in some or all of leads V₁ through V₆. The location of the occlusion within the left anterior descending coronary artery, that is, whether proximal or distal, is suggested by the chest leads in which the ST-segment elevation occurs and the presence of ST-segment elevation or depression in other leads.

Occlusion of the proximal left anterior descending coronary artery above the first septal and first diagonal branches results in involvement of the basal portion of the left ventricle, as well as the anterior and lateral walls and the interventricular septum. This will result in the ST-segment spatial vector being directed superiorly and to the left and will be associated with ST-segment elevation in leads V₁ through V₄, I, aVL, and often aVR. It will also be associated with reciprocal ST-segment depression in the leads whose positive poles are positioned inferiorly, that is, leads II, III, aVF, and often V₅.^{15,16} Typically, there will be more ST elevation in aVL than in aVR and more ST-segment depression in lead III than in lead II, because the ST-segment spatial vector will be directed more to the left than to the right.

When the occlusion is located between the first septal and first diagonal branches, the basal interventricular septum will be spared, and the ST segment in lead V₁ will not be elevated. In that situation, the ST-segment vector will be directed toward aVL, which will be elevated, and away from the positive pole of lead III, which will show depression of the ST segment.¹⁴ When the occlusion is located more distally, that is, below both the first septal and first diagonal branches, the basal portion of the left ventricle will not be involved, and the ST-segment vector will be oriented more inferiorly. Thus, the ST segment will not be elevated in leads V₁, aVR, or aVL, and the ST segment will not be depressed in leads II, III, or aVF. Indeed, because of the inferior orientation of the ST-segment vector, elevation of the ST segment in leads II, III, and aVF may occur. In addition, ST-segment elevation may be more prominent in leads V₃ through V₆ and less prominent in V₂ than in the more proximal occlusions.¹⁶

Recommendations

1. ECG manufacturers are encouraged to develop software capable of displaying the spatial orientation of the ST-segment vector in both the frontal and transverse planes.
2. Wherever possible, the ECG diagnosis provided by automated algorithms should refer to the occluded vessel and to the site of the occlusion within that vessel, as well as to the region involved.
3. When ST-segment elevation is present in I and aVL, as well as in leads V₁ through V₄ and sometimes in V₆, and ST-segment depression is present in leads II, III, and aVF, the automated interpretation should suggest an extensive anterior wall or anterobasal ischemia/infarction due to occlusion of the proximal portion of the left anterior descending coronary artery.

4. When ST-segment elevation is present in leads V₃ through V₆, and ST-segment depression is not present in leads II, III, and aVF, the automated interpretation should suggest anterior wall ischemia/infarction due to occlusion of the mid or distal portion of the left anterior descending coronary artery.

Inferior wall infarction that results in ST-segment elevation in only leads II, III, and aVF may be the result of occlusion of either the right coronary artery (RCA) or the left circumflex coronary artery (LCx), depending on which provides the posterior descending branch, that is, which is the dominant vessel. When the RCA is occluded, the spatial vector of the ST segment will usually be directed more to the right than when the LCx is occluded. This will result in greater ST-segment elevation in lead III than in lead II and will often be associated with ST-segment depression in leads I and aVL, leads in which the positive poles are oriented to the left and superiorly.^{17,18} When the RCA is occluded in its proximal portion, ischemia/infarction of the right ventricle may occur, which causes the spatial vector of the ST-segment shift to be directed to the right and anteriorly, as well as inferiorly. This will result in ST-segment elevation in leads placed on the right anterior chest, in positions referred to as V_{3R} and V_{4R}, and often in lead V₁.^{19–21} Lead V_{4R} is the most commonly used right-sided chest lead. It is of great value in diagnosing right ventricular involvement in the setting of an inferior wall infarction and in making the distinction between RCA and LCx occlusion and between proximal and distal RCA occlusion. It is important to recognize that the ST elevation in the right-sided chest leads associated with right ventricular infarction persists for a much shorter period of time than the ST elevation connoting inferior wall infarction that occurs in the extremity leads. For this reason, leads V_{3R} and V_{4R} should be recorded as rapidly as possible after the onset of chest pain.¹⁴ The joint task force of the AHA and the American College of Cardiology, in collaboration with the Canadian Cardiovascular Society, has recommended that right-sided chest leads V_{3R} and V_{4R} be recorded in all patients presenting with ECG evidence of acute inferior wall ischemia/infarction.²²

Recommendations

1. ECG machines should be programmed to suggest the recording of the right-sided chest leads V_{3R} and V_{4R} when ST elevation greater than 0.1 mV occurs in leads II, III, and aVF.
2. ECG machines should be equipped with a mechanism to label leads V_{3R} and V_{4R}.
3. Descriptive and interpretative statements should be developed to describe and interpret abnormalities that might occur in leads V_{3R} and V_{4R}.

ST-segment depression in leads V₁, V₂, and V₃ that occurs in association with an inferior wall infarction may be caused by occlusion of either the RCA or the LCx. This ECG pattern has been termed *posterior* or *posterolateral* ischemia since the recommendations of Perloff²³ and Horan et al²⁴ and is based on anatomic and pathological studies of ex vivo hearts. However, recent in vivo imaging techniques, including echocardiography and magnetic resonance imaging, have demon-

strated the oblique position of the heart within the thorax. These studies demonstrated that the region referred to as the posterior wall was lateral rather than posterior and led to the suggestion that the term posterior be replaced by the designation lateral.²⁵ Bayés de Luna et al⁶ correlated the ECG patterns of healed myocardial infarctions to their anatomic location as determined by magnetic resonance imaging. They reported that the most frequent cause of abnormally tall and broad R waves in leads V₁ and V₂ in patients known to have experienced a recent acute infarction was involvement of the lateral and not the posterior wall of the left ventricle. They suggested that the terms *posterior ischemia* and *posterior infarction* be replaced by the terms *lateral*, *inferolateral*, or *basal-lateral* depending on the associated changes in II, III, aVF, V₁, V₅, and V₆. Such terminology has been endorsed by the International Society for Holter and Noninvasive Electrocardiography.²⁶

Recommendation

1. It is the opinion of the committee that for the present, and not withstanding the recommendation of the International Society for Holter and Noninvasive Electrocardiography, the current ECG terminology of posterior be retained to describe ST depression in leads V₁ and V₂ and the subsequent development of broad R waves in these leads, as described in the previously published section concerning terminology.²⁷ Further collaborative studies involving larger groups of patients having a variety of ages and body builds and having acute ischemia as well as established infarctions are recommended to provide further data on this topic. This recommendation, as well as the others in this report, will be reviewed periodically to determine whether changes should be recommended.

It is not possible to determine whether the RCA or LCx vessel is occluded when changes of inferior wall ischemia/infarction are accompanied by depression of the ST segment in leads V₁, V₂, and V₃; however, the absence of such changes is more suggestive of RCA than LCx occlusion. When the LCx is occluded, the spatial vector of the ST segment in the frontal plane is more likely to be directed to the left than when the RCA is occluded. For this reason, the ST segment may be elevated to a greater extent in lead II than in lead III and may be isoelectric or elevated in leads I and aVL.²⁸ When a dominant RCA is occluded proximally, left posterolateral and right ventricular wall involvement will be present, and the posteriorly directed ST-segment vector associated with this involvement may cancel the ST-segment elevation in lead V₁ anticipated by right ventricular involvement and vice versa.

We have stated above that ST depression is always the reciprocal of ST-segment elevation in leads located opposite to the body surface leads recording the ST depression; however, in some cases, only ST elevation or depression (but not both) will be present on the routine 12-lead ECG. For instance, when ST elevation is localized to leads with positive poles that overlie a particular anatomic region, such as the midanterior left ventricular walls (eg, V₃ through V₆), there may not be reciprocal ST-segment depression on the body surface ECG because no body surface leads are routinely placed opposite to leads V₃ through V₆. In some cases, ST-

segment depression may be localized to specific leads such as V₂ and V₃, and in this situation, there will not be ST elevation on the 12-lead ECG because no body surface leads are routinely placed opposite to these leads.

ST-segment elevation in more than 1 discrete region is characteristic of pericarditis involving large portions of the epicardial surface.⁷ ST-segment depression in more than 1 discrete region that occurs in the absence of ST-segment elevation in leads I, II, III, aVL, and V₂ through V₆ implies the presence of currents of injury directed away from the body surface and toward the ventricular chamber and may indicate the presence of ischemia in more than 1 region of the heart. In these situations, leads aVR and V₁, in which the positive poles are located to the right, superiorly and anteriorly, may reveal ST elevation that reflects the spatial vector of the injury currents. Such diffuse ST-segment depression usually implies nontransmural ischemia or injury and is characteristic of 2 situations. The first is in association with stable angina pectoris and can be brought about by treadmill or bicycle exercise and other forms of stress. This is usually associated with subtotal occlusion of 1 or more coronary arteries and occurs when the oxygen demand of the myocardium is increased beyond the ability of coronary flow to meet this increased demand. Identification of the obstructed coronary artery or arteries on the basis of the ST-segment change is not possible in this setting.

In the second situation, ST depression in multiple leads occurs at rest in patients with unstable angina pectoris. This is frequently associated with severe multivessel or left main coronary artery stenosis.²⁹ It has been reported that in patients with angina at rest, ST-segment depression in 8 or more body surface ECG leads, combined with ST elevation in aVR and V₁, is associated with a 75% predictive accuracy of 3-vessel or left main stenosis.¹⁴

Recommendation

1. When the resting ECG reveals ST-segment depression greater than 0.1 mV (1 mm) in 8 or more body surface leads coupled with ST-segment elevation in aVR and/or V₁ but is otherwise unremarkable, the automated interpretation should suggest ischemia due to multivessel or left main coronary artery obstruction.

Postischemic T-Wave Changes

Often, after ischemia and infarction, T waves become inverted in leads with previous ST-segment elevation and remain inverted for varying periods that range from days to permanently. There is an important subgroup of patients in whom deeply inverted T waves, that is, greater than 0.5 mV, occur in leads V₂, V₃, and V₄, and occasionally V₅, often with significant QT prolongation, after an episode of chest pain but without further ECG evidence of evolving infarction or ongoing ischemia. This ECG pattern is similar to that which may occur after an intracranial hemorrhage (the CVA [cerebrovascular accident] pattern) and in some forms of cardiomyopathy.⁷ Coronary angiography in this subgroup of patients usually reveals severe stenosis of the proximal left anterior descending coronary artery with collateral circulation.^{30,31} If these changes are not recognized and the patients

are not evaluated and treated appropriately, a high percentage may experience an acute anterior wall infarction, with the risks associated with occlusion of the proximal left anterior descending coronary artery.³²

Recommendation

1. The specific pattern of deeply inverted T waves with QT prolongation in leads V₂ through V₄ should be interpreted as consistent with severe stenosis of the proximal left anterior descending coronary artery or with a recent intracranial hemorrhage (CVA [cerebrovascular accident] pattern).

Diagnosis of Ischemia/Infarction in the Setting of Intraventricular Conduction Disturbances

ST-segment criteria for the diagnosis of acute ischemia/infarction are not affected by the presence of fascicular blocks or by right bundle-branch block. They are affected by the presence of left bundle-branch block because of the more pronounced secondary ST- and T-wave changes that occur in this setting. Criteria for infarction in the presence of complete left bundle-branch block have been published³³ based on a retrospective analysis of patients enrolled in the GUSTO (Global Utilization of Streptokinase and Tissue plasminogen activator for Occluded coronary arteries) I trial. These include ST-segment elevation greater than or equal to 0.1 mV (1 mm) in leads with a positive QRS complex and ST depression greater than or equal to 0.1 mV (1 mm) in leads V₁ through V₃, ie, leads with a dominant S wave. These are referred to as concordant ST-segment changes. A third criterion is ST-segment elevation greater than or equal to 0.5 mV (5 mm) in leads with a negative QRS complex. These are referred to as discordant ST-segment changes. Concordant ST-segment changes and ST depression in leads V₁ through V₃ have been reported to demonstrate high specificity but low sensitivity,^{33,34} and the most recent study³⁵ reported that discordant ST changes

had very low specificity and sensitivity. It also reported that the presence of left bundle-branch block with concordant ST-segment changes was associated with a higher 30-day mortality than associated with left bundle-branch block and an enzyme rise but without concordant ST-segment changes.

Recommendation

1. Automated ECG algorithms should suggest the possibility of acute ischemia/infarction in patients with left bundle-branch block who have ST-segment changes that meet the above criteria.

Quantitative QRS Changes for Estimation of Infarct Size

One method for determining the presence of a remote myocardial infarct has been the Minnesota Code.³⁶ This method was developed for the diagnosis of infarction rather than the quantification of its size and correlates poorly with anatomically measured infarct size.³⁷ An improved correlation of changes in the QRS complex with infarct size was the development of a QRS scoring system by Selvester et al using a computer model of the human myocardial activation sequence reported by Durrer et al.³⁸ The Selvester QRS scoring system included 54 weighted criteria from the QRS complexes in 10 of the standard leads (leads I, II, aVL, aVF, and V₁ through V₆), which totaled 32 QRS points, each equivalent to approximately 3% of the left ventricular wall.³⁹ The specificity of the Selvester method has been established in normal subjects, and its ability to detect and estimate the anatomically determined sizes of prior infarctions has been documented.⁴⁰ However, these estimates are most useful in the setting of a single infarct.

Recommendation

1. Algorithms capable of determining the Selvester score in tracings that meet the criteria for prior infarctions should be developed and available for use by the reader if so desired.

Disclosures

Writing Group Disclosures

Writing Group Member	Employment	Research Grant	Other Research Support	Speakers' Bureau/Honoraria	Ownership Interest	Consultant/Advisory Board	Other
James J. Bailey	National Institutes of Health	None	None	None	None	None	None
Rory Childers	University of Chicago	None	None	None	None	None	None
Barbara J. Deal	Northwestern University	None	None	None	None	None	None
Leonard S. Gettes	University of North Carolina	None	None	None	None	None	None
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E. William Hancock	Stanford University Medical Center (retired Professor Emeritus)	None	None	None	None	Philips Medical Systems†; Covance Diagnostics†	None
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Peter Macfarlane	University of Glasgow	Cardiac Science Corp†; Medtronic Physio Control†; Spacelabs Health Care†; Draeger Medical†; Heartlab†; McKesson†	None	None	None	Cardiac Science Corp†; Medtronic Physio Control†; Spacelabs Health Care†; Draeger Medical†; Heartlab†; McKesson†	None
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Gerard van Herpen	Erasmus Medical Center	None	None	None	None	None	Welch Allyn*
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Hein Wellens	University of Maastricht	None	None	None	None	Medtronic*	None

This table represents the relationships of writing group members that may be perceived as actual or reasonably perceived conflicts of interest as reported on the Disclosure Questionnaire, which all members of the writing group are required to complete and submit. A relationship is considered to be "significant" if (1) the person receives \$10 000 or more during any 12-month period, or 5% or more of the person's gross income; or (2) the person owns 5% or more of the voting stock or share of the entity, or owns \$10 000 or more of the fair market value of the entity. A relationship is considered to be "modest" if it is less than "significant" under the preceding definition.

*Modest.

†Significant.

Reviewer Disclosures

Reviewer	Employment	Research Grant	Other Research Support	Speakers' Bureau/Honoraria	Expert Witness	Ownership Interest	Consultant/Advisory Board	Other
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Mark Eisenberg	McGill University	None	None	None	None	None	None	None
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Cindy Grines	William Beaumont Hospital	None	None	None	None	None	None	None
Mark Hlatky	Stanford University	None	None	None	None	None	None	None
Peter Kowey	Lankenau Medical Office	None	None	None	None	CardioNet†	Transoma*; CardioNet†; NewCardio*	None
Rachel Lampert	Yale University	Medtronic†; Guidant/Boston Scientific†; St. Jude†	None	None	None	None	Medtronic*	None
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*Modest.

†Significant.

References

- Kligfield P, Gettes LS, Bailey JJ, et al. Recommendations for the standardization and interpretation of the electrocardiogram: part I: the electrocardiogram and its technology: a scientific statement from the American Heart Association Electrocardiography and Arrhythmias Committee, Council on Clinical Cardiology; the American College of Cardiology Foundation; and the Heart Rhythm Society. *Circulation*. 2007;115:1306–24.
- Kléber AG, Janse MJ, van Capelle FJ, Durrer D. Mechanism and time course of S-T and T-Q segment changes during acute regional myocardial ischemia in the pig heart determined by extracellular and intracellular recordings. *Circ Res*. 1978;42:603–13.
- Surawicz S, Saito S. Exercise testing for detection of myocardial ischemia in patients with abnormal electrocardiograms at rest. *Am J Cardiol*. 1978;41:943–51.
- Myocardial infarction redefined: a consensus document of the Joint European Society of Cardiology/American College of Cardiology Committee for the Redefinition of Myocardial Infarction. *Eur Heart J*. 2000;21:1502–13.
- Birnbaum Y, Herz I, Sclarovsky S, et al. Prognostic significance of the admission electrocardiogram in acute myocardial infarction. *J Am Coll Cardiol*. 1996;27:1128–32.
- Bayés de Luna A, Cino JM, Pujadas S, et al. Concordance of electrocardiographic patterns and healed myocardial infarction detected by cardiovascular magnetic resonance. *Am J Cardiol*. 2006;97:443–51.
- Surawicz B, Knilans T. *Chou's Electrocardiography in Clinical Practice*. 5th ed. Philadelphia, Pa: WB Saunders; 2001.
- Macfarlane PW. Age, sex, and the ST amplitude in health and disease. *J Electrocardiol*. 2001;34(suppl):235–41.
- Macfarlane PW, Browne D, Devine B, et al. Modification of ACC/ESC criteria for acute myocardial infarction. *J Electrocardiol*. 2004;37(suppl):98–103.

10. Wu J, Kors JA, Rijnbeek PR, et al. Normal limits of the electrocardiogram in Chinese subjects. *Int J Cardiol.* 2003;87:37–51.
11. Surawicz B, Parikh SR. Prevalence of male and female patterns of early ventricular repolarization in the normal ECG of males and females from childhood to old age. *J Am Coll Cardiol.* 2002;40:1870–6.
12. Macfarlane PW, Petryka J, Kaczmarska E. Normal limits of ST_{Tj} in V3R-V6R and V7-V9. *Eur Heart J.* 2006;27(suppl):918. Abstract.
13. Sgarbossa EB, Birnbaum Y, Parrillo JE. Electrocardiographic diagnosis of acute myocardial infarction: current concepts for the clinician. *Am Heart J.* 2001;141:507–17.
14. Wellens HJJ, Gorgels APM, Doevendans PA. *The ECG in Acute Myocardial Infarction and Unstable Angina: Diagnosis and Risk Stratification.* Boston, Mass: Kluwer Academic Publishers; 2004.
15. Birnbaum Y, Sclarovsky S, Solodky A, et al. Prediction of the level of left anterior descending coronary artery obstruction during anterior wall acute myocardial infarction by the admission electrocardiogram. *Am J Cardiol.* 1993;72:823–6.
16. Engelen DJ, Gorgels AP, Cheriex EC, et al. Value of the electrocardiogram in localizing the occlusion site in the left anterior descending coronary artery in acute anterior myocardial infarction. *J Am Coll Cardiol.* 1999;34:389–95.
17. Herz I, Assali AR, Adler Y, et al. New electrocardiographic criteria for predicting either the right or left circumflex artery as the culprit coronary artery in inferior wall acute myocardial infarction. *Am J Cardiol.* 1997;80:1343–5.
18. Zimetbaum PJ, Krishnan S, Gold A, et al. Usefulness of ST-segment elevation in lead III exceeding that of lead II for identifying the location of the totally occluded coronary artery in inferior wall myocardial infarction. *Am J Cardiol.* 1998;81:918–9.
19. Braat SH, Brugada P, de Zwaan C, et al. Value of electrocardiogram in diagnosing right ventricular involvement in patients with an acute inferior wall myocardial infarction. *Br Heart J.* 1983;49:368–72.
20. Zalenski RJ, Rydman RJ, Sloan EP, et al. Value of posterior and right ventricular leads in comparison to the standard 12-lead electrocardiogram in evaluation of ST-segment elevation in suspected acute myocardial infarction. *Am J Cardiol.* 1997;79:1579–85.
21. Correale E, Battista R, Martone A, et al. Electrocardiographic patterns in acute inferior myocardial infarction with and without right ventricular involvement: classification, diagnostic and prognostic value, masking effect. *Clin Cardiol.* 1999;22:37–44.
22. Antman EM, Anbe DT, Armstrong PW, et al. ACC/AHA guidelines for the management of patients with ST-elevation myocardial infarction: executive summary: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Writing Committee to revise the 1999 guidelines for the management of patients with acute myocardial infarction) [published correction appears in *J Am Coll Cardiol.* 2005;45:1376]. *J Am Coll Cardiol.* 2004;44:671–719.
23. Perloff JK. The recognition of strictly posterior myocardial infarction by conventional scalar electrocardiography. *Circulation.* 1964;30:706–18.
24. Horan LG, Flowers NC, Johnson JC. Significance of the diagnostic Q wave of myocardial infarction. *Circulation.* 1971;43:428–36.
25. Cerqueira MD, Weissman NJ, Dilsizian V, et al; American Heart Association Writing Group on Myocardial Segmentation and Registration for Cardiac Imaging. Standardized myocardial segmentation and nomenclature for tomographic imaging of the heart: a statement for healthcare professionals from the Cardiac Imaging Committee of the Council on Clinical Cardiology of the American Heart Association. *Circulation.* 2002;105:539–42.
26. Bayés de Luna A, Wagner G, Birnbaum Y, et al; International Society for Holter and Noninvasive Electrocardiography. A new terminology for left ventricular walls and location of myocardial infarcts that present Q wave based on the standard of cardiac magnetic resonance imaging: a statement for healthcare professionals from a committee appointed by the International Society for Holter and Noninvasive Electrocardiography. *Circulation.* 2006;114:1755–60.
27. Mason JW, Hancock EW, Gettes L, et al. Recommendations for the standardization and interpretation of the electrocardiogram: part II: electrocardiography diagnostic statement list: a scientific statement from the American Heart Association Electrocardiography and Arrhythmias Committee, Council on Clinical Cardiology; the American College of Cardiology Foundation; and the Heart Rhythm Society. *Circulation.* 2007;115:1325–32.
28. Bairey CN, Shah PK, Lew AS, Hulse S. Electrocardiographic differentiation of occlusion of the left circumflex versus the right coronary artery as a cause of inferior acute myocardial infarction. *Am J Cardiol.* 1987;60:456–69.
29. Gorgels AP, Vos MA, Mulleneers R, et al. Value of the electrocardiogram in diagnosing the number of severely narrowed coronary arteries in rest angina pectoris. *Am J Cardiol.* 1993;72:999–1003.
30. De Zwaan C, Bär FW, Gorgels AP, Wellens HJ. Unstable angina: are we able to recognize high-risk patients? *Chest.* 1997;112:244–50.
31. De Zwaan C, Bär FW, Wellens HJ. Characteristic electrocardiographic pattern indicating a critical stenosis high in left anterior descending coronary artery in patients admitted because of impending myocardial infarction. *Am Heart J.* 1982;103:730–6.
32. Simon K, Hackett D, Szelier A, et al. The natural history of postischemic T-wave inversion: a predictor of poor short-term prognosis? *Coron Artery Dis.* 1994;5:937–42.
33. Sgarbossa EB, Pinski SL, Barbagelata A, et al; GUSTO-I (Global Utilization of Streptokinase and Tissue Plasminogen Activator for Occluded Coronary Arteries) Investigators. Electrocardiographic diagnosis of evolving acute myocardial infarction in the presence of left bundle-branch block [published correction appears in *N Engl J Med.* 1996;334:931]. *N Engl J Med.* 1996;334:481–7.
34. Shlipak MG, Lyons WL, Go AS, et al. Should the electrocardiogram be used to guide therapy for patients with left bundle-branch block and suspected myocardial infarction? *JAMA.* 1999;281:714–9.
35. Wong CK, French JK, Aylward PE, et al; HERO-2 Investigators. Patients with prolonged ischemic chest pain and presumed-new left bundle branch block have heterogeneous outcomes depending on the presence of ST-segment changes. *J Am Coll Cardiol.* 2005;46:29–38.
36. Blackburn H, Keys A, Simonson E, et al. The electrocardiogram in population studies: a classification system. *Circulation.* 1960;21:1160–75.
37. Pahlm US, Chaitman BR, Rautaharju PM, et al. Comparison of the various electrocardiographic scoring codes for estimating anatomically documented sizes and single and multiple infarcts of the left ventricle. *Am J Cardiol.* 1998;81:809–15.
38. Durrer D, van Dam RT, Freud GE, et al. Total excitation of the isolated human heart. *Circulation.* 1970;41:899–912.
39. Startt-Selvester RH, Wagner GS, Ideker RE. Myocardial infarction. In: Macfarlane PW, Lawrie TDV, eds. *Comprehensive Electrocardiology: Theory and Practice in Health Disease.* New York, NY: Pergamon Press; 1989:565–629.
40. Hindman NB, Schocken DD, Widmann M, et al. Evaluation of a QRS scoring system for estimating myocardial infarct size, V: specificity and method of application of the complete system. *Am J Cardiol.* 1985;55:1485–90.

KEY WORDS: AHA Scientific Statements ■ electrocardiography ■ infarction ■ ischemia ■ electrophysiology

AHA/ACCF/HRS Recommendations for the Standardization and Interpretation of the Electrocardiogram: Part VI: Acute Ischemia/Infarction: A Scientific Statement From the American Heart Association Electrocardiography and Arrhythmias Committee, Council on Clinical Cardiology; the American College of Cardiology Foundation; and the Heart Rhythm Society: Endorsed by the International Society for Computerized Electrocardiology

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Circulation. 2009;119:e262-e270; originally published online February 19, 2009;
doi: 10.1161/CIRCULATIONAHA.108.191098

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

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