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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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

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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 predetermined thresh-

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old 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 appear (6) 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 (i.e., 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 V_1 and V_2, 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 V_8 and V_9 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-

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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 V_1 or V_2, 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 V_1 and V_2 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 V_1 through V_6. 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 V_1, V_2, and V_3 and greater than 0.1 mV in all other leads (4). In the classic ECG display, the chest leads V_1 through V_6 are displayed on the ECG paper in their anatomically contiguous order from right anterior (V_1) to left lateral (V_6); 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 (i.e., lead aVR with reversed polarity), II, aVF, and III. With this configuration, −aVR exists at 30° in the hexaxial frontal plane, that is, 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 guidelines (4) 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 V_2 and V_3 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 V_2 and V_3 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 V_2 and V_3 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 V_2 and V_3 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 V_3R and V_4R 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 V_7 through V_9 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 V_2 and V_3 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

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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 V_1 through V_6. 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 V_1 through V_4, 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 V_5 (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 V_1 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 V_1, 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 V_3 through V_6 and less prominent in V_2 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 V_1 through V_4 and sometimes in V_6, 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 V_3 through V_6, 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 V_1 (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 V_1, V_2, and V_3 that occurs in association with an inferior wall infarction may be caused

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by occlusion of either the RCA or the LCx. This ECG pattern has been termed *posterior* or *posterolateral* ischemia since the recommendations of Perloff (23) and Horan et al (24) 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 demonstrated 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 al (6) 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 V_1 and V_2 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, V_1, V_5, and V_6. 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 V_1 and V_2 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 V_1, V_2, and V_3; 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 1 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 V_1 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 (e.g., V_3 through V_6), there may not be reciprocal ST-segment depression on the body surface ECG because no body surface leads are routinely placed opposite to leads V_3 through V_6. In some cases, ST-segment depression may be localized to specific leads such as V_2 and V_3, 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 V_2 through V_6 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 V_1, 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 V_1, 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 V_1 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 V_2, V_3, and V_4, and occasionally V_5, often with

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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 V_2 through V_4 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 published (33) based on a retrospective analysis of patients enrolled in the GUSTO (Global Utilization of Streptokinase and Tissues 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 V_1 through V_3, that is, 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 V_1 through V_3 have been reported to demonstrate high specificity but low sensitivity (33,34), and the most recent study (35) 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 V_1 through V_6), 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.

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Disclosures

Writing Group Disclosures

Writing Group Member	Employment	Research Grant	Other Research Support	Speakers' Bureau/ Honoraria	Ownership Interest	Consultant/Advisory Board	Other
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Galen S. Wagner	Duke University Medical Center	Medtronic†; Physiocontrol†; Welch Allyn†	None	None	None	None	None
Hein Wellens	University of Maastricht	None	None	None	None	Medtronic*	None

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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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Mark Hlatky	Stanford University	None	None	None	None	None	None	None
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Rachel Lampert	Yale University	Medtronic†; Guidant/Boston Scientific†; St. Jude†	None	None	None	None	Medtronic*	None
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Stuart A. Winston	Michigan Heart, PC	Medtronic*; Boston Scientific*	None	Boston Scientific*	None	None	None	None

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*Modest.

†Significant.

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