Special Article

Thoughts About the Abnormalities in the Electrocardiogram of Patients with Acute Myocardial Infarction with Emphasis on a More Accurate Method of Interpreting S-T Segment Displacement: Part I

J. WILLIS HURST, M.D., M.A.C.P

Emory University, Atlanta, Georgia, USA

Summary

The QRS, S-T, and T wave abnormalities produced by the usual myocardial infarction are discussed in Part I of this manuscript.

The recent guidelines supplied by the ACC/AHA Practice Guidelines divide primary S-T segment displacement into S-T segment elevated myocardial infarction (STEMI) and non S-T segment elevated myocardial infarction (NSTEMI). Accordingly, the electrophysiologic mechanisms responsible for epicardial and subendocardial injury that produce the S-T segment abnormalities are discussed in this manuscript. In this regard, the author suggests that the Grant method of interpreting the 12 lead electrocardiogram, which uses basic principles and vector concepts, is a more accurate method of identifying epicardial injury and subendocardial injury than memorizing the rules provided by the guidelines. An electrocardiogram is shown to illustrate this point. In Addition, the author expresses his personal view that labeling an electrocardiogram as NSTEMI but not stating what is actually present in the tracing is a cumbersome method of communication. The author believes it is better communication to state what *is present* rather than to state what is *not present*. At best, the result of both assessments should be stated in the interpretation.

Part II of the manuscript is devoted to a discussion as to why treatment with thrombolytic or percutaneous coronary intervention is more effective in patients whose electrocardiograms reveal epicardial injury than it is in patients whose electrocardiograms reveal subendocardial injury or no injury.

Key words: epicardial injury, subendocardial injury, electrocardiographic signs of infarction, errors interpreting the electrocardiogram, S-T segment displacement

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The displacement of the S-T segment in the electrocardiogram has become one of the most important abnormalities in all of medicine because: ACC/AHA Practice Guidelines indicate that thrombolytic therapy is beneficial for patients with acute myocardial infarction when there is “S-T segment elevation.”^{1,2} The results of nine clinical trials have indicated that thrombolytic therapy is not beneficial for patients with acute myocardial infarction when there is “S-T segment depression,” when the S-T segment remains at baseline, or when only T wave abnormalities are present.³

Each day a countless number of physicians who do not have access to percutaneous coronary intervention, use the S-T segment to guide them in the use of thrombolytic therapy for the treatment of acute myocardial infarction. Some of them have memorized the criteria needed to identify S-T segment elevated myocardial infarction (STEMI) and non-S-T segment elevated myocardial (NSTEMI), but may or may not realize that they are trying to discover the presence of predominant epicardial injury and predominant subendocardial injury in the electrocardiogram. Whereas the pattern recognition method of identifying the location of injured myocytes seems to be adequate most of the time, a more accurate method of electrocardiographic analysis is available. In addition, the mechanisms responsible for epicardial and subendocardial injury need emphasizing. Therefore

Address for reprints:

J. Willis Hurst, M.D., M.A.C.P

Emory University

1462 Clifton Road, N.E.

Suite 301

Atlanta, Georgia 30322

e-mail: jhurst@emory.edu

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it seems proper to review the subject of S-T segment displacement as well as Q wave and T wave abnormalities in the electrocardiogram of patients with acute myocardial infarction; to ask a worrisome question—is there a more accurate way of describing S-T segment displacement due to epicardial and subendocardial injury; to show an electrocardiogram that illustrates how an error of interpretation can be made; to list some of the problems related to the correlation of data; to discuss why thrombolytic therapy is beneficial for patients with a myocardial infarction who show epicardial injury in the electrocardiogram, but is not beneficial for patients who do not show epicardial injury; and to mention some unsolved problems.

Important Background Information

When an atherosclerotic plaque in a coronary artery ruptures or cracks it may initiate an acute coronary syndrome. This causes either sudden death, unstable angina pectoris, acute myocardial infarction, a serious nonfatal cardiac arrhythmia, or abrupt conduction system abnormalities. Which one of these events occurs is determined by the location and size of the obstructed coronary artery, the intensity of the ischemia that is produced by the occlusive process, the duration of the occlusive process, the amount of coronary artery thrombosis and spasm that develops, the amount of isoemboli that occur, and the presence or absence of collateral circulation. This communication deals mainly with only one of the subsets of the acute coronary syndrome—acute myocardial infarction.

There are three major electrocardiographic abnormalities associated with an acute myocardial infarction. They are as follows: abnormal Q waves, abnormal S-T segments, and abnormal T waves. Appley and his colleagues reported in their review of nine clinical trials, that described the electrocardiographic findings of 60,000 patients with acute myocardial infarction, that 4% had bundle branch block, 68% had S-T segment elevation, 7% had S-T segment depression and 17% had other abnormalities (inverted T waves or some other nonspecific pattern), and 5% had near normal electrocardiograms.³ Currently, non-S-T segment elevated myocardial infarction is being seen more often than it was previously because the clinical recognition of myocardial infarction is improving.

Abnormal Q Waves

Abnormal Q waves are produced when an acute myocardial infarction destroys a segment of the subendocardium of the left or right ventricles making the area nonconductive (Fig. 1). Electrical forces produced by the intact diametrically opposite ventricular myocardium

will then influence the electrical field and produce an abnormally directed Q wave vector.

There are many reasons why the electrocardiogram may not show abnormal Q waves in patients with an acute myocardial infarction. Obviously, it takes a finite number of dead myocytes to accomplish this and small infarcts may not produce abnormal Q waves. The dead cardiac muscle may be located mid-way between the endocardium and epicardium, high in the right ventricle, or in a papillary muscle where an abnormal Q wave cannot be generated. Also, if the diametrically opposite myocardium has been previously damaged by an old infarct, a new abnormal Q wave may not be generated by a new infarct. The same is true when an infarct is located in, or near, the apex of the heart—there is no myocardium opposite the cardiac apex, only the aortic and mitral valves. Accordingly, new abnormal Q waves may not develop in such patients. Also, abnormal Q waves may gradually become smaller after weeks or months, perhaps because the scar becomes smaller. Finally, every observer has seen abnormal Q waves disappear when they are caused by an acute myocardial infarction suggesting that, for a brief period of time, the myocytes act as if they are electrically dead, but are able to recover.

Using the Grant method of analysis, the mean spatial direction of electrical forces that occur during the initial 0.02–0.04 s of the QRS complexes can be easily computed.⁴ The initial 0.02–0.04 s of the QRS complex can be represented as a vector that is directed away from a *segment* of dead myocardium. The area of dead nonconductive tissue of most acute infarctions is larger in the subendocardial area than it is in the epicardial area of the ventricles (Fig. 1). Such infarcts are not referred to as subendocardial infarcts. A *subendocardial infarction* is said to be present when the dead area is located in a rather large portion of the subendocardium (Fig. 2). Abnormal Q waves may not be seen in these patients initially, and as will be discussed subsequently, the condition is diagnosed electrocardiographically by the persistence of an S-T segment vector that is directed away from the centroid of the endocardium.

As stated, the Q wave vector tends to point away from the segment of the subendocardium that is dead. However, it does not point exactly away from the dead area because the direction of the abnormal Q wave vector is created by the vector sum of the remaining normal Q wave electrical forces plus the new electrical forces produced by the infarct.

The duration of the abnormal Q wave is often, but not always, 0.03–0.04 s in duration.⁵ For example, a septal infarct may produce an abnormal Q wave that is 0.02 s or less in duration. After all, the septum is normally depolarized in less than 0.02 s—a septal infarct cannot produce a Q wave that is 0.04 s in duration. It can, of course, contribute to the creation of a QS complex, but this is not a Q wave followed by an R wave—it is a

FIG. 1 This conceptual illustration is designed to show the electrocardiographic abnormalities produced by the usual myocardial infarction.

The mean spatial *Q* wave vector is directed away from the *dead* portion of the myocardium. Note that the dead area does not extend to the endocardium or to the epicardium. The dead area is larger in the subendocardium than it is near the epicardium. The mean spatial *S-T* vector is directed toward the area of predominant *epicardial injury*.

The mean spatial *T* vector is directed away from the area of predominant *epicardial ischemia*.

Each of the three vectors influences all 12 of the routine electrocardiographic leads, and any other lead that might be used, in a very predictable way. Note how the deflections would appear in leads I, AV_F , and V_3 .

The diagram illustrates how *S-T* segment elevation is created.

QS complex. Only the first part of the QS complex is produced by the septal infarct.

When abnormal *Q* waves are noted in the electrocardiogram of patients with an acute myocardial infarction the condition is usually referred to as a *Q* wave myocardial infarction.

Abnormal S-T Segments

Any deviation of the *S-T* segment from the baseline of the electrocardiogram must be identified and explained. Errors are commonly made when the interpreter looks at the leads showing moderate *S-T* segment displacement, but ignores lesser *S-T* segment displacement that is present in other leads.

There are *secondary causes* of abnormal *S-T* segment displacement and *primary causes* of abnormal *S-T* segment displacement.^{6,7}

Secondary Causes of S-T Segment Displacement

Secondary causes of *S-T* segment displacement occurs in patients with right and left ventricular hypertrophy

due to systolic pressure overload; diastolic pressure overload of the left ventricle; and in patients with right or left ventricular conduction system block.^{6,7} When these conditions are present the *S-T* segment displacement, when represented as a mean spatial vector, is directed relatively parallel with the mean spatial *T* wave vector. The displaced *S-T* segments are due to repolarization forces and are part of the *T* waves. A notable exception to this rule is a vector that represents the *S-T* segment displacement due to digitalis, which is directed opposite to the mean *QRS* vector. Digitalis does not produce a change in direction of the mean spatial *QRS* vector. Also, the direction of the mean spatial *T* vector does not change significantly—it simply becomes shorter. As the *S-T* segment vector becomes larger, the *T* wave vector becomes smaller and smaller and the *Q-T* interval becomes shorter. The so-called *S-T* segment displacement is due to early repolarization and, when due to digitalis, begins when the very first part of the *QRS* complex is produced. Its direction is reversed because the transmural pressure gradient seems to be partially eliminated by digitalis.⁸ This

FIG. 2 This conceptual illustration is designed to show the vector arrangement of electrocardiographic abnormalities produced by subendocardial injury and subendocardial infarction.

The mean spatial S-T vector is directed away from *subendocardial injury*. The area of injury may be segmental or more generalized. It may occur transiently due to spontaneous angina pectoris or during an exercise stress test, or it may be persistent, indicating subendocardial infarction. When it is localized it may morph into the abnormality shown in figure 1. When there is a large, generalized subendocardial infarction it may create a critical state (see text).

The abnormally directed S-T vector influences all 12 of the routine electrocardiographic leads, and any other lead that may be used. Note how the S-T segment deflection would appear in leads I, AV_F and V_5 .

This diagram illustrates how S-T segment depression is created.

reverses the direction of the early stage of repolarization.

Normal young adults may exhibit displacement of the S-T segments. The direction of the mean spatial S-T vector in such cases parallels the direction of the large normally directed mean spatial T wave vector. The S-T segment displacement in the normal young adults is due to early repolarization. The repolarization does not begin as early as it does when it is due to digitalis medication and therefore the T producing process is not reversed.

Diastolic pressure overload of the left ventricle may be accompanied by a mean S-T segment vector that is parallel with the large, but normally directed mean T vector. This type of secondary S-T segment abnormality is seen in athletes and in patients with aortic regurgitation or mitral regurgitation who have diastolic overload of the left ventricle.

Primary Cause of S-T Segment Displacement

A primary S-T segment abnormality is due to myocyte injury, such as occurs with hypoxia, or from the myocyte

damage that occurs with pericarditis.^{6,7} The electrical charges that are created when the myocytes are polarized are not sustained—they leak and produce an *injury* current, which is a new *primary* electrical force. This type of S-T segment displacement is referred to in electrocardiographic language as *injury—not ischemia*. The T wave abnormality due to hypoxia occurs when the repolarization process is simply delayed in a specified portion of the heart—there is no leakage of electrical charges. A-T wave abnormality due to hypoxia is referred to as being *due to ischemia*. Accordingly, it is not correct to describe a primary S-T segment abnormality as being ischemic—it is *injured*. These terms have been used for nearly a century, and although they are a bit confusing, they do signify the status of the myocytes when there is a primary S-T segment abnormality.

The mean spatial S-T vector points toward predominant epicardial injury (Fig. 1).⁹ When it is associated with the usual myocardial infarction it is located in a *segment* of the left or right ventricle. Pericarditis is usually generalized so the mean spatial S-T segment vector is almost always directed toward the cardiac apex unless

there is a reason for it to be segmental such as an acute infarction, surgery, or trauma.

The mean spatial S-T vector is directed away from predominate subendocardial injury (Fig. 2).⁹ This abnormality occurs transiently with spontaneous episodes of angina pectoris and it is the electrocardiographic abnormality that occurs during or after a stress exercise test. When the S-T segment abnormality persists it heralds the development of a subendocardial infarction.

There are approximately ten different types of myocardial infarction. The direction of the mean spatial S-T segment vector determines their location (Fig. 3).

It is not clear when in the electrical cycle the injury current is actually produced. One school of thought holds that the depolarization process removes all of the electrical charges from all of the depolarized cells, including those that are injured. If injured cells are stripped of their charges, they cannot create electrical forces. The electrical charges are built back on the myocytes, including those that were injured, by the repolarization process suggesting that the injury current actually occurs during the T-Q interval.¹⁰ A spatial vector representing the T-Q segment displacement would be directed away from epicardial injury. When the stylus of the electrocardiograph machine is moved to the center

of the recording paper the battery enters an electrical force that is equal to, but opposite to the electrical force created by the injured myocytes. This creates an artificial S-T segment displacement that abuts the last portion of the QRS complex. When this occurs, the mean spatial S-T segment vector points toward predominant epicardial injury. Others state that the S-T segment displacement is due to the injured cells and that the depolarization process does not wipe off all of the electrical charges.¹¹ Still others believe that S-T segment displacement is due to a combination of the two mechanisms.

The important point that is germane to the subject of this essay is: except when it is associated with a left ventricular aneurysm, S-T segment elevation due to predominant epicardial injury does not persist—it usually disappears by returning to the baseline in hours, days, or weeks after the onset of an acute myocardial infarction. The injured cells either die and join the dead area of the infarct, join the ischemic T waves, which may be abnormal due simply to a delay in repolarization, or return to normal. *Thrombolysis encourages the injured cells to join the cells that produce the abnormal T waves or return to normal. In other words, thrombolytic therapy prevents the additional death of myocytes by eliminating*

FIG. 3 This conceptual illustration is designed to show the approximate direction of the mean spatial S-T segment vector that is produced by ten different myocardial infarcts. Note how the direction of the spatially oriented mean S-T segment vector identifies epicardial and subendocardial injury, and determines the leads in which the S-T segment is elevated or depressed.

The names of the infarcts are listed below.

(i) Septal (ii) Anteroseptal, (iii) Anterolateral, (iv) Posterolateral, (v). apical, (vi) inferior, (vii) infero-posterior (viii) right ventricular (ix) true posterior (the vector is directed straight back), and (x) subendocardial.

When the location of the ventricles and coronary arteries are visualized, an interpreter can identify the area of the heart muscle that is infarcted, the culprit artery causing it, and the location of the obstruction in the artery most of the time. This is not always possible because some areas of the heart are nourished by more than one coronary artery.

or decreasing primary S-T segment displacement. As discussed later, thrombolytic therapy is beneficial and useful in decreasing the size of the S-T segment elevation produced by epicardial injury, but is not useful or beneficial when used for S-T segment depression that is produced by subendocardial injury.³

Prolonged subendocardial injury may morph into abnormal Q waves and epicardial injury and act like the usual acute myocardial infarction. Accordingly, an electrocardiogram should be recorded at frequent intervals in a patient with the characteristic chest pain of myocardial ischemia and S-T segment displacement due to subendocardial injury in order to record S-T segment elevation that may develop later. On the other hand, subendocardial injury may become a massive subendocardial infarct due to necrosis of a large circumferential area of the left ventricular endocardium.

The direction of the mean spatial S-T segment vector produced by predominant epicardial injury can be used to determine the location of an infarction. It does so with more precision than the direction of the mean spatial Q wave vector or the direction of the mean spatial T wave vector. This is true because the mean spatial S-T segment vector is usually a relatively pure vector whereas a mean spatial Q wave vector and a mean spatial T vector are produced by the vector sum of new and old electrical forces. Even so, it is wise to remember that the dead zone, injured, and ischemia areas of an acute infarction are not perfectly concentric and that vectors representing the dead and ischemic areas may not be located exactly where the mean spatial S-T vector is directed. The direction of the mean spatial S-T vector is an excellent indicator of the location of the occluding thrombus in the culprit coronary artery. This fact reinforces the point that an interpreter must visualize the anatomic location of the ventricles and coronary arteries and identify where the mean S-T segment vector crosses the coronary artery. This commonly indicates the location of the area of coronary artery occlusion.

Currently, it is customary to identify STEMI, S-T segment depression myocardial infarction, or T wave myocardial infarction. It is popular to use the term NSTEMI. This term is not a satisfactory one because it does not describe what is present in the electrocardiogram. For example a T-wave infarct may be called a NSTEMI—it is always better communication to state what is present rather than to state what is absent—it is better to call it a T-wave infarct. The worrisome question is—is this common method of identifying epicardial and subendocardial injury the best way to do so?

Abnormal T Waves

The T-waves may become abnormal when there is an acute myocardial infarction. *The mean T-vector points away from an area of predominant epicardial ischemia*

(Fig. 1). Predominate epicardial ischemia may be localized, as it is with the usual myocardial infarction, or generalized as it usually is with catecholamine produced epicardial coronary spasm and direct myocyte damage in patients with an acute cerebral catastrophe and in patients with the “tako tsubo” syndrome.¹² In addition, a generalized epicardial T-wave abnormality usually occurs late in the course of pericarditis.

A T-wave vector may point toward localized subendocardial ischemia that occurs early in the course of myocardial infarction and is referred to as a hyperacute T-wave abnormality.¹³ This abnormality probably occurs more often than is appreciated, but it is transient and usually disappears before an electrocardiogram can be recorded.

It is sometimes useful to calculate the direction and size of the ventricular gradient in order to identify secondary and primary T-wave abnormalities when there is left or right hypertrophy or left or right ventricular conduction system block.^{14,15}

The direction of the T-wave vector is a rather poor indicator of the location of a myocardial infarction because the T-wave abnormality is the vector sum of the normal T-forces plus the abnormal T-forces created by the ischemia of infarction.

T-wave abnormalities may persist forever or gradually return to normal, or near normal, following a myocardial infarction. The abnormal myocytes that are responsible for the T-wave abnormality seldom shift to enlarge the dead area. The persistence of abnormal T-waves deserve a comment. Electrical charges are not leaked as they are with more severe myocyte injury. The repolarization process is simply delayed. This reverses the direction of repolarization of the affected myocytes and, when a sufficient number are involved the direction of the mean spatial T-vector will be altered. Dyskinetic areas may be seen in the echocardiogram in patients with persistent T-wave abnormalities but this does not indicate that there is an area of dead myocardium. The T-wave abnormality is due to a delay in repolarization of myocytes, which delays depolarization and myocardial contraction in the affected area.

When there is *only a T-wave abnormality* the myocardial infarct should be labeled as being a T-wave infarction. It should not be labeled as NSTEMI. As stated earlier, it is better communication to state what is present rather than to state what is not present.

A Worrisome Question

Is the commonly used method of identifying predominant epicardial injury and subendocardial injury the best way to do so? The guidelines state that STEMI can be identified in patients with myocardial infarction when there is S-T segment elevation in two contiguous extremity leads or two adjacent chest leads. Some investigators

only recommend thrombolytic therapy if the S-T segment elevation is equal to, or is more than, 1 mm in the extremity leads and equal to, or is more than 2 mm in the precordial leads.¹⁶ These measurements are stressed because S-T segment depression, or no S-T segment displacement, should not trigger the use of thrombolytic therapy.

The interpreters of electrocardiograms are trying to identify predominate epicardial and subendocardial injury when they describe S-T segment elevation and depression. A major purpose of this article is to emphasize that the use of vector concepts and basic principles as described by Grant may produce a more accurate identification of the location of predominate epicardial and subendocardial injury than the descriptive method that is commonly used.¹⁷

This is not the place to repeat the details of the Grant Method of analysis. The details of his work can be found in Refs. 17–19. Using his method it is relatively easy to determine the approximate direction and size of the electrical forces that create the deflections seen in the 12 routinely recorded leads. The direction and size of the normal diagnostic cardiac vectors is known and the frontal and transverse view of the cardiac anatomy, including the location of the coronary arteries can be visualized. Accordingly, it is possible to *measure* the duration of the P-wave, QRS complex, Q-T interval and to identify, and to *measure* in degrees the direction and size of the eight diagnostic cardiac vectors. The eight diagnostic cardiac vectors are as follows: the mean direction and size of a vector representing the P-wave, the direction and size of a vector representing the first half of the P-wave, the direction and size of a vector representing the last half of the P-wave, the direction and size of a vector representing the mean QRS complex, the direction of a vector representing the first 0.02–0.04 s of the QRS complex, the direction of a vector representing the last 0.02 s of the QRS complex, and the direction and size of a vector representing the mean spatial S-T segment and a vector representing the mean spatial T-vector. These electrical forces are called diagnostic vectors because they are used to identify the different abnormalities that are produced by heart disease. They were chosen after identifying them in cathode ray produced vectorcardiograms. They identify the portion of the P-QRS-T loops that are altered by heart disease.

Grant's method of using vector concepts and basic principles to interpret electrocardiograms fulfils Lord Kelvin's admonition about the absolute need to measure "*what you are speaking about.*" The simple method of memorizing patterns in the electrocardiogram does not do that.

Lord Kelvin wrote—

*When you can measure what you are speaking about, and express it in numbers, you know something about it; but, when you cannot express it in numbers, your knowledge is of a meager and unsatisfactory kind: it may be the beginning of knowledge, but you have scarcely, in your thoughts, advanced to the stage of science.

Stated briefly, an interpreter who uses the Grant method of interpretation should: know how the six extremity leads and six precordial leads are created; understand why the center of the hexaxial reference system can be assumed to be located in the middle of the ventricles; and be able to visualize the spatial location of the atria, ventricles, conduction system, and coronary arteries in the frontal and transverse planes. The interpreter should then identify the portion of the electrocardiogram that he or she wishes to study and apply the following rules.

Step one: Inspect the six extremity leads. The *area* subtended by the part of the electrocardiogram being studied should be used.

When an electrical force, which can be represented by an arrow, is parallel to a given extremity lead axis, it records its largest deflection on that axis. When an electrical force is perpendicular to a given extremity lead axis, it records its smallest deflection on that axis. The polarity of the electrical force being measured must fit the polarity of the six extremity leads.

This approach is used to establish the *frontal plane direction* of an electrical force within about 5% of its actual location.

Step two: Inspect the six precordial leads. The area subtended by the part of the electrocardiogram being studied should be used.

The frontal plane direction of the electrical force that was calculated in step I must be retained. A zero potential plane is located at the origin of the force. The plane extends to the body surface where it becomes the transitional pathway. This plane is perpendicular to the force that produces it. One inspects the deflections recorded in the chest leads and identifies the precordial lead that reveals the smallest representation of the electrical force that is being studied. The precordial electrode position that contains the smallest representation of the electrical force is recording from the transitional pathway, which is the edge of the zero potential plane that is perpendicular to the electrical force. This enables the interpreters to determine how many degrees the force is located anteriorly or posteriorly.

Note: The largest complex seen in the precordial leads cannot be used to state that the electrical force is parallel to it. One can state that the smallest complex is recorded from the transitional pathway, which identifies the location of the zero potential plane, which is perpendicular

* Source: Williams Thompson, Lord Kelvin: Popular lectures and addresses. 1891–1894.

to the direction of the force that produces it. Remember, the smallest complex is identified by algebraically adding the area of the complex—or part of a complex—that is above the base line to the area of the complex that is located below the base line.

This concept is used to establish the anterior or posterior direction of an electrical force within about 15° of the actual location.

Grant's methods can be used to measure the direction and size of the 8 cardiac vectors. This enables the interpreter to *measure* in degrees the normal and abnormal relationship of one electrical force with other electrical forces.

The Grant method of analysis, being more scientific than memorizing—should minimize errors and improve understanding. For example, it should be no surprise that an electrocardiogram showing the mean S-T vector located at +60° in the frontal plane and S-T segment depression in leads V₁, V₂ and V₃ plus S-T segment elevation in leads V₇ and V₈ is usually caused by obstruction in the circumflex artery. Although such a tracing could be memorized, it is not likely that all interpreters can memorize all of the exceptions they will encounter. Also, when the Grant method of analysis is used it is not necessary to record leads V₃R and V₄R searching for an elevated S-T segment due to right ventricular infarction that is caused by an obstruction in the proximal portion of the right coronary artery because these abnormal deflections can be predicted when the spatial direction of a vector representing the S-T segment is calculated using the 12 routine leads.⁷ Then too, an anterior infarct due to obstruction of the left anterior descending artery may occasionally produce S-T segment elevation in right sided precordial leads.²⁰ This will cause an error of interpretation if the physician has only memorized that an elevated S-T segment recorded in leads V₃R and V₄R always indicates a right ventricular infarct. To memorize deflections without understanding the possible distribution of the electrical fields that are created by the electrical forces is dangerous.

The diagram shown in Fig. 3 illustrates the difference in the direction of the mean spatial S-T segment vector in patients with 10 types of myocardial infarction. Four of them show "S-T segment depression," but only one of the four is associated with subendocardial injury. Three of them are caused by predominant epicardial injury. Thrombolytic therapy might not be given to patients with those three types of infarcts because an interpreter might diagnose NSTEMI. When the direction and size of electrical forces are calculated, it is not necessary to memorize the direction of the S-T segment in all of the 12 leads of the electrocardiogram.

Errors are made when the distribution of electrical fields is not understood or when the interpreter cannot remember which leads to memorize. For example, several articles have appeared in the literature pointing

out that the S-T segment is elevated in leads V₁–V₃ in patients with a right ventricular infarct suggesting the presence of an anterior infarction.^{21–23} The authors were surprised to discover that a right ventricular infarct could produce such a finding. They had memorized that such a finding indicated the presence of an anterior infarction. They did not realize that the S-T segment is usually elevated in lead I in patients with an anterior infarct, but is depressed when there is a right ventricular infarct. The authors were disassociating the abnormalities shown in the chest leads from those shown in the extremity leads. They did not realize that the same underlying electrical forces influenced all twelve of the electrodes. It is much more accurate to calculate the direction of the mean spatial S-T segment vector than it is to memorize a nonspecified number of deflections. The mean spatial S-T vector associated with a *right* ventricular infarction is commonly directed to the right about 120° or more in the frontal plane and anteriorly where the right ventricle is located.²⁴ Accordingly, the rightward direction of the mean S-T segment vector in the frontal plane projects a negative quantity on lead I, which produces S-T segment depression. On the other hand, the mean spatial S-T vector associated with an anterior infarction is usually directed to the *left* and *anteriorly*.

An Electrocardiogram that Illustrates how An Error can be Made

Many clinicians memorize the abnormalities that permit them to identify S-T segment elevated myocardial infarcts. Errors can be made using that approach to identify predominant epicardial injury. Such errors can be avoided if the interpreter uses vector concepts and basic principles to analyze the electrocardiogram.

The electrocardiogram shown in Fig. 4 might be interpreted by pattern readers as showing obvious S-T segment depression in the precordial leads and very little S-T segment displacement in the extremity leads. Having made that determination, they might discontinue any additional thought about the matter. When an interpreter plots the direction of the mean S-T segment vector, a completely different assessment can be made. The mean S-T segment vector is very small in the frontal plane but is directed about +150° to the right and is directed about 80° posteriorly. *It is directed toward epicardial injury in the inferoposterior region of the heart.* This region of the heart is served by the right coronary artery and the circumflex.

Coronary arteriography revealed 100% occlusion of the mid right coronary artery, 90% diameter occlusion of the proximal portion of the circumflex coronary artery, and 70% occlusion of the distal portion of the left anterior descending coronary artery.

FIG. 4 This electrocardiogram was recorded from a 66-year-old woman who experienced the characteristic pain of an acute myocardial infarction and developed enzymatic evidence of considerable cardiac muscle destruction.

The mean spatial QRS vector is directed at -20° in the frontal plane and about 75° posteriorly. The initial 0.04 s vector is directed to the left and slightly anterior. The mean spatial T vector is directed at about $+80^\circ$ in the frontal plane and about 60° anteriorly. The mean spatial S-T segment vector is directed at $+150^\circ$ in the frontal plane and about 75° posteriorly. This indicates infero-posterior predominant epicardial injury.

The direction of the mean spatial S-T segment vector is not directed as it usually is when it is caused by subendocardial injury. The mean spatial S-T segment vector due to subendocardial injury is usually directed between -90° and $\pm 180^\circ$ in the frontal plane and is usually about parallel with the frontal plane or directed slightly anterior. Here, in this tracing, it is directed $+150^\circ$ in the frontal plane and 75° posteriorly. An electrocardiogram recorded from the appropriate place on the back would show S-T segment elevation. The direction of the mean spatial S-T segment vector indicates epicardial injury in the infero-posterior portion of the left ventricle.

It is likely that some interpreters would classify the tracing as showing S-T segment depression and think no more. Interpreters who understand that they are searching for predominant epicardial injury and can plot the direction of the mean spatial S-T segment vector can arrive at the correct diagnosis.

Coronary arteriography revealed 100% occlusion of the mid right coronary artery, 90% occlusion of the proximal portion of the circumflex coronary artery, and 70% occlusion of the distal portion of the left anterior descending coronary artery.

The P-Q segment displacement suggests the likelihood of an atrial infarction. The patient had an extension of the infarction, developed A-V block and died. As reported by Jim *et al.*, atrial infarction in the setting of inferior-posterior infarction predicts a high mortality from arrhythmias and rupture.²⁵

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