Clin. Cardiol. 28, 219–224 (2005)

Thoughts about the Ventricular Gradient and Its Current Clinical Use (Part II of II)

J. WILLIS HURST, M.D., MACP

Emory University, Atlanta, Georgia, USA

Part I of this paper, including Figs. 1–5, appeared in the April issue of Clinical Cardiology (Clin Cardiol 2005;28:175–180).

Summary: The concept of the ventricular gradient was conceived in the mind of Frank Wilson in the early 1930s. Wilson, a mathematical genius, believed that the calculation of the ventricular gradient yielded information that was not otherwise obtainable. The method of analysis was not utilized by clinicians at large because the concept was not easy to understand and because the method used to compute the direction of the ventricular gradient was so time consuming that clinicians could not use it. Grant utilized the concept to create vector electrocardiography, but he believed that if his method of analysis was used, it was not often necessary to compute the direction of the ventricular gradient. He did, however, describe an easy way to compute the direction of the ventricular gradient. The current major clinical use of the ventricular gradient is to identify primary and secondary T-wave abnormalities in an electrocardiogram showing left or right ventricular hypertrophy or left or right ventricular conduction abnormalities. In addition, the author uses the term ventricular time gradient instead of ventricular gradient in an effort to clarify the concept. Finally, the author discusses the possible clinical significance of a normally directed, but shorter than normal, ventricular time gradient, an attribute that has not been emphasized previously.

Key words: ventricular time gradient

Address for reprints:

J. Willis Hurst, M.D., MACP
Emory University
1462 Clifton Road, N.E.
Suite 301
Atlanta, GA 30322, USA
e-mail: jhurst@emory.edu

Received: February 4, 2005
Accepted: February 4, 2005

The Mechanism for and Characteristics of the Normal and Abnormal Spatial Ventricular Time Gradient in Adult Human Subjects

Mechanism for the Production of the Normal Spatial Ventricular Time Gradient

As stated earlier, one theory that explains the endocardial time delay of the repolarization process present in the normal human heart is that the transmyocardial pressure is higher in the subendocardium of both ventricles than in the subepicardium. This delays the time that the endocardium begins to repolarize. Stated another way—the duration of the excited state is prolonged in the endocardium. Remember, when the repolarization process is directed in a certain direction it produces an electrical force that is directed in the opposite direction. This force is referred to as the mean T-wave vector. To restate for emphasis—the reason the mean spatial repolarization vector (T-wave vector) in the normal adult heart is not directed in exactly the same direction as the mean spatial QRS vector may be that the endocardial pressure that produces the repolarization delaying process may not be equally distributed throughout the ventricles. This is why a spatial QRS-T angle of 0º–60º is present in most normal adult human hearts.

The Direction of the Normal Spatial Ventricular Time Gradient

The frontal plane direction of the normal mean ventricular time gradient is located between + 20º and + 70º (Fig. 6A).[7] Its normal direction is almost parallel with the mean anatomic axis of the ventricles that may be more vertical or horizontal than suggested by Figure 6A. Accordingly, one can assume that the range of normal is 0º to + 90º (Fig. 6B).

Although it is more difficult and less accurate to determine the anteroposterior direction of the normal ventricular gradient of the normal adult subject, it is usually located about 10º to 20º anteriorly or posteriorly to the frontal plane.

The Size of the Normal Ventricular Time Gradient

The normal ventricular time gradient is usually larger than the mean spatial QRS vector; this is illustrated in Figure 7.

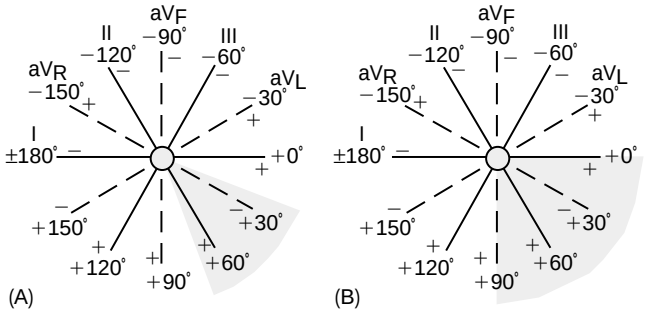


FIG. 6 (A) The shaded frontal plane area indicates the location of the frontal plane direction of the normal ventricular time gradient in adults. The direction seems to parallel the anatomic axis in most hearts. (B) Because the anatomic axis of the normal heart may occasionally be more vertical or more horizontal than shown in (A), it is acceptable to consider that the normal frontal planes direction of the ventricular time gradient is located between 0˚ and + 90˚.

Note that the direction of the mean spatial T vector is determined by the summation of the hypothetical T-wave vector and the ventricular time gradient.

When a tracing is viewed, it is useful to ask—given the direction and size of the mean spatial QRS vector that is present in an electrocardiogram—where should the direction of the actual mean spatial T vector be located to be considered normal. The answer is arrived at by determining the vector sum of the mean spatial hypothetical T-wave vector plus a vector representing the normal spatial ventricular time gradient which should be directed 0º–90º in the frontal plane, no more than 10º to 20° anteriorly or posteriorly, and is larger than the mean QRS vector.

Mechanisms Responsible for an Abnormally Directed Spatial Ventricular Time Gradient

When the spatial ventricular time gradient is directed in any direction except 0º to + 90º in the frontal plane, or when it is directed more than 20º to 30º posteriorly or anteriorly, it is likely to be due to localized nonphysiologic causes such as ischemia, fibrosis, or some other pathologic reason (Fig. 8). The abnormally directed spatial ventricular time gradient may occasionally be directed away from the centroid of the epicardial damage produced by severe generalized epicardial ischemia or severe generalized epicardial damage due to pericarditis (Fig. 9).

Mechanisms Responsible for a Normally Directed but Shorter than Usual Spatial Ventricular Time Gradient

When the spatial ventricular time gradient is normally directed between 0º and + 90º and is no more than 10º to 20º posterior, but is shorter than the mean spatial QRS vector, it is usually due to a physiologic response of left or right ventricular hypertrophy due to systolic pressure overload or uncomplicated left or right ventricular conduction system block (Fig. 10A and B and later discussion). It may rarely be due to generalized ischemia or epicardial damage due to pericarditis.

The Clinical Use of the Ventricular Time Gradient

There are only two common electrocardiographic abnormalities in which the determination of the spatial direction and size of the ventricular time gradient gives additional clinical information that is not otherwise available. They are left or right ventricular hypertrophy and left or right ventricular conduction system block.

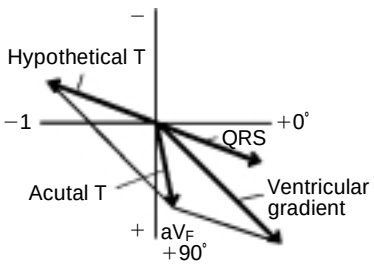


FIG. 7 This diagram illustrates how the direction of the recorded mean T vector is created. It is the vector sum of the hypothetical T-wave vector, which is equal in size to the mean QRS vector and is precisely opposite to it, and the ventricular time gradient.

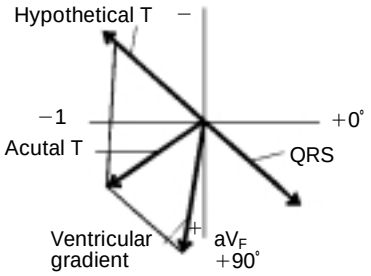


FIG. 8 This figure illustrates the frontal plane representation of a ventricular time gradient that is located outside the normal 0˚ to 90˚ area of the frontal plane display system. It indicates that the excited state of the myocardial cells is prolonged in a localized area of the superior portion of the left ventricle. Stated another way, there is an abnormally long time delay before repolarization begins in that area.

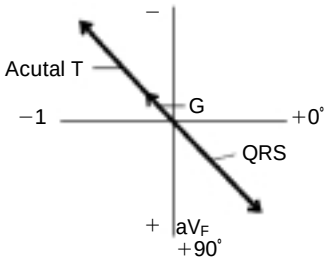


FIG. 9 This figure illustrates what may happen when there is a generalized epicardial delay in the repolarization of the ventricles. The direction of the mean T vector will be opposite to that of the mean QRS vector. When it is longer than the QRS vector, the ventricular gradient will be directed as shown above.

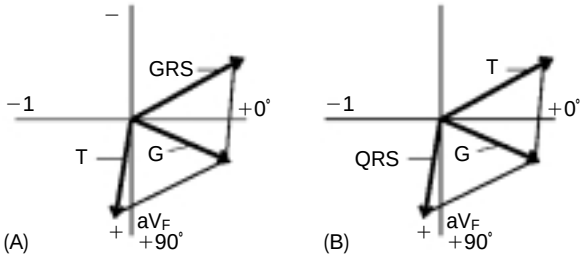


FIG. 10 (A) A normally directed, but abnormally short ventricular gradient, produced by left ventricular hypertrophy or left ventricular conduction system block. (B) A normally directed, but abnormally short ventricular gradient produced by right ventricular hypertrophy or right ventricular conduction system block.

Left Ventricular Hypertrophy

Systolic pressure overload of the left ventricle: Initially, the electrocardiogram may be normal when there is mild left ventricular hypertrophy due to systolic pressure overload of the left ventricle. The mean QRS vector is directed from −20º to +90º in the frontal plane and 30º to 50º posteriorly. The duration of the QRS complex is ≤ 0.10 s. At times, there may be only an increase in QRS amplitude as determined by a 12-lead QRS amplitude that is greater than 185 mm.[12] However, an increase in QRS voltage alone only permits an electrocardiographic diagnosis of probable left ventricular hypertrophy. When there is also a left atrial abnormality, or the appropriate S-T- and T-wave vectors, a definite diagnosis of left ventricular hypertrophy can be made. Initially, the direction and size of the ventricular time gradient may be normal. When the mean spatial T vector is directed about 130º away from the mean spatial QRS vector, the ventricular time gradient becomes shorter than the mean spatial QRS vector, but can be normally directed (Fig. 11A). The T-wave abnormality is referred to as a secondary T-wave abnormality. When the mean spatial T vector becomes 180º away from the mean spatial QRS vector, the spatial ventricular time gradient becomes zero (Fig. 11B).

The mean spatial S-T segment vector is usually directed relatively parallel with the mean spatial T vector and is also due to the forces of repolarization. It is referred to as a secondary S-T segment vector.[13, 14]

Mechanism involved: It is proper to ask why there is an ST-T-wave abnormality in the electrocardiogram of patients with left ventricular hypertrophy due to systolic pressure overload. Whereas there is no definite answer to this question, deductive reasoning suggests the following anatomic-physiologic explanation. When the left ventricular systolic pressure is sufficiently elevated and the ventricular wall has a certain thickness, the transmyocardial pressure gradient may be diminished or eliminated although the transmyocardial pressure itself may be higher than normal. This would cause a reversal of the repolarization process. The ventricular time gradient would remain normally directed although its size may become shorter. This abnormality is referred to as a secondary T-wave abnormality.

How does one determine whether or not there is an additional cause for the alteration of the direction of the mean spatial T vector? After all, a condition such as myocardial ischemia due to coronary atherosclerotic heart disease is common in patients with left ventricular hypertrophy. It is useful to plot the spatial ventricular time gradient in patients with left ventricular hypertrophy due to systolic pressure overload because the small electrocardiographic changes produced by the added abnormalities due to ischemia, or some other condition, may escape the casual inspection of the tracing. When the ventricular time gradient is directed abnormally in such electrocardiograms, it is possible to conclude that there are two abnormalities of repolarization—one due to the left ventricular hypertrophy itself and the other due to ischemia or some other condition (Fig. 12). This abnormality is referred to as a primary T-wave abnormality.

Diastolic pressure overload of the left ventricle: The electrocardiographic abnormalities due to diastolic pressure overload of the left ventricle initially are different from those occurring with systolic pressure overload of the left ventricle. The amplitude of the QRS complexes may be increased and a left atrial abnormality may be present. The direction of the mean QRS vector may be from −20º to 90º in the frontal plane and 30º to 60º posteriorly. The mean spatial S-T vector and mean spatial T-wave vector may become larger than usual, but may be normally directed. The S-T vector is due to large forces of repolarization; therefore, the mean spatial S-T vector tends

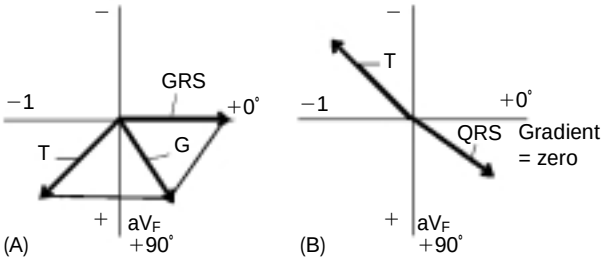


FIG. 11 (A) When the mean spatial T vector becomes more than +130˚ away from the mean QRS vector, the ventricular time gradient will be normally directed but shorter than the mean QRS vector. (B) When the mean spatial T vector is directed opposite to the mean QRS vector, the frontal plane representation of the ventricular time gradient will be zero.

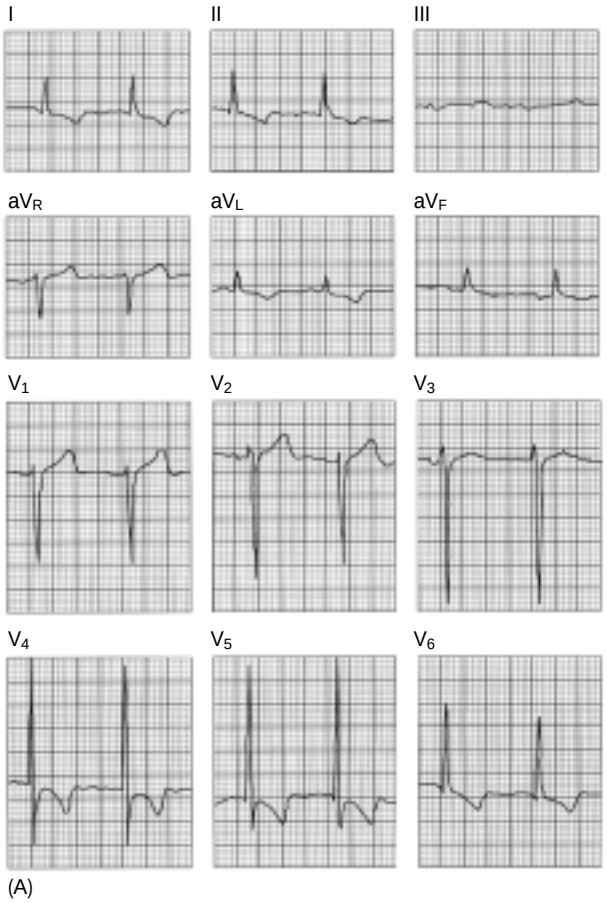
to be parallel with the large mean spatial T vector. The S-T segment vector is referred to as a secondary ST-segment change, because it is actually part of the repolarization process. The ventricular time gradient may be normally directed, and its size may be normal or greater than normal. The direction of the ventricular time gradient may later shift to be similar to the abnormalities described under systolic pressure overload.

Mechanism involved: When there is left ventricular hypertrophy due to left ventricular diastolic pressure overload, the left ventricular chamber is larger than it is when the left ventricular hypertrophy is due to systolic pressure overload. Therefore, the endocardial surface area of the left ventricle is enlarged and this, plus the fact that the systolic pressure within the left ventricle is lower than it is when there is systolic pressure overload, explains to some degree why the mean S-T and T vectors and the ventricular time gradient are initially different from the abnormalities caused by systolic pressure overload of the left ventricle.

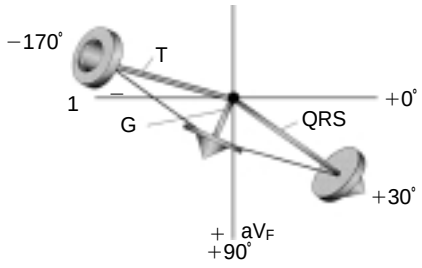
Right ventricular hypertrophy: The same concepts mentioned above are also operative when there is right ventricular hypertrophy (Fig. 10).

Additional comments: Using deductive reasoning it is possible to postulate that when the ventricular time gradient is normally directed but is smaller than normal in patients with left or right ventricular hypertrophy, a generalized alteration of the ventricles might be present. In the setting of left or right ventricular hypertrophy, it may be related to the decrease in transmyocardial pressure gradient that develops in the course of hypertrophy due to systolic pressure overload and later on in the course of diastolic pressure overload. Such an abnormality could also occur when there is generalized epicardial damage such as occurs with generalized ischemia or generalized pericarditis.

When the ventricular time gradient is directed abnormally in patients with left or right ventricular hypertrophy, there may be an additional localized condition, such as ischemia or fibro-



(A)



(B)

Source: Reproduced from Hurst JW and Woodson GC Jr: *Atlas of Spatial Vector Electrocardiography*, p. 91. New York: The Blakiston Company, Inc., 1952. Copyright J. Willis Hurst.

FIG. 12 (A) The mean P vector is difficult to plot because the p wave is difficult to see, but suggests interatrial block. The mean QRS vector is directed at + 30° in the frontal plane and about 40° posteriorly. The duration of the QRS complex is 0.08 s and its 12-lead amplitude is about 204 mm. The mean T vector is directed at about −170° in the frontal plane and about 40° anteriorly. The number of small squares subtended by the T wave in leads I and II suggests that the area under the T wave is slightly greater than the area under the QRS complexes.

The ventricular time gradient is computed as shown above. Note that it is not directed normally—it lies outside the left lower quadrant of the hexaxial display system: *This is a primary T-wave abnormality*.

This tracing shows a greater than normal 12-lead amplitude of the mean spatial QRS vector and a T-wave abnormality indicating left ventricular hypertrophy. These two abnormalities indicate systolic pressure overload of the left ventricle. The mean S-T vector in this tracing is part of repolarization because it is parallel to the mean T vector. The ventricular time gradient is directed abnormally indicating that something in addition to left ventricular hypertrophy is producing the T-wave abnormality. It is described as being a primary T-wave abnormality that would be difficult to recognize unless the ventricular time gradient was calculated.

The patient was 51 years old and had essential hypertension. The abnormal ventricular time gradient indicates that a primary T-wave abnormality is present in addition to the abnormality caused by left ventricular hypertrophy.

(B) Vectors are used to illustrate the direction and size of the mean QRS complex and mean T wave that are described narratively in the legend of Figure 12A. Note that the ventricular time gradient is directed abnormally outside the left lower quadrant of the frontal display system. This indicates that there is a primary T-wave abnormality in addition to the T-wave abnormality that is secondary to left ventricular hypertrophy.

sis, that is responsible for the T-wave abnormality. In such cases, the abnormally directed ventricular time gradient has produced a primary T-wave abnormality.

Left or Right Ventricular Conduction System Block

Left or right ventricular conduction system block is said to be present when the duration of the QRS complex is ≥ 0.12 s. A conduction defect can also be present when the QRS duration is normal, but the instantaneous spatially oriented QRS vectors are not created in the normal sequential manner.

Left Ventricular Conduction System Block

The term left ventricular conduction system block should replace the term left bundle-branch block.[15] The new term fits our current anatomic and electrophysiologic knowledge of the subject more accurately than the older term.

Uncomplicated left ventricular conduction system block: When there is uncomplicated left ventricular conduction system block, the QRS duration is 0.12 s, the mean spatial QRS vector is directed < 30º to the left and is always directed slightly posteriorly. The terminal spatially oriented 0.02 QRS vector is directed to the left and posteriorly toward the left ventricle. The mean spatially oriented T vector may be directed no more than 90º away from the mean spatial QRS vector, but is commonly directed almost 180º from that vector. When the block is uncomplicated, the T waves appear to be abnormal, but the ventricular gradient is normally directed and is shorter than normal. The T-wave abnormality is referred to as a secondary T-wave abnormality. The direction of the mean spatial S-T-segment vector is normally directed within 60º of the mean spatial T-wave vector. This S-T vector is called a secondary S-T-segment vector and is actually due to repolarization; it is part of the T wave.[13, 14]

What causes the abnormal looking T wave when there is uncomplicated left ventricular conduction system block? Most text books are silent on this matter, but using deductive reasoning one can reasonably suspect that when the S-T and T waves are produced in the left ventricle there is an alteration of the transmyocardial ventricular pressure gradient in microsegments of the myocardium. Should this occur, the repolarization process could possibly be reversed in the microsegments of the myocardium. Accordingly, when the ventricular gradient is normally directed but shorter than normal, the T-wave abnormality is caused by the altered sequence of depolarization.

Complicated left ventricular conduction system block: When there is complicated left ventricular conduction system block, one or more of the following abnormalities may be present: the QRS duration may be > 0.12 s, the mean spatial QRS vector may be directed more than −30º to the left and may be directed abnormally, posteriorly, or anteriorly, a primary T-wave abnormality is identified when the ventricular gradient is directed abnormally, and a primary spatially oriented S-T-segment vector may be directed > 60º away from mean spatial T vector, indicating that it is not part of the repolarization process.

A primary T-wave abnormality is caused by a mixture of the T-wave abnormality associated with the conduction defect, which is due to the reversal of the repolarization process throughout most of the microsegments of the left ventricle, plus an additional abnormality that is localized, such as ischemia or fibrosis. The ventricular time gradient is directed abnormally in such tracings. When the condition is not obvious by simple inspection of the tracing, it is useful to plot the ventricular gradient (Fig. 12).

Right ventricular conduction system block: When there is uncomplicated right ventricular conduction system block, the QRS duration is 0.12 s, the mean QRS vector is directed < + 120º to the right and anteriorly, the initial 0.02 QRS vector is normally directed, the terminal 0.02 s QRS vector is directed to the right and anteriorly where the right ventricle is located, and the QRS-T angle approaches 180º. There is a secondary T-wave vector and a secondary S-T-segment vector, and the ventricular gradient is normally directed, but shorter than normal.

Complicated right ventricular conduction system block: Complicated right ventricular conduction block is said to be present when the duration of the QRS complex is > 0.12 s, or the mean spatial QRS vector is directed > + 120º to the right in the frontal plane, or when the mean spatial initial 0.02–0.04 s QRS vector is directed abnormally due to infarction, or when the mean spatial S-T-segment vector is directed ≥ 60º from the mean T vector, or when there is a primary T-wave abnormality identified by an abnormally directed ventricular time gradient (Fig. 13).

The mechanisms responsible for these electrocardiographic findings are conceptually similar to those discussed under left ventricular conduction system block.

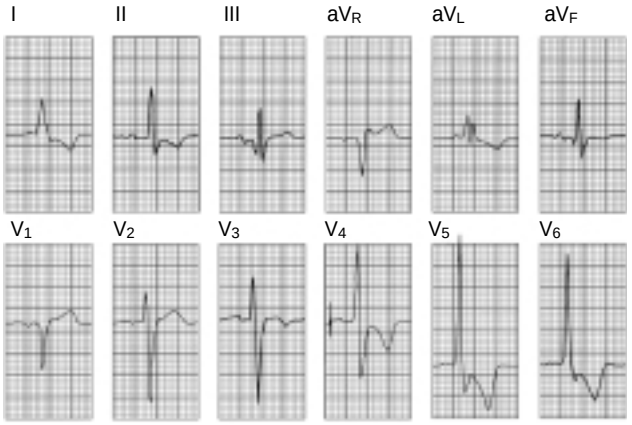
Conclusion

The concept of the ventricular time gradient is 70 years old. Following the teaching of Frank Wilson,[1] several investigators and clinicians pointed out the importance of understanding the concept, but failed to convince clinicians that its actual measurement was of great clinical value.

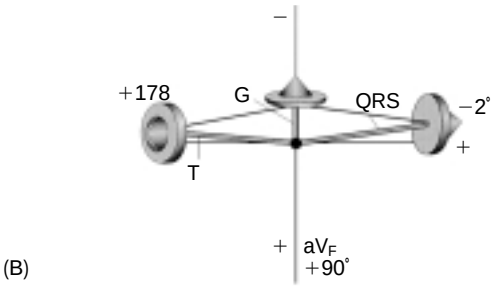
In retrospect, it is easy to discern the two major reasons why the determination of the ventricular time gradient remained the tool of only a few experts. First of all, the concept is not easy to grasp, and second the methods used to calculate its direction and size were too time consuming to be used by busy clinicians.

The use of vector concepts, as described by Grant,[9] to interpret electrocardiograms makes it possible to determine the direction and size of the spatial electrical forces that create the deflections in electrocardiograms.[9] Accordingly, it is relatively easy to determine the direction and size of the mean spatial QRS vector, the mean spatial T vector; and the ventricular time gradient. Accordingly, it seems wise to look once again at the concept that Wilson believed would create a new and better way of interpreting certain aspects of electrocardiograms.

Grant taught us that the identification of the spatial QRS–T angle contains most of the information obtained by calculating



(A)



(B)

FIG. 13 (A) The mean P vector is directed normally. The duration of the QRS complex is 0.14 s in lead I indicating complicated left ventricular conduction system block. The mean QRS vector is directed at about −2° in the frontal plane and is about 40° posterior. The mean T vector is directed at about −178° in the frontal plane and about 30° anteriorly. The amplitude of the mean QRS is larger than usual, but this can occur in patients with ventricular conduction defects and does not always indicate hypertrophy. The area subtended by the QRS complexes is about the same as the area subtended by the T waves. *Note that the ventricular time gradient is directed abnormally, signifying that a primary T-wave abnormality is present in addition to that produced by the conduction defect.* The mean ST-segment vector is directed parallel to the mean T vector, which indicates a secondary S-T-segment abnormality. The patient may well have an area of segmental ischemia of the left ventricle in addition to the T-wave abnormality caused by the conduction defect. Such an abnormality would be difficult to identify without calculating the ventricular time gradient.

This tracing was recorded on an 82-year-old man with triple-vessel coronary atherosclerotic heart disease. He had multiple myocardial infarcts, a large heart, and heart failure.

(B) Vectors are used to illustrate the direction and size of the mean QRS complex and mean T wave that are described narratively in the legend of Figure 13A. Note that the ventricular time gradient is directed abnormally outside the left lower quadrant of the frontal display system. This indicates that there is a primary T-wave abnormality in addition to the T wave abnormality that is secondary to left ventricular conduction system block.

the ventricular time gradient. There are two times, however, when it is sometimes useful to determine the direction and size of the ventricular time gradient in the electrocardiograms of some patients with left or right ventricular hypertrophy and in some patients with left or right ventricular conduction system block. This is true because the interpreter must be able to determine whether the T-wave abnormality is a secondary or a primary T-wave abnormality.

In addition, it is reasonable to believe that the ventricular time gradient, which is directed away from the myocardium where repolarization is delayed, is a more accurate indicator of the location of an abnormal time delay of myocardial repolarization than the direction of the mean T-wave vector.

Finally, in this manuscript, our emphasis has also been placed on the possible meaning of a short but normally directed ventricular time gradient.

Acknowledgment

The author recognizes that this discussion of the ventricular time gradient is not based on scientific information; rather, as is the case for many aspects of electrocardiography, the concept is created by deductive reasoning. However, the concept and practical value of the ventricular time gradient must be understood by those who interpret electrocardiograms and those who are struggling to create improved software for computer interpretation.

References (Parts I and II)

1. Wilson PF, MacLeod AG, Barker PS, Johnston FD: The determination and the significance of the areas of the ventricular deflections of the electrocardiogram. *Am Heart J* 1934;10:46
2. Bayley RH, Monte LA: Acute local ventricular ischemia, or impending infarction, caused by dissecting aneurysm: Case report with necropsy. *Am Heart J* 1943;25:262–270.
3. Ashman R, Byer E: The normal human ventricular gradient. I. Factors which affect its direction and its relation to the mean QRS axis. *Am Heart J* 1943;25:16–35
4. Ashman R, Byer E: The normal human ventricular gradient. II. Factors which affect its manifest area and its relationship to the manifest area of the QRS complex. *Am Heart J* 1943;25:36–57
5. Ashman R, Gardberg M, Byer E: The normal human ventricular gradient. III. The relation between the anatomic and electrical axes. *Am Heart J* 1943;25:473–494
6. Ashman R: The normal human ventricle gradient. IV. The relationship between the magnitudes, A_{QRS} and G, and deviations of the RS-T segment. *Am Heart J* 1943;25:495–510
7. Burch G, Winsor T: *A Primer of Electrocardiography*, pp. 179–189. Philadelphia: Lea & Febiger, 1945
8. Barker JM: *The Unipolar Electrocardiogram: A Clinical Interpretation*, p. 578. New York: Appleton-Century-Crofts, Inc., 1952
9. Grant RP: *Clinical Electrocardiography: The Spatial Vector Approach*, pp. 1–225. New York: The Blakiston Division, McGraw-Hill Book Company, Inc., 1957 (a) pp. 132–134; (b) pp. 71–72
10. Burch G, Winsor T: *A Primer of Electrocardiography*, pp. 23–31; (a) p. 181. Philadelphia: Lea & Febiger, 1945
11. Grant RP: *Clinical Electrocardiography: The Spatial Vector Approach*, p. 69; (a) p. 31. New York: The Blakiston Division, McGraw-Hill Book Company, Inc., 1957
12. Odum H II, Davis JL, Dinh H, Baker BJ, Roberts WC, Murphy ML: QRS voltage measurements in autopsied men free of cardiopulmonary disease: A basis for evaluating total QRS voltage as an index of left ventricular hypertrophy. *Am J Cardiol* 1986;58:801–804
13. Hurst JW: Abnormalities of the S-T segment—Part I. *Clin Cardiol* 1997; 20:511–520
14. Hurst JW: Abnormalities of the S-T segment—Part II. *Clin Cardiol* 1997; 20:595–600
15. Hurst JW: A new terminology for describing left and right ventricular conduction abnormalities. *Clin Cardiol* 2003;26:540–545