Spatial Vector Electrocardiography
The Clinical Characteristics of S-T and T Vectors

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The application of a spatial vector method of interpretation to the S-T and T deflections in the conventional electrocardiogram is described, and the vector abnormalities responsible for the more commonly encountered deformities are discussed. In addition, the isolated T-negativity syndrome is described, a rare type of precordial T-wave inversion which may be encountered in normal subjects and is often indistinguishable from the ST-T deformity of recent myocardial infarction. The spatial vector method of interpretation is found to provide a simple yet rational basis for the interpretation of these often bizarre wave forms.

In the past, the clinical interpretation of the S-T segment and the T wave in the electrocardiogram has been based upon the size and direction of the deflections in individual leads. With the development of multiple precordial leads and unipolar limb leads, this method of interpreting ST-T contours has become more and more difficult and arbitrary. Recently it has been shown that, in the human subject, all body surface deflections can be treated as, in effect, recordings from the same central resultant electrical forces or vectors. This means that, by and large, the deflections are different on the various leads in a given patient because each lead records a different projection of essentially the same vectors. It is possible to determine the characteristics of these central resultant vectors from the deflections on the conventional leads and the interpretation of the electrocardiogram can be based upon these data. Such a method for interpretation simplifies clinical electrocardiography because it makes it possible to express the many different contours and amplitudes of QRS and T deflection on the various leads in a given patient in terms of the spatial directions of the responsible vectors. In addition, it is a more objective and rational method of interpretation than methods based upon deflection “patterns” in individual leads.

In a previous paper, a method was described for determining the direction in three dimensional space of instantaneous and mean vectors from simple inspection of conventional limb and precordial leads, and the characteristics of normal and abnormal spatial QRS vectors were presented. In the present paper the S-T and T vectors will be discussed with particular reference to the way in which precordial ST-T abnormalities are produced. The material will be presented descriptively with little discussion of the bioelectric abnormalities responsible for these deformities. However, the spatial vector method has made it possible to apply objective and semiquantitative technics to the study of the mechanisms of S-T and T abnormalities in the human subject, and this material will be presented elsewhere.

Material and Methods

The cases were obtained from 3000 consecutive clinical electrocardiograms taken at the Grady Memorial Hospital. In all cases, the three standard limb leads and six precordial V leads had been taken. In many subjects unaugmented unipolar extremity leads were also available. In certain subjects the projection of T forces on the chest surface was studied by recording unipolar V leads at two inch intervals along a series of vertical lines from above the clavicles to the umbilicus.

The method for study was the spatial vector method described in detail previously. In brief, the frontal plane projection of the mean spatial vector is determined from the three limb leads on a triaxial reference figure. Then, the spatial direction of this mean vector is determined by identifying the precordial electrode position where a transitional deflection was recorded from that vector. These two

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Aided by a grant from the Life Insurance Medical Research Fund.
data make it possible to plot the transitional pathway for the vector, which is the line around the chest where a plane perpendicular to the vector would intersect the body surface. Plotting the transitional pathway makes graphic the direction of the responsible vector and indicates the distribution of positive (upright) and negative (inverted) deflections on the chest for that vector.

The S-T and T contours are recordings of the variations in magnitude and direction of resultant vectors from one instant to another during a single ST-T cycle. When these instantaneous vectors are translated to the point of relative zero in a drawing of the electrical field of the heart, they define an ellipse which is called the spatial ST-T loop or vectorcardiogram. The method for constructing the ST-T loop from conventional leads is the same as that for calculating the QRS loop and has been described previously.

It will be recognized that this method for studying the electrical field of the heart treats the body as if it were a regular cylindrical volume conductor of relatively homogeneous conductive properties with the heart at its center. Accordingly, variations in the contour of the chest and in the eccentric position of the heart as well as variations of the placement of the precordial electrode will be sources of error in the calculations of spatial vectors by this method. Such errors are, however, rarely large enough to significantly influence the interpretation or impair the validity of the method for clinical purposes. It must be remembered that body surface deflections give at best only a vague and general notion of the electrical forces of the heart, no matter how accurate the recording instrument, or how precise or elaborate the method for interpreting them.

Space will not permit a discussion of secondary T wave abnormalities. These are the T wave changes which accompany ventricular conduction disturbances and can be expected whenever the QRS interval exceeds 0.12 to 0.14 second. However, vector methods are applicable to these disorders, and, indeed, by the spatial vector method of interpretation it is possible to detect primary T-wave abnormalities when they are superimposed on the secondary T-wave changes.

In the illustrations, the three standard limb leads and precordial leads V₁ to V₆ are shown. Below the tracings, frontal and coronal plane ST-T loops and mean QRS vectors are drawn; and the mean spatial QRS, S-T and T vectors are shown three-dimensionally with drawings of the transitional pathways on the chest for the mean spatial S-T and T vectors for each subject. To be sure, the coronal plane loop is not entirely accurate in these drawings because the V₁ to V₆ electrode positions from which the coronal projection of the loop is constructed do not all lie in the same coronal plane of the body. Furthermore, in order to make the illustrations as simple and legible as possible, complete accuracy in drawing the loop and plotting the directions of the spatial vectors had to be sacrificed in certain instances. However, these inaccuracies are slight and do not interfere with the general validity of the reconstructions. Finally, in some illustrations, the QRS complexes were retouched, but in no instances were ST-T waves retouched.

**Results**

From a descriptive point of view, three vector abnormalities account for nearly all the variations in ST-T contours which are encountered in limb and precordial leads. They are (1) abnormalities in the direction of the mean spatial T vector, accounting for variations in the distribution of positive, negative, and transitional T waves among the various leads, (2) abnormalities in the contour of the ST-T loop, accounting for abnormal S-T segments in these leads, and (3) apparently extracardiac factors which locally affect the propagation of T forces, as is seen in what is called the isolated T-negativity syndrome. They will be discussed in that order.

1. **The Direction of the Mean Spatial T Vector**

In a given subject, the mean spatial T vector in effect divides the body surface in half, as far as the type of T wave recorded from various regions of the body surface is concerned. One half, the half toward which the vector is pointing, is an area of resultant electrical positivity, and unipolar electrodes placed anywhere on this region will record positive or upright T waves. The half of the body surface away from which the vector is pointing is the area of resultant electrical negativity, and negative or inverted T waves are recorded by unipolar electrodes placed in this region. The two areas are separated by the transitional pathway which is the line of intersection on the body surface of a plane perpendicular to the vector. Electrodes placed on the transitional pathway will record isoelectric T waves, or T waves with as much positivity as negativity. Accordingly, the most rational way to interpret the T waves recorded in the various leads of a given electrocardiogram is to determine from them the direction of the mean spatial T vector for that subject and base the interpretation on this information.
The simplest of rational methods for defining normal and abnormal directions of the mean spatial T vector is to evaluate the angle it makes with the mean spatial QRS vector in the given subject, and compare this with established criteria of normal and abnormal QRS-T angles. There are several reasons for using this as the basis of the clinical interpretation. First, it is easy to do, requiring no special equipment. With a little experience, the spatial QRS-T angle can be calculated from inspection of the limb and precordial leads in no more time than it takes to interpret the tracing by any other method. Second, the direction of the mean spatial QRS vector is altered by relatively few intracardiac conditions while, as will be seen, the direction of the mean spatial T vector is influenced by a host of different subtle physiologic and pathologic conditions. Accordingly, the direction of the QRS vector provides a relatively stable reference datum for the evaluation of the direction of the T vector. Third, as Wilson and his colleagues have shown, there is a fundamental electrical relationship between the manifest QRS forces and manifest T forces in a given individual and this relationship when expressed quantitatively is called the ventricular gradient for that subject. The abnormal electrocardiogram represents a disturbance in this relationship, and the location and intensity of the disturbance is manifested by a relatively specific change in the magnitude and direction of the T vector as compared with the QRS vector. Accordingly, the QRS-T angle is a property of the ventricular gradient and its use brings the rational and objective aspects of the ventricular gradient concept to routine clinical interpretation. Fourth, a change in the location of the heart in the chest will alter QRS and T deflections in all leads; however, it will not influence the QRS-T angle, provided the myocardium has remained unchanged. This is useful in the interpretation of follow-up tracings because it helps to differentiate changes in the QRS and T waves which are due to alterations in the position of the heart in the chest from those which are due to intrinsic myocardial abnormalities.

In the vast majority of normal subjects, the mean spatial QRS vector tends to be directed leftward, inferiorly, and somewhat posteriorly to the frontal plane of the body. In young subjects it tends to point relatively vertically, while in older subjects it tends to have a more horizontal direction. Normally the mean spatial T vector tends to be relatively parallel with the mean spatial QRS vector. Usually the spatial QRS-T angle is less than 40° and only infrequently does it exceed 50° in normal subjects. Its precise relationship to the QRS vector varies somewhat with age and body build. In young subjects, it is directed somewhat posteriorly to the frontal plane and the transitional T wave is therefore commonly encountered at electrode position V3 or V4. Occasionally this juvenile pattern persists into the third decade. In older age groups, the T vector gradually rotates anteriorly and rightward so that V2 or V1 or even electrode positions to the right of V1 write the transitional T wave. These variations are illustrated in figure 1D.

The variations in the direction of the T vector with age are no doubt related to hematic and structural differences between right and left ventricle in these age groups. The T process takes place during ventricular systole, and the magnitude and direction of the mean T force therefore varies with the systolic sizes, wall thicknesses and intramyocardial pressures of the two ventricles. In young subjects, the right ventricle is relatively dominant and the T vector tends therefore to point somewhat away from the location of the right ventricle in the chest. In adults, on the other hand, the left ventricle is relatively dominant and the T vector tends to point more or less away from its location in the chest.

In older age groups another process appears which is believed to alter repolarization. This is myocardial ischemia, secondary to the coronary vascular changes which occur in nearly all older subjects. Myocardial ischemia delays repolarization, causing the T vector to point away from the affected region. Since in general these coronary vascular changes affect the left ventricular myocardium more than the right, the T vector tends to point away from the location of the left ventricle in the chest in these subjects. The QRS vector is relatively unaltered by ischemia and accordingly QRS-T
angles over 60° are not uncommon in this older age group with little or no other evidence of myocardial disease. The wide QRS-T angle of left ventricular ischemia is probably the commonest abnormality of the ventricular complexes encountered in clinical electrocardiography. It is well to remember, however, that the electrical evidence of ischemia precedes the actual histologic evidence of myocardial damage, and the clinical implications of this electrocardiographic abnormality in a given case therefore will usually depend upon other clinical findings.

In the electrocardiographic syndrome of left ventricular ischemia, the T vector usually comes to be more and more anteriorly directed, pointing away from the left ventricle. Accordingly, more and more of the anterior chest the heart. When the heart is in intermediate electrical position, as indicated by the direction of the QRS vector, the T vector usually rotates anteriorly and rightward producing a low or inverted T1 while T2 and T3 remain upright (fig. 1A). When the heart is in horizontal electrical position, the T vector tends to rotate nearly directly anteriorly, causing the frontal plane projection of the T vector to become smaller without change in direction so that the

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**Fig. 1.** A, B, C: The changes in direction of the mean spatial T vector as left ventricular ischemia develops. Three electrical positions of the heart are shown: A, horizontal, B, leftward, and C, vertical.

D: The distribution of positive and negative T waves on the body surface at various age groups. The black band indicates the distribution of transitional deflections for the given T-vector direction, and the black dots represent the standard precordial lead electrode positions.

E, F: The relationship between the effective location of myocardial electrical "injury" and the direction of the S-T vector.

G: The relationship between the directions of the S-T and T vectors and the effective location of a myocardial infarction.
limb lead T waves become small in amplitude but remain upright (fig. 1B). When the heart is in a vertical electrical position, the T vector tends to rotate anteriorly and slightly leftward, causing inversion of T2 and T3 while T1 remains upright (fig. 1C).

As far as the T loop in the normal subject is concerned, cathode tube vectorcardiograms show it to be a nearly straight line; that is, the various instantaneous vectors which write the T wave are relatively parallel with one another in a given normal subject. However, because the instantaneous vectors are not entirely parallel with one another, their difference in direction will cause some to project small positive components and others to project small negative components on the axis of a lead taken in the transitional pathway. This is the explanation for the bizarre, multiphasic T waves frequently recorded from the transitional pathway in normal subjects. The deflections at V2 and V3 in figure 2A, obtained from a subject with no evidence of heart disease, illustrate this. Here, the instantaneous T vectors are directed first anteriorly, then posteriorly, then anteriorly to the vector which is perpendicular to the transitional plane. That the variations in direction of instantaneous T vectors responsible for the bizarre T waves are actually very slight can be seen in the coronal plane drawing of the ST-T loop for this subject. Accordingly, with present knowledge of T forces, these are normal ST-T waves.

The reason for the different directions of the instantaneous T vectors in the normal subject is not known. The variation in direction is extremely slight, for the bizarre transitional T waves are recorded from only a narrow region in the transitional pathway. Indeed, in many subjects this region is so narrow that it passes between two precordial electrode positions and the multiphasic wave forms are not seen in the routine tracing. However, careful exploration of the transitional region in such subjects will usually demonstrate the multiphasic complexes. Occasionally, this pathway runs through the electrode position of one or another limb lead in which case the bizarre T wave will be recorded in the limb leads, as in leads II and III of figure 2B. The bizarre contour is not usually as striking in these leads, however, because the amplitude of the various components of the electrocardiogram is much smaller in more remote leads.

As the ST-T loop becomes wider, the pathway of multiphasic transitional complexes forms a broader band around the chest so that more and more precordial electrode positions are included in the transitional pathway. Such loops are abnormal and will be discussed in greater detail in the next section. However, it is important to recognize that the occurrence of the bizarre complex at more than one precordial electrode position may be due to the unusual direction of the mean spatial T vector and not to an abnormal loop. For example, when the T vector has such a direction that its transitional pathway runs through two or more precordial electrode positions, each of these leads may show multiphasic T waves. This is exemplified in figure 2C, obtained from a normal subject. Here the transitional pathway runs through V5, V6, and V1 electrode positions causing three of the six V leads to record bizarre transitional complexes, although, as can be seen in the drawings, the difference in direction of successive vectors is quite slight and the ST-T loop cannot be differentiated from the normal.

When the mean spatial T vector has an abnormal direction, the transitional T wave is, of course, recorded at different precordial electrode positions. For example, with left ventricular ischemia, the bizarre transitional T waves may be recorded at V6 or V5 instead of V1 or V2 as in the normal subject.

It is important to realize that when the transitional T pathway runs near or through one or another precordial electrode position, slight differences in the placement of that electrode in follow-up tracings will result in definite changes in the contour of the T wave it records. In left ventricular ischemia, for example, a difference in the location of the V6 electrode of only a few centimeters may change it from lying in the area of relative positivity and writing an erect T wave to lying in the area of relative negativity where an inverted T wave will be written. Similarly, slight variations in the position of the heart in the chest or differences in the re-
This figure illustrates how minor variations in the direction of successive instantaneous ST-T vectors account for bizarre precordial ST-T segments, discussed more fully in the text. Frontal and coronal plane projections of the ST-T loop, transitional pathways, and spatial directions of mean vectors for portions of the loop are shown.

cumbent position of the patient in serial tracings will change the location of the transitional pathway on the chest so that the T waves recorded from a region of the chest near this pathway may be positive one time and negative the next.
There is only one other commonly encountered cause of abnormal direction of the T vector in addition to ischemia. This is ventricular hypertension, causing what is perhaps inappropriately called the ventricular strain syndrome. From an electrocardiographic point of view it is probably justified to distinguish between the T vector abnormalities of “ischemia” and “strain.” In the first place, there is reason to believe that the bioelectric disturbance responsible for each is different; that is, while the ischemia abnormality can apparently be correlated with actual vascular insufficiency in most instances, the strain abnormality appears to be in large measure due to a direct effect on excitation of increased myocardial membrane tension. In the second place, the ischemia abnormality frequently represents a relatively localized disturbance of repolarization in the left ventricle with the T vector pointing away from the affected region; accordingly, the QRS-T angle is not commonly over 120°. In the strain syndrome, on the other hand, the T vector abnormality appears to be a result of altered repolarization throughout an hypertrophied ventricle. Accordingly, in addition to the QRS vector being increased in magnitude and altered in direction, the T vector is nearly completely opposite to it in direction; that is, the QRS-T angle is from 150° to 180° (fig. 3C).

These two processes, ischemia and strain, appear to account for nearly all abnormalities in the direction of the T vector clinically. It is nearly certain, however, that with further study of the mechanisms of T vector abnormalities, many abnormalities at present indistinguishable from ischemia effects and currently so designated will be found to be due to other subtle disorders. Similarly, intramyocardial pressure variations analogous to those seen in the strain syndrome but not as severe and not accompanied by hypertrophy can cause transient abnormalities of T vectors, and are no doubt responsible for many of the so-called functional abnormalities of T waves. Accordingly, it is perhaps wisest to consider the electrocardiographic diagnoses of “ischemia” and “strain” as strictly electrical terms, and their clinical implications in a given case as dependent upon other clinical data.

2. The S-T Vector and Abnormalities of the ST-T Loop

The portion of the electrical cycle of the heart which follows the QRS complex is made up of instantaneous resultant spatial vectors which are believed to represent, with extremely rare exceptions, solely electrical forces of repolarization. In the normal subject, there is relative quiescence of this process in the immediately post-QRS interval. The electrocardiogram shows little deviation during this period and it is descriptively called the S-T interval. Later in the post-QRS period, the repolarization forces swell in magnitude, writing what is descriptively called that T wave. Frequently, however, repolarization forces of measurable magnitude appear during the S-T interval and they may have different directions from the forces during the T interval. Accordingly, it is convenient to treat the electrical events in the post-QRS period in terms of a mean spatial T vector and a mean spatial S-T vector. In this sense, the mean S-T vector is the vector sum of the forces preceding the T wave, whether or not there is also a shift of J. Although this may one day prove to be an oversimplification of post-QRS forces, at the present time the vast majority of abnormalities of the ST-T segment encountered in clinical electrocardiography can be accounted for by variations in the directions of these two mean spatial vectors.

When the ST-T loop is abnormal in contour, the transition from the direction of the vectors during the S-T interval to their direction during the T interval tends to be gradual and orderly. This is important in clinical electrocardiography because, with a wide loop, the peak of the T wave will not be written at the same time after the onset of QRS complex in all leads. The reason for this is that the portion of the ST-T loop which accounts for the maximum projection of ST-T vectors on the axis of one lead is not the same portion which accounts for the maximal projection on the axis of another lead, and the two portions represent
different instants in the time course of the ST-T loop. This explains why the Q-T time may lead, late on another, and be bowed or curved on a third. In figure 2D, for example, what

be different on various leads in a given subject, and why the S-T segment may show its maximum deviation early in the S-T interval on one would be called T wave in V1 coincides in time with the last part of the S-T segment in V6, and the bizarre, deep negative portion of the T

Fig. 3. Similar to figure 2. See text for discussion.
wave in $V_2$ coincides with an isoelectric interval between ST-T forces and what would be called a U wave on leads II and III of the same subject. Obviously, to designate an S-T segment, T wave or U wave in the individual leads in this case would be difficult and arbitrary. However, the electrical abnormality responsible for these contours can be easily understood when the ST-T loop is drawn, and the interpretation can be based upon the mean directions of S-T, T, and U vectors.

Since the S-T period is normally a period during which the repolarization forces are gradually increasing in magnitude it should not be surprising that occasionally normal subjects will show significant deviations of S-T segments simply because of an over-all increase in the magnitude of repolarization vectors. Indeed, S-T displacement due to normal repolarization during the S-T interval is regularly seen at $V_2$ and $V_3$ in normal subjects because the proximity of these electrodes to the heart results in a relative magnification of all cardiac electrical forces in the recorded deflection. However, in young adults the repolarization vectors are often unusually large in magnitude, producing large T waves and measurable S-T segment deviation in the limb as well as the precordial leads. Under these circumstances the spatial S-T vector is relatively parallel with the spatial T vector. Figure 3A illustrates such a case. This was obtained from a young student nurse who had no evidence of cardiovascular disease. The striking elevation of the S-T segment at $V_3$ to $V_6$ persisted for over a year without change.

The cause of this increase in magnitude of normally directed repolarization vectors in the young adult is not known. However, because the mean T vector is a resultant of repolarization forces from all regions of the ventricular myocardium, in these cases there must be an alteration in repolarization of roughly the same intensity and duration of all regions of the heart. This would suggest that systemic or general metabolic factors are responsible rather than local myocardial or hemodynamic factors. Indeed a change in the magnitude of the T vector without a change in its direction may be seen in a host of systemic physiologic and pathologic conditions: after meals, during exercise, during sleep, in the presence of anemia, anoxia, acidosis, in hyperthyroidism and hypothyroidism, beri-beri, hyperpotassemia and other conditions. On the other hand, when the repolarization processes are altered asymmetrically in the heart, the mean spatial T vector is changed in direction, tending to point away from the affected region. In the vast majority of clinically manifest forms of heart disease there is an asymmetric hemodynamic or structural alteration in the heart, and the T vector therefore becomes abnormally directed, with or without a change in its magnitude. Indeed, it was largely because there is a change in direction of the T vector in most clinical heart diseases that the electrocardiogram came to have its great clinical differential diagnostic value.

Occasionally, the S-T vector due to normal early repolarization forces is difficult to distinguish from the S-T vector due to acute pericarditis. The reason for this is that in pericarditis the cell injury results in a dissipation of repolarization forces in such a way that S-T segment deviations appear, representing an S-T vector which points toward the center of the injured region. In pericarditis the center of the injured region is usually the apex of the heart. Accordingly, the S-T vector in this disorder has a direction which is relatively parallel with the normal T vector, resembling the situation in the subject with normal early repolarization. Often not until pericardial ischemic changes appear, causing the T vector to have an abnormal direction, can the two causes of S-T deviation be differentiated. Of additional help in differentiating them is the fact that the S-T vector is transient in pericarditis, lasting only a week or so, and the T vector is not increased in magnitude. On the other hand, in the subject with normal S-T deviation, the S-T vector persists for years and the T vector is always unusually large in magnitude.

When the spatial T vector is abnormal in direction and increased in magnitude, there will be an S-T vector due to early repolarization forces which is relatively parallel with this abnormally directed T vector. In general, such an S-T vector is not seen in the electrocardio-
gram of uncomplicated ventricular ischemia but is characteristically seen in the ventricular strain syndrome (fig. 3C). In these latter cases, the S-T vector usually is not perfectly parallel with the T vector and therefore the S-T and T transitional pathways do not coincide on the chest surface. Because of this, there are usually small regions of the chest where S-T elevation with T inversion or vice versa may be recorded while elsewhere the S-T and T deviations are concordant. In the individual tracings, the discordant patterns may sometimes closely resemble the ST-T deformity of myocardial infarction, as can be seen at V1 in figure 3D. This tracing was obtained from a 43 year old man with aortic insufficiency due to rheumatic fever, but with no clinical evidence of coronary artery disease; the tracing showed no change during 18 months of observation. When vector methods are used there is rarely any difficulty in distinguishing the two causes of S-T and T discordancy, regardless of what an individual deflection looks like, for in myocardial infarction the S-T and T vectors are nearly completely opposite in direction (fig. 1G), while in the strain pattern they are relatively parallel with one another.

Most abnormal ST-T wave forms, and therefore most abnormal ST-T loops, can be represented by differences in the directions of just two vectors, a mean spatial S-T vector and a mean spatial T vector. When studied in this light, it is found that two clinical circumstances account for the great majority of instances where the two vectors have significantly different directions. These are (1) digitalis administration, and (2) injury current. Among less common causes of differences in the directions of the two vectors are marked tachycardia and hyperkalemia, and these are usually easily rec-
ognized on other grounds. Undoubtedly, with further research, other factors which influence the directions of forces during the ST-T interval will come to light and require modification of these generalizations. In the present discussion the electrocardiographic effects of digitalis administration and injury current will be considered.

In brief, following digitalis administration, the entire repolarization process is accelerated so that the majority of repolarization vectors are generated during the S-T interval and the Q-T interval is measurably shortened. In addition, there is an alteration in fiber energetics during systole in both the normal and abnormal subject which causes the accelerated repolarization forces to be different in direction from those generated during the T interval. Accordingly, following digitalis administration, the mean S-T vector comes to be directed nearly 180° from the direction of the mean QRS vector, while the mean T vector remains unchanged in direction, gradually dwindling in magnitude as the S-T vector becomes larger and larger at each stage of digitalization. This is illustrated in figure 3B, obtained from a normal subject after digitalization. It will be noted that the QRS-T angle is normal and the S-T vector is about 160° from the QRS vector in space.

The fact that the T vector is unchanged in direction following digitalis administration, other factors remaining the same, is of importance in clinical electrocardiography, for it means that an abnormal T vector can be recognized whether or not digitalis effects are present in the electrocardiogram. Thus, for example, the wide QRS-T angle and rightward and anteriorly directed T vector of left ventricular ischemia can be recognized even when digitalis effects are present as manifested by an S-T vector relatively opposite in direction to the QRS vector.

When digitalis is given in the presence of the ventricular strain syndrome, the Q-T interval is shortened without change in the ST-T contours, presumably because the repolarization forces are already rotated nearly 180 degrees from the direction of the mean QRS vector (fig. 3C). Accordingly, digitalis effects cannot usually be detected in the presence of the strain pattern. When amounts of digitalis are given which are too small to produce characteristic electrocardiographic alterations slight hemodynamic alteration in the heart may result in the pattern of complete digitalization. For example, simple breath-holding in full inspiration in a subject who has received digitalis will cause an S-T vector to appear, opposite in direction from the mean QRS vector, and will cause the T vector nearly to vanish. For this reason it is difficult to evaluate the degree of digitalization in a given subject from the degree of electrocardiographic change. The mechanism whereby digitalis brings about these electrocardiographic changes and their relationship to the mechanical properties of the heart will be discussed in greater detail in a later paper.

The second cause of abnormally directed S-T forces is "injury current," which represents a steady-state electrical force resulting from actual cell membrane injury. Without going into details of its mechanism, the injury current acts as an electrical "leak" during the period that the myocardium is polarized and is effectively absent when the myocardium is depolarized. Accordingly, it produces an apparent S-T segment deviation which can conveniently be represented by an S-T vector. The magnitude of the S-T vector depends upon the size of the injured region and the intensity of injury, and its direction depends upon the location of the injured region of the myocardium. Thus, when the injury is in epicardial layers, as in pericarditis and classical myocardial infarction, the S-T vector tends to point toward the affected region of the ventricle (fig. 1E). When the injury is more endocardial in location, as in angina pectoris, coronary insufficiency, and subendocardial infarction, the S-T vector in general points away from the affected region (fig. 1F).

When an "injured" epicardial region is also ischemic, as is often the case, the ischemia causes the T vector to point away from this region, as was described earlier. Under this circumstance, the S-T and T vectors are oppositely directed, and this explains the discordant S-T and T contours seen in myocardial infarc-
Fig. 5. Illustrating other aspects of isolated T negativity.
tion and the “coved” of the ST-T segment during recovery (fig. 1G). However, in the very early stages of myocardial infarction, the S-T and T vectors are often relatively parallel, both greatly increased in magnitude and both pointing toward the infarcted region. Perhaps this “against the rule” behavior of the T vector is due to an intense subendocardial ischemia at the onset of the infarction, for this would cause the T vector to point toward the affected region.

3. Isolated T Negativity

In the course of the survey of 3000 consecutive clinical electrocardiograms using vector methods, 11 instances were encountered where the T waves at V₄ and V₅ were not consistent with the spatial concept of the electrical forces of the heart. In these 11 cases, the direction of the mean spatial T vector was such that the left anterior chest was an area of relative electrical positivity, and positive T waves should have been recorded from V₁ to V₅. Instead, at V₄ and V₅ negative T waves were recorded in these cases, while at all the other V-lead positions, positive T waves were recorded conforming with the direction of the mean spatial T vector.

As can be seen in figures 4 and 5 the contour of these negative T waves often closely resembles the “coved” ST-T pattern of recent myocardial infarction. Only when multiple precordial leads have been taken and are interpreted by vector methods can the two be confidently differentiated. Such differentiation is of considerable clinical importance because 5 of the 11 subjects were young adults with no clinical or historical evidence of organic heart disease, demonstrating that isolated T negativity may occur in the apparently normal subject. Others have described atypical precordial T-wave inversions which may be instances of isolated T negativity. However, the relationship of this deformity to the electrical field of the heart as a whole and a rational method for its differentiation have not previously been presented.

In these 11 cases, the T-wave deformities were encountered only at electrode positions overlying the apical impulse, an extremely small region of the chest surface. In figure 4 are shown deflections recorded from all regions of the anterior chest and the T transitional pathway in 1 of these 11 subjects. The deflections were recorded at two inch intervals along a number of vertical lines on the anterior surface of the chest, and it can be seen that the area of isolated T-wave negativity occupies only a few square inches of chest surface with a narrow area of less marked T deformity surrounding it. Deflections from the remainder of the chest conform with the characteristics of the spatial T vector and the ST-T loop. This limited distribution of T deformity indicates that the responsible electrical force must be extremely small in magnitude. Furthermore, it indicates that the force must be generated from tissue immediately beneath the electrodes, for if it were generated from a deeper region it would be manifest over a larger area of chest surface. And finally, it will be noted that the force is only evident during the last third of the ST-T interval, having the appearance of a brief extrinsic electrical disturbance superimposed on an otherwise normal T wave. Not infrequently it appears as a V-shaped notch between what are usually identified as T and U waves; perhaps in figure 5E the deformity at V₄ and V₅ represents an instance of isolated T positivity.

How can one be certain this isolated T deformity is not simply due to the proximity of the electrodes to the myocardium at this region. That is, while at other regions of the chest the electrode is sufficiently remote from the myocardium so that all regions contribute relatively equivalently to the recorded deflections, here at the apex, potentials from the underlying portion of the myocardium dominate the recorded deflections, making them no longer consistent with the spatial concept of precordial lead deflections. There are two principal reasons why this is probably not the case. (1) Analogous deformity of the QRS complexes should have been observed at these same electrode positions, and was not. (To be sure, the frequency characteristics of potentials during the QRS period are different from those
during the T period, and this might have made a localized QRS deformity difficult to recognize. Technics have not been available to study this possibility.) (2) The mean spatial T vector is the resultant of T vectors from the various regions of the ventricular myocardium. In these cases, the direction of the mean spatial T vector was such that the T vectors for the individual regions of the myocardium were, in general, directed from endocardium to epicardium. Accordingly, if potentials from a given region of the heart should, because of proximity of the recording electrode, dominate the T forces as recorded at this region of the chest, an unusually high positive T wave should have been recorded, rather than the inverted T wave encountered in these cases.

This is of course not to deny that there are variations in the characteristics of T vectors generated from one region of the heart or another, nor that there are variations in the effective electrical remoteness of a given precordial electrode from various regions of the heart. Rather these cases indicate how rarely such differences have recognizable effects on body surface deflections when studied by present-day methods. It seems quite clear that, for clinical purposes in the vast majority of subjects, the resultant of all the T vectors, the mean spatial T vector, can with reasonable accuracy be considered to be the sole effective electrical force writing T waves in body surface leads wherever on the chest they may be taken.

Of course, it will always remain a possibility that an extremely small apical epicardial lesion will occur in a subject with a thin chest wall, and the electrical effects of this lesion will be too small in magnitude to recognizeably alter the mean spatial T vector as registered in the remoter leads of conventional electrocardiography, but will influence the precordial deflection taken directly over it. However, from the present survey of several thousand consecutive tracings examined for this possibility, it is apparent that such instances must be extremely infrequent. In most subjects all regions of the body surface, including the region of the apex, are too remote from the heart to pick up these potentials selectively, with present methods for interpreting the deflections.

Because the electrical force causing the isolated T deformity is small in magnitude, superficial in origin, generated late in ventricular systole, and different from the majority of T forces in effective direction, it is possible that it is somehow related to the mechanical impact of the heart against the left anterior chest wall during systole. Suggestive evidence for this hypothesis was found in a subject with isolated T negativity who had auricular fibrillation with a markedly irregular ventricular rhythm (fig. 5A). In this subject, the contour of the T deformity at V1 varied strikingly with the length of the preceding R-R interval, while T waves in remoter leads showed only slight variations in amplitude and no irregularity in contour in this regard. Thus, the magnitude of the electrical force causing the deformity varied with changes in the length of the preceding diastolic interval. Since the T wave is written during ventricular systole, the variations in the magnitude of the force must have been related in some way to variations in the systolic size of the heart as a consequence of the varying diastolic filling period. Such variations in systolic size would alter the intensity of the contact of the heart against the chest wall, accounting for the variations in the potentials generated at the region of contact.

Other studies were undertaken to elucidate the mechanism of isolated T-wave negativity. (1) It is well known that the application of cold to the myocardium markedly delays repolarization at the cooled region. It seemed conceivable that the outer layers of the myocardium at the apex might be “cooled” by their contact with the chest wall. However, application of heat and cold to this region of the precordium did not produce significant changes in the T deformity. (2) On full inspiration it was found that the T deformity dwindled or vanished. However, this finding did not greatly clarify the mechanism because it not only reduced the intensity of the mechanical contact of the heart against the chest wall, but also increased the distance between the heart and the electrode, thereby diminishing the recorded
amplitude of the force. The variation in isolated T negativity with the respiratory cycle is seen at V4 in figure 5B. Chest compression was not found to produce isolated T negativity in normal subjects. (3) In 2 of the subjects with isolated T negativity but no other electrocardiographic abnormality 25 Gm. of potassium citrate were given orally. In normal subjects, this amount of potassium will produce a marked increase in the magnitude of the mean spatial T vector without altering its direction. In the subjects with isolated T negativity, the T waves on the precordium were increased in size but an isolated T deformity persisted.

There is, however, a serious shortcoming in this mechanical explanation for isolated T negativity. If it were due to the impact of the heart against the chest wall, one would expect the deformity to be encountered most commonly in subjects with cardiac enlargement and ventricular hypertrophy. However, this was not the case. Indeed, of these 11 cases, only 2 had arterial hypertension, 1 with cardiac enlargement, the other without. A third subject had cardiac enlargement due to arteriosclerotic heart disease without ventricular hypertrophy. The remaining subjects were free of recognizable organic heart disease, and 5 of these 8 subjects were males under 35 years of age.

In concluding this discussion of isolated T negativity, it is well to point out that an abnormal mean spatial T vector may occasionally have such a direction as to produce an apparent instance of isolated T negativity. Figure 5C illustrates this. Here, a 55 year old man with a clinical story and electrocardiographic evolution characteristic of recent myocardial infarction, the T vector has an abnormal direction and the transitional T pathway runs obliquely across the chest in such a way that V4 lies in the area of relative negativity while all the other precordial leads lie in the area of relative positivity. The ST-T contours in the precordial V leads in this subject with myocardial infarction are nearly indistinguishable from those in figure 5D, an example of isolated T negativity obtained from a 27 year old male with no history or physical evidence of cardiovascular disease. The electrocardiogram in this latter case has been unchanged during six months follow-up.

**Summary**

1. A simple method for calculating spatial S-T and T vectors from conventional limb and precordial leads is presented, and the characteristics of these vectors in normal subjects are described.

2. It is suggested that the angle between the mean spatial QRS vector and the mean spatial T vector can be the basis for the clinical interpretation of electrocardiograms. This greatly simplifies interpretation and provides a rational and objective basis for the analysis of the electrocardiogram.

3. The commoner causes of abnormalities in the direction of the mean spatial S-T and T vectors are described.

4. A method for differentiating the normal from the abnormal electrocardiogram in the presence of digitalis effects is described.

5. An uncommon type of precordial T wave deformity is described: isolated T negativity. This deformity may be encountered in normal subjects and yet may present an ST-T contour indistinguishable from that of recent myocardial infarction. It is suggested that it may be related to the mechanical impact of the heart against the chest wall, and a method for differentiating it from other causes of T abnormality is presented.

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Circulation. 1951;3:182-197
doi: 10.1161/01.CIR.3.2.182
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
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