1963

Vector analysis of the electrocardiogram in acute myocardial infarction

Ralph Henry Keill
University of Nebraska Medical Center

VECTOR ANALYSIS OF THE ELECTROCARDIOGRAM IN ACUTE MYOCARDIAL INFARCTION

Ralph H. Keill, Jr.

Submitted in Partial Fulfillment for the Degree of
Doctor of Medicine

College of Medicine, University of Nebraska

March 24, 1963

Omaha, Nebraska

TABLE OF CONTENTS

INTRODUCTION

The electrocardiogram has been of special interest to me since my introduction to it early in my medical studies. However, I have never felt that I had an adequate understanding of the basic principles or, more important, the clinical application of this important diagnostic tool. It was my desire to come to a better understanding of the electrocardiogram that prompted me to select this general area as a topic for this paper.

The electrocardiogram serves two main purposes in clinical medicine.[1] First, it may be used in the recognition and differentiation of the cardiac disorders which affect the sequence of auricular and ventricular contractions and the time relations between them. Disorders of this type include the various cardiac arrhythmias. Secondly, the electrocardiogram may be used as an aid in the detection of abnormalities which alter the relative order in which the different parts of the ordinary ventricular or auricular muscle pass through the various stages of the excitatory and recovery process. It is in this second area where the ECG is of particular value, as there are few other diagnostic aids for the practi-

tioner. Myocardial infarction lies in this area, and for this reason this discussion will be limited to the acute phase of the uncomplicated variety of this disorder.

It is generally recognized that there are two methods of clinical interpretation of the electrocardiogram.[1,2,3] The first method is commonly known as the pattern method of analysis. This method is based on the measurement of the voltages of the different deflections of a tracing, the classification of abnormal ventricular complexes on the basis of the relative incidence of each type in various kinds of heart disease, and the analysis of series of ECGs taken on groups of patients who presented similar signs and/or symptoms, or similar structural changes in the heart after death.[1]

The second method is the vector analytic method. There are two quite different ways in which this method can be used; in the first method, four or more surface electrodes and a cathode-ray oscilloscopic recording system are used to define the properties of the resultant electrical forces generated within the heart. So far, however, this approach has been of little clinical usefulness.[2,4]

In the other method of vector analysis, the vector is used as a way to collect and integrate the information contained in the various leads of the clinical tracing.

One of the serious limitations of the pattern method lies in the likelihood that many of the electrocardiographic phenomena emphasized by the classification will be those that are most conspicuous to the eye, and not those which are most fundamental and most significant. To arrive at a rational classification of abnormal ventricular complexes and to interpret them on a logical basis requires a clear understanding of the operation of the many factors determining the character of the variations in potential at the body surface which are represented by the electrocardiographic deflections. Such an understanding cannot be acquired without some knowledge of the origin of the cardiac action currents and the principles which govern their distribution in the body.

An electrocardiographic deflection is, in the final analysis, simply a measurement of components of electrical forces generated within the heart, and therefore the most rational and objective method for

handling this information is to express it in the form of directed electrical forces, or vectors. The vector, when used in this way, is nothing more than the electrical force which, if generated at the center of a cylindrical volume conductor (the body) , would produce a distribution of potential on the surface of the cylinder similar to that which is encountered in the leads of the clinical tracing. It is for these reasons that this discussion shall be limited to the vector method of analysis.

I shall not include is this paper a discussion of infarction of the myocardium complicated by such disorders as peri-infarction block or bundle branch block, as I feel that the subject matter must be limited in order that the material is adequately covered.

In summary, after a brief review of the historical development of our knowledge of the electrocardiogram, this paper will proceed with a discussion of the basic elements of electrocardiography and of the vector approach to analysis. This will be followed by a discussion of the vector analytic method as it pertains to the electrocardiographic diagnosis of acute uncomplicated myocardial infarc-

tion. Lastly, I will discuss the vector method as it can be used to determine the location of infarctions in the myocardial tissue.

HISTORICAL REVIEW

In considering the historical background for this paper, it seems only logical to first consider the disease process with which we are concerned.

In 1768 William Heberden introduced the name angina pectoris and applied it to a type of substernal chest pain with radiation, particularly to the left arm, which was closely related to exertion.[5] He pointed out that people with this complaint may recover, but that many die in a sudden paroxysm. Shortly after this, Edward Jenner observed extensive sclerotic and calcific changes in the coronary arteries of patients dying of angina pectoris, and attributed the pain to the involvement of these vessels.[6]

In 1896 Dr. George Dock described four cases of coronary thrombosis, one of which the diagnosis was made before death.[7] However, we are indebted to Dr. James B. Herrick for our ability to differentiate this condition from ordinary angina pectoris.[8] He was the first to realize that coronary occlusion is a common condition and that many of the patients recover completely. He appreciated also that the electrocardiogram would be of great value in the diagnosis of the milder and less typical cases.

The first studies concerned with the electrical currents produced by the heart were made in 1855 by Kolliker and Muller, who isolated the frog gastrocnemius muscle and its nerve, and then placed the nerve in contact with the beating heart from the same animal. They showed that twice during each beat the heart produced electrical currents of sufficient intensity to stimulate the nerve and thus cause the attached gastrocnemius to contract.[9]

The electric currents produced by the human heart were first recorded by Waller in 1889.[10] He placed electrodes on the extremities and connected them to the terminals of the capillary electrometer invented by Lippman.

Willem Einthoven worked for several years with the capillary electrometer; knowing the peculiarities of this instrument, he was able to devise a mathematical method, which allowed him to compute from the distorted records which it furnished, curves which accurately represented the human electrocardiogram.[11] He was then able to determine the characteristics which an instrument suitable for his purposes must possess. In 1903 he constructed the first model of what has since been known as the string galvanometer.

In the period from 1906-1908 Einthoven published several articles in which he introduced his now famous triangle composed of the three standard bipolar leads, and propsed Einthoven's Law.[12,13] He also carried out simple experiments with strips of isolated muscle bounded by a dielectric. He attempted to show that if this muscle was stimulated at one end, that end of the muscle became active and hence electrically negative with respect to the inactive muscle at the other end.[14] This was referred to as the "Negativity Hypothesis."

Sir Thomas Lewis made many contributions to the field of electrocardiography, but perhaps his greatest was his "Theory of Limited Potential Differences." Lewis studied the spread of the impulse in the ventricular muscle and found that during the QRS interval, all the ventricular muscle passes into an excited state. This observation led to the conclusion that the QRS deflections represent the electric forces produced by the spread of the excitatory process over the ventricular muscle, and to a distinction between what has been called excitation, depolarization, or accession, on one hand, and what has been called recovery, repolarization or regression, on the other hand.[16] These studies led Lewis to the conclusions

that the two deflections of the biphasic curve of the hypothetical experiment on the isolated muscle strip became different, not merely in direction, but in origin. The direction of electric forces liberated by the spread of the depolarization process coincided with the direction in which the process was advancing.[15]

The field of electrocardiography stands squarely on the shoulders of Einthoven and Lewis. The advances which have been made in this field since their work have been almost solely connected with the understanding of the form of the electrocardiogram and its interpretation.

Smith, in 1918,[17] noted that ligation of the branches of the left coronary artery in the dog was followed by progressive changes in the T deflection. In 1920 Pardee[18] was the first to record similar T wave changes following occlusion in man. He noted fusion of the T to the end of the QRS in the early stage and the displacement of the S-T segment from the isoelectric level.

Several years later, Parkinson and Bedford[19] noted that the pattern of the T deflections in infarction could be divided into two groups which

they labeled T1 and T3, and thus originated a system for determining the location of the infarction.

Levine and Brown, in 1929,[20] observed the frequency of a large Q wave in lead III in coronary thrombosis. Fenichel and Kugel[21] in 1931 suggested that Q deflections in other leads might have similar significance to that in lead III. They felt that the large Q wave in lead III was due to infarction of the posterior portion of the ventricular septum.

Barnes and Whitten[22] concluded that the ST-T deflections of the T1 type were due to infarction of the anterior wall and apex of the left ventricle, and that those of the T3 type were due to infarction of the basal portion of the posterior wall of the left ventricle.

In 1933, Wilson and others divided the initial Q deflection into the Q1 and Q3 types[23]; they related that often in some infarctions Q1 and T1 types would occur together, and in others the Q3 and T3 types would occur together. They noted that the changes in the initial deflections were usually more persistent than the changes in the final deflections. They also raised the question as to the possibility that the position of the infarction in relation to

the endocardial and epicardial surfaces of the heart might play a part in determining the forms of complexes.

Wilson and his associates made great advances in the interpretation of the electrocardiogram with their work in the area of unipolar leads. Wilson[24] introduced the idea of unipolar precordial leads in 1932, and several years later they were in common usage. He also applied this concept in the development of unipolar limb leads.[25,26] Wilson's work in this area and the introduction of the central terminal have been of great significance in the field of electrocardiography.[27,28,29]

With this material for a background, I shall now begin a discussion of the fundamentals of the understanding of the electrocardiogram, with particular reference to its use in the diagnosis of myocardial infarction.

BASIS OF ELECTROCARDIOGRAPHY

In order to discuss the use of the electrocardiogram in the diagnosis of myocardial infarction, it is imperative to first have in mind the basic principles on which the subject is founded. Therefore, I shall proceed with a discussion of these basic concepts with particular reference to cardiac vector and its clinical application.

The myocardial cell at rest is polarized with a layer of evenly distributed positive charges outside and another layer of negative charges inside the cell membrane. In a biological system these charges are ions, whereas in other electrical systems these charges are due to the shift of electrons. The electrical energy which produces the deflection of the electrocardiogram is caused by the movement of these charged particles across the membranes of the myocardial cells. Such a movement of charged particles constitutes a flow of electrical current. A combination of a positive and a negative charge is known as a dipole; the direction of current flow in it is from negative to positive. There is an electrical potential behind this current flow which controls the magnitude and direction of the flow,

and it is this property of the electrical events of the heart which determines the electrocardiographic deflection.

The flow of currents in the heart is confined to the immediate environment of the membranes surrounding the myocardial cells and does not extend significantly outside the heart.[2] However, the body tissues surrounding the heart make up a relatively poor conducting medium. Since the human body is a three dimensional structure, it is a volume conductor which, for the sake of simplicity, is considered uniform. The electrical potential within the heart creates electrical fields throughout the body. These fields extend to the body surface, and it is this property that makes it possible to record the electrical events of the heart from the surface of the body.

The galvanometer consists of a delicate string or other writing element suspended movably within a magnetic field.[30] A wire extends from each end of the string; one wire is the positive electrode while the other is a the negative electrode. When the two electrodes are attached at the different points on the body surface, a lead is formed. Since

the two electrodes are at different locations in the electrical field, they are at different points of electrical pressure produced by the cardiac potential. Therefore, there is a flow of currect through the electrodes and the string. It is the flow of current through the string which causes it to deviate, and it is the recorded movement of the string which forms the deflection on the electrocardiogram. Thus the electrocardiographic deflection is a measurement of the way in which the lead intercepts the electrical field of the cardiac potential.

It would seem that the electrodes of any, or all, leads would have to be equidistant from the heart to record the electrical activity of the heart on the same scale. Einthoven[30] realized this when he began his work on the electrocardiogram; he attempted to solve this problem by applying a property of electrical fields to that produced by the heart. This property is that the intensity of an electrical field diminishes algebraically with distance from its center. This means that as the electrodes are moved farther and farther away from the center of the field, the drop in field intensity becomes less and less. Therefore, it can be reasoned that if the electrodes are placed as far from the

heart as possible they might be considered equidistant electrically even though they are not equidistant from it anatomically.

Since the extremities are the part of the body most remote from the heart, Einthoven placed the electrodes on the, and this method is still in use today. The electrodes are placed on the right arm, left arm, and the left leg. These leads are known as the standard, or bipolar, limb leads, and the polarity of these leads is as follows:

Lead I---LA(+) RA(—)
Lead II--LL(+) RA(—)
Lead III-LL(+) LA(—)

Einthoven represented these electrodes as the apices of an equilateral triangle and considered the cardiac dipole to be at its center.[30] This arrangement is shown below in Figure 1.

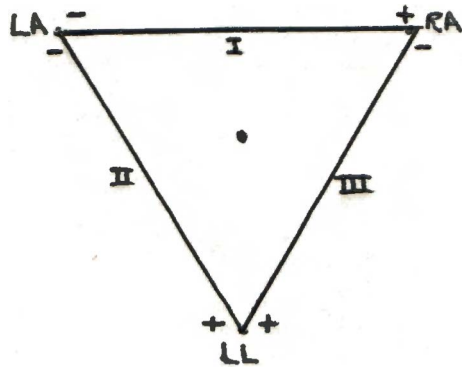


Fig. 1

When represented in this way, the bipolar limb

leads may be shown in a graphic manner. It is customary in constructing a graph to draw the various axes of the graph through the same zero point. Bayley[31] devised such a graph by drawing the three limb lead axes in their proper direction and with their proper polarity but with all of them sharing the same zero point. With this type of a system, the limb lead axes form a reference figure consisting of three axes, 60° from each other, and this is called the triaxial reference system. A diagram of this system is shown below in Figure 2.

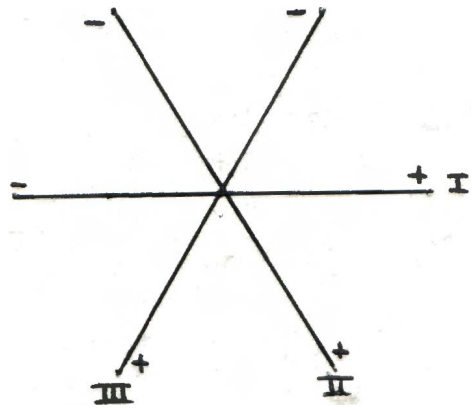


Fig. 2

In 1934, Wilson introduced three new leads to obtain a better picture of the heart's electrical activity in the frontal plane.[26] In these leads the

positive electrode is attached to one of the limbs and the negative electrode is attached by wire to all three limb leads. This negative electrode adds the potential of all three standard limb leads which by Einthoven's Law (to be discussed later) means that the negative electrode must be at zero potential at all times. It follows that the axis for this type of a lead is a hypothetical line from the limb where the positive electrode is placed to the zero point of the electrical field of the heart. Since this type of a lead has only one electrode, it is referred to as a unipolar lead. An example of such an arrangement is diagrammed in Figure 3.

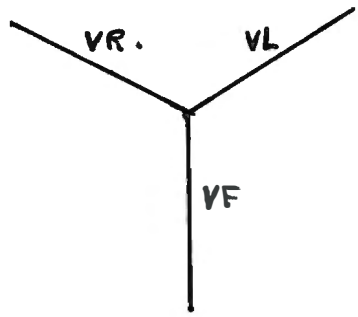


Fig. 3

These leads are labeled by the limb which has the positive electrode, and are preceded by the letter "a" to denote that these leads are augmented, and

the letter "V" to denote that they are unipolar leads. A triaxial reference system can be formed for these leads also, as shown in Figure 4.

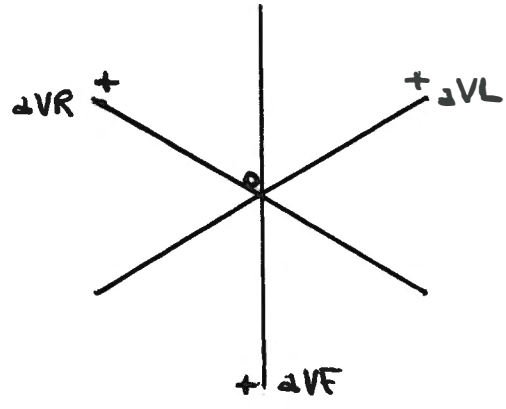


Fig.4

The reference systems of the two types of limb leads may be combined so that a single hexaxial reference system is produced as shown in Figure 5. It is this type of a system which is commonly used in the interpretation of the frontal plane ECG by vector methods.

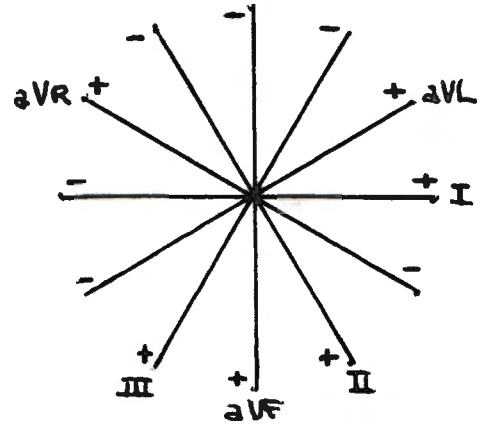


Fig. 5

The potentials generated within the heart are measurable, and the measurable properties of an electrical potential are its magnitude and its direction. Any quantity that has these properties may be represented by a symbol known as a vector; it is represented by an arrow where the length of the arrow indicates the magnitude, and the direction of the arrow indicates the direction. The arrowhead indicates the location of electrical positivity.[32] Vectors can be used to explain the relationship between the ECG deflection and the electrical activity taking place in the heart. The graphic line drawn between the two electrode positions for a given lead is called the axis for that lead. The ECG deflection is a measurement of the projection of the cardiac vector on that lead.

If the vector is parallel to a lead it will have a large projection on that lead; if the vector is perpendicular to a lead it will have a very small projection on that lead. If the deflection is positive in a lead, the vector will point in the direction of the positive pole of that lead, and vice versa. It follows that if the net deflection is zero, the vector will be perpendicular to that lead.

An example of a vector and its projection on the triaxial reference system of the bipolar limb leads is shown in Figure 6.

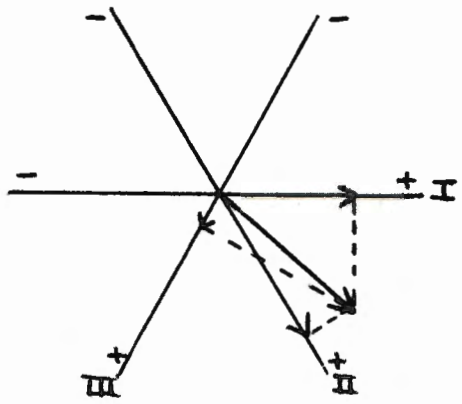


Fig. 6

Einthoven's Law states that the magnitude of the deflection in Lead I plus its magnitude on Lead III equals the magnitude of the deflection on Lead II. This can be stated algebraically:

Lead I + Lead III = Lead II

Since the length of a vector represents the magnitude of the force it represents, vectors can be used to illustrate this law. If the projections of the vector in Fig. 6 are added, we have:

In the electrocardiogram, the P wave represents the depolarization of the atria, the QRS complex the

depolarization of the ventricles, and the T wave the repolarization of the ventricles. All the aspects of the QRS forces that can be used in clinical interpretation are contained in three components of the QRS forces:[2]

1. The direction of the mean QRS vector
2. The direction of the mean vector for the first 0.04 second of the QRS interval
3. The direction of the mean vector of the last 0.04 second of the QRS interval

Depolarization and repolarization spread through the ventricles in such a way that at each region QRS and T vectors can be considered to be directed from endocardium to epicardium. At any single instant during either the QRS or T cycles, the galvanometer records the sum of all electrical activity taking place at that instant as if there were but a single force or vector responsible for it. This vector is known as the instantaneous resultant vector. Thus, depolarization of the ventricles is recorded by the galvanometer as if there were a series of single instantaneous QRS vectors of successively changing magnitude, direction, and with the same zero point at the center of the chest (Fig. 7A). It is often useful to consider the average direction of all vectors of the QRS interval; this vector is called

the mean QRS vector. It can be determined by adding all the instantaneous vectors by the origin to terminus method as shown in Figure 7B.

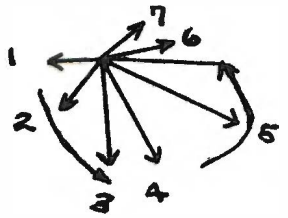


Fig. 7A

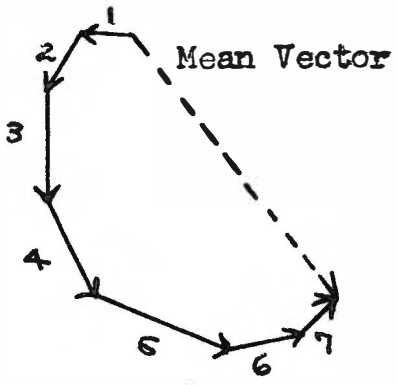


Fig. 7B

There are certain conditions which tend to affect only the early or late QRS forces. Therefore, it is sometimes valuable to determine the mean vectors of the first 0.04 second and the last 0.04 second of the QRS interval. In a like manner it is possible to determine the mean vector for the P and T intervals.

When the mean QRS vector is plotted on the triaxial reference system, it should lie somewhere within the range as indicated in Figure 8. Its position depends to some extent upon the age of the subject and of the electrical position of the heart. In a normal subject the angle between the mean QRS

vector and the mean T vector should not exceed 45° in the frontal plane.[2]

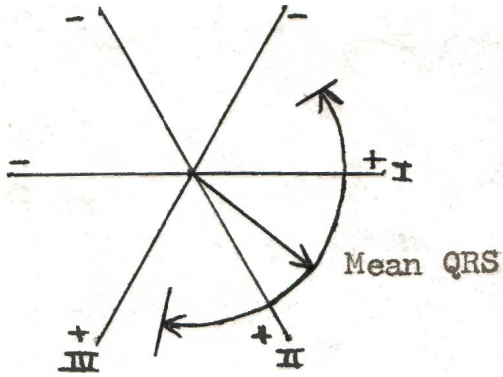
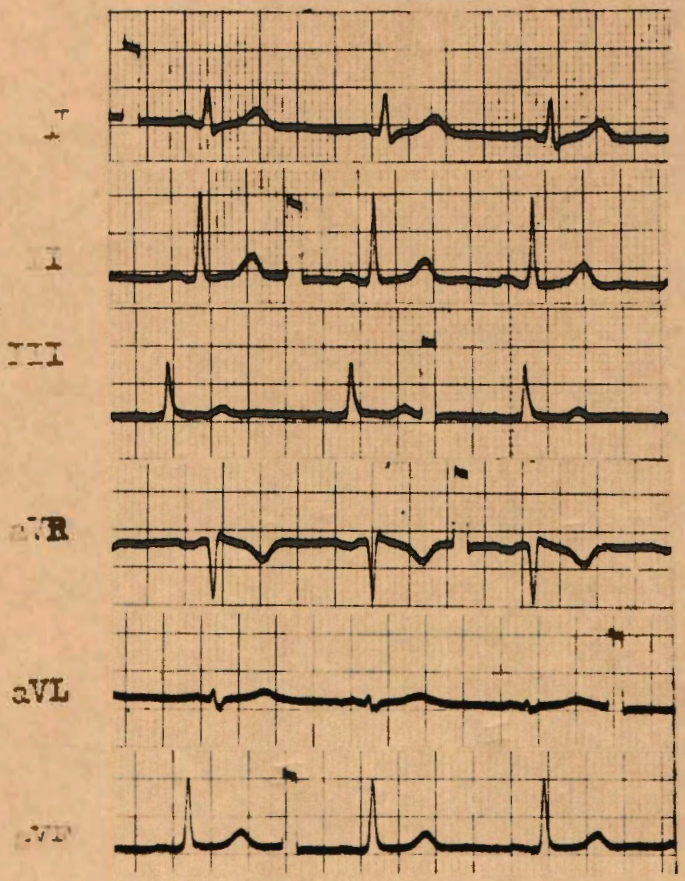


Fig. 8

Now with an understanding of the vector concept and the relation of the deflection on a lead to the projection of a vector onto that lead, it is possible to analyze a frontal plane electrocardiogram by vector methods. Also, with a knowledge of the normal range of the mean QRS and T vectors on a reference system, it is possible to determine the normality or abnormality of these vectors. A frontal plane electrocardiogram and its vectoral interpretation is shown in Figure 9.



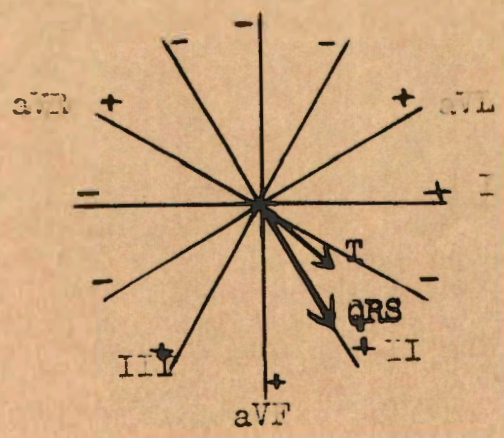


Fig. 9

In the tracing shown in Figure 9 the QRS complex has a net zero, or isoelectric, deflection in lead aVL. In other words, the mean QRS vector has a very minute projection on lead aVL, so the direction of the mean QRS vector must be perpendicular to lead aVL. The QRS complex has a net positive deflection in leads II, aVF, and III, so the sense of the vector must be towards the positive pole of lead II as shown in Figure 9. The T wave is closest to being isoelectric in lead aVL but is slightly positive in this lead. It is strongly positive in lead II and strongly negative in lead aVR. From these observations it is possible to locate the T vector as being nearly perpendicular to lead aVL but angled slightly towards the positive pole of lead aVL. It can also be determined that the sense of this vector is towards the positive pole of lead II and the negative pole of lead aVR as shown in Figure 9.

This discussion has thus far been limited to the interpretation of the frontal plane electrocardiogram. In other words, the heart has been considered in two dimensions only. However, the heart and the human body have three dimensions, and it seems likely that a great deal more could be learned

from a tracing if the third dimension could be recorded.

Wilson[24,27] introduced a method by which electrical activity of the heart could be recorded in the anterior-posterior projection. He showed that this could be done by using a unipolar lead in which the positive electrode was placed at different points on the precordium, and the negative electrode was connected to all three limb electrodes. Since Einthoven's Law states the sum of the three limb leads equals zero, the negative electrode of the unipolar chest lead is at zero potential relative to the frontal plane throughout the cardiac cycle. Thus, the axis on which the chest lead measures the cardiac vector is a line from the position of the chest electrode to the zero point of the electrical field of the heart. A positive deflection on this lead means that the sense of the vector is towards the precordial electrode, and a negative deflection means that the vector points away from the precordial electrode.

In present-day electrocardiography there are six locations for unipolar precordial leads which are in common use; they are as listed below:[33]

V1: Fourth intercostal space, at the right sternal border.

V2: Fourth intercostal space, at the left sternal border.

V3: A point equidistant between V2 and V4

V4: Fifth intercostal space, in the mid-clavicular line.

V5: The anterior axillary line at the level of V4.

V6: The mid-axillary line at the level of V4 and V5.

These are the standard precordial leads which are in common usage. However, there are other lead systems which are used on occasion, and the principles of application are the same as for the common unipolar precordial leads.

The interpretation of the precordial ECG can be confusing, but it can be greatly simplified by the application of vector methods. If the human body is considered a cylindrical volume conductor, then the electrical activity of the heart can be represented as a vector, or vectors, generated from the center of this cylinder. If unipolar leads are recorded from the surface of the cylinder, each deflection will be a measurement of the projection of the vector on the axis of that lead. Whenever an electrode is placed at such a point on the surface that the axis of the lead is perpendicular to

the direction of the vector, it will record a zero, or isoelectric deflection.[34] There are a number of points on the surface of the cylinder where such isoelectric deflections will be recorded. Since they lie on axes which are perpendicular to the vector, they form a pathway around the chest (cylinder), which forms a plane perpendicular to the vector and intersects the surface of the cylinder. This pathway around the cylinder is known as the null contour for that vector and is illustrated in Figure 10.

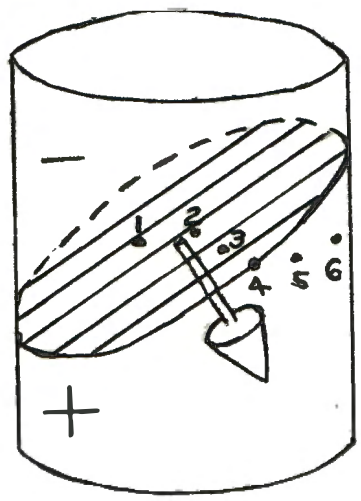


Fig. 10[34]

At all points on one side of the null contour, the unipolar leads will record positive deflections for that vector. This is the side toward which the

vector points, and is called the area of relative positivity. At all points on the other side of the null contour, all unipolar leads will record negative deflections; this area is the area of relative negativity. Therefore, in Figure 10 lead 4 will record an isoelectric deflection. Leads 1, 2, and 3 will record a negative deflection and leads 5 and 6 will record a positive deflection.

It can be seen from this that if one can identify a null contour, one can plot the direction of the vector, for it must lie relatively perpendicular to the plane defined by the null contour. It follows that one can determine the direction of a vector in space and thereby analyze any portion of the cardiac cycle.

As a first step in the analysis of a cardiac vector, it is necessary to determine the direction of the vector in the frontal plane as described previously and illustrated in Figure 9. Next, it is necessary to determine which of the unipolar chest leads has an isoelectric deflection, for this lead must lie on the null contour. Finally, one must draw or imagine a plane perpendicular to the vector and running through the isoelectric lead to visualize the vector in space.

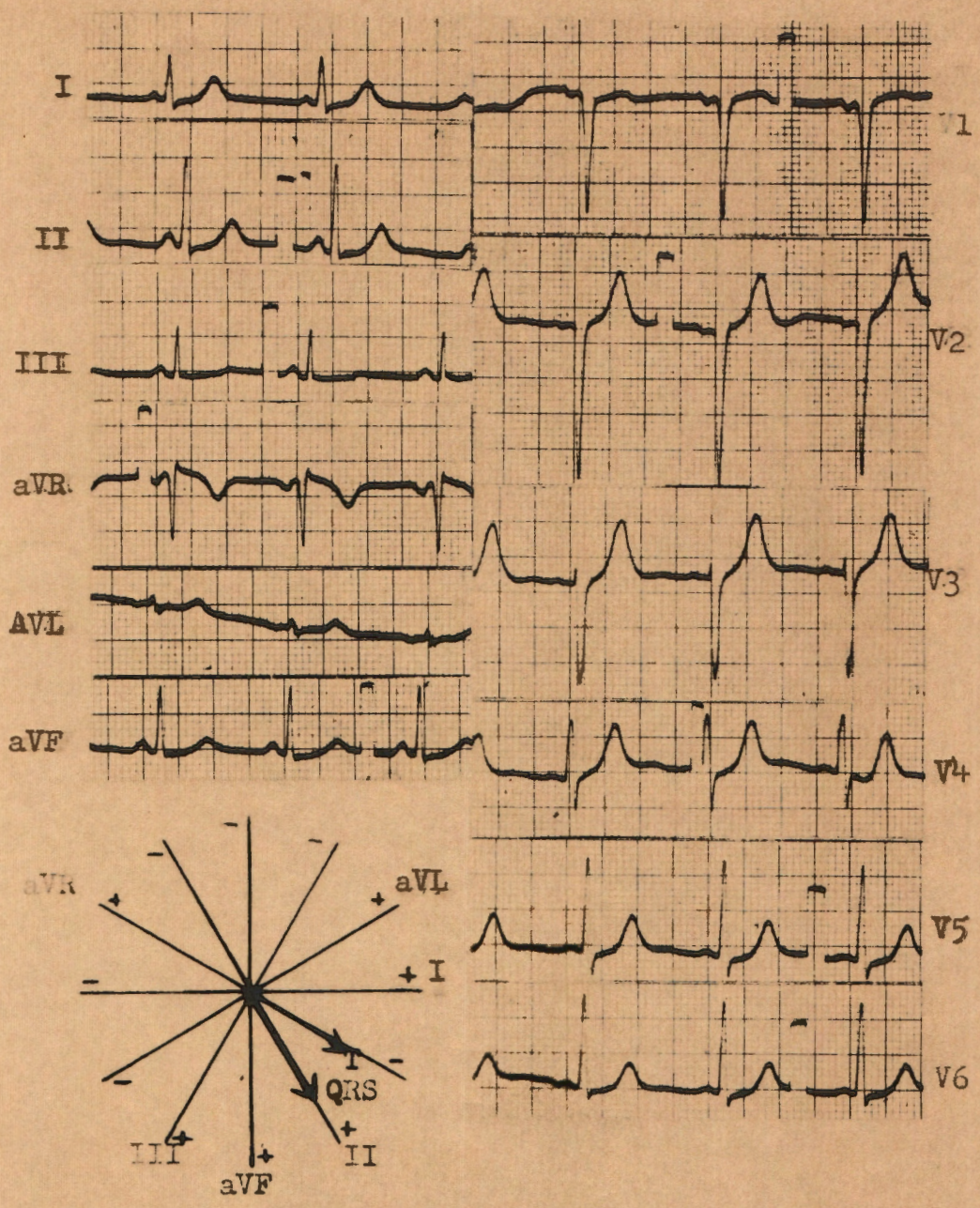

I
II
III
aVR
AVL
aVF
V1
V2
V3
V4
V5
V6
aVL
+ I
T
QRS
III
aVF
II
I
QRS
aVF
I
T
aVF

Fig.11

A normal ECG tracing and its spatial vector analysis is shown in Figure 11; it can be analyzed in the frontal plane as discussed previously. The mean QRS deflection is isoelectric in lead aVL and strongly positive in lead II; thus it is perpendicular to lead aVL and its direction is towards the positive pole of lead II as shown. The T wave is isoelectric in lead III and strongly negative in lead aVR, so its vector is perpendicular to lead III and its direction is towards the negative pole of lead aVR as shown.

The next step is to determine the position of the vectors in space. The null contour can be imagined as a round plane which is perpendicular to the vector and to the page. If this plane is now rotated so that its leading edge passes through the isoelectric chest lead, the position of the vector can be determined in space. This is demonstrated in Figure 11 where the mean QRS complex is isoelectric between leads V3 and V4. If the leading edge of the null contour is drawn between V3 and V4, it is seen that the mean QRS complex is pointing posteriorly since, by definition, the vector must be perpendicular to the null contour.

Similarly, the T wave is isoelectric slightly to the left of lead VI, so if the null contour is drawn in this position, it is shown that the T vector points anteriorly (Fig. 11).

Since the direction of the mean QRS vector is within the normal range (Fig. 8) and the angle between this vector and the T vector is less than 45^{o}, these two vectors can be interpreted as normal.

Now, applying these principles of vector analysis, it is possible to proceed with a discussion of the electrocardiographic diagnosis of acute myocardial infarction. It must be remembered that any interval of the cardiac cycle can be analyzed in the same manner.

VECTOR ANALYSIS OF MYOCARDIAL INFARCTION

When an area of the myocardium becomes infarcted by loss of blood supply, there are several changes which result. The actual infarction is a region of necrosis that is not capable of electrical activity, hence it is dead electrically. This is surrounded by an area of injury which has impaired electrical activity. Surrounding this is another area with impaired activity which is due to ischemia without any injury or death to the tissue involved.

There are four abnormalities of the electrocardiogram in acute myocardial infarction; all four may be present in any given case.[2] These four abnormalities, as represented by vectors, are;

1. Alteration in direction of the first part of the QRS interval. This is best represented by the initial 0.04 sec. vector; it tends to point away from the site of the infarction.

2. Alteration in the direction of the mean T vector due to the electrical ischemia surrounding the infarct. This vector also tends to point away from the site of the infarct.

3. Injury current surrounding the infarct produces an S-T vector which tends to point towards the site of the infarction.

4. In some cases of infarction, the last part of the QRS complex is altered due

to peri-infarction block. This is best represented by the terminal 0.04 sec. vector which tends to point towards the site of the infarct in these cases.

From the clinical point of view, it is important that there are four abnormalities associated with myocardial infarction; there are other conditions which can produce one or another of these abnormalities, but only infarction can produce all four. However, all four are present only in the acute stage of infarction.

It has been shown that the initial portion of the Q wave is altered in 95 per cent of the cases of myocardial infarction.[34] In most cases of acute infarction, all four vector abnormalities will be present. However, cases have been described of severe, even fatal infarction, in which no electrocardiographic abnormality could be demonstrated.[23,35] Thus, while the ECG is relatively diagnostic of infarction when all four abnormalities are present, a normal electrocardiogram does not rule out an infarction.

During the first 0.04 sec. of the QRS interval, excitation of the subendocardial layers of the heart takes place.[36] QRS vectors are generated simultane-

ously from all regions of the inner layers of the left ventricles during this interval as shown in Figure 12A. The mean vector of all these vectors is known as the initial 0.04 vector. Normally, this vector is approximately parallel with the mean QRS vector for the subject and points leftward, inferiorly, and slightly posteriorly as in Figure 12E. With infarction the subendocardial layers at the site of the infarct are made electrically inert, or dead, and do not contribute vectors during the first 0.04 sec. of the QRS interval. As a result, the forces developed in portions of the heart located opposite the infarcted area are no longer opposed[3], as shown in Fig. 12C. This results in a displacement of the sum of these forces away from the site of the infarct. This change produces a shift in the direction of the mean 0.04 initial vector from a normal to an abnormal position (Fig. 12B-D).

It can be seen in Fig. 12E that when the initial 0.04 vector is directed normally, it has positive projections on the three standard limb leads and writes no Q waves in these leads. However, if this vector is caused to deviate outside of this area, it will have negative projections in one or

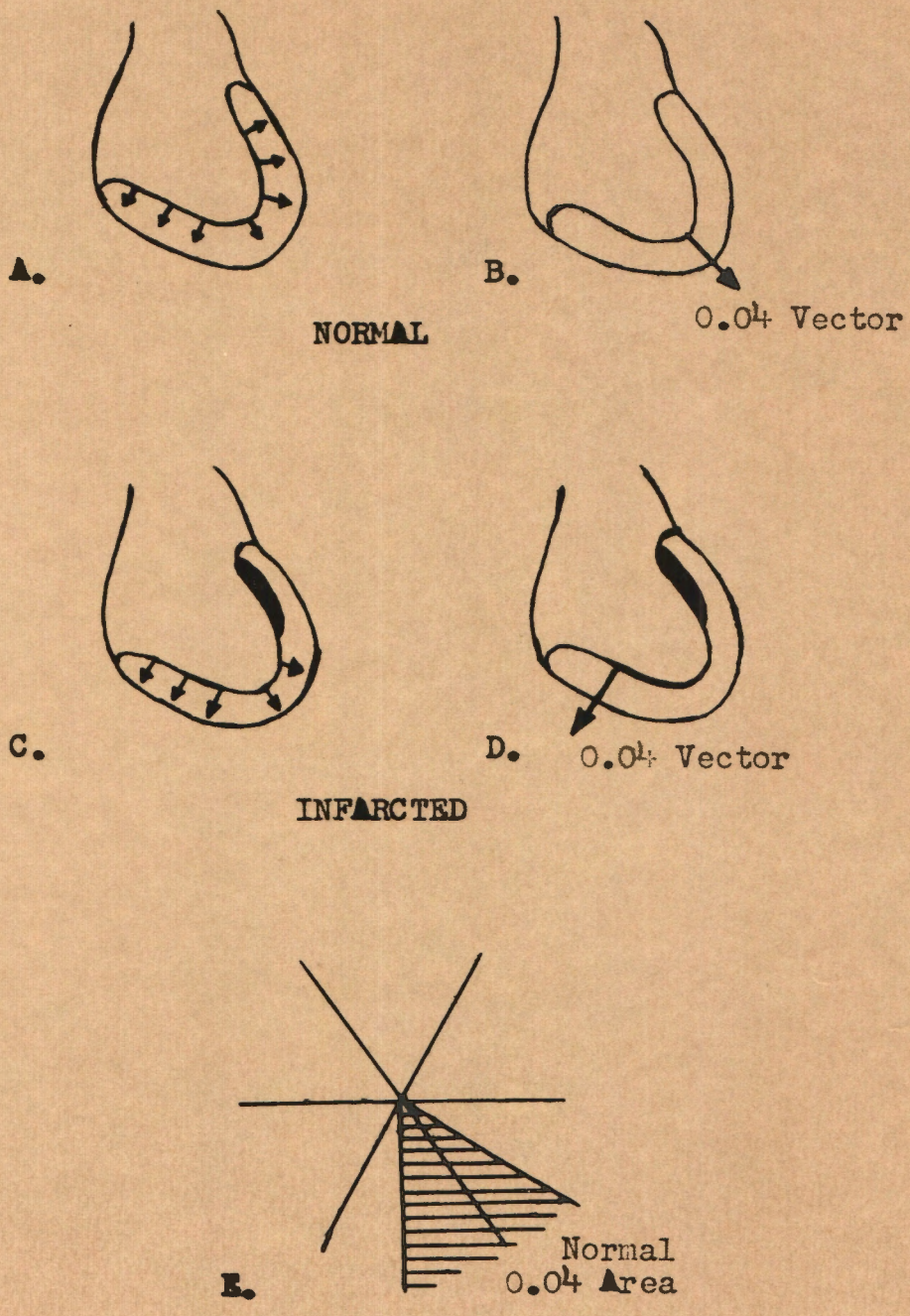

A.
B.
0.04 Vector
NORMAL
C.
D.
0.04 Vector
INFARCTED
E.
Normal
0.04 Area

Fig. 12

more of these leads, resulting in Q waves. This is the basis for the abnormally-directed initial 0.04 vector being of importance in the diagnosis of infarction.

If, however, an infarction produces an initial 0.04 vector that remains within the normal area, no Q waves will be produced. This is why tracings from some subjects with infarctions may fail to have waves. It is in these cases where a normal ECG tracing may have to be disregarded in the presence of clinical evidence of infarction.

In general, there are two types of T vector abnormalities. The first, called a primary T vector abnormality, is due to alterations of myocardial metabolism. The other type, known as a secondary T vector abnormality, is seen in ventricular conduction disorders.[2] It is the primary type with which we are concerned in myocardial infarction.

Wilson,[37] felt that the T wave changes were due to a local physiologic gradient at the margins of the infarct, and that the gradient was of a kind that affected the subsidence (repolarization) phase. As a result, the T vector from the uninvolved regions of the myocardium dominate the mean T vector, caus-

ing it to point away from the site of the infarction.

The above is true if the predominant location of ischemia is epicardial, which is the usual case in infarction. Rarely, an infarction will produce predominantly endocardial ischemia, and in this situation the mean T vector will point towards the site of the infarction.

The third abnormality that is present in myocardial infarction is the presence of an S-T vector. Normally, the S-T segment has zero potential and thus has no vector. This vector is thought to be due to injury of the cell membranes of some of the tissue in an infarction.[37] If the membrane of a cell is injured so that there is an imperfection in the semipermeability, then the cell is such that it is incapable of maintaining the polarized state. As soon as repolarization places charges on the membrane, they leak off. This leak represents a flow of ions and is called an injury current.

The injury current produces a displacement of the baseline between the T wave and the next QRS complex. However, the S-T segment is a much easier portion of the tracing to study, so it is conventional to interpret the injury current in terms of the amount of S-T segment displacement. When the S-T segment, or vector, is used in this way, it

represents the reciprocal of the vector generated by the injury current.

Most infarctions of the myocardium produce injury in the epicardial region at the site of the infarct. Since the S-T vector is the reciprocal of the true injury current, it tends to point towards the site of the infarction. If, however, the infarct is subendocardial in location, the injury current will be produced in the endocardial regions; as result, the S-T vector will point away from the site of the infarction.

The last abnormality which may occur in infarction is alteration in the last 0.04 sec. of the QRS interval. This is represented by the terminal 0.04 vector, and points towards the site of the infarction in these instances. This was first described in 1950 and was termed peri-infarction block.[38]

The mechanism for this alteration is not known, but the most common explanation is that in a subendocardial infarction, the normal perpendicular spread of excitation from endocardium to the uninfarcted epicardium is prevented. Therefore, the excitation can reach this region only by circuitous spread around the infarct, with resultant delay of the terminal segment of the QRS interval. This results

in a terminal 0.04 vector that points towards the site of the infarction.

This abnormality, along with other conduction defects and arrhythmias associated with infarction, are beyond the scope of this paper and will not be discussed further.

The relations of the initial 0.04 vector, the S-T vector and the T vector are shown in Fig. 13.

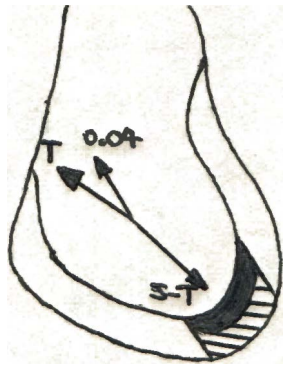


Fig. 13

The darkened area represents an area of infarction, while the striped section represents an area of injury and ischemia. It is most important to remember the direction of these three vectors in relation to the site of the infarction:

Initial 0.04 Vector ..... away
T Vector ..... away
S-T Vector ..... towards

The remainder of this paper will be devoted to the discussion and demonstration of how all the principles previously discussed can be applied for

the diagnosis and localization of infarctions of the myocardium.

LOCALIZATION OF INFARCTIONS

The cardiac vectors previously discussed can be used to determine the approximate location of an infarction on the myocardium. All that need be remembered is that the initial 0.04 vector and the T vector tend to point away from the site of the infarct, and that the S-T vector tends to point towards the infarction.

However, there are a few situations in which the direction of these vectors may not be as previously described. For example, consider a case in which a patient has an old anterior wall infarct. If this patient would later suffer posterior wall infarction, the initial 0.04 vectors from these two sites would tend to cancel each other, resulting in a normal initial 0.04 QRS vector. The diagnosis in this case would have to be made on the basis of the direction of the T and S-T vectors. The T vector would point away from the site of the new infarction, so it would be directed anteriorly. The S-T vector would point posteriorly towards the site of the acute infarction.

The mean T vector may point towards the region of the infarct at the very onset of the infarction.

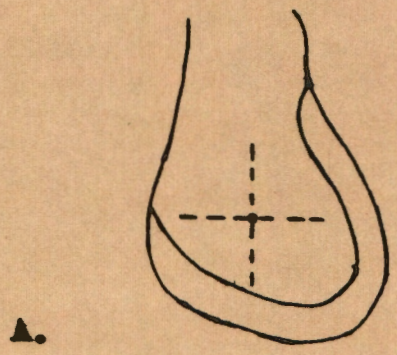
However, it will assume an opposite direction in a day or two, due to the ischemia which develops.[39]

The range of direction of the mean initial 0.04 vector for various locations of infarctions is illustrated in Figure 14. An outline of the heart is included to help in visualizing the region of infarction represented by each vector.

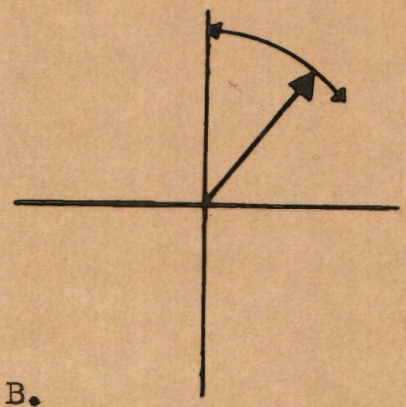
The initial 0.04 vector in inferoseptal infarction is directed into the left upper quadrant of the triaxial reference system (Fig. 14B). In lateral infarction it is directed into the right lower quadrant (Fig. 14C). The initial 0.04 vector in apical infarction is usually directed into the right upper quadrant, but it may be shortened and pointing toward the apex (Fig. 14D). This vector is directed posteriorly in anterior wall infarction and directed anteriorly in infarction of the posterior wall (Fig. 14E-F).[2,40]

The T vector tends to parallel the initial 0.04 QRS vector in infarction and the S-T vector usually is directed opposite to these two vectors towards the region of infarction.

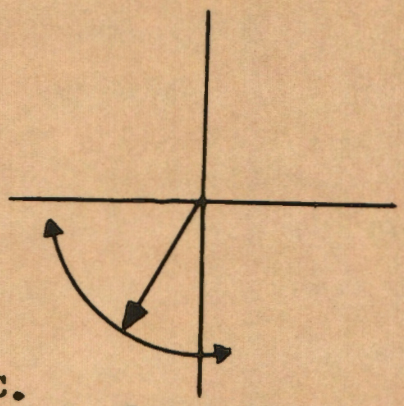
Now, let us see how these principles apply to clinical applications. Several ECG tracings are



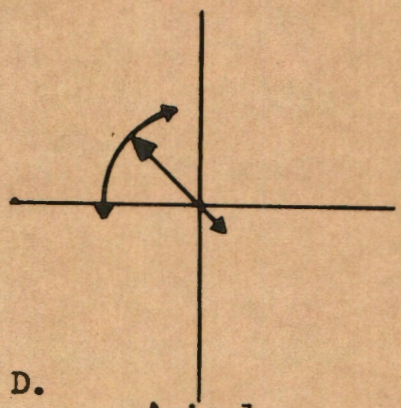
A.



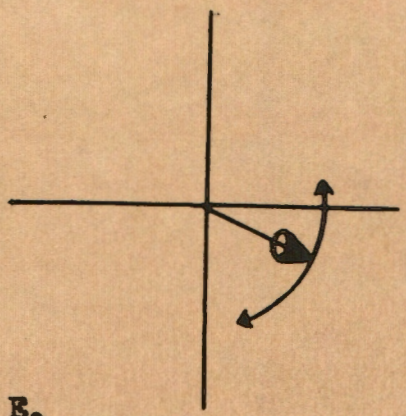
B. Inferoseptal



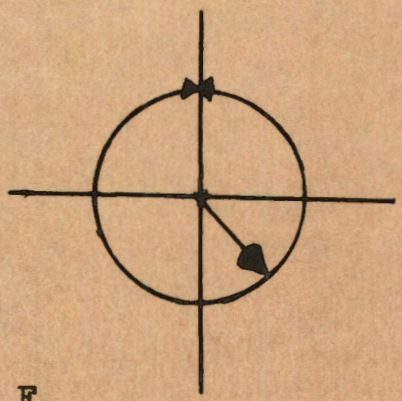
C. Lateral



D. Apical



E. Anterior



F. Posterior

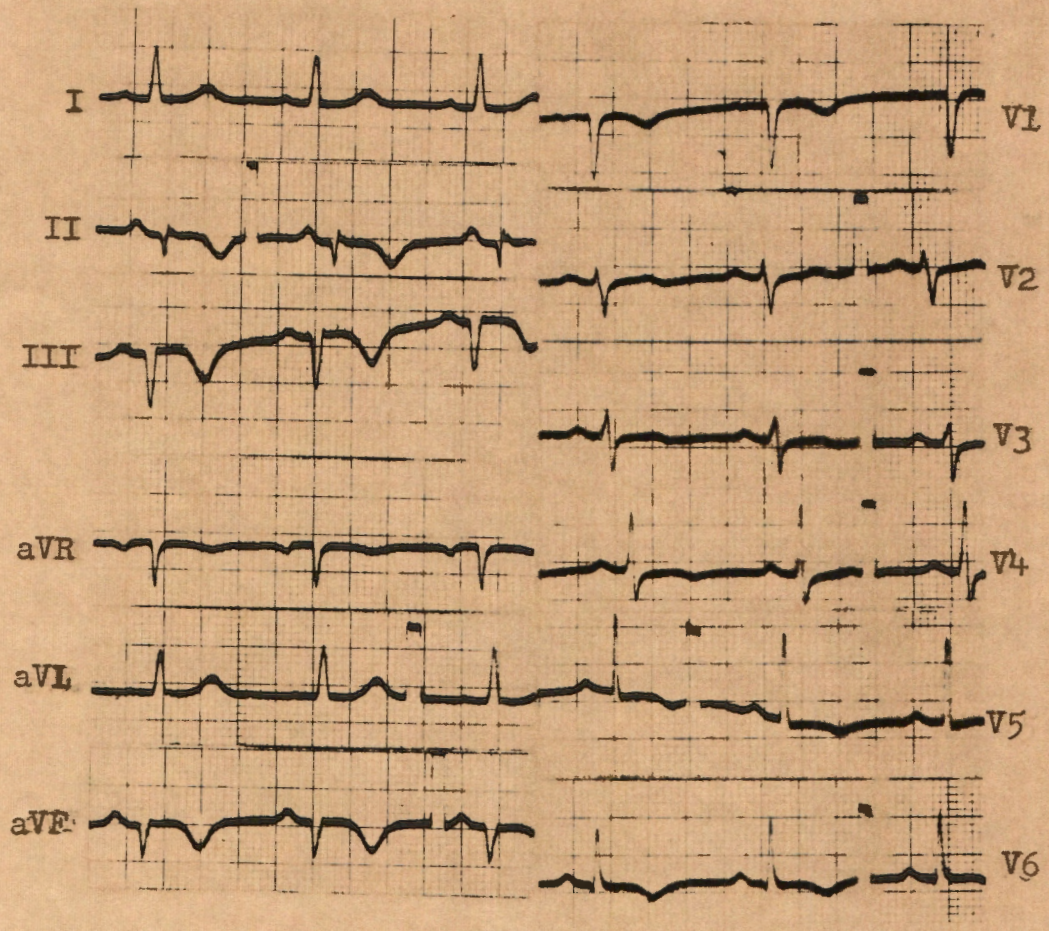
Fig. 14

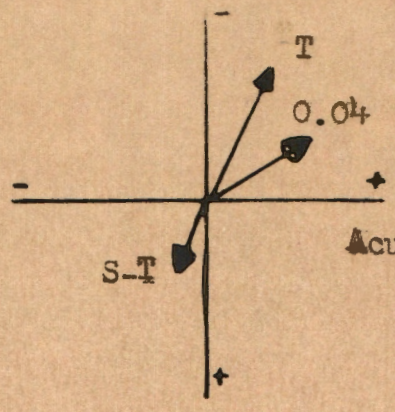
included in the following pages. Accompanying each tracing will be the vector analysis of it and a brief discussion of the methodology and interpretation.

The first ECG tracing to be considered is shown in Figure 15 and its vector interpretation is diagrammed in Figure 16. The initial 0.04 QRS vector is nearly isoelectric in lead II, positive in aVL, and isoelectric in V2-V3, so it is directed to the left, superiorly, and posteriorly. The T wave is isoelectric in aVR, positive in lead I, and isoelectric in lead V4, so that its vector almost parallels the initial 0.04 vector, except that it is directed slightly anterior. The S-T segment is isoelectric in aVR, positive in III and isoelectric in V3, so that it is directed opposite the T vector and slightly posterior (Fig. 16D).

From the direction of these three vectors, this tracing can be diagnosed as acute inferoseptal infarction with the S-T vector pointing towards the site of the infarction and the T and 0.04 vector pointing away from the infarction (Fig. 15).

The next tracing and analysis are shown in Figure 17-18. An analysis of this tracing shows that the initial 0.04 QRS vector is isoelectric between





Myocardial Infarction, Acute, Inferoseptal

Fig. 15

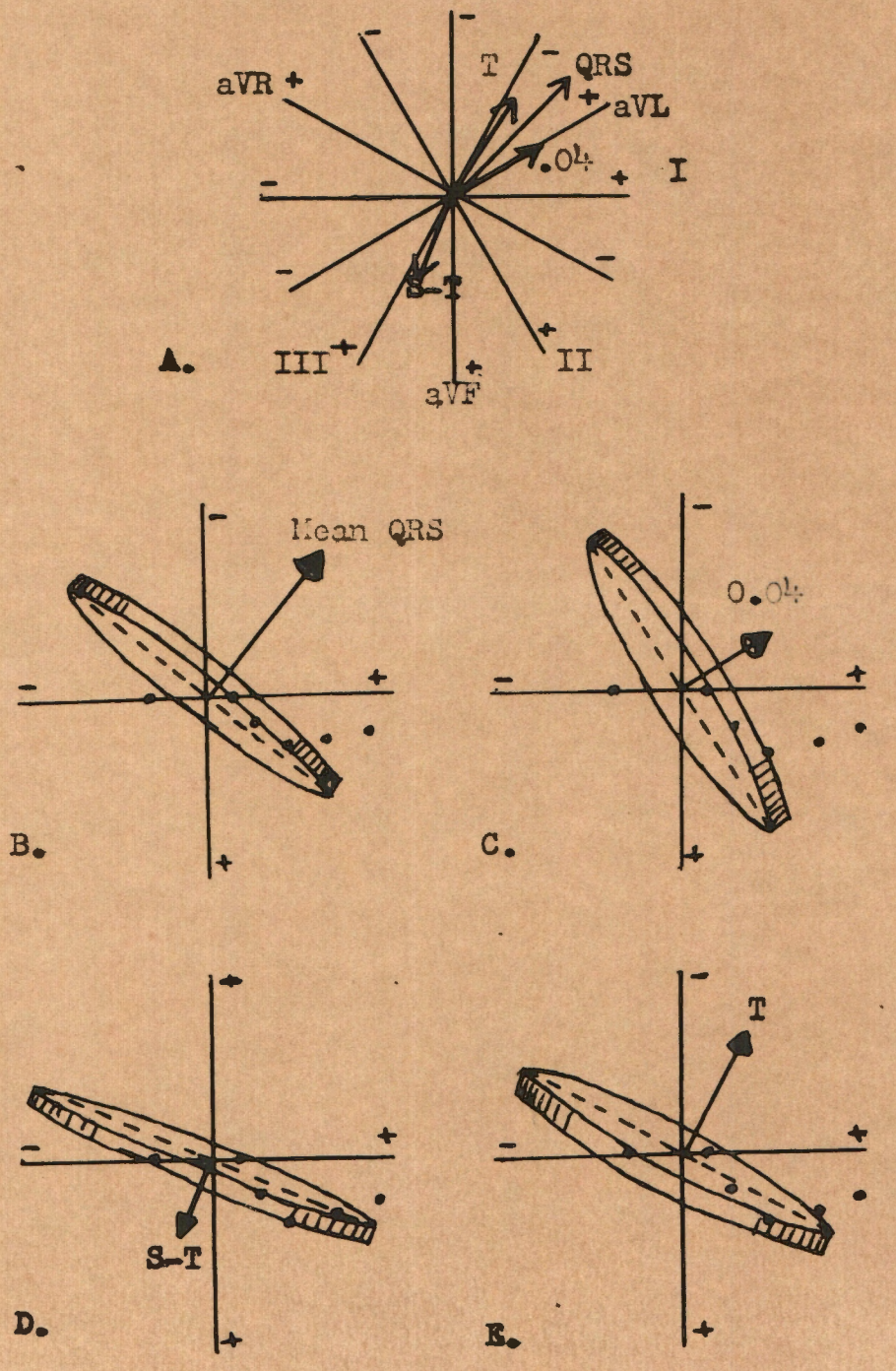
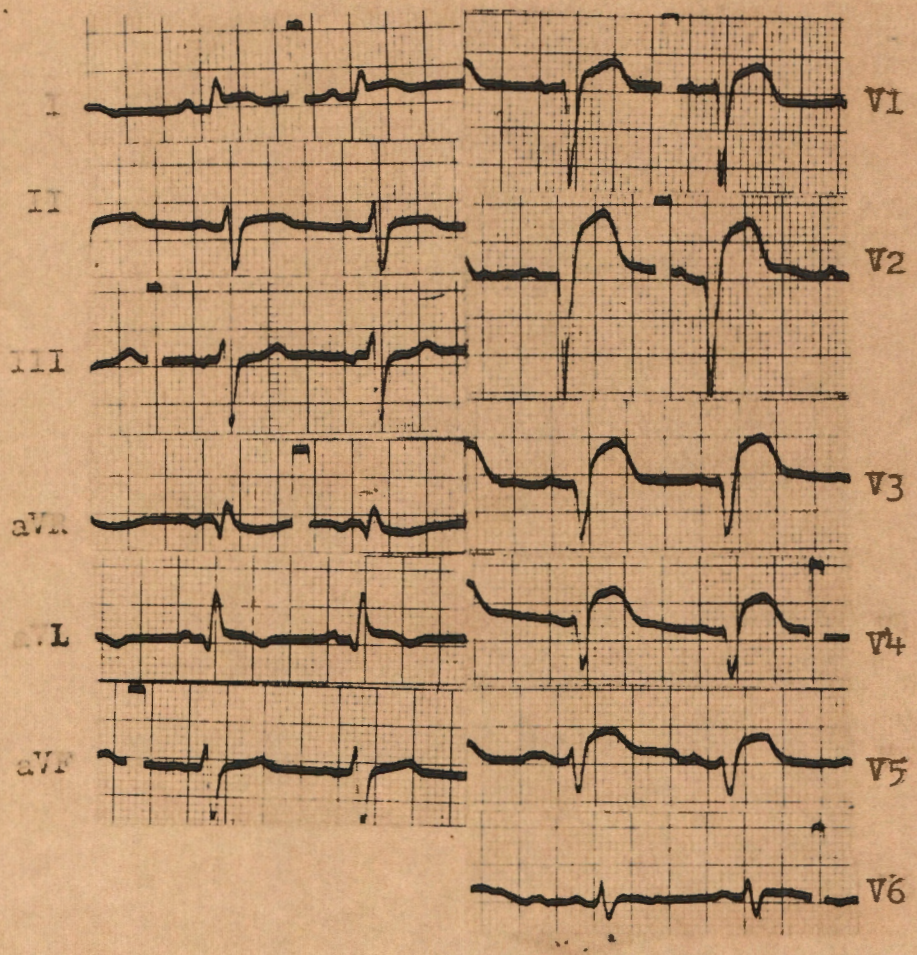
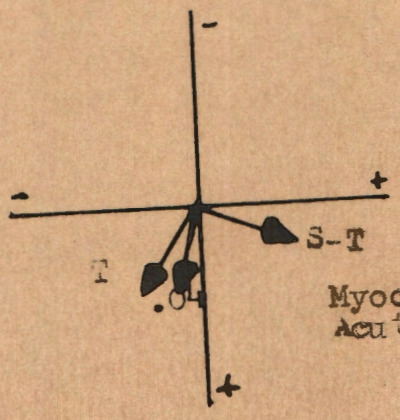


Fig. 16

leads I and aVR, positive in aVF, and isoelectric between V4 and V5, so it is directed slightly to the right, inferiorly and posteriorly (Fig. 18C). The T vector parallels the initial 0.04 vector closely, but is directed anteriorly. The S-T segment is nearly isoelectric in leads III and aVL, positive in I, and isoelectric somewhere to the right of chest lead V1; thus it is directed to the left, almost horizontally, and markedly anteriorly. The position of the vectors indicates that this tracing represents an acute anterolateral infarction (Fig. 17).

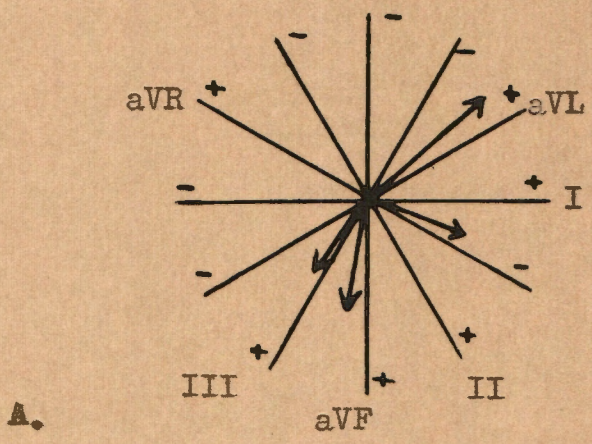
The third tracing and its vector analysis are illustrated in Figures 19-20. The initial 0.04 QRS vector is isoelectric in lead aVR, positive in aVL, and isoelectric in V1, so it is directed far to the left, almost vertically, and anteriorly. The T vector is approximately parallel to the initial 0.04 vector, but is more anterior. The S-T segment is isoelectric in aVR and pointing towards the positive pole of lead III. It is isoelectric in V1, so the S-T vector is directed slightly to the right, inferiorly, and posteriorly (Fig. 20D). This arrangement of the vectors signifies an acute



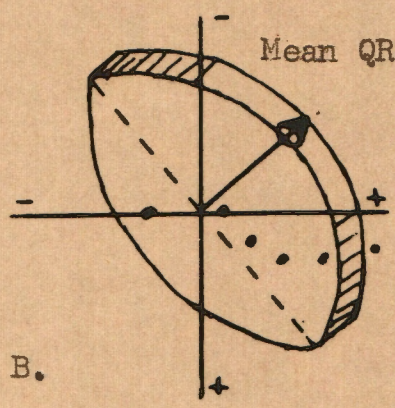


Myocardial Infarction,
Acute, Anterolateral

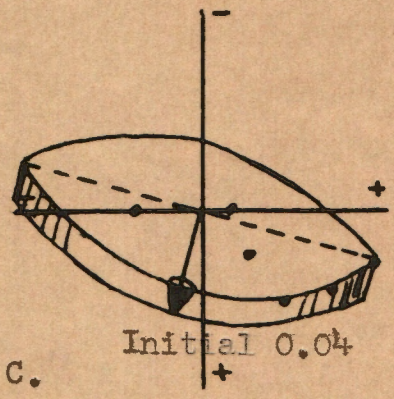
Fig. 17



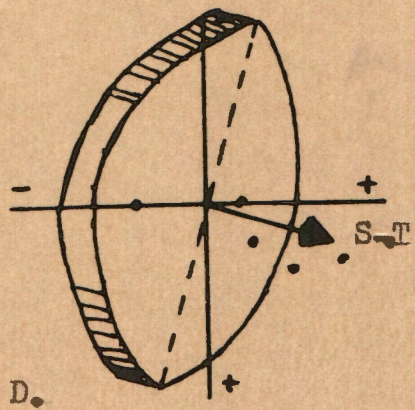
A.



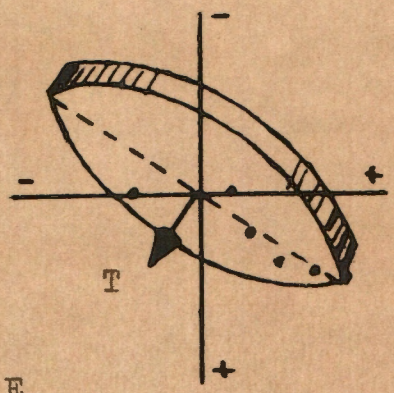
B.



C.

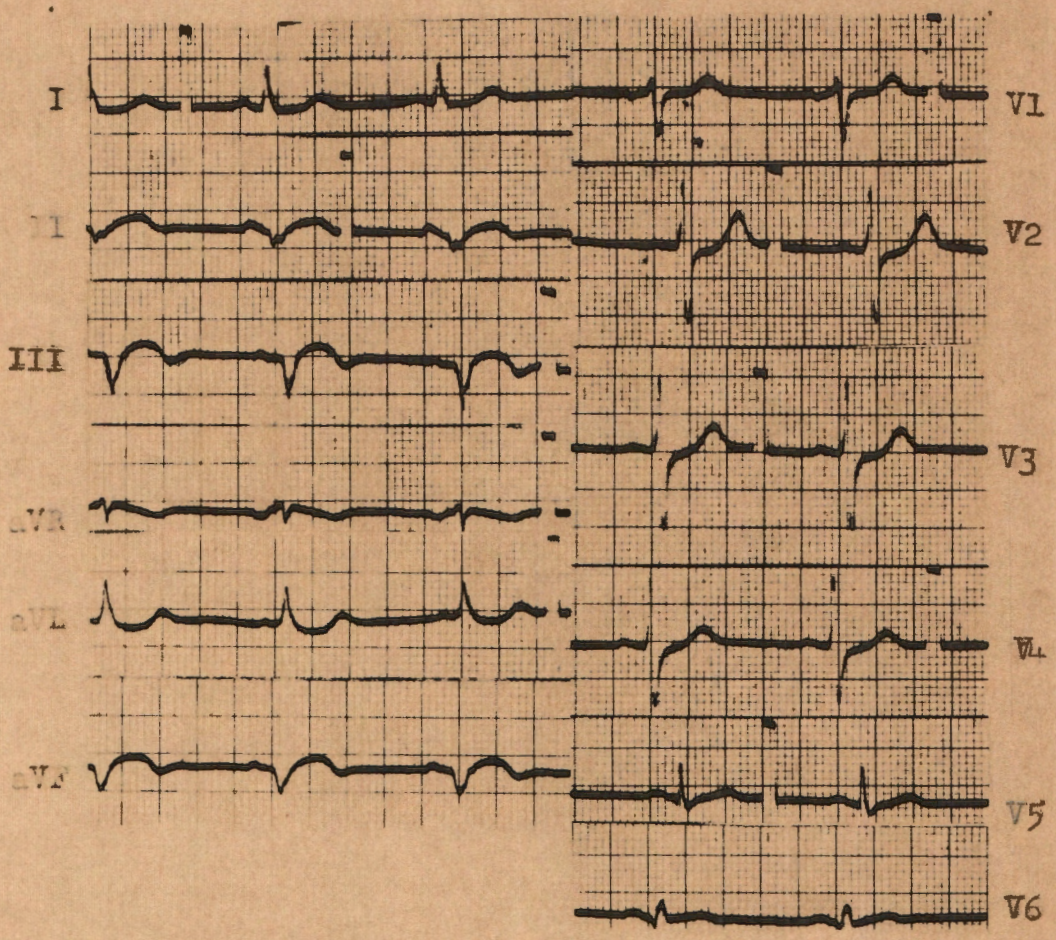


D.



E.

Fig. 18


I
III
V1
V2
V3
V5
V6

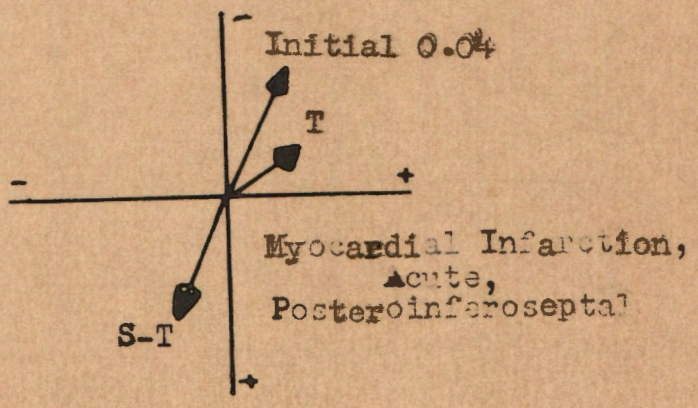
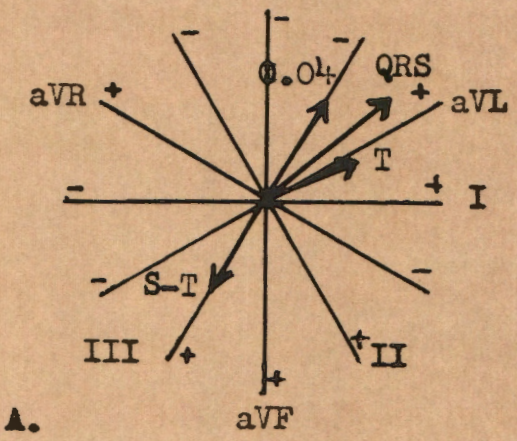
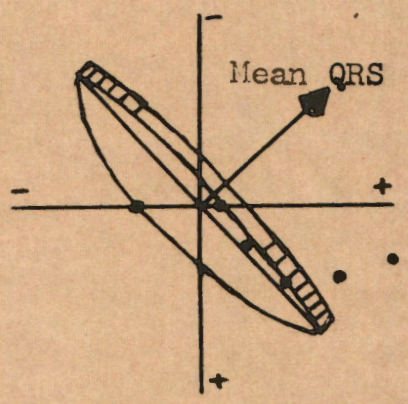

Initial 0.04
T
S-T
Myocardial Infarction,
Acute,
Posteroinferoseptal

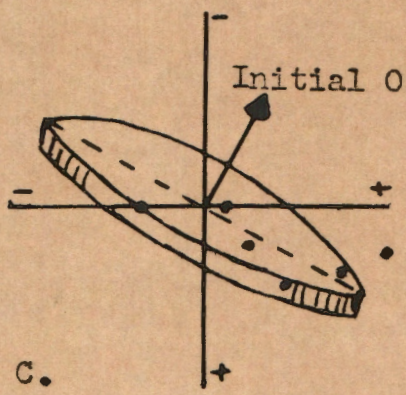
Fig. 19



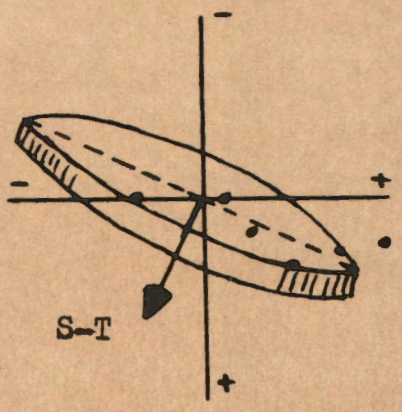
A.



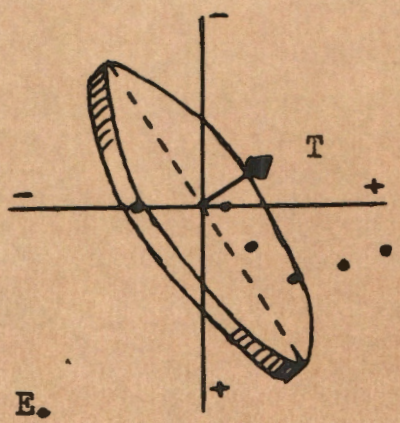
B.



C.



D.



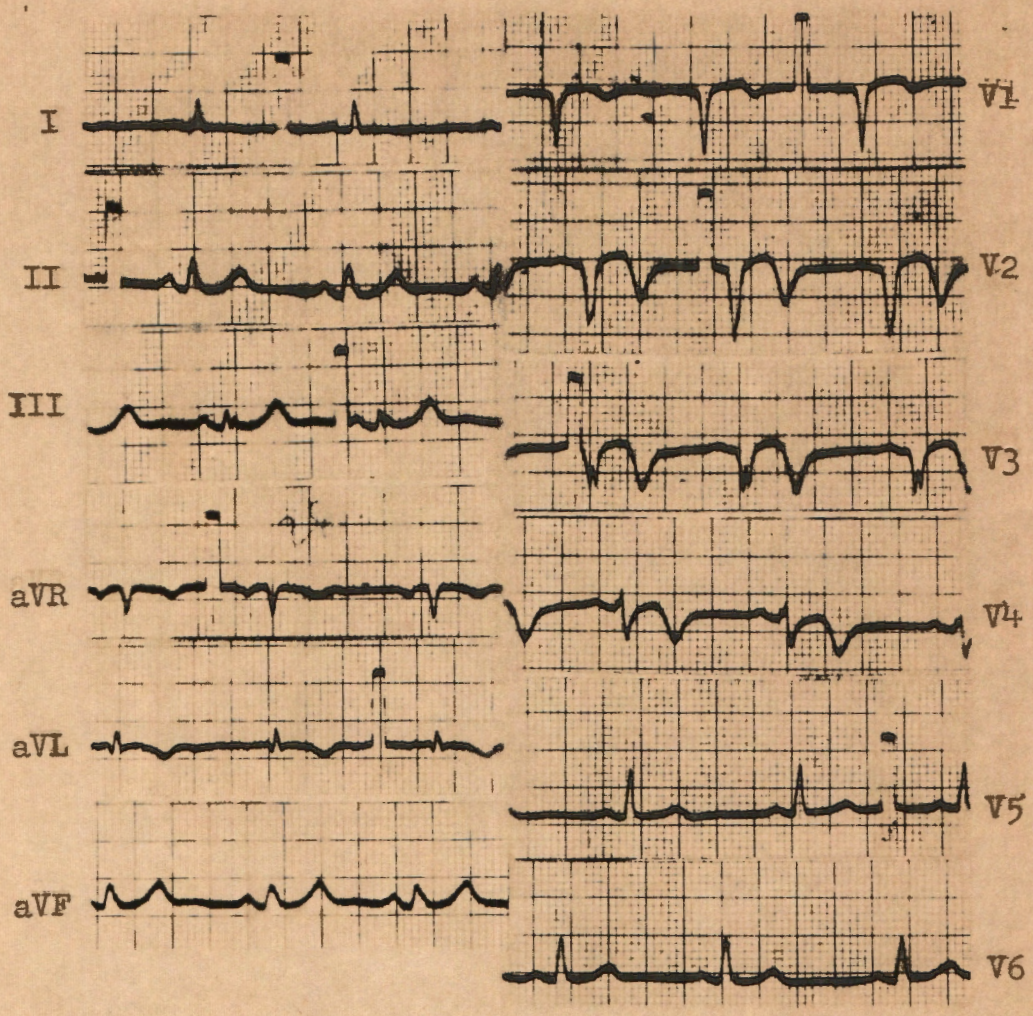
E.

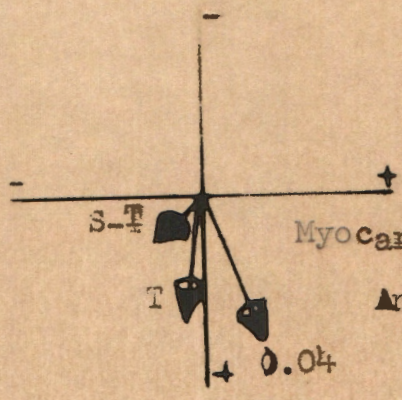
Fig. 20

infarction of the posteroinferoseptal portion of the myocardium.

The last ECG tracing included in this paper for illustrative purposes is shown in Figure 21 with its vector interpretation diagrammed in Figure 22. The first 0.04 of the QRS interval is almost isoelectric in lead aVL, positive in II and isoelectric between chest leads V2-V3. Thus, the initial 0.04 vector is directed inferiorly, posteriorly, and slightly to the left (Fig. 22C). The T wave is isoelectric in I, positive in aVF, and transitional between V4-V5. Therefore, it is directed almost strai ht inferiorly and posteriorly. No S-T segment deviation can be detected in the limb leads. However, there is persistent S-T elevation in the chest leads, so the S-T vector must be pointing almost directly anteriorly.

Thus, the S-T vector is pointing towards the anterior wall, and the 0.04 and T vectors are pointing away from the anterior wall. Therefore, this tracing must represent an acute infarction of the anterior portion of the myocardium. This tracing strongly shows the advantage of using unipolar chest leads in routine ECG recordings. They are





Myocardial Infarction,
Acute,
Anterior

Fig. 21

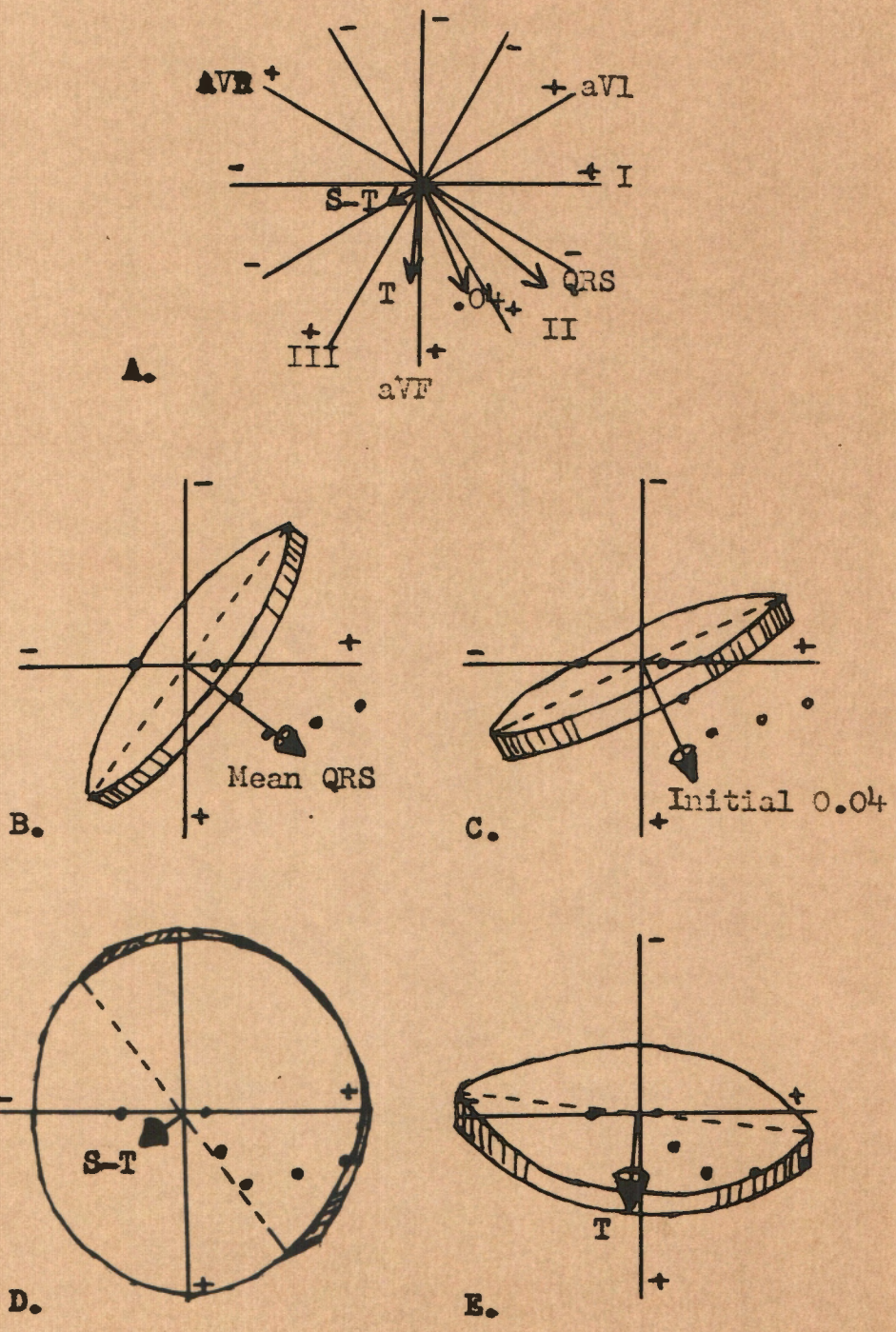


Fig. 22

very useful in recording the electrical activity of the heart in the anteroposterior dimension.

It is hoped that these tracings and their respective analyses have illustrated the mechanics of vector analysis, and their practical application to clinical medicine. The vector method of analysis has its basis in the fundamental principles of physics and the foundations of electrocardiography. It seems only logical to say that analysis by vectors is the key to an understanding of the fundamental activity of the heart as it is recorded by the electrocardiogram.

SUMMARY

This paper is concerned with the electrocardiographic diagnosis of acute myocardial infarction using the method of vector analysis. Two different methods of ECG interpretation exist--by patterns and by vectors. This paper discusses only the vector method and no attempt is made to compare the two methods.

A historical review of the clinical recognition of infarction of the myocardium as a specific entity, as well as the development of the field of electrocardiography, is included. Three men are particularly outstanding in the field of electrocardiography. Einthoven is well known for his original string galvanometer and his fundamental law of the relation of the standard limb leads. Lewis is best known for his theory of limited potential differences, while Wilson made many contributions to the field, including the unipolar limb and precordial leads.

Of necessity, much of this paper is devoted to the discussion of the electrical activity of the heart and how this activity can be recorded by the ECG. The common lead systems and the various reference systems are reviewed. The vector quantity,

its characteristics, and its application to the ECG tracing are thoroughly discussed. The deflection on the tracing is really a projection of the vector onto that lead; it is this fact which makes vectors useful in the interpretation of an electrocardiogram.

The method of determing the position of a vector in space is reviewed, and the importance of the three dimensional electrocardiogram is stressed.

Loss of blood supply to an area of the myocardium results in several changes in the myocardium which are represented by certain changes in the ECG tracing. These changes are:

1. Death - Initial 0.04 QRS vector points away from the site of the infarction
2. Injury - S-T vector points towards the site of the infarction
3. Ischemia - T vector points away from the site of the infarction and tends to parallel the initial 0.04 vector

These changes are basic to an infarction, but the absence of these findings on a tracing from a patient with clinical evidence of infarction does not eliminate the possibility of an infarction.

Several examples are included to illustrate the practical aspects of vector analysis, and how

these principles may be applied to the clinical interpretation of the ECG tracing from subjects with myocardial infarction.

This paper mentions only briefly some of the complications of infarction, and the discussion is limited only to the acute phase of infarction.

CONCLUSIONS

1. The field of electrocardiography stands squarely on the shoulders of Willem Einthoven, Sir Thomas Lewis, and Frank N. Wilson. These three men have made the most important contributions to the field.

2. The electrical potential generated within the heart creates electrical fields throughout the body. These fields extend to the body surface where they can be recorded by surface electrodes.

3. There are twelve leads used commonly in recording electrocardiograms, six limb leads and six precordial leads. A lead system may be either bipolar or unipolar.

4. The electrical potential generated within the heart has the measurable properties of magnitude and direction. Therefore this potential can be represented by a vector or vectors.

5. The deflection on an ECG lead represents the projection of the cardiac vector onto that lead. This is why vectors representing various phases of the cardiac cycle can be determined from the various leads of a tracing.

6. There are three vector abnormalities associated with infarction which correspond to three changes within the heart.

   1. Death - Initial 0.04 QRS vector points away from infarction
   2. Injury - S-T vector points towards infarct
   3. Ischemia - T vector points away from infarct

7. These changes together are found only with infarction of the myocardium, but t eir absence does not rule out the possibility of infarction.

8. The electrocardiogram is useful as a diagnostic aid when considering the possibility of myocardial infarction.

9. The vector method is a useful and practical means of interpretating the ECG tracing when considering the diagnosis of myocardial infarction.

ACKNOWLEDGMENT

I wish to express my gratitude to William D. Angle, M. D. His encouragement, advice, and assistance were of great value to me in the preparation of this paper. I would also like to thank him for his permission to include in this paper ECG tracings from his book, Elements of Vector Cardiography.

BIBLIOGRAPHY

1. Wilson, F. N., Johnston, F. D., and Kossmann, C. E., Interpretation of the Ventricular Complex of the Electrocardiogram, Advances in Internal Medicine, 2:1-63, Interscience Publisher, Inc., New York, 1947.

2. Grant, R. P., Clinical Electrocardiography: The Spatial Vector Approach, McGraw-Hill Book Co., Inc., New York, 1957, Preface, Chapters 1, 2, 4, 6.

3. Gelfand, David, and Bellet, Samuel, Recent Advances in Clinical Electrocardiography, Med. Clinics of N. A., 38:6, Nov., 1954, pp. 1583-1599.

4. Wilson, F. N., and Johnston, F. D., The Vectorcardiogram, Am. Heart J., 16:14-28, July, 1938.

5. Heberden, William, 1768. Cited by: Wilson, F.N., The Electrical Currents Associated with the Heart Beat, Michigan Alumnus Quarterly Review, 47:12-21, 1940.

6. Jenner, Edward. Cited by: Wilson, F. N., Ibid.

7. Dock, George, 1896. Cited by: Wilson, F. N., Ibid.

8. Herrick, J. B. Cited by: Wilson, F. N., Ibid.

9. Kolliker and Muller, 1855. Cited by: Wilson, F. N., Ibid.

10. Waller, 1889. Cited by: Wilson, F. N., Ibid.

11. Einthoven, Willem. Cited by: Wilson F. N. Ibid.

12. Einthoven, Willem. Cited by: Wilson, F. N., (Arch. Internat. d. Physiol. 4: 132, 1906.) The Accuracy of Einthoven's Equation, Am. Heart J., 7:203-206, 1931.

13. Einthoven, Willem, Arch. f. d. ges. Physiol., 122:517, 1908. Cited by Wilson, F. N., Ibid.

14. Einthoven, Willem, Uber die. Deutung des Elektrokardiogramms, Pfluger's Archiv., 149, 1912. Cited by Wilson, F. N., The Origin and Nature of the Process Made in our Understanding of the Electrocardiogram during the Last Three Decades., Selected Papers of Dr. Frank N. Wilson, J. W. Edwards, Inc, Ann Arbor, Mich., 1954.

15. Lewis, Thomas, Interpretations of the Initial Phases of the Electrocardiogram with Special Reference to the Theory of "Limited Potential Differences," Arch. Int. Med., 30:269-285, 1922.

16. Wilson, F. N., The Origin and Nature of the Process Made in our Understanding of the Electrocardiogram during the Last Three Decades; Selected Papers of Dr. Frank N. Wilson, J. W. Edwards, Inc., Ann Arbor, Mich., 1944.

17. Smith, Archiv, Intern. Med., 1918, XXII, 8. Cited by: Wilson, F. N., MacLeod, A. G., Barker, P. S., Johnston, F. D., and Klostermeyer, L. L., The Electrocardiogram in Myocardial Infarction with Particular Reference to the Initial Deflection of the Ventricular Complex., Heart, 16-155-199, June, 1933.

18. Pardee, Archiv. Intern. Med., 1926, 26:244. Cited by: Wilson, F. N. and others, Ibid.

19. Parkinson and Bedford, Heart, 1927-29, 14:195. Cited by: Wilson, F. N. and others, Ibid.

20. Levine and Brown, Medicine, 1929, 8:24-5. Cited by: Ibid.

21. Fenichel and Kugel, Amer. Heart Journ., 1931-32, 7:235. Cited by: Ibid.

22. Barnes and Whitten, Amer. Heart Journ., 1930, 5:142. Cited by Ibid.

23. Wilson, F. N. and others, The Electrocardiogram in Myocardial Infarction with Particular Reference to the Initial Deflection of the Ventricular Complex, Heart, 16:155-199, June, 1933.

24. Wilson, F. N. MacLeod, A. G. and Barker, P. S., The Order of the Ventricular Excitation in Bundle Branch Block, Am. Heart. J., 7:305-330, 1930.

25. Wilson, F. N. Johnston, F. D., MacLeod, A. G., and Barker, P. S., Electrocardiograms that Represent the Potential Variations of a Single Electrode, Am. Heart J., 9:447-458, April, 1934.

26. Wilson, F. N., Johnston, F. D., Cotrim, N. and Rosebaum, F. F., Relations between the Potential Variations of the Ventricular Surfaces and the Form of the Electrocardiogram in Leads from the Precordium and the Extremities., Assn. Am. Physicians, 56:258-271, 1941.

27. Wilson, F. N., Johnston, F. D., Rosenbaum, F. F., Erlanger, H., Kossman, C. E., Hecht, H. H., and others, The Precordial Electrocardiogram, Am. Heart J., 27:19-85, 1944.

28. Wilson, F. N., and others, Old Einthoven's Triangle, the Theory of Unipolar Electrocardiographic Leads, and the Interpretation of the Recordial Electrocardiogram, Am. Heart J., 32:277-310, 1946.

29. Bryant, J. M., Johnston, F. D., and Wilson, F. N., Unipolar Electrocardiographic Leads. Effects Produced by Eliminating the Resistors between the Limb Electrodes and the Central Terminal, Am. Heart J., 32:321-332, March, 1949.

30. Einthoven, W., The Different Forms of the Human Electrocardiogram and Their Signification, Lancet, March 30, 1912.

31. Bayley, R. H., Fundamental Relations of the Instantaneous Electrical Axis of Cardiac Accession, M. Times, New York, 67:558, 1939.

32. Bayley, R. H., Biophysical Principles of Electrocardiography, Paul B. Hoeber, Inc, New York, 1958, Chapter 3.

33. Sodi-Pallares, Demetrio, and Calder, Royall, New Bases of Electrocardiography, The C. V. Mosby Co., St. Louis, 1956, Chapter 6.

34. Grant, Robert P., and Murray, Raymond H., The QRS Complex Deformity of Myocardial Infarction in the Human Subject, Am. J. of Med., 17:587, 1954.

35. Wilson, F. N., Hill, I. G. Q., and Johnston, F. D., The Form of the Electrocardiogram in Experimental Myocardial Infarction. I. Septal Infarcts and the Origin of the Preliminary Deflections of the Canine Leuocardiogram, Am. Heart J., 9:596-614, June, 1934.

36. Scher, A. and Young, A., The Pathway of Ventricular Depolarization in the Dog, Circulation Res., 4:461, 1956.

37. Wilson, F. N., and others, The Electrocardiogram in the Later Stages of Experimental Myocardial Infarction, Trans, Assn. Am. Physicians, 48:154-163, 1933.

38. First, S. R., Bayley, R. H., and Bedford, D. P., Peri-infarction Block; Electrocardiographic Abnormality Occasionally Resembling Bundle Branch Block and Local Ventricular Blocks of Other Types., Circulation, 2:31, 1950.

39. Bayley, R. H., Vector Analysis of the T-Deflection of the Electrocardiogram, Am. Heart J., 50:844, 1955.

40. Angle, William D., Elements of Vectorcardiography, University of Nebraska College of Medicine, Postgraduate Medicine, Feb., 1961.