1951

Spatial vectorcardiography : its relationship to the electrocardiogram

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SPATIAL VECTORCARDIOGRAPHY
ITS RELATIONSHIP TO THE ELECTROCARDIOGRAM

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Submitted in Partial Fulfillment
for the Degree of Doctor of Medicine
College of Medicine, University of Nebrasks
December 20, 1951
Omaha, Nebraska

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INTRODUCTION

There are two ways to learn electrocardiography. The first method is based on the memorization of electrocardiographic patterns, and is largely empirical because the actual pathology of the heart cannot be determined until autopsy, which cannot often be accomplished, and which can never be done simultaneously with the recording of the heart tracing. The second method is to base the interpretation of the ECG on a thorough understanding of the nature of the genesis of the recorded electrical currents or potentials. This last method is theoretical, but with its theorems abstracted from verifiable phenomena and capable of being expressed mathematically.

The purpose of this paper is to adequately define the theoretical method so that electrocardiograms, both scalar and spatial, may be interpreted by reason and be utilized to explain the specific pathology of the patient uncolored by previous empirical electrocardiographic patterns from other patients with perhaps somewhat similar pathology.

HISTORY

Galvani (22) first discovered bioelectric currents and developed the instrument which bears his name for their measurement.

Waller, in 1898, (49) measured the action currents of the heart with a capillary electroscope. Einthoven, (20, 21) improved upon this so that more accurate measurements of these currents could be undertaken. At any one instant the electromotive force of the heart may be represented by a number indicating magnitude. Einthoven gave an immense stimulus to the investigation of electrical phenomena in the heart when he conceived of the electrical axis of the heart as a vector force and showed the relationship of the electromotive force to the three standard bipolar limb electrodes by a system of polar coordinates on the frontal plane of body which was represented by an equilateral triangle but it was Mann (41) who conceived of representing the instantaneous electromotive forces by a system of rectangular coordinates, thus giving them not only magnitude but direction. These forces may thus be shown on the frontal plane as a number of scalar vectors with their length indicating magnitude and their arrowheads, direction. Mann connected the termini of these vectors with a curved line, producing a loop which he called the monocardiogram.

Later the true nature of these vectors and their loop was shown by Wilson (58) who gave spatial direction to them when he devised the equilateral tetrahedron (based

on the three standard limb-leads with a fourth electrode on the back) which is the geometrical figure representing these electrode positions.

Although Mann (42) developed an electrical system for recording monocardiograms by a cathode ray oscilloscope, no more progress in visualizing this loop beyond his two dimensional monocardiogram occurred for a number of years. Thus the only way in which the spatial nature of the loop could be visualized was mathematically and geometrically by wire models.

Finally a system using three cathode ray oscilloscopes, each recording from a different plane, was perfected by Duchosal and Sulzer (16) which by superimposition enabled accurate visualization of the loop in space, or if viewed separately on two or more planes enables an accurate mental construction of the loop in space.

Action Currents and Resting Potentials

One of the properties of life is the ability to react to stimuli which property is called irritability. Whenever a cell is stimulated the chemical processes which occur in it as a result of stimulation generate electrical currents. These electrical currents are called action currents.

There are two ways of measuring these electrical

phenomena. One is by the galvanometer which records action currents, and the other is by the oscilloscope which records the electrical potentials associated with the current. The results by either method are similar.

The electrodes recording these action currents or potentials (the terms are interchangeable) may be placed directly upon the cell, near the cell, or at a distance from the cell. Or one electrode (called the indifferent electrode) may be placed at a distance and the other (the exploring electrode) may be varied in distance so as to be direct or indirect. Indirect positions may be semi-direct or distant.

When two electrodes are used which are electrically equidistant from the heart and both record different potentials (as a result of their positions in respect to the electrical field), they are called bipolar electrodes -- bipolar meaning they each have a different polarity or potential.

If one of the electrodes is connected to the three standard bipolar electrodes, with appropriate resistances as shown in Figure 1a, the sum of the electrical forces recorded from these three positions will be zero (according to Kirkhoff's second law). This electrode is

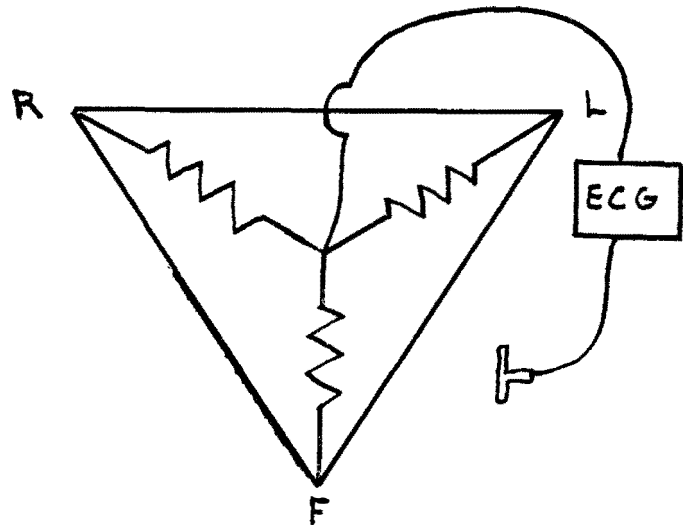

Fig. 1 a. Unipolar electrode connections.

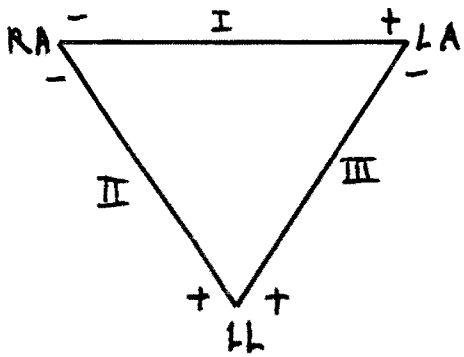

Fig. 1 b. Einthoven's Triangle: standard bipolar limb leads.

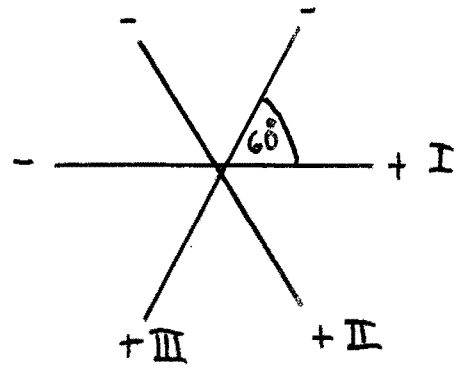

Fig. 1 c. Standard triaxial reference system.

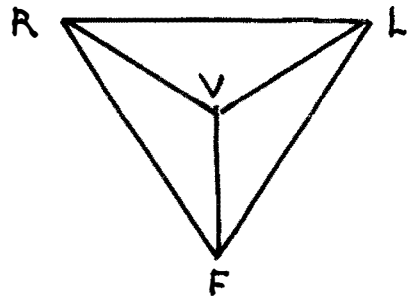

Fig. 1 d. Einthoven's Triangle: unipolar limb leads.

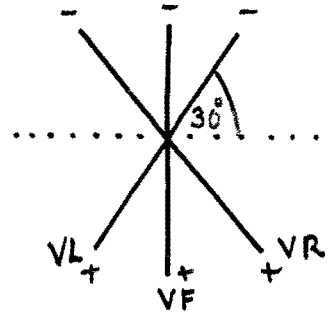

Fig. 1 e. Unipolar triaxial reference system.

called, as before, the indifferent electrode and is indicated by the symbol V, while the exploring electrode is called, in this case, the unipolar electrode. If it is placed on any extremity it is called an unipolar limb lead. While if it is placed on the chest it is called a precordial lead.

In electrocardiography the galvanometers are so arranged that a positive deflection is recorded as an upward deflection of the base line, and the various bipolar limb positions are so recorded that in Lead I the left arm is positive, and in Lead II and III the left leg is positive. As seen on Einthoven's triangle it appears as shown in Figure 1b. It is better pictured on the triaxial reference system, which is derived from the triangle by transposing the three lead axes so that a point on the center of each axis is superimposed in all cases and so that the direction of the axes is preserved, as shown in Figure 1c.

Of course in the case of the unipolar electrode, the unipolar electrode is considered to be positive in relation to the indifferent electrode.

It is to be noted that this system of polarity is used only in reference to the triaxial reference systems which represent a transposition of the various limb

electrodes as explained above. The standard triaxial reference system refers to one constructed from the three standard bipolar limb electrodes, while the unipolar triaxial reference system refers to one constructed from a unipolar electrodes recording successively from the right arm (R), the left arm (L)and the left foot (F). It is to be noted that an electrode may be placed anywhere upon an extremity with no change in its recorded potential. They are therefore considered electrically equidistant from the electrical center of the heart no matter where placed on the extremity. Therefore, the system of notation while different in the standard and unipolar refers to identical positions. Thus RA = R, LA = L, LL = F in the standard and unipolar systems respectively.

The unipolar limb electrodes with their indifferent electrode are designated by a combination of the separate electrode symbols, thus VR, VL, VF. If these electrodes are augmented by the introduction of a transformer so as to magnify their potentials they are called the "augmented unipolar extremity leads" and are designated by the symbols AVR, AVL, AVF.

The unipolar limb electrode positions are shown electrically equidistant by a frontal plane isoceles

triangle similar to the Einthoven triangle, except that, since the indifferent electrode is zero for all three electrode positions, its geometrical position must be located in the center of the triangle. The leads are then pictured as shown in Figure 1d, each terminating in the electrical center or zero point of the field.

The unipolar triaxial system will be constructed from a transposition of the lead lines entirely similar to the method before mentioned. It will appear as in Figure 1e. It is similar to the standard system except it has been rotated 30^{o} in a clockwise direction.

The oscilloscope which utilizes a cathode ray is only different from the galvanometer in that it records action potentials instead of action currents, and that it is more accurate since it has practically no inertia.

In clinical electrocardiography only indirect electrodes can be used to record the electrical activity of the heart. Of the indirect electrodes we have considered the standard bipolar and unipolar limb and precordial electrodes. However there are as many indirect electrode positions as there are points on the body surface, besides some within it, i.e., intratracheal, esophageal, gastric, and intracardiac. For the most part such accessory leads are entirely unnecessary when one knows the

nature of the electrical field.

We have stated that action potentials or currents are caused by chemico-electrical changes which occur during activity of the cell, and we have discussed the manner in which these electrical changes are measured by indirect electrodes. It should be added that action potentials are of two types: that due to normal cellular activity, and that due to currents of injury.

It must not be thought that the cell is constantly active in the sense defined above. There are periods of rest necessary for regeneration of energy. The potentials or currents recorded from resting cells are called resting potentials or currents, and these are recorded by electrodes as periods of isopotentiality. The resting potential will shift its base line if it is taken with one electrode internal and the other external as compared to its former position recorded with both electrodes external.

In Figures 2 and 3 are shown the normal and injured resting potentials and action currents. These records are those recorded from the sciatic nerve of a frog, (50) and from the squid giant axon (13) by direct electrodes. The latter would be similar to those recorded

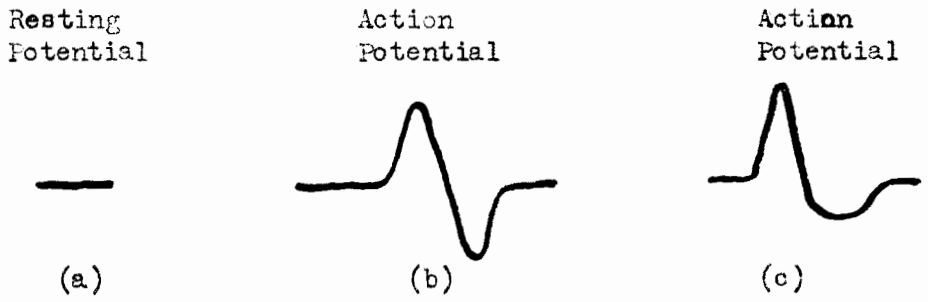

Fig. 2. Parts (a) and (b) are diagrammed from records taken from the uninjured sciatic nerve of the frog. Part (c) is taken from the uninjured squid giant axon. (See text).

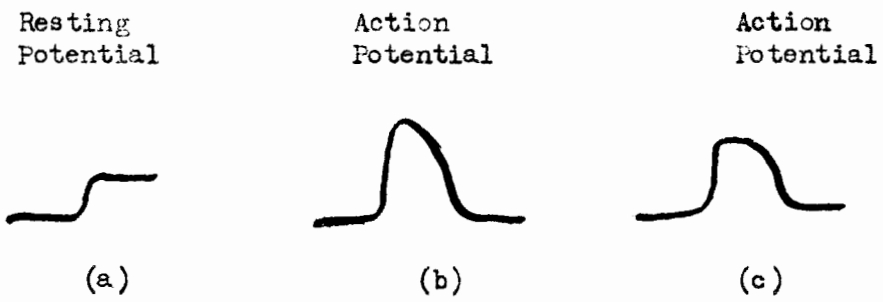

Fig. 3. Parts (a) and (b) are diagrammed from records taken from the injured sciatic nerve of the frog. Part (c) is recorded from the injured squid giant axon. (See text).

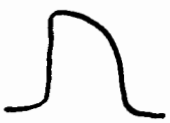

Fig. 4. Injured action potential from heart taken with direct electrodes.

with the same leads from a single heart cell, if that were possible. Injured action potentials recorded from a portion of the heart comprised of many cells with directo electrodes appear as in Figure 4. The appearance of normal and injured action potentials as recorded from the heart with indirect electrodes is very similar to the above except that the negative phase of the biphasic wave (inverted portion) is now positive (the T wave).[1] This is due to the fact that the regenerating force in the heart takes the opposite direction from that in the nerve. If the regenerating force is made to take the same direction as that in the nerve it appears as in Figure 5.

Thus the biphasic wave appears to be the normal action potential wave, and the monophasic wave appears to be that due to a current of injury. Such observations led Hoff, Nahum, and their collaborators (33, 34) to state that the biphasic wave was composed of two oppositely oriented monophasic action potentials from the right and left side of the heart respectively. Thus a current of injury would be due to improper functioning

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[1]The appearance of the normal and injured action potentials in the human heart may be seen in Figure 31 above blocks A and D.

of one-half the heart, or in a single cell to diminished or complete inactivity of one-half the cell. While the latter statement is true for one cell the former statement for the heart is not only a serious oversimplification (5) but is actually false, for while it may serve to explain certain facts it fails entirely to explain others.

A misunderstanding of what an injured area is led to a further conflict between cardiologists and physiologists when they attempted to explain the monophasic wave (50, 59). In order to understand the nature of this conflict we must consider the current of injury in more detail.

CURRENTS OF INJURY

The current of injury is that current due to the difference in potentials occasioned by an injured area. It now becomes necessary to explain precisely what is meant by an injured area. An injured area is one where the activity of the cell (comprising depolarization and repolarization) is tardy in relation to the normal, and where there is also a sizeable reduction in the number of electrically active constituents (dipoles) which would ordinarily be present during the normal cycle of activity.[2] The causes of injury are many, chief of

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[2]The processes of depolarization and repolarization will be discussed in detail in a later section.

these being ischemia (6).

The patterns of the resting and injured action potentials in nerve and heart have already been given. It remains for us to discuss the manner in which the direct electrodes, which recorded these waves, were applied.

In Figure 2 the electrodes in parts a and b were direct and place a few centimeters apart upon the surface of the uninjured sciatic nerve. In Figure 3a and 3b the end of the nerve was injured and one electrode inserted in that end. The other electrode was on the external uninjured surface. Again the electrodes were a few centimeters apart. In Figure 3c and Figure 3c the electrodes were direct, but in 2c one was placed inside the cell and the other was directly opposite it upon the surface. The cell was not injured. In Figure 3c one electrode was on the uninjured surface and the other was a few millimeters away on the injured end.

From these considerations it can be seen that direct electrodes may be classified as: (a) uninjured cell: both external and apart; and one external, the other internal and directly opposite; (b) injured cell: both external and apart; and one external, the other internal and directly opposite. The direct external

electrodes for injured cells may have the electrode in the injured end either placed on the surface of the injured area or inserted just within it. The results for either case will for all practical purposes be entirely similar.

When the nature of the electrical processes which occur at the cell membrane is understood one can reason out for himself the pattern of the recorded wave. The following diagram shows what occurs in heart muscle. The situation is probably entirely similar in nerve except that nerve being more irritable repolarizes at a faster rate. It is this slower repolarization in heart muscle which accounts for the S - T segment.

As will be explained in greater detail below the most satisfactory method for visualizing the nature of the electrical changes which occur on the surface membrane of the cell, is to picture this membrane as being composed of transverse dipoles, each dipole being comprised of a negative and a positive pole and arranged with their long axis perpendicular to the cell membrane. (See Figure 6). It is only necessary to consider one side of the cell in this description. These dipoles are arranged so that during rest they have their positive poles directed outward from the center of the cell.

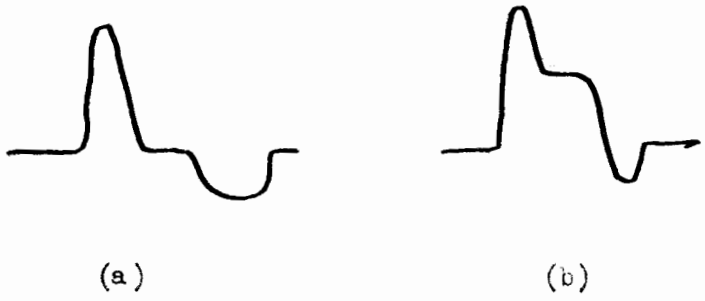

Fig. 5. Normal (a) and injured (b) action potentials from heart taken with indirect electrodes.

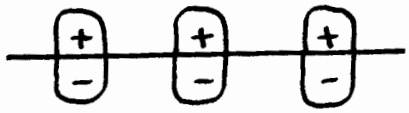

Fig. 6. Dipole on resting surface membrane.

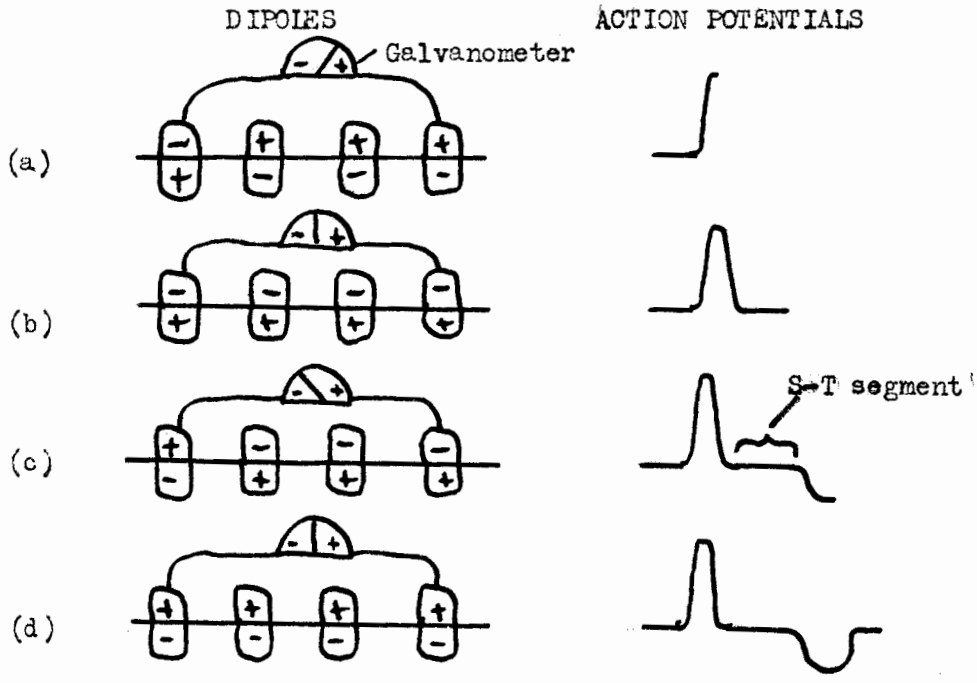

Fig. 7. Action currents from an uninjured heart cell where both electrodes are external and apart. Parts (a) and (b) show depolarization; (c) and (d) show repolarization. Here the process of repolarization is in the same direction as depolarization. It may be seen that this is probably how Figures 2b and 5a were inscribed.

When a stimulus is applied at one end of the cell, the dipole at that end reverses its direction and this reversal sets up a "chain reaction" which travels in both directions completely around the cell until all the dipoles are reversed (depolarized). After a short time (which in the heart is recorded as the S - T segment) the dipoles, if they repolarize in the same direction as they depolarized, again reverse their position beginning at the point where stimulation was first applied and again proceeding on around the cell in an orderly succession but at a somewhat slower rate. This is what happens in heart muscle. In nerve, being more irritable, the process of repolarization commences immediately with each dipole as soon as it has depolarized, so that at no time is the entire cell depolarized at once, as in the heart.

Now, to consider the relation of the various direct leads to these processes, we see that the galvanometer will record the electrocardiographic patterns. (See Figures 7 and 9). (Note that a positive deflection of the galvanometer is recorded as an upward wave.)

The derivation of the normal and current of injury action potentials in the actual ECG recorded by indirect leads from the heart will be diagrammed later.

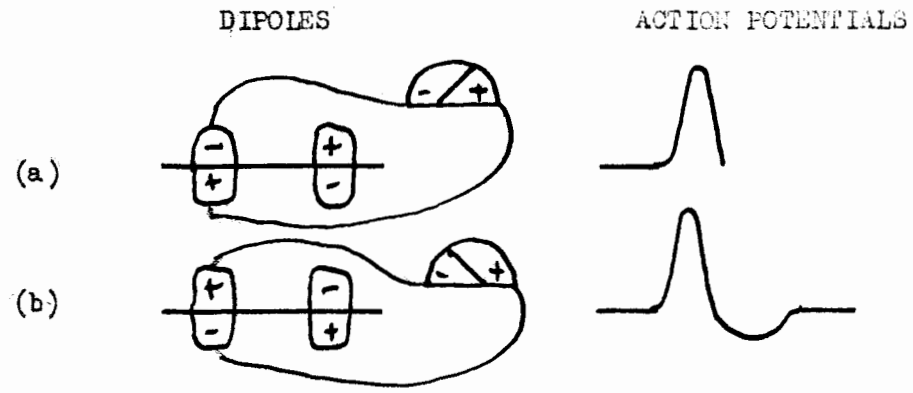

Fig. 8. Action currents taken from an uninjured nerve cell where one electrode is external and the other internal and opposite. Part (a) is depolarization, and part (b) is repolarization. It may be seen that this was probably how Figure 2 c. was inscribed.

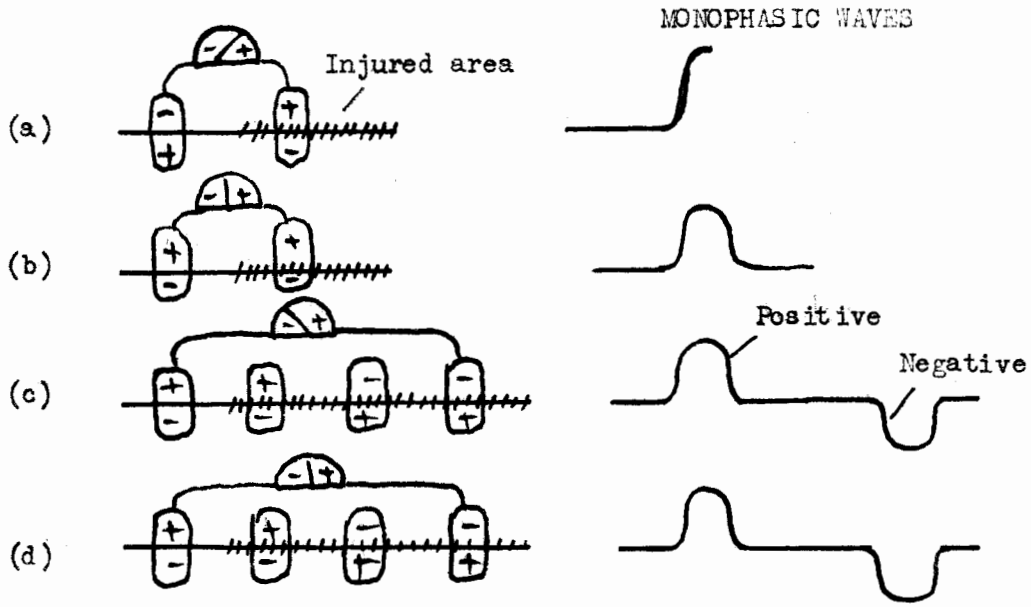

Fig. 9. Action currents in injured cell, both electrodes external and apart. In (a) and in (b) the electrodes are close; in (c) and (d) they are far apart. See the definition of an injured area in the text. If the injured area were killed there would be no negative monophasic wave recorded. If the electrodes were placed with one external and the other internal and opposite in the uninjured portion of the injured cell the record would be as in Figure 8.

It may be seen from these diagrams that if in the uninjured cell where direct external electrodes were used which were far enough apart on the cell, a monophasic wave would result followed by another monophasic wave in the opposite direction. If these electrodes were close enough a biphasic would result. The same holds true for an uninjured cell where direct electrodes, with one internal and the other external, are used.

Thus to return to our previous discussion, the monophasic wave is not necessarily due to a current of injury. However, for all practical purposes it is impossible to obtain a single heart cell so elongated as to allow the electrodes to be far enough apart. Furthermore, in the nerve cell, the fact that the process of repolarization succeeds immediately that of depolarization for each dipole, precludes the possibility of recording physiological normal monophasic waves from such a cell. Therefore, to all intents and purposes the monophasic wave is always due to current of injury. Similarly the normal wave of the intact dipole is always biphasic. The same holds true for the heart muscle cells when (as stated above) the process of repolarization is made to take the same direction as the process of depolarization.

Thus, in partial rebuttal to Hoff, Nahum et al (33, 34), by definition a monophasic wave is a wave of injury, and therefore, a biphasic wave, which by definition is a physiologically normal wave, can never be composed of two waves of injury, i.e., of two monophasic waves. A further rebuttal to this concept will appear in the discussions to follow concerning repolarization, depolarization, and ventricular gradients.

In the heart pure monophasic waves are difficult to record (35, 50) even with direct electrodes because the electrodes usually pick up potential variations in other parts of the heart as well as that immediately beneath the electrodes. The farther one gets away from the heart, as with indirect electrodes, the more likely will be the electrodes to record such variations in other parts of the heart.

Some investigators thought that the precordial electrode since it was so much closer than limb leads was practically a direct electrode. Others preferred to call it a semi-direct electrode, as they thought it was mid-way between direct and the more distant indirect limb leads. In reality the precordial electrode is both a direct and a "spatial electrode". (A spatial electrode is one which is remote enough from generating tissue to

be recording relatively equally from all parts of the heart, as is the case with limb leads). Thus Wilson and his co-workers (59) recognized that to treat precordial leads as direct or as spatial leads is largely a matter of interpretation, depending upon the purposes of the study, either interpretation might be valid. While Grant (27) states that "strictly speaking there is no such thing as a perfectly direct lead as far as the intact heart is concerned. Even with the electrode placed directly on the surface of the heart it records potentials from remote regions of the heart as well as from the region with which it is in contact." Yet, the same thing holds true for direct electrodes on one cell for not only do they record from the dipole to which they are immediately adjacent, but they also record from other dipoles nearby. Therefore, to quote Grant again, "indeed, no matter how remote on a radius from the heart a recording electrode is placed, it is probable that the deflection will always resemble in contour a direct lead deflection taken on the same radius." This matter will be further discussed under intrinsic deflections.

To return to the conflict between the cardiologists and physiologists over the origin of the monophasic

wave, the cardiologists were"of the opinion that the monophasic curve obtained by leading from an injured region on the epicardial surface (V electrode) to a point distant from the heart (the common terminal) represented potential variation of the injured region and not the latter (common terminal). This assumption was considered rank heresy by the physiologists who were accustomed to thinking of an injured region as electrically indifferent (59)." Therefore, the conflict is a matter of definition since it is to be noted that the physiologists' "injured area" is in reality a killed area. Thus they recorded only one monophasic wave as compared to the two opposite monophasic waves which would be recorded from an injured area. The precordial electrodes of the cardiologists may be treated as a direct electrode which is placed over the injured area, while the common terminal (although a spatial electrode) may be treated in this instance as another direct electrode placed on resting muscle (since its potential is zero). Thus the situation as far as electrodes are concerned is identical, and the general contours of the waves should be similar if one takes into account the different direction which repolarization takes in the heart. If they are not it can only be due to the fact

that one area is killed and the other injured. If this latter is the case both groups are correct in their assumptions that in each case different areas are responsible for the potential variations.

Theories As to the Modes of Genesis of Activity

It now becomes our task to take up the subject of the theories as to the modes of genesis of the action potential, and to consider secondarily, the genesis of the resting potential. We shall first consider the purely electrical phenomena.

There are two criteria by which any such theories must be considered. First, is the theory in accord with the electron theory? Second, does it enable prediction of electrical effects which can be verified by experimental results?

The electron theory supposes that for all practical purposes an atom of matter in the neutral state is composed of an equal number of positive and negative charges known as protons and electrons. When an atom loses an electron, it becomes positively charged; when it gains an electron it becomes negatively charged; and a continuous flow of electrons forms a current. It follows that for every quantity of positive electricity, there is an equivalent amount of negative electricity.

In view of this conception of the electrical field, it seems necessary that any theory which attempts to explain the genesis of the action potential, which is an electrical phenomenon, must consider the presence of

both positive and negative charges; which moreover, would apparently have to be in close proximity during some stage, if not throughout the entire duration, of the action potential.

The earliest conclusion as to the nature of the electrical processes occurring in excited muscle to gain any general measure of approval was the "negativity hypothesis" of DuBois-Reymond (15) and Bernstein (7). Lewis (40) called this theory the "hypothesis of distributed potential differences." According to this view, that part of the muscle which is active is negative, while the whole remainder of the muscle is relatively positive in relation to the negative area. This theory implies that in determining the direction of the electromotive force, you are to join the center of the active muscle to the center of the inactive mass.

Now, in analyzing this and the subsequent theories to follow, we might consider three possible divisions of the heart muscle, i.e., the cell, the muscle strip composed of many cells, and finally the intact heart. But since it is held that the heart being a syncitium may be considered to behave electrically like one large cell (9, 35); and, since, therefore, we may consider a portion of this large cell as being similar to a

portion of heart muscle, we need only consider the large hypothetical muscle cell and its portions in this analysis.

Thus to diagram the negativity hypothesis (Figure 10), this theory requires that only negative forces be shown in the active part of the cell, while the remainder of the cell is neutral and therefore relatively positive. The theory definitely does not say that the remainder of the inactive cell is positive in charge. Therefore if we consider one end of the cell to be the base and the other the apex, and connect the two by a vector indicating the electromotive force we have two possibilities, i.e., either the negative charge is within or without the membrane. Since it is now known that only the surface membrane is involved in these electrical processes (14, 17) we need not consider the cytoplasm of the cell as ever carrying any charge. Since we further know that the wave of activity may be considered, in general, to pass from the base to the apex of the heart, we may therefore have considered the "wave of negativity" to have passed from the base to the apex at the stage in which the cell is pictured. Unless there is some defect in the membrane the wave should spread evenly from the base in all directions over the cell as

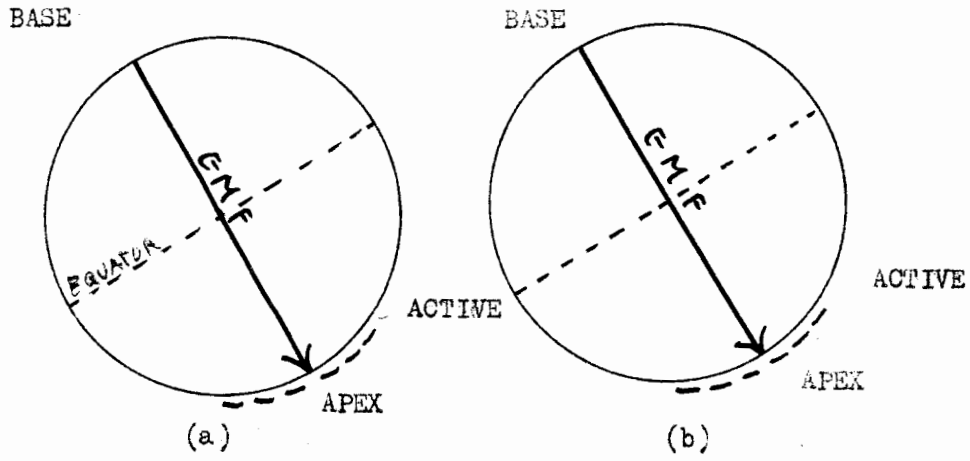

Fig. 10. Negativity Hypothesis.

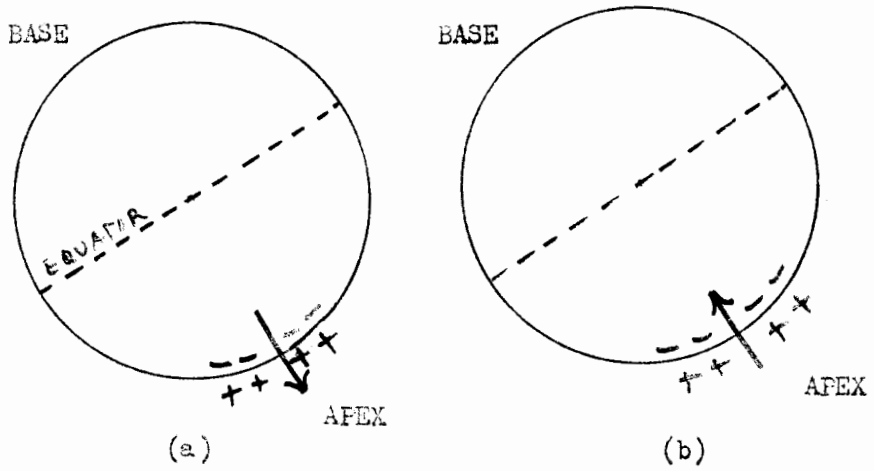

Fig. 11. Craib's Dipole Theory.

a circular band to meet at the apex. Thus it is apparent that the electromotive force completely reverses its direction during the process of excitation, pointing first towards the base and last towards the apex, and that when the band of excitation is at the equator there are two equal and opposite electromotive forces which cancel each others electrical effect.

Craib (10) criticizes this theory from two standpoints, first the electron theory is not satisfied because there must be an equal positive charge present, and secondly, "galvanometric records obtained from one lead directly applied to excited cardiac tissue with the other electrode at a distance may show positive deflections from the direct lead." This is incompatible with Bernstein's view (7). Furthermore, as will be shown later, the direction which the electromotive forces actually take is exactly the reverse of that shown by this theory.

The next theory which was advanced was the "limited potential difference hypothesis" of Lewis (40). Lewis found his experimental results difficult to reconcile with the negativity hypothesis of his predecessors and was compelled to advance the view that in cardiac muscle excitation at any given point results in a double elec-

trical effect. The actual point excited exhibits "relative" electrical negativity and the tissue immediately adjacent, "relative positivity". This theory is different from the foregoing in that it postulates two closely adjacent electrical charges of opposite sign. The wave of excitation is similar in its mode of spread to the "wave of negativity" except that it is composed of two electrical waves -- the foremost positive and the hindmost relatively negative. No specific statement is offered as to the duration of positivity or negativity at each point excited, nor as to the nature of the wave of recovery. Craib (10) criticizes this theory on the grounds that Lewis implied that each cell of heart muscle in the excited positive area was entirely positive while its adjacent cell in the negative area was entirely negative. Craib held that such a separation of positive and negative forces was too distant for the electron theory, and that further, the theory is incomplete as regards the wave of repolarization. Both these criticisms are valid, but in addition this theory would result in a shifting null line (see below) which is incompatible with the facts. (This last criticism also holds for the negativity hypothesis.)

Craib (10) then advanced his "doublet or dipole hypothesis" which specifically defines the excitation doublet as existing in each element excited for a very short time. He does not attempt to explain just what this element is, but clearly implies that it is smaller than a cell, the wave of excitation thus gives rise to what may be described as a succession of temporary doublets. The electrical counterpart of the "wave of recovery" (repolarization) is defined as a further succession of doublets each of which occurs in a given element as soon as the wave of recovery reaches it, and lasts a relatively prolonged period of time, "possibly even waxing and waning in regard to its intensity, and differing further from the previous dipoles in that its orientation is reversed." "The wave of recovery thus follows a course identical to that of excitation and with an equal velocity, but gives rise to prolonged electrical effects and associated galvanometric deflections in which the sharp angles and rapid deflections of the wave of excitation are replaced by the rounded curves due to a slowly moving (galvanometric) string."

Craib (10) defines an "electrical doublet" or "dipole" as two near poles situated in a conducting medium and maintained at equal and opposite potentials.

These poles when connected by a straight line having a magnitude defined by the absolute potential difference between them and a direction as determined by their position in space, constitute a vector representing the electromotive force. Current will flow from the positive to the negative pole through all the regions of the medium, and will continue to flow while there is a difference of potential and so long as the medium remains a conductor.

If we overlook Lewis' implication that the different poles were in two different adjoining cells we arrive at Craib's dipole theory as far as the wave of depolarization is concerned. The method of spread of the entire wave of depolarization when applied to a single large cell representing the intact heart follows the same pattern of spread as previously defined in preceding theories. Figure 11a shows this process at a late stage of depolarization. When the wave of repolariation occurs we have the situation at a late stage as shown in Figure 11b. Here, as Craib states the wave of repolarization has proceeded in an identical direction as the wave of depolarization, i.e., from base to apex.

Considering this theory by the first criteria of

the electron theory, we can see that it is entirely satisfactory. Considered by the second criteria there are only two criticisms of it in the light of modern experimental findings concerning the heart. First, if the wave of repolarization should follow the same path as depolarization there would be no ventricular gradient. This is contrary to fact. Secondly, if the waves spread from pole to pole leaving unexcited areas behind them, the null lines of the QRS and T waves would shift. This they do not do. (See below).

We must now consider the resting potential because in attempting to explain its presence the dipole theory as originally advanced by Craib has been altered.

Curtis and Cole (14) while not the first to measure the resting potential have given us some very accurate measurements. They found with direct electrodes, in an uninjured cell, with one electrode external and the other internal, that this potential in the squid giant axon has a value of about 50 mV. Other measurements with direct electrodes one of which was placed on or within a killed end and the other on the external uninjured surface have shown that the external resting surface of the axon is positively charged while the internal surface is negatively charged.

Now from Craib's experiments, as well as the observations of others, it is apparent that during depolarization there is a passage of a wave of negativity which is immediately preceded by and associated with a wave of positivity. Hence one is led to think of the passage of a series of sources (/) and sinks (-) or dipoles in a longitudinal direction along the cell membrane.

These considerations led Wilson to modify Craib's original doublet theory so that he showed the resting cell with a number of dipoles arranged along its surface having their positive poles without and their negative poles within. (See Figure 12). These may be considered as inactive dipoles whose charge is yet maintained during the resting state so as to account for the difference of potential in the resting cell between its internal and external surfaces, which amounts to 50 mV.

Wilson pictures this cell to undergo a serial alteration of the membrance so that the active dipole instead of having its axis (represented by a line from one pole to the other) located in a transverse direction to the membrane, as shown by Craib, has its axis located in a longitudinal direction to the cell membrane (52, 59). The area immediately behind the active

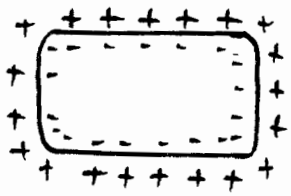

Fig. 12. Resting dipoles: transverse orientation

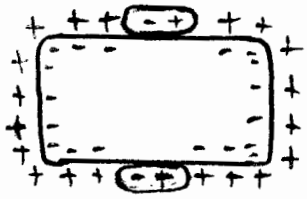

Fig. 13. Active dipoles: Wilson's longitudinal orientation

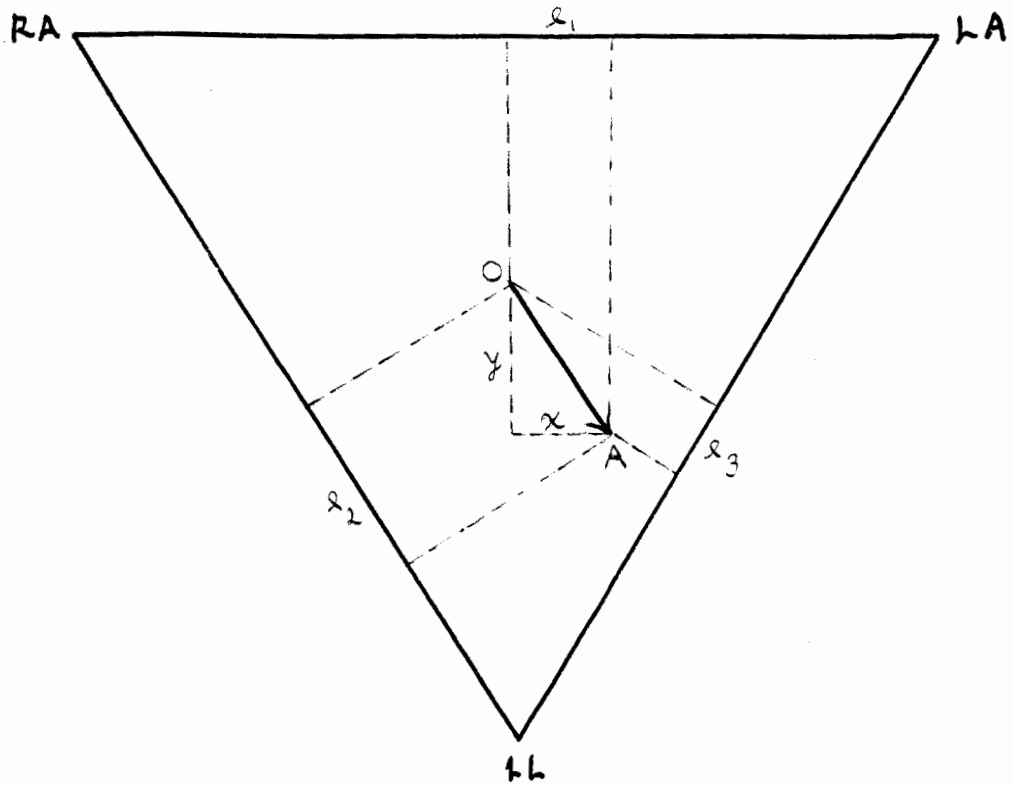

Fig. 14. Geometrical relationship of the electrical axis (AO) to the manifest potentials (ℓ). (See text).

dipole reverts to its former state. This would appear as in Figure 13, with the direction of serial depolarization indicated by the arrow (50). We may call this theory the longitudinal dipole theory, while Craib's original theory will be called the transverse dipole theory.

This theory must be criticized from at least two standpoints. First, as with all the preceding theories such a process of depolarization would result in a shifting null line (see the "null line" below). And second, it is apparent that in such a cell, the positivity never exceeds that of a resting section.

However, a number of investigators (10, 14, 31) have shown that the positive potential preceding negativity rises above that of the resting surface potential. Curtis and Cole state that during the passage of an impulse the membrane potential is momentarily reversed in sign, so that the outside may be as much as 110 mV negative with respect to the interior. This fact throws doubt on the simple explanation of the action potential as a passive depolarization of the membrane or abolition of the resting potential. Referring to their record of the depolarization of the squid giant axon as shown in Figure 2c, one can see the

record of such a dipole. This positivity is called the "overshoot" and Huxley attempted to explain it by means of four possibilities to which may be added a fifth. (1) A selective permeability may develop for anions formed in the axoplasm as a result of activity. (2) A change in orientation of theoretical dipoles across surface membranes may develop. (3) Inductance of the membrane due to a piezoelectric effect may occur. (4) Inductance may cause a reversal of polarity if it is permissible to regard the resting electromotive force as in series with membrane capacity instead of being parallel to it as is generally supposed. (5) The release of acetylcholine may augment action potentials produced by passive depolarization of the membrane.

In considering the first of these possibilities, i.e., that a selective permeability may develop for anions formed in the axoplasm during activity, Hodgkin and Huxley (31) state that his is unlikely since it is hard to imagine the motility of lactate or any other organic ion could be sufficient to swamp the contribution of potassium and sodium to the membrane potential. As concerns the mobility of these last two ions, it has long been known that in the resting cell the potassium concentration is great within and small without. The

reverse holds true for the sodium ion. Hodgkins and Katz (32) show that during activity there is not only a migration of potassium ions out of the cell, but there is also a migration of sodium within.

According to McIntyre (43) this process might be due to an alteration of the lipid and water phases in the cell membrane so that during the period of increased permeability the water phase becomes continuous whereas during rest the lipid phase is continuous. This would allow the migration of these ions which might be explained as being due to the law of diffusion. This migration would explain part of the potential, but as McIntyre states, some further mechanism is necessary to account for the total potential change. The mechanism by which the lipid and water phases become altered is as yet not agreed upon.

Considering the second possibility it seems clear that there is definitely a reversal of the dipoles as hypothesized by Hodgkins and Huxley and Curtis and Cole, but this reversal in itself, while present and associated with the changing potentials of the biphasic wave, will perhaps only account for the return of the positive and negative wave to isopotentiality thus exhibiting a neutralizing effect (see below) but not accounting for the

actual positive and negative potentials. If we accept the migration of potassium and sodium ions as contributing to part of these potentials, we have yet to consider the additional factor contributing to the total potentiality.

This additional factor is probably the inductance of the membrane. Curtis and Cole (14) thought this might be possibly due to a piezoelectric effect, but since they could not conceive of a mechanism to explain such inductance they were reluctant to accept this explanation. However, Dunn (17) has shown that theoretically the cell could possess the properties of inductance, capacitance, and resistance gives excellent evidence to support this contention that inductance is indeed the additional factor.

As concerns the fourth possibility Dunn (18) states it is difficult to believe that the resting electromotive force could be in series with the capacitance. While surges can be produced in pure capacitance-resistance circuits, no oscillatory discharges can be set up, thus the biphasic wave cannot be accounted for by this concept.

In relation to the possible mechanism by which the membrane of the cell might become permeable to the

migration of potassium and sodium ions, Nachmansohn (45) suggests that the increased permeability and its associated fall in resistance is due to the release of acetylcholine and its effect on the lipoproteins of the membrane. He states that in muscle, and perhaps nerve, the source of energy for the synthesis of acetylcholine is derived from the release of energy-rich phosphate bonds by adenosinetriphosphate and that phosphocreatine is probably the source of energy for the resynthesis of adenosinephosphate to adenosinetriphosphate. In turn the energy required for the resynthesis of phosphocreatine would be derived from the oxidative breakdown of pyruvic acid, adenosinetriphosphate again acting as the catalyst.

While Bremer (8) objects to this theory on the basis that acetylcholine is not present in lower forms of life, he states that "comparative biochemistry offers instances of substitution of different substances" so that the objection is not decisive.

The resting cell of the heart has a surface membrane which is composed of elements called dipoles. These dipoles are perhaps large lipoproteins, but no matter what their chemical composition they possess a positive charge at one end and a negative charge at the

other. These charges are constantly maintained in the living cell by the flow of electrons from one pole to the other. The charges are oriented during rest so that the positive pole is away from the center of the cell and the negative pole is directed towards this center.

The negative pole attracts positive ions, but since potassium has a higher place in the electromotive series than sodium, it has a greater affinity for this pole than sodium, which it displaces if present. The potassium partially neutralized the negative charge, but since the electron transfer is a constant process, the negativity of that pole is never completely neutralized. This accounts for the resting potential, and possibly for auto-excitation which would be due to a spontaneous reversal of dipoles occasioned by the necessity to discharge the everincreasing potential difference of the two dipoles.

The other end of the dipole being positive probably attracts some anion such as chloride.

Upon release of acetylcholine either the flow of electrons from the positive to the negative pole is reversed, or the entire element is reversed. The former more probably occurs, but the mechanism is unknown. Therefore, momentarily there is no current flow.

During this period of time the potassium and chloride ions exert a force which is unbalanced and accounts for part of the "overshoot" potential of Curtis and Cole (13). The reversed pole of the dipole quickly builds up its charge and contributes to the rest of the positive charge.

At this stage the positive pole of the dipole is oriented towards the center of the cell. Thus it repels the potassium ion. A similar repelling force is exerted by the now external negative pole upon the chloride ion. Thus these forces as well as the law of diffusion cause the migration of potassium outward and chloride inward. Besides these forces we probably have to consider the law of diffusion which likewise affects these ions and others. It seems reasonable, therefore, to suppose that sodium migrates into the cell by diffusion to replace potassium and is accompanied by chloride. Also by diffusion, bicarbonate would migrate out to replace chloride. With the migration of these charges the potential should be neutralized. Thus the positive wave returns to the base line.

It is not necessary to think of the water and lipid phase as being altered by acetylcholine to account for the migration of these ions, but this theory does

not exclude that possibility.

The negative charge of the dipole which faces outward from the center of the cell, before it neutralized probably favors the further synthesis of acetylcholine which in turn affects those dipoles adjacent to it, and so on until all the dipoles in the cell are reversed. At this point most of the intracellular potassium should be extracellular.

Finally, after all the dipoles have been neutralized and thus the negativity of the cell has vanished, and before it can continue to build up its charge by a continuing electron flow, the acetylcholine synthesis is no longer favored and with its breakdown the entire process reverses to its former state. This accounts for the negative phase of the biphasic wave.

The mechanism of depolarization and repolarization will be discussed in greater detail and diagrammed.

The Laws of the Electrical Field of the Heart

In 1913 Einthoven (21) gave an immense stimulus to the investigation of the electrical phenomena associated with heart muscle when he defined the electrical axis of the heart and demonstrated the use of the equilateral triangle in its derivation. He based his method on the assumption that the source of the electrical

field in a large homogeneous conducting equilateral triandle, which is taken to represent the three standard limb leads on the frontal plane of the body, consists of two near points, situated at the center of the triangle and showing a potential difference, constituting a dipole. He represented the potential difference between these points by a straight line of given length and passing through the points in question. It is assumed that the differences between the potentials at the apices of the triangle under these conditions is proportional to the perpendicular projections of this line onto the sides of the triangle. The potential difference at the center is treated as if it were a vector quantity subject to vector analysis. This constitutes the Einthoven Triangle Theory.

It must be understood that although the equations thus developed may be true in experience, their validity depends on pure empiricism unless it can be shown mathematically that the electrical field under the conditions described will in fact conform with the assumptions of Einthoven's Triangle. Next it must be demonstrated that the potential difference between the dipoles may be considered as a vector quantity for the purposes of determining the potential difference

between equidistant points in the associated field on the human subject, or under suitable experimental conditions (10).

The electrical axis may be determined geometrically from the Einthoven Triangle in the following manner. Draw the triangle. (See Figure 14). Represent the potential differences on the three sides by lines of appropriate length (e) measured from a point determined on each side by a line drawn perpendicular to that side and passing through the electrical center of the heart (point O). The direction of the vector (AO), representing the mean electromotive force or the electrical axis, is found by point (A) where perpendiculars meet drawn from the other end of the manifest potentials (e).

The value of the abcissa (x) is that of e, but if one wishes to find the value of the ordinate (y) by mathematical means he must divide the algebraic sum of leads II and III by the square root of 3,

$$y = \frac{e_2 + e_3}{\sqrt{3}} \qquad (41).$$

Before considering the Einthoven Triangle Theory in more detail, we shall discuss Kirkhoff's Second Law which is derived from Ohm's Law and upon which the Einthoven Equation is based. Kirkhoff's Law states

that the sum of the products of the current by the resistance taken around any closed path in a network of conductors is just equal to the sum of the electromotive forces which one passes in going around the closed circuit. The sum of the current by the resistance is the voltage. In going around the circuit regard must be had for the direction in which the current is flowing. Currents which are flowing in the direction in which the circuit is traced out are positive, while those flowing in the opposite direction are negative. In more general terms, this law states that whenever we go around a closed loop, of any shape, and return to the starting point, the net amount of work done is zero. This is true for any number of stopping points above two. In the electrocardiograph the sum of the manifest potentials (e) from the three standard leads equals zero.

$$e_1 + e_2 + e_3 = 0$$

If we reverse the sign of the work done between any two points, then obviously this work will equal the sum of all the other work done. This is exactly what is done in electrocardiography. The attachments to the galvanometer are reversed for lead II, and therefore e_2 is arbitrarily given a negative sign and we

obtain:

$$e_1 - e_2 + e_3 = 0$$

or $$e_1 + e_3 = e_2 \qquad (30).$$ This last is Einthoven's Equation.

We shall now discuss in greater detail the assumptions of the Einthoven Triangle Theory. These are: (1) The electrical activity is assumed to originate from a single dipole located at the center of the body. (2) The body is assumed to be a cylindrical homogeneous volume conductor. (3) The right arm, left arm, and left leg are assumed to be electrically equidistant from the electrical center of the heart. (4) The heart and the three extremities are assumed to lie on the same plane of the body, namely the frontal plane (24).

Canfield, in an appendiz to Craib's paper (10), in discussing the mathematical properties of dipoles has shown that dipoles may be summated according to the laws of vector analysis, so that a number of dipoles present in a limited space, such as the heart, will give rise to a single resultant dipole so far as their combined effect at a distance is concerned. Such a dipole is pictured in Figure 15. The deduction is therefore justified, that if the resultant effect of a number of electrically active muscle elements may be

summated so as to be considered a single dipole, then each element may be considered as contributing an elementary dipole to the total field (10, 59).

The first assumption also states that this single dipole is located at the center of the body. Katz (35) objects to this assumption when he states that the heart is anatomically eccentrically located, but Grant (25, 26) makes it clear that the distribution of the electrical field in space is expressed by laws which are different from those governing anatomic distance and structure.

In the second assumption the body is assumed to be a cylindrical homogeneous volume conductor. This subject has been a point of controversy for years, but recent theoretical and experimental studies in the human subject leave little doubt that this assumption is reasonably valid (10, 24, 27, 37, 59).

In the third assumption the right arm, left arm, and left leg are considered to be electrically equidistant from the electrical center of the heart; thus, they constitute an equilateral triangle which, in the fourth assumption, is assumed to lie on the frontal plane of the body.

Craib (10) has shown that in a volume conductor

the amplitude of a deflection varies inversely with the square of the distance from the dipole at the center of the conductor to the recording electrode. This means that beyond a certain distance from the center of the field, further displacement of the electrodes will result in negligible further change in the deflection amplitude (26). In the human subject this distance has been found to be 10 to 12 centimeters from the heart (51). Therefore, electrodes more remote than this from the heart are in effect electrically equidistant from the heart, no matter what their anatomic remoteness. Conversely, at such an electrode position, all parts of the heart are electrically equidistant from the electrode (26). These findings confirm both the third and fourth assumptions as to the limb leads. And since the general contours of the deflections become constant at electrode positions much less than 12 centimeters from the heart (51), the contour of a deflection and its resultant electrical sign as recorded by precordial leads, as well as limb leads, reflect the mean electrical activity of the heart in the extrinsic components of the deflection (26, 27).

These premises permit one to apply to the problem of precordial leads, as well as limb leads, the physical

laws governing the distribution of an electrical field arising from a vector at the center of an homogeneous cylindrical volume conductor.

This brings us to a discussion of the mathematical and physical laws which prove that the electrical field does conform to the assumptions of the assumptions of the Einthoven Triangle Theory.

Canfield in an appendix to Craib's paper (10) has given mathematical justification for the use of Einthoven's formulae in the determination of the electrical axis of the heart. Using Laplace's equation based on Kirkhoff's Second Law he devised formulae which will give the electrical field set up by a doublet placed at the center of a conducting medium of definite size and spherical form.

It is not difficult to see that the body may be considered to be a sphere if one remembers that any electrode over 10 to 12 centimeters from the heart may be considered electrically equidistant no matter what its anatomical distance, and that, if the extrinsic deflections are considered, even precordial electrodes may be similarly pictured. From such a sphere it is easy to see how the circle on the frontal plane as shown in Figure 15 may be derived, and it is further apparent

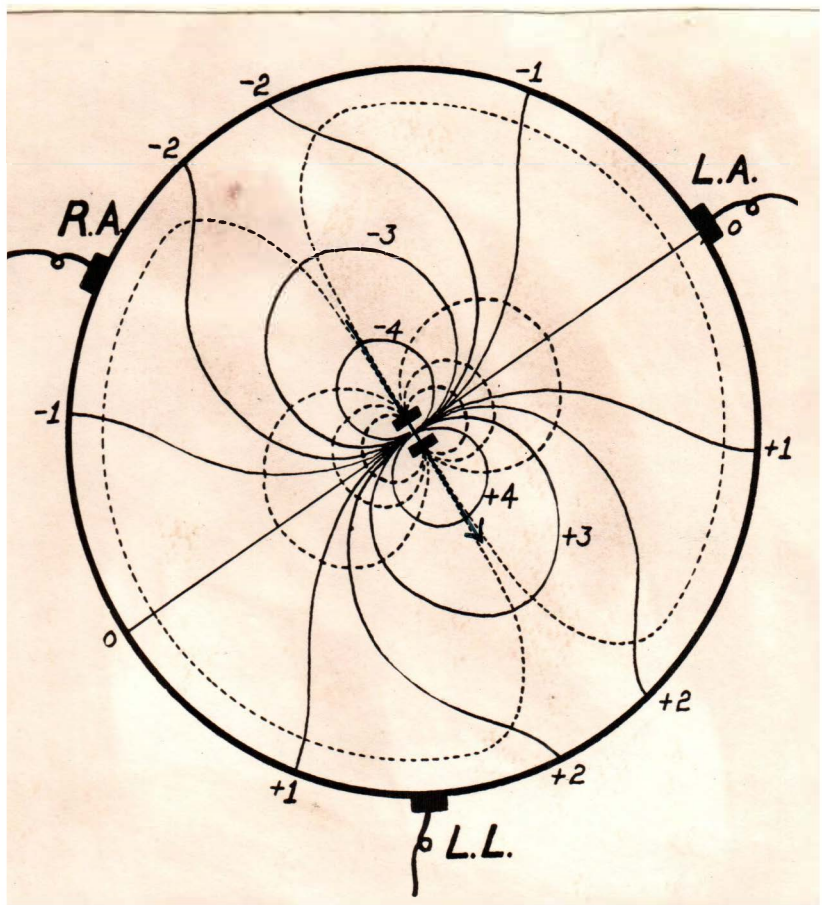

FIG. 15.—Diagram representing the electrical field in a circular homogenous medium resulting from a single bipole (solid rectangles) placed in its center. The solid lines indicate lines of equal potentials; the broken lines, the lines of current flow. The figures on the equipotential lines give their values in arbitrary units. RA, LA, and LL represent three electrodes (with their lead wires) placed at the apices of an equilateral triangle. (Katz, Am. Heart J., 13, 17, 1937.)

that from such a circle an equilateral triangle may be constructed. However, in the determination of the size and direction of resultant electrical forces in space it actually will not matter what geometrical figure is used provided it resembles the electrical relationships of the heart to the equidistant electrode positions and to the two or more planes in the body which they define. (25). Thus, the sphere (10), the equilateral tetrahedron (58), the cube (28), the cylinder (26), and other figures have been used (48). Nevertheless, the chest proves more similar to a cylinder with the heart at its center, than to any other geometrical form, as stated above.

From the sphere Canfield showed that the electrical potential for any point within or on the surface of the volume conductor may be determined by the following formulae derived from a central dipole as shown in Figure 16.

$$V = 2V_o \text{ ab } \cos\theta \left[\frac{1}{r^2} + \frac{2r}{R^3} \right]$$

At the surface of the volume conductor the potential is determined by:

$$V = K \cos\theta$$

Where $K = 2V_o \text{ ab } \left[\frac{1}{r^2} + \frac{2r}{R^3} \right]$, and is constant over the surface of the bounding sphere.

To the cylinder Grant (26) showed that the precordial

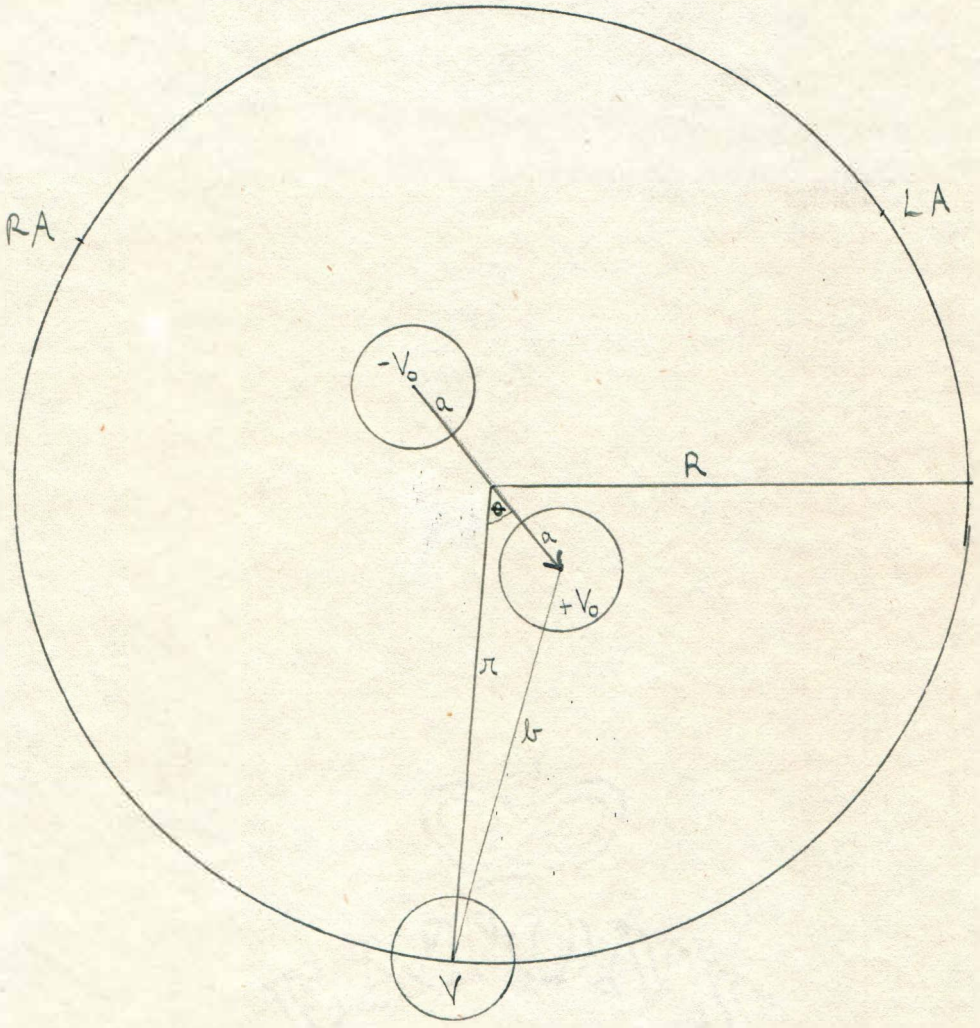

Fig. 16. Spherical volume conductor and central dipole.

θ = angle between r and the line joining the centers of the dipoles.
V = potential at the surface of the volume conductor.
V_o = potentials at the negative and positive poles.
a = radius of each spherical pole.
b = distance between the recording electrode and the positive pole.
R = radius of the spherical volume conductor.
r = radius between recording electrode and electrical center.

electrodes could be used to determine the distribution of an electrical field arising from a vector at the electrical center by the use of the "null line." In such a field, a plane of zero isopotentiality extends from the center of the vector to the surface of the cylinder, separating the surface into an area of positive potential on one side of this plane and negative potential on the other side. This line is shown in two dimensions in Figure 14, and in three dimensions in Figure 17, and is called the null line. Its position is determined by the direction, but not by the magnitude, of the vector at the center of the cylinder. It can be calculated for a cylinder, then, if the direction of the vector and the size of the cylinder are known. Or this calculation can be reversed, and the direction of a vector at the center of a cylindrical volume conductor can be determined if the distribution of positive, negative, and null potentials on the surface of the cylinder are known.

The null lines of the cylinder are analagous to the pathways of the transitional QRS and T complexes of the chest which record null or zero potentials since their deflections have as much upright as inverted components.

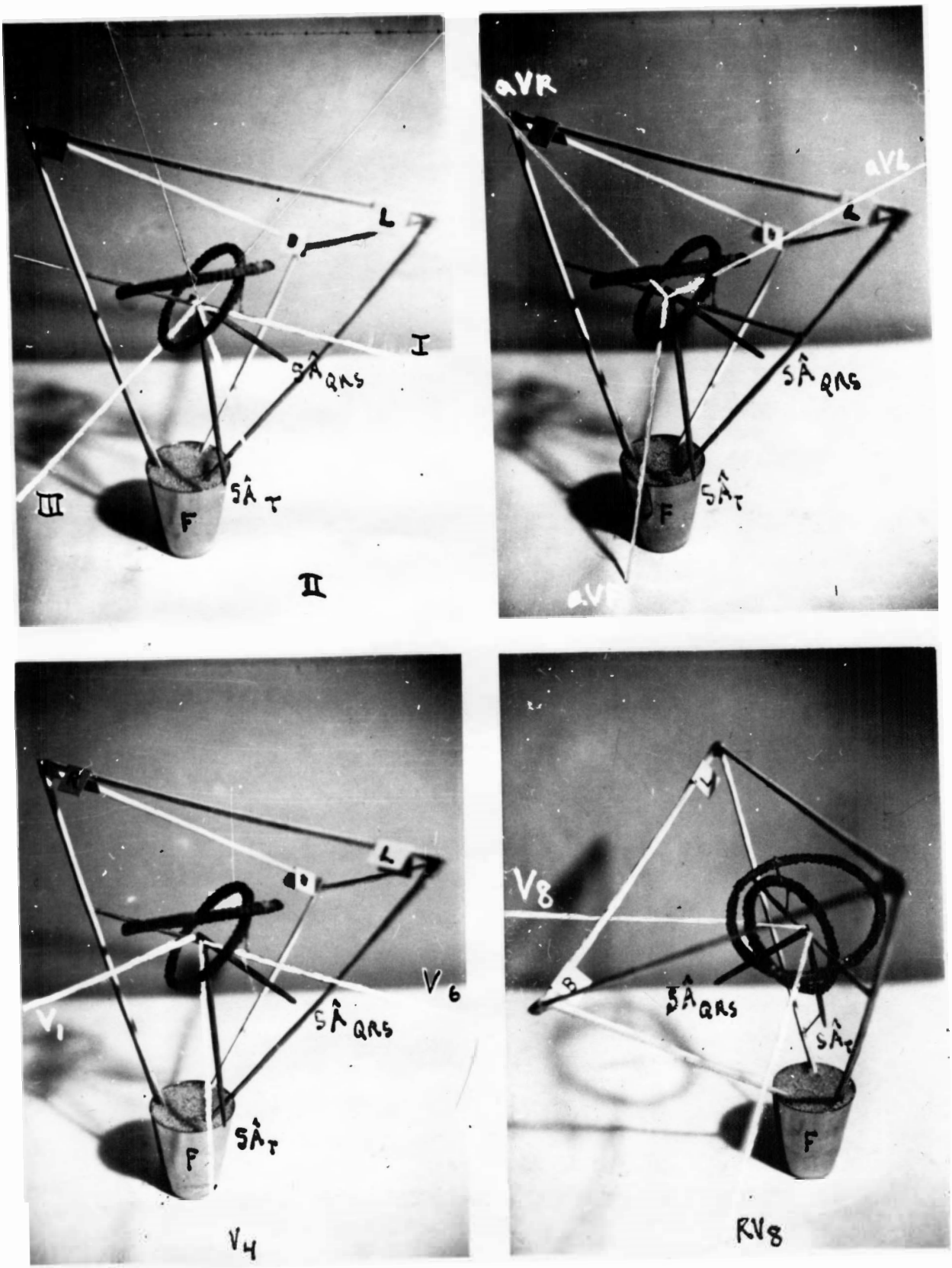

Fig. 17. Spatial model of QRS and T-vectors with their null lines in equilateral tetrahedron. Positive electrode axes are shown. The last picture is a sagittal view; the others frontal views. (See text).

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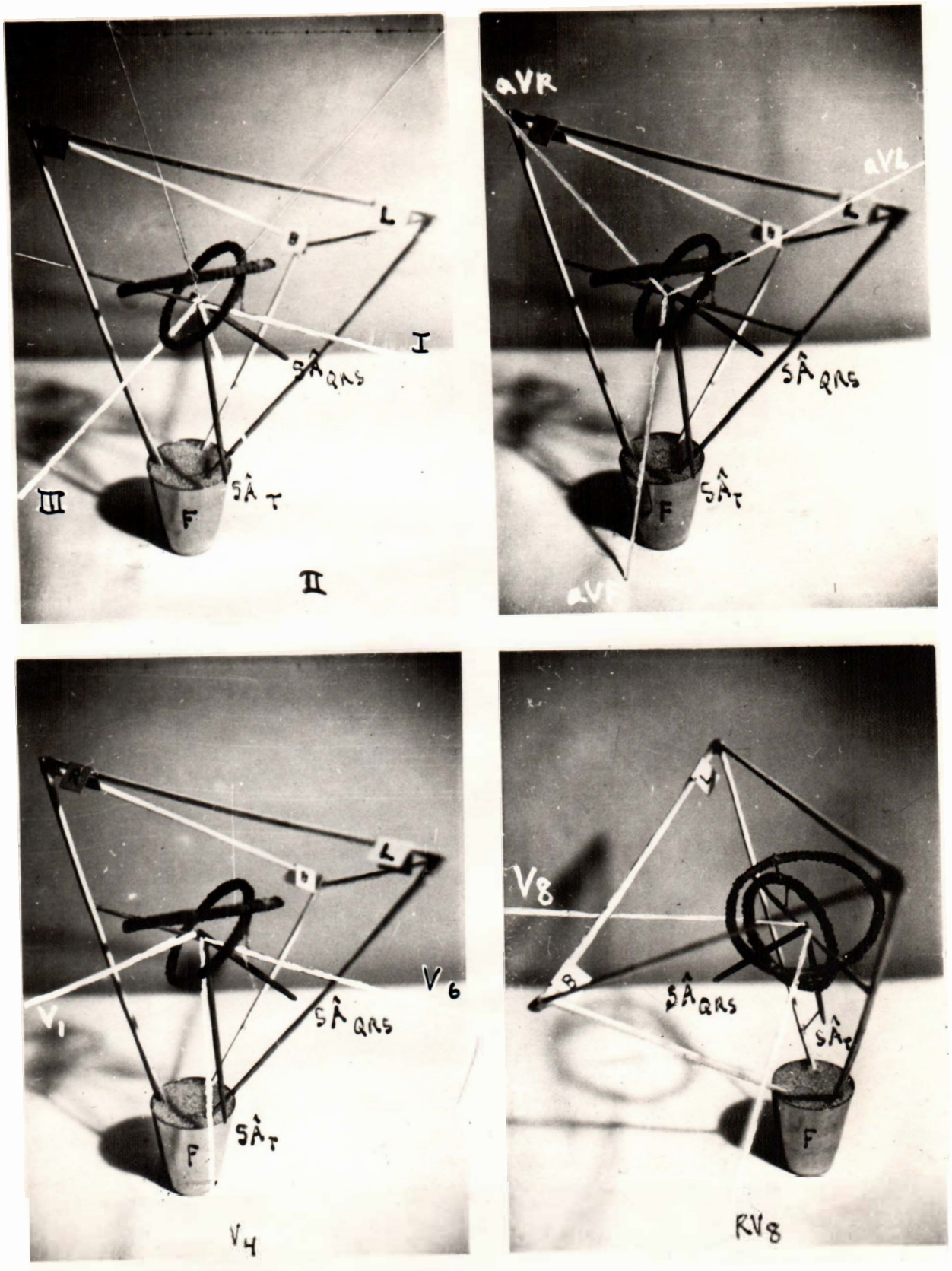

Fig. 17. Spatial model of QRS and T-vectors with their mull lines in equilateral tetrahedron. Positive electrode axes are sho n. The last picture is a sagittal view; the others frontal views. (See text)

For the counterpart of the vector at the center of the cylinder, a method is available for calculating the magnitude and direction of the mean QRS and T electrical forces in the three-dimensional space in the human subject with reasonable accuracy from the electrocardiograms (25, 58).

These mathematical formulae show that the electrical field does conform with the assumptions of the Einthoven Triangle Theory. Finally, it must be demonstrated that the dipoles vector quantity does actually determine the potential difference between equidistant points experimentally.

Goldberger (24) gives experimental proof for the human subject showing that the dipole vectors do determine the potential difference between equidistant points, and Wilson (57) does the same in another way. Although Katz (35), and Wolferth and Livezy (60) have objected to the Einthoven Triangle Theory the experimental evidence for its validity has been well established (19, 23, 57).

The Electrical Phenomena of Activity

In our discussion of the electrical phenomena of the cell we have spoken of the resting and active states. The former is that state wherein the cell is

unstimulated. In terms of the dipole theory, it is characterized by having all its dipoles oriented with their positive poles away from the center of the cell. Thus, as will be shown, the electrical force of each dipole is opposed by an equal and opposite force on the other side of the cell, which reduces the electrical potential of the cell as measured by external electrodes to zero. Yet there is electrical activity present in the resting cell as shown by the presence of a resting potential. (See the discussion of the Dipole Theory).

The active state of the cell is the stimulated state, and is divided into two components, i.e., the stage of depolarization and the stage of repolarization. In terms of the Dipole Theory, in the former stage the cell is characterized at the completion of the process by having all its dipoles reoriented so that the positive poles now point towards the center of the cell. In the stage of repolarization, the cell is characterized, at the completion of the process, by having the dipoles again reversed to their former resting position.

As has been previously shown, all the dipoles of the heart during activity may be summated by the laws of vector addition so that their effect may be considered to be that of a large single dipole located at

the electrical center of the heart. The mean electromotive force of this dipole may be represented by a vector passing through the poles of the dipole and having its direction determined by the orientation of these poles in space. The magnitude of the vector is determined by the potential difference between the two poles. Such a vector and its accompanying electrical field, in one plane only, is shown in Figure 14. In such a field the plane of zero isopotentiality (or the null line) extends, as stated before, perpendicular from the center of the dipole to the surface of the cylinder, separating the surface into an area of positive and negative potentials on either side. The null line is determined by the direction of the vector, or stated in another manner, the null line determines the direction of the vector.

If we consider the null line, as it is oriented in space, we see that it describes a circle which may be taken as representing an imaginary plane, S, upon each side of which are accumulated positive and negative charges respectively. The area of this plane is proportional to the number of charges on it. These charges represent the summation of all the charges on each elementary dipole, which when summated give rise to

the electromotive force, E, whose magnitude is determined by the absolute potential difference. The direction of the electromotive force in space is represented by E. If e is the magnitude of a single vector representing the summation of all the elementary vectors of each small dipole drawn normal to S and $\hat{e}$ represents its direction in space, then: $\hat{E} = S\hat{e}$ (6). This may be diagrammatically shown as in Figure 18.

As was stated earlier depolarization occurs at a faster rate than repolarization, even in a single cell. One would think that repolarization should begin where depolarization started. While this seems to be true in nerve cells, it does not seem to be the case in the heart, where repolarization begins opposite to the point where depolarization begins. This will be discussed under the ventricular gradient.

The processes of depolarization and repolarization as viewed by the Dipole Theory may be diagrammatically shown as in Figure 19. In Figure 19, the active dipoles have their poles reversed and are cross-hatched if unopposed by and equal and opposite force on the other side of the equator. They are shown blank if opposed. The effective resting dipoles, i.e., those which act in conjunction with the active dipoles to produce the

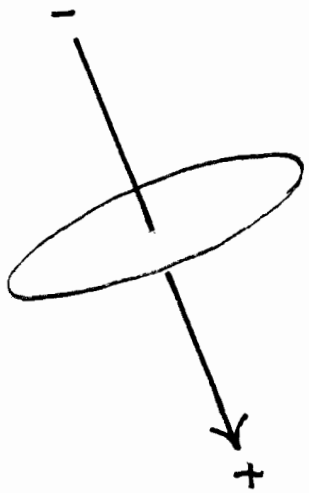

Fig. 18. The spatial vector drawn normal to its circular plane, S.

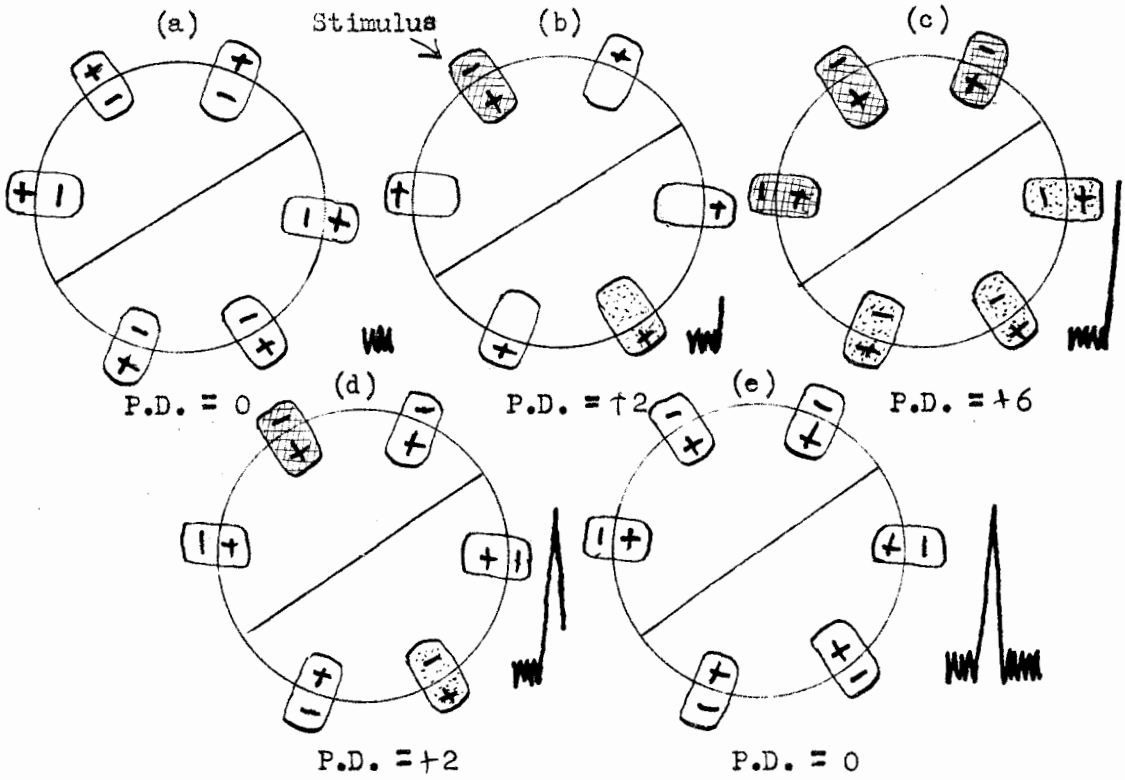

Fig. 19. Depolarization of dipoles with potential differences and electrocardiograms.

total electromotive force are dotted. The other resting dipoles are ineffective (shown as blank) because their force is balanced by an equal and opposite force on the other side of the equator. The absolute potentials (PD) as recorded by a unipolar electrode located at points P are shown for each stage of depolarization as well as the accompanying electrocardiographic record.

If bipolar electrodes were used the absolute potential, or the potential difference, would be the algebraic difference of the potentials as recorded by each electrode and would be twice as great as for a unipolar electrode recording at the same distance.

Actually the effective resting dipoles probably produce about half the electromotive force that the active dipoles do, but for simplicity we shall consider them as producing the same electromotive force as the active ones. Thus, in Figure 19, Part B, the electromotive force from both sides of the cell produces a potential difference of two.

In Figure 20, the vectors from the active and effective resting dipoles for Figure 19 are pictured.

If all the vectors in Figure 20 were added together according to the laws of vector addition one would obtain the mean electrical axis for that cycle of

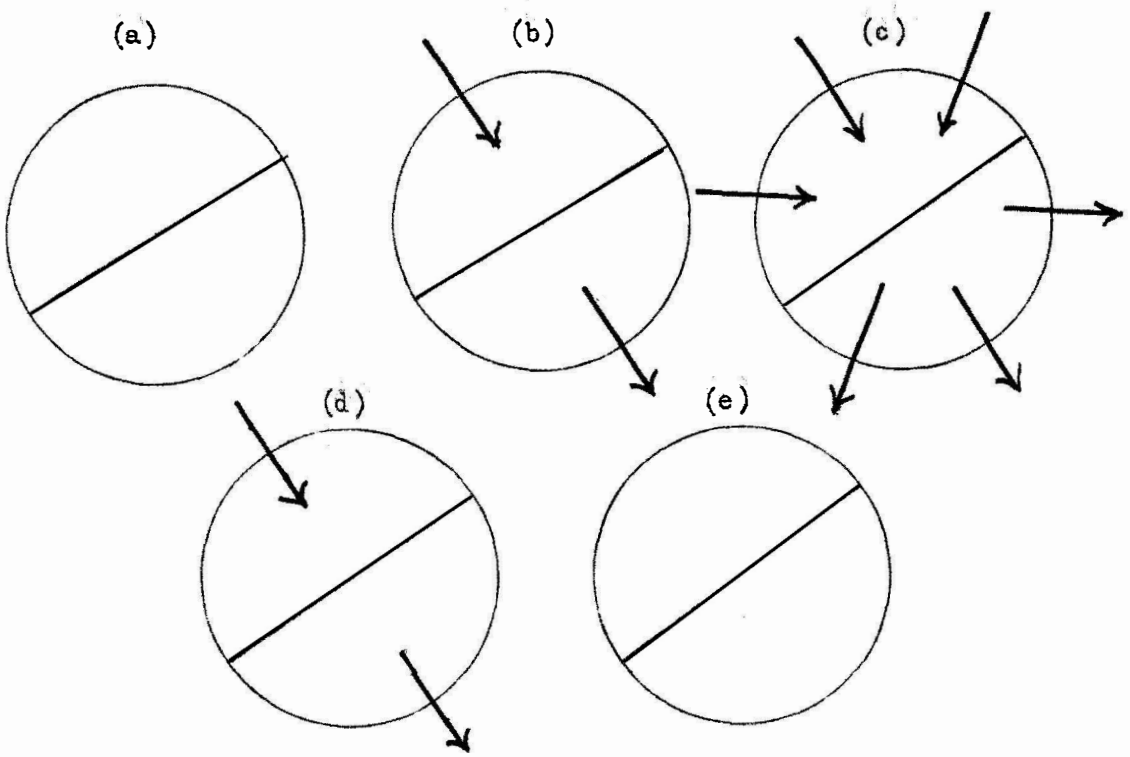

Fig. 20. Depolarization vector from active and effective dipole. See Figure 19.

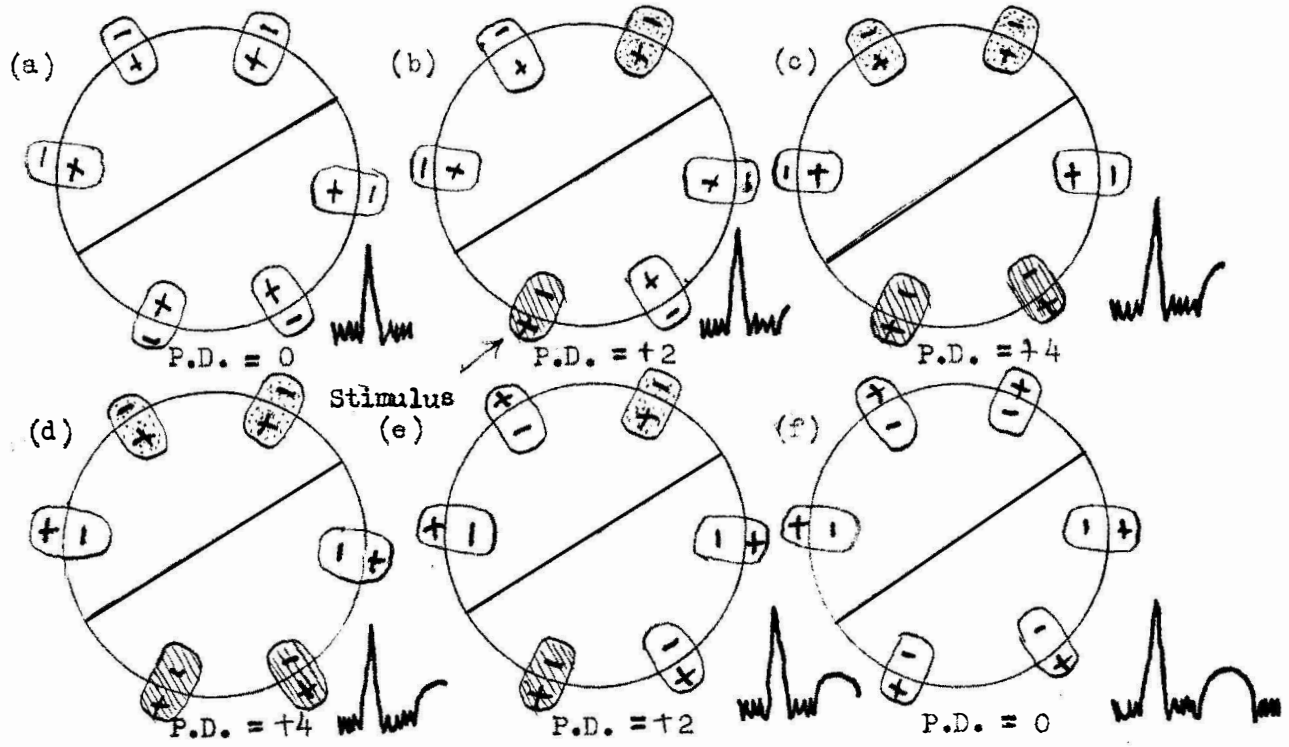

Fig. 21. Repolarization of dipoles with potential differences and electrocardiograms.

depolarization, and the result, shown diagrammatically, would appear as in Figure 18. This mean vector for depolarization is comparable to the mean QRS vector of the cardiac cycle.

We have now to consider repolarization in the single cell. As stated previously, repolarization occurs at a slower rate than depolarization and begins at a point opposite to where depolarization begins. These events are shown in Figure 21 and the vectors for this process in 22.

If now the mean electromotive force (the electrical axis) for repolarization is diagrammed (See Figure 23) we see that while the orientation of the positive and negative poles of the vectors are similar to that of depolarization, the direction of the mean vector of repolarization (which corresponds to the mean T vector of the heart) is somewhat different. This difference is what accounts for the ventricular gradient. If the positive portions of these two vectors are drawn from the electrical center of the cell with their appropriate directions, magnitudes, and null lines in relation to the equator, they will appear as shown in Figure 23. It will be noted that the null line of the depolarization vector is here parallel to the equator of the cell,

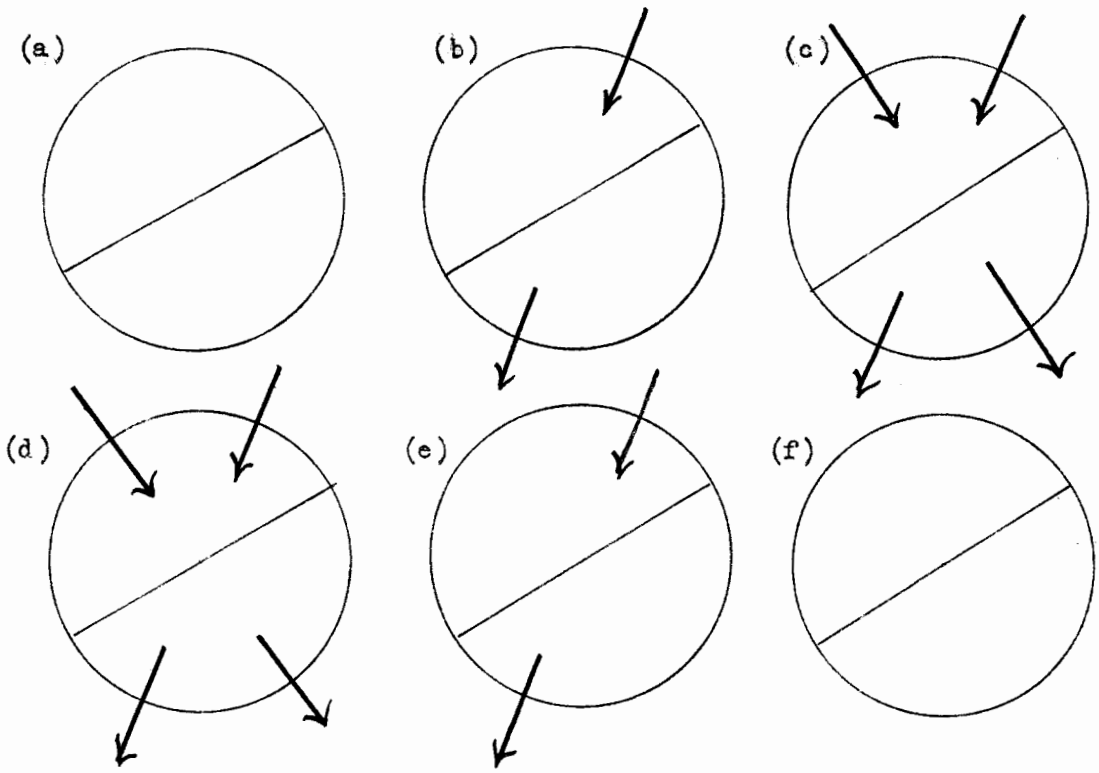

Fig. 22. Repolarization vectors from active and effective dipoles See Figure 21.

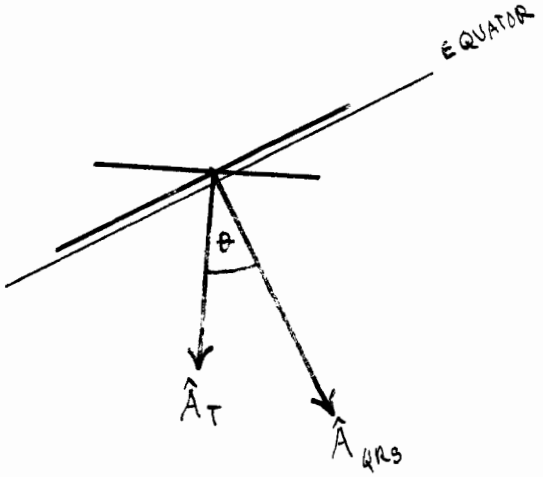

Fig. 23. Depolarization and repolarization vector and their null lines. $\hat{A}_{QRS}$ = the mean depolarization vector with a manifest area of $\hat{G}$. $\hat{A}_T$ = the mean repolarization vector with a manifest area of 4. θ = the polar angle ($\hat{A}_T$, $\hat{A}_{QRS}$).

and that the length of the null line is proportional to the size of the vector.

If this single cell is punched in at one end, it resembles the contour of the ventricles without a septum. In such a cell, shown in Figure 24, there is no difference in the pathway of depolarization except that the direction of the vectors from both sides of the cell are now parallel, giving a greater magnitude to the total electromotive force. The sum of the two parallel dipoles, i.e., the unopposed active and its opposite effective resting dipole, will henceforth be called the double-layer instantaneous vector.

The heart in toto may be diagrammatically visualized with the ventricles represented by such a punched in sphere, as seen in Figure 25, having a septum, and capped by a smaller slightly flattened sphere representing the auricles. That such a diagram is a quite accurate representation of the heart may be seen by inspection and by consideration of the P and P_u waves, and the QRS and T waves. Apparently, the septum of the auricles has little or no electrical effect in contradistinction to that of the ventricles.

It should be apparent that the depolarization and repolarization of the auricles will be quite similar to

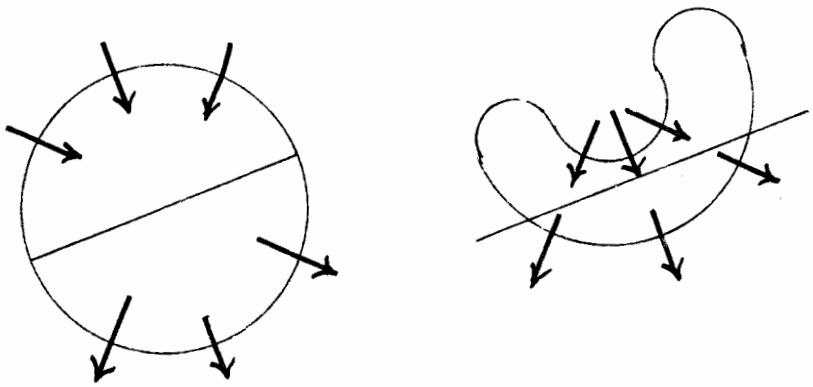

Fig. 24. Showing the effect of invagination on the vectors.

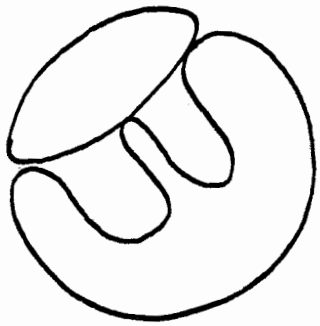

Fig. 25. Schematic representation of auricles and ventricles.

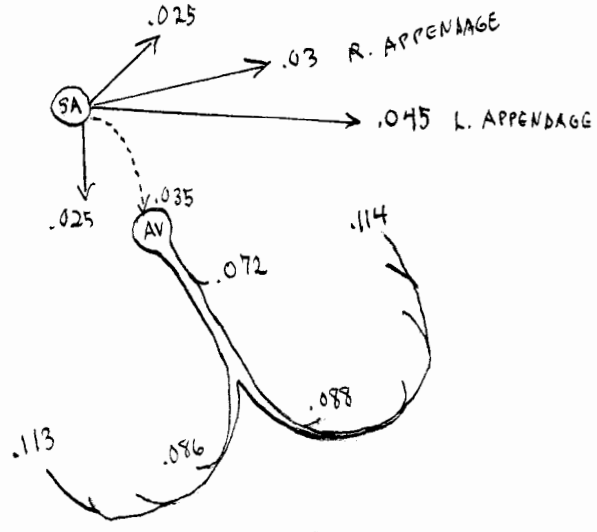

Fig. 26. The conductive system of the heart and time sequences.

that of the ventricles with the only differences being that the point of stimulation is more lateral in the auricles and that, therefore, the direction of the vectors will be in a more lateral position (pointing to the opposite side), and their magnitude will be much smaller due to the less amount of muscle in the auricle.

The processes of activity in the heart would be almost entirely similar to those of our hypothetical cell, except that the left ventricle of the heart is usually thicker than the right and the heart is endowed with specialized stimulus conducting system of its own, i.e., the Sino-Auricular node, the Bundle of His and the Purkinje network. This system alters the rate and points of stimulation so that the auricles and ventricles have most of their dipoles normally activated almost simultaneously.

Wiggers (50) has shown the rates of impulse conduction in this system (See Figure 26). One can see that the left side of the septum is activated first and then the right ventricle slightly before the left.

In addition changes in the amount of muscle, such as by hypertrophy, or decreases in the active and effective dipoles, as by infarction, alter the processes of activity.

The processes of electrical activity in the heart as shown by the double-layer vectors, and the instantaneous electromotive forces, which are derived by vector addition of the double-layer vectors, as affected by the normal muscle masses of the ventricles, and by the conducting system of the heart, are shown in Figure 27. In this figure, the greater the muscle mass of a ventricle the more dipoles are present upon its surface, and the slower is the impulse to travel through it from the endocardium (which is stimulated first) to the epicardium. Thus the longer are the double-layer vectors effective. The rate of travel through the myocardium is shown in the diagrams by the successive progression of shaded muscle lamellae. Normal hypertrophy of the left ventricle is what accounts for the normal left (electrical) axis deviation.

The stimulation of the left septal wall first is what accounts for the Q wave as recorded by unipolar leads, or bipolar leads having their positive electrode "facing" this left septal wall, such as Lead I (See Figure 28).

The relation of these electrical forces to the standard limb lead electrode positions are shown in Figure 28, as well as the accompanying electrocardio-

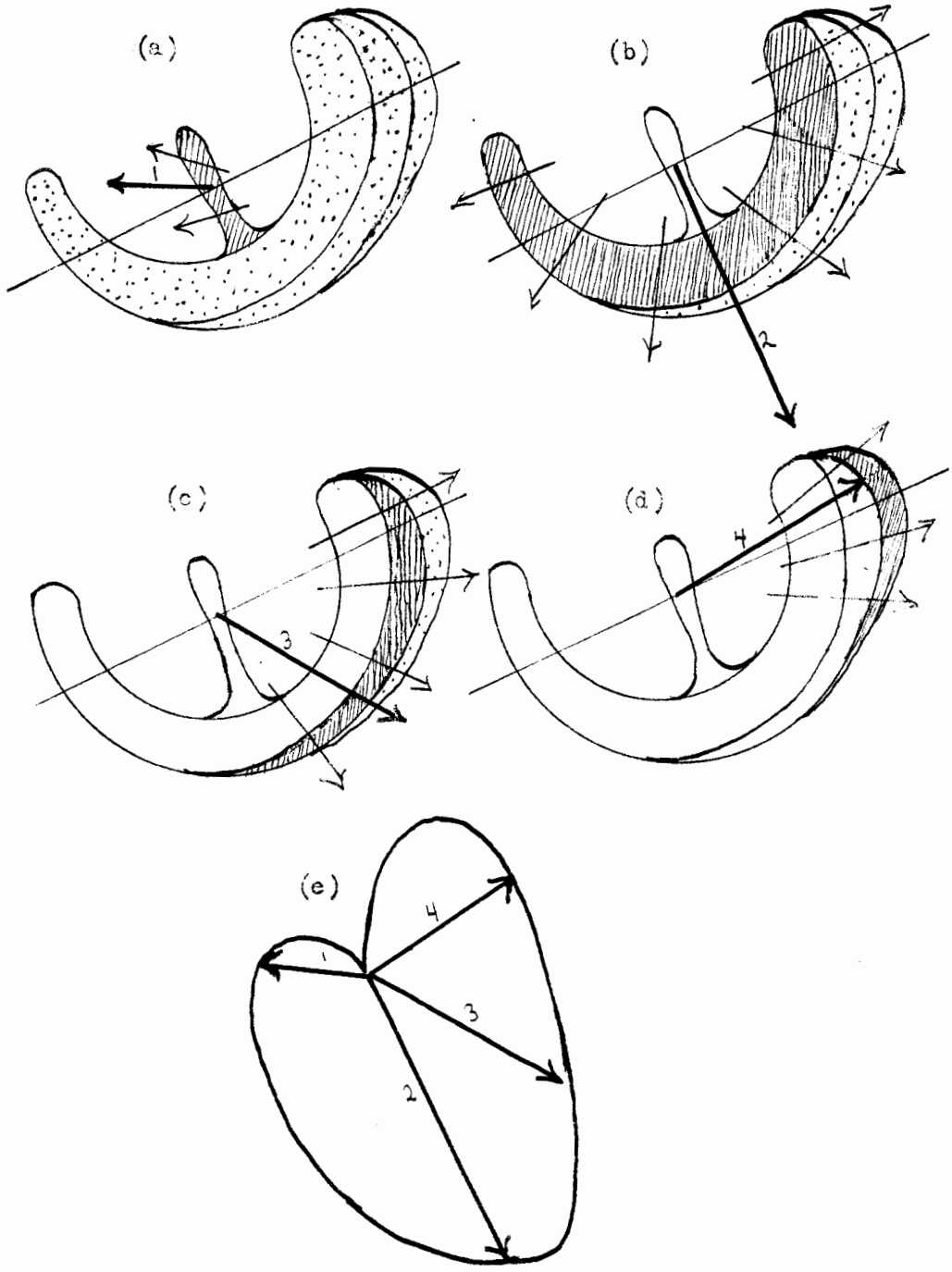

Fig. 27. Depolarization double layer and mean instantaneous vectors, with the derived frontal QRS loop. Shaded is active area; dotted is resting; blank is depolarized.

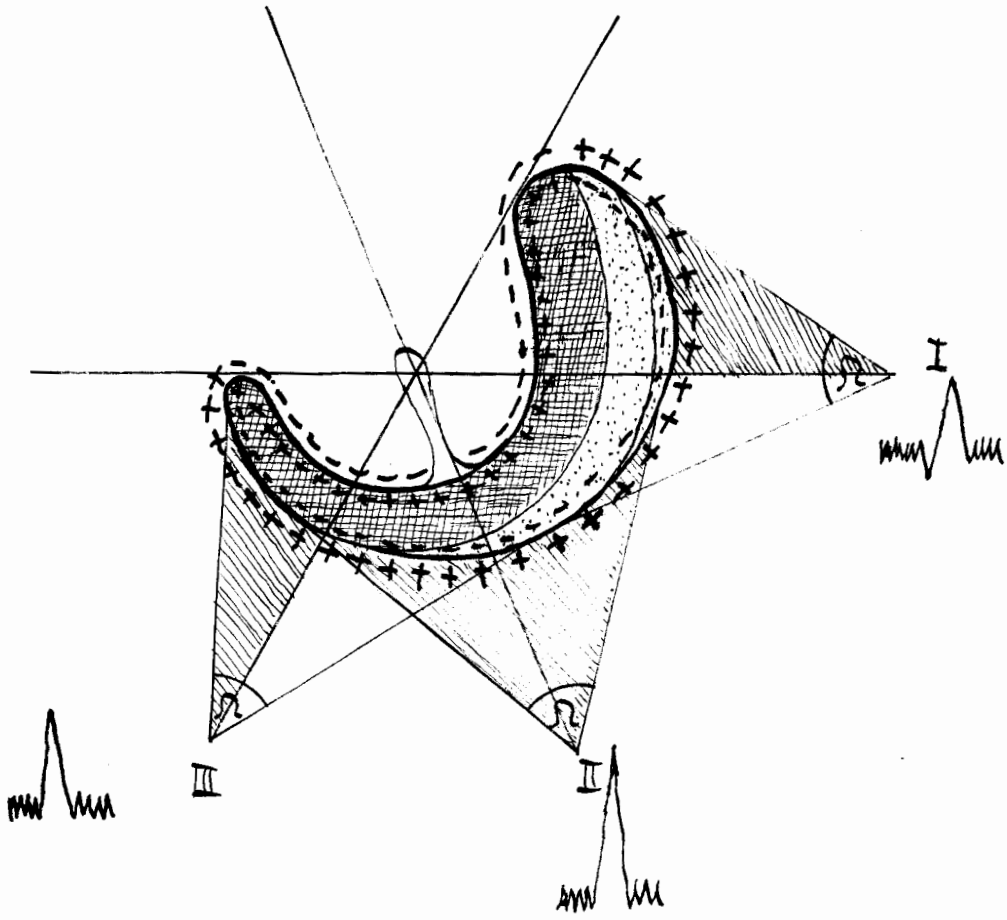

Fig. 28. Depolarization and the relationship of the solid angle, Ω, to the potential difference as shown on the electrocardiograms. The legend is similar to that in Fig. 27. The shaded portion of the solid angle is effective. (See text).

graphic record.

The potential at point P, may be expressed by the formula: $V = \phi \Omega$ (6).

V denotes the potential at a point in the field produced by arbitrary double layers and represented by the magnitude of the electrocardiographic wave. ϕ has the dimensions of an electromotive force across the double layers and its value is not definitely known at the present. Ω is the solid angle defined by the area of spherical surface cut off the unit sphere (inscribed about point P) by a cone formed by drawing lines from P to every point on the boundary of the double layers.

In Figure 28 the effective part of the solid angle, Ω, for each electrode position is shaded. The unshaded angle from point P to the active muscle is ineffective because the effect of the dipoles in the muscle mass nearest the electrode cut off by this angle is opposed by an equal and opposite force in the other ventricle.

A more simple method of determining the resultant electrical effect of the double layers as to sign upon the electrode position is to determine the direction of the mean vector, and then noting the position of the electrode in relation to the direction of the vector

under consideration. If the electrode is parallel with the vector, i.e., has the vector pointing towards it, then the recorded potential will be maximal and will be either positive or negative depending upon which sign the half-vector carries. If the electrode is perpendicular to the vector, the recorded potential will be zero, and the electrode will be found to be on the null line. This method of analysis is shown in Figure 17.

If now all the instantaneous vectors are recorded and their termini are connected by a curved line we will have a loop on the frontal plane as shown in Figure 27. This loop may be recorded upon three planes as shown in Figures 29a and 29b (48). Thus the loop has spatial dimensions just as the vectors have spatial directions. One may construct a spatial model of the loop with wire, or he may construct a mental image of the same. This loop oriented in space is called the spatial loop or the vectorcardiogram (16). Since it is a record of all the instantaneous vectors, it may be used, just as the mean vector was used, to determine the potential of any electrode placed anywhere upon the body. Its relationship to the electrocardiogram is shown later.

We have already spoken of the mean vectors of

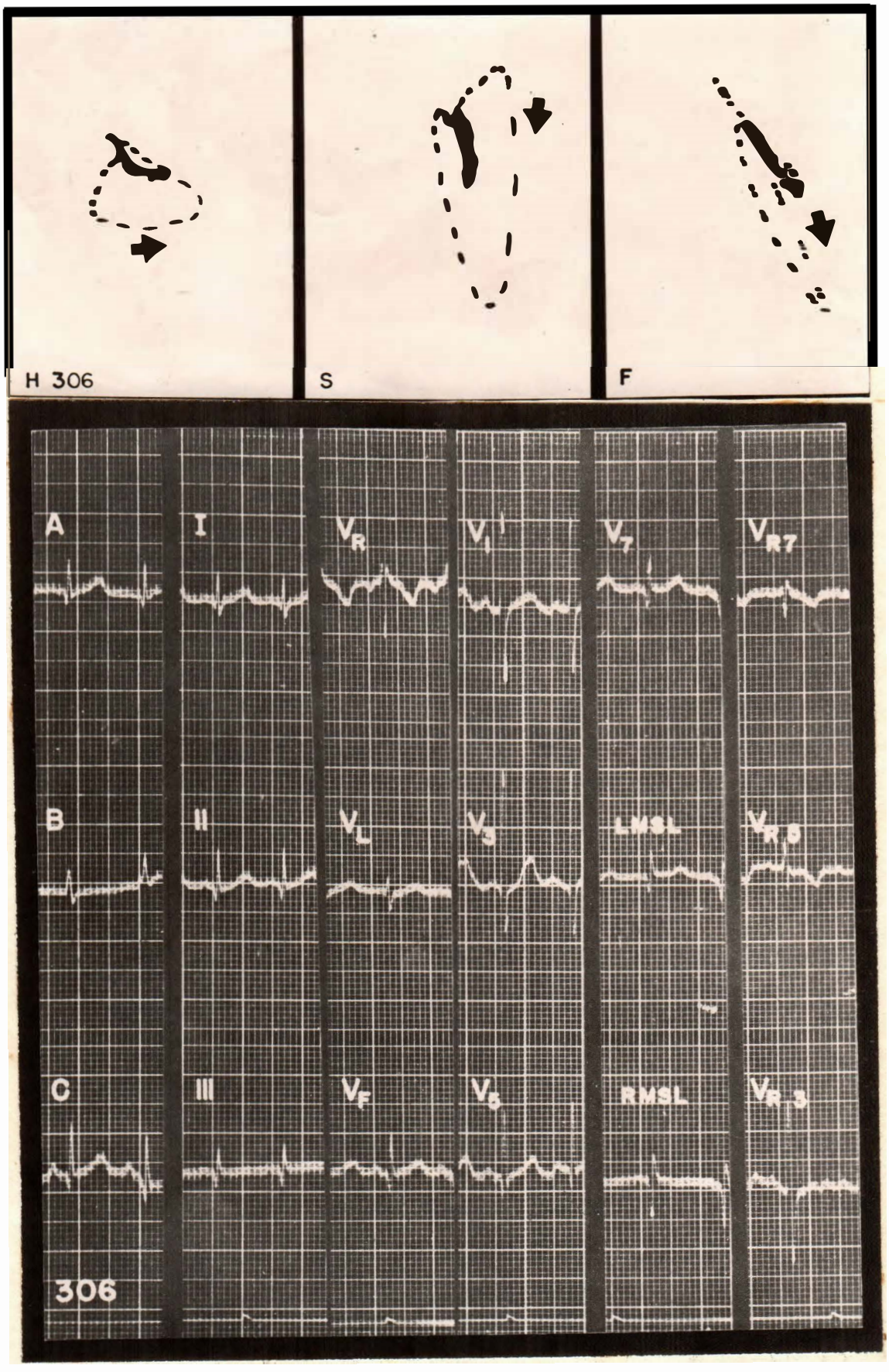

Fig. 29a. Normal vectorcardiogram from 8 month old child. A B C in the electrocardiogram refer to the horizontal, sagittal, and vertical bipolar vectorcardiogram leads. H, S, F = horizontal, sagittal, and frontal planes.

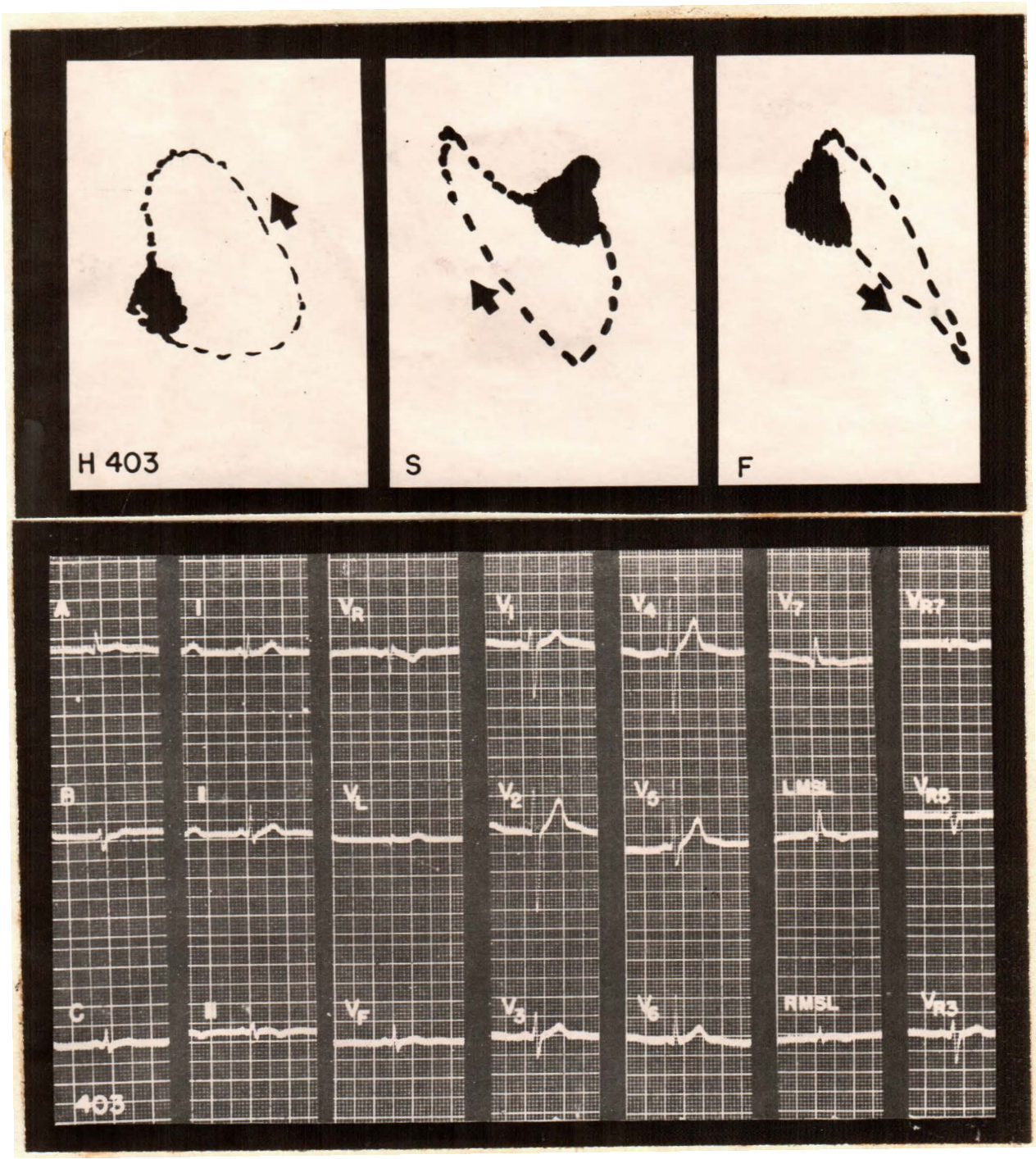

Fig. 29b. Normal vectorcardiogram and electrocardiogram in a 24 year old adult.

depolarization and of repolarization as being the mean QRS and T vectors (AQRS, AT) of the heart. These vectors are pictured in Figure 30. The sum of these two vectors comprises the ventricular gradient, which may be obtained by the parallelogram method pictured in Figure 30.

If the sequence of repolarization began at the same point where depolarization began and the two were equal in magnitude there would be no gradient, because the two vectors would comprise two equal and opposite forces, the sum of which would be zero. Therefore, the ventricular gradient is an expression of those electrical forces which appear when the sequence of repolarization is different from that of depolarization (4).

In discussing the relationship of these vectors to one another the formula: AQRS + AT = G may be rewritten so that: AT = -AQRS + G (6). (The method for determining AQRS and AT will be given below). Thus it is apparent that the manifest area, i.e., that area on the frontal plane, of T depends upon the manifest area of QRS and G. Changes in T caused by alterations of the gradient are known as primary T-wave changes, while those changes in T caused by alterations in QRS are called secondary T-wave changes. The primary changes are

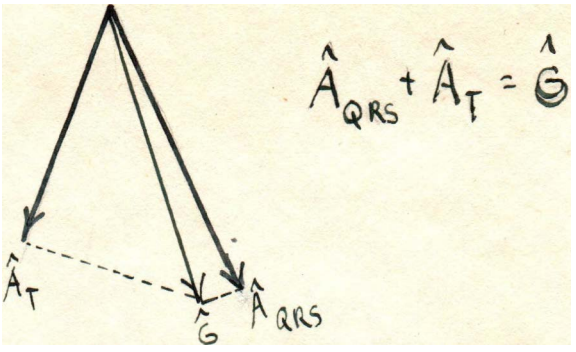

Fig. 30. Ventricular gradient. Perpendiculars are dropped from the termini of the vectors. The point where they meet is the terminus of the gradient.

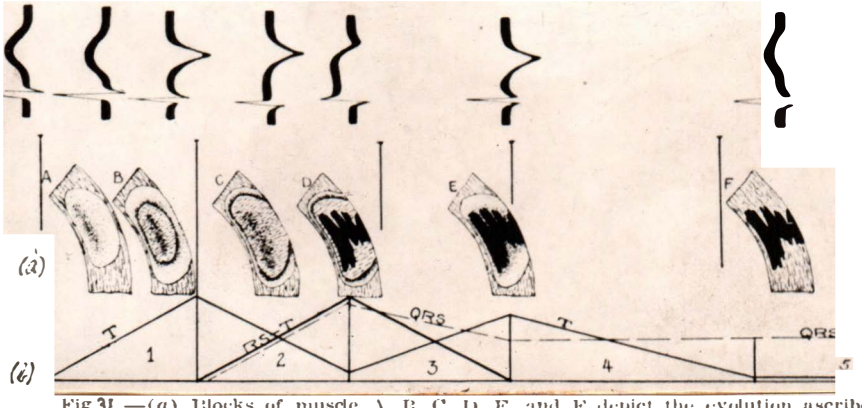

Fig. 31.—(*a*) Blocks of muscle A, B, C, D, E, and F depict the evolution ascribed to myocardial infarction. Superimposed are curves which display the associated electrocardiographic changes as produced in a unipolar lead from a superjacent point. In block A, local ischemia has developed without producing detectable electrical effects. In block B, ischemia has reached the epicardial surface and has accounted for a primary T-wave change. An injured zone has also appeared within the ischemic zone, and theoretically minor QRS changes may be present. In block C, the injured zone has reached the epicardial surface, so that RS-T junction displacements, further QRS changes, and reduced T-wave changes are present. In block D, infarction has occurred, and RS-T junction displacements are maximum. Concurrently, the primary T-wave changes are reduced to a minimum. In block E, healing has commenced. The injury zone has vanished and the effects of local ischemia are again pronounced (compare curves three and six from the left). In block F, the infarct is depicted as healed. No perifocal ischemia is present. (*b*) Diagram showing the time relationship of the appearance and (or) disappearance of the various kinds of electrocardiographic changes, QRS, T, and RS-T junction, which characterize the electrocardiographic evolution of myocardial infarction. Note the reciprocal relationship between primary T-wave changes and RS-T junction displacements, which is likewise shown by the development, at the epicardial surface, of the two zones, ischemic and injured.

caused by a local alteration in the duration of the excited state in the subepicardial and subendocardial (or the double) layers such as by ischemia or hypertrophy as shown in Figure 31 (6). The T vector (and the ventricular gradient as influenced by it) tends to point away from areas where the excitated state is longest, as in hypertrophy and ischemia, to areas where it is least (2, 26). This last may be understood by reflecting that the process of repolarization must begin late in areas of hypertrophy or ischemia. (A more complete explanation of these two conditions will follow shortly). Secondary changes are like those found in bundle branch block, and are related to QRS changes and not to muscle changes. In some cases, both factors operate (1).

Before we discuss abnormal QRS, T, and G vectors, it is important that we know the normal magnitudes and spatial relationship of these vectors to one another.

Bayley in an appendix to Ashman (1) standardized the nomenclature concerning these forces. By his designation the magnitude or the manifest area (on the frontal plane) in microvolt-seconds, as determined by Ashman's method (see below) for the electrocardiogram is represented by the symbol, A, which is to be followed by the appropriate symbol for the phenomenon being considered.

For example, A_{QRS} is the manifest area of the QRS force. If the force is considered a vector, the cupola, $\wedge$, is added above the A. Thus $\hat{A}_{QRS}$ represents the manifest QRS vector. If the true spatial direction of the force is to be considered, the symbol, S precedes the designation ($S\hat{A}_{QRS}$).

The angle made by any two lines radiating outward from the electrical center of the heart is called the polar angle, and it is indicated by writing in brackets the two lines by which the angle is formed.

The position of any of the spatial mean axes is not sufficiently defined by its manifest position. Hence, the polar angle made by a spatial mean axis is described as positive or negative according to whether the shortest motion through which the spatial axis appears rotated out of the frontal plane is counterclockwise or clockwise to an observer stationed at the patient's left, thus viewing the saggital plane (1).

The magnitude of a manifest mean axis is always equal to the product of the magnitude of its related spatial mean axis by the cosine of the angle made by the spatial mean axis and its projection upon the frontal plane. For example: $G = SG \cos (S\hat{G}, \hat{G})$.

In a similar manner, the areas under the curve of

the extremity lead electrocardiograms are always equal to the product of the magnitude of the related mean axis (spatial or manifest) by the cosine of the angle made by the limb of the triaxial reference system on which the area is plotted and the magnitude of the mean axis is considered. For example:

$$\text{Area QRS} = A_{QRS} \cos (-\text{RL}, \hat{A}_{QRS}) \quad (1).$$

Grant (4) studied twenty normal young adults and found that in the majority, the mean $S\hat{A}_{QRS}$ and $S\hat{A}_T$ vectors pointed within a range of $+20^\circ$ (anteriorly) and -45° (posteriorly). In all instances the manifest direction of the T wave, $\hat{A}_T$, was directed leftward and anterior to the A_{QRS}. The average $S\hat{A}_T$ pointed $+30^\circ$, and the average $S\hat{A}_{QRS}$ pointed -20°.

The magnitudes of the vectors varied more than their directions: the mean $S\hat{A}_{QRS}$ from 20 - 60 microvolt-seconds, the $S\hat{A}_T$ from 20 - 80 microvolt-seconds, and the $S\hat{G}$ from 40 - 120 microvolt-seconds (4). All these are within the normal range of Ashman's analysis (1). In most instances the T vector was larger than the QRS. Occasionally the reverse was true, or both would be strikingly smaller or greater than average. Because the range in magnitude was so wide, it was impossible to anticipate the degree of anterior or pos-

terior deviation of a vector by its size projected on the frontal plane (25).

The polar angle between the mean $\hat{SA}_{QRS}$ and $\hat{SA}_{T}$ was found to be within a range somewhat greater than has been anticipated in the literature (1). This angle was 14° to 49°, with the majority under 20°. The polar angle between the spatial T and the spatial gradient varied from 5° to 27°, all within the normal range as defined by Ashman (1). In this study no attempt was made to calculate the ventricular gradient --anatomical axis polar angle (25). However, this last correlation has been accomplished by Ashman (3), but time does not permit its discussion.

We shall now briefly discuss the effect of local ischemia and hypertrophy as they alter dipole activity and the spatial loops.

There are two kinds of local ventricular ischemia: the acute and chronic. There is no difference in their electrocardiograms except that in the latter the rate of appearance, duration, and disappearance of the primary T wave changes is longer.

Local ischemia is characterized by a local prolongation of the effective duration of the excited state, or as a local tardy onset of regression (6).

In terms of the double layer dipole theory, the ischemic state would be diagrammed as in Figure 32 a and b.

The potential difference point P in Figure 32 at half repolarization would normally be +18, but due to the ischemic dipole the potential is reduced 3 units. Immediately after complete repolarization of the normal dipoles, the ischemic or tardy dipoles repolarize restoring the potential difference to zero and bringing the inverted portion of the T wave back to the base line of the electrocardiogram.

If the area of ischemia is sufficiently large there will be none or only a slight positive segment early in the T wave, and this will be followed by a large inverted segment such as seen in Figure 31 above block B.

The next grade of impaired function in ischemia appears to be an inability to complete locally the delayed regression process (6).

After these stages of impaired function, injury occurs. The difference between an injured zone and an ischemic zone is that in the former not only is regression tardy in its onset in the involved area, but accession in this area is also tardy. In addition, the number of dipoles in the injured zone is reduced. Diagrammed at the end of normal depolarization, an injured

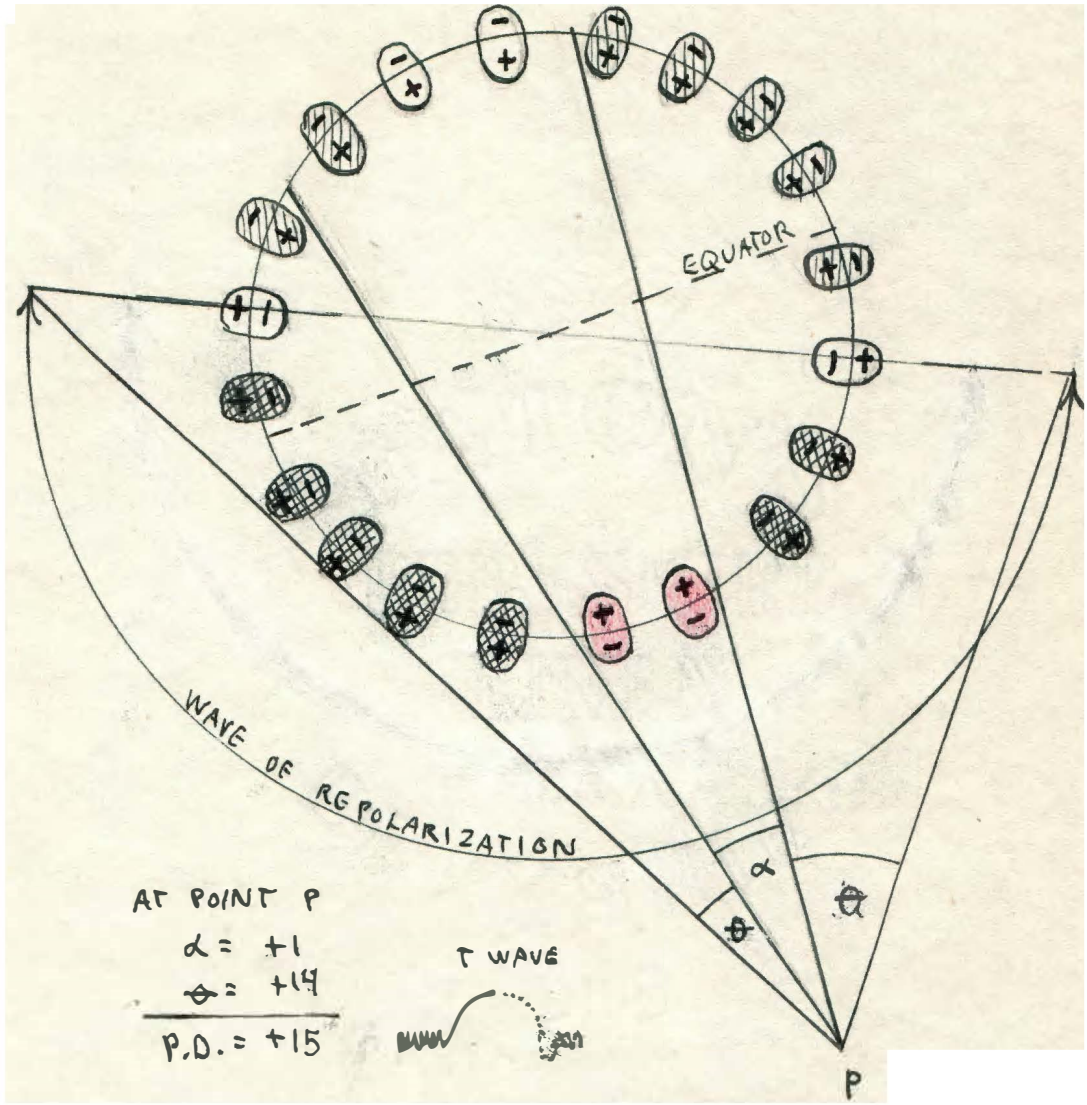

Fig. 32a. Ventricular repolarization in an ischemic zone at half-repolarization. Legend: cross-hatched dipoles are active, shaded are effective depolarized or repolarized dipoles, blank are ineffective, red are ischemic dipoles (tardy repolarization). α = portion of the solid angle, Ω, transected by the boundaries of the ischemic zone. θ = the solid angle as determined by the termini of the wave of repolarization minus α.

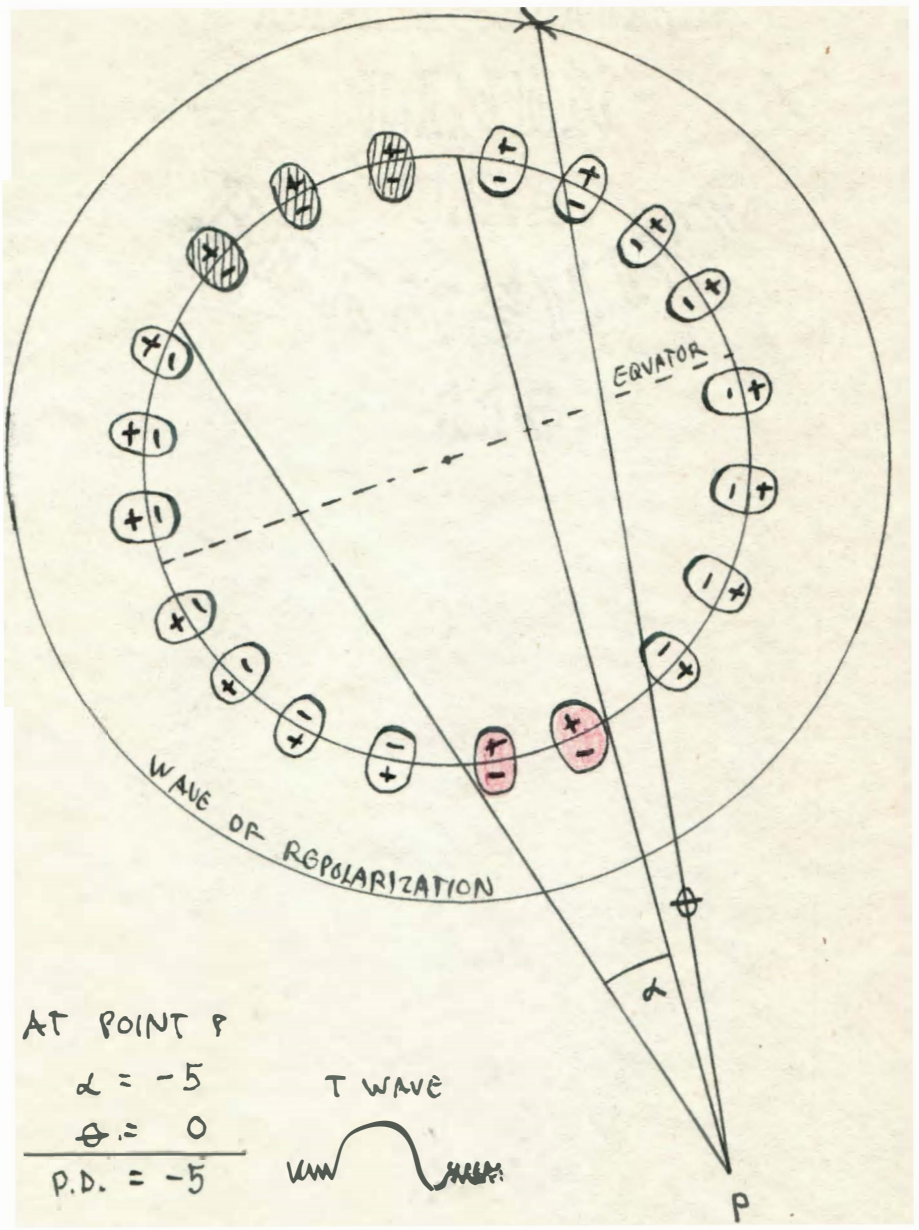

Fig. 32b. Complete repolarization of normal dipole in ischemic zone (tardy repolarization of ischemic dipoles). (See legend Fig. 32a.)

area would appear as in Figure 33.

The potential difference at the end of normal depolarization would be zero, here due to the injured zone there is a potential difference of + 4 -- this constitutes the current of injury or the elevated RS-T segment as seen in the injured electrocardiogram of Figure 31 above block D. A moment later the injured dipole depolarizes and negates the current of injury so that it returns to the base line.

It is to be noted that the height of the R wave is also decreased by the absent dipole in Figure 33 by one unit. Thus instead of being 18 units as in Figure 32, the R wave would be only 17 units in magnitude. In actual infarction the number of absent dipoles is much greater.

In Figure 34 the effect of half repolarization is shown in the injured zone. The injured electrocardiogram is shown in Figure above block D.

Actually, the height of the T wave would not be + 15 units as shown in Figure 34, because here we have shown, for simplicity, the rate of repolarization as being as rapid as that of depolarization.

At the end of normal repolarization it can be seen, by referring to Figure 32b, that the potential difference

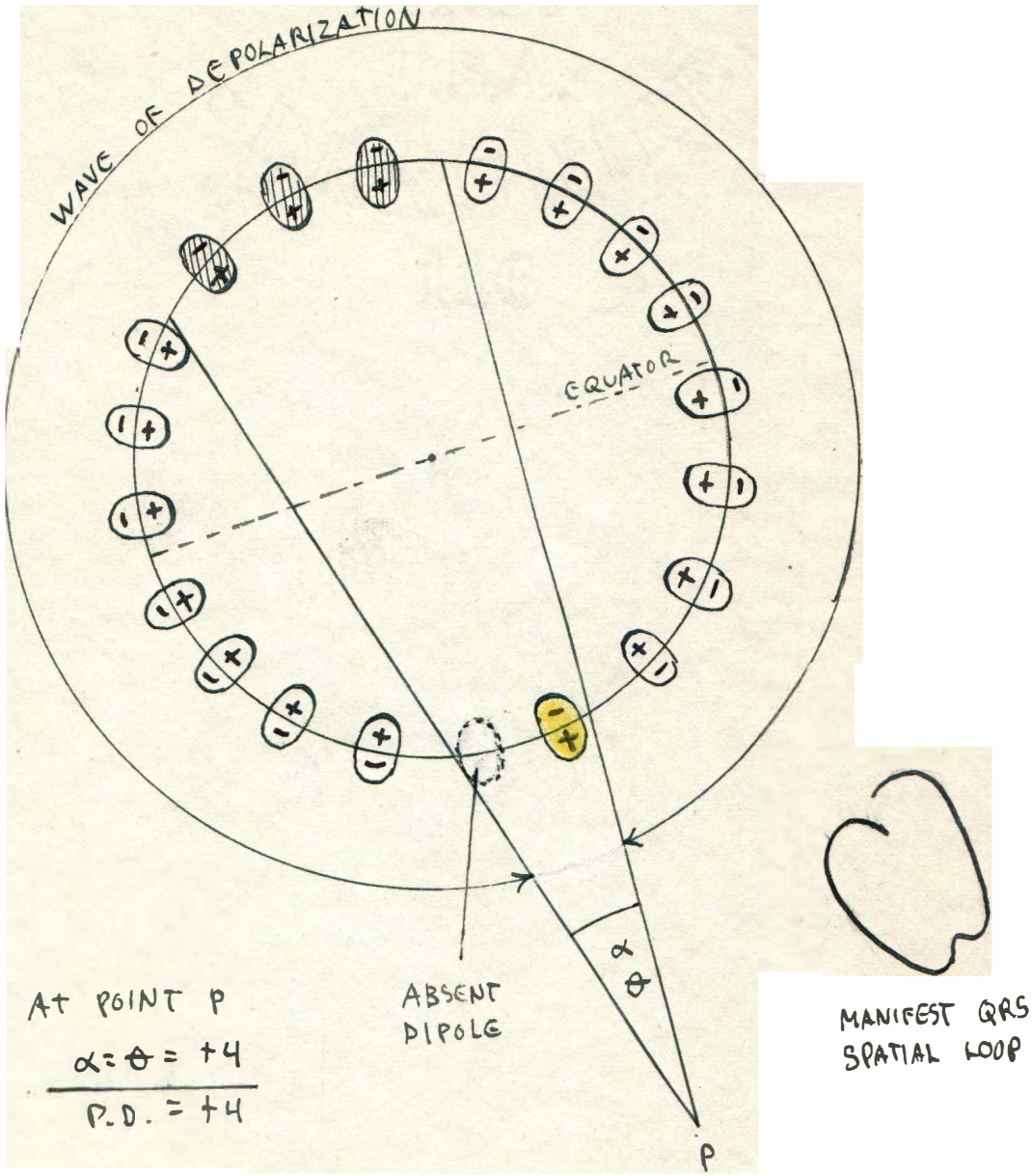

Fig. 33a. Ventricular depolarization in an injured zone at the end of normal depolarization. (Legend same as in Fig. 32a.) Yellow = injured dipole (tardy depolarization and repolarization). The spatial loop on the frontal plane is also shown. (See text).

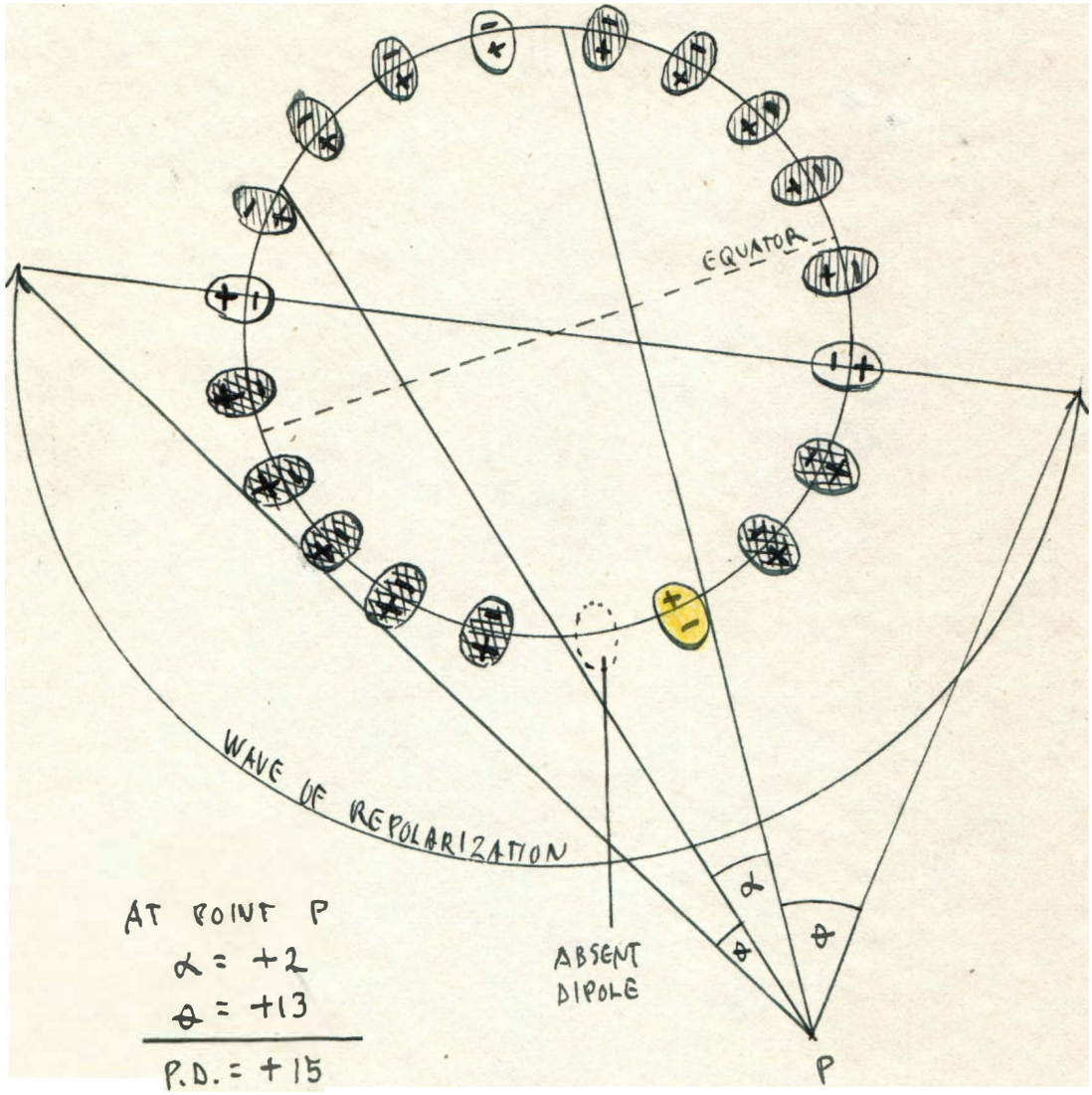

Fig. 33b. Ventricular repolarization in an injured zone at half repolarization. (See Figures 32a and 33).

at point P, in Figure 34, would be -4.

After the injured dipole repolarizes the inverted segment, as shown in Figure 31, returns to the base line of the electrocardiogram.

It can be seen that the spatial QRS loops in myocardial infarctions would show an indentation in their normal contour on the side of the injured zone (Figure 33). This is also shown in Figure 34 (47). The reason for this indentation can be inferred if one remembers that the loop is written by the instantaneous vectors which are the sum of the instantaneous double layer vectors. Since these forces are decreased in the injured zone, they affect the contour of the loop as shown; however, they do not constitute vectors in the direction as shown in Figure 34, but are actually a reduction of the true vectors which act in the opposite direction.

The effect of progressive hypertrophy would be to increase the duration of the active dipoles on the hypertrophied side so that the loop would be progressively distorted in the direction of the forces acting in this area of prolonged activity. The effect of progressive right, and of left hypertrophy is shown in Figures 35, 36 and 37 (38), and in Figure 38 (46).

Abnormalities in timing of QRS vectors, i.e.,

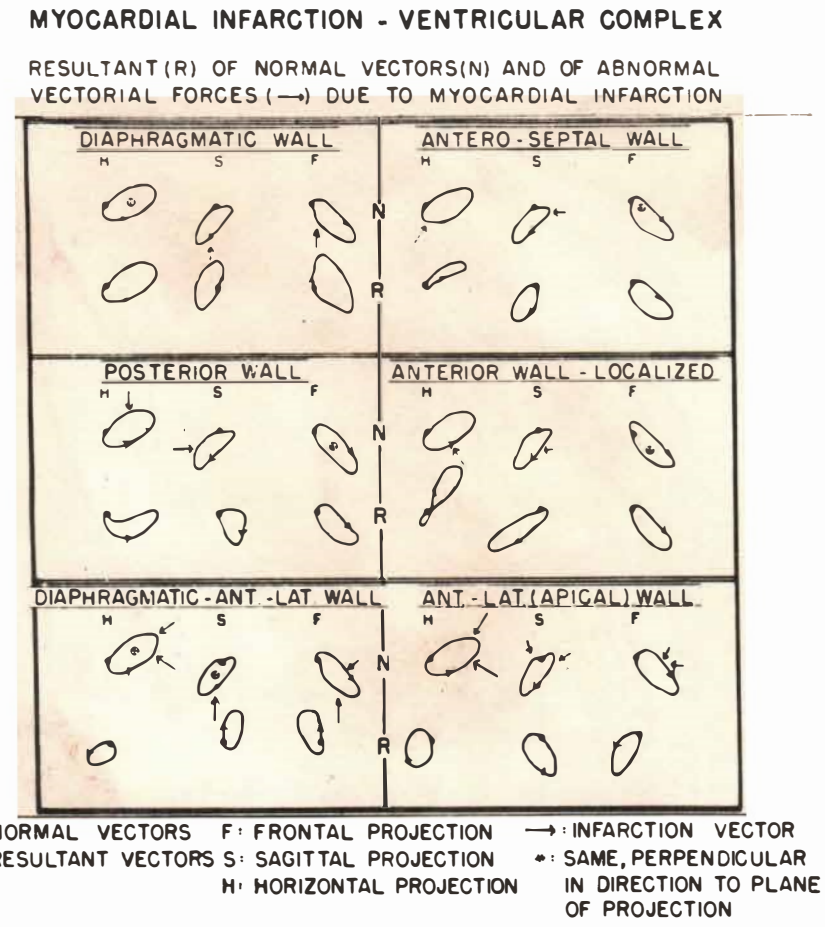

Fig. 34. Schematic representation of the spatial projections of the vectorcardiograms in the horizontal, sagittal, and frontal planes. The resultant vectorcardiograms following various localizations of myocardial infarction are shown with the arrows indicating the direction in which the abnormal vectorial force displaces the QRS sÉ loop in each plane.

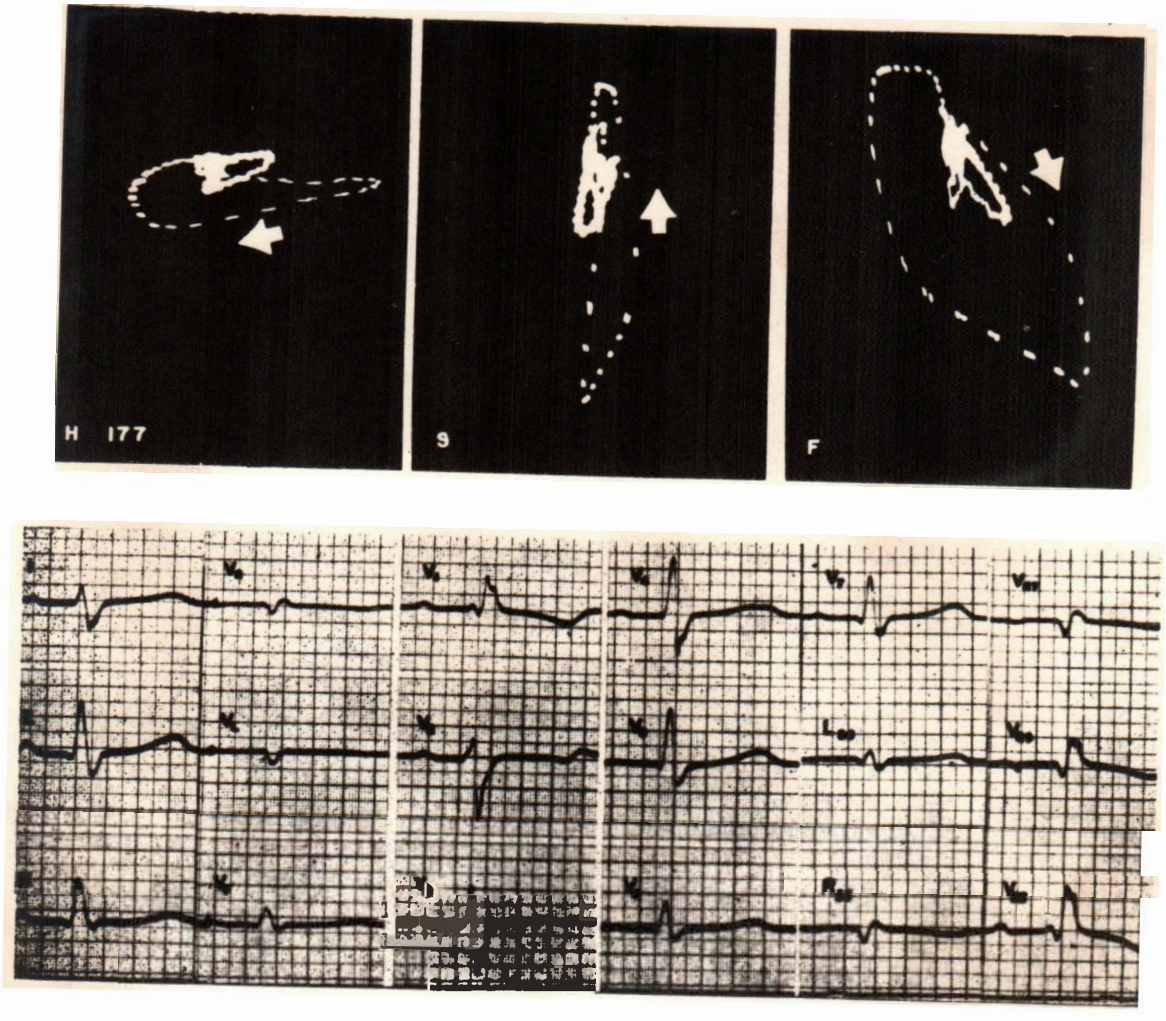

FIG. 35. Right ventricular hypertrophy, type I. See text for detailed analysis and discussion.

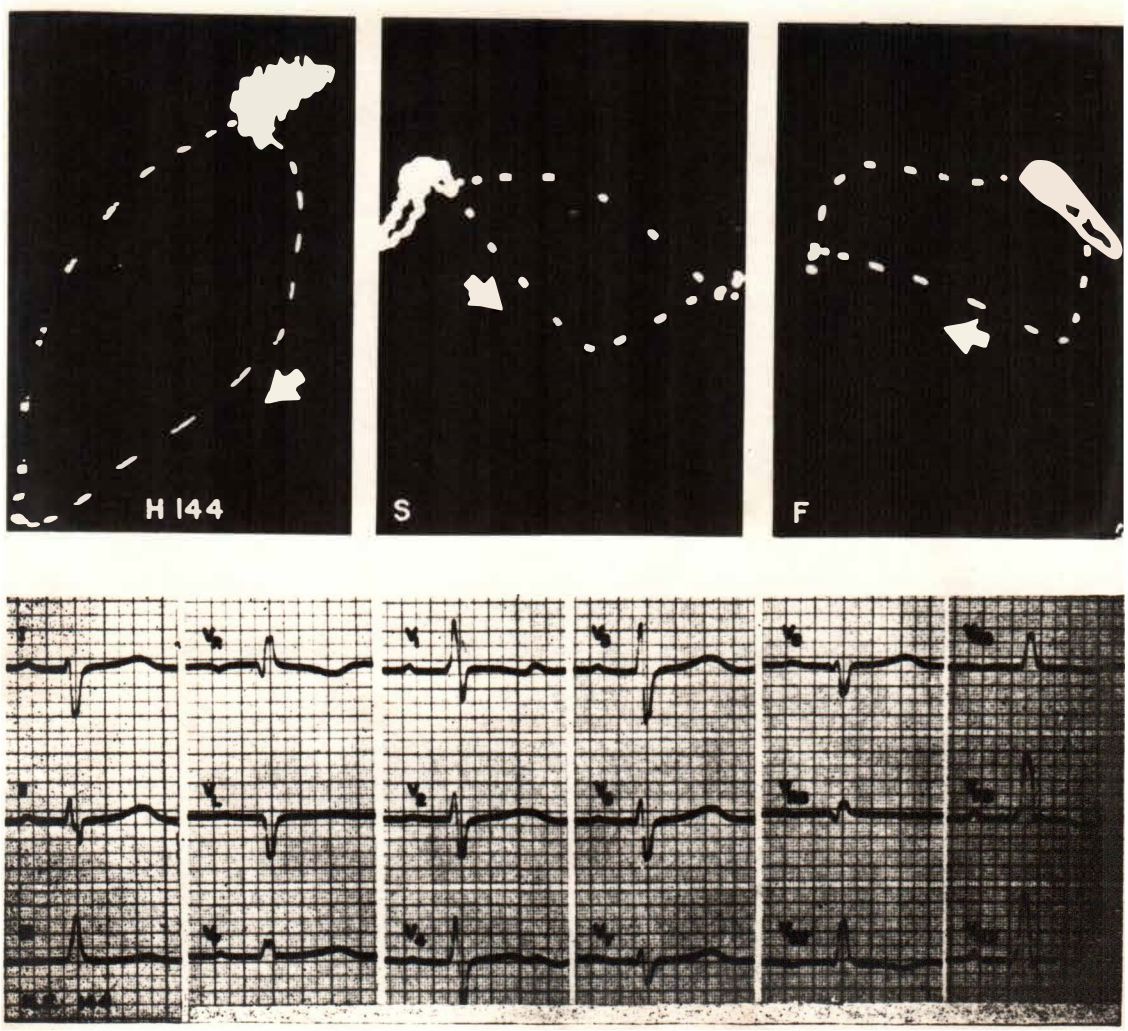

Fig 36. Right ventricular hypertrophy, type II. See text for detailed analysis and discussion.

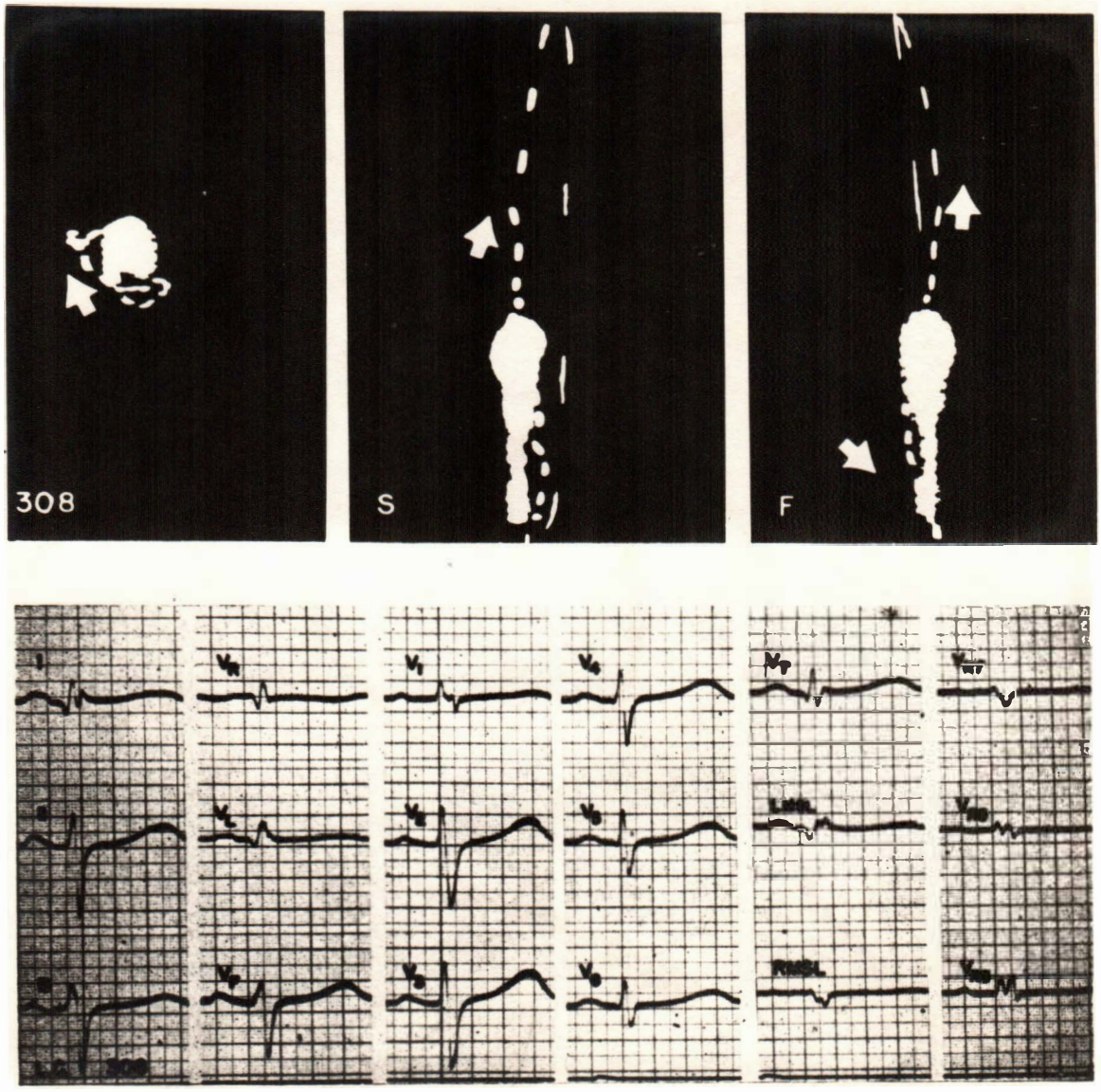

Fig. 37.—Right ventricular hypertrophy, type III. See text for detailed analysis and discussion.

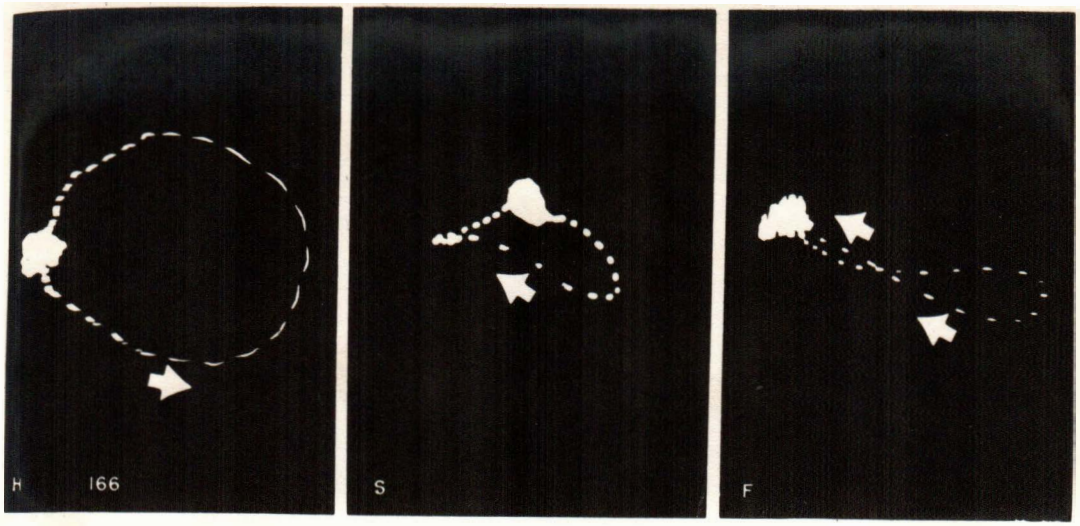

Fig. 38a. Vectorcardiograms in left ventricular hypertrophy.

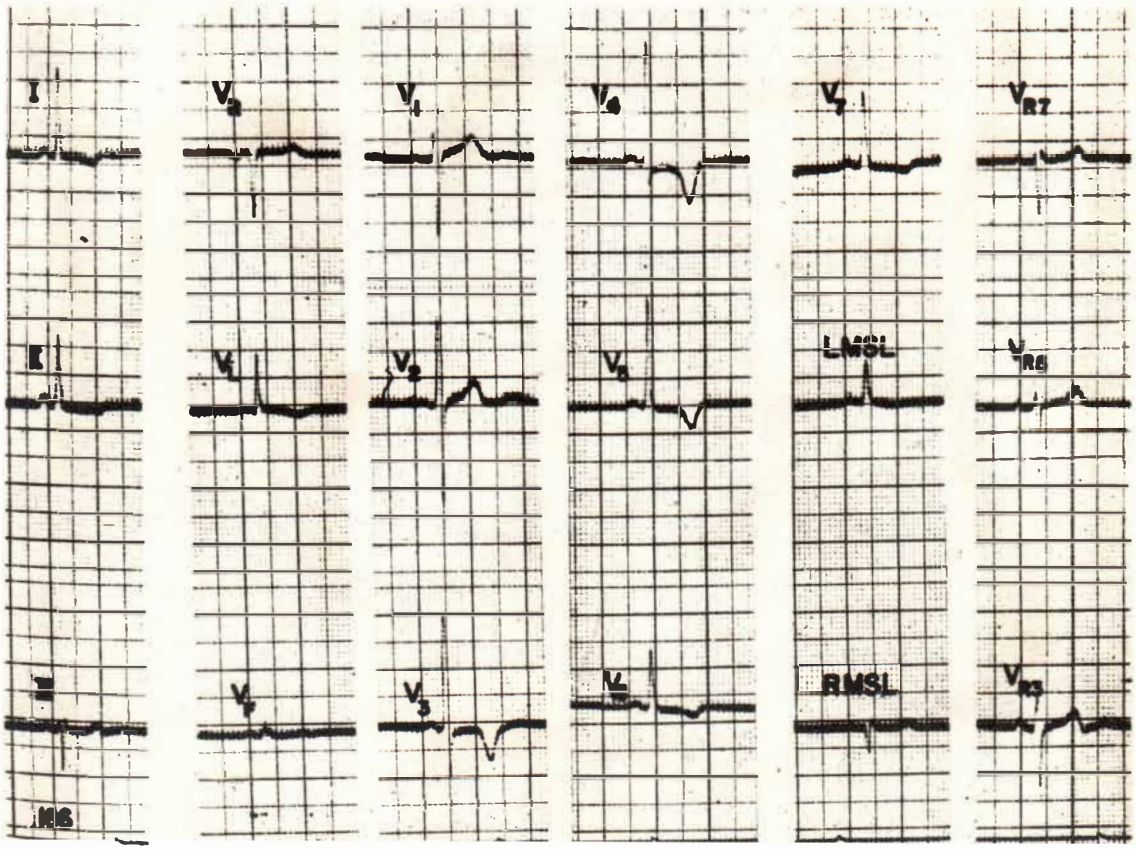

Fig. 38b. Thoracic and routine electrocardiograms in Case 166. *LMSL* = left mid-scapular line; *RMSL* = right mid-scapular line; V_{R7} = right posterior axillary line; V_{R6} = right anterior axillary line; V_{R3} = medial to right mid-clavicular line. (All the above were recorded at the level of V_4.)

abnormalities in the activation of some of the dipoles in relation to their normal time of activation, with resultant abnormalities in the dipole forces themselves, occur when one or the other bundle branch is blocked. The QRS interval is prolonged by the late activation of the dipoles in the blocked ventricle, which gives rise to vectors which distort the terminal portion of the QRS spatial loop. These terminal vectors will point in the general direction of the blocked ventricle, wherever in the chest it may lie (27). (See Figure 39 (46)).

The relative speed with which the deflections are inscribed can be seen by comparing the distances between interrupted segments of the vector loops. A slow inscription is thus evidenced by the close proximity of these segments. The absolute speed can be measured by counting the segments since the beam is interupted every 0.0025 second. It is important to analyze each loop for evidence of such delayed inscription since a tangential projection of a normally inscribed loop may present the appearance of a fairly solid line. Evidence of delay, to be considered significant, should be present in all three projections. The direction of inscription in the QRS spatial loop must be noted in each plane since an analysis of the sequence of the

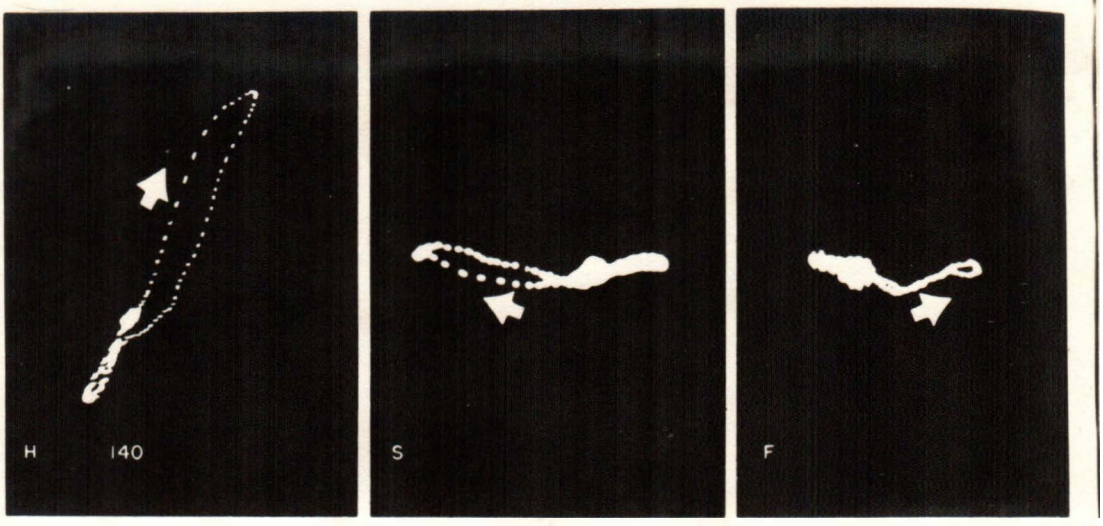

Fig. 39a Vectorcardiogram in left bundle branch block. Case 140.

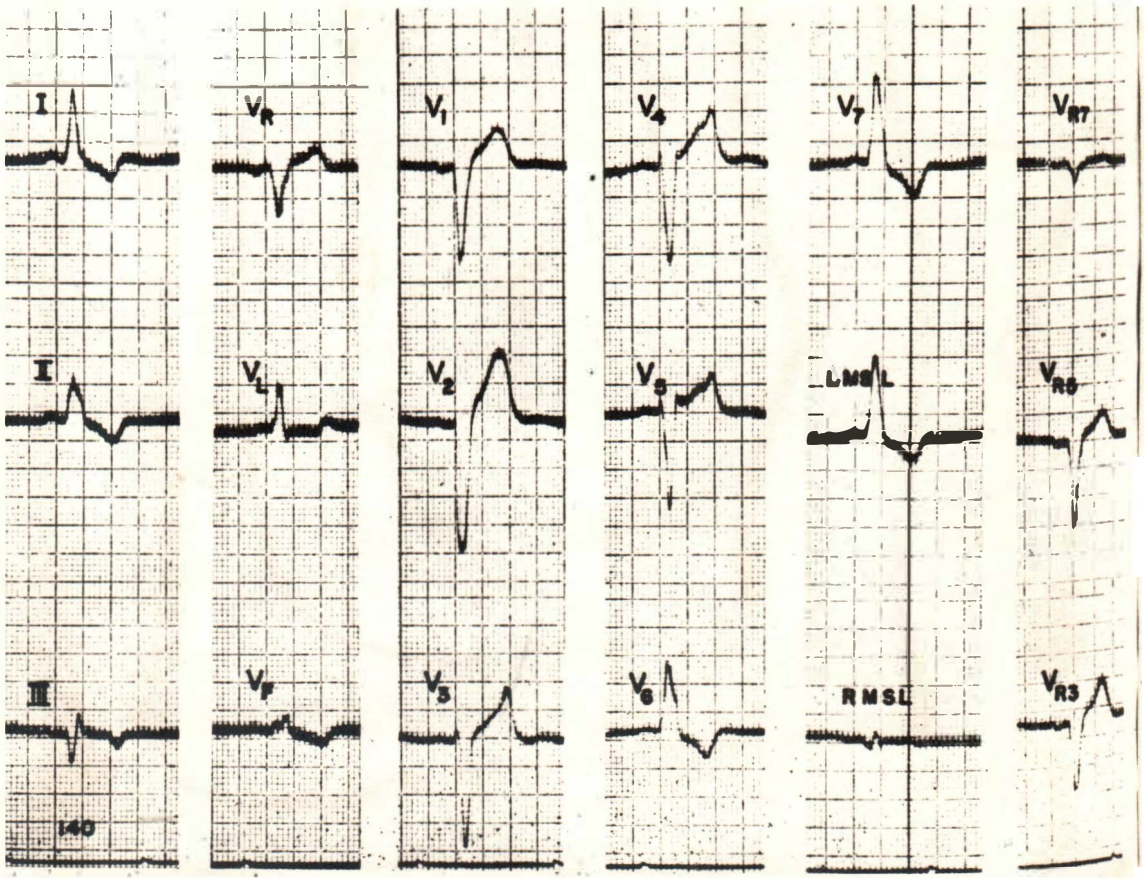

Fig. 39b Thoracic and routine electrocardiograms in Case 140.

spread of the wave of accession is of vital importance, the normal direction of rotation in the horizontal plane is counterclockwise, in the saggital plane clockwise, and in the frontal clockwise or counterclockwise, according to the position of the loop.

The normal vectorcardiogram for the child and adult as seen in Figures 28 and 29 (52) consists of a white center with a rapidly inscribed large loop (QRS $\hat{SE}$ loop), and a much more slowly inscribed smaller loop (T $\hat{SE}$ loop). The white center represents the isoelectric line of the normal electrocardiogram. The spread of the wave of accession over the atria is recorded as a small loop (P $\hat{SE}$ loop) which is not well visualized with the standardization employed in the routine recording of vectorcardiograms. Each loop is normally recorded as a closed loop. Failure of the beam to return to center will be recorded as an open loop. This occurs when the RS - T vector is not zero, but has either a positive or negative sense. In the electrocardiogram it is seen as a deviation of the RS - T segment (48).

The Methods of Analysis

The first method of analysis to be presented here is that derived from the spatial cardiac vectors. These vectors may be analyzed according to their direction

and magnitude, or by their direction alone. The former method is determined from appropriate calculations made from stand limb-lead electrocardiograms, while the latter is made merely from inspection of these leads as well as the unipolar precordial leads. We shall first present the method by calculations.

Ashman (1) perfected this method. The algebraic sum of the total inclosed negative and positive areas (in microvolt seconds) is determined on the electrocardiogram for the QRS and T waves in leads I and III as measured from that edge of the P-R interval which is opposite to the direction in which the deflection moves. The resultant area represents the magnitude of the vector (QRS or T) as projected on that lead. In the case of the QRS vector the area is determined by counting the height of the wave in microvolts and multiplying this by the length of the inclosed area (in seconds) on the base line as determined from the P-R interval. This sum is then divided by two.

The magnitude of each vector in each lead is then transferred to the standard triaxial reference system, as shown in Figure 40. From these points perpendiculars are dropped which determine the terminus of the spatial vector as projected on the frontal plane.

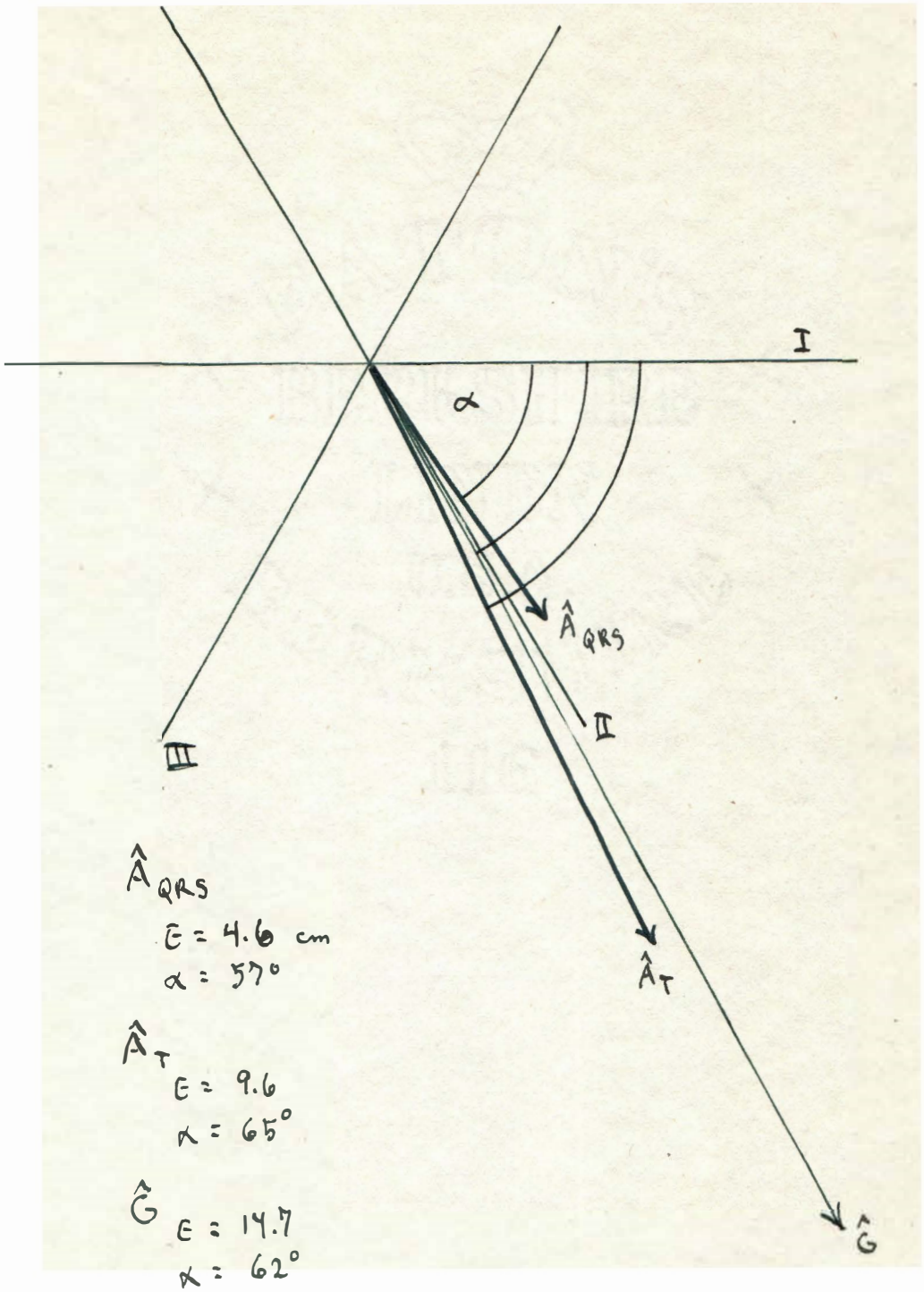

Fig. 40. Normal manifest vectors. Subject X.

$\hat{A}_{QRS}$ and $\hat{A}_T$ are then drawn in.

The ventricular gradient may be determined from these two vectors by the laws of vector addition or by the parallelogram method. The parallelogram method is shown in Figure 40.

The unipolar limb-leads and the unipolar triaxial reference system may be utilized in a similar fashion to calculate the magnitude and direction of these vectors on the frontal plane, but in this case the magnitudes of $\hat{A}_{QRS}$ and $\hat{A}_T$ must be corrected by multiplying them by the square root of three, if they are to be equal in magnitude to those on the standard triaxial reference system (30). While $\hat{G}$ on the unipolar system must be multiplied by 1.154 to be equal to that on the standard system (24).

The next step in determining the spatial vectors is to obtain their projection on another plane -- either the horizontal or sagittal, Grant (25) and Wilson (58) use the bipolar back electrode which is placed on a point located just below the inferior scapular angle and mid-way from the spinal column. This electrode defines the saggital plane when it is used with the projections of the frontal vectors on the 90^{o} frontal-median plane. The magnitudes of the areas under the

QRS and T waves on this back electrode electrocardiogram are plotted on a bi-axial coordinate system with a 45° angle between the two axes. This is shown in Figure 41. The magnitudes should be multiplied by the square root of 1.5, but this correction is so small that it can be ignored (27).

In Figures 42 and 43 the electrocardiograms from which the above vectors in Figures 40 and 41 were calculated on the frontal and sagittal planes are shown.

At this point one can construct a spatial geometrical model from swab sticks and pipe cleaners as shown in Figure 17. The true spatial direction and magnitude of the vectors as well as their polar angles can be readily measured on such a model. Or if one wishes he may calculate the magnitude and polar angles of such vectors.

This model (Figure 17) also shows the relationship of the vectors and their null contours to the positive pole of the electrode positions from which the positive portion of the electrode axis has been drawn in (See Figure 47 -- text below). The electrocardiograms from which the model was derived are shown in Figures 42 and 43.

The magnitude of the mean spatial vectors calculated from their projections on the frontal and sagit-

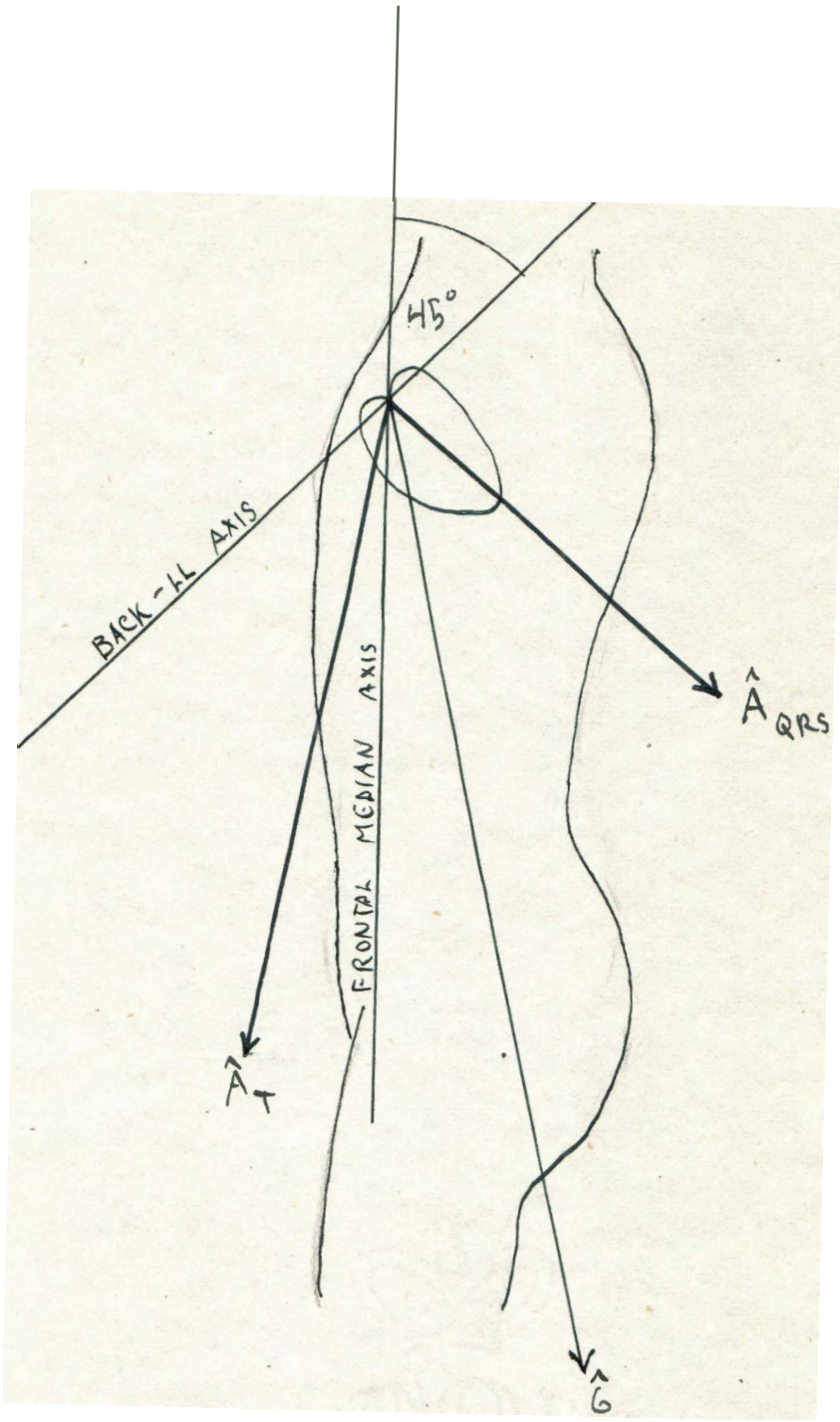

Fig. 41. Standard sagittal biaxial reference system. Sagittal loop and vectors. Subject X.

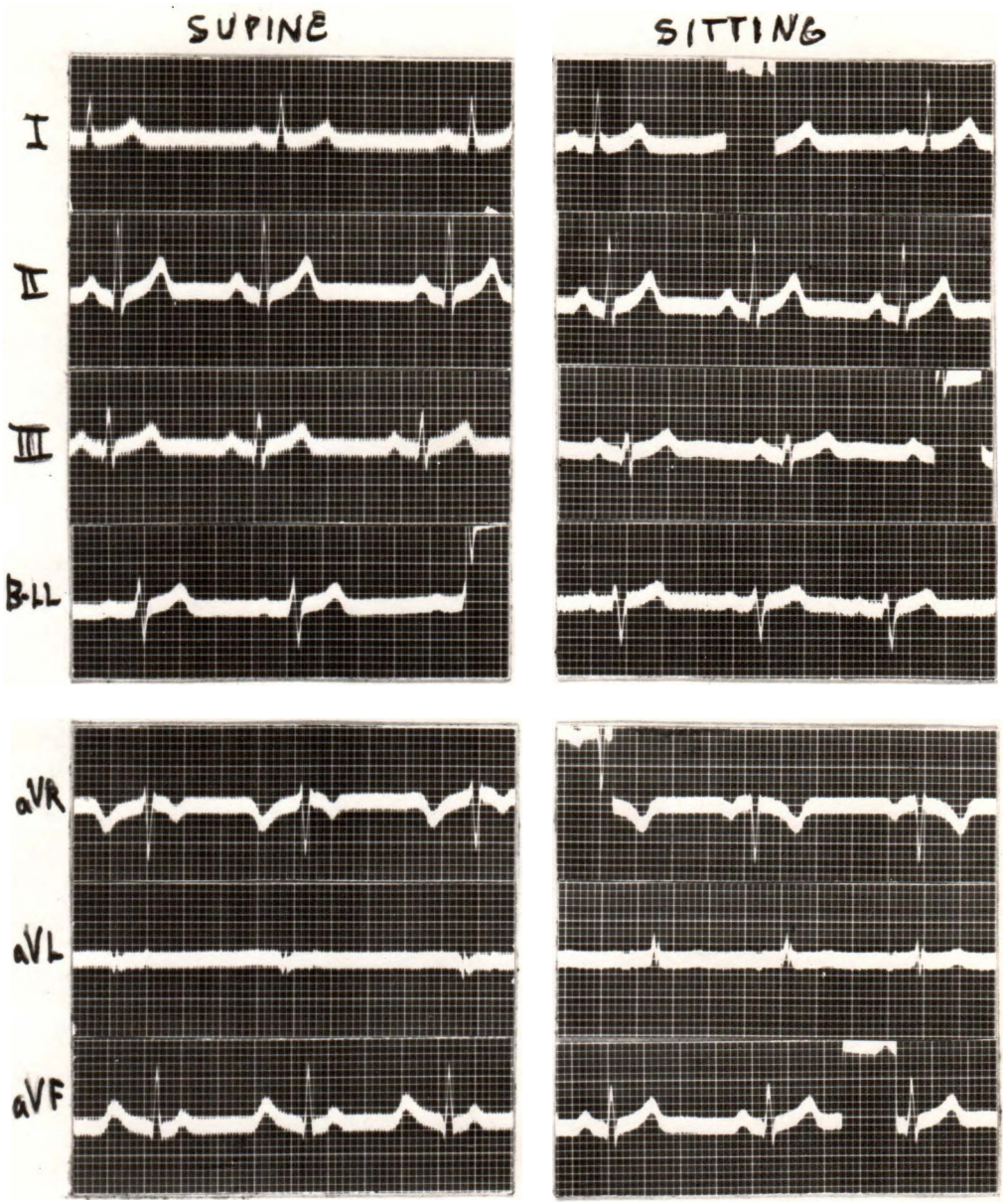

Fig. 42. Normal electrocardiogram. Young adult. Subject X.

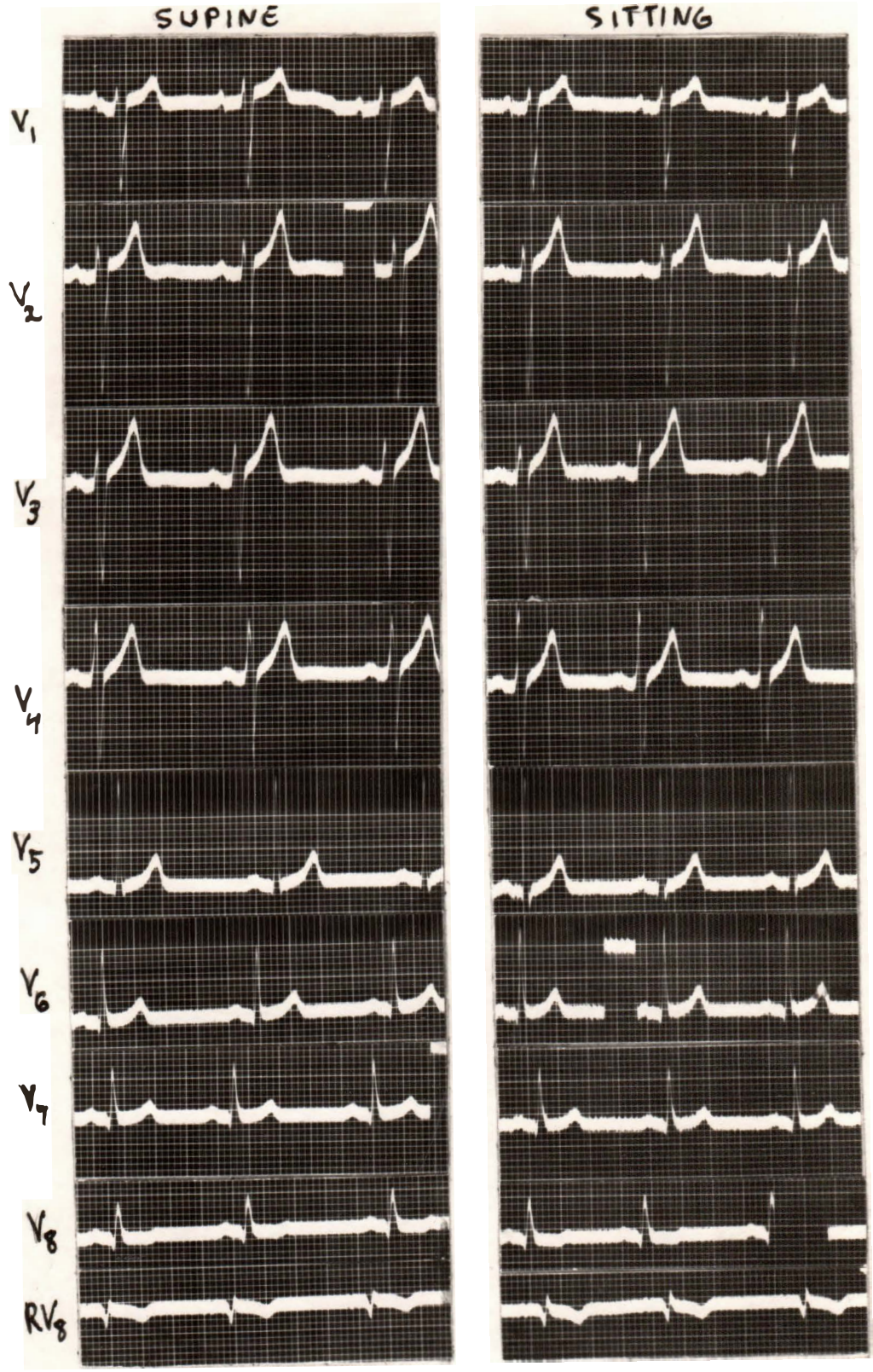

Fig. 43. Normal electrocardiogram. Young adult. Subject X.

tal planes using a Cartesian coordinate system consisting of three mutually perpendicular axes: (1) the vertical axis (median at 90° in the frontal plane) = a. (2) the anteroposterior axis which extends from the origin (in the center of the frontal plane) to the apex of the tetrahedron in the sagittal plane = b. (3) the horizontal axis which is lead I of the frontal plane = c. The components of the vectors on these axes are determined by dropping perpendiculars from the axes to the ends of the vectors in the appropriate plane. The magnitude of the mean vectors in space were calculated from the equation:

Magnitude of mean spatial vector = $V = \sqrt{a^2 + b^2 + c^2}$

The components of QRS, T and G were each in turn substituted in the equation giving the mean spatial QRS, T, and G. In Bayley's nomenclature these are SA_{QRS}, SA_T, and SA_G.

The angles between the mean spatial vectors were calculated from these same components by appropriately substituting in the equation:

$$\cos \theta = \frac{(QRS_a \times T_a) + (QRS_b \times T_b) + (QRS_c \times T_c)}{SA_{QRS} \times SA_T}$$

where θ is the angle between the mean spatial QRS and T vectors (27). Wilson (62) gives another method for calculating these values.

Grant's method (27) for determining the direction of the vectors remains to be discussed. This method does not enable any accuracy in the determination of the magnitude of the vectors, but the magnitude is not nearly as important clinically as the direction, unless the heart has symmetrical pathology in which case the vector would have its direction unaltered. Such an occurance is rare (27). This method for determining the direction of the mean spatial QRS and T vectors by inspection is very rapid, practical, and has only a 5% error in skillful hands.

The direction of these mean vectors on the frontal plane is determined by drawing the appropriate triaxial reference system. Next one decides which limb-lead has the conspicuously largest or smallest electrocardiographic deflection. (Remember the algebraic sum of the total positive and negative areas inclosed is the essential factor determining the size of the deflection.) The vector is parallel or perpendicular, respectively, to these deflections.

Now the anterior or posterior projection of the spatial vector is determined. First draw a chest, upon which the triaxial reference system (lightly drawn), with its mean QRS and T vectors, is represented. Next,

draw the six precordial electrode positions. Identify the one with the transitional QRS complexes (or null contours). (See Figure 44). Using the vectors obtained above, they must have a direction anterior or posterior from the frontal plane so that they are perpendicular to the axis of the precordial electrode which recorded the transitional complex. Now draw in the null contour as shown in Figure 44. All electrodes on the positive side of this contour (which represents a plane circle) will record resultant positive deflections. The opposite side will record negative deflections.

The second method of analysis is that employing spatial loops. Here again there are two methods. The first is again one of inspection (27) and is obtained from the limb-leads and the precordial leads, while the second is based upon the actual spatial loops as they are recorded by vectorcardiograms (16).

In the method of determining the spatial loop by inspection, the loop is first drawn on two or three planes. From two or preferably three of these planes either a single mental image or a single wire loop construction can be made representing the spatial loop.

In this first method let us begin by examining the relationship of the loop on the frontal plane to the standard limb leads, as redrawn from Mann (41) and

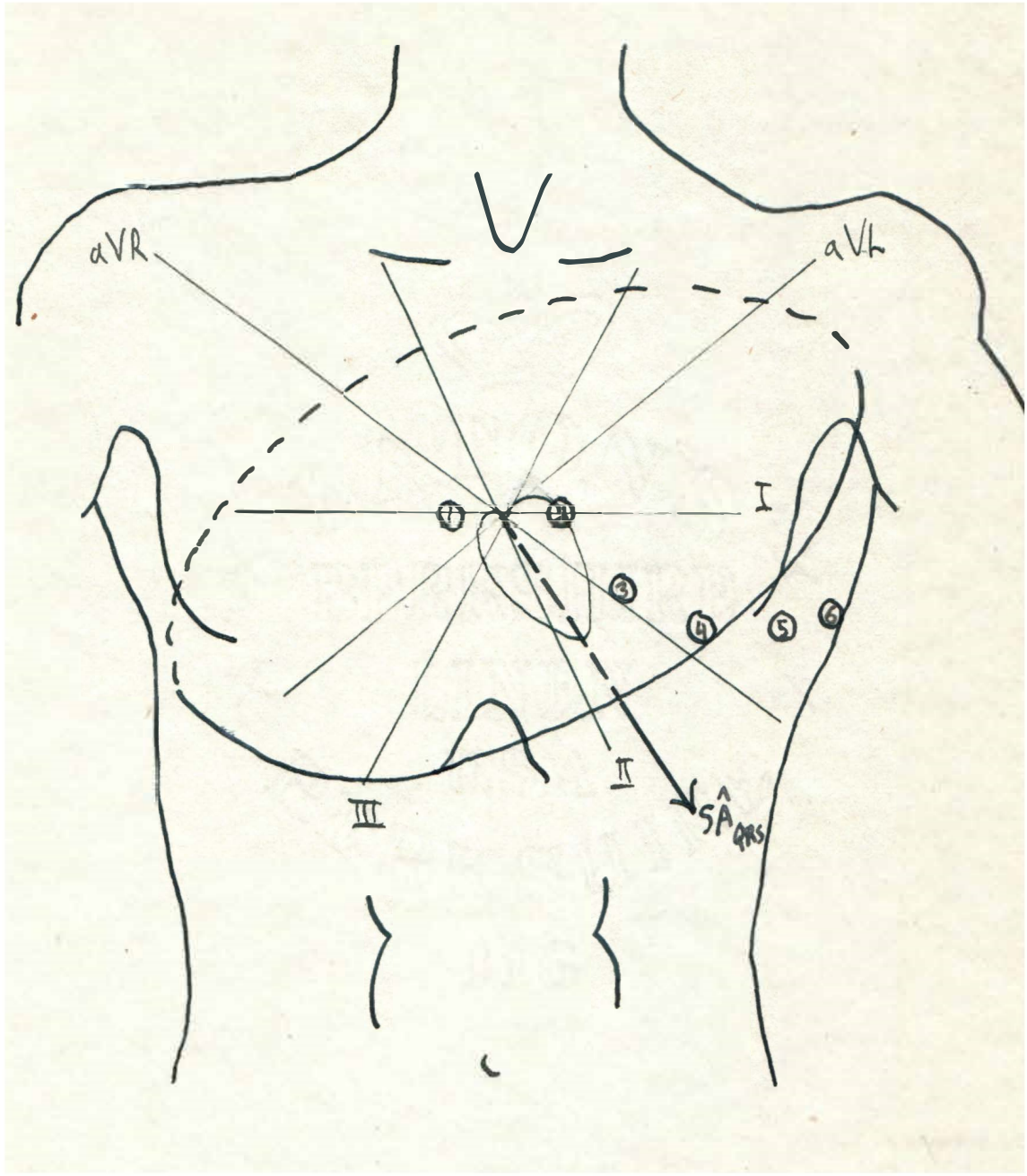

Fig. 44. Grant's method for finding vectors and loops by inspection. Subject X.

Wilson (55). (See Figure 45).

It can be seen in Figure 45 that depending upon the direction in which the loop is inscribed there are two possible electrocardiograms for each lead. Now while the resultant manifest QRS vector for each of these loops is the same, the electrocardiograms are not. Thus we can see that if we are given the electrocardiogram we can determine the normal loop on the frontal plane, but the reverse does not appear to be true, unless the direction of inscription is known. Fortunately, in the vectorcardiograms the direction of inscription is always shown by an arrow.

What is the significance of the difference in direction of inscription and in the two different electrocardiograms? We have already seen, that analyzed by the vector method, there was no difference on the frontal plane. The answer is that the difference in inscription and that in the electrocardiograms represents a different anatomical position for the heart which does not appear to be shown by the vector.

If we remember that the instantaneous vectors tend to exert a more pronounced effect where the myocardium is thickest, then the spatial QRS loop tends to follow that portion of the myocardium which is thickest at each

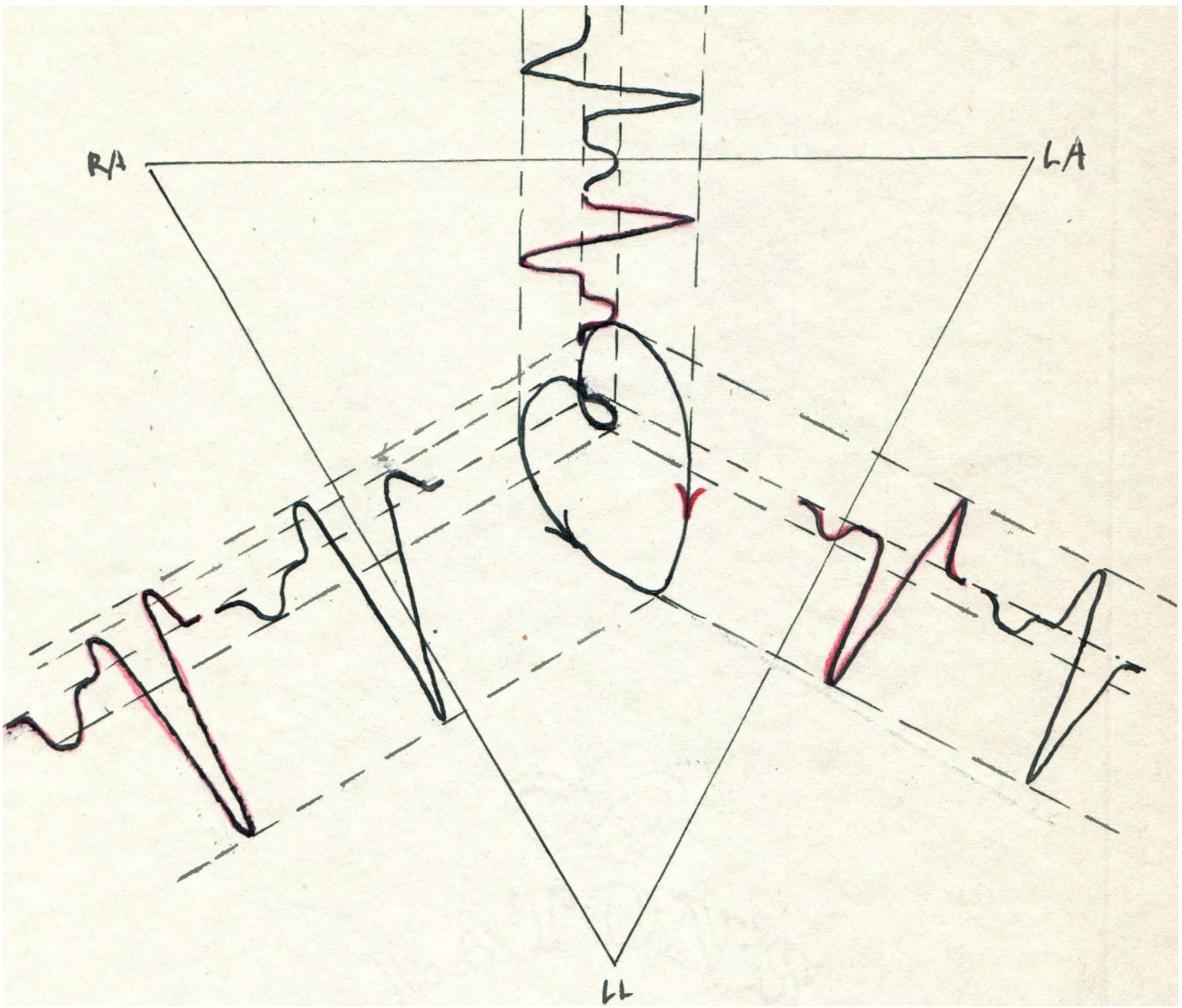

Fig. 45. Showing the effect of different directions of inscription in the manifest QRS loop on the QRS complex. This diagram also shows the relation of the loop to the bipolar limb leads.

given instant of the cycle. Now if this portion of the myocardium is rotated far enough on its longitudinal axis the inscription of the loop will be in the opposite direction. Thus Scherlis (48) says the frontal loop may have the direction of its inscription either clockwise or counterclockwise. One might think that there would be two possible normal electrocardiograms for the loop on each of the other planes, depending on the direction of its inscription. However, this is not the case because there is only one normal direction of inscription for the sagittal and horizontal planes. Scherlis (48) states that the normal direction of inscription is counterclockwise on the horizontal, and clockwise on the sagittal plane.

The direction in which the loop is inscribed on the frontal plane, if the loop were constructed from the electrocardiograms, and the direction was not known, could be inferred from a determination of the heart's anatomical position. The loop will tend to follow the thickest portion of the myocardium which may normally be inferred to be in the direction of the left ventricle. There are various means of determining this position.

Ashman (3) has shown the relationship of the anatomical and electrical axes. From the above discussion

it would not appear that the vector could be used to determine the heart's position but this is only because at least two vectors with their polar angles must be considered in order to determine position.

The normal vector directions and range of the polar andles must first be known for at least two planes. Next these directions and these angles for the heart under consideration must be determined. Then remembering that the heart may be rotated on three axes, i.e., the longitudinal, the anterio-posterior, and the transverse, the direction of rotation may be determined. Some of these values were given above, the remainder are to be found in Ashman's article (3). These values apply to the normal heart, but whether they apply or not in the determination of the abnormal heart's position cannot be discussed in this article.

Another method for determining the position of the heart is that of Goldberger (24) which is based on the precordial QRS complex pattern.

Grant discusses the relationship of the QRS patterns in the determination of the frontal loop. He states, "if the Q wave is conspicuously briefer in duration on one of these leads than the other two, the loop must next extend towards a point on the triaxial system

which is perpendicular to the axis of that lead. Then if the next part of the QRS wave on these three leads is an R wave, the loop will now extend to a point on the axis of the lead with the largest R wave, as far out along this axis towards the positive pole as the amplitude of the R wave indicates, and so forth (27)."

Grant also states that certain QRS deflections on the sagittal plane may be determined from V_1 and V_2. "For example, if in given case these deflections have an initial R wave followed by an S wave, the instantaneous vectors forming the first part of the frontal plane loop must also be directed anteriorly to the frontal plane of the body, and the vectors making up the remainder of the frontal plane loop must be directed posteriorly." If the biaxial reference system vectors have been calculated, one can draw the loop on this plane from V_F and the back (B) to LL lead, as shown in Figure 41.

On the horizontal plane, if the relationship of the precordial leads in the electrocardiograms is known (See Figure 47), an accurate loop can also be drawn. If now we reverse the procedure and derive the electrocardiogram from the spatial loop it can be seen from Figures 45 and 47, that for either bipolar or unipolar electrocardiograms the same rule applies.

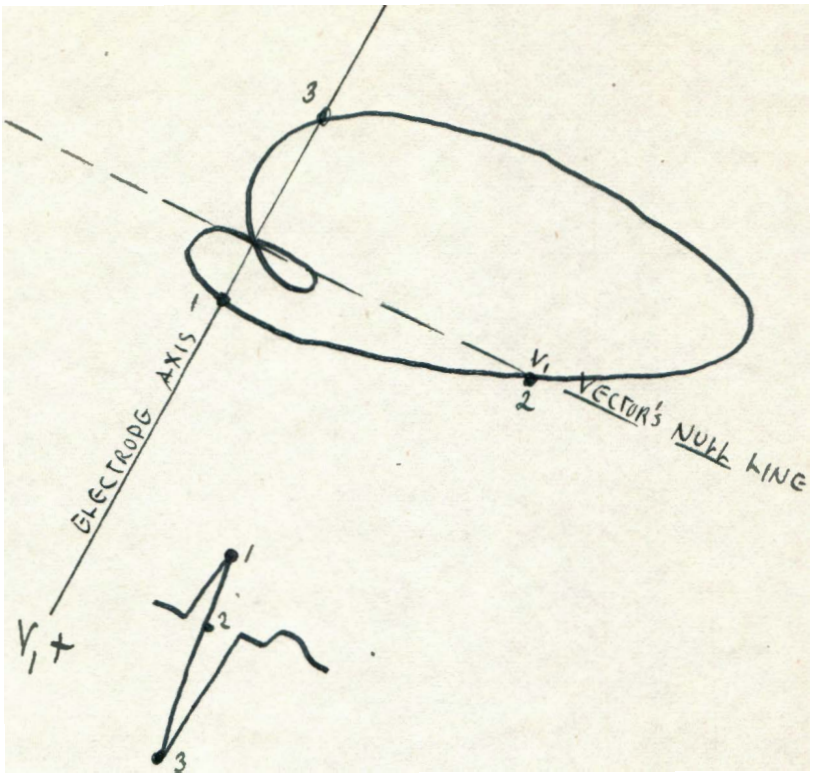

Fig. 47. Relationship of unipolar electrocardiogram to the horizontal loop.

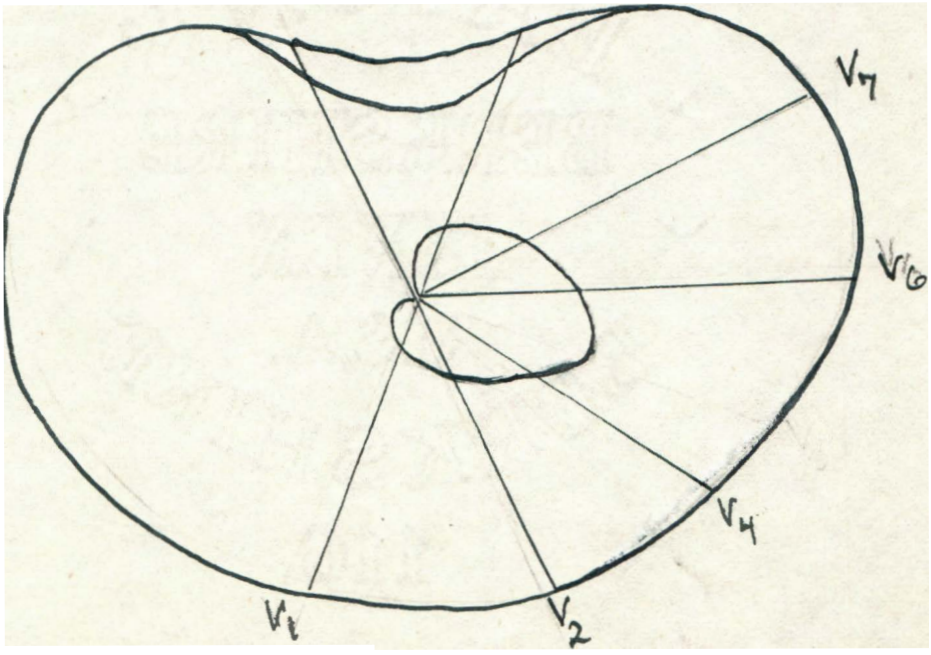

Fig. 48. Horixontal loop. Grant's method. Subject X.

Which is, draw a line passing from the positive electrode passing through the center of the heart (if bipolar electrodes are used, this line will terminate at the negative electrode). This is the electrode axis. Now draw a perpendicular to this line which also passes through the heart. This perpendicular represents the null line for the instantaneous vector which is parallel to the axis of the electrode. Therefore, any part of the loop on the positive side of the null line will write a wave on the electrocardiogram which is upright. The reverse holds true for portions of the loop on the negative side. The maximum deflection of the electrocardiogram will naturally be written by the instantaneous vector which is parallel to the electrode axis. The magnitude of this vector will be proportionate to the magnitude of the deflection. The reverse of this procedure will enable one to draw the loop from the electrocardiograms.

The relationship of the horizontal loop to the unipolar electrocardiograms is shown in Figure 48. The loops shown in Figures 44, 46, and 48 were taken from the electrocardiograms in Figures 42 and 43.

SUMMARY

The purpose of electrocardiography is to record

as accurate and as complete a picture of the electrical activity of the heart as is possible with the final aim of arriving at a precise diagnosis. After having obtained such a record in the most convenient manner, there are two methods by which such records may be interpreted. The older method is an empirical one and is based on the memorization of electrocardiographic patterns and deflection sizes. It came into being because the speed with which electrocardiographic records could be recorded far outstripped the advance of knowledge concerning their true nature. The second method is a more rational one which is individualized to each patient and which is based on understanding.

As a basis for understanding the newer methods, the dipole theories were analyzed, and that theory which seemed to explain all electrical phenomena known to date was adopted. The processes of depolarization and repolarization were explained in the light of this theory, and the origin of spatial vectors and loops was shown. Then the effect of abnormalities in the dipole activity was discussed and the resultant alterations in the vectors and loops was presented.

Finally, two rational methods of complete analysis of the electrical field of the heart were discussed.

The first is derived from the method of Grant and uses the spatial vectors and the spatial loop determined from electrocardiograms by the method of inspection. The second method is that which electrically records the spatial loop of Duchosal and Sulzer and which employs the vectorcardiogram. It has been most widely used in this country. This method eliminates the element of human error present in the first system and is more practical as regards the loop. However, the mean spatial vectors can only be poorly estimated by this method. Thus a combination of these methods is the most satisfactory for complete analysis.

Such a combined method would use the spatial vectors as determined by the first system, and the spatial loops as determined by the second. As shown by Grant the magnitude of these vectors is not as important as their direction, and since the direction of the vectors can be satisfactorily determined by inspection, calculations are not necessary. However, the spatial loop cannot be as easily determined by inspection and therefore, the method of spatial vectorcardiograms is more practical for determining the spatial loop.

Such methods can be used more rapidly than the more common method in use today of comparing electrocardiographic patterns in several leads and would be much

more individualized to the patient as well as more accurate since they are based on reason and understanding.

Either the first method of analysis by spatial vectors or the second of analysis by spatial loops could be used alone in those cases exhibiting an alteration of normal dipole activity, such as in infarction, hypertrophy, and bundle branch block. But, the latter method could not be used in those cases where the conduction system alone, instead of the myocardial dipole activity was at fault, such as in auricular fibrillation. However, if either method was used alone (in suitable cases), that of the spatial loop would perhaps be the easier, while at the same time more accurate and would obviate the use of multiple leads. But it would seem apparent that if one wishes to make a complete analysis of the heart for any condition whatsoever, and desires his analysis to rest upon reason instead of upon empiricism, he should use both the first method of spatial vectors and the second of spatial loops.

However, it should be understood that modern electrocardiographic methods give only extremely general notions of the electrical characteristics of the heart, and so, while no evaluation of the heart is complete without such electrocardiographic records, these records,

like all other laboratory procedures, should be evaluated in the light of clinical findings.

CONCLUSIONS

1. A dipole theory which explains all the known electrical phenomena of the heart and which is in accord with the mathematical and physical laws governing the electrical field of the heart is presented.

2. Two rational methods of complete analysis of the electrical field derived from such dipoles are discussed. These methods are based on spatial vectors and spatial loops.

3. The relationship of spatial vectors and spatial loops to all semi-direct and distant electrocardiographic leads is shown.

4. Thus, the interpretation of the electrical field of the heart may be based on reason and the accuracy of such an analysis is enhanced as well as is the simplicity of interpretation.

ACKNOWLEDGEMENT

The author wishes to express his sincere gratitude to Miss Anna Nielsen, R.N., ECG Department, Dr. A.R. McIntyre, Professor of Physiology and Pharmacology, and Dr. Arthur Dunn, Assistant Professor of Physiology and Pharmacology, all of the University of Nebraska College of Medicine for their interest and help.

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