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ON EINTHOVEN'S TRIANGLE, THE THEORY OF UNIPOLAR ELECTROCARDIOGRAPHIC LEADS, AND THE INTERPRETATION OF THE PRECORDIAL ELECTROCARDIOGRAM

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EINTHOVEN'S TRIANGLE

IN 1913, Einthoven, Fahr, and de Waart[1] published a method of estimating the direction and manifest magnitude, at a given instant in the cardiac cycle, of that component of the heart's electromotive force which is parallel to the plane defined by the standard limb leads. They utilized this method to study and to explain the modifications of the electrocardiographic deflections of these leads produced by respiratory variations in the position of the heart. The chief purpose which they had in mind seems to have been to find a way of distinguishing electrocardiographic phenomena due to extrinsic causes of this sort from those originating within the heart itself. More than thirty years have passed since this fundamental paper by Einthoven and his associates was written. No other has had so great an effect upon the development of our knowledge of the electrocardiogram; none has been the source of more inspiration; and none has been the subject of so much misunderstanding, so much critical examination, and so much controversial discussion. Why after all these years should there still be a wide difference of opinion regarding the correctness of the views expressed in this paper?

Unlike Einthoven and Fahr, the vast majority of those who have been engaged in the study of the human electrocardiogram have had small acquaintance

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with, and little interest in, mathematical attacks upon physical problems of the sort encountered in attempts to apply the classical theory of electricity to the analysis of the varying electrical field associated with the heartbeat. In theoretical investigations of this kind the actual situation under consideration is always far more complicated than any of those that can be treated mathematically, and it is necessary to make many simplifying assumptions that are not strictly in accord with the facts. To assert that all deductions based on such assumptions are *ipso facto* worthless is, so to speak, to deny that mathematics has contributed anything worth while to the physical sciences. To maintain, on the other hand, that deductions of this kind represent anything more than a first approximation to the truth or have any great value except in so far as they are supported by experience and by experiments designed to test their validity would be equally unreasonable. It is imperative that those who make use of conclusions of this sort as a guide to further investigations, or who attempt to extend them, clearly understand and constantly bear in mind the postulates upon which they rest.

Most of the controversies to which Einthoven's work has given rise seem to have originated in differences between the participants in respect to their familiarity with and their attitude toward its theoretical background. In our opinion, there is no reason to suppose either that Einthoven and his associates had any false notions as to the general character of the heart's electrical field or that they considered their method of determining the position of the electrical axis of the heart entirely free of error. In 1921, a paper by Lewis, Drury, and Iliescu[2] on the electrical axis of the auricle in clinical cases of auricular flutter raised a question as to the conditions under which the principles of Einthoven's triangle are applicable. A letter to Einthoven concerning this matter was answered by him on Nov. 21, 1921, as follows:

"In regard to the equilateral triangle I fully agree with you. I assumed in my original paper 'Ueber die Richtung und die Manifeste Grösse der Potentialschwankungen etc.,' in the center of the triangle a 'bipole,' that is to say two points lying very close together and showing a potential difference. The triangle was supposed to be a homogeneous sheet of conducting material and in regard to the distance between the two points of the bipole, of a large, let us say infinite extent.

"The applicability of this scheme to the ordinary leads of the human body depends indeed on the fact that the electrodes are at a relatively great distance from the heart. If they are placed near the heart the errors are greater and the more so the closer they get to the heart. Even in the case of the ordinary leads from the limbs the results cannot be absolutely exact."

A number of attempts have been made to test the validity of Einthoven's triangle by impressing a constant or variable voltage upon two metallic electrodes thrust into the heart of a cadaver and comparing the position of the electrical axis, computed by Einthoven's method from the potential differences recorded in the standard limb leads, with the direction of the impressed potential difference. The first experiment of this kind was performed by Fahr and Weber.[3] The heart was exposed and two small zinc needles were thrust into its

wall, one in the region of the sinus node and the other at the apex. When 1/5 volt was applied to these electrodes the deflection in Lead I was 10 mm., that in Lead II was 46 mm., and that in Lead III was 36 millimeters. The angle between the line defined by the two electrodes and the direction defined by Lead I was estimated at 75 degrees. The corresponding angle computed from the deflections in the three leads was approximately 3 degrees larger.

A similar experiment was performed by Wagner[4] on the cadaver of an infant who had died eight days after birth. In this instance two zinc needles were thrust through the precordial tissues into the heart and a potential difference of approximately 6 volts was impressed upon them. Three milliammeters were used to measure the resulting potential differences between the extremities. In the first test the currents in Leads I, II, and III were 6, 10, and 4 ma., respectively; when the input voltage was increased, these currents rose to 8, 13, and 5 ma., respectively. The chest was then opened, and it was found that one of the needles had entered the heart near its base, and the other entered just above the apex. The currents in the three leads were the same after opening the chest as before. When the electrodes were replaced so that the line defined by them made an angle of approximately 60 degrees with the direction of Lead I the currents in the three leads were 3, 6, and 3 ma., respectively. When the electrodes were arranged so that the line defined by them made an angle with the frontal plane, the currents in the standard leads were 3, 5, and 2 ma. when the projection of this line on the frontal plane was parallel to Lead II, and 4, 2, and −2 ma., respectively, when it was parallel to Lead I. This experiment and a large number of experiments on models of various types led Wagner to conclude (contra Groedel and Straub) that the theory of the equilateral triangle was in all respects well founded.

On March 1, 1934, Johnston, Kossmann, and Wilson[5] performed an experiment on the cadaver of a man who had died of carcinoma of the face complicated by pneumonia more than a week before. During the interim, the cadaver had been stored in the morgue in the supine posture, and it was suspected that in addition to pronounced post-mortem changes there had been some gravitation of the body fluids into the more dorsal tissues. The input electrodes consisted of two small brass rods, insulated except at the sharpened tips and fixed in a wooden frame. The frame permitted the rods to be moved endwise so that when they were thrust into the precordium the depth of the tip of each rod was independently adjustable. By means of a rotating contact breaker a potential difference of approximately 18 volts was rhythmically impressed upon these electrodes after they were in place. The thickness of the chest, measured from precordium to back, was 21 centimeters. The electrodes were first thrust through the chest wall in the third intercostal space, one just to the right and the other just to the left of the sternum. The depth of the tip of the former (the negative electrode) was 5.7 cm. and that of the tip of the latter (the positive electrode) was 8.8 centimeters. The deflections recorded in Leads I, II, and III measured 26, 12.75, and —13 mm., respectively. Moving the left leg electrode to the pubis had no appreciable effect. Increasing the depth of the positive elec-

trode to 10 cm. and decreasing the depth of the negative electrode to 5 cm. produced only very minor changes in the potentials of the three extremities, measured with respect to that of a central terminal connected to these electrodes and also to an electrode in the left interscapular region through resistances of 10,000 ohms. This procedure increased the positivity of the electrode on the back from 2 to 4.5 tenths millivolt.

When the positive electrode was in the third intercostal space near the left sternal edge with its tip 10.7 cm. below the skin, and the negative electrode in the fourth intercostal space and on the same vertical line but with its tip 5.5 cm. below the skin, the deflections in Leads I, II, and III measured 1.5, —30.5, and —32 mm., respectively. In this case, however, increasing the depth of the positive electrode to 15 cm. increased the deflection in Lead I to 12 and that in Lead III to —35 mm. and reduced the deflection in Lead II to —23 millimeters. The factor responsible for this unexpected result was not discovered.

These cadaver experiments by different workers support Einthoven's belief that when a potential difference is generated between two points lying within or close to the heart, the deflections in the three standard limb leads are very nearly proportional to the cosines of the angles made by the frontal projection of the axis of this potential difference with the corresponding sides of his equilateral triangle. It is, of course, true that the conductivity of dead tissues is by no means the same as the conductivity of living tissues. If experiments of the kind described could be performed on living subjects would the results be vastly different? In 1920, Wilson and Herrmann[6] made a crude test of the validity of Einthoven's triangle in the course of some experiments on dogs in which the heart was stimulated rhythmically for the purpose of studying its refractory period. The stimulus was the current delivered by the secondary coil of an inductorium when the circuit through the primary coil was broken. Sharp deflections representing the induction shocks were observed in the limb leads. A stimulating electrode was then attached to each terminal of the secondary coil and the two electrodes were thrust into the ventral wall of the heart, one near the base and the other near the apex, in such a way that the line joining them was nearly parallel to the long axis of the body. The deflections produced by the induction shocks in the limb leads measured 2, 16, and 14 mm., respectively, under these circumstances. When the electrodes were so placed that the line joining them was perpendicular to the long axis of the body, these deflections measured 9, 3, and —6 mm., respectively. Except for the response to those shocks which fell outside the refractory period, the heart continued to beat normally. Its ventral surface was exposed and the lungs were not fully inflated. We doubt that the string galvanometer was capable of recording the very brief induction shocks with great accuracy. Nevertheless, it will be noted that the direction and relative size of the deflections in the limb leads were about what would be expected on the basis of the principles of the equilateral triangle.

It is clear that Einthoven regarded the electrical field associated with the heartbeat, in so far as it is represented by the potential differences recorded by the standard limb leads, as approximately equivalent at any given instant to

that of a dipole or doublet located in a homogeneous isotropic medium of large extent. In all probability this view was suggested by a well-known theorem on the potential of a complex of electric charges distributed in a dielectric and enclosed by a spherical surface of the smallest adequate radius. The potential of such a complex at any point outside this surface may be expressed in the form of an infinite series of spherical harmonics. When the net charge of the complex is zero, the successive terms of the series represent the potential of a dipole, the potential of a quadrapole, the potential of an octupole, and the potentials of multipoles of increasingly higher order.[7] At points sufficiently distant from the center of the sphere the field may legitimately be regarded as closely approaching that defined by the first term alone, in other words, that of a dipole.[7]

Between the electrical field of a complex of charges of the kind described and the electrical field associated with the heartbeat, there is an obvious analogy. The sources and sinks of the heart's field corresponding to the positive and negative charges of the complex all lie within a circumscribed region: the smallest sphere in which the heart can be enclosed. The action current which flows out of any given cardiac fiber re-enters the same fiber in a neighboring region. Each source is, therefore, associated with a sink of equal strength, and it is clear that the cardiac field is not only comparable to that of a distribution in which the net charge is zero, but to a complex consisting of doublets only. Between an electrostatic field and the cardiac field there are, however, some obvious differences. In the first place, the latter, unlike the former, varies with the time. Nevertheless, the cardiac field at any given instant has always been treated as if it were stationary; the effects of induction have been neglected. The justification for this procedure lies in the low frequency of the cardiac currents, the relatively small size of the conductor involved, and the relatively small conductivity of the body tissues, and also in the results of experiments of the kind we have already described in which the distribution of variable currents of low frequency has been studied. In the second place, the heart is imbedded in a medium which is neither strictly homogeneous and isotropic nor infinite in extent. The effect of the requirements imposed by the boundary conditions involved is to superimpose upon the field of the cardiac sources and sinks, as it would exist in free space, the field of a layer of doublets at the body surface[8] and the fields that would be produced by the presence of a single layer of charge on every surface separating tissues of unlike conductivity. The double layer is required to annul the field of the cardiac sources and sinks outside the body and each of the single layers to make the product of the conductivity and the electric intensity normal to the boundary surface the same on both sides of it. The effect of the double layer will, in general, be greatest at the body surface and least at points most distant from it; the effect of each single layer will be greatest near the surface on which it lies. It is, of course, out of the question to compute the exact effect of the boundary conditions that must be met in the case of conductors like the body which are irregular in shape and complicated as regards the arrangement and electrical properties of their constituent parts. It is possible, however, to compute the field of a centric or eccentric doublet in a sphere made up of spherical

shells of specified conductivities. On the basis of such computations, of the available experimental knowledge of the specific conductivities of the body tissues, and of the results of experiments of the kind described in previous paragraphs it seems to us that Einthoven's views as to the nature of the heart's electrical field, in so far as they are expressed in, or may be inferred from, his published work, are still in accord with all the known facts.

UNIPOLAR LEADS

In 1932, Wilson, Macleod, and Barker[9] described a new type of electrocardiographic leads in which a central terminal connected through equal resistances to electrodes on the right arm, left arm, and left leg is paired with an exploring electrode placed on the precordium or upon any other part of the body. They held that leads of this kind are essentially unipolar in the sense that they record the potential variations of the exploring electrode with respect to a reference point which remains at very nearly the same potential throughout the cardiac cycle. It was shown that the sum of the differences in potential between any number of electrodes and a nodal point connected to these electrodes through equal resistances must be zero as a consequence of Kirchhoff's first law. The potential of the central terminal is consequently equal at every instant to the mean of the potentials of the electrodes on the extremities. On the basis of the assumptions upon which the equilateral triangle of Einthoven, Fahr, and de Waart is based plus the additional assumption that electrical forces of cardiac origin which are perpendicular to the plane of the standard limb leads have no significant effect upon the potential variations of the extremities, it was also shown that the potential of a central terminal connected through equal resistances to electrodes on the right arm, left arm, and left leg is not materially affected by the heartbeat and may be considered nearly constant throughout the cardiac cycle.

This conclusion promises to become the subject of a controversial discussion no different in character and not less lengthy than the one that has revolved around Einthoven's triangle. Several kinds of experiments bearing on its validity have been reported. Burger and Wuhrmann[10] mention that one of them compared the potential of the central terminal of Einthoven's triangle with that of other central terminals each connected to three electrodes equidistant from the heart and lying at the apices of a triangle enclosing it. No details are given, but it is stated that the differences in potential between the various terminals were negligibly small. Arrighi[11] is known to have carried out experiments of a similar kind. So far as we know his work has not yet been published, but all of his experiments that we have knowledge of yielded results comparable to those reported by Burger and Wuhrmann. We have performed one experiment of the same kind and the results of such experiments are predictable on the basis of Arrighi's published work. In his doctoral thesis[12] he described his experience with three leads which formed the sides of a sagittal triangle that enclosed the heart. One electrode was placed in the left submaxillary region close to the chin, the second 3 or 4 cm. to the left of the midpoint of a line joining the umbilicus with the center of the pubis, and a third in the left interscapular space, approximately at

the level of the spinous process of the seventh thoracic vertebra. In almost all of the more than fifty cases of various types that were studied, it was found that the voltage of the deflection recorded at a given instant in the cardiac cycle by leading from the electrode on the jaw to that on the abdomen was very nearly equal to the sum of the simultaneous voltages recorded in Leads II and III divided by the square root of 3. It is not difficult to demonstrate algebraically that whenever this is the case the difference in potential between a central terminal connected to the usual extremity electrodes and a central terminal connected to Arrighi's submaxillary and abdominal electrodes only cannot be appreciably greater than that between his abdominal electrode and the left leg electrode. Since these two electrodes are similarly situated with reference to the heart we may expect that they will always be at nearly the same potential. A lead from the central terminal of Einthoven's triangle to a central terminal connected to all three of Arrighi's electrodes will, therefore, ordinarily yield deflections similar to, but approximately one-third as large as, those obtained by leading from the first of these terminals to the electrode on the back.

The tracings obtained in the only experiment of this kind that we have carried out are reproduced in Fig. 1. In addition to the standard and the unipolar limb leads (taken by Goldberger's method) the following special leads (taken with the electrocardiograph at twice the normal sensitivity) are shown: (1) a lead from the central terminal of the Einthoven triangle to a terminal connected to all three of the Arrighi electrodes; (2) the same lead after the electrode on the back had been disconnected from the second terminal; (3) the same lead after reconnecting the electrode on the back and disconnecting the electrode on the jaw; (4, 5, and 6) leads from the central terminal of the Einthoven triangle to each of the three Arrighi electrodes in turn; (7) a lead from the same terminal to one connected through equal resistances to two electrodes, one on the left back near the base of the neck and the other just to the left of the sacrum; (8 and 9) leads from the same terminal to each of these electrodes in turn. It will be noted that the greatest potential difference between the central terminal of the Einthoven triangle and that of the Arrighi triangle did not exceed 0.15 mv and that the first of these terminals was negative with respect to the other. It should also be noted that the deflections of Lead $V_{T'} - V_T$ are similar to those of Lead $V_B - V_T$ but about one-third as large.

In the case of normal subjects, an electrode placed on the back directly behind the heart is ordinarily positive with respect to the central terminal of the Einthoven triangle throughout the greater part of the QRS interval. For the time being we may assume, therefore, that this terminal is normally slightly negative and that of the Arrighi triangle slightly positive when the sagittal component of the heart's electromotive force has an anteroposterior direction. By connecting these two terminals together or by connecting an electrode on the back to the central terminal of Einthoven's triangle we might perhaps obtain a reference point more nearly indifferent than either.

Several investigators have attempted to ascertain the magnitude of the potential variations of the central terminal of Einthoven's triangle by means of

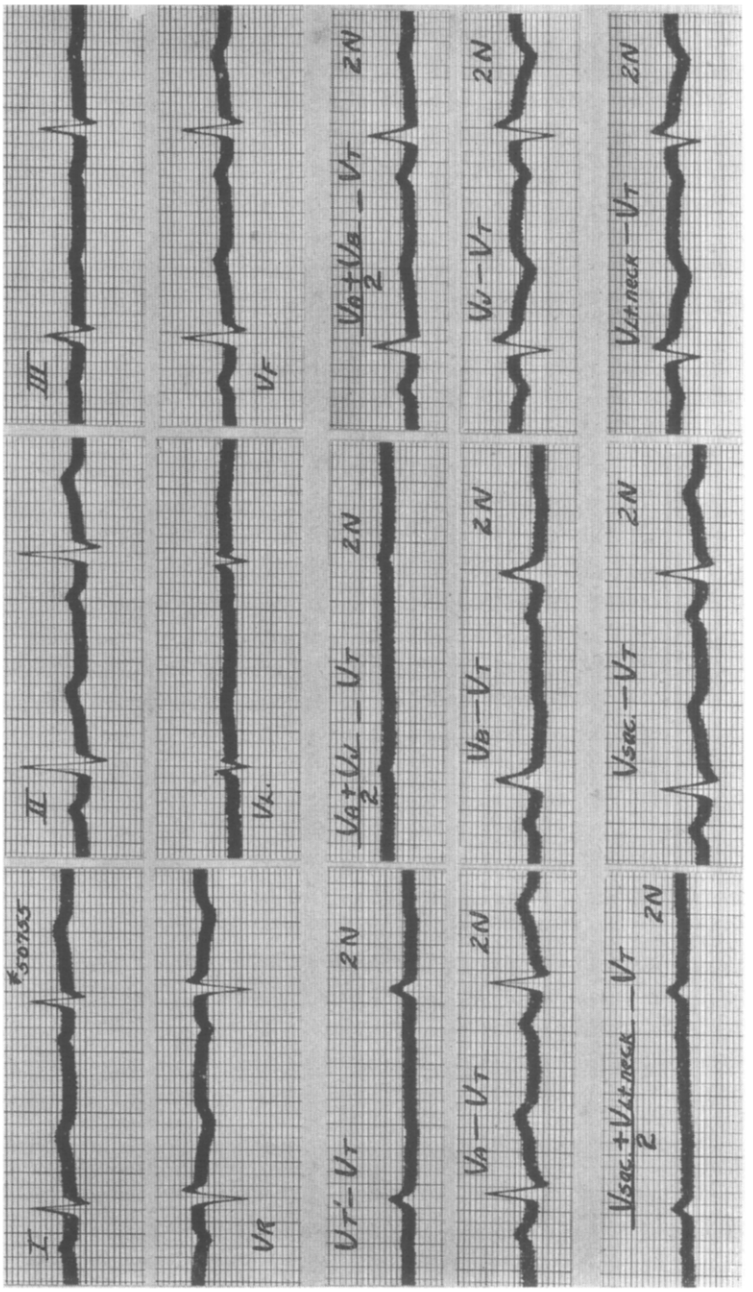

Fig. 1. - Comparison of the central terminal of the Einthoven triangle with that of the Arrighi triangle. A deflection of 2 cm. represents a voltage of 1 millivolt.

immersion experiments. Eckey and Fröhlich[13] placed their subjects in a large wooden tub lined with metal and filled with distilled water; contact between the subject and the metal lining was prevented by a suitable wooden support. The surface of the water was screened by a sheet of metal placed beneath it and in contact with the metal lining of the tub. The subject breathed through a glass tube brought out through a small hole in this metal lid; other small openings accommodated the electrocardiographic cables. The electrodes employed were not insulated. It was found that immersion of the subject in distilled water did not materially reduce the size of the deflections in the standard limb leads and that the cardiac field did not extend to the water outside the metal screen. The largest potential variations of the central terminal with respect to this screen were of the order of 0.2 to 0.3 mv in all of the unspecified number of experiments performed.

Burger[14] employed a tub lined with zinc and filled with tap water, and he did not immerse the face of his subjects. He insulated his electrodes from the bath with rubber sheeting. Immersion reduced the deflections of the standard limb leads to approximately 75 per cent of their original size. In five experiments on normal subjects the voltage of the largest deflection obtained by leading from the metal screen to the central terminal was about 0.26 millivolts. In four of the five cases the central terminal was slightly negative with respect to the zinc shield during the greater part of the QRS interval.

We have performed one immersion experiment of a somewhat different kind. After the standard limb leads had been taken, the subject was immersed up to the chin in a small fresh-water lake. The short-circuiting effect of the water reduced the size of the deflections in these leads to approximately one-half their original size (Fig. 2). There was also a slight change in the form of the ventricular complexes, probably because, when in the water, the subject was not able to assume exactly the posture in which the control curves were taken. The potential variations of the limb electrodes and the central terminal with respect to a large metal electrode suspended in the lake at a distance of about 11 feet from the body were recorded with an amplifier-type electrocardiograph at twice its normal sensistivity. The largest potential variation of the central terminal measured 0.15 mv; it was negative to the reference electrode (Fig. 3). The distant and the left leg electrode remained at practically the same potential throughout the cardiac cycle; we assume that in a series of experiments this would happen only rarely. Both arms and a point on the right scapula were negative with respect to the distant electrode during the greater part of the QRS interval.

Burger was uncertain as to whether the magnitude of the potential variations of the central terminal could be ascertained by the method which he employed for this purpose. Wolferth and Livezey[15] have expressed the opinion that "the reason advanced by Eckey and Fröhlich to support the claim that their immersion procedure can be used to obtain unipolar leads has no merit." The lack of agreement exemplified by this comment is basically similar in origin to the controversy between the proponents of the "negativity hypothesis" and the proponents of the "doublet hypothesis" which began some ten years ago. As the

years have passed it has become more and more apparent that the chief sources of this controversy are differences in point of view, in opinion as to the proper choice of a reference point for the measurement of bio-electric fields, and in the sense in which the word "potential" is employed between those who are mainly interested in the action currents of isolated nerves bounded by a dielectric and those who are mainly concerned with the action currents of the heart which is imbedded in a conducting medium.* Because many who do not understand the nature of the dispute have become uncertain as to whether unipolar precordial and unipolar limb leads are desirable and as to whether they are theoretically or practically possible, we have re-examined and attempted to clarify the ideas upon which the concept of an indifferent electrode is founded.

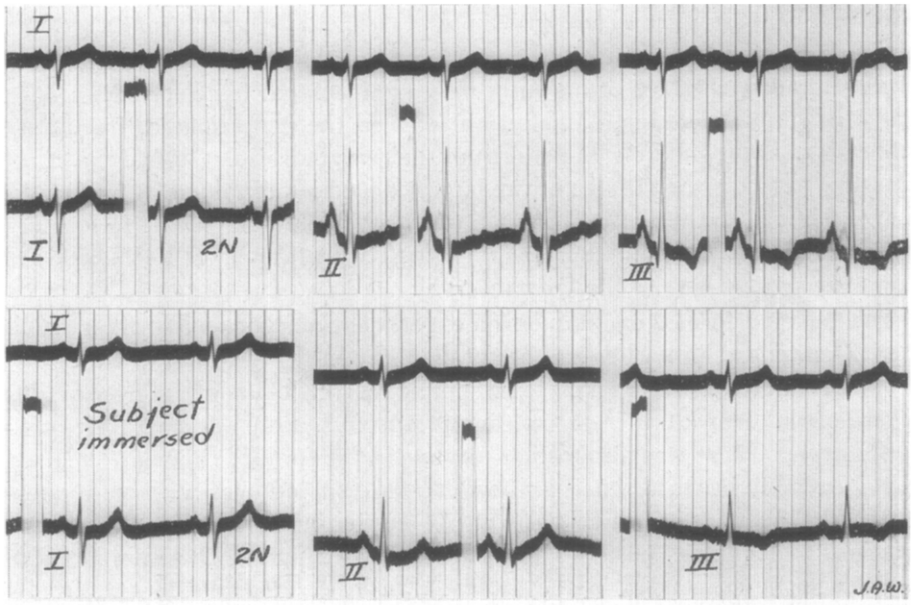

Fig. 2.—Electrocardiograms of a normal subject taken with the electrocardiograph at twice the normal sensitivity (2 cm. equals 1 mv).

The sequence of events and the considerations which led to the introduction of the central terminal for the purpose of obtaining unipolar leads are in outline quite simple. In 1916, Lewis and Rothschild[17] had difficulty in recognizing the "intrinsic" deflection in leads in which paired contacts were placed on the exposed ventricular surface. They attributed this difficulty to the arrival of the impulse beneath both electrodes almost simultaneously. To avoid it they left one electrode in place and moved the other, sometimes to another part of the heart's surface, and sometimes to the chest wall. They found the last procedure par-

*The champions of the "negativity hypothesis" focus their attention upon the action potential, or time-course of the voltage across the cell membrane during excitation and therefore choose an injured region which is incapable of responding to the excitatory process as their reference point. (For a discussion of such leads see Col and Curtis.[16]) Cardiologists who are forced to deal with the distribution of the cardiac action currents in a volume conductor are confronted by problems of an entirely different sort. They cannot apply the same principles to the interpretation of their tracings, must use the term "potential" in a different sense, and, consequently, must find another point of reference more useful for their purposes.

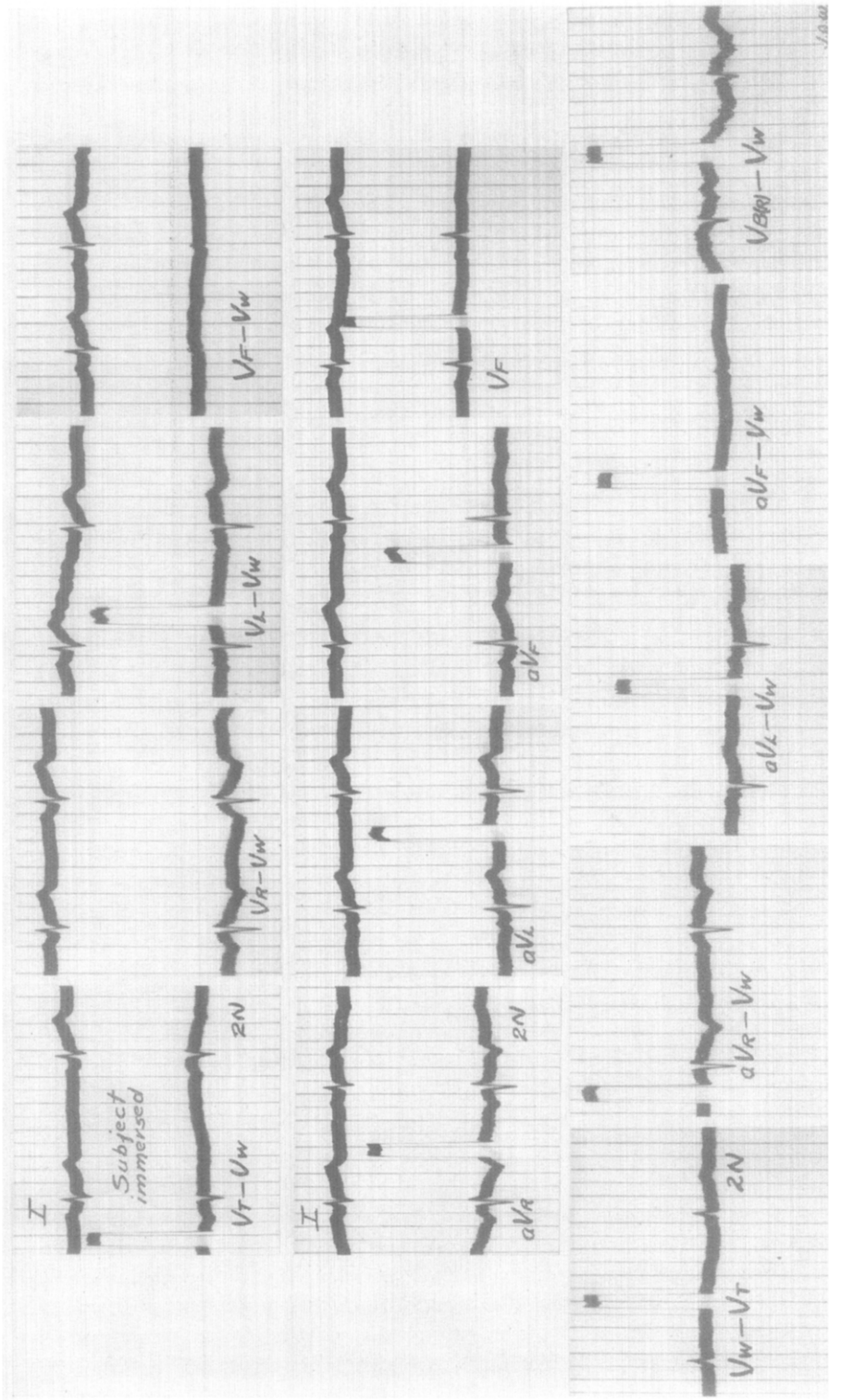

Fig. 3.—Records showing the potential variations of the central terminal and of electrodes on the extremities with reference to an electrode 11 feet from the subject. These tracings were taken with the electrocardiograph at twice the normal sensitivity. The symbols used have the following significance: V_R, V_L, and V_F represent the potentials of the electrodes on the right arm, left arm, and left leg, respectively, when connected to the central terminal through resistances of 5,000 ohms; aV_R, aV_L, and aV_F represent the potentials of these same electrodes when disconnected from the central terminal; V_T is the potential of the central terminal; V_W is the potential of the distant reference electrode; and $V_{B(R)}$ is the potential of an electrode on the right scapula. Leads from an extremity electrode to the central terminal are labelled V_R, etc., or aV_R, etc., in the usual way.

ticularly serviceable, and they clearly regarded the chest contact as without influence upon the position of the intrinsic deflection in the QRS interval. It is not certain that they considered this contact as indifferent in other respects; or that they believed the potential variations of this contact too small to have any significant influence upon the form of the tracings they obtained. In this laboratory the electrocardiograph is employed at one-twentieth of its normal sensitivity when leads of the kind in question are taken. So long as the distant contact is not placed close to the heart, its location can, therefore, have no important effect upon the size or form of the deflections recorded. On the other hand, moving the direct contact from one part of the heart's surface to another is almost certain to give the resulting curve an entirely different character. If we think of the cardiac field in terms of the current density, it is obvious that it is very intense in the vicinity of the epicardial surface, and, in comparison, of negligible strength in the neighborhood of the distant electrode. In the former region, the variations in the intensity of the field during the cardiac cycle are very large; in the latter they are very small. It is logical, therefore, when employing leads of this sort, to regard the potential variations recorded as characteristic of the region upon which the direct contact rests and to think of the distant electrode as indifferent and without influence upon the form of the curve; in other words, to consider leads of this kind unipolar.

In 1920, Wilson and Herrmann[18] performed the following experiment. A line was drawn from the fourth left costal cartilage to a point on the left leg just below Poupart's ligament. A small electrode (A) was placed at the upper end of this line and similar electrodes (B, C, D, and E) were spaced along its course at points 5, 10, 15, and 20 inches, respectively, below the first. With the electrocardiograph at half the normal sensitivity, Leads A-B, B-C, C-D, and D-E were then taken. The largest QRS deflection measured 20 mm. in the first, about 3 mm. in the second, and about 1 mm. in the third of these leads. No deflection of any kind was visible in the fourth. The results of this experiment suggested that, if an electrode on the central part of the precordium were paired with a contact at a considerable distance from the heart, the form and size of the ventricular deflections obtained would be nearly the same regardless of whether the second electrode were above, below, to the right of, to the left of, or behind this organ. The experiment was tried and this conclusion was confirmed.[19] Theoretical considerations and the resemblance in general contour between the ventricular complexes of leads in which a precordial electrode was paired with a contact far from the heart and those which Lewis and Rothschild obtained by leading from the epicardial surface of the exposed ventricles to some point on the chest wall led to the belief that leads of this kind are actually semidirect leads from the anterior ventricular surface, and this conclusion was published by Wilson, Wishart, and Herrmann[20] in 1926. A preliminary report of experimental and clinical observations bearing upon the value of such leads for the purpose of differentiating left from right bundle branch block was published in 1930 by Macleod, Wilson, and Barker.[21] The publication of the complete account[22] of these observations was postponed until the components of the human pre-

cordial curves which could legitimately be ascribed to potential variations of the distant electrode,[23] which had been placed on the left leg, could be computed and eliminated. The central terminal was introduced[9] with the object of accomplishing the same purpose more directly by reducing the potential variations of the reference contact to a minimum. This seemed desirable in order to make precordial leads of the kind in question as nearly unipolar, and therefore as nearly comparable to direct leads of the sort used by Lewis and Rothschild, as might be possible.

The central terminal is founded upon the idea that, so far as the limb leads are concerned, the electrical field of the heart is approximately equivalent to that of a dipole lying in or near the plane of these leads and that the principles upon which Einthoven's equilateral triangle is based are sound. If this view is tenable the potential of this terminal should remain at nearly the same level throughout the cardiac cycle. It is true that the sum of the potentials at the apices of an equilateral triangle enclosing a centric dipole which varies in strength will not remain constant unless the plane which passes through the dipole and is perpendicular to its axis separates the conducting medium involved into two identical parts. It is also true that the body is not symmetric with respect to any plane that passes through the heart. On the other hand, the magnitude of the effects produced by a lack of symmetry with respect to any such plane must decrease as the distances from the heart to the boundaries responsible for it increase. It was shown, for example, by Wilson[19] that when a coil of copper wire is placed in the field generated in a layer of electrolyte by a centric source and sink close together, the resulting modification of the field increases as the disturbing factor is brought closer to the region where the current density is maximal.

With respect to immersion experiments and the like, it is evident that factors which increase the asymmetry of the conducting medium surrounding the hypothetical cardiac dipole will tend to increase, and factors that have the opposite effect to decrease the potential variations of the central terminal with reference to a point that is completely indifferent. Placing the body in a lake or in a smaller body of water bounded by a metal screen, cannot change the location of the boundaries which define differences in tissue conductivity and it is hardly possible that it can significantly increase the flow of current across them. It does alter the heart's field by modifying the conditions at the body surface. The short-circuiting effect of the conducting fluid naturally reduces the potential differences between the various parts of this surface including those between one extremity and another and between the extremity electrodes and the central terminal. The magnitude of this effect is proportional to the conductivity of the water in which the body is immersed. The conductivity of distilled water is of the order of 2×10^{-4} mhos per meter and that of lake water and tap water is five to fifty times as great.[7] If the potential variations of the three extremity electrodes are reduced proportionately and in the same measure as the differences in potential between them, the potential variations of the central terminal will be diminished in the same degree.

Even when it has comparatively little effect upon the size of the deflections in the limb leads, as in the experiments of Eckey and Fröhlich, or reduces the size of all these deflections in the same proportion, as in those of Burger, immersion of the body will not have the same effect upon the potential variations of all three extremity electrodes if it brings about differences in their spatial relations with respect to the new bounding surfaces. In experiments of the kind performed by the investigators just mentioned, a contact or near-contact between one of these electrodes and the shielding metal screen would bring both to the same or nearly the same potential. The difference in potential between the screen and the central terminal would then become equal or nearly equal to the difference in potential between the latter and the extremity electrode concerned. In other words, the effect of the asymmetric arrangement of the electrodes would be to make the potential variations of the central terminal with respect to the screen larger rather than smaller.

Whether bringing one of the extremity electrodes very close to the screen would alter the potential of the former, that of the latter, or that of both depends upon what is considered the proper reference point for the measurement of the potential of the cardiac field. By connecting the electrode or the screen to earth the absolute potential of either could be maintained at zero. Since the conductivity of metal is roughly fifty billion times that of distilled water, the intensity of the electric forces produced by the heart must be infinitesimal outside the metal shield, and we agree with Eckey and Fröhlich that the potential of the screen should be considered completely indifferent with respect to the cardiac field.

Differences of opinion on questions of this kind have led to much confusion. Their source lies in the circumstance that the absolute potential of a given point on an isolated conductor in which electric currents are flowing is indeterminate unless the total charge on the conductor is known or the potential of one of its points has been fixed by grounding it. This difficulty arises because an isolated conductor may carry a static charge of unknown magnitude. Such a charge over its surface will raise or lower the absolute potential by the same amount at every point of the conductor but will have no effect upon the currents flowing through it. In the case of an infinite conductor this situation cannot arise. If the conductivity of the isolated conductor under consideration is large enough, we may think of it as in contact over its whole surface with an infinite conducting medium possessing a very much smaller conductivity and thus make it possible, at least theoretically, to choose infinity as our reference point for the measurement of the field.

The electrical field associated with the heartbeat presents some additional complications because it varies with time. We have been treating it as though, at any given instant, it had the same characteristics that it would have if it were not changing. Let us suppose, therefore, that there is a static charge on the body (or any conductor of which it is a part) which varies in magnitude from instant to instant, and that the inductive effects of this varying charge may be neglected. The potential variations produced by it will then be of the same magnitude at every point of the conductor and will have no effect upon the cardiac currents.

Potential variations of this sort are imposed upon the cardiac field by selecting some arbitrary point on the body and grounding it, or what amounts in effect to the same thing, making it the reference point for the measurement of the potential. It is obvious that if the potential of any chosen point was not constant before, and is constant after it has been grounded, this procedure must either impose upon every other point of the conductor variations in potential of the same absolute magnitude as those abolished, or alter the distribution of the cardiac currents. Connecting the body to earth does not have an effect of the latter kind* large enough to be detected by the electrocardiograph.

If one investigator places his reference electrode on a freshly injured spot on the ventricular surface and connects it to earth, he will arrive at the conclusion that all ventricular complexes represent a combination of two monophasic responses. Another who places his reference electrode on an uninjured part of the ventricular surface will not find this view attractive. In leads from all parts of the body surface each will record large complexes that are practically identical in form, and both will disagree with a third investigator who has placed his reference electrode as far from the heart as possible and believes that the magnitude of the potential variations produced by the heartbeat diminishes rapidly as the distance from the heart increases. As to the variations in the difference in potential between two specified points on the body surface, all will come to the same conclusion only if they compare them directly by leading from one to the other, for neither of the first two investigators will be able to estimate these potential differences by comparing leads from each of the two points to his reference electrode unless he makes use of a measuring machine. It is clear that the arbitrary choice of a reference point for the measurement of the cardiac field in terms of the potential, and also a purely empirical approach to the selection of the most useful bipolar leads, is likely to yield a harvest of confusion rather than enlightenment. We can, of course, give up the concept of the potential and think of the field of the heart in its vector form; that is to say, as a distribution of electric currents. Unfortunately, vector fields, in which three numbers must be associated with every point, are much more difficult to visualize and to analyze than scalar fields.

Three-dimensional fields of any kind, vector or scalar, are difficult to visualize unless they have some degree of symmetry. In the case of a field that has this property, it is profitable to fix the attention upon the point, line, or plane with respect to which the symmetry subsists. There is nothing to be gained by choosing a reference point for the measurement of the potential in such a way as to give the measured cardiac field a less symmetrical aspect than that which it has when expressed as a system of current lines and isopotential surfaces. If this is to be avoided the potential of the reference electrode should be the same as that of the point or points with respect to which the cardiac field is most nearly symmetrical;

*It would seem that in this case the fluctuating charge on the body is represented by a flow of charge into and out of a condenser of which the plates are the body and the earth. Both the capacity of this condenser and the resistance in series with it are small, so that the time constant of the circuit involved must be very short, and the static charge involved very small. There is no chance that the redistribution of the amount of electricity required to change the potential of the body with reference to the earth by a few millivolts could be detected by any instrument used to take electrocardiograms.

better still, if it were possible, the same as that of points far enough from the heart to be beyond an appreciable influence of this field. In the latter case the potential would be zero where the intensity of the field was negligible. The potential of the central terminal is the mean of the potentials of the apices of Einthoven's triangle and these are nearly as far from the heart as any other points on the trunk. If the field of the heart, so far as its least intense parts are concerned, may be regarded as nearly equivalent to that of a dipole located within the heart, the potential of this terminal is also that of the center of the dipole, the point about which the cardiac field is most nearly symmetric, provided that the electric forces perpendicular to the plane of the limb leads have no significant effect upon the mean potential of the extremities.

If the potential variations of the reference electrode are large, the ventricular complexes of all leads from regions where the cardiac field is considerably weaker and therefore varies less will be very much alike in form. The occurrence of strikingly similar complexes in leads from points that are widely distributed over the body and differ greatly in respect to distance and direction from the heart is a clear indication that the reference electrode is far from indifferent. If the cardiac field at points far from the heart is nearly equivalent to that of a doublet, leads from two points equidistant from this organ and at opposite ends of a line which passes through its center should yield complexes exactly opposite in character if the leads employed are unipolar. The average potential over a spherical surface, due to charges within it, is zero if the net charge is zero[24] as in the case of dipoles. It seems probable, therefore, that the average of the *cardiac potential** over the body surface must have a small value. If the reference electrode is indifferent and complexes of one kind are obtained from all parts of a region close to the heart, such as the precordium, complexes of the opposite type should be obtained from a still larger diametrically opposite region, such as the back, which is farther away from the heart. So far as we are able to judge from our experience with the central terminal, its potential is ordinarily close to the average of that of the body surface.

In concluding this discussion we may emphasize the fact that all of the available data which have a bearing upon the questions at issue are consistent. This is a very important consideration in estimating their significance. The cadaver experiments indicate that in spite of the irregular shape of the body and the somewhat eccentric position of the heart it is possible to ascertain the orientation of the frontal projection of the heart's electrical axis with considerable accuracy by Einthoven's method. All the immersion experiments that have been carried out gave substantially the same results. In the case of normal subjects the potential of the central terminal with respect to an electrode which bore essentially the same relation to all or nearly all parts of the body surface was slightly negative throughout the greater part of the QRS interval and did not vary through a range of more than 0.3 millivolts. The chief question that arises in connection with these experiments concerns the relative magnitude of the potential variations of the central terminal before immersion with respect to its potential

*We use this term to indicate the potential of the cardiac sources and sinks under the boundary conditions imposed upon the field to which they give rise.

variations after immersion. Due allowance has been made for the short-circuiting effect of the water on the basis previously indicated. That other factors, such as large variations in skin resistance from point to point, which might alter the magnitude of the potential variations in question when the subject was immersed could have had substantially the same effect in all the experiments performed seems very improbable. The observations on the difference in potential between the central terminal of the Einthoven triangle and that of the Arrighi triangle suggests that that the small negative potential of the former observed in the immersion experiments was due to the effect of electric forces having an antero-posterior direction. It is desirable to know the magnitude of the error involved in determining the inclination of the sagittal projection of the heart's electrical axis by Arrighi's method. This organ is closer to the anterior wall of the chest than to any other part of the body surface and its position with respect to his triangle is not precisely the same as its position with respect to the triangle of Einthoven.

THE INTERPRETATION OF THE PRECORDIAL ELECTROCARDIOGRAM

A comprehensive article[25] on this subject has recently been published from this laboratory. We propose here to supplement and not to repeat what was said in that article. We shall confine our remarks to a few examples of types of precordial electrocardiograms that have not been adequately discussed.

Incomplete Right Bundle Branch Block.—The electrocardiogram reproduced in Fig. 4 is that of an obese boy, aged 9 years, who had a speech defect and displayed evidence of general hypoplasia and mental retardation. The heart was not enlarged, no significant murmurs were heard, and the blood pressure was 96/40. There was no history of cyanosis at birth and none was present at the time of the examination. The limb leads show rather pronounced left axis deviation, a QRS interval which measures approximately 0.09 second, and both an R and an R′ wave in Lead III. Double R waves are also present in precordial leads V_1, V_2, and V_E. We believe that many precordial electrocardiograms of this kind represent incomplete right bundle branch block. We have encountered them frequently in all types of heart disease and also in many instances in which there was no other evidence of heart disease. Our interpretation of these curves is based on the occurrence of ventricular complexes of the same form in an electrocardiogram which was discussed in the article previously referred to (see Figs. 14 and 15 of that paper[25]). In that instance they alternated with complexes characteristic of complete right branch block, and the initial phases of the two types of complexes were identical in all leads. The difficulty is that one often sees an embryonic secondary R, that is to say, a conspicuous notch on the ascending limb of S, or a small terminal R′ deflection in Lead V_1 in cases in which there is not only no other evidence of heart disease but no increase in the QRS interval and no trace of a similar deflection in Lead V_2 or Lead V_E. The diagnosis of incomplete right bundle branch block must, therefore, be made with caution. We think that this diagnosis is more likely to be correct when the secondary R

wave is conspicuous and is present in Lead V_E as well as in Lead V_1 and the QRS interval measures at least 0.10 second in the limb leads. This diagnosis should not be made unless the R′ deflection rapidly decreases in size as the exploring electrode is moved toward the left side of the precordium as it invariably does in complete right branch block. In some cases Lead V_1 displays an unusually prominent R deflection with a prominent notch or slur on its ascending limb, and if the exploring electrode is moved farther to the right a broad bifid R or a final R′ deflection is recorded. This situation is illustrated in Fig. 5, in which is reproduced the electrocardiogram of a boy, aged 17 years, who had a very loud, rasping

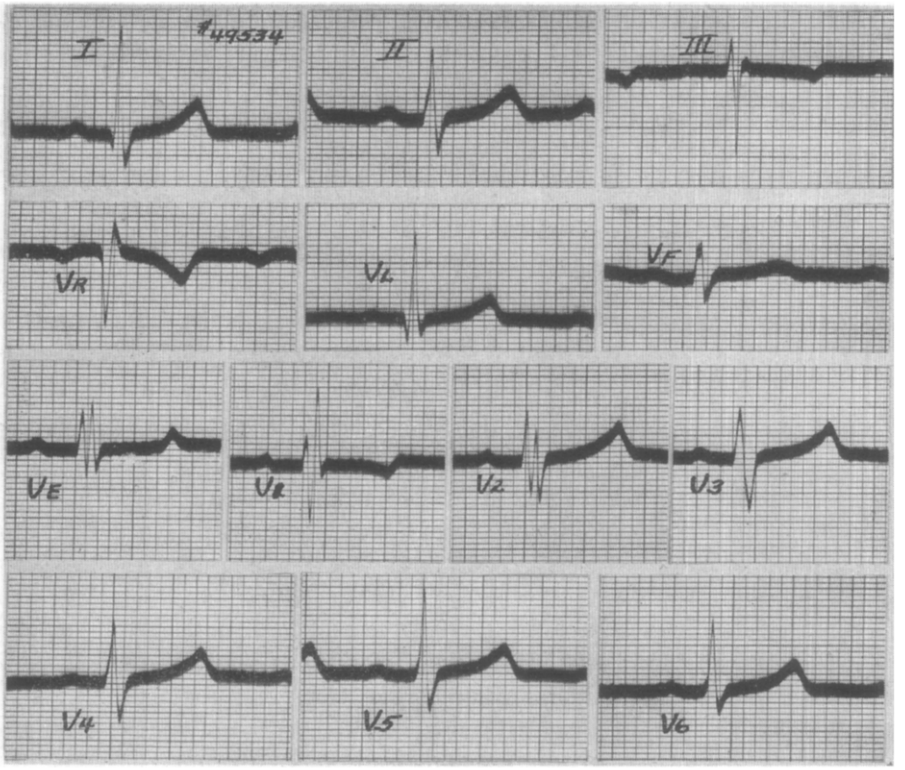

Fig. 4.—Incomplete right bundle branch block.

systolic murmur accompanied by a thrill in the pulmonic area. He was not blue at birth but a cardiac abnormality was noted a year later. There was no cyanosis and roentgenographic examination of the heart was negative. The position of the electrical axis, the small size of the R wave in Lead V_6 and in the leads from the left back (V_7 and V_8), and its large size in the leads from the right side of the precordium suggest right ventricular hypertrophy. However, this abnormality, which was suspected on clinical grounds also, does not satisfactorily explain the occurrence of secondary R waves in the leads from the right side of the chest.

Occasionally, we have seen precordial electrocardiograms which had all the characteristics of those that are diagnostic of complete right bundle branch block

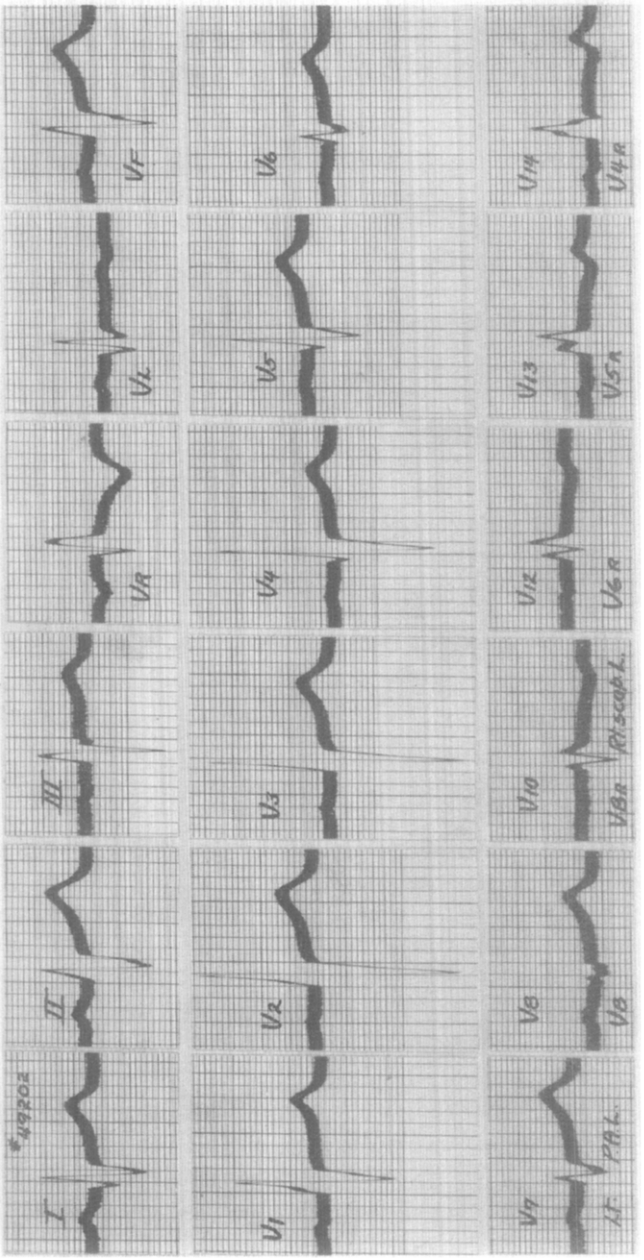

Fig. 5.—Somewhat questionable example of incomplete right branch block in a patient with a congenital heart lesion and right ventricular hypertrophy.

except that the QRS interval did not exceed 0.10 second. Most, if not all of these have been obtained in cases in which there were clinical reasons for supposing that the right ventricle was carrying an abnormally heavy burden. We are inclined to believe that such tracings represent the combined effect of right ventricular hypertrophy and incomplete right branch block, possibly resulting from the high pressures sustained by this chamber.

Incomplete Left Bundle Branch Block.—This condition is still more difficult to diagnose with confidence than incomplete right branch block. It is probable that it often gives rise to electrocardiograms that are indistinguishable from those considered characteristic of left ventricular hypertrophy. This opinion is supported by an observation made by Dr. John B. Levan. He has been kind enough to send us for teaching purposes the electrocardiogram of a young man who was able to engage in strenuous exercise and appeared to be healthy in every respect. Ordinarily, his electrocardiogram was of the normal type but on one occasion it displayed, off and on, sequential complexes showing pronounced left axis deviation and deeply inverted T deflections in Lead I. The QRS interval of these complexes was slightly longer than that of the normal complexes, and the transitions from the abnormal to the normal mechanism were abrupt. It is evident that disturbances in intraventricular conduction that behave in this manner must involve only a single strand of specialized tissue, for it is hardly likely that several bundles would always cease to function and always recover at the same instant. Transient incomplete left branch block seems, therefore, to be the logical diagnosis in this case.

The electrocardiograms reproduced in Fig. 6 are those of a man, aged 49 years, whose blood pressure had been extremely high for a period of at least five years and who died of congestive cardiac failure in June, 1944. The first tracings, taken on May 23 of that year, are quite characteristic of complete left bundle branch block. The QRS interval measures approximately 0.17 second. On May 29, however, the QRS interval had decreased to between 0.09 and 0.10 second although the QRS deflections of the limb leads still showed conspicuous slurring and notching. The precordial curves of the same date are similar to those obtained in many cases of hypertensive heart disease. Note, however, that no Q wave is present in either Lead V_5 or V_6. We have observed the same sequence of events in a number of other instances. The question arises as to whether the second set of curves represents incomplete left branch block, some other conduction defect, left ventricular hypertrophy alone, or a combination of the last two. If the first is the case, the earliest phases of the QRS complex of the same lead should have exactly the same outline in both sets of tracings. Unfortunately, this valuable criterion is often less useful than might be expected. The delay in the activation of the left ventricle may be nearly as great in incomplete as in complete left branch block or it may be very slight. If the latter is the case, the decision must be made on the basis of the form of the QRS complex during the first 0.01 or 0.02 second of the QRS interval; it will be noted, in the present instance, that in Leads V_1, V_2, V_3, and V_4 the resemblance between the earliest phases of QRS in the two sets of curves is pronounced. In Leads V_5 and V_6,

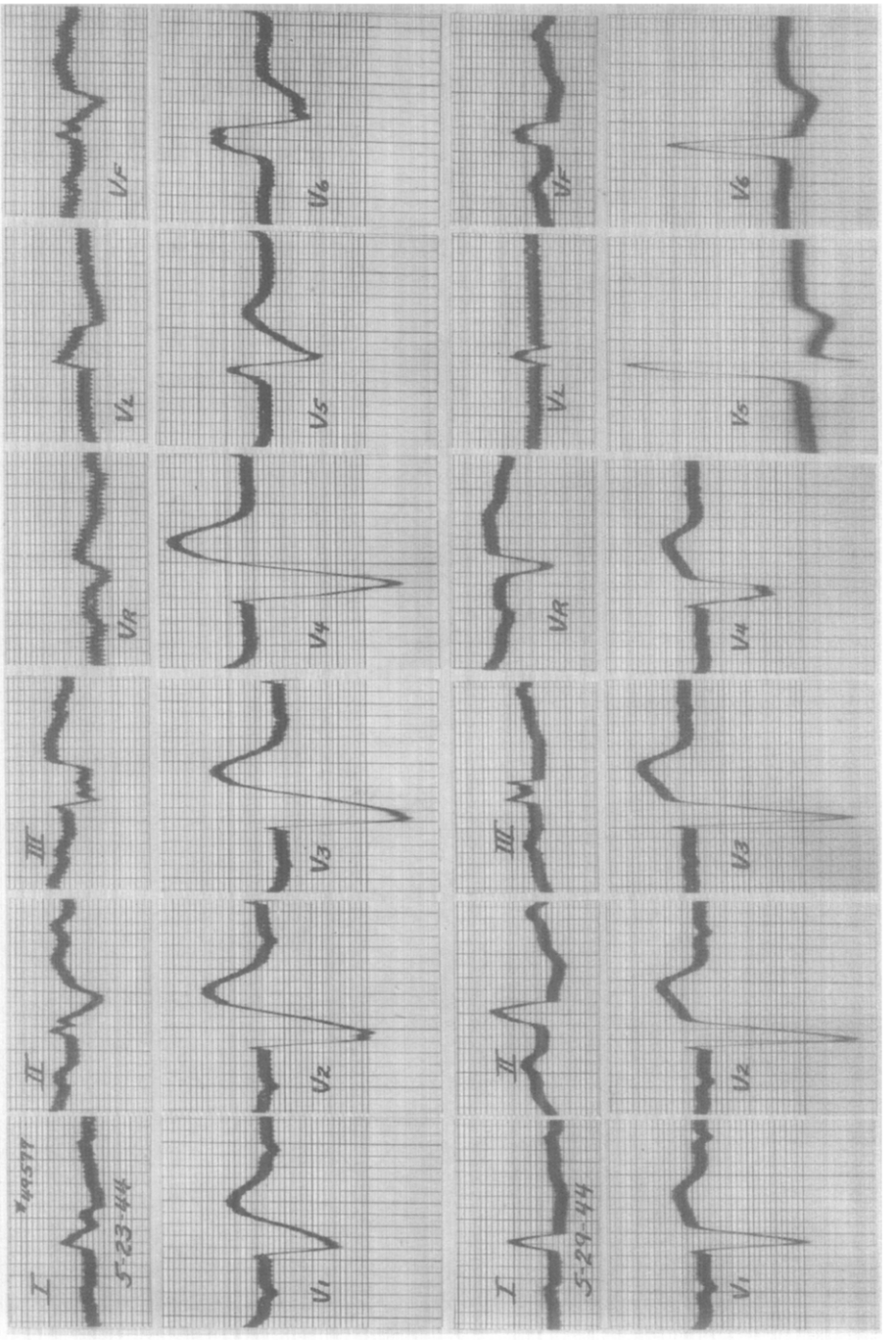

Fig. 6.—Transient complete left bundle branch block in a case of left ventricular hypertrophy, possibly complicated by incomplete left bundle branch block.

the R wave begins with a slowly rising portion in the curves of the second set, but the slope of this initial component appears to be much steeper than the corresponding part of the R wave in those of the first set. About the only thing that can be said is that if the last set of tracings represents left ventricular hypertrophy plus incomplete left branch block, the delay in the activation of the left ventricle caused by the latter was slight. If a Q wave were present in the second set of leads from the left side of the precordium, the presence of this conduction defect could be ruled out with reasonable certainty.

In some cases in which incomplete left branch block is suspected, the precordial and extremity curves are like those of complete left branch block in every respect except that the QRS interval is less than 0.12 second.

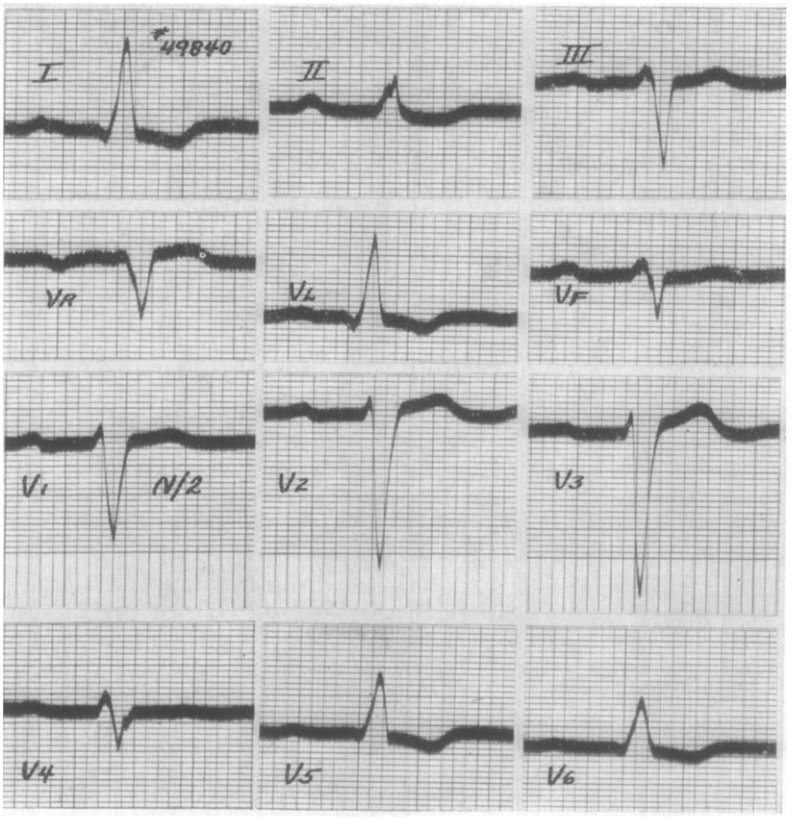

Fig. 7.—Left ventricular hypertrophy possibly complicated by a defect in intraventricular conduction.

Left Ventricular Hypertrophy.—A problem closely related to the one just discussed is presented by the electrocardiogram reproduced in Fig. 7, which is that of a man, aged 35 years, who had mitral stenosis, aortic regurgitation, and pronounced cardiac enlargement. The standard limb leads show conspicuous left axis deviation and inversion of the T wave in Leads I and II. The P-R interval is abnormally long and the QRS interval measures 0.12 second. Because

of the large voltage of the QRS deflections, the precordial leads were taken with the electrocardiograph at one-half its normal sensitivity. There is a conspicuous Q wave in Lead I and a small Q in Lead V_6. The R wave of the last of these leads is not broad-topped or bifid, as it usually is in complete left branch block, but there is some slurring and notching of the QRS deflections of the limb leads. The large voltages recorded in Leads V_1, V_2, and V_3 strongly support the diagnosis of left ventricular hypertrophy, but was this condition present alone, in combination with complete or incomplete left branch block, or in combination with some other conduction defect? In our opinion, the presence of a Q in Lead I and particularly in Lead V_6 plus the absence of a broad-topped or bifid R wave in the latter make the second possibility very unlikely. It is difficult to decide between the other two.

The electrocardiogram reproduced in Fig. 8 is that of a physician, aged 29 years, with mitral stenosis, aortic insufficiency, and pronounced cardiac enlargement. The limb leads show slight right axis deviation and changes in the P waves of the type commonly associated with an advanced mitral lesion. The P-R interval is slightly prolonged and the QRS interval measures approximately 0.105 second. The QRS deflections are slurred. The precordial curves are much more like those seen in left ventricular hypertrophy than like those associated with extreme right ventricular hypertrophy. The voltages of the deflections are not, however, extremely large and the T waves are normal. This electrocardiogram represents either auricular hypertrophy plus left ventricular hypertrophy, or plus hypertrophy of both ventricles. An increase in the QRS interval is rarely encountered in the electrocardiograms which are typical of preponderant right ventricular hypertrophy.

Pulmonary Embolism.—The electrocardiograms shown in Fig. 9 are those of a woman, aged 39 years, who was subjected to a subtotal hysterectomy plus appendectomy on May 26, 1944. On June 6 it was noted that Homans' sign was present, and on the following day at 8 P. M. the patient had a severe attack of chest pain accompanied by faintness and dyspnea. The blood pressure fell to 70/50. The first electrocardiogram was taken at 9:40 P. M. on June 8 and the second was taken at 4:45 P. M. on June 9. The patient died about five hours later and the post-mortem examination showed massive pulmonary embolism, pulmonary arteriosclerosis with organizing and recanalized thrombi, and some active pulmonary arteritis. The heart was not grossly abnormal. The two sets of limb leads are very similar; both show prominent S waves in Lead I and rather conspicuous Q waves in Lead III. The T waves are pointed in Leads II and III and there is a sharp bend in the initial limb of the T complex in Leads I and III. The QRS interval is a little longer in the second set of curves.

The two sets of precordial curves are very different. The first set is notable chiefly for the slight downward RS-T displacement in Leads V_3, V_4, and V_5 and for the sharp angulation of the ascending limb of T in these leads. The second set shows late R waves in Leads V_E and V_1 and sharply inverted T waves in the same leads and is strongly suggestive of incomplete right bundle branch block. It is well known that transient complete right branch block often occurs

in pulmonary embolism and that it is frequently followed by incomplete right branch block of gradually decreasing grade. In many cases, a conduction defect of this sort may account for all of the electrocardiographic abnormalities present. In the present instance, however, there were changes of the kind that have been considered characteristic of pulmonary embolism at a time when the precordial leads showed no evidence of a defect in conduction of the kind in question.

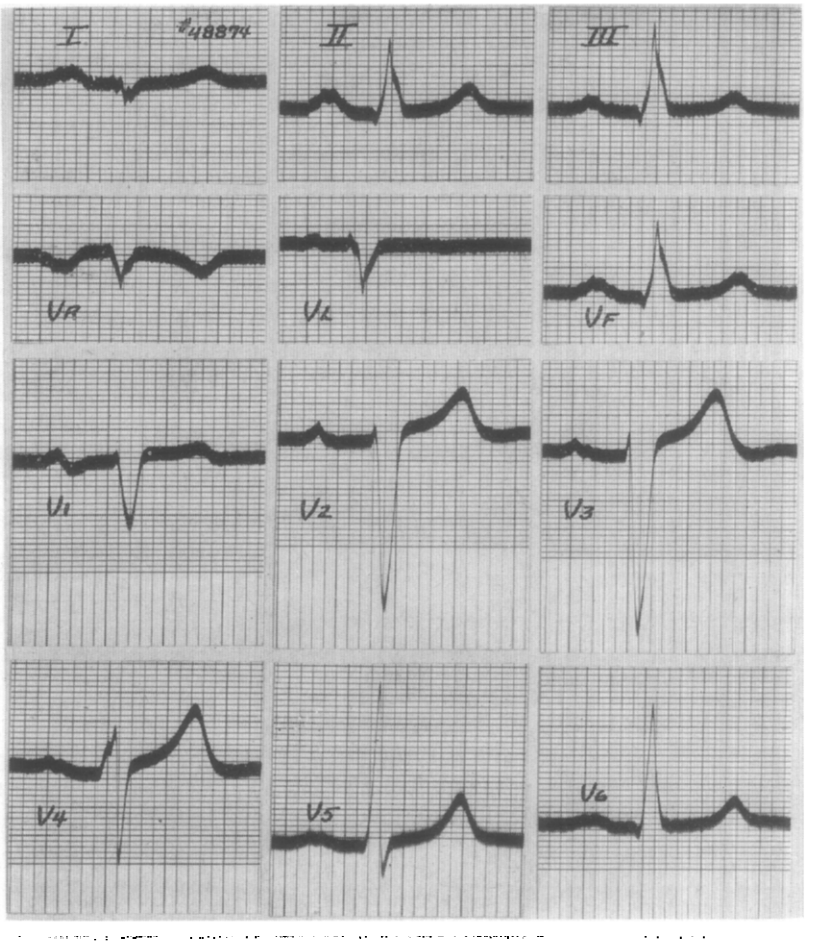

Fig. 8.—Left ventricular hypertrophy or hypertrophy of both ventricles.

Anterior Infarction.—When the anterior wall of the left ventricle is infarcted the resulting changes in the QRS and T complexes are seldom more pronounced in Lead I than in precordial lead V_5. If the anteroseptal wall of the left ventricle is involved, the diagnostic electrocardiographic signs are usually confined to one or more of the first four precordial leads and the complexes of the limb leads are either of the normal type or show modifications of the T waves only. If the anterolateral wall is involved, diagnostic changes are present in Lead I and Lead V_L and in some combination of the precordial leads which includes Lead

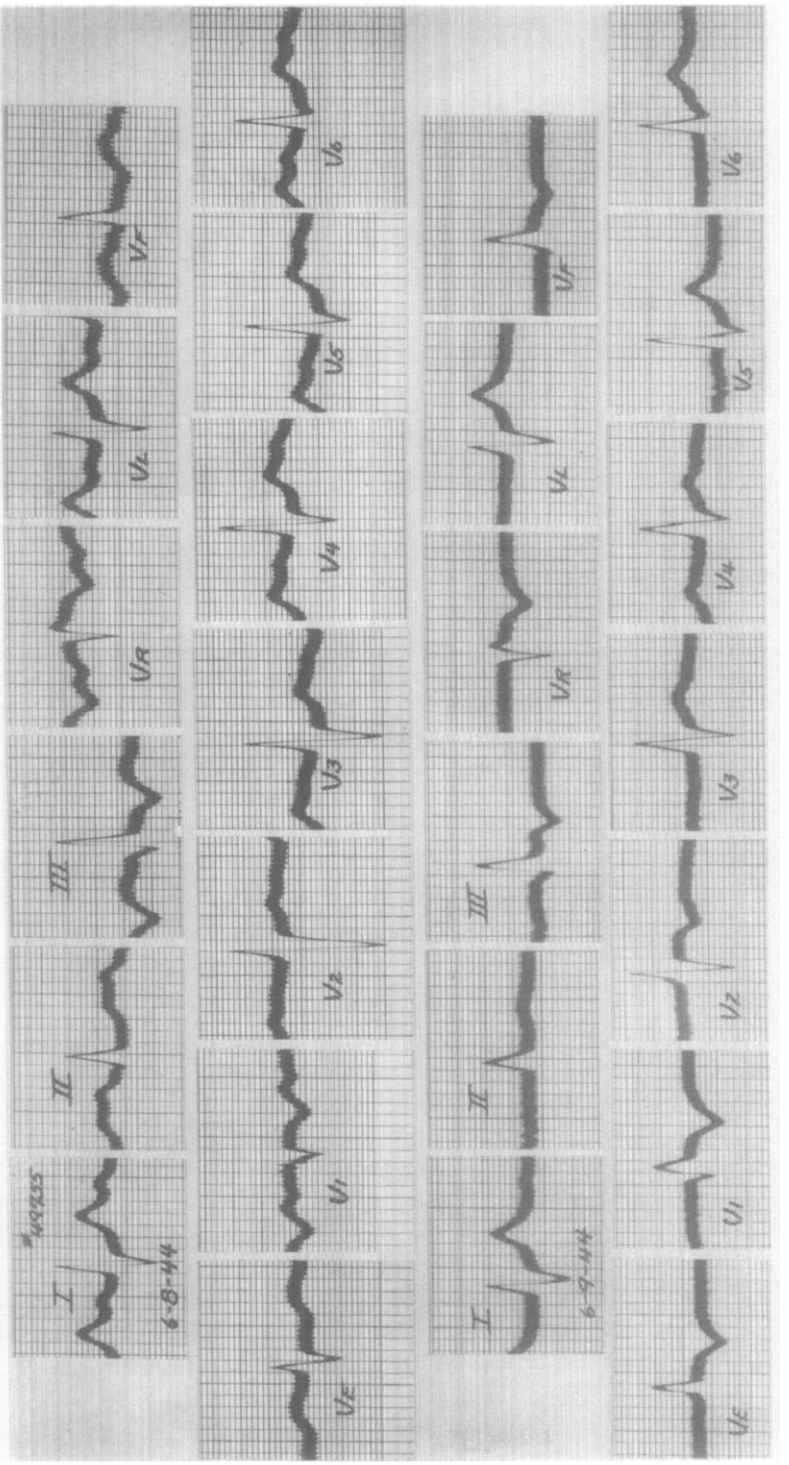

Fig. 9.—Pulmonary embolism.

V_5. There are, however, some striking exceptions to these general rules. We have seen, for example, conspicuous flattening of the T waves in Lead I, terminal inversion of this wave in Lead V_L, and a large pointed positive T wave in Lead III when the complexes of Leads V_1, V_2, and V_3 were diagnostic of infarction and those of Leads V_4, V_5, and V_6 were normal in every respect. More interesting still are those cases in which the complexes of Lead I are diagnostic or very strongly suggestive of anterior infarction while those of the precordial leads are either of the normal type or show only minimal changes of the kind characteristic of this lesion.

The electrocardiograms reproduced in Fig. 10 are those of a man, aged 41 years, who gave a history of severe attacks of chest pain in 1943 and developed a persistent left hemiparesis in May of that year. He had been told that his blood pressure was elevated, but at the time when he was first seen it was only 120/80. The heart was slightly to moderately enlarged; no murmurs were heard. There were no signs of congestive cardiac failure. The extremity curves show conspicuous Q waves and terminal inversion of the T waves in Lead I and Lead V_L. The usual precordial leads are negative except for low R waves preceded by tiny Q waves in V_3 and V_4 and terminal inversion of the T waves in V_3, V_4, and V_5. The leads taken from higher levels, particularly those from the 3rd and 4th intercostal spaces in the left midclavicular and the left anterior axillary line, show considerably more striking changes. The electrocardiograms of this patient differ from those attributed to high lateral infarction in a previous report.[25] The latter showed unusually large R and T waves in the leads from the right side of the precordium. Such changes suggest posterior rather than anterior infarction.

Posterior Infarction.—In some cases of posterior infarction in which there are abnormally large Q waves and sharply inverted T waves in Leads II, III, and V_F, the same kind of changes are present in Lead V_6. The leads from the right side of the precordium may, or may not, display unusually large R waves and tall pointed T waves. Tracings of this kind have been ascribed to infarction of the posterolateral wall of the left ventricle.[25]

The electrocardiogram reproduced in Fig. 11 is that of a man, aged 61 years, who was first seen on June 18, 1944. There was a history of severe chest pain which radiated to both arms in November, 1943. A diagnosis of coronary thrombosis was made at that time and the patient remained in bed for eight weeks. Some days before he was brought to the hospital he had a second attack of chest pain following moderate exertion. A short time after this, tarry stools were noted. At 4:00 A. M. on June 18 he was awakened by severe pain in the region of the left scapula, through the chest, and in the left abdomen. When he was examined some hours later the blood pressure was 70/50, the pulse rate was 130 per minute, and the rectal temperature was 102° F. The heart was enlarged, the heart sounds were faint, and no murmurs were heard. The abdomen was somewhat rigid and tender on the left side. Death occurred at 3:35 P. M. on June 19, shortly after another attack of severe pain in the chest. The location of the infarcted regions disclosed by the post-mortem examination is shown by the sketch reproduced in

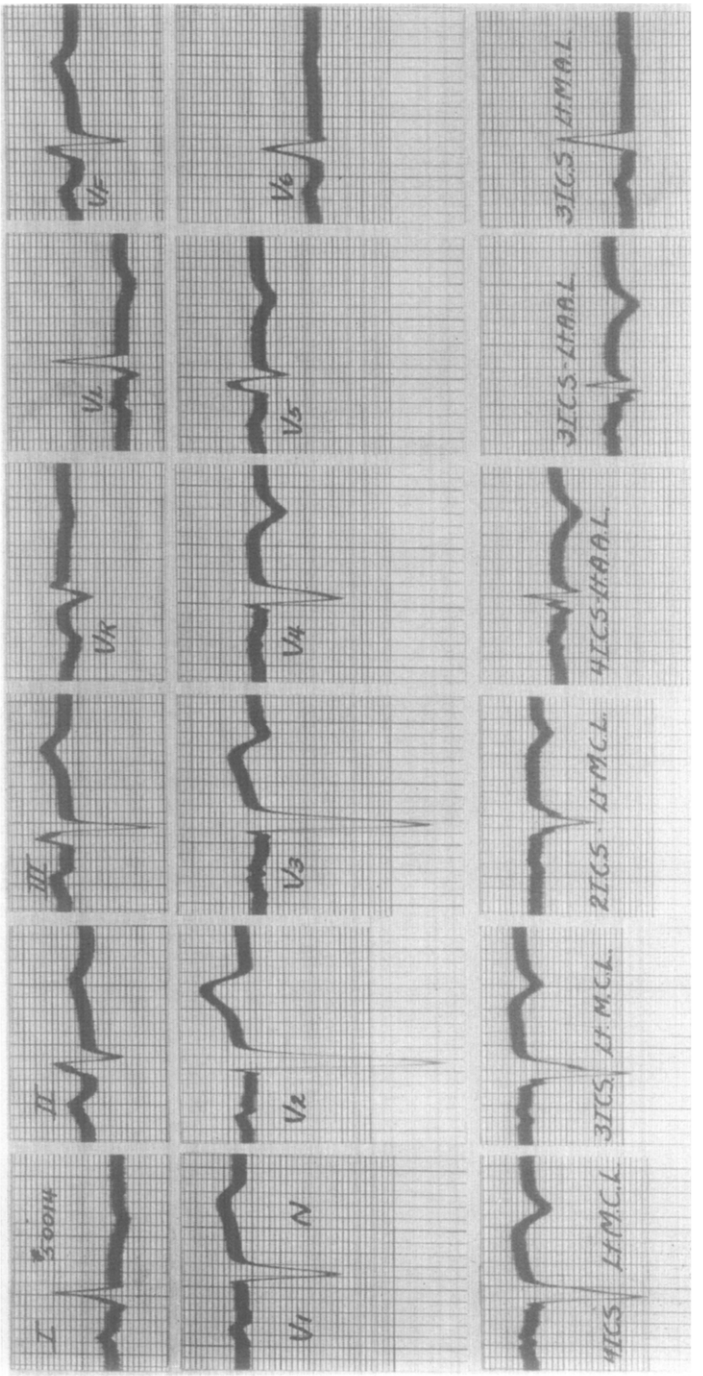

Fig. 10.—Anterior infarction with pronounced signs of infarction in precordial leads from points at higher levels than those from which the standard precordial leads are obtained.

Fig. 12. The autopsy findings included a perforating gastric ulcer complicated by a subphrenic abscess and fibrinopurulent peritonitis.

The electrocardiogram shown in Fig. 13 is that of a man, aged 46 years, who had chest pain of short duration on Sept. 10, 1944. On the following day he had a second attack which lasted one hour. He was then kept in bed, and was told that his blood pressure was 200. He continued to have pain and on September 16 had an unusually severe attack. At the time of the physical examination on the same day, he was still complaining of pain. The blood pressure was

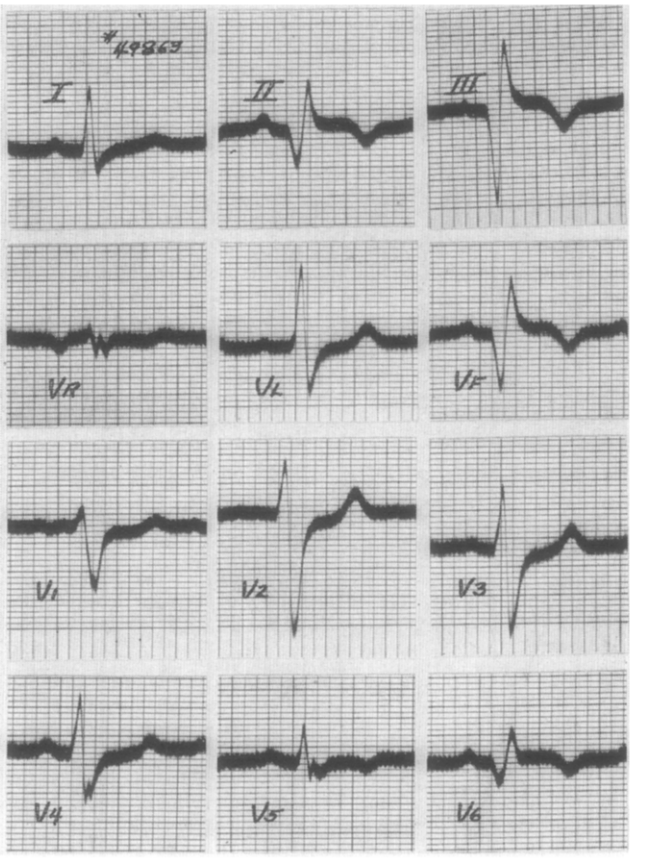

Fig. 11. Posterolateral infarction.

150/90 and the white blood count was 11,000 per cubic millimeter. The abdomen was distended and tender. The heart was not enlarged, but subsequent roentgenographic studies showed some broadening of the aorta. There was a past history of intermittent claudication, and no pulsation could be felt on palpation of the left dorsalis pedis artery. The patient made a good recovery from the coronary accident.

The first electrocardiograms taken on September 18 were considered within normal limits. There was a slight flattening of the T waves in the limb leads and a slight concavity of the RS-T segment in Leads V_1 and V_2. A number of tracings

taken during the next few days were of similar form. On September 27, however, there was a sharp dip at the end of the T wave in Lead I. The precordial electrocardiogram of the same date shows large pointed upright T waves in Leads V_1, V_2, and V_3 in which these waves had previously been small, and terminal inversion of T in Lead V_6. More striking inversion of T is present in leads from a high point in the left axilla, from the left posterior axillary line at the level of the fourth costosternal junction, and from the left scapular region. These findings suggest that the infarct was on the posterolateral wall of the left ventricle well toward the base. This case illustrates the desirability of taking serial electrocardiograms when the first tracing is negative and of caution in ruling out infarction on the basis of the absence of characteristic electrocardiographic changes.

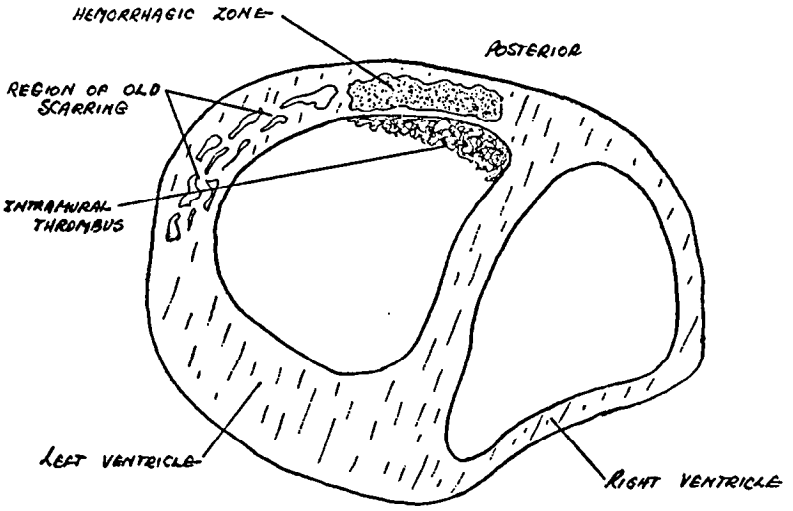

Fig. 12.—Compare with Fig. 11. Location of the infarcted areas found at autopsy.

The electrocardiogram reproduced in Fig. 14 is that of a man, aged 78 years, who was awakened at 2:00 A. M. on Sept. 29, 1944, by severe epigastric pain radiating to the left scapula. The pain was followed by coughing and the expectoration of frothy blood-tinged sputum. When seen at the hospital some hours later, he was cyanotic and the blood pressure was 110/76. On previous examinations the systolic pressure had been in the neighborhood of 150 to 160. The heart was borderline in size; the sounds were extremely faint; no murmurs were heard. Coarse moist râles were audible over the entire lung field. Death occurred about forty-eight hours after the onset of symptoms.

The limb leads are diagnostic of right bundle branch block, but also show large Q waves in Leads II and III and upward RS-T displacement in the last of these leads, which are characteristic of posterior infarction. The precordial leads, however, in addition to the late R waves in Leads V_1, V_2, and V_E, which are attributable to right branch block, show pronounced upward RS-T displacement in these same leads and in Leads V_3 and V_4 as well. These findings suggest antero-

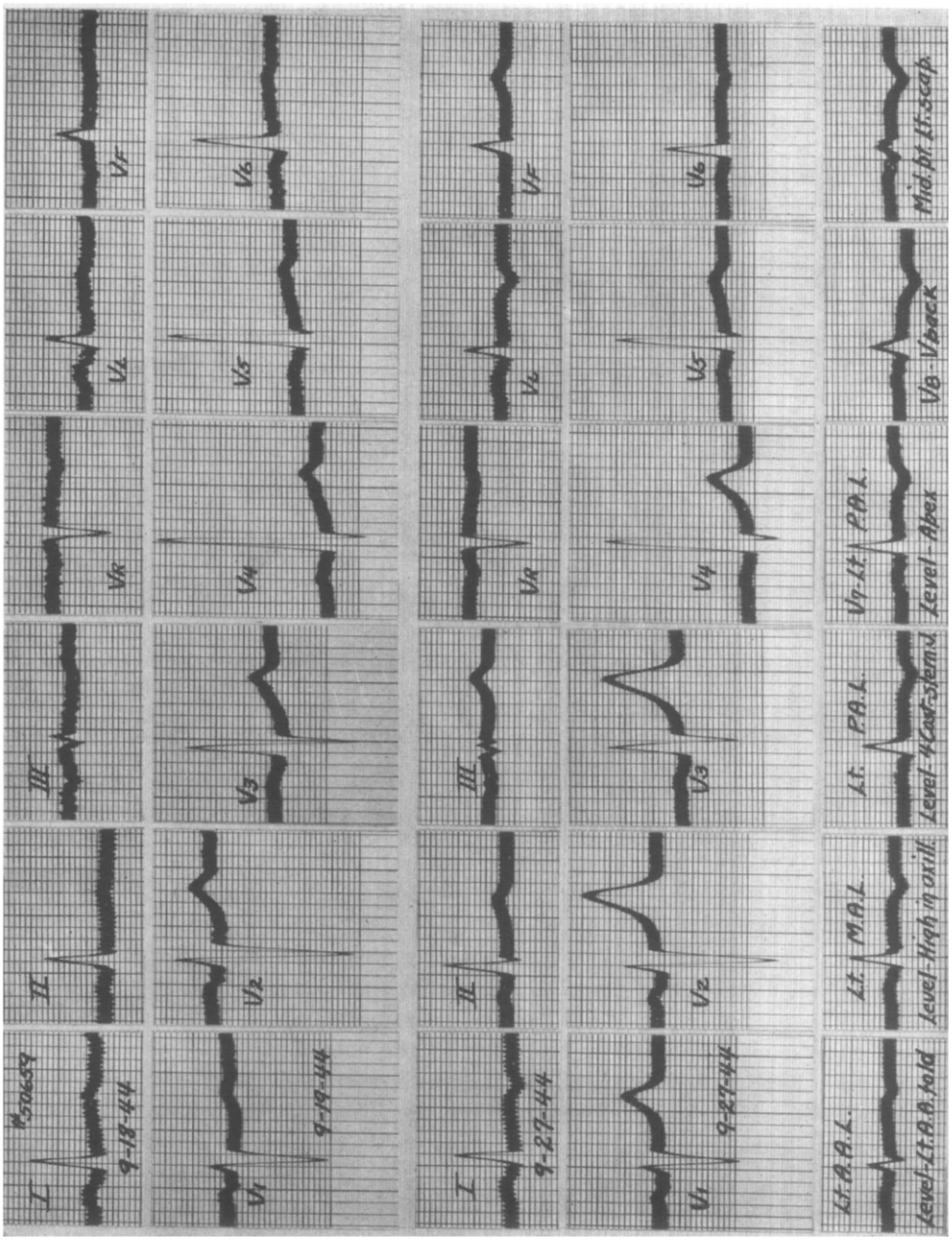

Fig. 13.—High posterolateral infarction. Electrocardiographic evidence of infarction delayed.

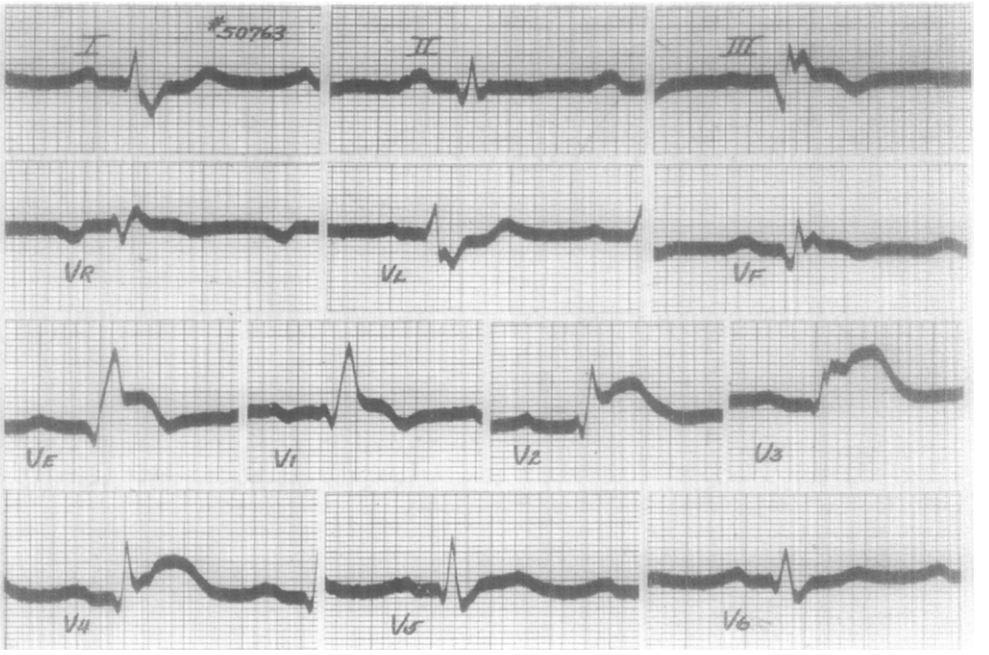

Fig. 14.—Right bundle branch block associated with signs of posterior infarction in the limb leads and signs of anteroseptal infarction in the precordial leads.

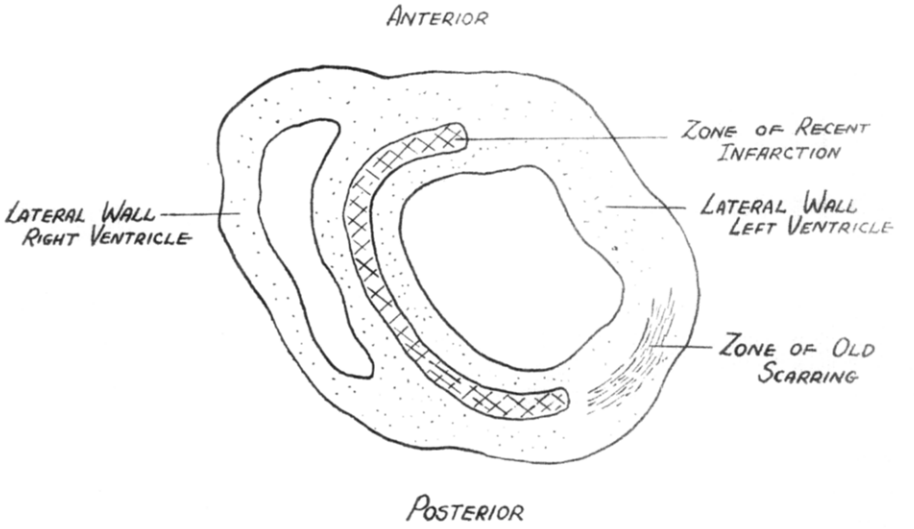

Fig. 15.—Compare with Fig. 14. Location of the infarcted areas found at autopsy.

septal infarction. The location of the infarcted regions disclosed by the post-mortem examination is shown in Fig. 15. Both coronary arteries showed pronounced atherosclerotic changes and the lumen of the anterior descending branch of the left was nearly obliterated. No thrombi were found in these vessels.

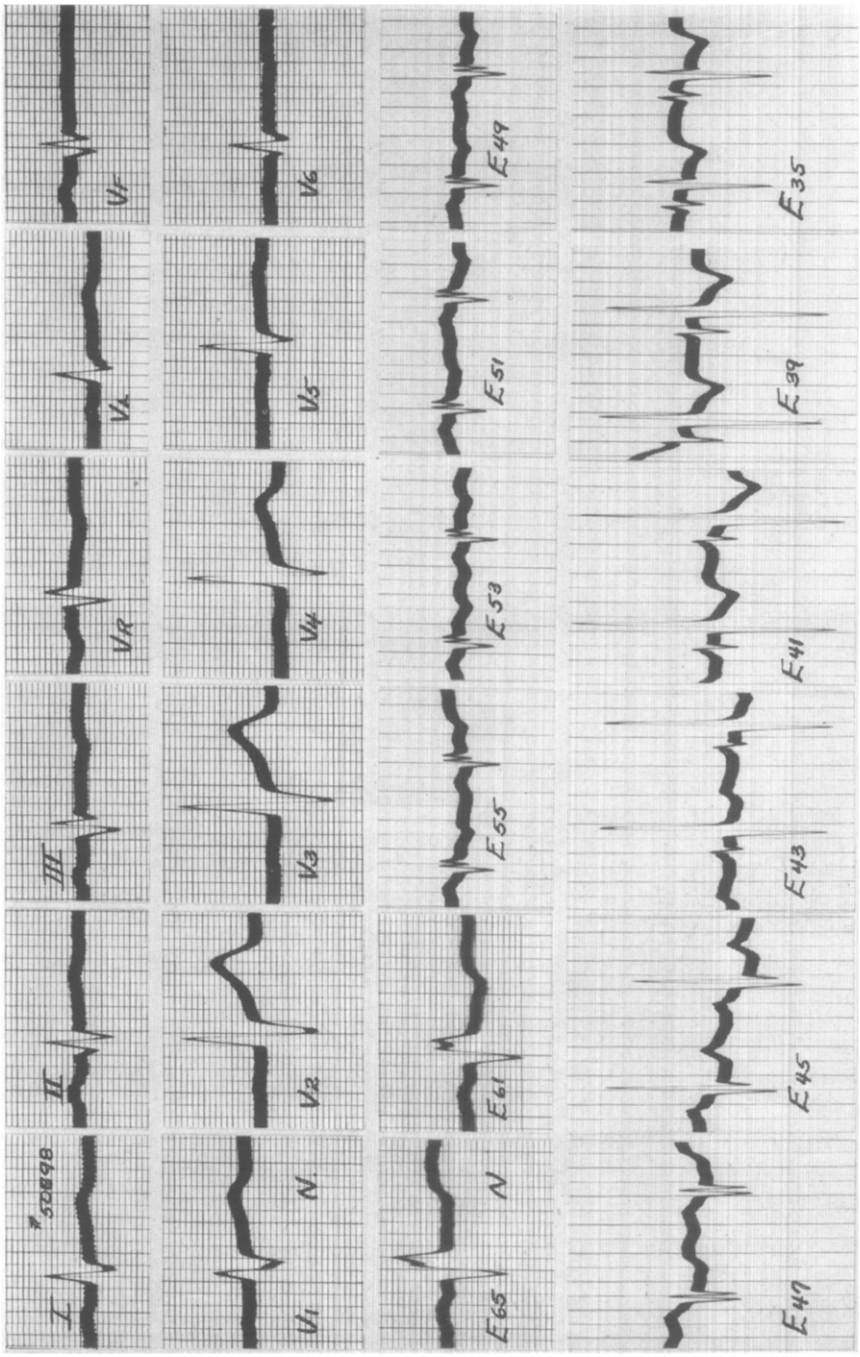

Fig. 16. Changes suggesting old posterior infarction in a case in which the clinical history made it seem very unlikely that infarction had occurred. The symbol *E* is used to designate an esophageal lead. The numeral attached to this letter gives the distance (in centimeters) of the exploring electrode from the nares.

The electrocardiograms reproduced in Fig. 16 are those of a man, aged 39 years, who had two spontaneous attacks of anginal pain in June, 1944. The first pain was felt in the region of the lower sternum and persisted throughout the day; it was not particularly severe. The second attack occurred about thirty-six hours later; the pain was under the midsternum and lasted for about thirty minutes. Subsequently, there was mild anginal pain on brisk exertion. Physical examination on Sept. 21, 1944, was negative except for a moderately loud late systolic murmur at the apex. The blood pressure was 128/75. There was nothing in the past history which threw any light on the development of angina pectoris.

The electrocardiogram shows large Q waves in Leads II, III, and V_F and in all of the leads from the ventricular levels of the esophagus. There are also rather prominent Q waves in Lead V_6. No changes in the T deflections suggesting myocardial infarction are present, but when such changes are present initially they may disappear in the course of three or four months. We consider the electrocardiograms in this case characteristic of old posterolateral infarction, but a diagnosis of infarction could not be made because standard limb leads taken in 1936 during a physiologic experiment showed exactly the same peculiarities as those taken at the time of our examination. We do not know what the correct explanation of these observations may be. We feel, however, that it is imperative to avoid making a clinical diagnosis on the basis of electrocardiographic examination when, after adequate investigation, it is certain that this diagnosis is not supported by the history and other clinical data.

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