ON THE DIRECTION AND MANIFEST SIZE OF THE VARIATIONS OF
POTENTIAL IN THE HUMAN HEART AND ON THE INFLUENCE
OF THE POSITION OF THE HEART ON THE FORM
OF THE ELECTROCARDIOGRAM

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GENERAL CONSIDERATIONS

(1) THE position of the heart influences the form of the ECG. Nevertheless, it is
our main purpose in electrocardiographic investigation to study better the
action of the heart, and one understands easily that if, by a change of position of
this organ, an alteration in the form of the curve has been produced, difficulty
will be encountered in deciding about the activity of the heart by means of
this form.

(2) This difficulty can best be resolved if one previously has learned to recognize
exactly the influence of position.

(3) The simplest and at the same time the most striking example of the influence
of change of position on the form of the ECG is furnished by a case of situs inver-
sus viscerum. Waller has already studied such cases, and several others have
since been published. We are reproducing (Fig. 1) a curve taken with Lead I
from a 7-year-old boy with situs inversus viscerum.

(4) One sees immediately that the peaks of Fig. 1, compared with those of a
normal ECG, are reversed. If one changes the right- and the left-hand leads of
the boy, there develops a curve which cannot be distinguished from a normal
ECG and which is reproduced in Fig. 2. Figs. 1 and 2 are complete mirror images
of each other.

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While the ECG in situs inversus viscerum can be explained in a very simple manner and requires no further discussion, in other cases of change in position of the heart phenomena appear which are more complicated and which must be analyzed more completely to be understood. We shall first consider those changes in position of the heart which are associated with respiratory movements.

Most respiratory movements in woman as well as in man are to a lesser or greater extent diaphragmatic, and, as the diaphragm rhythmically assumes a high and a low position, the heart must also rhythmically be displaced within the thorax.

Even before the construction of the string galvanometer, Samojloff had already demonstrated in his lectures, with the aid of the capillary electrometer, that exaggerated respiration has a definite influence on the amount of the displacement of mercury. In Lead I the excursions become greater during the phase of expiration and smaller in the phase of inspiration.

Among the numerous other investigators who have studied the influence of respiratory movements on the form of the ECG we can mention especially Vaandrager, Kahn, and Grau.

In many individuals the influence of the usual normal respiration is hardly noticeable, and at times one sees that even definitely deepened respiratory movements remain without remarkable consequences. One sees an example of this in a patient who has been described before and who showed the Cheyne-Stokes phenomenon in a high degree. Periods of superficial, hardly noticeable respiratory movements alternated regularly with periods in which the patient was strongly dyspneic and made deep, vigorous, frequent respiratory movements.
ECG's taken from this patient during periods of apnea and dyspnea were hardly distinguishable from each other.

(10) In contrast to these results, there occur in other people even during normal respiration regular changes in the ECG which have the same rhythm as respiratory movements and therefore must be most intimately associated with them. If respiratory movements are deep enough, striking changes appear in all individuals.

(11) To study this more closely we carried out a systematic investigation on ten healthy men whose ages varied from 19 to 65 years. As an index of the depth of respiration, the vital capacity of each individual was measured previously, and he was instructed to make several deep inspirations and expirations in a Hutchinson spirometer during the photographic registration of the ECG. The amount of air to be displaced was to be about two-thirds of the vital capacity. This amount of respiratory movement was chosen because on the one hand it was large enough for our purposes, and on the other hand it presented no difficulties to the experimental subject.

Fig. 3. — Bl. Lead I. During inspiration. Abscissae, 1 division = 0.04 sec.; ordinates, 1 division = $10^{-4}$ volts. The upper curve is the pneumogram.

Fig. 4. — Another portion of the same photographic record as the previous figure, during expiration.

(12) In Figs. 3 and 4 one can see two portions of a record taken with Lead 1 from Bl., one of the ten subjects. Movements of the spirometer into which he breathed were registered on the photographic plate by means of a special arrangement of pulleys in such a manner that an upward movement of the writing arm corresponded to an inspiration and a downward movement corresponded to an expiration. The writing arm moved exactly perpendicularly in front of the slit of the registering device, and one scale division corresponded to 250 c.c. of inspired or expired air.
(13) The pneumogram, that is, the upper line in the two figures, shows waves whose amplitude varies between 11.3 and 11.5 scale divisions; the movement of air between maximal inspiration and maximal expiration thus amounts to 2.8 to 2.9 L., while a figure of 4.4 L. was obtained in a preliminary measurement of vital capacity in the subject.

(14) The pneumogram reproduces the changes in volume of the thorax without a significant delay, as can be proved by the tremors which the subject shows at the height of inspiration. One sees the tremors clearly appearing in the ECG. This is caused by the vigorous muscular exertion which takes place in extreme inspiration. As soon as inspiration is ended, the muscles relax and passive expiration begins; the tremors in the ECG stop. We noticed that this arrest of tremor corresponds almost exactly to the peak of the curve of the pneumogram in Fig. 3, whereby for our purposes we can easily neglect small differences of, for instance, five scale divisions (0.2 second).

(15) Apart from the form and the height of the peaks of the ECG, it is striking that the rate of the heart is subject to great differences during respiration. In Figs. 3 and 4 this is clearly in evidence, and at the same time one sees that the maxima and the minima of heart frequency do not coincide with the peaks of the pneumogram. Thus, for instance, the longest heart pause is found at the end of Fig. 3 and the shortest pause at the beginning of the same figure, although in both places the filling of the lungs with air is about the same amount. Thus also in Fig. 4 the filling of the lungs is the same at the beginning and at the end of the figure, while the frequency of the heart in these positions shows significant differences.

(16) The explanation of the phenomenon is known: during slow, deep respiration the gas content of the blood varies in sufficient amount to influence the tonus of the vagus nerves, in particular at the point where they take origin in the medulla oblongata, and since the maximum and the minimum of the vagal tonus do not correspond with the extremes of inspiration and expiration, the times of the least and the greatest heart frequencies must, in general, be displaced in relation to these positions.

(17) The displacement is not equally great in all men. In some of our experimental subjects the maximum of heart rate is present at the height of inspiration, in others at the depth of expiration, while in still others it shows up at various intermediate phases of respiration.

(18) We have mentioned this circumstance here particularly because it must be taken into account if the influence of respiratory movements on the form of the ECG is to be discussed. For we know that by means of the vagus nerves the form and the height of the T wave and especially of the auricular P wave can be altered significantly.

(19) In Figs. 5 and 6 we reproduce two portions of a tracing which was taken with Lead III from the same person. Here the influence of vagus stimulation on the P wave is clearly portrayed. After long heart pauses P is small, sometimes diphasic, sometimes entirely negative, while after the short intervals, that is, with greater frequencies and diminished vagal tone, P regains its normal shape and size.
(20) The maximal changes in P are not synchronous with the changes in the form of the ventricular EG. In Fig. 5, for instance, the first and the third heart beats show ventricular waves which can hardly be told apart, while P is positive in the first heart beat and negative in the third.

(21) Further, it is apparent from Figs. 5 and 6 that the maximal changes in P are not synchronous with the extremes of inspiration and expiration. This is perhaps demonstrated best in another curve (see Fig. 7). The center of the figure corresponds roughly with the position of extreme inspiration, as is apparent from the respiratory tracing. To the right and left of the center the inflation of the lungs with air is roughly the same, while the heart rates are very different. On the left side where the cardiac interval is short, one sees that a high P wave is present; on the right side of the figure where the heart pause is long, a low P wave occurs.

(22) It follows from the above considerations that one runs into difficulty if one wishes to discover by means of respiratory movements the influence to which the form of the ECG as a whole is subjected by a change of position of the heart, for the P wave is exposed to many influences during respiratory movements.

(23) In the discussion which follows, we shall attempt to separate these influences from each other and make it possible to discuss each separately, but for the present it is better to turn our attention to some other waves of the tracing.

(24) It is evident from Figs. 3 to 6 that in Lead I the peaks of the ventricular EG become greater with expiration and smaller with inspiration, while in Lead III, on the contrary, they become smaller on expiration and larger on inspiration.
If we consider this fact in relation to the formula, Lead III = Lead II − Lead I, we must conclude that in Lead II the alterations in the waves of the ECG are insignificant. This is the case in Bl. as well as in most of the individuals studied, which leads to the practical conclusion that one should employ this lead in a patient when one wishes to study the form of his ECG as free as possible from the influence of respiratory movements.

(25) It must, nevertheless, be emphasized that even in Lead II noticeable variations in the size of the waves at times come to light, opposite in direction to the changes in Lead I. A typical example of such behavior is shown by Wi., whose ECG in Lead II is reproduced in Figs. 8 and 9. One sees how the respiratory tracing, whose waves show an amplitude of approximately 10.5 scale divisions, runs always very near the peaks of the R waves. A line drawn through the actual peaks of the R waves would run almost parallel to the pneumogram. The figures show that the waves increase during inspiration and diminish during expiration. The quantity of air displaced with each respiration amounts to 2.6 L., while the vital capacity of the experimental subject concerned amounts to 4.1 L.

![Fig. 7.—Br. Lead II. The respiratory tracing reaches its highest point at Inspir. Carot., carotid pulse.](image)

(26) In all of the foregoing figures from 3 to 9, the R and T waves are approximately equally diminished or increased during the various phases of respiration, so that the height relationships of the waves in a given person and in a given lead remain unchanged, and the tracing thus retains in general its characteristic appearance.

(27) For instance, when we measure out the waves of Figs. 3 and 4, we find in the position of maximum inspiration $R = 6$, $T = 2.8$ and $\frac{R}{T} = 2.14$; in extreme expiration $R = 11$, $T = 0.5$ millivolt, and $\frac{R}{T} = 2.20$. The relation of $\frac{R}{T}$ is thus nearly the same in both circumstances.

(28) But similar changes were not observed in all individuals. In some, one finds exceptions which on superficial observation appear to be completely irregular and thereby become somewhat puzzling.
We will first describe some of these exceptions separately and then attempt to unite them in a single point of view.

First, we point out that in many cases the change undergone by R can be significantly greater than the alteration in T. As examples we reproduce Figs. 10 and 11. Fig. 10, A and B shows the ECG of Bak. taken from Lead I. Two portions were cut out of the original tracing, and A represents a recording in inspiration and B in expiration. One sees that in expiration the R wave is significantly more increased than T, whereby the tracing takes on a completely different shape.

This inequality becomes even more marked in Fig. 11, A and B, taken from Wi. Both parts are again reproductions of portions of one and the same tracing, which was taken in Lead I; A corresponds to inspiration and B to expiration of the subject. In the first position R is smaller than T; in the second the relationship is reversed.

Another exception consists in this, that in the same phase of respiration a wave that grows in Lead I also becomes larger in Lead III. This phenomenon appears when the waves in the two above-mentioned leads point in opposite directions. As examples we point to Fig. 12, A and B, which reproduces the ECG in Lead III of the same person, Bak., from whom also the tracing of Fig. 10 is taken. The T_{III} wave is here negative, and we see that during expiration T_{III} (Fig. 12), as well as T_{I} (Fig. 10), become larger.

Entirely similar considerations are applicable to the QRS group as to T.

We will, nevertheless, attempt to answer the question of how the apparently irregular influence of respiratory movements on the form of the ventricular ECG can be explained.
We notice, first, that without exception in the persons studied the maxima and minima of the waves coincide exactly with the extreme respiratory positions, and that therefore one ought to take this phenomenon into consideration in every attempt at an explanation. Second, we exclude the possibility that such potential differences as appear in our curves are generated by some epiphenomenon, as, for example, muscle action related to the respiratory movement or movements of the electrodes. For in case some such accidental cause exerted an influence synchronous with respiration, the tracing reproducing a series of successive ECG's would move rhythmically up and down as a whole. We see, however, that the line that could be drawn through the diastoles of the heart shows no such rhythm but, in fact, as can be easily established with the help of the square millimeter grid, runs completely straight in many photographs.

As a second possibility to be considered, we mention the influence of altering body resistance to which Samojloff has called attention. We assume that during respiration the resistance of the body between the leads remains constant. If very small variations did occur here, they could be neglected without hesitation in view of the great resistance of the galvanometer. But during respiration the electric resistance of the organs surrounding the heart must undergo rhythmic variations on account of the altered air and blood content of the thorax, and these must necessarily exert an influence on the amplitude of the waves of the ECG. Apparently this influence is by itself very small, as is indicated, in our opinion, by the fact that the tracing in Lead II undergoes in general only small changes while the changes seen in Lead III are usually opposite those obtained in Lead I.

Were change in resistance the cause of the alterations in amplitude of the waves, one would have to assume that in a respiratory movement the resistance
of the thorax diminished in the horizontal direction while at the same time it increased in the vertical direction and remained unaltered in the oblique direction. Such an assumption seems to us very unlikely, especially because in not too vigorous respiratory movements the expansion of the thorax in the various directions is pretty much the same.

(38) Also, the fact that the relation of the size of the waves often undergoes a significant change is not explainable on the basis of a change in body resistance. Samojloff has already pointed this out himself, and he rightly concludes that in any event change in resistance cannot be the sole cause of the phenomenon.

(39) Although on physical grounds we must assume a priori that an influence of changing body resistance does exist, the above analyses justify the conclusion that it is small and for practical purposes may be disregarded in most cases.

(40) As a third cause, we can point out that the manner and way in which the heart contracts can be influenced by the altered pressure relations within the heart as well as in the thorax. The negative intrapleural pressure, which is increased during inspiration and diminished during expiration, exerts a definite direct mechanical influence upon the circulation. On account of its weaker musculature the right heart will be more sensitive to this influence than the left, and if the activity of one-half of the heart is more or less changed than that of the other, then the QRS group in the ECG would in particular be modified. It is, in fact, this group that undergoes the clearest changes during respiration.

Fig. 12.—Bak. Lead III. Fig. 12, A and B are portions of one and the same continuous tracing. Fig. 12, A during inspiration; Fig. 12, B during expiration.

(41) For an exhaustive study of the influence which extreme changes of intrathoracic pressure can exert on the ECG, we direct attention to the investigations of Kahn, who made ECG's of men while they carried out the Valsalva experiment. The changes thereby appearing in the ECG were described by Kahn as very significant.

(42) Nevertheless, in ordinary and even in quite deep respiration, pressure relationships do not constitute the main reason for the changes in form and amplitude of the ECG, for they cannot explain the above-described, and apparently irregular, variations.

(43) We discuss finally the influence of changes in position which the heart undergoes in the thorax during respiration. This influence has already been brought to the foreground by Grau, who carried out his investigations especially on “men with movable hearts,” in whom he controlled the position of the heart roentgenologically.
Even earlier we had considered it not at all surprising that the rhythmic changes in position of the heart caused by respiration should also modify rhythmically the form of the ECG. Apparently the change in position caused by normal respiration is alone insufficient in most people to evoke noticeable changes in the form of the ECG. This fact, which is reinforced by the circumstance that occasionally quite deep respiration remains without notable effect, is by itself something remarkable. For the respiratory motions are but seldom purely thoracic, and the anatomic relations make it necessary for the heart to experience a change in position in abdominal respiration. Transilluminating the human chest with roentgen rays, one sees these changes in position come clearly to view.

Still more questions emerge when one wishes to explain the changes in form of the ECG by alterations in position of the heart. How is it possible that the several waves often become greater or smaller quite unequally? Why does the ECG show so much more inconsiderable modifications in Lead II than in Leads I and III?

All these difficulties are resolved at a single stroke if one does not restrict oneself to the form of the registered tracings, but attempts from the ECG as recorded by the various leads to derive the direction and manifest size which the resulting potential differences in the heart itself possess.

RELATIONSHIP OF THE TRACINGS TAKEN BY THE THREE CONVENTIONAL LEADS

Before we go on to describe the manner and means whereby one can ascertain the resultant potential difference within the heart, it is desirable to review in a few words the mutual relation which exists among the three leads now generally employed.

The formula, Lead III = Lead II – Lead I, has validity only if the current flowing through the galvanometer exerts no influence on the potentials present at the extremities. Now this factor is so small that it can be neglected without causing any noticeable error. For the total resistance of the circuit in which are found the galvanometer and the human body is very large in comparison with the resistance of the heart musculature. We can thus assume a priori that the formula mentioned is correct.

On superficial inspection of many curves some doubt of the practical applicability of the formula seems to arise. This concerns particularly those ECG's which show great variations in form in the three leads. One can compare, for instance, the tracings of Fl. published previously (Figs. 13, 14, and 15). These and similar curves give a false impression with respect to their mutual relationship, which is caused by the circumstance that often the waves that we are accustomed to designate with the same letter do not fall in the same phase of a cardiac cycle. One can demonstrate this in various ways, among others by comparing the distances between the peaks P, R, and T in the tracing from Lead I with the corresponding distances in the tracings from Lead II and Lead III. It is apparent then that the summits mentioned are in general closer to each other in Lead I.

That our formula is in complete agreement with fact can be shown without too great difficulty. One would gain one's objective most simply if one recorded
two or three tracings from the several leads simultaneously on the same photographic plate. A simultaneous double registration has already been carried out by other investigators, who, as far as I know, employed two small-model string galvanometers and did not attempt an exact measurement of the tracings. An oscillograph is not suitable for the purpose mentioned.

Another method has been employed by Fahr. He recorded, as did Kahn, the ECG and the heart sounds simultaneously. In one and the same person the ECG was recorded from the three leads under identical conditions, while the tracing of heart sounds was combined with each ECG. In this way a fixed point of time could be marked, which fell always in the same phase of a cardiac cycle. With the aid of this fixed point of time the phases of the tracings in the various leads could then be mutually identified with exactness.

The investigation of Fahr shows that the ventricular EG. as a rule begins earlier in Leads II and III than in Lead I, and that the summit of R_I is formed somewhat earlier than the summits of R_{II} and R_{III}. Making use of this data it is no longer difficult to demonstrate the validity of the formula even in apparently complicated forms of the ECG.

As an example we reproduce here in Fig. 16, the QRS group of the ECG of Fl. (cf. Figs. 13, 14, and 15), schematically and in one and the same coordinate system. In its construction the abscissae are, for the sake of clarity, four times more extended than the ordinates, so that the whole figure seems expanded in breadth: one scale division on the abscissa corresponds to 0.01 sec.; one scale
division on the ordinate corresponds to $10^{-4}$ volts. The vertical lines represent various phases of a cardiac cycle, and at each phase the rule holds: Lead II – Lead I = Lead III.

One sees that the waves $Q_{II}$ and $Q_{III}$ begin 0.01 sec. before $R_I$ while the summits of the first-named waves correspond with a point of the anacrotic portion of the latter wave. The entire wave $S_I$ corresponds to the catacrotic part of $R_{III}$, while the whole wave $S_{II}$ corresponds with the anacrotic part of $S_I$.

Fig. 16.—Construction of the QRS group of $P_I$ in the three leads. Each ordinate corresponds to identical phases of a cardiac cycle. Abscissae, 1 division = 0.01 sec.; ordinates, 1 division = $10^{-4}$ volts.

Similar constructions which are capable of clarifying our understanding of the significance of the QRS group can always be carried out without great difficulty and also applied to the $P$ and $T$ waves. One must then, however, always bear in mind that these constructions lose their value if one changes the conditions under which the three leads are taken. Understandably one must give the subject the same body position during the recordings, while the scale divisions of the three systems of coordinates must correspond to the same time intervals and the same potential differences. Further, the galvanometer must react quickly enough to indicate the potential variations to be registered, immediately and without a significant error.
In conclusion, still another remark should be made regarding the calculation of such tracings. Inasmuch as absolute precision can naturally never be achieved and small errors, say of the order of a few per cent, can always occur, the accuracy with which one derives a small low wave by subtraction of two waves of nearly the same size will be very small.

If, for instance, $T_I$ and $T_{II}$ are nearly of equal size, the calculated value of $T_{III}$ from the formula, $T_{III} = T_{II} - T_I$, will be very inexact. $T_{III}$ as actually recorded then gives its size more exactly than the calculated, and in a comparison of the three tracings it is proper in these circumstances to start from one of the first two and the third lead in order to derive the tracing of the remaining lead from the data obtained hereby.

![Diagram of the equilateral triangle](image)

The above discussion makes it clear that one should not as a rule content oneself with two leads in the belief that the tracing of the remaining lead is sufficiently known from the data of the two former. For the dimensions of a curve constructed by calculation can be derived not only more simply but also more exactly by direct registration.

**The Schema of the Equilateral Triangle: Direction and Manifest Size of the Resulting Potential Difference in the Heart**

How, from the tracings taken in the three leads, is one to determine the actual direction of the potential difference in the body?

If one attempts to answer this question, one attains the goal in the simplest way by schematizing the human body. The following schema, that can be well-designated as the schema of the equilateral triangle, can be recommended as especially useful. Herein the human body is represented as a flat, homogeneous plate in the form of an equilateral triangle, $RLF$, (Fig. 17). Current is led off to the galvanometer from the corners. $R$ corresponds to the right arm, $L$ to the left arm, while $F$ represents the potential of both feet. A lead from $R$ and $L$ corresponds, therefore, to Lead I, from $R$ and $F$ to Lead II, and from $L$ and $F$ to Lead III.
A small spot, $H$, in the middle of the triangle, represents the heart. We assume that at a certain instant the potential differences in the heart are so distributed that their resultant has the direction of the arrow portrayed in the figure. A lead from the heart in this direction shows a maximal potential difference of such a nature that in the direction of the head of the arrow the heart is positive, in the opposite direction, negative.

We can also represent the matters thus: that between two closely adjacent points of the small spot $H$ a potential difference is developed. The arrow drawn in the figure coincides with the line which joins these two points and represents the direction of the maximal potential difference in the heart. The distance separating the points is very small compared with the length of one side of the triangle.

The angle that the arrow makes with the side $RL$ is called $\alpha$ and reckoned as positive when the arrow is turned in the clockwise direction and negative when turned in the opposite direction. In determining this the standpoint of an observer who faces the anterior chest wall of the subject is assumed.

We now assume further that an ECG has been recorded from a subject that has in each of the three leads a simple form, so that the summits $R_I$, $R_{II}$, and $R_{III}$ fall at identical phases of a cardiac cycle. According to the well-known formula, it can then be easily verified that $R_{II} - R_I = R_{III}$. If we transfer the values found in the experimental subject over to the schema, we can determine in him the direction of the potential difference which was present in the heart during the registration of the $R$ wave, and thus was the cause of the formation of this wave.

The ECG of Bak. in inspiration can serve as an example. One finds in the three leads the following values. These are expressed in tenths of a millivolt: $R_I = 3.2$, $R_{II} = 12.5$, $R_{III} = 9.3$.

In the homogeneous equilateral plate which contains the heart at its center, these values can only be produced by a resulting potential difference whose direction coincides with the arrow drawn in such a manner that $\alpha = 76$ degrees.

In expiration there is found in the same person $R_I = 9.2$, $R_{II} = 11.2$, $R_{III} = 2.0$, from which one can calculate that under these circumstances $\alpha_1 = 40$ degrees (cf. Fig. 18).

By means of the schema we are in this manner in a position to show that during expiration the heart has rotated itself in the body around a sagittal axis. The extent of the rotation in the schema is $\alpha_1 - \alpha = -36$ degrees.

In the construction of our schema we have assumed that the heart lies as a material point in a homogeneous mass and that the distances of the heart from the three leads and also the resistances concerned are equally great. This is, understandably, not the case. The electrical resistance to conduction in the lungs is different from that in the heart itself and in the chest wall. Only one point is assumed in the schema for both feet, although small but still measurable potential differences are produced between left and right foot.

Nevertheless, we have sufficient reason to assume that the actual rotation of the heart in the body for practical purposes corresponds sufficiently exactly with the rotation in the schema, and therefore in the following lines we shall
identify the direction which the potential difference assumes in the human body with the direction of the arrow in the schema and designate it as $\alpha$.

(73) The manner and way in which one calculates the angle $\alpha$ from the measured potential differences will be elucidated more closely in the appendix. We mention here only that the calculation is simple and can be carried out quickly.

(74) It is possible to attempt to verify the results thus found by means of an investigation with roentgen rays. Nevertheless, great demands on roentgen technique are made if one wishes to measure exactly the angle by which the axis of the heart turns during respiration. One would have to be able to make instantaneous exposures which correspond with a definite phase of the cardiac cycle, as, for example, they have been carried out by Koranyi and v. Elischer.\(^{15}\)

(75) Further, it must be noted that in different men the motility of the heart shows great differences and that therefore for achievement of our goal the roentgenograms and the ECG's must be taken in the same person and under the same conditions. A few experiments which we have carried out in this direction have not progressed far enough to be discussed here in greater detail.

![Diagram](image)

Fig. 18.—For explanation of the illustration, see Fig. 17. $\alpha_1 = 40$ degrees.

(76) In the roentgenogram the direction of the heart's axis can be determined with any certainty only with difficulty, and in so far as we are now aware, nowhere in the whole of the roentgen literature is a detailed, decisive treatment of the question of the rotation of the axis of the heart encountered.

(77) Nevertheless, data have been published which are not without value for our purpose. We mention especially here the book of Groedel,\(^{16}\) in which a cinematogram of the heart in forced respiration is reproduced. Groedel comes to the conclusion that in respiration the left heart border is displaced more strongly than the right, namely in the proportion of 6.5 to 4.8. The heart thus rotates itself, so to speak, around a point which lies at the place where the right diaphragm and the right auricle meet.

(78) In general, these results agree well with the results of electrocardiography. They cannot, however, be a satisfactory control on this latter because the rotation of the cardiac axis can be measured with greater accuracy electrocardiographically than roentgenographically.
The schema of the equilateral triangle not only gives the direction of the potential differences, but it places us also in the position to make a comparison between the magnitudes of these differences as they are found in the heart itself. For clarity we can introduce a designation "the manifest potential difference in the heart" and, when particular waves, for instance, P, R, or T, are considered, indicate them by the figures \( P_m, R_m, \) and \( T_m \). By these figures, then, the manifest value of each summit is meant.

We define the manifest potential difference in the heart as the magnitude given in one of the three leads when the direction of the current between the leads corresponds to the direction of the resulting potential difference in the heart. Only the potential differences in the frontal plane come into consideration therein.

If we assume that a given potential difference in the heart produces the summits \( T_I, T_{II}, \) and \( T_{III} \), and that the direction of current in the schema is represented by an arrow parallel to the side \( RL \) (Fig. 19), then \( T_I \) is the greatest of the three summits while \( T_{II} \) and \( T_{III} \) will be opposed to each other and attain half the size of \( T_I \). In these circumstances \( T_m = T_I \).

The manifest size of a wave can always be calculated from the height of the recorded wave and can thus always be expressed in absolute terms, for instance, in tenths of a millivolt.

The calculation, just as that of the angle \( \alpha \), can be carried out easily and rapidly, as will be more completely described in the appendix. Applying the calculation to the R wave of Bak., the data of which have been given previously, (see Figs. 10 and 12), we find that \( R_m = 13 \) in inspiration and in expiration \( R_m = 12 \) tenths of a millivolt.

In Table I the similar values of four subjects are collected.

One sees from the figures recorded in the table that the rotation of the resulting potential difference R, present in the heart, takes place in the same direction in all subjects during respiration, but, however, as was to have been expected, the extent of this rotation is not the same in the various people. The rotations vary from \(-12^\circ\) to \(-36^\circ\).
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TABLE I.

<table>
<thead>
<tr>
<th>SUBJECT</th>
<th>$R_1$</th>
<th>$R_2$</th>
<th>$R_3$</th>
<th>DIRECTION OF R AND ROTATION IN EXPIRATION</th>
<th>MANIFEST SIZE OF R IN TENTHS OF A MILLIVOLT</th>
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<tbody>
<tr>
<td>Bak., inspiration</td>
<td>3.2</td>
<td>12.5</td>
<td>9.3</td>
<td>$76^\circ \setminus -36^\circ$</td>
<td>13</td>
</tr>
<tr>
<td>Bak., expiration</td>
<td>9.2</td>
<td>11.2</td>
<td>2.0</td>
<td>$40^\circ \setminus$</td>
<td>12</td>
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<td>6.2</td>
<td>20.0</td>
<td>14.0</td>
<td>$73^\circ \setminus -20^\circ$</td>
<td>20</td>
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<td>12.2</td>
<td>20.2</td>
<td>8.0</td>
<td>$53^\circ \setminus$</td>
<td>20</td>
</tr>
<tr>
<td>de Bl., inspiration</td>
<td>6.0</td>
<td>26.0</td>
<td>20.0</td>
<td>$77^\circ \setminus -12^\circ$</td>
<td>27</td>
</tr>
<tr>
<td>de Bl., expiration</td>
<td>11.0</td>
<td>26.0</td>
<td>13.0</td>
<td>$65^\circ \setminus$</td>
<td>26</td>
</tr>
<tr>
<td>Br., inspiration</td>
<td>6.5</td>
<td>21.0</td>
<td>14.5</td>
<td>$73^\circ \setminus -23^\circ$</td>
<td>21</td>
</tr>
<tr>
<td>Br., expiration</td>
<td>13.0</td>
<td>20.0</td>
<td>7.0</td>
<td>$50^\circ \setminus$</td>
<td>20</td>
</tr>
</tbody>
</table>

(86) The manifest size of R which we have called $R_m$ remains unchanged during respiration in one and the same person, within the limits of error of the method, although it shows great differences in various people. In Bak. it has a value of approximately 12.5, and in Bl. of about $26.5 \times 10^{-4}$ volts.

(87) The constancy of the manifest size of R in the same person demonstrates, first, that the influence of altering body resistance on the size of the peaks of the ECG is but slight, and, second, it can be considered as a proof of the validity of the schema employed.

(88) We must now determine the direction and the manifest size of T in the same way as for R, but must remember that the T wave is not suitable to bring to light clearly the change of position of the heart caused by respiration. The changes which T shows during respiration have two causes, of which the first is associated with the change in position, the second with the change in frequency of the heart. It is not difficult, however, to determine from the data of the tracings the part which each of the two causes has in the modification of T.

(89) Attention should be first directed to the manifest size of T. It is apparent from Table II that $T_m$ regularly increases during expiration, and, in our subjects, from a minimum proportion of 1.2 to a maximum proportion of 1.9.

(90) In accord with the schema of the equilateral triangle, increase or diminution of the manifest size of a wave can never be produced by a rotation of the axis of the heart. Neither will a displacement of the heart noticeably influence the manifest size of a wave when it is confined to normal limits. A change of this magnitude must therefore be ascribed exclusively to other influences, and as one such we have come to recognize the action of vagal tonus with which the heart rate is also associated.

(91) The rotation of a wave, however, is a different matter than the manifest size. We know from the data of Table I how much the axis of the heart will be rotated in the four subjects mentioned during the respiratory movements carried out by them and measured by the spirometer. The data of Table II concern the same respiratory movements as those of Table I and are taken from the same
ECG. If one therefore finds a different angle for the rotation of T than for the rotation of the axis of the heart, the difference between the two rotations must be caused by the action of additional influences. In our experiments only the changeable vagal tone can be held responsible for the latter.\(^{17}\)

As a matter of fact, we find values for the rotation of T which are mostly significantly smaller than for the rotation of R. Thus, for instance, in Bak. a value for the rotation of R, that is, for the rotation of the heart axis, of \(\beta_R = -36\) degrees is found, while in the same ECG a value for the rotation of T of \(\beta_T = -24\) degrees is found. The difference \(\gamma = \beta_T - \beta_R = 12\) degrees is then the rotation caused by the altered vagal tone.

It is apparent that \(\gamma\) is, as a rule, positive, whether or not the manifest size of T increases. One can receive confirmation of this finding by taking the ECG of a number of people in whom the vagal tonus is changed in other ways than by forced breathing. For instance, one can study the subject at rest and immediately after strenuous physical exertion when the heart frequency is increased.

The results of such an investigation, which will be reported in the material which follows, confirm the above-mentioned conclusions.

P is as little suitable as T to indicate the changes in position of the heart caused by respiration, for P is even more greatly influenced by a change in heart rate than T. In addition, P is often small and less simple. In so far as it was possible to determine the direction of this auricular current in our ten subjects, it indicated without exception that during respiration a rotation takes place in the same sense as with R and T.

The small and rather inconstant peaks Q and S occur mainly where the QRS group is complicated so that their measurement is rendered difficult. But the same thing can be said of them as of P. Where it was possible to carry out the measurement and to draw a not too uncertain conclusion from small modifications, the same result was always found, that the two waves rotate in respiration in the same direction as the other waves of the ECG.
The cause of all these rotations taking place in the same sense is clear. For the whole heart is turned around a sagittal axis by the change in position of the diaphragm during respiration.

It would not be without interest to ascertain whether the axis of the heart retains the same direction in diastole and systole. During diastole the heart has, to all intents and purposes, no tendency to assume a characteristic shape and is able to follow the movements of the diaphragm better than during systole. P falls in a phase where the ventricles still remain in diastole, while the QRS group is produced in the very beginning of ventricular contraction and apparently therefore falls in such a phase of this contraction where the musculature has not yet entirely attained its systolic hardness. Finally, T falls in a phase where the ventricles show their full hardness.

Were one able to determine exactly the direction of each wave and the influence which respiration exerts on its rotation, it would be possible to determine separately the rotation of the heart’s axis in diastole and in systole. But the difficulties of measurement of P and the complication because of altered cardiac frequency do not permit one to determine with certainty whether the rotation of P and the QRS group caused by change of position are as marked or more marked than those of T.

It is better to leave this point undecided and to remark further that during respiration no noteworthy rotation of the heart takes place around a vertical axis. This is proved by the behavior of the peak $S_I$. As will soon be discussed more fully, $S_I$ shows in some individuals very great modifications during rotation of the heart around the vertical axis, while this wave is only slightly changed during respiration in the same individual.

Finally, we return to the question brought up in the beginning, and apparently presenting many difficulties, but which now can be answered easily.

First, the question, how is it possible that in ordinary respiratory movements in which roentgenographic study often demonstrates clear changes in position of the heart, one usually observes no significant changes in the ECG?

The explanation must be sought therein, that during an ordinary, not excessive respiration in many cases there occurs mainly a displacement of the heart parallel to itself. Such a displacement exerts only a slight influence on the form of the ECG while a small rotation of the heart on a sagittal axis as it is produced during a deeper respiration already suffices to modify noticeably the form and height of the various waves of the tracing. This arises directly out of the schema of the equilateral triangle.

That the various waves of the ECG are often unequally increased or diminished by an altered position of the diaphragm, and that the tracing in Lead II shows so much less important modifications than in Lead I and III, is explained in the most easy and satisfactory manner by the triangle schema. A potential difference present in the heart, whose direction according to the schema would be designated in the one heart position by $\alpha = 80$ degrees and in the other by $\alpha_1 = 40$ degrees, produces a wave which in Lead II remains unchanged and in Leads I and III on the contrary would be modified significantly.
If the wave in Lead II in the first heart position had an amplitude of $9.4 \times 10^{-4}$ volts, it would retain this value in the same lead in the second heart position. In Lead I, on the contrary, it would be increased from $1.7 \times 10^{-4}$ volts in the first heart position to $7.7 \times 10^{-4}$ volts in the second position of the heart. In Lead III it would be diminished from $7.7 \times 10^{-3}$ volts in the first heart position to $1.7 \times 10^{-4}$ volts in the second heart position.

When the resultant potential difference $T$ has a different direction than $R$, the waves $T_I$, $T_{II}$, and $T_{III}$ must be modified in a different manner than $R_I$, $R_{II}$, and $R_{III}$ by the rotation of the heart around a sagittal axis. Therefore, in general, it is not permissible to conclude from the fact that $T$ behaves differently under certain conditions than $R$, that the development of electricity whereby $T$ is produced has another cause than the development of electricity whereby $R$ is evoked.

It is likewise clear why a wave which is directed similarly in Leads I and III undergoes opposite changes through a change in position of the diaphragm, while another wave is increased in both leads or diminished in both leads, as long as in the former it has a positive and in the latter a negative value. The latter takes place when the angle $Q$ lies between $-90$ degrees and $+30$ degrees.

There exist good grounds to assume that the most important peaks of the ventricular $E G$, $T$ and $R$, are produced by potential changes which are localized in approximately frontal planes of the heart. This can be demonstrated by the behavior of the S wave in Lead I. When a person lies first horizontally on his left side and later turns to his right side, the striking phenomenon appears that the $S_I$ wave, which in the first position was not present at all or was only very small, appears clearly and sometimes reaches a considerable height when the body is turned.

Figs. 20 and 21, which were both taken from Lead I, are examples of the development of a large S wave upon turning the body to the right side. Fig. 20, which was recorded during the left-sided position, shows a small S wave which can almost be overlooked, while Fig. 21, which was taken from the right-sided position, shows so large an S wave that one is inclined to wonder somewhat that both tracings were taken from the same person with the same lead, and, with exception of body position, under completely similar circumstances.

The most satisfactory solution of this phenomenon, so puzzling on superficial consideration, will be found if one assumes that on turning the body from the left to the right side the heart is rotated in the thorax around the longitudinal axis of the body.

When there is present in the heart a potential difference having a sagittal direction, a small rotation of the heart around the long axis of the body can cause a definite change in the amplitude of the waves involved, and this will occur especially markedly in Lead I. In case the potential difference is directed exactly sagittally, the wave disappears completely. It is clearly apparent that the great differences in the S wave in various individuals as well as its general inconstancy must be explained by this circumstance.
In Figs. 20 and 21 we notice that the P, R, and T waves undergo almost no change. If we conclude from the enormous modification of the S wave that a rotation of the heart has taken place along the longitudinal axis of the body, we must also assume that the P, R, and T waves are produced in approximately the frontal plane, or better, in such planes as form relatively large angles with the sagittal plane. For in this manner it is easiest to explain their slight variability under these circumstances.

The immutability of the P, R, and T waves gives occasion for another remark. Anatomic relationships require that in turning the body to the left side, the heart is displaced in the thorax somewhat to the left; on turning the body to the right side, it is displaced somewhat to the right. That this change of position evokes no modification in the P, R, and T waves confirms the result which investigation of respiration yielded, namely, that a displacement of the heart parallel to itself exerts only a very minor influence on the form of the ECG.

In five of the ten persons studied, as well as in the above-mentioned case of Fl., the S_I wave became greater as soon as the body was turned from the left to the right side. In three subjects S_I was lacking in both positions of the body, and in the two others this wave retained the same size.

When we compare the ECG's which were taken from men lying upon the back and upon the abdomen successively, it appears that in turning over on the abdomen the heart rotates on a sagittal axis in the same sense as in expiration. The ten individuals studied showed this rotation without exception.
<table>
<thead>
<tr>
<th>SUBJECT</th>
<th>FREQUENCY OF HEART IN BEATS PER MINUTE</th>
<th>TI</th>
<th>TII</th>
<th>TIII</th>
<th>DIRECTION OF T AND ROTATION WITH INCREASED HEART RATE</th>
<th>MANIFEST SIZE OF T IN TENTHS OF A MILLIVOLT (T m)</th>
<th>RELATION BETWEEN MANIFEST SIZES $T_m$ AT INCREASED HEART RATE $T_m$ AT REST</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ca. at rest</td>
<td>72</td>
<td>5.0</td>
<td>4.5</td>
<td>-0.5</td>
<td>25°, 21°</td>
<td>5.5</td>
<td>1.3</td>
</tr>
<tr>
<td>Ca. after exercise</td>
<td>112</td>
<td>5.0</td>
<td>7.0</td>
<td>2.0</td>
<td>46°</td>
<td>7.2</td>
<td></td>
</tr>
<tr>
<td>v.d.Sch. at rest</td>
<td>76</td>
<td>4.0</td>
<td>5.0</td>
<td>1.0</td>
<td>41°</td>
<td>5.3</td>
<td>1.6</td>
</tr>
<tr>
<td>v.d.Sch. after exercise</td>
<td>97</td>
<td>6.0</td>
<td>8.0</td>
<td>2.0</td>
<td>44°, 3°</td>
<td>8.3</td>
<td></td>
</tr>
<tr>
<td>Fu. at rest</td>
<td>86</td>
<td>1.5</td>
<td>2.0</td>
<td>0.5</td>
<td>44°, 2°</td>
<td>2.1</td>
<td>1.7</td>
</tr>
<tr>
<td>Fu. after exercise</td>
<td>137</td>
<td>2.5</td>
<td>3.5</td>
<td>1.0</td>
<td>46°</td>
<td>3.6</td>
<td></td>
</tr>
<tr>
<td>Tj. at rest</td>
<td>91</td>
<td>3.0</td>
<td>3.3</td>
<td>0.3</td>
<td>35°, 16°</td>
<td>3.6</td>
<td>1.6</td>
</tr>
<tr>
<td>Tj. after exercise</td>
<td>131</td>
<td>3.5</td>
<td>5.5</td>
<td>2.0</td>
<td>51°</td>
<td>5.6</td>
<td></td>
</tr>
<tr>
<td>Jo. at rest</td>
<td>89</td>
<td>3.0</td>
<td>4.2</td>
<td>1.2</td>
<td>46°, 19°</td>
<td>4.3</td>
<td>1.6</td>
</tr>
<tr>
<td>Jo. after exercise</td>
<td>96</td>
<td>3.0</td>
<td>7.0</td>
<td>4.0</td>
<td>65°</td>
<td>7.0</td>
<td></td>
</tr>
</tbody>
</table>
The explanation imposes no difficulties, for in the prone position pressure in the abdominal cavity is increased and through the pressure of the abdominal viscera against the diaphragm this organ is pushed upward.

Apart from the rotation around the sagittal axis there can also be recognized in the same circumstances, by the behavior of S1, a minor rotation around the longitudinal axis of the body.

We mention now briefly the differences which the ECG shows when an individual is studied in the sitting position and while lying on the back. In the ten persons studied the differences were in general but slight; in some it was clear that the heart in the recumbent position had rotated on a sagittal axis in a similar manner as in inspiration and in the sitting position in a similar manner as in expiration. In others these rotations were, however, hardly noticeable.

Because this question has a practical importance for the clinic, it must be recalled here that in our investigation the trunk of the subject was really horizontal during recumbency and vertical while seated. As a rule, one is not able to fulfill these conditions in the clinic. For when a patient is studied seated, one permits him to lean back comfortably in the chair—at least we ourselves always proceed in this way, generally also with a patient lying in bed, the shoulders are at a somewhat higher level than the pelvis.

In this way the two body positions approach each other so that the difference between the ECG's becomes smaller, and, as a rule, one can without much hesitation compare directly the tracings registered while lying with those recorded while sitting.

THE INFLUENCE OF INCREASED HEART RATE

With the help of the schema of the equilateral triangle one is in a position better to form an opinion about the form of the ECG in all sorts of circumstances. For instance, let us consider more closely the influence of an increased heart rate caused by bodily exertion. In "Weiteres über das ECG" a number of this kind of tracings have already been described,19 but the description at that time had to be restricted to the separate ECG's taken from the three leads. Now we can combine the results, and, for instance, deduce from the measurements of PI, PII, and PIII, that the manifest size of the potential difference present in the heart, Pm, increases with increasing heart rate, therefore with diminished vagal tonus.

Worthy of notice is the change that the ventricular EG. undergoes under the influence of diminished vagal tonus. We point especially to the changes in the T wave and give in the following Table III the height reached by this wave in five persons.20

From the values in the sixth column it is evident that in all of the cases studied the direction of T is rotated a bit in the positive direction when the heart rate increases, while the seventh and eighth columns indicate that then the manifest size of T also increases.

The investigation is fairly troublesome. For the construction of the above table it was necessary to measure out six ECG's for each subject, or thirty ECG's in all. So long as one studies the person in the resting state his heart rate is as a
rule relatively constant during the recording of the three leads. But if one takes
the photograph immediately after bodily exertion while the heart rate is increased,
a difficulty arises because it is not always possible to catch the right moment
when the frequencies are the same. It should not remain unmentioned here that
the ECG's of v.d. Sch. and Fu. appear from the table to satisfy these require-
ments better than those of the three other subjects who have therefore not fur-
nished as reliable results.

The rotation of T, which, as is apparent from the table, is positive in all
persons studied, is not caused by a rotation of the heart's axis. In the first place,
there is no reason for the assumption that under the above-mentioned conditions
a rotation of the axis of the heart took place, and second, the unaltered direction
of this axis can be proved directly by measurement of the R wave.

The positive rotation as well as the increase in manifest size of T therefore
depends exclusively upon the increase in the heart rate and must be explained
by the diminution of the vagal tonus. Vagal tonus thus influences the path
which excitation and the wave of contraction take in the heart muscle.

PATHOLOGICAL CONDITIONS

In conclusion, several examples from pathology can be discussed.

In myodegeneratio cordis and in many cases of auricular fibrillation T is
small in all three leads. It need not be discussed in greater detail that under
these circumstances T must also be small.

In many cases of hypertrophy of the left heart one finds a negative QRS III.
What conclusion can one draw from this? Entirely in general, the modification
of the QRS group could perhaps be caused by a change in position of the heart,
especially by a rotation of the heart around the sagittal axis. In fact, the clinician
is often in doubt whether he should ascribe the phenomena seen in his patients
to an actual hypertrophy or simply to a change in the position of the heart.
(130) The schema of the equilateral triangle in such a case can often provide the solution.

(131) We found, for instance, in several cases of hypertrophy of the left ventricle that the most important peaks of the QRS group, or at times the QRS group as a whole, had a direction which could be represented by \( \alpha = -40 \) degrees, while for this wave or group in normal hearts the amplitude of \( \alpha \) varied between +40 degrees and +90 degrees.

(132) If one wished to explain the rotation of the QRS group in these cases by a rotation of the heart around its sagittal axis, one would have to assume that this rotation amounted to 80 degrees and even 130 degrees—which must be considered as impossible.

(133) The application of the schema permits therefore the conclusion that we are concerned in these cases with a significant change in the conduction of excitation and the progress of the wave of contraction through the heart muscle. It is entirely possible that one would be able to diagnose a beginning hypertrophy of the left heart through electrocardiographic investigation of such a change sooner than in any other existing manner.

(134) Hypertrophy of the right heart and also atypical contractions of the heart can be adjudged in the same manner. The latter are of great importance and worth a special exhaustive review. But the above-mentioned examples may suffice. They prove adequately that by means of the schema one is in a position to separate from each other the real changes in the heart’s action and the apparent ones, which are caused only by change in the position of the heart.

**APPENDIX**

*On the Manner and Way in Which One Calculates the Direction and Manifest Size of the Resulting Potential Difference*

(135) **A. The Direction of the Resulting Potential Difference.**—In the equilateral triangle \( RLF \) in Fig. 22 an arrow is drawn through the center \( H \) making an arbitrary angle \( \alpha \) with the side \( RL \). On this arrow a portion \( pq \) of arbitrary length is marked off. The projection of \( pq \) on the side \( RL \) is \( p_1q_1 \), that on the side \( RF \) is \( p_2q_2 \), and that on the side \( LF \) is \( p_3q_3 \).

(136) If we make \( pq = E \), \( p_1q_1 = e_1 \), \( p_2q_2 = e_2 \), and \( p_3q_3 = e_3 \), thus:

\[
\begin{align*}
  e_1 &= E \cos \alpha & \\
  e_2 &= E \cos (\alpha - 60 \text{ degrees}) & \\
  e_3 &= E \cos (120 \text{ degrees} - \alpha) & \\
  e_3 &= e_2 - e_1
\end{align*}
\]

(137) We assume that the equilateral triangle \( RLF \) represents a homogenous flat plate, that in two points lying very close to each other in the immediate neighborhood of \( H \) a potential difference is created, and that a line joining the two points together coincides with the arrow drawn in the figure. Under these circumstances the potential differences present between the corners of the triangle must be related to each other as \( e_1 : e_2 : e_3 \).
When \( \alpha \) is unknown, this angle can be calculated from the relationship of two of these three potential differences. Employing \( e_1 \) and \( e_2 \) in the calculation, one finds with the help of the formulas (1) and (2):

\[
\tan \alpha = \frac{2e_2 - e_1}{e_1 \sqrt{3}}
\]

If one employs \( e_1 \) and \( e_3 \), one finds:

\[
\tan \alpha = \frac{2e_3 + e_1}{e_1 \sqrt{3}}
\]

and employing \( e_2 \) and \( e_3 \), one finds:

\[
\tan \alpha = \frac{e_2 + e_3}{(e_2 - e_3) \sqrt{3}}
\]

The angle \( \alpha \) represents the direction of the resulting potential difference in the body. By means of any one of the formulas (5), (6), and (7), one is also in a position to calculate this direction from the potential differences as these are found in the three conventional leads.

Nevertheless, it is not always worth the recommendation to employ the above-mentioned formulas because one can often achieve his purpose much more quickly. If one is content with a lesser accuracy so that an error of 10 degrees or 15 degrees is still permissible, one can use a geometric construction whereby \( \alpha \) is derived directly from the figure. One sees easily that with angles of 0 degrees, 30 degrees, 60 degrees, 90 degrees, etc., the relationship of \( e_1: e_2: e_3 \) is very simple and, in fact,

\[
\begin{align*}
\text{at 0 degrees} & : e_1: e_2: e_3 = 1 : 0.5: -0.5 \\
\text{at 30 degrees} & : e_1: e_2: e_3 = 1 : 1 : 0 \\
\text{at 60 degrees} & : e_1: e_2: e_3 = 0.5 : 1 : 0.5 \\
\text{at 90 degrees} & : e_1: e_2: e_3 = 0 : 1 : 1 etc.
\end{align*}
\]

The angles of values lying in between must then be estimated.

If greater accuracy is desired, one can employ a table of figures to advantage. Such a table is Table IV. With its help one is in a position to read off the value of \( \alpha \) to within a minute in any case that may occur. The added table of interpolation, Table V, gives directly the desired value for every two degrees.

**Example 1:** For the R wave of Bak, in inspiration there were found in tenths of a millivolt: \( R_I = 3.2, R_{II} = 12.5, R_{III} = 9.3 \). We have thus for this wave: \( e_1: e_2: e_3 = 3.2 : 12.5 : 9.3 \). Since the table shows the relationship of potential differences for a maximum value of \( \pm 10 \), we must multiply the three above figures by \( \frac{10}{12.5} \) and obtain thus: \( e_1: e_2: e_3 = 2.6: 10: 7.4 \). It can be seen from the table that this proportion corresponds to an angle \( \alpha \) which lies between 70 degrees and 80 degrees. As shown in the table the value of \( e_1 \) diminishes from 3.5 to 1.8 between 70 degrees and 80 degrees. An interpolation table which lies between those figures, 3.5 and 1.8, must therefore be consulted. Such a one is Table V, II.
Table IV. Table of Recorded and Manifest Potential Differences for Various Values of $\alpha^*$

<table>
<thead>
<tr>
<th>Recorded Potential Differences ($e$)</th>
<th>Manifest Potential Differences ($E$)</th>
</tr>
</thead>
<tbody>
<tr>
<td>$e_1$</td>
<td>$e_2$</td>
</tr>
<tr>
<td>0°</td>
<td>10</td>
</tr>
<tr>
<td>10°</td>
<td>10</td>
</tr>
<tr>
<td>20°</td>
<td>10</td>
</tr>
<tr>
<td>30°</td>
<td>10</td>
</tr>
<tr>
<td>40°</td>
<td>8.2</td>
</tr>
<tr>
<td>50°</td>
<td>6.5</td>
</tr>
<tr>
<td>60°</td>
<td>5.0</td>
</tr>
<tr>
<td>70°</td>
<td>3.5</td>
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<tr>
<td>80°</td>
<td>1.8</td>
</tr>
<tr>
<td>90°</td>
<td>0</td>
</tr>
<tr>
<td>100°</td>
<td>-1.8</td>
</tr>
<tr>
<td>110°</td>
<td>-3.5</td>
</tr>
<tr>
<td>120°</td>
<td>-5.0</td>
</tr>
<tr>
<td>130°</td>
<td>-6.5</td>
</tr>
<tr>
<td>140°</td>
<td>-8.2</td>
</tr>
<tr>
<td>150°</td>
<td>-10</td>
</tr>
<tr>
<td>160°</td>
<td>-10</td>
</tr>
<tr>
<td>170°</td>
<td>-10</td>
</tr>
<tr>
<td>±180°</td>
<td>-10</td>
</tr>
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<td>-170°</td>
<td>-10</td>
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<td>-160°</td>
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<td>-40°</td>
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<td>-30°</td>
<td>10</td>
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<tr>
<td>-20°</td>
<td>10</td>
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*For each group of three leads the maximal value for the recorded potential differences is set at ±10.

(144) Here one finds for $e = 2.5$ (that is, for the value which lies closest to the value 2.6) the corresponding angle $\alpha = 6$ degrees. It follows therefrom that the value sought is $\alpha = 70$ degrees $+ 6$ degrees $= 76$ degrees.

(145) Example 2: The following were found for the T wave of Bak. in expiration, in tenths of a millivolt: $T_I = 4$, $T_{II} = 1.5$, $T_{III} = -2.5$. We have thus for these waves: $e_1: e_2: e_3 = 4: 1.5: -2.5$. Since the table expresses the relationship of
the potential differences in terms of a maximal value of ±10, the above figures must be multiplied by \( \frac{10}{4} \). We get then: \( e_1: e_2: e_3 = 10: 3.75: -6.25 \). It can be seen from the table that this proportion corresponds to an angle \( \alpha \) which lies between -10 degrees and 0 degrees.

(146) According to the information in the table, the value of \( e_2 \) decreases between 0 degrees and -10 degrees from 5 to 3.5. An interpolation table is therefore sought which lies between these values 5 and 3.5. Such a one is Table V, III. Here one finds for \( e = 3.8 \) (that is, a figure that lies closest to value 3.75) the corresponding angle equals 8 degrees. It follows from this that the value sought is \( \alpha = -8 \) degrees.

(147) It occurs but rarely that one has at his disposal data with which he can calculate \( \alpha \) more exactly than to within 1 degree or 2 degrees. We have already pointed out that it is at times not easy to decide whether the potential differences which occur in any one of the three leads fall in exactly the same phase of a cardiac cycle. Further, there are unavoidable errors in measurement, which, when an adequate string galvanometer is used, are caused less by the imperfections of the instrument or by variations in body resistance than by difficulties in setting the sensitivity of the string exactly at a previously determined magnitude.

(149) We must, however, keep in mind the fact that errors are attached to the measurements, even if only a low per cent. It is therefore often desirable to correct the results of direct measurement before one goes on to calculate the angle \( \alpha \). One takes care thereby that the corrected value always satisfies the

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Table V. Interpolation Table
condition of formula (4), namely, that $e_3 = e_2 - e_1$, for only under these circumstances can it be calculated with the desired accuracy.

The application of the correction can be illustrated by an example. If we assume that one finds for a given wave of the ECG, for example, for the T wave in the three leads, the following values expressed in tenths of a millivolt: $T_I = 10$, $T_{II} = 10$, $T_{III} = -1$. These values must be corrected. Assuming that the percentage of error in the amplitude of the waves in each of the three leads is about the same size, the absolute value of the error will be the least in the small wave $T_{III}$. After percentile correction and rounding out of hundredths of a millivolt, the amplitude of $T_{III} = -1.0$ remains unchanged, while $T_I$ and $T_{II}$ are modified in the sense that $T_I = 10.5$ and $T_{II} = 9.5$. With the help of these corrected values which now fit the requirement of formula (4), the angle $\alpha$ is calculated.

It is worthy of notice that those ECG's are particularly suitable for the calculation of the direction of the resulting potential differences which have a simple form, and in which, for every wave, the formula (4) is almost completely applicable without correction. In ECG's of complicated form in which one can with difficulty recognize the identical phase of a cardiac cycle in the three leads, the calculation of $\alpha$ can be time consuming.

Fortunately many simple forms occur in which, as has been pointed out above, the measurement can be profitably employed without a great expenditure of time.

B. The Manifest Size of the Resulting Potential Difference.—The manifest size of the resulting potential difference is represented in Fig. 22 by the length of $pq$, which we have made equal to $E$. This value must be distinguished sharply from the actual potential difference in $H$ itself. The relation between $E$ and the potential difference in $H$ itself is dependent upon the mutual distance apart of the two points lying in $H$ between which the potential difference is developed.

In our discussion we assume that the distance between the two points mentioned is small compared with the size of the triangle, because otherwise we have no way of accounting for this distance. For this reason nothing can be said of the size of the potential difference in $H$ other than that compared with $E$ it is very large.

The value of $E$ is to be calculated from the values which the leads from the corners of the triangle give. We express it in the angle $\alpha$ and in one of the three projections $e_1, e_2, or e_3$ and write thereby the formulas (1), (2), and (3) in the form

$$E = \frac{e_1}{\cos \alpha} \quad \ldots \quad (7)$$
$$E = \frac{e_2}{\cos (\alpha - 60 \ degrees)} \quad \ldots \quad (8)$$
$$E = \frac{e_3}{\cos (120 \ degrees - \alpha)} \quad \ldots \quad (9).$$
For instance, when $\alpha$ has the value 0 degrees or ±180 degrees, the potential difference in $H$ is parallel to the side $RL$. In these circumstances $pq = pq_1$ or $E = e_1$, and the manifest size of the potential difference is given directly by the amplitude registered in Lead I. In the case $\alpha = 60$ degrees, $E = e_2$.

In the case $\alpha = 120$ degrees we get $E = e_3$.

If one wishes to derive the value of $E$ for any arbitrary value of $\alpha$, one can use any one of the formulas (7), (8), or (9). By means of relatively simple formulas one can express the value of $E$ directly in $e_1$ and $e_2$, $e_1$ and $e_3$, or in $e_2$ and $e_3$. It is, however, simpler to employ again the table of figures.

Example 1: It was stated in Example 1, Section A, that, for Bak. in the state of inspiration, the values recorded were $R_I = 3.2$, $R_{II} = 12.5$, and $R_{III} = 9.3$ tenths of a millivolt while the direction of the resulting potential difference for the $R$ wave was determined by means of the interpolation Table V, II as $\alpha = 76$ degrees.

The value of 10.4 for the manifest potential difference corresponding to this angle is read off directly from the interpolation table concerned. But since the table gives the values in the instance that the maximum of the registered potential difference equals 10, while in our case in fact the maximal value of $R_{II}$ amounts to $12.5 \times 10^{-4}$ volts, the manifest size of $R$ must be calculated as $R_m = \frac{12.5 \times 10.4}{10} = 13.0 \times 10^{-4}$ volts.

Example 2: It was stated in Example 2, Section A, that for Bak. the recorded values in expiration amounted to $T_I = 4$, $T_{II} = 1.5$, $T_{III} = -2.5$ tenths of a millivolt, while the direction of the resulting potential difference of the $T$ wave was determined by means of the interpolation Table V, III as $\alpha = -8$ degrees.

The value of 10.1 for the manifest potential difference corresponding to this angle is read off directly from the interpolation table concerned. Since, however, the table gives the value for the case in which the maximum of the recorded potential difference, $e = 10$, while in our case the maximum value of $T_I$
actually amounts to $4 \times 10^{-4}$ volts, the manifest size of $T$ must be calculated as

$$T_m = \frac{4 \times 10.1}{10} = 4.0 \times 10^{-4} \text{ volts.}$$

(163) If one does not wish to use the interpolation table, one may read off the size of the manifest potential difference directly from Table IV accurately enough.

(164) The sign of the manifest potential difference should always be positive. One needs to introduce no negative values and should consider that the description of the manifest potential difference is complete when one states when it occurs, its direction, and its amplitude.

(165) In Table VI the directions and the manifest sizes of the QRS group of $F_1$, as it was constructed in Fig. 16, are recorded for each one hundredth of a second.

REFERENCES AND NOTES

5. Compare Weiteres, etc., ref. above, p. 564.
6. In these and all subsequent figures one scale division on the abscissa corresponds exactly with 0.04 sec., and one scale division on the ordinate with $10^{-4}$ volts.
7. See ref. above, p. 183.
8. See ref. above, p. 307.
9. See ref. above.
11. For the meaning of the expression “manifest size” consult text to follow.
17. In considering this, no account is taken of the possibility that perhaps the mobility of the heart is greater in the state of diastole than in systole. Consult on this point the text a few lines further.
19. See ref. above, p. 565 et seq.
20. The data are taken from the tracings employed in “Weiteres über das E.K.G.”

*See following page for Concordance in Paragraphs Between Pflüger’s and Lancet Articles.*
**Concordance in Paragraphs Between Pflüger's and Lancet Articles**

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*The concordance is at times exact with no changes other than different paragraphing. At other times, however, a single sentence in the Lancet article is expanded into several paragraphs in Pflüger’s article, and new illustrative material is added. The impression is clear that the Lancet article was written first and was reorganized, edited, and enlarged for publication in Pflüger’s Arch. f. d. ges. Physiol.*

**Concordance in Figures Between Pflüger’s and Lancet Articles**

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The Different Forms of the Human Electrocardiogram and Their Signification

By Professor Wilhelm Einthoven, M.D.,

Of Leyden

The application of electrocardiography to clinical examination gives valuable data in two different ways. In the first place, it shows the time relations between the action of the ventricles and the auricles. The difference in time between the contraction of these two parts of the heart can be measured easily and accurately. Diseases such as Stokes-Adams, in which the auricles have a rhythm different from that of the ventricles, and which sometimes give difficulties when examined by the older methods, are always immediately recognized electrocardiographically. And further it may be mentioned that a new insight has been obtained into several other diseases, among which auricular fibrillation takes a prominent place.

But this evening we shall pass over this first group of symptoms, and we shall limit our attention to the second one, which is concerned with the form of the EKG. If this form is perfect, it must be the exact expression of the changes in potential difference which take place between two parts of the human body. It is self evident that a perfect form can only be obtained by means of a perfect instrument, and it need not be mentioned that such an instrument does not exist. The capillary electrometer is not rapid enough when made sufficiently sensitive, and therefore not suitable for clinical purposes. The oscillograph suffers from the same fault, although in a lesser degree, and has, moreover, the great disadvantage that the movements of the recording mirror are oscillatory. On the contrary, the string galvanometer is in general sufficient for clinical purposes, and by means of this instrument we are able to approximate the exact form of the EKG, so closely that the remaining divergences from it become practically imperceptible.

Not every model of the string galvanometer is equally suitable. Even if we have at our disposal a good instrument in all other respects, the correctness of the work done with it depends in a high degree upon the qualities of a small part of the apparatus, the string, and we have further to take into consideration the magnification and the sensitiveness used.

How is the clinician to know if the curves which he has obtained with the instrument are reliable reproductions of the electrical variations under investigation? He can, it is true, correct the curves obtained by means of data determined from the properties of the instrument, and which are to be found by means of control curves. But such a correction requires a fairly detailed calculation and a reconstruction of the curve, for which reason he will be loth to apply it. And happily this is unnecessary in most cases. It is only necessary to record a control curve in such a way that an equal resistance from a rheostat is introduced into the galvanometer circuit in place of the body resistance, which usually has a value of from 1000 to 2000 ohms.

The tension of the string must be so regulated that an excursion of the image of the string of 1 mm. is equal to 10⁻⁴ volts. At a given moment a constant potential difference is thrown into the circuit. If the string reaches its new position of equilibrium within about 0.01 second or less, the instrument is rapid and at the same time sensitive enough for recording EKG.'s with sufficient accuracy. The deviations from an ideal EKG. which a curve recorded under these conditions exhibits are so small that in the great majority of cases they may be neglected.

To prove this we reproduce in Figs. 1 and 2 two EKG.'s which were taken from the same person by lead I. The sensitiveness of the galvanometer was accurately adjusted in both records, so that 1 scale division corresponded to 10⁻⁴ volts. All other conditions were also kept the same as nearly as possible, with the one exception that in recording Fig. 2 a magnification was used twice as large as in Fig. 1. Therefore the string was stretched twice as much in Fig. 2, and in accordance with this the duration of a deflection was reduced to half.

It is possible to determine the duration of a deflection to a certain degree by means of Fig. 3, which was recorded immediately after Fig. 2 upon the same photographic plate. A resistance of 2300 ohms was introduced into the galvanometer circuit from a resistance box, this resistance being equal to that found for the body of the person investigated under the conditions of the

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1A paper read before the Chelsea Clinical Society on March 19th, 1912.

experiment. The introduction of a potential difference of 3 \times 10^{-3} \text{ volts} causes a deflection of 3 \times 10 \text{ scale divisions}, and each deflection is completed in about 0.5 \text{ scale divisions}—i.e., 0.01 second. The control curve for Fig. 1, which is not reproduced here, shows deflections requiring twice as much time for their completion—i.e., 0.02 second.

Notwithstanding this important difference in deflection time, Figs. 1 and 2 show EKG’s whose forms agree in all details, and whose various peaks have the same absolute heights within the errors of adjustment—i.e., within a few tenths of a scale division.

We are now justified in making the following conclusion. If the movements of the string could be made 10 or 100 times faster, the sensitiveness remaining the same, or even if, theoretically spoken, an instrument were available with an infinitely small deflection time, the form and dimensions of the recorded EKG’s would not be thereby perceptibly changed.

![Fig. 1.-Br. Lead I. Magnification of the movements of the string 660. Abscissa 1 scale division = 0.02 sec. Ordinate 1 scale division = 10^{-4} \text{ volts.}]

![Fig. 2.-Same person and same lead as in Fig. 1. Magnification of the movements of the string about 1200. Value of abscissae and ordinates as in Fig. 1.]

On the other hand, if the movements of the string are sufficiently retarded, the form of the EKG. will also be altered. This is illustrated by Fig. 4. This reproduces the EKG. of the same person taken under the same conditions. The sensitiveness of the galvanometer is, just as in Figs. 1 and 2, accurately regulated so that a deflection of the image of the string of 1 mm. corresponds to 10^{-4} \text{ volts.} But the string moves much more slowly, and we now see that all the peaks of the EKG. are reduced in size, whilst the ratio of the peaks to one another is markedly changed.

Allow me to make a comparison here of the requirements to be demanded of the galvanometer and the accommodation of the eye. A definite adjustment of the eye corresponds to a definite distance from an object that must be focused sharply. Therefore two objects at different distances from the eye can not form absolutely sharp images at the same time, but the physician knows that if the image of an object at 6 metres distance is sharp, the diffusion circles caused by points farther removed are so small that they may be neglected. The accommodation for infinite distance does not differ practically from the accommodation for 6 metres. In the same way we may say that an EKG. recorded with a galvanometer, of which the deflection time is about 0.01 second, does not differ practically from the same EKG. recorded with a galvanometer whose deflection time is infinitely small.

Within these limits the form of the EKG. is independent of the instrument used, and every EKG., when and where it may have been recorded, is immediately comparable with every other EKG.

A parallel can be drawn between image formation in the eye and the recording of the EKG. in still another direction. Suppose that the accommodation of the eye is faultless in a certain case. Even then the retinal image will not be absolutely sharp and exact. The errors of spherical and chromatic aberration, and of defective centration inherent in all optical systems are also found in the eye; and, moreover, the diffraction of the light rays suffices to exclude the formation of an exact image. In the same way there are sources of inaccuracy in the recording of the EKG.; their influence may be decreased, it is true, but can never be completely avoided. I have here
in mind not so much the dexterity of the observer, who must regulate the tension of the string exactly, so that the curves may be recorded in absolute measure, as the unavoidable changes in resistance which the human body offers to the electrical current.

(14) We must make a distinction between the real and the apparent resistance in measuring the resistance of the body. The real resistance is measured by means of an alternating current, in the same way as the resistance of an electrolyte is determined, whereas the apparent resistance is found by making use of a constant current. Under certain conditions the apparent resistance can have a value of more than twice as much as the real resistance, and the question arises, which resistance are we to take into account when regulating the tension of the string. For example, we wish that a deflection of the image of the string of 1 cm. shall exactly represent 1 millivolt, when the human body is in the galvanometer circuit. If the body resistance is large, the string must be relaxed a little; if on the other hand the body resistance is small, the string must be stretched somewhat more tightly, all other conditions remaining equal.

(15) In case the difference between the apparent and the real resistance of the body reaches an important value, the sharp peak $R$ of short duration will be too high in relation to the slow wave $T$, this being an error which is just the opposite of the error produced by a slow instrument.

(16) How can we best reduce these errors and how large is the error remaining finally? In the first place we must make the resistance of the body as small as possible by putting those parts from which the current is lead off, the hands and the foot, deeply into a strong salt solution. Under these conditions the difference between real and apparent resistance is also reduced to a minimum. Further we must use an instrument with a thin string, thus with a comparatively large internal resistance, so that the variations of the body resistance will only have a small influence upon the current strength.

(17) In regulating the tension of the string we can make use of a rheostat resistance, the value of which is the mean of the real and the apparent body resistance. The difference between these two values need not be larger than 400 ohms under the above mentioned favourable conditions, so that the deviation from the mean will not be more than 200 ohms. If we take the resistance of the string to be 10,000 ohms the maximum error in the dimensions of the curves is 2 per cent.

(18) These errors are much less in reality, because peak $R$ is not recorded in an infinitely short time and peak $T$ not in an infinitely long time. Moreover, an error of 2 per cent. in the absolute values of the potential difference is so small that it may as a rule be neglected in physiological as well as in clinical investigations. The error would be 0.4 mm. for a large peak $R$ of 20 mm. height, and only 0.1 mm. for a large peak $T$ of 5 mm.

(19) Although we cannot attain an absolute accuracy at present, yet we may consider an EKG., when properly recorded, the exact expression of the changes in potential difference, just as we may consider the retinal image an exact reproduction of the object observed.
It is desirable to apply three leads in electrocardiographic investigations: (1) from the two hands, across the body; (2) from the right hand and left foot, obliquely; (3) from the left hand and left foot, lengthwise. The galvanometer must be connected to the human body in such a way that the image of the string is deflected upwards when the base or the right half of the heart is negative in respect to the apex or the left half.

Each of the three leads produces an EKG, of special form so that each has its own peculiar value. Lead I has the advantage of being applied easily in the clinic. The curve obtained is of especial value in judging peak T and certain atypical EKG's. With lead II the peaks are usually larger. Especially the auricular peak P is clearly shown and may therefore be best studied with this lead. In judging the wave T in cases of cardiac insufficiency lead II has the same importance as lead I. On the other hand, many atypical forms of the EKG, can be studied better with lead I than with lead II.

Lead III is most suited for diagnosing hypertrophy of the right or left ventricle. Atypical EKG's are also well shown by lead III. On the other hand, the wave T obtained by this lead is of no value in drawing conclusions as to the functioning of the heart. T of normal EKG's, is often negative, and often practically absent.

From the nature of the case there must be a connexion between the curves obtained by the three different leads from the same person. If two forms are known, the third may be calculated from them. The difference between the electrical tensions of leads I and II must be equal to the electrical tension of lead III. This may be formulated: lead II - lead I = lead III. The formula holds good only in case the current which is flowing through the galvanometer exercises no influence upon the tensions present at the extremities. As a matter of fact, this influence is so small that we may neglect it without making a perceptible error. For the total resistance of the circuit containing the galvanometer and the human body is very large when compared to the resistance of the heart muscle. We may therefore assume a priori that the formula is exact.

We might attempt to make use of this formula when examining patients, for the sake of simplicity contenting ourselves with the recording of two leads. The curve of the third lead would then be constructed from the other two. Although the construction seems to be easy, yet its execution gives difficulties, the main cause of which lies in the fact that the peaks, which we generally indicate by means of the same letters, do not occur in identical phases of a heart's revolution. It is possible to show this in various ways—e.g., by comparing the distances between the peaks P, R, and T in the curves obtained by lead I with the corresponding distances in the curves obtained by lead II and lead III. It then appears that the peaks mentioned stand closer to one another in lead I.

A superficial examination of certain curves—for example, those of Fl. (Figs. 5, 6, 7), in which the three leads exhibit fairly large differences—might appear to favour a doubt as to the practical validity of the formula. But it is not difficult to show that the rule is also in accordance with the facts.

The simplest way to do so would be to record two or three curves by the various leads immediately under one another. Two or three large string galvanometers should be used and the images of the three strings should be projected by means of mirrors upon the same photographic plate. It seems to me that the use of a small model of the galvanometer, in which two or more strings are placed in the same magnetic field, or the use of an oscillograph containing a number of mirrors, is not suited to this purpose, because these instruments do not reproduce the form of the EKG, accurately, whereas it is especially desirable in this case to have the form as exact as possible.

Another method has been applied by Fahr and de Waart. They have recorded the EKG, together with the heart sounds, according to the method of Kahn. The EKG's obtained by the three leads were taken from the same person under exactly the same conditions, the curve of the heart sounds being combined with each EKG. By this means a fixed point falling always in the same phase of a cardiac cycle could be marked, and corresponding phases in the curves of the various leads could be accurately identified.
The investigation of Fahr and de Waart shows that the ventricle EG, when obtained by leads II and III begins, as a rule, a little earlier than when obtained by lead I, whereas the summit of R\textsubscript{I} is reached a little earlier than the summits of R\textsubscript{II} and R\textsubscript{III}. If we make use of these data it is not difficult to show the validity of the formula, even in complicated forms of the EKG. To illustrate this we have reproduced in Fig. 8 QRS-group of the EKG.'s of Fl. shown in Figs. 5, 6, and 7. The three curves are constructed in the same system of coordinates. The abscissae

Fig. 5.—Fl. Lead I. Abscissa 1 scale division = 0.04 sec. Ordinate 1 scale division = 10^{-4} volts.*

Fig. 6.—Id. Lead II.

Fig. 7.—Id. Lead III.

have been increased 4 times more than the ordinates, so that the whole figure has been stretched in the direction of the abscissae; one scale division of the abscissae represents 0.01 sec., one scale division of the ordinates 10^{-4} volts. Each vertical line corresponds to one phase of a cardiac cycle and the formula lead II - lead I = lead III holds good for every phase. We see that the peaks Q\textsubscript{II} and Q\textsubscript{III} begin 0.01 sec. before R\textsubscript{I} and fall in the anacrotic branch of this peak. The whole peak S\textsubscript{I} falls in the catacrotic branch of R\textsubscript{II} and R\textsubscript{III}, whilst the whole of peak S\textsubscript{II} only corresponds with the anacrotic branch of S\textsubscript{I}.

*If not otherwise mentioned, the abscissae and the ordinates in all figures have the same value as in Fig. 5.
Similar constructions can be made, as a rule, without great difficulty and can be applied to peaks $P$ and $T$ as well, but it is often difficult to attain a great degree of accuracy. This is especially true when the height of a small peak must be determined from the difference in the heights of two peaks of nearly the same size. If $T_I$ and $T_{II}$ are of approximately the same height, as is often the case, then the value of $T_{III}$, calculated by means of the formula, is very inexact.

Fig. 8.—Construction of the QRS group in the three leads. Each ordinate represents identical phases of a cardiac cycle. Abscissa 1 scale division = 0.01 sec. Ordinate 1 scale division = $10^{-4}$ volts.

It is thus evident that we should not content ourselves with taking only two leads, for the dimensions of a curve constructed by calculation can be determined not only more simply but also more accurately by directly recording it by the third lead. The great advantage of a construction as in Fig. 8 is the better insight obtained into the QRS-group.

This group deserves a closer examination because it forms the beginning of the ventricular EG. and shows the path by which the excitation wave is conducted from the auricles to all parts of the ventricular wall. This group takes very different forms in normal hearts, so that it is sometimes impossible to point out the peaks $Q$, $R$, and $S$. The examination of even a small number of curves will show that the inconstant peak $Q$ fails in some EKG.'s, the inconstant peak $S$ fails in others. In some EKG.'s $S$ is as large as $R$, and in some others both $Q$ and $S$ fail so that only $R$ remains.
We reproduce here an EKG of Wi. (Fig. 9), who possesses a normal heart. The curves obtained by leads I and II show large peaks R, as is often the case with normal hearts. The curve reproduced above and taken by lead I exhibits an entirely different form of the QRS-group. This group is here represented by a slight elevation, about as high as P and about one-third as high as T. On closer examination it is seen to consist of two small upwardly directed peaks, placed close beside one another. It is impossible to say whether they are Q, R, or S. The same is true of many normal as well as many pathological curves taken with lead III. A single example of such a (QRS) group may suffice (Fig. 10).

We have here again two upwardly and two downwardly directed peaks, no one of which may possibly be distinguished as Q, R, or S. T III is negative in the EKG of this person.

The explanation of the ventricular EG. is based upon this great variability in the QRS-group. As soon as the excitation wave has reached the ventricular walls through the atrio-ventricular bundle and the Purkinje fibres, the ventricular musculature begins to contract in many places at the same or nearly the same time. The atrio-ventricular bundle, according to the investigations of Aschoff-Tawara, consists of a system of muscle fibres, well likened to a tree with its branches. From their origin in the auricular septum to their arborisation in the walls of the lower chambers of the heart, these fibres are separated from the rest of the heart muscles by connective tissue. The bundle does not enter into connexion with the heart musculature excepting with its end ramifications, which are spread over a large surface in the walls of the ventricles, fusing with them at many points. The great variety of forms of the QRS-group is caused by small variations in the architecture and the conductivity of the bundle and the Purkinje fibres, so that the excitation wave in some hearts reaches the right side 0.01 second sooner than the left, or the base sooner than the apex, whereas the opposite sequence occurs in other hearts.

The contraction wave spreads over the whole ventricular musculature within a few hundredths of a second, and all changes in potential disappear temporarily. This happens in the time between group QRS and the beginning of the T wave.

The potential difference indicated by the galvanometer in any given moment is the resultant of the joint potential differences existing at that moment between the various parts of the heart. If we consider the phenomena from this point of view we may agree with Seienin, who regards the EKG. as the algebraic sum of the changes in potential, caused by the contraction of the right and left chambers. We also may agree with Eppinger and Rothberger, who assume that the EKG. is the resultant of the forces in the constrictor fibres and in the longitudinal fibres of the heart musculature. Neither is there any objection to the idea of the EKG. being the expression of the antagonism between base and apex. For we may divide the heart into two arbitrary parts, and the EKG. will always be the resultant of the changes in potential differences in these parts. The curve must represent, under all circumstances and in every moment, the algebraic sum of all the potential differences which at that moment are developed in the heart.

We must now point out two peculiarities of the EKG. which up to the present have been considered worthy of only slight consideration. In the first place, the existence of a wave, which
comes after the T wave and may have a considerable height in pathological cases. This wave U is also present in the curves from normal hearts, but in these cases it is very low as a rule. Lewis estimates that wave U is present in three-quarters of all EKG.'s taken by lead II. After an examination of my collection of EKG.’s I also conclude that the presence of peak U is very common, at least in half of all persons.

Figs. 11 and 12 are Illustrations of EKG.'s with fairly high peaks U, obtained from persons with normal hearts. Fig. 11 is from a sportsman, aged 37 years, with a strong musculature; Fig. 12 is from a well-built young man, 19 years of age, of somewhat more than the average height. Wave U reaches a value of about $0.5 \times 10^{-4}$ volts in both curves, but is not of equal height in all heart beats. This wave must be regarded as an inconstant one.

The significance of it and the reason for its inconstancy are for the present not known with surety. The method of recording the heart sounds, together with the EKG., has shown that the second sound is produced almost immediately after peak T is finished. The end of U lies after the second sound, so that there is no doubt that wave U fails wholly or partly in that phase of the cardiac cycle which follows the closing of the semilunar valves. The heart is in diastole after the closing of these valves. But the heart muscle, which does not begin to contract at all points at the same time, does not relax at all points at the same time either. A large part of the heart can have already relaxed, so that the pressure in the heart’s cavities may be practically diminished to zero, and at the same time a few fibres may still be in the condition of contraction. Peak U will not have reached its end until these latter fibres are fully relaxed.

The Interval between Auricular and Ventricular Contraction.

In the second place, we wish to direct attention to the position of the galvanometer in the interval between $P$ and the $QRS$-group. In many cases the string takes the same position here as that held during diastole, so that a horizontal line is recorded at the level of the line of diastole. But in many other cases there is a perceptible difference in the positions of the lines. We wish to illustrate this with only one example (Fig. 13). We see in this figure that the curve between $P$ and the $QRS$-group lies about 1 mm.—i.e., $10^{-4}$ volts lower than during diastole. To what must this be ascribed?

Is the conduction through the atrio-ventricular bundle accompanied in this case by the development of a perceptible electrical current, or is a potential difference developed even during the diastole? Have we perhaps to consider a generation of electricity effected by the same causes that produce peak U? For the present we shall not choose between the possibilities mentioned, but shall content ourselves with the information that curves like Fig. 13 are often obtained from persons with normal hearts.

The Influence of the Position of the Heart.

The position of the heart influences the form of the EKG. In electrocardiographic investigations we are chiefly interested, however, in getting a better insight into the functioning of the heart and it is easy to understand that a difficulty must arise in drawing conclusions as to this point from the form of the curve, if a displacement of the organ is sufficient in itself to produce a change in the EKG.

The difficulty is best solved by making ourselves accurately acquainted with the effects of a shifting and a rotating of the heart. The easiest and at the same time the most striking example of the influence of the position of the heart upon the form of the EKG, is offered by a case of situs inversus viscerum. Waller was the first to investigate such a case, and since then a number of other cases have been published. I permit myself here, moreover, to publish a curve taken by Dr. Fahr from a boy aged 7 years by lead I. We see immediately that the peaks of Fig. 14 are exactly the reverse of a normal EKG. If we interchange the conducting wires to right and left hands we obtain a curve which cannot be distinguished from a normal EKG, as is shown in Fig. 15.

Whereas the EKG, in situs inversus may be very easily explained and gives no cause for a more extended discussion, other cases of change in position of the heart are accompanied by more complex phenomena, and these require a more detailed analysis for their understanding. We
Fig. 10.—Jo. Lead III. A remarkable QRS group.

Fig. 11.—Br. Lead II. Wave U visible.

Fig. 12.—Sch. Lead II. Wave U visible.

Fig. 13.—Em. Lead II. The level of the curve between P and Q is different from the level during diastole.

Fig. 14.—Situs inversus viscerum. Lead I. Abscissa 1 scale division = 0.02 sec. Ordinate 1 scale division = 10^{-4} volts.
shall here discuss in particular the deviations in the position of the heart which accompany the
movements of respiration.

(45) Movements of the diaphragm participate to a greater or less extent in the respiratory move-
ments of women as well as of men, and the heart must be displaced in the thorax whenever the
diaphragm takes a higher or a lower position.

(46) Even before the construction of the string galvanometer Samojloff demonstrated to his
classes that increased respiratory movements had a perceptible influence upon the oscillations
of the mercury meniscus of the capillary electrometer connected with the human body. By
lead I the oscillations became larger in the phase of expiration and smaller in the phase of inspira-
tion. Many other investigators have examined the same phenomena. In our description we
shall make use of curves which were taken with the help of Vaandrager and later with the help
of Fahr and de Waart. In Figs. 16 and 17 we see pieces of a curve which was taken from Bl.
by lead I. The upper curve is the pneumogram.

(47) The pneumogram was obtained by recording the movements of a Hutchinson spirometer
into which the person under investigation breathed. A movement of the recording lever upwards
corresponds to an inspiration, a movement downwards to an expiration. Each scale division
of the ordinates represents 250 c.c. inspired or expired air; the amplitude of the oscillations of the
pneumogram varies between 11.3 and 11.5 scale divisions—i.e., 2.8 to 2.9 litres. The vital capacity
of the person amounted to 4.4 litres; thus the respiratory movements during the recording of the
curves were about two-thirds of the vital capacity. These movements were sufficient for our
purpose, and at the same time the subject could execute them with ease.

(48) The pneumogram reproduces the changes in the volume of the thorax without practically
any delay. This may be shown by means of the trembling of the subject at the height of inspira-
tion. The effect of this trembling caused by the strong muscle contractions necessary for the ex-
cessive movements of inspiration is to be seen clearly in the EKG. As soon as the inspiration is
ended the oscillations in the EKG. disappear, for the muscles relax and passive expiration begins.
We may say that the end of the trembling coincides with the top of the wave of the pneumogram,
if we neglect small differences in time—as, for example, differences of five scale divisions, or 0.2
second.

(49) Apart from the form of the EKG. and the height of its peaks there is a striking variation in
the heart's frequency during the movements of respiration. I should like to point out that the
maxima and minima of the heart's frequency do not correspond with the crests and troughs of the
pneumogram. For instance, the longest pause in the heart's action is found at the end of
Fig. 16, the shortest pause at the beginning of the same figure, whereas the volume of the lungs
is about the same in both places. The volume of the lungs is also the same at the beginning and
at the end of Fig. 17, whereas the heart's frequency differs markedly in these places.

(50) The explanation of this phenomenon is generally known; during very slow and deep respiratory
movements the gases of the blood change sufficiently to influence the tonus of the vagi, especially
at their origin in the medulla. As the maximum and minimum of vagus tonus do not correspond
to the extreme positions of inspiration and expiration, the time of the greatest and the time of the
least frequency of the heart will also be displaced in respect to these positions. This displacement
varies largely from person to person. We have mentioned this circumstance here because it
must be taken into account whenever the influence of the respiratory movements upon the form
of the EKG. is discussed. For we know that the form and magnitude of peak P as well as of wave
T can be materially changed by the action of the vagi.

(51) In Figs. 18 and 19 we reproduce two pieces of a curve, which was taken from the same person
by lead III. The influence of the vagi upon the form of peak P is shown very clearly in these
figures. After long heart pauses P is small, sometimes diphasic, other times totally negative,
whereas P has its normal form and magnitude after small pauses—i.e., with increased frequency
and decreased vagus tonus.

(52) The above mentioned facts make it evident that we meet with a difficulty when estimating
the influence upon the form of the EKG. of a displacement of the heart caused by the respiratory
movements. Peak P is influenced by two factors, and it is not easy to separate them from one
another nor to estimate the magnitude of each alone. We shall therefore concentrate our atten-
tion upon some other peaks of the curve.
Fig. 15.—Id. The connexions of the electrodes reversed.

Fig. 16.—Bl. Lead I during inspiration.

Fig. 17.—Id. Lead I during expiration.

Fig. 18.—Id. Lead III during inspiration.

Fig. 19.—Id. Lead III during expiration.

Fig. 20. Bak. Lead I during inspiration.
We see from Figs. 16-19 that the peaks of lead I are larger during expiration, whereas by lead III they become smaller during the same phase of respiration. If we consider these facts in connection with the formula, lead III = lead II - lead I, we must conclude that the changes in the peaks of lead II will be very slight. And this is, in fact, the case with BI. and with the majority of the persons investigated. The practical result of this is that we must apply this lead when examining a patient if we wish to investigate the form of the EKG. as free from the influence of the respiratory movements as possible.

Further, we wish to remark in the above figures that peaks R and T are both decreased or both increased in the same degree as well as lead I during the various phases of breathing. Thus the relative heights of the peaks remain the same, so that the curve retains its form.

But similar changes are not observed in all persons. In some persons we find differences which upon superficial examination seem to be wholly irregular and thereby puzzling. We shall first describe some of these differences separately, and shall then try to unite them all under one point of view.

In the first place we shall direct attention to the fact that the changes which peak R undergoes are, in many cases, materially larger than the corresponding changes in T. Figs. 20, 21, 22, and 23 are examples of this. Figs. 20 and 21 reproduce the EKG. of Bak. by lead I. Two pieces have been cut out of the original curve in such a way that Fig. 20 renders that portion of the record taken in inspiratory position, and Fig. 21 renders that portion taken in expiratory position. In this latter figure we see that the enlargement of peak R is materially greater than that of the T wave, whereby the curve receives quite another shape.

An even greater disparity is brought to light in Figs. 22 and 23, which were obtained from Wi. Here we have again two pieces of a curve taken by lead I; Fig. 22 corresponds with the inspiratory, Fig. 23 with the expiratory position of the person under investigation. In the first position R is larger than T, in the second the relation is reversed.

Another difference found is that a peak which becomes larger in lead I also increases in lead III. This phenomenon occurs whenever the peaks have opposite directions in the above-mentioned leads. Figs. 24 and 25 furnish an example of this. They reproduce the EKG. in lead III of the same person whose EKG.'s in lead I were shown in Figs. 20 and 21. Wave T is negative in lead III and we see that T in lead I has increased as well as T. Phenomena similar to those described above are to be observed in the QRS group.

But we shall now try to explain the apparently so irregular effects of the respiratory movements upon the form of the EKG. In the first place we exclude the possibility that such potential differences as are shown by our curves might be due to accidental causes—as, for instance, the muscle contractions necessary for the respiratory movements, or to a displacement of the electrodes. For if such secondary causes should exercise an influence synchronous with the movements of respiration, then the curved line which reproduces a series of EKG.'s would have to rise and fall in the same rhythm as the movements of breathing. But we see that the line drawn through the heart pauses does not show such a rhythm. On the contrary, it has in many curves an absolutely straight course, as may easily be seen with the aid of the system of square millimeters.

As a second possibility to be taken into account we may mention the influence of a changing body resistance, a hypothesis pointed out by Samojloff. The electrical resistance of the organs surrounding the heart must show during breathing rhythmic changes due to changes in the blood and air content of the chest, and these changes in resistance must necessarily influence the heights of the peaks of the electrocardiogram. But this influence is obviously small, for the curve of lead II shows only slight changes as a rule, whereas the changes produced in lead III are as a rule the opposite of those in lead I. Moreover, the fact that the relation between the heights of the peak is often changed cannot be explained by a change in the resistance of the body.

As a third cause we may mention that the manner in which the heart contracts depends upon the pressure in the thorax. The negative interpleural pressure, which increases during inspiration and decreases during expiration, mechanically influences the circulation perceptibly. But this influence is also insufficient to explain the phenomena described.

The difficulties are solved at once, however, if we apply a schema in which the human body is represented by a plane, homogeneous plate in the form of an equilateral triangle, RLF. (Fig. 26.)
Fig. 21.—Id. Lead I during expiration.
Fig. 22.—Wl. Lead I during inspiration.
Fig. 23.—Id. Lead I during expiration.
Fig. 24.—Bak. Lead III during inspiration.
Fig. 25.—Id. Lead III during expiration.
From its corners the current is lead off to the galvanometer. \( R \) corresponds to the right and \( L \) to the left arm, while \( F \) indicates the potential of both feet. Thus, a lead from \( R \) and \( L \) corresponds to lead I, from \( R \) and \( F \) to lead II, and from \( L \) and \( F \) to lead III. A small spot \( H \) in the middle of the triangle represents the heart. We assume that in a given moment the potential differences in the heart are so distributed that their resultant takes the direction of the arrow drawn in the figure. This direction indicates the direction of the current in the heart. The angle which the arrow forms with the side \( RL \) is called \( \alpha \). We shall call the angle positive if, when looking towards the front wall of the chest of the patient, the arrow rotates with the hands of the clock; negative if it rotates in the opposite direction.

We now suppose that the EKG of a patient has a simple form in each of the three leads, so that the peaks \( R_I, R_{II}, \) and \( R_{III} \) fall in corresponding phases of a cardiac cycle, and \( R_{II} - R_I = R_{III} \). If we transfer the values found with the patient to the schema we are able to determine the direction of the potential difference which has been present in the heart during the recording of \( R \).

The EKG of Bak., in the position of inspiration may be taken as an illustration. In his curves the following values are found for \( R \): \( R_I = 3.2, R_{II} = 12.5, R_{III} = 9.3 \) millivolts. In the homogeneous triangular plate with the heart in the centre these values can only be produced by a potential difference, having the direction of the arrow drawn—i.e., that \( \alpha = 76^\circ \).

In the position of expiration we find for the same person: \( R_I = 9.2, R_{II} = 11.2, R_{III} = 2.0 \); wherefrom we can calculate that under the circumstances \( \alpha = 40^\circ \) (Vide Fig. 27.)

Thus we are able to demonstrate by means of the schema that the heart has rotated in the chest around a sagittal axis during the movement of expiration. The amount of rotation according to the schema is \( \alpha = -36^\circ \). As we have reason to believe that the real amount of rotation in the body corresponds sufficiently well for practical purposes with the amount of rotation in the schema, we shall hereafter simply consider the direction of the potential difference in the body as parallel to the direction of the arrow in the schema. The way in which \( \alpha \) can be calculated from the potential differences recorded need not be explained in detail. It may suffice to state that the calculation is simple and can be executed very quickly.

The schema can show more than the direction of the potential difference only. It also makes it possible for us to compare the values of the potential differences present in the heart itself. To make this clearer we shall introduce a new term, "the manifest potential difference in the heart," and when speaking of certain peaks, \( P, R, \) or \( T \), we shall indicate them with the figures \( P_m, R_m, \) or \( T_m \). By these figures is meant the manifest values of the peaks.

We define the manifest potential difference in the heart as the amount shown in one of the three leads, when the direction of the current between the points of leading off is parallel to the direction of the potential difference in the heart. We here consider only potential differences in a frontal plane. Suppose that a given potential difference in the heart forms the peaks \( T_I, T_{II}, \) and \( T_{III} \), and that the direction of the current in the schema is represented by an arrow parallel to the side \( RL \) (see Fig. 28), then \( T_I \) is the largest of the three peaks, \( T_{II} \) and \( T_{III} \) being opposite to one another, and each equal to one half of \( T_I \). In this case \( T_m = T_I \).

The manifest value of a peak can always be calculated from the height of the recorded peaks and thus can always be expressed in absolute measure—e.g., in tenths of a millivolt. This calculation is also easily and quickly made. If we apply it to peak \( R \) of Bak., the dimensions of which have already been given, we find \( R_m = 13 \times 10^{-4} \) volts in the position of inspiration, \( R_m = 12 \times 10^{-4} \) in the position of expiration.

In another person a rotation of the heart of \(- 12^\circ\) was brought about by expiration, \( R_m \) amounting to \( 27 \times 10^{-4} \) volts in a position of inspiration and to \( 26 \times 10^{-4} \) volts in a position of expiration. In a third person a rotation of \(- 20^\circ\) was found, whilst \( R_m \) kept the constant value of \( 20 \times 10^{-4} \) volts. In all persons examined till now the rotation takes place in the same direction in expiration, whilst the manifest height of \( R \) keeps constant within the limits of the errors of the method.

We can investigate the direction and the manifest value for \( T \) in the same way as for \( R \). As is to be expected, \( T \) is always rotated by the movements of respiration in the same direction as \( R \), but the amount of rotation is not exactly the same. Moreover, the manifest value of \( T \) changes—a fact which points out that other factors besides the rotation of the heart are active in the formation of \( T \) during respiration. The vagus tonus is one of these factors.
Fig. 26. Schema of the equilateral triangle. R corresponds to the right hand, L to the left hand, and F to both feet. The heart, H, is in the centre. The arrow indicates the direction of the potential differences in the heart. $\alpha = 76$ degrees.

Fig. 27.—Id. $\alpha = 40$ degrees.

Fig. 28.—Id. $\alpha = 0$ degrees.

Fig. 29.—Fl. Lead I, patient lying on his left side.

Fig. 30.—Id. with same lead, lying on his right side.
There are good reasons for assuming that the most prominent peaks of the ventricle electrogram, \( T \) and \( R \), are brought about by potential differences localised in approximately frontal planes. This is proved by the behaviour of peak \( S \) in lead 1. When a person lying horizontally upon the left side turns upon the right side whilst his EKG. is taken, we obtain the striking phenomenon that peak \( S_1 \), which is often very small or not present at all in the first position, becomes visible and sometimes reaches a prominent height in the second position. Figs. 29 and 30, both of which were taken by lead 1, furnish an illustration of the production of a large peak \( S_2 \) by the turning of the body on to the right side. Fig. 29, which was recorded while the patient was lying upon the left side, shows a small peak \( S \), which may almost be neglected, whereas Fig. 30, which was obtained while the patient was lying upon the right side, shows a peak \( S \) so large that we are inclined to be astonished that both curves have been obtained from the same person, with the same lead, and, with the exception of the body position, under quite the same circumstances.

The most satisfactory explanation of this, upon superficial examination, so puzzling phenomenon is to be found in the assumption that the heart rotates in the chest about the longitudinal axis of the body, when the body itself turns from the right upon the left side. If there is a potential difference in the heart having a sagittal direction, then a slight rotation of the organ about the longitudinal axis of the body can produce a material change in the height of the corresponding peak, and this will be especially well shown by lead 1. If the potential difference has exactly the sagittal direction the peak entirely disappears. It is probable that the large differences in \( S \) in different persons, as well as its inconstancy, must, as a rule, be explained by this circumstance.

We direct attention to the fact that in Figs. 29 and 30 the peaks \( P \), \( R \), and \( T \) undergo hardly any change. If we conclude from the enormous change in \( S \) that a rotation of the heart has taken place round the long axis of the body, then we must also assume that the peaks \( P \), \( R \), and \( T \) are developed in approximately frontal planes, or in other words, in planes making relatively large angles with the sagittal plane, for thereby it can be most easily explained that under these circumstances the changes in these peaks are so small.

The application of the schema of the equilateral triangle accounts for a number of phenomena here treated of. First it is immediately obvious from the schema that a comparatively large displacement of the heart parallel to itself will only slightly affect the heights of the peaks recorded. This explains the fact that the EKG. of many persons is hardly perceptibly changed or is not changed at all during the ordinary movements of breathing. It also explains the fact that with the exception of peak \( S \), there are only slight changes produced in the EKG., when the body is turned from one side to the other, although we must expect from the anatomical conditions that the heart has hereby taken a lower position in the chest.

The schema explains easily and satisfactorily how the various peaks of the EKG. are often unequally increased or decreased by a change in the position of the diaphragm, and why the curve of lead II shows slighter changes than the curves of leads I and III. A potential difference which is present in the heart and has a direction which in the one position of the heart makes an angle of 80°, and in the other position an angle of 40°, according to the schema, causes the formation of a peak which remains unchanged in lead II, but which is very much altered in lead I as well as in lead III. If this peak in lead II has the value \( 9.4 \times 10^{-4} \) volts in the first position of the heart, it keeps this value in the second position. It increases in lead I from \( 1.7 \times 10^{-4} \) volts in the first position to \( 7.7 \times 10^{-4} \) volts in the second. It decreases in lead III from \( 7.7 \times 10^{-4} \) volts in the first position to \( 1.7 \times 10^{-4} \) volts in the second.

It is equally clear why a peak which has the same direction in leads I and III undergoes opposite changes by a displacement of the diaphragm, whereas it is either increased or decreased in both leads if it has a positive value in lead I and a negative in lead III. This is the case when \( \alpha \) has a value between \(-90°\) and \(+30°\).

With the aid of the schema we are in a better position to judge the form of the EKG. under all circumstances. For instance, it is possible therewith to show that an increased frequency of the heart brought about by bodily exercise is always accompanied by a positive rotation of \( T \) and by an increase in the manifest value of this peak. In myodegeneratio cordis and in many cases of auricular fibrillation \( T \) is small in all three leads. It need not be said that \( T \)'s must also be small under these circumstances.
In cases of hypertrophy of the left ventricle we sometimes find the most prominent peak of the QRS-group in a direction represented by $\alpha = 40^\circ$, whereas in normal hearts the value of $\alpha$ for this peak lies between $+40^\circ$ and $+90^\circ$. We see from these figures that the deformation of the EKG. in this heart affection is not, or not chiefly, caused by a change in the position of the heart. The manifest value of the most prominent peak of the QRS group in hypertrophy of the left ventricle can reach the enormous value of $26 \times 10^{-4}$ volts. In hypertrophy of the right heart we obtain, as is well known, EKG.'s of an entirely different form, to which, however, similar considerations may be applied. By means of the schema we can in general differentiate the real changes in the heart's action from the apparent ones, which are only caused by variations in the position of the heart.

Atypical contractions may also be examined with the aid of the schema. They are of great importance in the clinic, and are worthy of a special, detailed discussion, but I must conclude now.

It gives me an especial pleasure to bring to remembrance here that the human EKG. was first recorded by a London physiologist, Augustus D. Waller, who also introduced the term "electrocardiogram" into science; and that Dr. Thomas Lewis, whose extensive researches have been crowned with such exceptional success, was the first man in England who applied electrocardiography to clinical investigations.

The method of electrocardiography is still a young plant. We may reasonably expect that it will continue to bear good fruit.