ON THE ELECTROCARDIOGRAM. By W. A. JOLLY. (From the
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(With forty figures in the text.)

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THE interpretation of the electrocardiogram is not yet complete. Notwithstanding the large additions which have been made in recent years to our knowledge of the electrical changes accompanying the heart beat, much remains doubtful. Before the electrocardiogram can be used with certainty as a guide in the investigation of the course of excitation and contraction in the cardiac musculature, and still more in the diagnosis of pathological conditions, it is necessary that we should obtain a more exact knowledge than we possess at present of the nature of the electrical disturbance which passes over the heart when it is active.

There are two problems which await final solution relating to (1) the form of the electrical disturbance in active heart tissue, and (2) the course which excitation takes in passing over the heart. It is evident that if either of these is satisfactorily solved it will be possible to elucidate the other.

Much work has been undertaken by very various methods to determine the point or points of the cardiac musculature which first enter into activity in systole, and to trace the subsequent path of the wave of excitation and its accompaniments.

The electrocardiogram has in recent years been largely employed in the investigation. A brief survey of the results published shows how divergent opinion has been on the subject.

It was for a long time the accepted view (1), based on the work of Engelmann, Burdon-Sanderson and Page, Gaskell and others, that contraction passes as a wave over the ventricle from base to apex. MacWilliam (2) was able to observe directly the wave passing over the mammalian ventricle from base to apex when the beat was slowed by the action of potassium bromide.

Waller and Reid (3) found that contraction in the isolated mammalian heart begins earlier at apex than at base, and discussed the possibility that the excitation passes from the auricles through the papillary system to the apex and from here back to the base. In the excised cold-blooded heart the contraction begins almost always at the apex.

Bayliss and Starling (4) observed that in the carefully prepared

mammalian heart the base always enters into activity before the apex, but that on cooling this is reversed. The reversal is attributed to conduction by a nervous path from the base to the apex, and by a muscular path from apex to base.

Similar results were obtained by Schlüter (5), who applied the nerves of two rheoscopic nerve-muscle preparations to different parts of the ventricle and recorded the contractions of the muscles.

Albrecht (6) concluded on anatomical grounds that contraction begins at the apex.

Lohmann (7) states that as a consequence of slight injury the direction of the contraction wave is reversed.

Fauconnier (8) assumes that under normal conditions excitation passes from base to apex, but that in the case of artificially produced extrasystoles the wave of excitation may begin at any part of the heart and spread radially thence.

Gotch (9) found that in the tortoise ventricle the active process appears to pass as a wave from the auriculo-ventricular junction through the ventricular substance towards the apex, but returns to the neighbourhood of the aorta, the last part of the ventricle to commence contracting being that which lies near the exit of the aorta. The events are similar in the mammalian heart. Each contraction begins in the general mass of basal ventricular substance; it then develops at the apex, and finally in the ventricular mass near the aorta.

Kraus and Nicolai (10, 11) come to a similar conclusion with regard to the mammalian heart. The first part of the ventricle to become active, according to these authors, is the basal part of the papillary system, that is to say, activity begins in the internal part of the ventricular base and proceeds in the interior of the ventricle towards the apex. Near the apex, i.e. at the base of the papillary system, the contraction is transmitted to the general ventricular musculature, the fibres of which contract together. Finally, the base becomes again negative, not because this part has remained negative but because the excitation has ascended a second time to the base. Hofmann (12) states that the normal course of excitation in the frog ventricle is always from base to apex. This author quotes the results of Pagliani and Kaiser, who found that the contraction of the heart muscle in response to stimulation may begin at a distance from the point of stimulation, and of Bethe, who states that in strips of the dog's heart he has observed on stimulation at one end that the contraction begins at the other end. Hofmann regards these conclusions as erroneous.

Meek and Eyster (13) have traced the course of the negative change over the ventricle of the tortoise, and conclude that activity passes over the muscle in the form of a wave which begins at the anterior part of the auriculo-ventricular ring and terminates at the apex.

Eiger (14), from his study of the electrocardiogram in the dog, concludes that the excitation is not transmitted immediately to the base of the

ventricle but to a region between base and apex, and thence spreads simultaneously in two chief directions to the base and to the apex.

Clement (15), making use of the method of differential electrodes, finds that the wave of activity passes in the tortoise ventricle practically simultaneously from base to apex in the basal part of the ventricle and from apex to base in the apical part. In one case, where the preparation had been cooled to 4° C., it was observed that activity began in the frog's ventricle 13 σ earlier at the base than at the apex, but under normal conditions almost simultaneous appearance of activity at different points on the ventricle surface is found.

Boruttau (16), whose method is to injure the middle part of the frog's ventricle and to record monophasic variations, confirms Clement's findings that the action currents begin practically simultaneously at base and apex.

The electrical changes have been studied by Lewis and Oppenheimer (17, 18), Wybauw (19), Eyster and Meek (20), and others, with a view to determining the point which becomes primarily negative in the auricles. The method employed has been to apply electrodes to two points on the surface and to take the point which becomes primarily negative to the companion point as becoming active before it. The sequence of activity is traced in the same way. Erlanger (21) has criticised this method, and points out that primary negativity at a point on the surface of the heart may indicate that active tissue lies close to the surface at that point or that it is specially accumulated there, while the point of real initial activity may lie elsewhere in a part whose negativity affects the galvanometer less. Certainly on examination of Eyster and Meek's curves it does not always appear conclusive that the first movement of the string indicates negativity of the point mentioned by them. In fig. 9b of their paper, for example, the first small movement would seem rather to indicate negativity elsewhere.

The following paper gives the results of a study of the form of the electrical change which passes over the cardiac muscle when it enters upon activity. The experiments have been made upon the tortoise and frog hearts, either in situ with the pericardium open, or isolated. The galvanometer used is Einthoven's string-galvanometer (Edelmann's large model), and the movement of the string has been recorded photographically upon sensitised paper with a magnification of 660.

From recent work (14, 22, 23, 24) we may take it as proved that warm-blooded and cold-blooded hearts yield essentially the same form of electrocardiogram, and indeed each chamber of a heart on systole gives us a similar curve. The curves are not invariably the same. They differ in details, but we may assume that there is a characteristic variation given by cardiac tissue when it actively contracts, and that an adequate analysis of the curve will apply equally well to the chambers of the cold-blooded and human hearts.

We can therefore, in treating of the electrocardiogram generally, use the nomenclature introduced by Einthoven for the curve recorded from the human heart. In the present state of our knowledge it appears desirable to use designations for the summits which do not involve theories as to their production and meaning. Until interpretation is further advanced, to alter the lettering introduced by the observer who described for the first time all the peaks that we now recognise as present in the usual curve might lead to confusion, and would have little more value than to change the designation of an unknown from x to y . The ventricular variation may be described as consisting of an initial or QRS complex, an intermediate stretch, and a T complex. Such a description does not commit one to the statement that either Q or S is always present, or that the T complex consists of one or more deflections.

It seems advisable also to adhere to the form of record employed by Einthoven, in which the summits which would indicate galvanometric negativity of the base are directed upwards. In this paper the electrode connected with the lower end of the string, galvanometric negativity of which causes an upward movement in the record, is termed the right-hand or proximal electrode, and the other whose negativity depresses the curve is the left-hand or distal electrode. Eiger (14) has inverted the direction of the summits in his records, but this seems to introduce an additional complication into the literature on the electrocardiogram without obvious compensating advantages.

Einthoven (25) offers an explanation of the summits of the electrocardiogram on the following lines. The excitation is conducted from the auricles to the ventricles by the auriculo-ventricular bundle, and until it reaches the terminal branches of this conducting system the ventricular musculature does not contract, nor is the galvanometer affected. The QRS group, which initiates the ventricular curve, is variable in form on account of the anatomical structure of the heart. The presence of Q indicates that excitation is communicated to the ventricular wall at a point lying near the apex of the left ventricle, while the absence of Q means that opposite points are first reached by the stimulus. Summit Q is thus referred to relative negativity of the distal electrode. Einthoven draws an analogy between the electrocardiogram recorded from points on the external surface of the body and the curve which would be yielded by a band of muscle lying symmetrically in the body and in which excitation arises at one or other point within the muscle band.

Mines (26) adopts a comparatively simple view of the electrical variation in the ventricle. The ventricular complex begins, according to this observer, with a sharp deflection indicating negativity of the basal electrode, which shows that the base of the ventricle becomes excited before the apex. The excitation spreads to the apex, and when this is uninjured the whole ventricle again becomes isoelectric; since the second phase follows the first with great rapidity, owing to the rapid conduction of the excited

state through the muscle, the result is the characteristic "spike" of Burdon-Sanderson, "first ventricular wave" of Waller, or R wave of Einthoven. This wave is the electrical expression of the asymmetric orientation of the excited state in the muscle with regard to the two electrodes at the beginning of ventricular excitation. The final wave in the ventricular complex is due to the fact that the excited state does not, as a rule, pass off simultaneously with regard to the two electrodes. It is the electrical expression of the asymmetric orientation of the excited state in the muscle when the excitation is passing away. No account is taken by this author of the wave which was shown by Einthoven to precede the spike or R wave, and designated Q, nor of S which follows the spike. Only the waves of greatest constancy are discussed by Mines.

FIG. 1.—Electrocardiogram of frog heart spontaneously beating. Electrodes on right and left lateral margins of exposed ventricle. Time in half-seconds.

It is evident that an explanation, to be adequate, must take into account all the summits which may be present in the electrocardiogram. The initial position of the downward deflection Q is one of the most puzzling features of the ventricular curve, and it will be necessary to devote some attention to the question of its causation in our endeavour to determine the form of the electrical disturbance accompanying systole.

Fig. 1 is the curve obtained from the spontaneously beating heart of a pithed frog. The pericardium is slit open. A sheet of thin rubber is passed under the heart, and the non-polarisable electrodes are applied to the right and left lateral borders or margins of the ventricle respectively. The curve is essentially of the same form as the human electrocardiogram usually figured by derivation from the two hands. The auricular deflection P is followed after a horizontal stretch by the initial ventricular complex, which consists of a downward Q, upward R, and downward S. The

intermediate horizontal stretch runs a little above the zero line, and the curve is terminated by an upward T.

Fig. 2 represents a spontaneous beat from an isolated tortoise ventricle with the electrodes arranged as before. It differs from the previous figure

FIG. 2.—Isolated tortoise ventricle. Spontaneous beat. Induction shock at x. Electrodes on lateral margins.

FIG. 3.—Tortoise ventricle in situ. Spontaneous beats. Electrodes at base and apex of exposed ventricle.

in that the initial complex has the summits inverted. The curve has been recorded one hour after preparation and after the ventricle has been stimulated at intervals by induction shocks. The curve is interrupted by the effect of an induction shock which was thrown in immediately before T.

Fig. 3 is recorded from a tortoise heart beating spontaneously in situ. The right-hand electrode is applied to the middle point of the base of the ventricle and the left-hand electrode to the apex. An almost invisible

deflection has been lettered P in the curve, but it is probable that auricular action is here evidenced by the small upward deflection immediately preceding the initial ventricular complex.

FIG. 4.—Tortoise ventricle in situ. Spontaneous beats. Electrodes at base and apex of exposed ventricle.

FIG. 5.—Electrocardiogram of sinus and auricles from isolated tortoise heart. Electrodes on sinus and apex of ventricle. Spontaneous beat. Time in half-seconds.

Fig. 4 is from another tortoise heart with the same arrangement of electrodes.

These results obtained from the cold-blooded heart are in accordance with what has been found for the human heart. Einthoven (25) points out that he has published curves in which it is difficult to distinguish in the complex which summits should be taken as Q, R, or S.

From the trend of recent work on the cold-blooded heart it seems fairly certain that the electrical variations produced on activity by the different chambers of the heart are of similar nature, and that the principles of interpretation will be alike.

FIG. 6.—Isolated tortoise heart. Electrodes on auricles and apex of ventricle. Spontaneous beat. Time in half-seconds.

FIG. 7.—Isolated tortoise heart. Electrodes on auricles and apex of ventricle. Spontaneous beat.

Fig. 5 is the record obtained from the isolated heart of a tortoise where the electrodes are applied, the right-hand electrode to the sinus and the left-hand electrode to the apex of the ventricle. The sensitiveness of the instrument has been so regulated that the deflections corresponding to the sinus and auricles are distinctly shown on the curve.

Figs. 6 and 7 are from isolated tortoise hearts where the electrodes are applied to the auricles and apex of the ventricle respectively.

Fig. 8 is recorded from the sinus and right auricle alone, the other parts of the tortoise heart having been cut away.

It is clear that from each chamber of the heart we have a complex variation, the interpretation of which is rendered difficult by our uncertainty as to the point of primary activity in the wall of the chamber. The conditions are simplified if we select one chamber—the ventricle—and stimulate it artificially when in a quiescent state, varying the point of stimulation and the position of the leads and recording the electrical responses. The stimulation may be electrical or mechanical. The former has the disadvantage that the stimulating current affects the galvanometer circuit and

FIG. 8.—Electrocardiogram of isolated sinus and right auricle of tortoise heart. Spontaneous beat. Time in half-seconds.

thus complicates the curve. A touch with a glass point forms an efficient mechanical stimulation. I have chiefly made use of the tortoise ventricle cut away from the rest of the heart and preserved for some time in Ringer's fluid, so as to allow of quiescence and complete recovery from the effects of preparation. The special conducting system along which excitation is transmitted from the auricles impinges on the ventricle at the auriculo-ventricular ring, and may be regarded as branching and spreading in different directions from that position, and so conducting the excitation along definite channels to different parts of the ventricular wall. In view of this, I have preferred to stimulate the ventricle at some point other than that at which the excitation normally reaches the ventricle, so that theories of the electrocardiogram depending upon transmission of excitation along the normal channels need not arise at this stage.

The isolated ventricles have been kept in Ringer's fluid for varying lengths of time. The electrical response to stimulation remains of the

same type even although a considerable period has elapsed after preparation. The deflections, however, become slower and of less strength as the interval is increased.

Fig. 9 is the curve obtained from the isolated ventricle of a tortoise which has been preserved for twenty-four hours in Ringer's fluid. The right-hand or proximal electrode is applied to the middle of the dorsal surface, the left-hand or distal electrode to the left margin of the ventricle. Mechanical stimulation is given by a touch with a glass point at the right margin. The curve begins with a downward Q, followed by an upward R, and concludes with an upward T. Following the accepted method of interpreting such a curve, we would say that the downward deflection Q indicates that the left-hand electrode, i.e. the one farther from the point of

FIG. 9.—Isolated tortoise ventricle. Electrodes on middle of dorsal surface and left margin. Mechanical stimulation at right margin.

stimulation becomes negative before the other, and that consequently the tissue at the left margin of the ventricle becomes active before the middle of the ventricle, when excitation spreads from the right margin.

The following records would have the same significance. Fig. 10 gives the response from the same ventricle one hour after preparation. The right-hand electrode is applied to the middle point of the ventral surface, the left-hand electrode to the left margin, and the ventricle is stimulated mechanically at the right margin.

Fig. 11 is from the isolated ventricle of a tortoise after twenty-four hours' preservation. The right-hand electrode is applied on the ventral surface a little to the left of the middle point, and the left-hand electrode on the left margin. The ventricle is stimulated mechanically at the right margin.

Fig. 12 is from an isolated tortoise ventricle after one-half hour's preservation. The right-hand electrode is at the middle point of the ventral surface,

the left-hand electrode at the right margin. Mechanical stimulation is given at the left margin.

Fig. 13 is recorded from the same ventricle immediately after fig. 12. The position of the electrodes is altered, so that now the right-hand electrode remains as before on the middle of the ventral surface, but the left-hand electrode has been transferred to the left margin, and stimulation is given at the right margin.

FIG. 10.—Isolated tortoise ventricle. Electrodes on middle of ventral surface and left margin. Mechanical stimulation at right margin.

FIG. 11.—Isolated tortoise ventricle, after twenty-four hours' preservation in Ringer's fluid. Electrodes to left of middle of ventral surface and on left margin. Mechanical stimulation at right margin.

FIG. 12.—Isolated tortoise ventricle. Electrodes at middle of ventral surface and right margin. Mechanical stimulation at left margin.

FIG. 13.—Isolated tortoise ventricle. Electrodes at middle of ventral surface and left margin. Mechanical stimulation at right margin.

Fig. 14 is the response of an isolated tortoise ventricle one-half hour after preparation. The right-hand electrode is applied to the middle of the dorsal surface, the left-hand one is placed near the left margin. The right margin is stimulated mechanically.

Fig. 15 is the curve obtained from an isolated tortoise ventricle one-half hour after preparation. The electrodes are applied, the right-hand one on the right side of the base, the left-hand one on the apex, and stimulation is given at the auriculo-ventricular ring, i.e. close to the right-hand electrode by induction shocks. The make and break shocks both affect the

galvanometer, and this is evident in the curve. The heart is responding to the break shock, and the variation begins with a small downward Q. This is inconspicuous, but any possibility of its being due to polarisation caused by the stimulating current is excluded by observation of the form of the

FIG. 14.—Isolated tortoise ventricle. Electrodes at middle of dorsal surface and near left margin. Mechanical stimulation at right margin.

FIG. 15.—Isolated tortoise ventricle. Electrodes at right side of base and at apex. Electrical stimulation at auriculo-ventricular ring. Induction shocks at x.

disturbance produced in the galvanometer circuit in cases where the heart has failed to respond to the induction shock.

When the method of derivation employed by Clement (15) and Garten is employed, differences of potential are shown which are developed in a small stretch of the musculature. The clays of two non-polarisable electrodes are united by a single wick which rests on the surface of the heart by its central part, which is bent to an acute angle. The length of the stretch thus led off from will depend on the acuteness of the angle of

the wick, i.e. on the amount of contact between it and the heart. Clement has shown that the direction of the electrical wave can be determined by observing the plane relative to the heart in which the wick is placed, and indeed when a single "differential" electrode is employed the results are quite comparable to those given by two electrodes placed as close together as possible.

Fig. 16 is the record obtained from the isolated heart of a tortoise immediately after preparation. The heart is beating spontaneously, but the curve figured is the response to a mechanical stimulation at the apex. A differential electrode is applied to the middle of the base as though the right-hand electrode were at the base and the left-hand one very slightly nearer the apex. The arrangement is therefore the same as that figured

FIG. 16.—Isolated tortoise heart. Differential electrode at base. Mechanical stimulation at apex.

by Clement in Table VII. fig. 17 (upper curve) of his paper. Mechanical stimulation is, however, employed instead of electrical. This frees the record from the doubt which Clement felt as to what is due to the response and what to the stimulating current.

It is seen that the curve begins with a small upward deflection which would indicate that the point farther from the apex, that is to say, from the part stimulated, becomes negative first. Clement does not recognise such a preliminary upward deflection in the curve figured by him from the frog heart, but although it is complicated by polarisation effects, it is possible, I think, to make out that this initial deflection is present in his curve.

Fig. 17 is the curve obtained from the same heart on mechanical stimulation at the apex when the differential electrode lies close to the apex. It corresponds to the lower curve in Clement's fig. 17 and resembles it. It is not possible to make out a sharp preliminary upward deflection in this case, although stimulation is followed by a slow elevation

of the curve, but the initial complex is not monophasic. It agrees with Clement's curve in exhibiting an upward deflection following upon the sharp downward deflection.

Clement attributes this upward deflection to polarisation caused by the stimulating current, and concludes that the curve is purely monophasic. In view, however, of the presence of the deflection on mechanical stimu-

FIG. 17.—Isolated tortoise heart. Differential electrode at apex. Mechanical stimulation at apex.

FIG. 18.—Isolated frog ventricle. Differential electrode at left margin. Mechanical stimulation at right margin.

FIG. 19.—Same ventricle as in fig. 18. Differential electrode reversed.

lation, this explanation is not adequate. There is present here in fact an upward S.

Fig. 18 is the response of an isolated frog ventricle one and a half hours after preparation. The differential electrode is applied on the ventral surface close to the left margin of the ventricle, and mechanical stimulation is given at the right margin. The derivation is as though the right-hand electrode were at the left margin and the left-hand electrode slightly nearer the middle of the ventricle.

Fig. 19 is the curve obtained from the same ventricle, mechanically stimulated at the right margin, when the electrode is placed on the dorsal surface close to the left margin, as though the left-hand electrode were on the left margin and the right-hand one slightly nearer the middle.

In both these figures there is an initial deflection which would indicate primary negativity of the electrode farther from the point of stimulation.

It may be of advantage in recording the response of cardiac tissue to artificial stimulation, instead of using the complete chamber, to employ strips of muscle. As previously quoted, Bethe reports having seen the contraction begin under such circumstances at the farther end from the point stimulated. Fig. 20, *a*, is the curve obtained from a strip of auricular muscle of a tortoise after twenty-four hours' preservation. In order to show the preliminary deflection clearly the sensitiveness of the instrument has been increased to such a degree that the amplitude of the deflections is too great to permit of the whole curve being recorded on the paper, but the

FIG. 20.—(a) Strip of auricular muscle. Twenty-four hours after preparation. Electrical stimulation at one end. Electrodes near point of stimulation and at distal end. (b) Record with less sensitive instrument.

form of the curve, as shown by records with a less sensitive instrument, is given in fig. 20, *b*.

The strip of muscle is stimulated at one end by an induction shock, whose effect on the galvanometer is seen at *x*. It deflects the string in a downward direction. The non-polarisable electrodes are applied, the right-hand or proximal one near the stimulated end, the left-hand one on the distal end. The electrical response begins with a downward deflection *Q*, quite distinct from the disturbance caused by the stimulating current, and which would indicate negativity of the distal electrode.

As we have seen, Einthoven compares the conditions under which the electrocardiogram is recorded to those present in a strip of muscle where the leads are in relation to the two ends and the excitation arises at some point situated asymmetrically between the leads. Eiger and others also support the view that excitation begins in the ventricular wall at some

point between base and apex. It may therefore be of interest to consider the form of the curve from the tortoise ventricle when artificial stimulation is applied between the leading off electrodes. It would appear in such a case that the initial deflection of the curve should represent a change taking place in the tissue under the lead which is nearer the point of stimulation.

Fig. 21 is a curve recorded from the isolated quiescent ventricle of a tortoise two hours after preparation. The right-hand electrode is applied to the left margin of the ventricle, the left-hand electrode to the right margin. The stimulating electrodes, which are non-polarisable, are placed on the dorsal surface half-way between the mid-point and the right-hand electrode. Break induction shocks are given. The curve begins with a

FIG. 21.—Isolated tortoise ventricle. Electrodes at left and right lateral margins. Electrical stimulation near left margin. Induction shock at *x*.

downward *Q*, which would indicate negativity of the electrode farther from the stimulated point.

Fig. 22 is a curve obtained from an isolated tortoise ventricle where the arrangement of the experiment is the same as in the preceding figure, except that the stimulating electrodes—metal—are applied half-way between the mid-point and the left-hand electrode. The upward *Q* would have the same significance as the downward *Q* in the previous experiment.

Fig. 23, *a*, *b*, and *c*, are curves obtained from another ventricle where the leads are applied, the right hand at the right margin and the left hand at the left margin, and mechanical stimulation is given by a touch with a glass point half-way between the mid-point of the ventral surface and the left margin. An upward *Q* is present, as in the previous experiment.

Samojloff (27) has published curves from the frog ventricle, quiescent after the first Stannius ligature, where the leading-off electrodes are placed on base and apex, and where the heart is stimulated by induction shocks

applied to the base and apex alternately. If these curves are carefully examined it may be made out, although indistinctly, that the results are in accordance with those I have described. Basal stimulation yields a curve whose initial small deflection downwards would indicate negativity of the

FIG. 22.—Isolated tortoise ventricle. Electrodes at margins. Stimulation near right margin. Induction shocks at *x*.

FIG. 23 (*a*, *b*, and *c*).—Isolated tortoise ventricle. Electrodes at margins. Mechanical stimulation near left margin.

farther electrode, while a small initial upward deflection, visible in some of the responses to the apical stimulation, would have the same meaning.

The following records were obtained from spontaneously beating hearts :—

A frog's heart was isolated and the right-hand electrode placed on the middle of the ventral surface, and the left-hand one on the right margin. Fig. 24, *a*, is the variation corresponding to a spontaneous beat. The left-hand electrode was transferred to the left margin and curve 24, *b*, recorded.

The left-hand electrode was then placed at the base and curve 24, *c*, recorded. Finally, the left-hand electrode was applied to the apex and curve 24, *d*, gives the variation. These curves are all similar in form. They agree in com-

FIG. 24.—Isolated frog heart. Electrodes as follows: (*a*) middle of ventral surface and right margin; (*b*) middle of ventral surface and left margin; (*c*) middle of ventral surface and base; (*d*) middle of ventral surface and apex. Spontaneous beats.

encing with a downward deflection Q. This is distinct in *a*, *b*, and *c*, very minute in *d*, but in this curve the beginning of the R ascent is slowed. A downward S is visible in *a* and *d*. The form of the curves would seem to signify that the lateral margins and base and apex are each primarily negative to the middle of the ventricle when the heart is beating spontaneously.

The ventricle of the tortoise heart spontaneously beating was also explored by placing a differential electrode successively on the lateral margins and base and apex.

FIG. 25.—Isolated tortoise heart. Spontaneous beat. Differential electrode at base of ventricle.

FIG. 26.—Isolated tortoise heart. Spontaneous beat. Differential electrode at apex of ventricle.

Fig. 25 is the curve obtained when the electrode is applied to the middle of the base of an isolated heart, as though the right-hand electrode were on the base and the left-hand one slightly nearer the apex on the ventral surface. Fig. 26 gives the curve when the differential is transferred to the apex of the same ventricle and applied—left hand on apex, right hand nearer base.

Fig. 27 is from another tortoise heart beating spontaneously in situ, with basal application of the differential as though right hand on base and left hand nearer apex.

FIG. 27.—Tortoise heart in situ. Spontaneous beat. Differential electrode at base of ventricle.

FIG. 28.—Tortoise heart in situ. Spontaneous beat. Differential electrode at right margin of ventricle.

FIG. 29.—Tortoise heart in situ. Spontaneous beat. Differential electrode between right margin and middle of ventral surface of ventricle.

FIG. 30.—Tortoise heart in situ. Spontaneous beat. Differential electrode on left margin of ventricle.

Fig. 28 is from the same heart, with the differential applied to the right margin of the ventricle as though right hand at margin, left hand nearer middle.

In fig. 29 the differential is applied in the same way but moved a little nearer the middle of the ventral surface. In fig. 30 the differential is

applied to the left margin of the ventricle as though left hand on margin and right hand nearer middle.

Fig. 31 is from another tortoise heart, with the same arrangement as in fig. 30.

The initial deflection of all these curves would indicate from its direction that the electrode farther from the middle point of the ventricle surface becomes negative before the other, and this may be compared with what we have previously described, viz.: that the electrode farther from the point of artificial stimulation becomes primarily negative.

The law which has been generally accepted is that every functional change in a tissue is associated with the production of differences of electrical potential, and that in a muscle any part that is in a state of

FIG. 31.—Tortoise heart in situ. Spontaneous beat. Differential electrode on left margin of ventricle.

functional activity is galvanometrically negative to any other part of the muscle which is at rest. When a muscle is stimulated at one point an electrical disturbance is produced which travels along the muscle from the stimulated point at the same rate as the mechanical process of contraction. After a latent period, corresponding to conduction of the wave of electrical change to the leading-off electrode nearer the point of stimulation, the tissue under this electrode is involved in a change of potential such that a current would pass in the galvanometer circuit from the distal to the proximal electrode. That is to say, the proximal becomes primarily negative to the distal electrode, and this would hold good in the heart as well as in a voluntary muscle.

It is difficult to reconcile this simple conception with the results described in this paper. The curves would seem to indicate from the direction of their initial deflection that the distal electrode becomes negative before the proximal on artificial stimulation, and that in the

spontaneously beating ventricle negativity begins at the margins and spreads inwards towards the centre.

Various explanations have been offered for the presence of the initial deflection. Einthoven, who first described it in the human heart and termed it Q, takes the view that it indicates primary activity of the tissue under the distal electrode, the excitation being conducted from the auricles to the neighbourhood of this electrode in a manner which does not affect the galvanometer, through the subendocardial conducting system of specialised muscle present in the mammalian heart.

The explanation offered by Eiger (14) is on similar lines. He considers that the occurrences in the ventricle are quite analogous to those described by Cybulski (28) and Hoffmann (29), and Piper and Lee, in a cross-striped muscle such as the frog's gastrocnemius stimulated through its motor nerve.

These authors have shown that by suitable leads one obtains from a nerve-muscle preparation not only the usual strong deflection to one side but frequently also a preceding very small deflection in the opposite direction, which indicates that a current first predominates in the reverse direction. This is due to the fact that the motor nerve enters the muscle at the junction of the upper and middle thirds, and that the condition of excitation travels from the point of entry of the nerve simultaneously in two directions, to the upper and to the lower tendon. In the heart the excitation conducted from the auricles to the ventricle by the His-Tawara system does not pass over immediately to the base but to a point between base and apex, and there results a simultaneous spread of the current in two chief directions—to the base and to the apex of the ventricle. Summit Q therefore signifies, according to Eiger, that in the first moment of excitation, this condition spreading from the ventricular terminations of the conducting bundle to the base predominates over the effects of the excitation spreading towards the apex. Soon afterwards the current towards the apex predominates, and not only compensates the other current but produces the deflection R in the opposite direction.

Nicolai (11) takes the view that the normal electrocardiogram cannot be the expression of excitation passing only in one direction, and points to the fact that the negative deflection produced by artificial stimulation may appear double or treble as large as that accompanying a spontaneous beat. This proves that a single unidirectional wave of excitation does not pass over the heart but that normally several waves pass simultaneously, whose electrical equivalents partly counteract each other.

Meek and Eyster (13) in their work on the tortoise ventricle state that they assume the potential of the tissue under the electrodes to be indicated by the first clear-cut quick movement above or below the abscissa. In certain cases they observed small preliminary waves either in the same or in the opposite direction to the main movement. These they disregarded in interpreting the curves, and attributed their presence to the

possibility that, if a distant point becomes negative before either of the areas under the electrodes, the electrode nearer the point of negativity may show a slight change in potential, since the tissue of the heart acts as an indifferent conductor. This evidently amounts to the view that the initial deflection is due to a change of potential affecting the proximal electrode, but not dependent on processes occurring in the tissue under it.

Boruttau (16) assumes that the initial ventricular complex—that is to say, the summits R and S—forms the electrical expression of activity in the specialised conducting system, and that it is only in the horizontal line following on the initial complex, and in the final summit T, that the activity of the general ventricular musculature comes to light.

It is difficult to see how interpretations such as that put forward by Eiger of the summit Q are adapted to meet the case where the ventricle is stimulated at one lateral margin. The assumption would seem to be necessary that excitation passes by a special conducting system from the point stimulated towards the centre of the ventricle, and that then the waves of electrical change which affect the galvanometer spread out from the central region towards each margin of the ventricle—that in fact the electrical change which is recorded does not originate at the point stimulated, but at some other central point to which the excitation is conducted in a manner which is not galvanometrically perceptible.

We have so far assumed that the curves must be interpreted on the accepted lines, viz. that the difference of potential arising between two electrodes placed on muscle which becomes active is due to the involvement of the tissue under one electrode in a negative change before the other; but it is evident that the difference of potential which arises between the electrodes might be due to a positive change propagated through the tissue and reaching one electrode before the other. If such an assumption is made, then in cases where artificial stimulation has been employed the curves in this paper would indicate that the electrode nearer to the point of stimulation is involved in a propagated change before the distal, which is in itself not improbable. We cannot therefore entirely exclude the possibility that the first effect of the propagated electrical disturbance is to render the proximal electrode positive to the distal on account of its earlier involvement in a wave of “positivity” as evidenced by the preliminary deflection Q. In other words, the question presents itself as to whether Q is to be attributed to an electrical change dependent on a process occurring at the proximal or at the distal electrode.

Fig. 32 is the curve from an isolated tortoise ventricle preserved for some hours after preparation in Ringer's fluid. The leading-off electrodes are applied—the right-hand or proximal electrode at the right lateral margin of the ventricle, and the left-hand or distal electrode at the left margin. Non-polarisable stimulating electrodes are placed on the dorsal surface half way between the mid-point of the ventricle surface and the right-hand electrode, i.e. at the junction of the right and middle quarters of

the transverse diameter. Their wicks are separated by about $1\frac{1}{2}$ mm. The induction shocks are minimal. The make shock is short-circuited and the heart is responding to the break shock. The shocks affect the string and

FIG. 32.—Isolated tortoise ventricle. Electrodes on lateral margins. Electrical stimulation near right-hand electrode. Induction shock at x .

FIG. 33.—Isolated tortoise ventricle. Electrodes on lateral margins. Electrical stimulation near middle of dorsal surface. Induction shock at x .

mark the point of stimulation accurately. It is not possible with the rate of drum employed to measure the latent period of the downward Q in this response. It appears to follow immediately on stimulation. When the stimulating electrodes are moved some millimetres farther from the right-hand electrode towards the middle of the dorsal surface, curve 33 is obtained, in which Q has quite obviously a longer latent period.

Fig. 34 is a record obtained from the same ventricle $1\frac{1}{2}$ hours later. The right- and left-hand leads are placed as before on the right and left margins, and stimulation is given by metal electrodes applied near the middle of the dorsal surface. The break induction shock affects the string, and the

FIG. 34.—Isolated tortoise ventricle. Electrodes on lateral margins. Electrical stimulation near middle of dorsal surface. Induction shock at *x*. Alternating responses.

form of disturbance produced by it is shown at *x*, where an induction shock has been applied to which the ventricle did not respond. It will be seen that the responses of the ventricle to the successive induction shocks alternate. Curves *a* and *c* begin with a downward Q, followed by an upward deflection. The curve then falls slightly in *a*, runs almost horizontally for a space well above the zero line, and finally falls gradually to the zero line. Curve *c* is similar, but falls more continuously after the

first upward deflection than does *a*. But for the initial downward peak *Q* we should term these responses monophasic variations. The alternate responses *b* and *d* are of a form commonly obtained from the tortoise ventricle, and show a downward *Q*, upward *R*, horizontal stretch near the zero line, and final downward *T*. In curves *b* and *d* the downward *Q* is smaller than in *a* and *c*.

There can be little doubt, on examination of these curves, that we are dealing in *b* and *d* with the summation of two similar but oppositely directed variations, presumably the summed expression of the electrical disturbance accompanying activity at the right- and left-hand electrodes respectively; while in *a* and *c* one variation is absent or greatly weakened, and we have the electrical expression of the unbalanced activity of the tissue under one electrode. The deflection, however, is not monophasic. If curve *a* is subtracted from curve *b*, the ordinate difference will represent

FIG. 35.—Diagram of ordinate difference between alternating responses in fig. 34. The curve *c* gives ordinate difference.

the form of the electrical disturbance at the other electrode as shown diagrammatically in fig. 35, and it is seen to be of the same type (30). To explain the alternating character of the responses in fig. 34 we may assume that the stimulus given near the middle point of the ventricular surface affects in the one case only the tissue at the right side of the ventricle, and in the other case both sides. Functional dissociation has by lapse of time or repeated stimulation, or for some other reason, presumably set in between the two areas.

Light is thrown on the question as to which electrode is on active tissue when the responses *a* and *c* are recorded, by the results of injuring the ventricle, when the so-called monophasic response is obtained.

Samojloff (27) has published curves from the frog's heart where the electrodes are placed, the right-hand one at the base and the left-hand one at the apex, and has shown that the alteration which takes place when the apex is injured is an elevation of the intermediate part of the curve, and when the injury is complete the response assumes the form of a pure upward deflection—Samojloff's type A. Injury at the base produces a

pure downward deflection, but the author states that this final form practically never appears, because the injury at the base cannot be sufficiently carried out and the curves obtained always show an initial upward deflection (31).

Similar results have been obtained by Seemann (24) when apex or base of the frog's heart is damaged by burning.

Boruttau (16) has investigated the responses from the spontaneously beating ventricles of the frog and tortoise, when the middle of the ventricular surface is injured by burning or by the action of potassium, and one electrode is placed on the injured point, the other on a point nearer base or apex respectively. This observer has obtained monophasic curves similar to those figured by Samojloff. The reproductions in his paper are rather

FIG. 36.—Tortoise heart in situ. Spontaneous beat. Electrodes on base and apex of ventricle. Base injured by cutting.

indistinct, and it is difficult to say whether a preliminary deflection in the opposite direction to the main change is present or not. The author states that owing to the structure and course of the heart fibres it is rare to have pure cross-section derivation, but that usually the curve is complicated by the action current of fibres which remain uninjured, and that these give rise to preliminary and final deflections.

Fig. 36 gives the form of curve I have obtained from the spontaneously beating tortoise ventricle when the right-hand electrode is placed on the base of the ventricle, which has been extensively injured by cutting, and the other on the apex. It consists of an upward deflection followed by a downward deflection indicating unbalanced activity under the left-hand or apical electrode.

It may be observed incidentally that this curve does not regain the zero line immediately after the main change, but that there is a tail or wake succeeding it.

It is clear from the results yielded by injury that we must attribute the responses *a* and *c* of fig. 34 to unbalanced activity under the right-hand electrode, and if so, then it would appear that the preliminary deflections in these curves should also be attributed to this electrode, and that we have here an indication of the complete electrical disturbance in an active element of the heart tissue, viz. positive followed by negative change.

It may be of interest to attempt to explain the electrocardiogram on the assumption that this is true, and to see how far there would require to be modification of the views at present held. Instead of the form of curve representing the change under one electrode given by Bayliss and

FIG. 37.—Diagram representing at—*a*, suggested form of electrical change at a point in the cardiac musculature; *b* to *g*, curves obtained by superposition of *a* and a similar oppositely directed variation, differing from *a* in its commencement by varying time intervals.

Starling, Samojloff, and others, we would assume that it is represented by such a curve as is diagrammatically shown in fig. 37, *a*. If this is taken to represent the form of change under the right-hand electrode, then a similar but oppositely directed curve will give the variation under the other electrode, and a summation of the two differing by a very small interval of time in their commencement should yield a curve containing the summits which are present in the electrocardiogram. We may disregard the final T complex for the present. Curves *b* to *g* are produced by superposing the two variations and plotting the ordinate difference. Only the beginning of the curve up to the horizontal stretch, i.e. the QRS complex, is figured in each case. The right-hand variation leads in *b* by a time interval which becomes progressively less in *c*, *d*, and *e*; while the left-hand variation leads in *f* and *g*. In curves *b* to *e* we have downward Q and S and upward R, while in *f* and *g* the peaks

are oppositely directed. There are in addition interruptions or notches evident in the curves corresponding to those frequently found in the electrocardiogram.

Fig. 38 gives a series of initial complexes obtained from the tortoise ventricle, which is isolated and quiescent, in the following way:—

The right-hand or proximal electrode is placed on the ventral surface about 2 mm. from the right lateral margin, the left-hand electrode is placed on the left margin. The ventricle is stimulated mechanically by a touch with a glass point at the right margin, and response *a* recorded. The right-hand electrode is then moved about 2 mm. to the left—that is, towards the other electrode—and curve *b* recorded; and the subsequent curves are obtained by moving the right-hand electrode successively nearer the other by the same distance, until in *i* the electrodes are about 1 mm. apart. A downward Q is present in each curve. Except that R is not followed by S, this series is quite comparable to fig. 37, *b* to *e*, and would represent, like it, superposition of the right- and left-hand variations or phases where the right-hand phase precedes the left-hand one by a time interval which becomes progressively less. It is not difficult to understand how slight differences in the form of the summed variations as well as in their time relations can determine whether summit S will be evident in a particular curve or not.

It is worthy of attention in this series, that summit Q is a less distinct deflection when the proximal electrode is placed close to the point of stimulation than when it is removed further from it. In the former case the initial downward deflection runs a shorter course and is sooner interrupted by the upward R than in the latter.

With regard to the T complex of the electrocardiogram, the reason for its appearance seems to be fairly well understood. As was shown by Bayliss and Starling, it is the natural result of the asynchronous subsidence of the negative electrical change at the two electrodes. This is also Einthoven's view, and it has been abundantly confirmed by the work of Samojloff, Piper, Mines, Boruttau, and other observers.

As might be expected, T is a very variable phenomenon. In fig. 39, *a*, where the electrodes are placed about 5 mm. apart at the left margin of the ventricle, summit T is downward on mechanical stimulation. A slight approximation of the right-hand electrode to the left suffices to produce inversion of T (fig. 39, *b*).

Fig. 40 gives a good example of how T is inverted by stimulating the base of the tortoise ventricle with minimal induction shocks, whose effect on the string appears at *x*.

It will be observed that the form of curve which we have assumed to represent the response of an element of the cardiac tissue is not unlike that recorded from the retina on momentary illumination (32). Like the retinal response, it is susceptible to interpretation on the lines that two or more processes are evoked almost simultaneously whose electrical expressions

FIG. 38.—Series of electrocardiograms, initial group, from isolated quiescent tortoise ventricle. Left-hand electrode on left lateral margin. Right-hand electrode near right margin in *a* and successively 2 mm. nearer left margin in *b* to *i*. Mechanical stimulation at right margin.

interfere. The initial downward Q would indicate, on this view, that there is a process evoked tending to depress the curve with a shorter latent period than another process which tends to elevate the curve.

a

b

FIG. 39.—Isolated quiescent tortoise ventricle. Mechanical stimulation. *a*, electrodes 5 mm. apart at left margin. Summit T downward. *b*, electrodes 3 mm. apart. Summit T upward.

Several authors have published views as to the different nature of the initial and final deflections of the electrocardiogram.

Hoffmann (33, 34) regards the R group as due to the passage of a stimulation or excitation process through the heart muscle, preceding ventricular systole, a view which is in accordance with that of Brücke that the peaks R and T express two different metabolic processes.

According to Straub (22), summit T is the expression of an anabolic or restitution process following upon the contraction process.

Seemann (24) takes the view that the initial deflection and the terminal deflection or "Nachschwankung" (and by this term the author means

FIG. 40.—Isolated tortoise ventricle. Electrodes at base and apex. Electrical stimulation at base. T is upward in the earlier curves, downward in the later. Induction shocks at x.

all the curve after summit R) are actually different things. The initial deflection is an expression of conduction of excitation; it corresponds to the action current of cross-striped muscle. The terminal deflection is a concomitant of the contraction process. One cannot thus speak of the electrocardiogram as of a diphasic action current in the usual sense. The final deflection does not correspond, as Straub believes, to Fick's second

muscle process, since in Seemann's curves the mechanical contraction outlasts the electrocardiogram. While the terminal deflection must be regarded as at least a concomitant of the contraction process, the two need not necessarily always occur parallel with each other. This author does not consider that the terminal deflection indicates an anabolic process.

Samojloff (27) expresses himself rather ambiguously as to the summit T, which he regards as composed of two components, one not directly dependent on the spread of excitation.

Cybulski (35) rejects the generally accepted method of interpretation introduced by Hermann as inadequate to explain the form of the electrocardiogram, and regards it as the expression of the opposed processes of anabolism and katabolism.

Einthoven (25) inclines to the view that summit T can be most simply explained by assuming that it is brought about by the same general causes which produce the QRS group, and that the special changes which it undergoes and in which the QRS group does not take part can be caused by changes in conduction of excitation.

Rothberger and Winterberg (36), from the results of nerve stimulation, conclude that the QRS group and the T group of deflections are obviously of different origin.

Boruttau (16) considers that the peaks of the T group can be explained without exception as superposition or interference phenomena; they are produced by the longer-lasting excitation of the right or left halves of the heart, the base or the apex.

Leontowitsch (37) believes summit R and perhaps Q and S to be actual excitation currents of the heart, and summit T to be a very important but subsidiary phenomenon in the electrocardiogram.

Eiger (14) regards the peaks Q and R as representing a precontractual action current, while T is an entirely different phenomenon and requires a special explanation. After the precontractual process a new phenomenon appears which is intimately associated with the processes of anabolism and katabolism in the living heart tissue. There now arise potential differences caused by the chemical changes accompanying the mechanical action. The horizontal stretch in the curve corresponds to the systole and in part to the commencing diastole, and indicates that during that time there is compensation of the potential differences produced by the chemical processes taking place then. The author terms the period from S to the end of T the "biochemical" period, and regards deflection upwards according to the usual orientation of the curve as indicating that the balance has been disturbed in favour of katabolism, and conversely that when anabolism is predominant the curve falls. In support of this view he points out that a curve showing a downward deflection during this period can be inverted by the action on the heart of a narcotic, which inhibits anabolism. The author cites also the action of the vagus nerve, whose stimulation favours the anabolic process, and states that the curves then show increased down-

ward deflection. Cooling and warming favour anabolism with downward deflection and katabolism with upward deflection respectively.

This conception that processes of anabolism and katabolism taking place in the heart can be identified by means of the deflection of the galvanometer, and distinguished by the direction of such deflections, when two electrodes are placed at different points on the surface of the heart, is open to the criticism that a galvanometer deflection merely gives us information as to the differences of potential existing at the time between the two points from which we lead off. It is on this obvious ground that Bayliss and Starling and Einthoven have referred summit T and its direction to the longer-lasting activity near one or other electrode. As Mines (26) puts it: "If the final variation depends on some process in the muscle other than that responsible for the initial variation, it must be a process which takes place in one region of the musculature and not in another, or at different times in different parts, or to a greater degree in one region than in another. Apart from its orientation, no process taking place in the muscle generally can be thought of as yielding a difference of potential between two distant regions of the muscle."

In the present state of our knowledge such a criticism is unanswerable, and it is necessary, before discussion of anabolic and katabolic processes can be introduced, that a further step should be taken, and some evidence obtained as to the actual electrical disturbance taking place at one point of the tissue. A method such as Clement's might be expected to assist us in this respect; but as a matter of fact the curve derived from a "differential" electrode is of similar form to that obtained from the heart by the application of two electrodes, and what we actually record by such a method of leading off is the difference of potential between two closely adjacent almost coincident points.

If weight can be attached, however, to the evidence from systolic alternation brought forward in this paper, and Q can be referred to the same electrode as is affected in the production of R, then it may be possible to conclude that the form of the curve gives evidence of two opposite processes taking place almost simultaneously in an element of the cardiac musculature.

Since anabolism and katabolism must undoubtedly occur in living active tissue, the suggestion that they have opposite electrical expressions and underlie the components of the curve cannot be altogether ruled out of court.

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