The Oliver-Sharpey Lectures

ON

THE ELECTRICAL ACTION OF THE HUMAN HEART.

Delivered before the Royal College of Physicians of London on April 8th and 10th, 1913,

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LECTURE I.

Delivered on April 8th.

MR. PRESIDENT,—I may not expend too many of the minutes of my too short hour upon matters other than of strict business. But my strict business has this moment been interrupted by a gift that I value exceedingly, that of a portrait of Sharpey in his prime, side by side with his life-long friend, Allen Thomson. I am very grateful to Allen Thomson's son, Professor John Millar Thomson, for this gift, which recalls to my mind early memories of kindly advice given in 1874 by a wise master to a raw boy, and of still earlier table talk about the days when another Augustus Waller used to consult William Sharpey about the emigration of leucocytes and the action of the vago-sympathetic and the degeneration of nerves. And now that I see before me other pupils of William Sharpey—yourself, Sir, and George Oliver, the founder of these commemorative lectures—a teacher of us all in matters relating to the blood pressure in health and disease—I am again reminded that time passes but too quickly, and that it is a great honour for the raw boy of 1874 to follow in the footsteps of Sharpey and of Oliver.

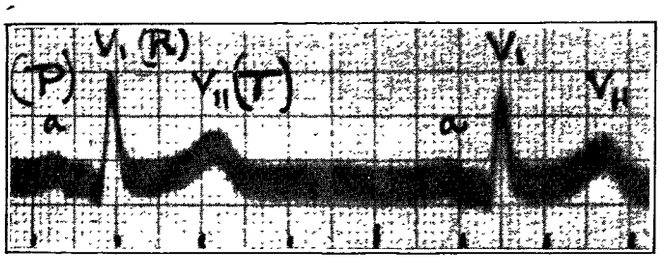
DEMONSTRATION OF THE CARDINAL FACTS.

The vertical spot of light upon the transparent screen in front of you is reflected from a minute mirror fixed to a stirrup of fine platinum wire in a powerful electro-magnetic field, and when a current of electricity traverses the wire—up one limb, down the other—the mirror and the reflected beam of light are deflected to your right and left. The apparatus is, in fact, a galvanometer or—if a condenser of large capacity be placed in circuit—an electrometer in one of its latest and most convenient forms. It is called an oscillograph.

Now that I dip my two hands into these two jars of saline which are connected with the two poles of the oscillograph, you see the spot of light sharply deflected in a rhythm which is obviously that of the pulse. Looking closely you see that each movement appears to be double; the two movements being what I designate as the first and second ventricular peaks, V_1 and V_{11}. The first and quickest which appears on the screen as a flash of the moving light I usually refer to as the systolic spike.

The two chief features (and one other—viz., a presystolic

FIG. 1.

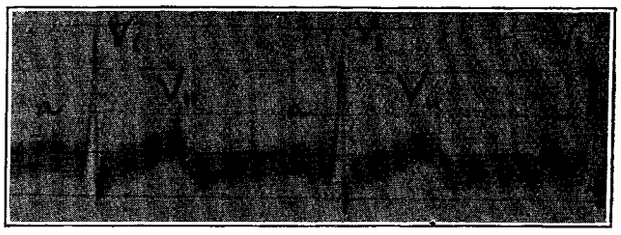


Electrocardiogram of a normal person recorded by a string galvanometer. a = auricular or presystolic (= P). V_1 = first ventricular or the "systolic spike" (= R). V_{11} = second ventricular (= T). Time in one-fifth second.

wavelet due to the auricular contraction) are fixed and made quite obvious by means of photography, the technique of which need not be considered. As a result we have on the developed record an electrocardiogram presenting three chief features.

I do not think it necessary or convenient to follow the rubric P, Q, R, S, T, and U, introduced by Einthoven. But as it is a customary rubric I have given in Fig. 1 the comparison between the two systems. Naturally I prefer my own rubric as being simpler and closer to the facts. I have used it for the last 20 years, and shall continue to do

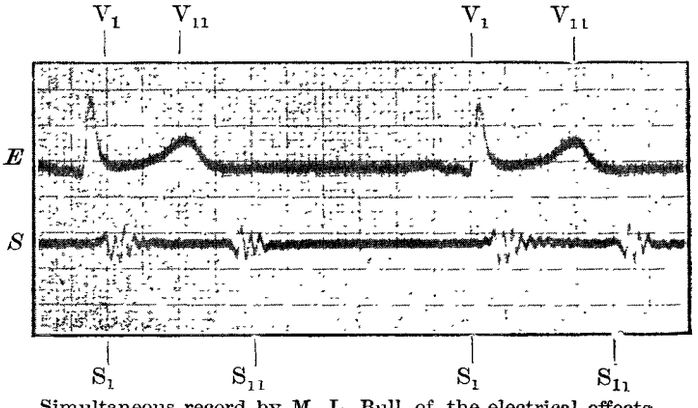
FIG. 2.



Electrocardiogram of a normal person recorded by an oscillograph.

so, partly because I find a difficulty in instantly recalling the application of the several letters of the other and newer rubric, partly because I hold that the simpler rubric keeps closer to the facts of the heart's cycle. The best justification of this simple nomenclature—first and second ventricular—is, however, to be found in simultaneous records of the sounds, where obviously first and second ventricular peaks of

FIG. 3.



Simultaneous record by M. L. Bull of the electrical effects E and of the sounds S associated with the beat of the human heart. The first and second sounds immediately succeed the first and second ventricular peaks. Time in one-fifth second.

the electrocardiogram correspond with first and second sounds of the phonogram. (Fig. 3.)

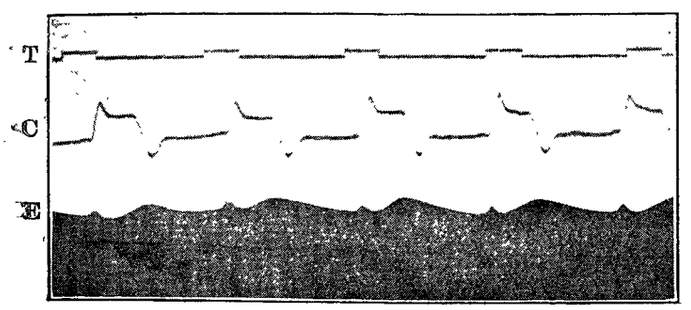
And this primitive record (Fig. 4), taken in 1887, of the movements of the capillary electrometer recorded simultaneously with the "apex-beat" cardiogram shows the same thing, in spite of the fact that the electrical effects, by reason of reversed pole connexions, were recorded downwards instead of upwards, as has now become the usual custom. With the more sluggish electrometer the auricular wavelet is not shown, but only the first and second ventricular waves, which are very nearly synchronous with the beginning and end of the ventricular systole. And with this instrument the first ventricular wave, which is very rapid, is comparatively small. But with a more rapidly acting instrument, such as a string galvanometer, it is tall as well as of short duration. We shall find it convenient to refer to this first ventricular wave as the systolic spike, and we shall learn to recognise this spike as a landmark in otherwise complicated records.

The electrical pulsations now visible to you on the screen are, of course, not the effect of any electrical currents applied to the body. They are caused by the contracting heart. And the fear that I have heard expressed, that the enfeebled heart of a hospital patient might be unable to bear such strong currents is entirely unfounded; nor is there better foundation for the fear lest an enfeebled heart be further exhausted by giving off electricity to the galvanometer. Neither are these pulsations in any degree due to the mere mechanical effect of the arterial pulse. The readiest proof of this—and at the same time a demonstration of the contrast between a favourable lead and an unfavourable lead—is given by the following experiment. Taking one of

the electrodes in my mouth—and for this experiment I must stop talking for a moment, because the movements of my tongue would interfere with the result—and dipping first my left and then my right hand into a vessel serving as the other electrode, pulsation will be clearly visible in the first, but not at all or very slightly visible in the second case. With the mouth and left hand we have a *favourable* or *strong* lead; with the mouth and right hand an *unfavourable* or *weak* lead.

This demonstration of the difference between the right and left sides might be completed by further experiments, showing, firstly, that there is hardly any effect when the right and left foot are led off to the galvanometer—i.e., that the two feet in this connexion are practically iso-electric; and, secondly, that if we lead off either of these two iso-electric parts—i.e., either foot in combination first with the right hand, then with the left hand—a difference is observable between these two cases similar to, if less pronounced than, the difference between the two hands taken

FIG. 4.



Simultaneous record of the apex beat C and of the electrical response E. The cardiogram C was recorded by the shadow of the lever of a Marey tambour. The electrical response is indicated by the shadow of the column of mercury in a Lippmann electrometer; the double movement with each systole is ventricular. T, Time in seconds. From the *Journal of Physiology*, vol. viii., 1887, p. 229.

in combination with the mouth. But taken in combination with either of the two feet it is the *right* hand that is "favourable" and the *left* hand that is "unfavourable," whereas in combination with the mouth the *right* hand was "unfavourable" and the *left* "favourable."

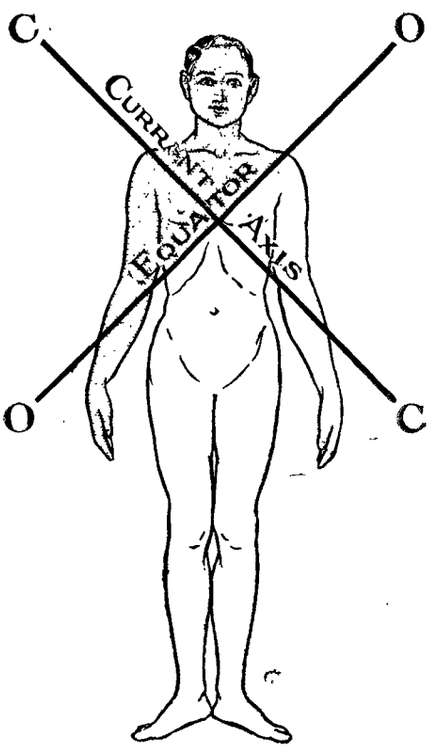
SYSTEM OF LEADS.

In review of all these facts I may now point to the following tabular summary giving the ten possible leads between mouth and extremities taken two by two, and the geometrical figure (Fig. 6) giving the five leads to which for all practical purposes these ten leads are reducible. The designations "right and left superior," "right and left lateral," "axial" and "equatorial" will, no doubt, be understood by the use I shall make of them, and they do not need therefore any formal explanation.

Lead I.*	Right hand and left hand	=	Transverse.
,, II.*	,, ,, ,, ,, foot	=	Axial.
,, III.	Left ,, ,, ,, ,,	=	Left lateral.
,, IV.*	Right ,, ,, right foot	=	Right ,,
,, V.	Left ,, ,, ,, ,,	=	Equatorial.
,, VI.	Right foot ,, left ,,	=	Inferior.
,, VII.*	Mouth and left hand	=	Left superior.
,, VIII.	,, ,, right ,,	=	Right ,,
,, IX.*	,, ,, left foot	=	Left inferior.
,, X.*	,, ,, right ,,	=	Right ,,

In my first investigation of the matter in 1887[1] I felt it necessary to study each and all of these ten leads, and I then divided them into two groups—favourable or *strong* leads, the starred cases in the list; unfavourable or *weak* leads, the unstarred cases. The former were the cases where each of the two led-off points were on opposite sides of the equator, or otherwise expressed, in accordance of direction with the current-axis. (Fig. 5.) I did not fail to observe in

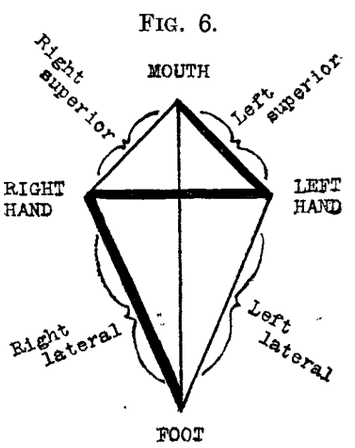
FIG. 5.



the course of that first survey that Lead VI.—i.e., the two feet—is the weakest of all, so much so, indeed, that for all practical purposes they may be regarded as iso-electric; and consequently Leads III. (left lateral) and V. (equatorial) were practically equivalent; also Leads II. (axial) and IV. (right lateral). As regards the four mouth leads, I observed no very marked difference of magnitude in the three strong leads, and for the purpose of further study of the effects on the two sides of the body I therefore confined my attention to the left and right superior—Leads VII. and VIII. of the list.

My system of leads was thus reduced to five—viz., transverse, right and left superior, right and left lateral, represented by the geometrical figure, which explains

FIG. 6.



The five leads—one across, two above, and two below. "Strong" leads are indicated by thick lines, "weak" leads by thin lines.

itself, and which I shall utilise for the further explanation of the calculation of the direction of the cardiac current axis. (Fig. 6.)

And now it must be evident to anyone familiar with Einthoven's three leads that, leaving out of account the two superior leads, the other three leads—transverse, left and right lateral—form a trio practically equivalent to Einthoven's trio—transverse, axial and left lateral. Practically it matters very little which of these two trios we

[1] Waller: On the Electromotive Changes Connected with the Beat of the Mammalian Heart and of the Human Heart in Particular, Philosophical Transactions of the Royal Society, p. 169, 1889.

[2] Einthoven: Ueber die Form des Menschlichen Electrocardiogramms, Pflüger's Archiv, vol. lx., p. 101, 1895. Weiteres über das Electrocardiogramms, Pflüger's Archiv, vol. cxxii., p. 517, 1908. In his latest paper Einthoven has described a method of measuring the cardiac angles based on physical considerations that cannot be discussed here, but must be studied from the paper itself. (W. Einthoven, G. Fahr und A. de Waart, Ueber die Richtung und die manifeste Grösse der Potentialschwankungen im menschlichen Herzen und über den Einfluss der Herzlage auf die Form des Elektrokardiogramms, Pfluger's Archiv, vol. cl., p. 275, 1913.)

may choose for observation. I prefer my own trio, because I have become used to it, and because it affords better and simpler thinking material for picturing to oneself the balance of potentials and the direction of currents on the two sides of the body, and because it leads quite naturally to a very simple formula for the determination of the direction of the current axis through the heart and body.

Except as regards one of the leads—the most important, however, as regards pathological valuation of the electrocardiograms—viz., the left lateral—the harmony of observation and of interpretation between Einthoven and myself has been complete. Obviously transverse is transverse, and right lateral is the equivalent of axial. And even the difference of view between us as regards the left lateral is more apparent than real. It depends on the fact that in 1887 I observed "broad" hearts, whereas in 1908 Einthoven observed "long" hearts, as will presently be explained.

It will have been evident from the foregoing description that the difference between us relates merely to the magnitude of the left as compared with the right lateral. I found it to be relatively small; Einthoven found it to be relatively large. A relatively small left lateral signifies a large angle; a relatively large left lateral signifies a small angle. If the left lateral is equal to the right lateral the angle is 0°—i.e., the current axis is vertical. If the left is larger than the right, as occurs with transposition of the viscera, the angle is to the right instead of to the left of the vertical, as indeed was shown in my first communications in the clearest possible manner.

My three cases of 1887 are happily still alive, and I have quite recently measured their angles with the following results: A. D. W. = 85°, A. M. W. = 51°, and Thomas Goswell = 45°. For Einthoven's two cases of 1908 I calculate the angles as being Fl. = 7°, Ei = 22°.

In 1887 I reckoned the left lateral as an "unfavourable" or "weak" lead. In 1908 Einthoven demurred to this and accounted for my supposed mistake by a supposition of his own into which I need not enter now. I have not altered my mind, or rather I have hardened it; I am again presenting the left lateral as a "weak" lead. I account for Einthoven's criticism by the fact that my first three cases were cases of "broad" heart, whereas Einthoven's two cases were cases of "long" heart.[3]

METHOD OF CALCULATION OF ANGLE.

And now it becomes necessary that I should explain how these angles have been calculated. Taking differences of electrical potential between a mesial point of the body—e.g., the mouth (or a foot) and its right and left sides—i.e., the two hands—we find that the differences on the two sides are unequal. In relation to the mouth, M, the potential difference between M and R is small, between M and L large. As we saw at the beginning of the hour the Right superior is a weak lead, the Left superior a strong lead. In analogy with the mechanical idea of a balance, M is the fulcrum or zero-point of a beam carrying a heavier weight at L than at R, and if we take the angle at M = 90°, then the inclination of the beam when the two arms are equally loaded will be given by the formula: $\tan \alpha = \frac{L-R}{L+R}$, where α is the angle formed with the vertical by a pointer fixed to the beam, R the weight (or the potential difference) on one side, and L the weight (or potential difference) on the other side.

The principle involved is illustrated by this rough model of a rectangular balance. With a weight of 2*d.* on one side and 1*d.* on the other the pointer is deflected through an angle α such that: $\tan \alpha = \frac{2-1}{2+1} = \frac{1}{3}$ $\therefore \alpha = 18°$.

With the weights 3*d.* on one side and 1*d.* on the other: $\tan \alpha = \frac{3-1}{3+1} = \frac{1}{2}$ $\therefore \alpha = 27°$.

Turning to the case of the electrocardiogram of the right and left superior leads, we have observed, e.g., a right superior spike = 0·0001 volt and a left superior spike = 0·0010 volt; the calculation is as follows:—

$$\tan \alpha = \frac{10-1}{10+1} = \frac{9}{11} = 0{\cdot}82 \quad \therefore \alpha = 39°.$$

From which we have concluded that in this particular case the current axis of the heart forms an angle of 39° with the vertical. Weighing the balance by 10*d.* on the left and 1*d.* on the right we see that the pointer indicates 39° on the scale. The balance is in fact an electrocardiac goniometer.

Similar considerations are applicable to the calculation of the angle formed by the current axis from the values of the left lateral and right lateral electrical effects (assuming, of course, that the two feet are iso-electric, and that the systolic spikes culminate synchronously on the two sides). But the angle at F must be taken as being more acute than the angle at M, and I have taken it such that $\cot \frac{1}{2} F = 2$ so as to obtain for the calculation of the inferior angle α the formula: $\tan \alpha = 2\frac{R-L}{R+L}$. In order to secure this relation the angle at F has been taken = 53° and the angle between the two arms of the balance = 127°.

EXAMPLES.

For example, in the case of Thomas Goswell the right lateral spike = 0·0009 volt and the left lateral = 0·0003 volt, so that $\tan \alpha = 2\frac{9-3}{9+3} = \frac{12}{12} = 1$ $\therefore \alpha = 45°$.

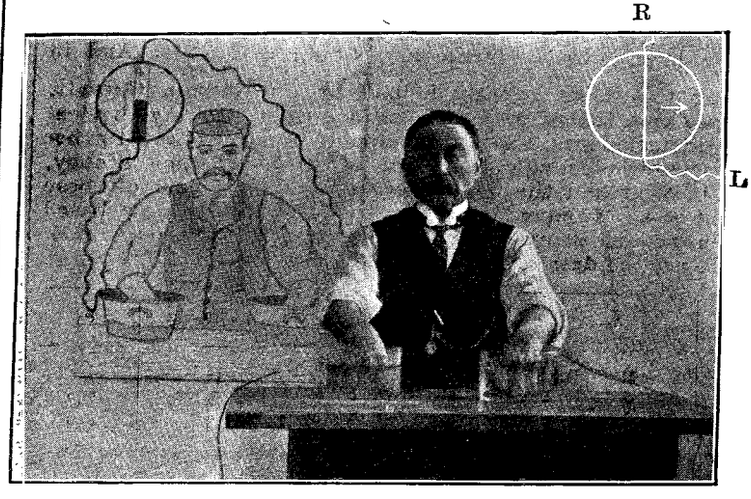
In Einthoven's case of Fl. I measure from the published records the axial spike as 18 mm. and the left lateral as 16, so that $\tan \alpha = 2\frac{18-16}{18+16} = \frac{4}{34} = 0{\cdot}12$ $\therefore \alpha = 7°$.

And, again, in my case of A. D. W. α comes out = 84°, whereas in that of Ei. it is only 22°.

The plotting of these four angles—45° and 84° in my two cases, 7° and 22° in Einthoven's two cases—will serve to make clear what was meant by the remark made a few minutes ago to the effect that my first observations happened to have been made upon "broad" hearts, whereas Einthoven's subsequent observations happened to have been made on "long" hearts. Or instead of the expression "broad" and "long" I might have used the expressions "soft" and "hard." But that would have been to anticipate the considerations illustrated below by Fig. 9.

I am sure you will forgive me if, in connexion with this subject, I take the opportunity of referring to the case of Thomas Goswell in more detail, and of showing a photograph

FIG. 7.



Thomas Goswell sitting with his hands in water connected with the poles of a string galvanometer, in front of a diagram made in 1887 in which he is represented as connected with the poles of a capillary electrometer. In 1887 the angle α formed by the current axis with the vertical was guessed as being 45°. In 1913 the following are the values of the systolic spike in right and left leads. R sup., 0; L sup., 8; R lat., 9; L lat., 3; from which the angle α is calculated as being 45° above and below the heart.

that was taken the other day in the laboratory where I am at present working. Thomas Goswell was my laboratory man at St. Mary's Hospital 25 years ago, and frequently served as my normal subject for purposes of demonstration. He was the "model" from whom my lecture diagrams were made, and from whom the figures were drawn illustrating an "Introductory Address on the Electromotive Properties of the Human Heart," delivered at St. Mary's

[3] Einthoven: Pflüger's Archiv, vol. cxxii., pp. 551–2, 1908. The records of his two cases, "Fl." and "Ei.," are given on pp. 554–5.

Hospital Medical School at the opening of the session 1888-89.[1] In the figures of that time the current axis is represented at an angle of 45°, which was not quite an accidentally chosen value, although I certainly could not have justified the choice by any precise arithmetical calculation. But in Goswell's case "it felt like it," and I chose 45°. A few days ago, Thomas Goswell visited my present laboratory, and had his electrocardiograms taken by the five leads, and his ordinary photograph taken with the diagram of 1887 hanging up as a background. The photograph indicates sufficiently what, indeed, was proved by physical examination of the chest, that Goswell, now 54 years of age, has no heart lesion. The electrocardiogram values were found to be: right lateral = 9, left lateral = 3. The inferior angle calculated from these data is as follows:—

$$\text{Tan } \alpha = 2\,\frac{9-3}{9+3} = \frac{12}{12} = 1 \therefore \alpha = 45°.$$

I cannot find any memoranda of the values of Goswell's leads in 1887, but I remember very well that I estimated his right-hand value at one millivolt and that his left-hand value was much smaller—less than half, but more than a quarter—i.e., that the cardiac angle must have been over 34° and under 50°. The coincidence between the actual result and my recollections and the angle I had guessed at 25 years previously was at the least very remarkable.

The Case of the Eight Young Ladies.

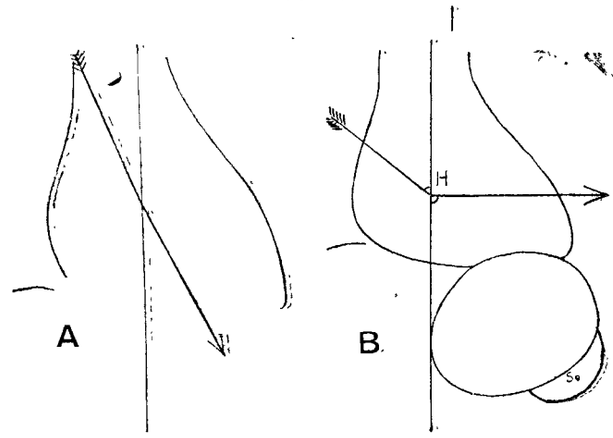
The next case that helped to open my eyes to the importance of paying special regard to the relative values of the right and left-hand spikes, and of calculating from these values the value in degrees of the inclination of the cardiac axis, was that of the eight young ladies. I say—perhaps a little disrespectfully—*the* case, because these eight cases formed one group, which I studied very closely, in fact, almost exclusively during a vacation fortnight undisturbed by the ordinary daily distractions of the laboratory.

During the previous session a young lady at the end of the lecture had asked to take electrocardiograms of herself, and had done so, not only on herself but on seven other young ladies. And I had taken away a memorandum of the right and left hand values in the case, or rather cases of these eight young ladies. The diversity of values was remarkable, and for a time disconcerting in a group of eight presumably healthy subjects, and I made various calculations and suppositions, in the course of which I was led to work out the simple formula for the angle $\tan \alpha = 2\,\frac{R-L}{R+1}$. And in doing this I was greatly helped by Dr. William Garnett, whose kind control and criticism as regards purely physical considerations were indeed invaluable to me. I have further to acknowledge the assistance of Miss Florence Stoney, M.D., who had the goodness to take skiagrams of these eight cases from which suppositions and inferences based upon the electrical signs were controlled and confirmed. Here are the data and the results summarised in tabular form.

No.	—	Right lateral.	Left lateral.	Tan α.	α.
1	M.G.B.	12	4	1	45°
2	M.J.H.	9	− 4	5·2	79°
3	N.L.	9	2	1·27	52°
4	P.W.	8	6	0·28	16°
5	H.S.	13	10	0·26	15°
6	M.N.A.	7	− 7	Inf.	90°
7	H.H.	9	4	0·78	38°
8	M.E.F.	9	3	1	45°

I cannot discuss this table in detail. I refer to it at present simply to illustrate the fact that the current axis varies normally within very wide limits, and that the variation can be due to causes other than age or gross cardiac lesion. It might no doubt be attributed to hypertrophy of the left side, or to dilatation of the right side, or to interruption or excitation of the right or of the left branch of the *a.-v.* bundle. But before we can discuss these possibilities we require to be familiar with what may be called physiological variations and with variations due to trivial causes. The heart muscle can be firm or flabby, hard or soft, long or broad within limits compatible with normal health. The organ as a whole can be displaced by a stomach full of gas, as, indeed, is shown by the skiagram of one of these eight young ladies. If we plot the angles of their current axes we notice that they form three groups that may be designated as vertical, horizontal, and intermediate, and we shall at once ask ourselves what can be the explanation of the contrasted types, vertical and horizontal. I hope to discuss this question more fully in the second lecture. For the present I am content to say that of several possible causes to which the difference can be due I am led to regard the tone of the heart muscle as a dominant factor. In my view of the matter a broad angle is the sign of a soft heart, and a narrow angle the sign of a hard heart, using these expressions in their literal physical sense. A soft or flabby heart, sessile upon the diaphragm; a hard or firm heart, standing upon the diaphragm—detached from its bed, as can be seen upon the fluorescent screen. A broad angle might be due to a flabby heart or to an atheromatous aorta or to a dilated stomach or perhaps to an hypertrophied left ventricle. A narrow angle may be due to a firm heart or perhaps to a dilated right side. And conceivably the heart may be pulled to one side or the other in consequence of a pleurisy or other cause of pulmonary retraction.

FIG. 8.



Outline sketches of the skiagrams of two of the eight young ladies to which have been added arrows to indicate the direction of the superior and inferior current axes, calculated from the observed values of the systolic spike in right hand and left hand leads. In A, the inferior current axis forms with the vertical an angle of 15°, and the muscular tissue is presumably firm. In B, the inferior current-axis forms with the vertical an angle of 90°—i.e., is horizontal; the circle below the heart is the outline of the shadow caused by an accumulation of gas in the cardiac end of the stomach; the muscular tissue in this case is presumably soft, as a consequence of imperfect digestion.

A reversed angle, large or small (I have witnessed both) can occur with complete transposition of the viscera. That, of course, is a rare and quite intelligible state of things, but what is much less rare and not nearly as intelligible is the fact that in certain cases, presumably of normal heart and also of manifestly diseased heart, we may find the relative magnitudes of the right and left leads such as to indicate that the cardiac current axis is inclined to the right instead of to the left. This is a memorandum of five such cases, three of which were presumably "normal."

		R.	L.	Value of α
A. G. W. ...	Normal	16	18	..... −10°
Dr. A.	,, ...	15	16	..... − 4°
G. S.	*a.-v.* Dissociation	20	24	..... −10°
F. F.	Morbus ceruleus	10	8	..... −12°
Dr. G.	Normal	12·5	15	..... −10°

This difference between broad and long, or soft and hard, does not seem to depend on sex or upon age, nor, I may add, upon coarse valvular disease. Cardiac muscle adequate to its mechanical task can be firm, with valves that are very imperfect. Failing cardiac muscle can be flabby, old cardiac muscle can be hard or soft, and with the normal or the premature atheroma of advancing years, inadequate cardiac muscle can be hard. Much further study of these matters is

[1] Brit. Med. Jour., Oct. 6th, 1888.

required; all that I am now urging is that the estimation of the electrical angle from right and left electrocardiograms forms an important element of information.

The Case of the Two Doctors.

The contrast just mentioned between broad heart and long heart, soft heart and hard heart, is particularly well-shown in the next case or pair of cases—that of the two doctors, Dr. D. and Dr. E. I have to thank Dr. J. S. Part, of the National Hospital for Diseases of the Heart, for the skiagrams that give value to this case. Indeed, the skiagrams, together with a memorandum of the electrical data, are all that are required to make the significance of

FIG. 9.

A. Case of Dr. E.; cor breve et molle.

B. Case of Dr. D.; cor longum et durum.

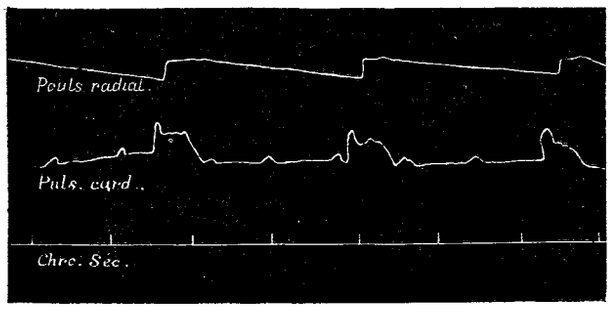
the contrast evident. That is not to say, however, that there is nothing to be discussed in this connexion. There is, but I must pass on.

SOME PATHOLOGICAL CONDITIONS.

I had intended in this first lecture to deal only with the physiological side of the electrocardiogram, reserving for the second lecture pathological considerations and cases. But in this matter, as in many others, it has proved impossible to talk pure physiology without trespassing upon the abnormal, and it will, I am sure, be equally impossible to discuss cases of diseased heart without constant reference to the physiological foundations of our knowledge.

Thus the grouped syndrome, familiar to us all under the designation of Adams-Stokes disease, is to a physiologist, considering the state of the heart, still more familiar as a common experiment of the laboratory. Auriculo-ventricular dissociation commonly occurs in the moribund heart. It was first pointed out in the diseased human heart—not by either Adams[5] in 1827 or by Stokes[6] in 1846, although both

FIG. 10.

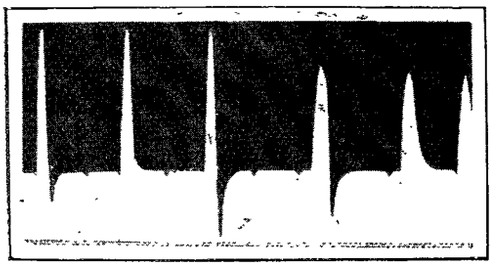


Auriculo-ventricular dissociation. The case of J. B. Damiron. Chauveau, Archiv de Médecine, 1885, p. 161.

of these most acute clinicians clearly recognised the syndrome—slow pulse plus *petit mal*, but by Chauveau in 1884, who described it perfectly and with perfect comprehension of its essential novelty and significance. Indeed, the case of Jean-Baptiste Damiron, as described by Chauveau[7] in 1885 under the title "Dissociation du Rythme Auriculaire et du Rythme Ventriculaire," is in my estimation a model description of a conclusive clinical inquiry.[8] I wish time allowed of the quotation of passages, but as it is we must be satisfied to glance at a reproduction of one of the figures (Fig. 10). You will see at once that it represents precisely what is now so familiar in the electrocardiograms of *a.v.* dissociation or heart-block.

Pulsus bigeminus, pulsus alternans, delirium cordis or muscular fibrillation, asystole of the left auricle, regular and

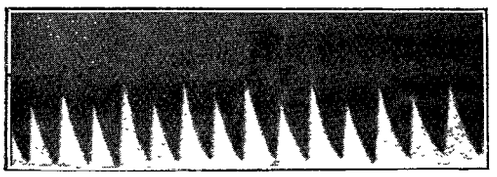
FIG. 11.



Variations accompanying spontaneous beats of puppy's heart. The variations of the auricle are visible as small black teeth on the tracing, and it may further be seen that the variations of the ventricle are in two instances monophasic, indicating negativity of apex, and in three instances diphasic, with first phase = apex negative to base, second phase = base negative to apex.

irregular irregularities, may also be mentioned as common phenomena of the laboratory that have only in quite recent times acquired clinical value and significance by the labours of Wenkebach, Mackenzie, George Gibson, Thomas Lewis, the younger Hering, and many others, following in the main the indications of Einthoven, who was the first to

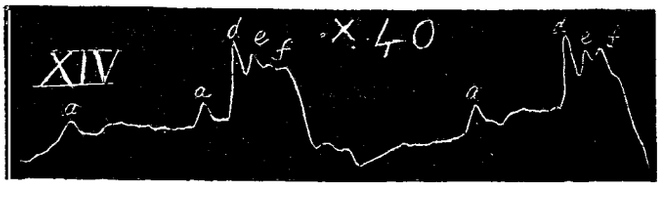
FIG. 12.



Electrocardiogram of a moribund cat's heart exhibiting true "pulsus alternans."

apply his own instrument, the string galvanometer, to the clinical study of cardiac irregularities. Indeed, in this respect the moribund heart in the laboratory has often afforded an epitome, so to say, of phenomena that unfold themselves clinically in the course of months and years. Gaskell's clamp and the immediate production of heart-block, partial or complete, are, of course, familiar to us all in connexion with the various clinical forms of heart-block. Here

FIG. 13.



Galabin's paper, Fig. 14. Guy's Hospital Reports, 1875, p. 261.

[5] Adams: Cases of Diseases of the Heart, accompanied with Pathological Observations, Dublin Hospital Reports, pp. 353-453, 1827.

[6] Stokes: On Slow Pulse, Dublin Quarterly Journal of Medical Science, vol. ii., p. 73, 1846; The Diseases of the Heart and the Aorta, Dublin, 1854, p. 305, et passim.

[7] Chauveau: De la Dissociation du Rythme Auriculaire et du Rythme Ventriculaire, Revue de Médecine, vol. v., p. 161, 1885.

[8] Dr. James Mackenzie has called my attention to a previous record by Mahomed in the Guy's Hospital Reports for 1875 (vol. xx., p. 261). The record is excellent and conclusive, but the description, while correct, is rather tentative than conclusive. The document is buried among several other cardiographic curiosities in correspondence with the title of the communication—"On the Interpretation of Cardiographic Tracings and the Evidence which they afford as to the Causation of Murmurs attendant upon Mitral Stenosis." But the figure itself is an obvious proof that the fact was actually observed. It is reproduced here with the addition of an approximate time-record. (Fig. 13.)

is another and perhaps less familiar example of the sort of rapid representation of clinical features that may sometimes be witnessed on the moribund heart. It is taken from a paper by Waller and Reid,[9] and shows the rapid development of true pulsus alternans ending with ventricular fibrillation in the case of the excised and therefore moribund heart of a cat.

Here is another figure (Fig. 11) taken from the same paper showing the electrical indications of *a.-v.* dissociation, and another (Fig. 12) showing the electrical indications of true alternation of the ventricular beat. But the hour has struck and I must reserve any further consideration of *a.-v.* dissociation for the next lecture.

ALIMENTARY TOXÆMIA:

A SUMMARY AND REPLY.[1]

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IT is obviously impossible to present in a quarter of an hour any adequate summary of a debate in which 60 speakers have taken part, but the speeches of the pathologists, chemists, and bacteriologists have probably interested the society most, for in an inquiry as to what clinical symptoms can with justice be attributed to alimentary toxæmia the first thing is to understand how the poisons are formed and how it is that they affect the body. There has been much said to show that it is not the formation of poisons which chiefly matters. Probably they are continually being formed from the contents of the alimentary canal, or they might even occur as endotoxins derived from dead bacteria, but no harm follows so long as the constantly active defences of the body against such poisons are in full play. But the liver may fail us, the thyroid may fail us, mechanisms in the intestinal wall may fail us, or other unknown defences may fail, and then poisoning symptoms will occur. It will be a great step forward when, with the help of experimental pathologists, we are able to say which cases of supposed alimentary toxæmia are really caused by disease of the liver, disease of thyroid, or disease of some other organ, as the case may be, or which are due to the formation of abnormal poisons or to an excess of normal poisons in the alimentary canal. Then sometimes it may be that the symptoms ascribed to alimentary toxæmia are really due not to the formation of more poison but to the fact that the wall of the bowel being diseased the poison is absorbed more readily than in health, or, again, some alteration in the intestinal contents may render rapid solution and consequent absorption of the poison easy.

Considering next the bacteria in the intestine, it is necessary to point out that if a person is poisoned by absorption from the intestine it does not follow that the poison is produced by bacteria; it may be that it is one of the normal results of the splitting of proteins, but that in all healthy subjects the poison is destroyed by the liver, which, however, is not performing this normal function in the person suffering from poisoning. We only know for certain that definite micro-organisms lead to poisoning when specific micro-organisms introduced from without cause specific diseases—e.g., in the case of typhoid, dysentery, cholera, and Gaertner poisoning; but many of those who have taken part in this discussion strongly suspect that under certain circumstances the micro-organisms constantly present in the alimentary canal lead to poisoning. But there is, I think, a consensus of opinion that the phrase "alimentary toxæmia" should be limited to the absorption of chemical poisons produced in the alimentary tract, and should not include the passage into the blood of bacteria themselves, although this probably occurs more often than is usually thought.

Which micro-organisms originate symptoms that are commonly included under the term alimentary toxæmia we do not know, even in comparatively simple cases. For example, we do not know the micro-organism which is the cause of enterogenous cyanosis; nor do we know whether, if the symptoms are not due to failure of protection against poisons, they are due to excessive number of bacteria, unusual supply of material for their growth, or absence of bacteria which are antagonistic to them; nor do we know whether the bacteria form the poisons from their medium, from their intrinsic metabolism, or from endotoxins liberated at their death. Although it has clearly appeared that we have a considerable knowledge of the bacterial flora of the human alimentary canal, it must be confessed that this discussion has shed very little light on the bacteriology of alimentary toxæmia originating in the gastro-intestinal tract, but we know more about that of pyorrhœa alveolaris, although even here we often only surmise which is the offending organism by the rather unsatisfactory method of the opsonic index. Probably among the intestinal bacteria the bacillus aminophilus intestinalis and its allies will most repay for the research, for much work has been done on them, but it certainly apppears that much of the statements of the French school on the intestinal flora need serious revision.

As the subject of this debate has been alimentary toxæmia it was to be hoped that from it we should learn something definite as to the toxins concerned. Unfortunately we have not. It is well known that the view of the Metchnikoff school is that poisons are manufactured in the intestine, especially the large, by micro-organisms, and these poisons produce the symptoms commonly comprehended in the term alimentary toxæmia, and that indol is one of the most important of these poisons, but although that view has been upheld by some speakers it has been severely criticised. More than one authority on bacteriology thinks it gratuitous to regard indol as the main toxic product of putrefactive organisms, and tells us that the conclusion drawn by Distaso that the flora of constipation is an index of intoxication is entirely unsupported by the evidence he adduces. I must confess it seems to me that the case for indol has not been proved, and that there are serious defects in the work of the French school on the bacteriological and biochemical properties of the intestinal flora. If the debate has done no other good it has helped to make widely known that much of the bacteriological and biochemical work which has been done upon the flora of the large intestine and the poisons found there does not satisfy the standards of scientific accuracy. But there is a general impression that the poisons of alimentary toxæmia may in many instances be comparatively simple chemical bodies derived from the proteins of the food. It is known that the ammonia present in the portal vein is derived from putrefaction in the large intestine. Tyrosine and histidine may be converted into poisonous substances by putrefactive bacteria, and experiments have been quoted which suggest that when certain of these poisons are absorbed arteriosclerotic changes in the vessels and disease of the kidney may follow. Confirmation of these experiments will be looked forward to with great interest. Of the poisons formed in the alimentary canal probably the study of derivatives, of tyrosine, leucine, and histidine will most repay further investigation.

In the clinical part of the discussion the dental surgeons who contributed to it seemed to be on surer ground than other speakers. Disease of the gums leading to periodontal disease is by far the most fruitful source of oral sepsis. Large numbers of people owe ill health to it, and it is often overlooked, and especially we are inclined to forget that mouth-breathing in children leads to persistent marginal gingivitis, and that this disease is more intractable and serious in its results in those who are mouth-breathers, from which it follows that not only must the teeth be treated but often it will be necessary to treat nasal obstruction also. Different speakers have laid a varying amount of stress on the use of autogenous vaccines. Probably the most important part of the treatment is local, and here much judgment is required. Some remove teeth needlessly, some do not remove enough of them, but in the more severe examples the intelligent use of autogenous vaccines may be of distinct help. The question has been raised as to whether in a few cases pyorrhœa alveolaris is not secondary to a toxæmia arising from some parts of the alimentary tract. It is quite possible that this may be so, or, at any rate, that such local pyorrhœa is increased by the fact that the general resistance of the body is lowered by an alimentary toxæmia of other origin, but obviously the question can only be settled by a discussion of each case in

[9] Philosophical Transactions, 1885. The examples referred to in the text are Tracings 1, 2, 3, and 4 of that paper, not here reproduced.

[1] A paper read at a special meeting of Fellows of the Royal Society of Medicine on May 7th, 1913, at the close of a debate on Alimentary Toxæmia.