THE VALUE OF ELECTROCARDIOGRAPHY IN DIAGNOSIS.*

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The use of the Electrocardiograph in diagnosis is that it gives us an additional mode of examination whereby to supplement the clinical methods we were taught as students and are still learning as practitioners. It is its conjunction with clinical findings that gives electrocardiography its peculiar charm. Lewis⁽¹⁾ has written that "Electrocardiography is the last court of appeal and its judgment is practically speaking infallible." Lest he, our greatest authority on electrocardiography, be judged to be unduly favourably inclined, let me quote from the obituary notice of Einthoven in "The Times," surely an impartial critic. "Of his many discoveries and inventions, those connected with the electrocardiogram and the so-called string galvanometer will remain the most famous. The heart has always been an organ of peculiar interest to laymen and to medical men alike. In many directions the diagnosis of its maladies has been improved in recent times, but the greatest single step forward was taken by Einthoven in his application of the string galvanometer to unravelling the mechanism of the electrical phenomena of the heart-beat" That was written in 1927, when in this country the clinical syndrome and characteristic electrocardiograms of coronary thrombosis were hardly known. At that time, too, we had not the portable electrocardiograph.

In the diagnosis of cardiac disease electrocardiography is concerned with disturbances of rhythm (there its value is recognized by all) and in gauging the condition of the myocardium (where recognition of its value is steadily gaining ground). The manifold disturbances of the orderly flow of impulses from sino-auricular node to the ultimate Purkinje fibres can all be followed and differentiated by the electrocardiogram.

In the sino-auricular node we have two varieties of abnormality. **Sinus arrhythmia** is caused by a variation of output of stimuli by the pace-maker, the rate quickening with inspiration and slowing with expiration. This irregularity affects the rhythm of the whole heart and consequently the radial pulse. There is no abrupt "miss" in the pulse, and I have known sinus arrhythmia to be mistaken for auricular fibrillation. In the electrocardiogram (Fig. 1) it is shown by a variation in the T-P interval, the period at which the string is at rest and the entire heart is in diastole. In **sino-auricular block** the transition is not gradual but abrupt (Fig. 2), the entire heart—auricles and ventricles alike—missing a beat. This condition is not common nor does it appear to be of any great pathological significance. It does, however, at times cause faintness and giddiness, and in the most extreme case I have met caused actual loss of consciousness.

The power of stimulus production is inherent to all heart-muscle fibres. Normally latent and not employed, the fibres merely passing on the wave of contraction by virtue of another of their inherent qualities, conductivity, this stimulus-production may break out in abnormal areas and thus a localized area of the auricles may take upon itself the rôle of pace-maker, for the time being usurping the function of the sino-auricular node. If such results in isolated beats we have one of the varieties of **extra-systoles**, if it results in a run of ectopic beats we have one of the varieties of **paroxysmal tachycardia**. In the electrocardiogram of these

* All the electrocardiograms were taken at the standard calibration and have been reduced one-third in the process of reproduction.

Figs. 1, 2 and 3.

1.—Sinus arrhythmia. 2.—Sino-auricular block. The R waves are broadened slurred and notched. 3.—Premature beats originating in an ectopic focus in the auricles. The main wave of contraction in the auricles is upwards and consequently the P waves are inverted. This electrocardiogram shows a blocked ectopic P the auricular systole failing to activate the ventricles, a very rare form of abnormality.

ectopic beats the P wave is always premature, it may be so premature as to be superimposed upon the T wave of the previous cardiac cycle, (Fig. 22) and if the main wave of auricular contraction is upwards, as commonly is the case, the P wave is inverted. (Fig. 3). The ventricular complexes are unaltered for the ventricles receive their impulses from above by the usual channels. Exceptionally the ventricular complexes shew aberration. (Fig. 3).

In the auricles there also arise auricular flutter and auricular fibrillation. These depend upon what Lewis has described as the "circus movement." Instead of the contractile impulse passing through the auricles to the auriculo-ventricular node from above downwards by the shortest route, a circular path is followed round and round the chambers. In auricular flutter the ring of the circus is large and constant, in fibrillation the ring is small and variable, but their essential similarity is shewn by the occurrence of intermediate forms.

Auricular flutter gives a beautiful electrocardiogram with the large peaked diphasic rapid auricular complexes, uniform in outline and usually regular in time, broken only by the R waves of the ventricle and with one peak to each ventricular beat sometimes modified in outline by the corresponding T wave. Their usual rate is 300 per minute, a pace obviously impossible for the ventricles to maintain and consequently a degree of heart-block, usually regular, is almost invariably present. The ordinary rate is 2:1 but it may be 4:1 less commonly 3:1, and the ratio may change. Auricular flutter, which puts a serious strain upon the heart, may easily be overlooked and should always be considered in persistent tachycardia, particularly in patients of middle age or over. An electrocardiogram will settle the matter at once, flutter with its characteristic picture, tachycardia with the curve altered only in shortening of the T-P interval. That is the point which ordinarily with persistent regular pulses of 140 or 150 requires clearing up—whether sinus tachycardia or flutter; for the rate in paroxysmal tachycardia is higher still. There are, however, further although uncommon pitfalls. I have met with patients who shewed signs of cardiac inadequacy but no evidence of auricular flutter. In them a 4:1 auricular flutter with regular ratio demonstrated

by the electrocardiogram (Fig. 4) proved the fallaciousness of the pulse with its normal rate and regular rhythm. Again, by its occurrence along with independent heart-block, auricular flutter may be associated with bradycardia (Fig. 5) while a varying ratio of the ventricular response to the auricular over-stimulation will simulate the pulse of auricular fibrillation.

Figs. 4 and 5.

4.—4 : I auricular flutter. Auricular rate 300, ventricular rate 75, patient's pulse consequently being unaffected by the flutter. 5.—Auricular flutter and heart block. Auricular rate 280, ventricular rate 40. The S waves are broadened slurred and notched.

In auricular fibrillation the ring of the circus is smaller and is variable, but the movements are more rapid, some 500 to the minute. Here the ventricular rate and the pulse rate generally do not correspond (Fig. 6). Far too many impulses reach the ventricles for the latter to respond to all and their arrival is itself irregular. In the electrocardiogram the R (or S) waves are irregular in spacing and also uneven in amplitude. The regular P waves have disappeared

Fig. 6.—Auricular fibrillation and pulse tracing.

and are not replaced by others plain to see like the peaks of flutter. In most records, however, some movements of the string are visible (Fig. 7) representing the auricular contractions, these usually—as in the case of flutter—being best seen in leads II and III. The T waves naturally are irregular in time but they are not otherwise altered by the fibrillation *per se*, although their outline may be obscured in greater or less degree by the waves produced by the fibrillating auricles. Auricular fibrillation nowadays is a commonplace but an electrocardiogram makes its differential diagnosis a bed-rock certainty and yields us further valuable information to which I shall refer later.

Fig. 7.—Auricular fibrillation showing the R-T dip of digitalis control. Fibrillation waves are evident and the ventricular complexes are normal.

In the auriculo-ventricular node and bundle we may have delay in conduction evidenced by lengthening of the P-R interval (Fig. 8), or failure to conduct (**heart-block**), such failure being incomplete or complete, irregular or regular. All these can be demonstrated by electrocardiograms. A partial heart-block (Fig. 9), sino-auricular block, multiple ectopic beats and slow auricular fibrillation are capable of being confused with one another, but if electrocardiography is available one's clinical diagnosis is easily tested by a graphic record which admits of no doubt.

Figs. 8, 9, 10 and 11.

8.—P-R interval 0.28 second instead of the normal 0.16 second. 9.—Partial heart-block every third P wave failing to activate the ventricles. The record also shows intra-ventricular block, the S waves being broadened and notched as in bundle-branch block but there is an iso-electric interval between the S and the T wave. 10.—Auriculo-ventricular nodal rhythm of the slow type, rate 40. Note the absence of auricular complexes. 11.—Complete heart-block. The RS waves show aberration of the intra-ventricular block type.

Here also originate ectopic contractions, Lewis's "junctional extra-systoles." These are the least common of the three varieties of premature beats. The upper and lower chambers are activated simultaneously, or nearly simultaneously, according to the level of the ectopic focus in the node and thus either the auricular P is lost in the ventricular R, or it appears immediately before or after that wave and it is inverted since the auricular stimulation is retrograde. So again massed nodal extrasystoles form the second variety of **paroxysmal tachycardia**, but whether the ectopic beats are merely interjected interruptions of the normal rhythm or occur continuously the ventricles are stimulated from above and their complexes in the electrocardiogram are unaltered in outline.

Further, whether from depression of the sino-auricular node or increased excitability of its own, the auriculo-ventricular node may assume the part of pacemaker (**nodal rhythm**), usually at a rate of about 40 (Fig. 10), and carry on thus over long periods of time without any special symptoms being produced. This is liable to be confused with heart-block and thus, if an electrocardiogram be not taken, an unwarranted bad prognosis may be given. The curve is as in nodal paroxysmal tachycardia so far as the complexes are concerned but at about one quarter its rate. Simple bradycardia, heart-block complete or 2:1 (Fig. 11), and nodal rhythm are clinically similar but are all clearly distinguishable by the electrocardiogram.

We now arrive at the ventricles and here originates the most common form of premature beats, set up by one or more localized foci of ectopic contractile activity in one or other ventricle, or in both.

Here the stimulus for ventricular contraction comes not *via* the normal channels but from the ectopic focus in slow irregular and retrograde fashion through the mass of the myocardium. Weird distorted complexes are thus produced. If the focus is in the left ventricle, in lead I there is a long broad slurred and sometimes notched R wave followed immediately without any iso-electric interval by a large inverted T wave so that the complex is diphasic. In lead III the initial deflection is downwards, an abnormally long and broad S with the ascending limb running into the ascending limb of an equally hypertrophied T. When the focus is in the right ventricle the picture is reversed, lead I becomes as lead III and lead III as

Fig. 12.—Premature beats from an ectopic focus in the right ventricle showing the blocked P waves, and simultaneous tracing from the radial pulse.

lead I. Thus in left ventricular premature beats we have the direction of the complexes that of the left axis deviation,* in right ventricular premature beats that of right axis deviation. In both varieties the outline of the complexes of premature beats occurring in lead II usually corresponds to lead III. (Figs. 12 & 13). Meantime the auricular rhythm is undisturbed, the auricles continue to beat regularly, paying no heed to the eccentricities of the ventricle and their P waves may be identified in the ventricular complexes (Fig. 12) or following them, blocked because they find the ventricular myocardium in contraction or refractory. Finally, when the heart's rate is slow, ventricular ectopic contractions may occur so early as not to interfere with the fundamental cardiac rhythm and so we have the "interpolated extra-systole" (Fig. 13), the only premature contraction which is a true extra beat. Once only have I met with an interpolated extrasystole of auricular origin.

Figs. 13 and 14.

13.—Interpolated extrasystoles, their origin being in the right ventricle. 14.—Paroxysmal tachycardia of ventricular origin.

All varieties of **ectopic beats** are clearly recognized in electrocardiograms. In mitral disease the occurrence of auricular premature contractions may draw our attention to threatened auricular fibrillation which can be postponed or averted by putting the patient at rest. In otherwise healthy subjects the occurrence particularly of auricular or junctional premature beats makes us search for some extrinsic cause (tobacco, gastric or intestinal disorders, septic foci), while with ventricular beats we search particularly for some intrinsic cause in the cardiovascular system which must be treated (hyperpiesia, rheumatic carditis, a patch of myocarditis left by a neglected influenza), while occurring with auricular fibrillation they may cause us to revise our digitalis dosage. Ventricular premature beats rarely open the aortic semilunar valves, hence the missed beat at the wrist (Fig. 12) for although in the electrocardiogram they may give immense complexes dynamically they are poor contractions. Thus in old people, and in them they are common, if the heart's rate is slow and the ectopic beats are numerous, there may be caused unpleasant symptoms of giddiness and faintness. Otherwise premature beats are no more than an annoyance, for any anxiety can be put at rest; their importance lies not in themselves but in what their presence may imply.

In the ventricles arises the third variety of **paroxysmal tachycardia** (Fig. 14), the rarest and much the most serious. I have met with but two examples.

* Throughout this article the old nomenclature is followed.

In the ventricles we have a further interesting and not very rare form of failure in the conduction path, where in one or other of the two main branches of the auriculo-ventricular bundle, right or left to the respective ventricle, conductivity fails (**bundle-branch block**). When this occurs the myocardium of the side affected is activated from the healthy side *via* the intra-ventricular septum. Characteristic electrocardiograms are produced and their essential features, as first enunciated by Carter ⁽²⁾, are that in leads I and III the initial ventricular deflection is over 0.1 sec., equals one-third or more of the total complex in time, is of large amplitude and of opposite sign, shews notching in one or more leads, and is followed by a well-marked T of opposite sign. In right bundle-branch block (Fig. 15) the initial ventricular complex in lead I is a large broad slurred notched R, in lead III a similar S so that in these waves we have the picture of left-ventricular preponderance, Lewis's levogram from the unaffected side, while in left bundle-branch the waves are reversed (Fig. 16) producing a dextogram. The initial and terminal ventricular deflections in leads I and III in both right and left types are in opposite directions and the normal iso-electric interval is absent, consequently the composite ventricular complex is diphasic.

Figs. 15, 16, 18 and 19.

15.—Right bundle-branch block. 16.—Left bundle-branch block. 18.—An extreme example of low voltage of the RS in recent coronary occlusion of T₁ type. The RS waves are hardly visible being a mere millimetre in amplitude. 19.—Initial electrocardiographic changes in a T_{III} coronary occlusion. This is a good example of the "high take-off." Note also the varying length of the P-R interval.

Just as in auriculo-ventricular block the fault in conduction is not of so much importance *per se* as in what it implies, viz., degenerative change within the heart as a whole, so also in bundle-branch block for the electro-cardiogram of the latter is indicative of established organic general myocardial disease. Our nomenclature is at present in a state of flux but up to recently the frequency of the defect on the right side of the heart, in opposition to heart disease generally, has been explained by the difference in anatomical structure and in blood supply of the two branches, the left bundle-branch being more spread-out and having a double blood-supply and therefore presumably being less vulnerable. In private practice I have records of 58 cases of right bundle-branch block, and of only 9 cases of left, and even that disproportion is less than in some published series. I have met with temporary bundle-branch block only in the course of myocardial infarction. Many die within a year and the prognosis is worse than in heart block.

There are other abnormal electrocardiograms which display some of the characters of bundle-branch records but do not fulfil all the requirements of Carter. These will be referred to later. Leaving them meantime out of consideration, we have now followed the path of conduction from the normal pace-maker (the sino-auricular node) through the auricles, the auriculo-ventricular node and bundle, and the two main branches of the latter, into the ventricular myocardium and by the electrocardiogram all disturbances of rate and rhythm are placed before us clearly differentiated from one another.

While the immense help the electrocardiogram has afforded in the diagnosis of disorders of rhythm is universally admitted, much had already been revealed by the painstaking genius of Mackenzie in the years immediately prior to Einthoven's discovery. Apart from pulsus alternans (Fig. 17), however, there is

Fig. 17.—Left bundle-branch block and alternation.

no disorder of rhythm concerning which equal or better information is not derived from the electrocardiograph than from the polygraph. Concerning the myocardium the polygraph is silent, but the diagnostic help of the electrocardiograph in myocardial disease, acute and chronic, is becoming more and more evident as time goes on. We have already seen how a fault in conduction revealed by the electrocardiogram is important not so much in itself as in what it infers, that it is but a special manifestation of a general degeneration, but the electrocardiogram can also give us direct guidance as to the state of the myocardium.

Among the acute myocardial lesions **coronary thrombosis**, a veritable cardiac catastrophe, at present stands out by itself. It is through the electrocardiograph that the clinical picture has been recognized. So well-established indeed, are the symptoms of the attack that there would seem to be arising a tendency to dispense with the electrocardiogram and to make and maintain the diagnosis on clinical signs and symptoms alone. This I submit is a mistake. An attack of coronary occlusion is a very serious matter in a patient's medical history. However successful the outcome of his case, there must in future be modification of his *modus vivendi*, which naturally in degree of severity will depend upon his former calling and habits. Thus a solicitor who earns his living with regular hours in an office is in a very different position when convalescent from a general medical practitioner who cannot earn his without climbing stairs at all hours of the day or night. The diagnosis ought therefore in the patient's interests to be made as secure as is humanly possible. On three occasions now I have been called to a case of

suspected coronary thrombosis and found instead a case of left-sided "spontaneous" pneumo-thorax. That statement is hardly accurate for in one of these cases the diagnosis had already been made and the patient had been under treatment for a fortnight, my co-operation only being sought for advice as to when it would be safe for him to get up. When the pain is referred low down to the xiphisternum or epigastrium and is accompanied by persistent vomiting, it may be mistaken for an acute abdominal affection such as a perforated viscus or biliary colic. An electrocardiogram, however, will settle the matter. I have examined patients reputed to have recovered from coronary thrombosis. In some of them the return to normality in the electrocardiogram has been suspiciously rapid, while in others recovery, as evidenced in the electrocardiogram, has been so speedy as to be impossible. Such cases of course are of infrequent occurrence. When met with there is no difficulty in dealing with them tactfully in such a way that no question of altered diagnosis is raised but the patient is impressed with the completeness of his recovery. Less uncommon are the ambulatory cases in the early weeks of their convalescence. In these patients there has been no initial shock, no "angor animi" which is a common symptom in coronary occlusion, but merely a prolonged attack of mid-sternal or epigastric pain which was attributed to indigestion. The doctor may not have been called in at the time but only later because the patient felt out of sorts generally, had a little palpitation or was pulled up by chest pain on walking. Until questioned he may have forgotten his original prolonged attack of painful "dyspepsia". These patients ought to be put to bed to serve what remains of their two months' term. The changes in the electrocardiogram are so well known that I need only go over them briefly. The special diagnostic point is the distortion upwards or downwards of the RS-T portion of the curve which normally is horizontal. This is always in contra-lateral direction in leads I and III, if upwards in lead I it will be downwards in lead III (Fig. 18) and vice-versa (Fig. 19). This occurs usually within twenty-four hours but if not met with in the first electrocardiogram further records ought to be taken. The lead in which RS-T elevation occurs is the more important to bear in

Figs. 20 and 21.

20.—T_I type of coronary thrombosis in the second stage showing the deeply inverted T wave in lead I and the coronary dome, with similar but less marked changes in lead II.
21.—T_{III} type of coronary thrombosis in the second stage showing the deep inversion of the T wave and also the long Q wave referred to in the text.

mind for in it follows within the next few days the second characteristic change, a sharp inversion of the T wave. Thus the cases fall into two main groups according to whether the inversion of T occurs in lead I (Fig. 20) or lead III (Fig. 21) and so we speak of coronary occlusion of T_1 type or T_{111} type as the case may be. This inverted T is frequently preceded by a convex bulge, the "coronary dome", the combination of the two making the "coronary T of Pardee". A T inverted by coronary occlusion may thus be distinguishable from the type described later. Similar changes but in less degree are seen in lead II (Fig. 20). All my T_{111} cases have shown inversion of T in lead II and about half my T_1 cases. That is the end so far as the infarction is concerned, for the subsequent changes are towards the regaining of normality. If T_{111} has been inverted, the inversion becomes less and less deep until that part of the curve is flattened out and then a positive wave reappears. So next with lead I or lead III as the case may be. Thus in a favourable case three months after the date of the occlusion the electrocardiogram will show a flattened or positive T_{11} with either a shallow inversion of T in lead I or an inversion of T in lead III which is less deep than formerly for it is in lead III that inversion of T is most deep of all. It is important to remember that when a solitary late record is taken a negative T in lead III may be the only abnormality remaining in the electrocardiogram⁽³⁾. Low voltage of the initial ventricular deflections is common especially in the T_1 type (Fig. 18) and these waves may be broadened, slurred or notched. Many of the T_{111} type show an abnormally long Q wave in that lead (Fig. 21) so long as to look like an S when the record is being taken. This Q may be the last relic in serial electrocardiograms. Less commonly

Figs. 22 and 23.

22.—An early coronary thrombosis of T_{111} type. In lead I there is normal rhythm except for the second beat which is premature and originated from an ectopic focus in the auricle. The P wave coincides with the T of the previous cardiac cycle. The rate is 120. Lead II in its entirety shows paroxysmal tachycardia from an ectopic focus in the auricles with rate 195. In lead III there is auricular fibrillation with a ventricular rate of 135. 23.—Lead I, early coronary thrombosis of the T_1 type showing extreme bradycardia there being only two heart beats during the five seconds of the curve. The pace-maker is the auriculo-ventricular node there being no auricular complexes visible in either beat.

a Q wave of sufficient size to merit attention is present in lead I in thrombosis of T₁ type, but such wave never attains the amplitude of that in lead III. As with the T wave lead II may shew a concomitant prominence of the Q wave in smaller degree. This long Q is of sufficient frequency in coronary occlusion and of rarity otherwise to require notice although it is not the essential diagnostic alteration in the curve. All forms of abnormal rhythm may be met with temporarily in the first few days. (Figs. 22 and 23).

Cardiac infarction nearly always occurs in the left ventricle, but as yet the lead in which inversion of T occurs is not diagnostic as to the precise site of the occlusion. Lewis describes electrocardiography as "a means of directly examining the all-essential heart-muscle."⁽⁴⁾ Unlike other organs, there is in the heart no sharp line of demarcation between the neural and the muscular tissue and *a priori* myocardial disease might be assumed likely to interfere with impulse conduction and thereby to be demonstrable in the electrocardiogram. In acute necrosis and chronic fibrosis of the myocardium it is reasonable to suppose that there may be a minimal area below which the record may be normal although the area can be capable of producing pain, and for all we know as yet there may be within the myocardium "silent areas". A clinical diagnosis of coronary thrombosis which is followed by negative electrocardiograms is one, however, which demands very careful reconsideration.

In cardiac infarction there is a small localised but complete failure in a portion of the coronary arterial circulation, in **angina pectoris** on the other hand there is generalized inadequacy of such circulation when increased call is made upon it. Coronary atheroma may be of sufficient degree to cause angina pectoris and yet leave unaltered the heart's borders, sounds and rhythm, the blood pressure may be within normal limits, indeed, a striking feature of angina is that it occurs as a subjective symptom in patients in whom on examination nothing objectively abnormal is found. We ought therefore to be prepared to welcome help from the electrocardiogram. Such we may obtain for the record may be frankly pathological. In contrast to what I have stated when considering coronary thrombosis a normal electrocardiogram must never be permitted to invalidate a clinical diagnosis of angina pectoris. Also there is no special type of electrocardiographic abnormality which is diagnostic of angina pectoris as such. The electrocardiogram may exhibit aberration of the QRS, (the long Q_{1,11} is common), inversion or flattening of the T waves, bundle-branch block or some degree of heart-block. If evidence of myocardial disease is found the clinical diagnosis is strengthened, but a physiological electrocardiogram I repeat must not be permitted to invalidate the clinical diagnosis. Ordinarily then in angina pectoris the electrocardiogram can be no more than helpful, but if we are fortunate enough to examine a patient during an attack of pain the record may be diagnostic. Parkinson and Bedford⁽⁵⁾ recorded their own observations on "Electrocardiographic changes during brief attacks of angina pectoris" and their bearings on the origin of anginal pain. They found that remarkably few examples were on record of electrocardiograms taken during attacks, opportunities of taking such arising but rarely and by chance. They reviewed what previous literature there was, and stressed the importance of all such records being reported so that in course of time a sufficient number would be available from which valuable conclusions might be drawn. They found temporary depression of the R-T and a diminution in the amplitude or inversion of the T waves in one or more leads, changes closely resembling, though not so pronounced as, those which follow cardiac infarction in the early stages. This

they held to be strongly in favour of a common origin (coronary disease), cardiac infarction being caused by occlusion and angina pectoris by spasm of an artery already atheromatous. I published records obtained in two patients by myself⁽⁶⁾ and have met with others since.

From time to time one meets with electrocardiograms resembling those of bundle-branch block but without their symmetry. The initial ventricular complexes are broadened-out, slurred and may be notched (Figs. 2, 5, 9 and 11) but they do not diverge or converge in leads I and III as in bundle-branch block nor are the complexes diphasic. This type has been designated "**intraventricular**" or "**arborization**" block and is believed to be caused by faulty conduction in the bundle-branch short of failure of function therein, or by lesions of the terminal divisions of the conductile fibres. Chronic myocarditis is the common finding *post mortem* and electrocardiograms of this type are to be regarded, in the same way as those of bundle-branch block, as pointing to generalized myocardial fibrosis. The prognosis is worse if later records shew further deviation from the normal as this reveals progressive degeneration.

The last point to be considered about the initial ventricular complexes is what significance can be placed upon a **fall of voltage** below normal. Cassidy and Russell⁽⁷⁾ included as of pathologically low voltage cases which shewed 7 mm. or less between the top of the R wave and the bottom of the S wave. If the tracing shewed only one of these waves its height or depth must be 7 mm. or less. Their main conclusions were that the prognosis in the case of a patient who has symptoms of myocardial degeneration, and also shows a low voltage curve in all leads, should be extremely guarded, as the great majority of such patients live a few months only and that on the whole the lower the voltage the worse the prognosis. Low voltage in all leads occurred in 1.75 per cent. of all electrocardiograms examined. In this respect a clear distinction must be drawn between permanent low voltage, which is the type referred to above, and low voltage which is only temporary, occurring as part of the early electrocardiographic picture of coronary occlusion (Fig. 18) or of acute myocarditis, particularly rheumatic and diphtheritic. In coronary thrombosis low voltage of the QRS is particularly met with in the T₁ type, in my experience in the more severe cases and its persistence is of unfavourable omen. I have noted it appear and disappear in diphtheria⁽⁸⁾ and also in acute pericarditis sometimes accompanied by flattening of the T waves. Electrocardiograms are thus of assistance in pericarditis where the friction often clears up early and we are left with a patient, usually young, who has a normal temperature, normal heart sounds and borders but persistent tachycardia, and in whom there is doubt whether the latter is functional or is dependent upon acute myocarditis otherwise unrecognizable objectively.

Inversion of the T wave may occur apart from any previous coronary thrombosis. Four types of negativity of the deflection, in lead I, in leads I and II, leads II and III, and leads I, II and III are recognised as pathological. Shallow negativity of T in lead III alone is met with, as are bizarre QRS waves, in a proportion of healthy subjects and is not considered pathological. Inversion of T in lead II alone, or in lead I and lead III without lead II does not occur. Statistics based on collections of all four groups have shewn a uniform high mortality, at least half the subjects having died within twelve months. It appears doubtful, however, whether bundle-branch block and coronary thrombosis cases

have been excluded and the time seems ripe for this subject to be revised. Lewis holds that the inverted T wave should not be used for prognostic purposes.⁽⁹⁾ Negativity of the T wave in lead I is particularly associated with left hypertrophy ventricular. The amplitude of T decreases with advancing years. Inversion of T in any of the four types enumerated above is diagnostically significant, or at least suggestive, of myocardial degeneration.

In mitral stenosis the P waves of the electrocardiogram may be of increased amplitude and so of some diagnostic help in a doubtful case of mitral stenosis. Similarly the picture of right axis deviation with high voltage of the RS waves may help in the differential diagnosis between congenital defect and acquired valvular disease. Otherwise the electrocardiogram is not concerned with heart murmurs.

In transposition of the heart all the deflections of lead I, auricular and ventricular alike, are inverted.

In alternation of the heart (Fig. 17) a pulse tracing is more reliable than the electrocardiogram. The latter may not show the abnormality at all and when it does the larger R waves may correspond with either the larger or the smaller radial waves.

In auricular fibrillation electrocardiography is of value apart from the differential diagnosis of the arrhythmia. A digitalized heart shews a dip after the R waves (Fig. 7) similar to that seen early in coronary occlusion. This may occur in all leads but is most common in leads II and III. There should, however, be no possibility of confusion for the deflection in all leads is in the downward direction, there is never the contra-lateral displacement in leads I and III. This R-T dip is more apt to be mistaken for inversion of the T waves and so an unfavourable view of the situation taken. This can be avoided by noting the fact that the dip comes immediately on the fall of the R wave without any iso-electric interval and by measurement when lead I is unaffected. Moreover, the true T wave may be recognizable following the dip. The importance of this R-T dip is that it shews that the heart is under digitalis control. Increase of dosage is liable to be followed by the appearance of ectopic beats and further increase by coupling. I have been asked to see cases of supposed heart-block which were really examples of coupling from digitalis with none of the ectopic beats reaching the wrist. In auricular fibrillation the outlook depends upon the all-important ventricular muscle. Given R waves of normal delicacy and amplitude (Fig. 7) and positive T waves similarly of normal voltage so much the better is the prognosis. On the other hand with lowered voltage, slurring, broadening or notching of the RS or inversion of the T wave the prognosis is unfavourable. Further, this gauging of the state of the myocardium by the electrocardiogram is helpful in assessing the safety of attempting to restore normal rhythm by quinidine.

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