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CLINICAL
ELECTROCARDIOGRAPHY.

THOMAS LEWIS.

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ELECTROCARDIOGRAPHY

BY

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Fourth Edition.

BY THE SAME AUTHOR.

"The Mechanism of the Heart Beat."

"The Mechanism and Graphic Registration
of the Heart Beat."

"Clinical Disorders of the Heart Beat."

"Lectures on the Heart."

"The Soldier's Heart and the Effort Syndrome."

"The Blood Vessels of the Human Skin
and their Responses."

PREFACE.

IN republishing an account of clinical electro-cardiography, I do so from conviction that this method of examination is essential to the modern study of heart disease. When some nineteen years ago I began to study disorders of the heart with the aid of the " string galvanometer " the method was in its early infancy; although it was regarded at that time as full of promise, yet its scope, in helping to perfect our knowledge of heart disease, could not then be foreseen. Electrocardiography has taken us far, having filled great gaps in our knowledge of these maladies; for it is a means of directly examining the all-essential heart muscle.

Cases of structural heart disease are few in which an electrical examination is superfluous, and in a not inconsiderable percentage of cases the records modify, more or less profoundly, our conceptions of the malady that we treat.

Electrocardiography has developed ; it has grown along technical lines ; it has embraced a terminology of its own, the inevitable result of progress in a new direction.

In *Clinical Disorders of the Heart Beat* I have attempted to collect the simple bedside signs of disordered heart action and to narrate their influence upon prognosis and treatment in a manner palatable to pure clinicians. The present chapters supplement that book. They are intended to serve as an introduction to students of electrocardiography and as a guide to practitioners and hospital physicians in understanding curves that may be taken by others from patients in their charge. They describe in outline the most precise method that we possess, when we attempt to identify the many forms of disordered heart action. It frequently happens that other graphic methods fail to analyse these disturbances ; electrocardiography is the last court of appeal, and its judgment is practically speaking

infallible. There are types of heart action upon which other methods are nearly, if not quite, silent; knowledge of these conditions is in the almost exclusive possession of electrocardiography. Finally, the electrical curves have revealed a number of new signs, associated with abnormal distribution of the muscular masses in the heart, with abnormalities in the course taken by the excitation wave in the heart, or with recent injury of the muscle.

In writing of *Clinical Electrocardiography* I confine myself to common varieties of disorder and to electrical signs that are frequently to be observed. I do not submit the evidences for my conclusions in this handbook, seeing that these are for the most part fully set forth in my larger treatise, *The Mechanism and Graphic Registration of the Heart Beat.*

T. L.

May, 1928.

CONTENTS.

Contents.

CHAPTER I.

THE ELECTROCARDIOGRAPHIC METHOD.

That electrical currents are developed in the heart at each contraction of this organ was demonstrated in the middle of the nineteenth century. Modern electrocardiography originated from this discovery. The currents are small, yet modern instruments are sufficiently sensitive to record them with facility. It is unnecessary that the heart should be exposed ; the currents will deflect a suitable galvanometer when the latter is connected to the limbs of the human subject, as Waller first showed. The study of the direction, time relations and magnitude of these currents constitutes modern electrocardiography.

The instrument that is described in the present chapter is known as the " string galvanometer," the invention of Einthoven, a Dutch physiologist. It consists essentially of a powerful electro-magnet, the poles of which are closely approximated (Fig. 1) ; and of an extremely delicate conducting fibre of silvered quartz or glass, stretched in the narrow gap between the two magnetic poles. If minute currents are led through this fibre, as it lies in the magnetic field, the fibre moves across the gap in response to the tested current in a plane at right angles to the lines of magnetic force. The magnified shadow of the fibre is vertical and is

projected by means of an optical system and powerful light
on to the horizontal slit of a camera ; the shadow moves at
right angles to and across the slit and the movements are
photographed upon a sensitive plate that travels behind the
slit. I do not propose to describe in detail the construction
of the galvanometer itself or of the accessory apparatus that
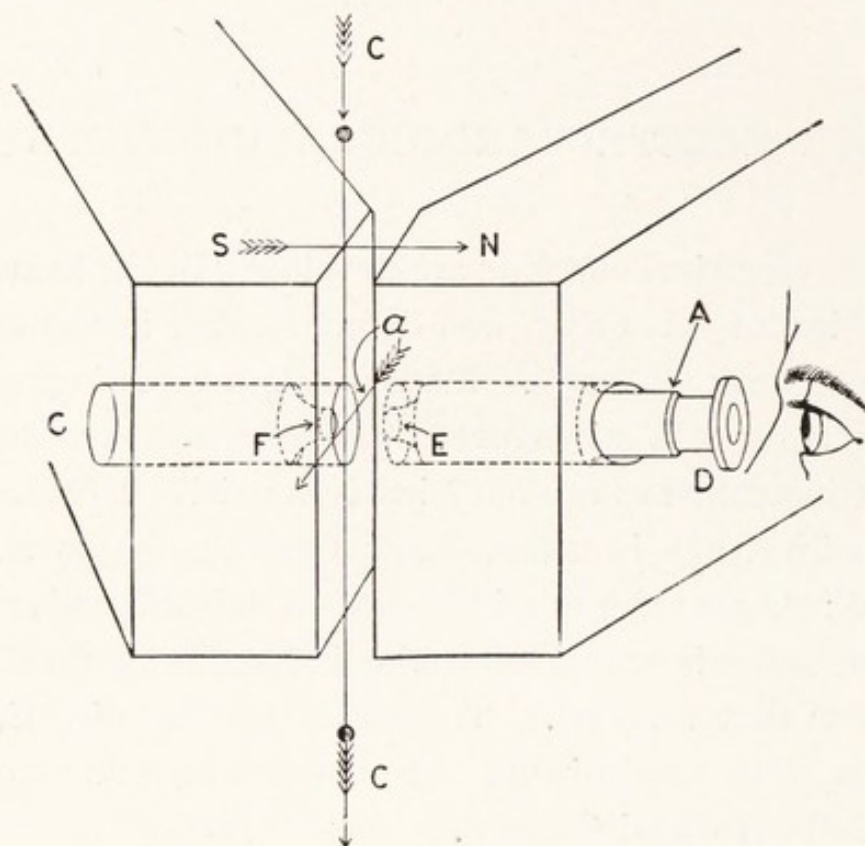
Fig. 1. A diagram illustrating the construction of the string galvanometer.
The poles of the magnet (*N*, *S*) are seen in outline ; the recording fibre
(*CC*) lies vertically between them ; its movements may be observed
through a microscope (*D*). The movements of the string are in a plane
parallel to the faces of the magnetic poles, as indicated by the central
arrow. *F* is a condenser.

is usually employed in obtaining human electrocardiograms.
Many different installations are now sold and some are
specially arranged for clinical purposes. The galvanometer
illustrated in Fig. 2 is perhaps as serviceable as any, and is
to be recommended for the simplicity of its construction and
the ease with which it is kept in order and manipulated. The
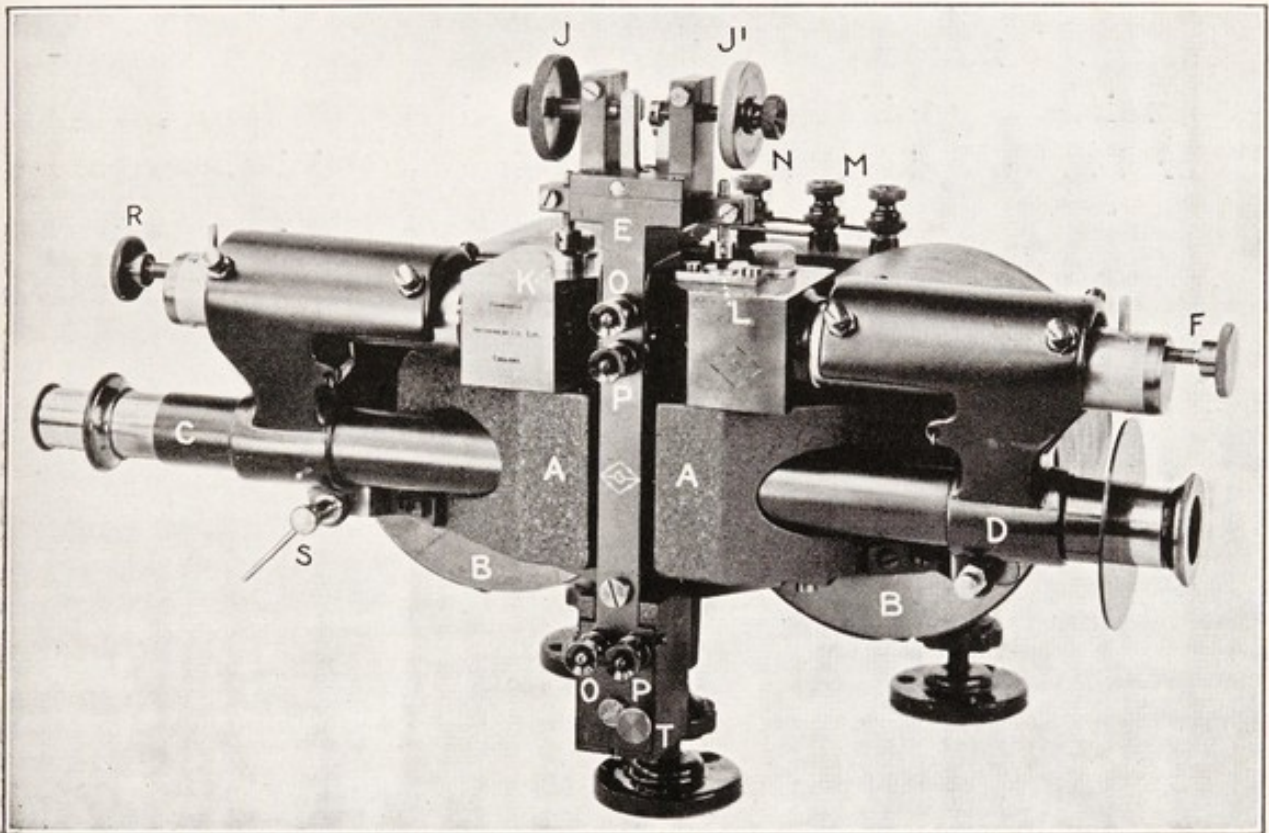
Fig. 2. ($\times \frac{1}{4}$.) A string galvanometer. *A, A* are the poles of the magnet, *B, B* its coils, and *M, N* the terminals carrying current to these. *D* receives and condenses the beam of light upon the string, which lies in the string carrier (*E*); the microscope (*C*) projects the image of the string on to the camera. *R* and *F* are focussing screws. The string carrier is pivoted on knife edges at *K* and *L*. Current is led into the strings through terminals *O* and *P*. The string tension is adjusted by the screws *J, J¹*. Carriers holding one string or two (as here depicted) may be used.

adjustments are simple and consist of mill-headed screws, which level the instrument, centre the fibre in the field, increase or decrease its tension, and focus its shadow upon the camera. Those who desire a more detailed account of the apparatus may refer to the special descriptions issued by its makers, or to my book *The Mechanism and Graphic*

Registration of the Heart Beat; but familiarity with galvano-metric outfits can be gained best by handling the instruments.

The galvanometer is connected to the patient through some special form of switchboard. Students of electro-cardiography utilise switchboards of different patterns, more or less complicated; those supplied for clinical use are
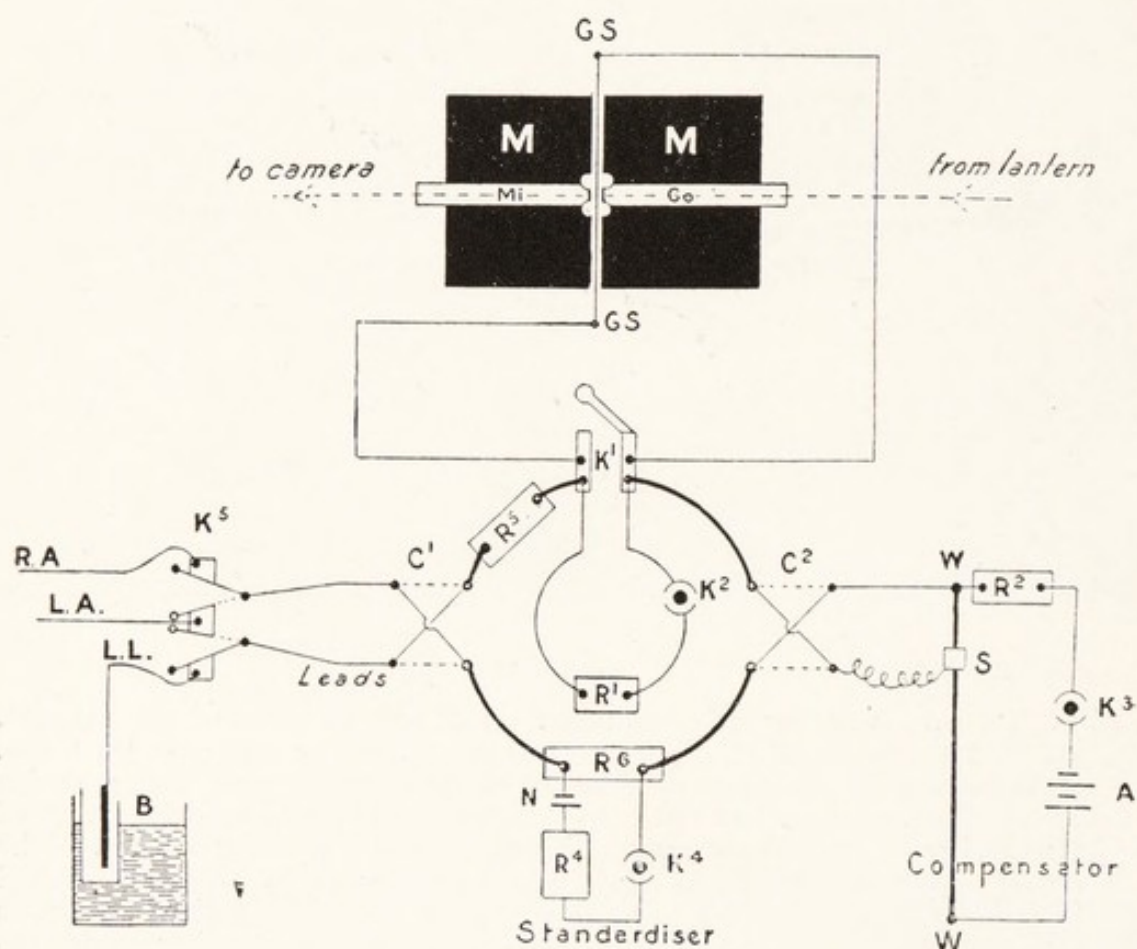
Fig. 3. A switchboard arranged as a group of simple circuits and connected to the galvanometer.

arranged to insure speedy and accurate work; the actual switches are therefore few in number but the wiring is intricate. I propose to describe a scheme that may be drawn as a group of simple circuits. This scheme, though serviceable, is no longer in clinical use, because the number of the keys makes the manipulation of the board relatively slow. But

I retain it as an illustration because its circuits and their use are easy to understand and because more complex boards are arranged on similar principles.

Fig. 3 is a diagram of the apparatus and its connections. The string of the galvanometer (GS) is connected to a key (K^1) which closes the string circuit. The closure of this key brings the string to rest and safeguards it from damage. The same key communicates with two circuits, the inner and outer circles of the diagram; the former is a shunt, containing a resistance and key (R^1 and K^2), so arranged that when closed it carries the greater part of the tested current, thus preserving the string from mishap. It is used when a current of unknown strength is thrown into the string circuit. The outer circle represents the main circuit; it is broken at three points; (1) at K^1 where it joins the string circuit, (2) at C^1, by a commutator, where it connects to the patient, and (3) at C^2, by a commutator, where it connects to the compensator. It contains also a dial resistance (R^5) and the standardiser. The separate parts of the apparatus may be described briefly and in rotation.

Connection of patient. A wire that runs from the commutator C^1 to the key K^5 may be connected at will to the right arm (*R. A.*) or left arm (*L. A.*) by means of the special key; a second or parallel wire may be connected to the left arm or left leg (*L. L.*) by the same key.* The actual contacts are made when the limbs are immersed in salt water,† and through porous pots containing zinc sulphate (Fig. 3 *B* and Fig. 4). The key (K^5) is arranged so that the two main wires may have the following paired connections.

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* The wires connecting to the limbs may be of any length; the patient who is observed may be in a separate building.

† The water should be warm and pieces of cotton wool may be added to form a bath of porridge-like consistency. By these means disturbing movements of hands and water are diminished.

Fig. 4. Photograph of a subject as connected for observation. The two arms and the left leg are used, and curves are taken from the three leads, which are represented by arrows in the figure. The zinc sulphate is placed in the outer vessels of the electrodes shown in this figure.

				Limbs connected.
Lead *I*	..	..	..	*R. A.* and *L. A.*
Lead *II*	..	..	..	*R. A.* and *L. L.*
Lead *III*	..	..	..	*L. A.* and *L. L.*

These are the connections that are employed in routine observation.

The compensating circuit. This is shown to the right in Fig. 3 ; and consists of an accumulator (A), key (K^3), suitable resistance ($R^2 = 19$ ohms) and slide wire ($W\ W = 1$ ohm). It is used to bring the string shadow to zero when the patient is connected to the galvanometer ; it serves to neutralise the current derived from the skin, generally referred to as the " skin current," and is fitted with a commutator, C^2, which reverses the direction of the compensating current.

The standardiser is formed by a similar circuit to that of the compensator, consisting of a battery, suitable resistances (R^4 and R^6) and key (K^4). It throws an E.M.F. of 3 millivolts into the main circuit, and is used to standardise the string excursion, so that all electrocardiograms may be comparable one with another.

The dial resistance R^5 serves many purposes ; amongst others, the measurement of the resistance of the string or of the patient's body.

The method of obtaining standardised electrocardiograms.

Standardised electrocardiograms are obtained in the following manner.

1. With the compensating and standardising circuits open, but with the shunt (K^2) closed and the patient in circuit (K^5 connecting to $R. A.$ and $L. A.$, the first lead), K^1 is opened. The string shadow immediately takes up a new position, as a result of skin current flowing into the galvanometer, and shows minute electrocardiograms (Fig. 5*a*).

2. K^3 is closed and a compensating current, or current to balance the skin current, is introduced for the purpose of bringing the string shadow back to zero (Fig. 5*b*).
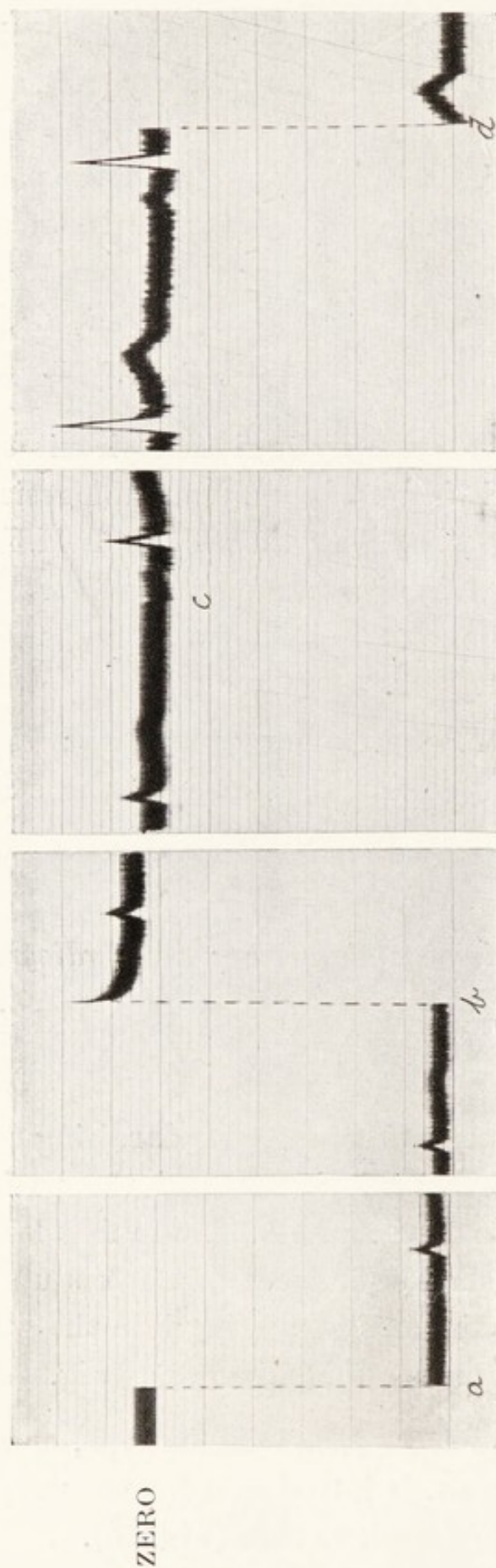
Fig. 5.   Portions of a continuous curve, illustrating the steps taken in obtaining a standardised electro-cardiogram.

This and all other curves used as illustrations read from left to right. The string is at first still. At a the main key (K^1 of Fig. 3) is opened ; the string is deflected in response to " skin current " and shows minute electrocardiograms. This current is compensated and the string brought back to "zero" by throwing a current of opposite direction into circuit (b). At c, the shunt K^2 is removed, the string moves a little from the zero and the electrocardiogram increases in amplitude. The string tension is then adjusted until a potential of 3 millivolts deflects the whole curve through 3 centimetres (d). The curve is then a standard one and the photograph may be taken, while the string is moving in the region of the original zero line.

3. The shunt (K^2) is opened and the string moves again* showing larger electrocardiograms (Fig. 5c), because the whole current is now allowed to flow through the string.

4. If the string shadow has moved much to one side, it is again brought to zero by means of the compensator.

5. The standardising circuit is now made (introducing 3 millivolts) and broken repeatedly until, by adjusting the string tension, the whole curve is deflected through 3 centimetres (Fig. 5d). Tightening the string reduces and slackening it increases the sensitiveness of the instrument.

6. A curve is taken from the first lead.

7. The process is repeated in the case of leads *II* and *III*. The curves obtained have the correct amplitude and a value of 3 millivolts to each 3 centimetres of excursion, or 1 millivolt to the centimetre of excursion.

For convenience of measurement, a screen, of millimetre lines, is placed immediately in front of the photographic plate. The light, falling through the screen, is intercepted by these lines and the film is ruled as the photograph is taken (Fig. 5).

Though the steps taken in producing standardised electrocardiograms may seem complicated and tedious in the description, yet in practice they become simple ; with modern apparatus a series of curves from the three leads should be obtained from a single subject within a few moments.

Checking the accuracy of standardised curves.

The accuracy of an electrocardiogram from a given subject may be checked in a simple fashion by increasing the resistance in the main circuit. When a curve has been

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* In Fig. 5c this movement is very small.

taken in the usual manner, a large resistance is placed in

series with the string and patient by altering the dial resistance (R^5). As a rule it is convenient to throw in a resistance, which is approximately equivalent to that of the string itself.* With this resistance in circuit the same procedure is followed, the tension of the string being suitably decreased, so as to give the original excursion of 3 centimetres to 3 millivolts. The second curve, the one taken with the added resistance in series, should be similar in every respect to the original one. This procedure is recommended as an occasional test; it is not necessary as a routine.
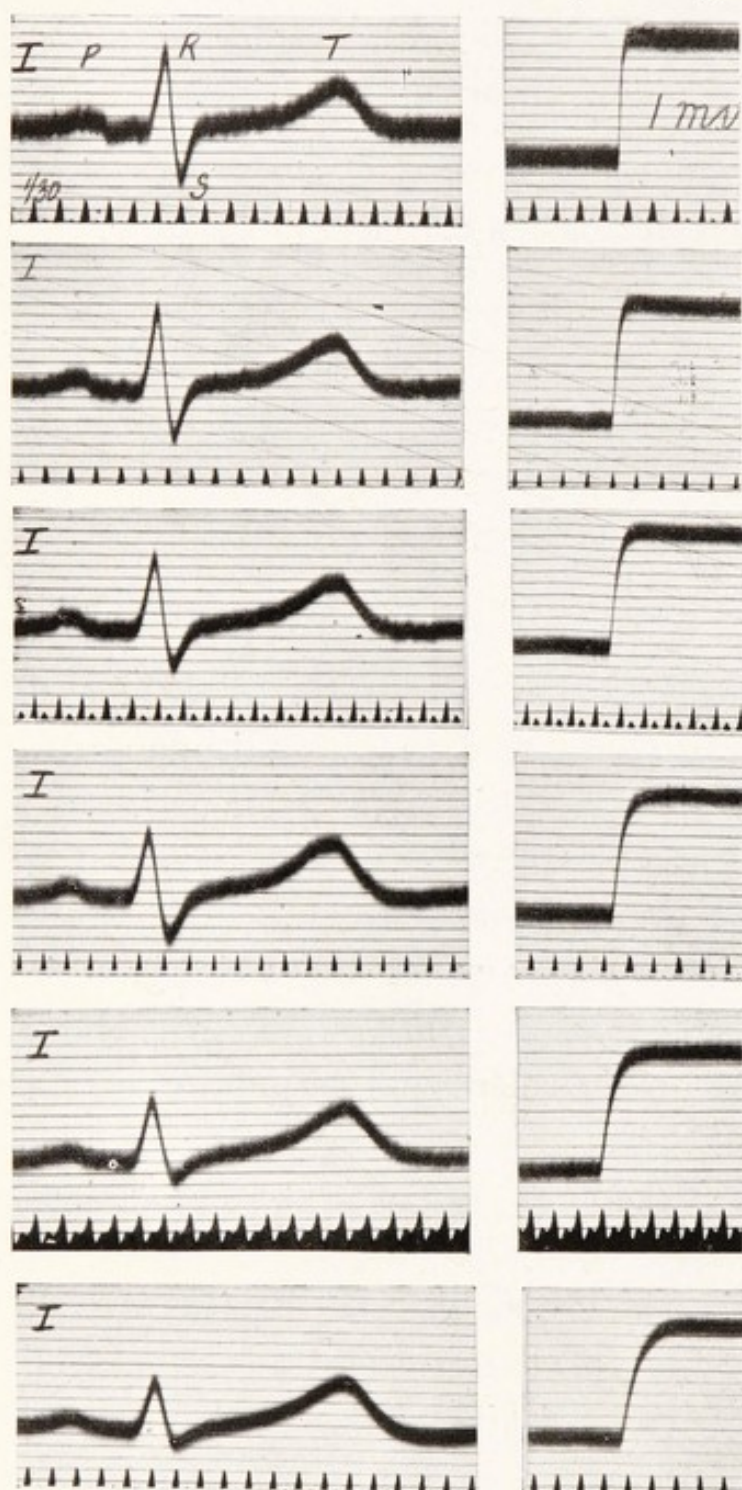
Fig. 6. Six electrocardiograms failed from lead *I* and from a single subject, and the six corresponding deflections in response to a test current. Illustrating the distortion of curves when the string tension is too slack. As the string is slackened beyond a certain limit and the deflection time (the time of a response to an E.M.F. of 1 millivolt over an excursion of 1 centimetre) increases, *R* and *S* are materially reduced in amplitude. Time-marker in thirtieths of a second in this and all other figures, unless otherwise stated.

* The string resistance may be measured by obtaining a deflection of 6 centimetres with 3 millivolts, and by placing such a resistance in series with the string as halves its excursion; *i.e.*, reduces it to 3 centimetres. The resistance of the string and the added quantity are then equal.

Testing certain properties of the string.

If standard curves of correct outline are to be obtained, the response of the fibre to simple current changes must be examined carefully from time to time ; for the excursion and shape of an electrocardiogram may be modified by the properties of the string. Supposing that an E.M.F. of 1 milli-volt is thrown into circuit (the patient being disconnected) and that the tension of the fibre is arranged so that it gives an excursion of 1 centimetre ; the curve obtained has an outline similar to those shown to the right hand of Fig. 6. The string moves when the current enters it and eventually takes up a position 1 centimetre away. But in arriving at the new position it describes a curve. The characters of this curve are important. Fig. 6 shows six electrocardio-grams from the same subject, and six responses to an E.M.F. of 1 millivolt. The curves differ because they were taken with different resistances in circuit, and consequently with different string tensions.* From above downwards the resistance was increased and the string was therefore slackened. All the curves were taken with the string at such a tension that 1 millivolt gave 1 centimetre of excursion (see deflections to right of strips).

In the first place, the movement of the string in response to 1 millivolt should be " dead beat " ; that is to say, there should be no over-shooting ; over-shooting tends to produce distortion of the electrocardiograms by increasing the amplitude of the initial deflections. Over-shooting is shown in Fig. 5 (above *b*) at a stage when the tension of the fibre was too great. In the second place, the movement should be of sufficient rapidity. The slacker the string, the more slowly

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* Adding resistance to the main circuit decreases the sensitiveness of the instrument and the string must be slackened to compensate this decrease if a constant excursion is to be maintained.

does it come to rest in its new position ; the deflection times
for the six strips of Fig. 6 are 0·013, 0·023, 0·028, 0·045, 0·060
and 0·070 of a second, respectively, from A to F. Now the
initial electrical changes that result from the heart beat are
rapid, and if the quickest movement of which the string is
capable is too slow to follow these changes, an accurate
electrocardiogram is not obtained. Undue slackness of the
string distorts the curve and this distortion is well illustrated
by the series of curves shown to the left in Fig. 6. As the
string is slackened, a stage is reached when the steepest
deflections (R, S) are shortened and their upstrokes and down-
strokes are rendered more oblique ; once distortion appears,
further slackening exaggerates it ; so that, as in the last strips
of Fig. 6, certain deflections (such as S and R) tend to
disappear. The electrocardiograms of the first two strips
are exactly alike, these electrocardiograms are accurate ; the
first change, a decrease in the amplitude of R and S in the
third strip, is seen when the deflection time of the string is
increased to 0·028 of a second.

For routine work the instrument and string should be
tuned, so that while on the one hand there is no over-
shooting, on the other hand the deflection time does not
exceed 0·02 of a second. The best strings for electrocardio-
graphic work are those which fulfil these conditions over
a considerable range of string tension. Intending students
of electrocardiography are strongly advised to satisfy
themselves fully of the adequacy in these respects of any
instrument with which they propose to work. It is also
necessary that the resistance of the string should be large
compared with the resistance of the body, so that small
variations of the latter do not appreciably affect the result.
Strings having resistances of about 5,000 ohms are
suitable.

Accessories.

The electrocardiogram is taken simultaneously with a time signal ; a tuning fork or other vibrator, which casts its shadow on the camera slit, suffices for this purpose, and such a device was used in obtaining most of the figures for this book. To-day it is customary to use a toothed wheel, rotating at a constant and controlled speed. Such a time-marker momentarily obliterates the whole of the light falling on the slit and rules vertical lines at regular intervals across the plate (see Fig. 84).

Simultaneous curves from the pulse (Fig. 62) or jugular vein may be obtained by allowing the levers of suitable recording devices to cast their shadows upon the camera slit.

CHAPTER II.

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THE PHYSIOLOGICAL ELECTROCARDIOGRAM.

The normal electrocardiogram consists of a series of deflections, some of which are rapid and of short duration, while others are slow and of longer duration. They have been named in a purely empirical fashion, P, Q, R, S and T. The electrocardiogram opens with a blunt summit P, which occupies presystole, and is due to the contraction process in the two auricles (Figs. 7 and 8). Following upon this deflection the string shadow either maintains the zero position or dips somewhat. I have named these portions of the electro-cardiogram the " auricular complex "; this complex begins with the upstroke of P and terminates at the opening of the " ventricular complex." The latter varies in the number of its component deflections; in its full form it comprises Q, a small dip, R, a tall spike, S, a steep valley of variable depth, and T, a broad blunt summit. The period occupied by all these deflections is approximately that of the ventricular systole with which they are associated. The earliest sign of contraction that can be recorded by other means from the ventricle occurs a little after the commencement of R, and usually during its upstroke. The contraction ends where T passes into the horizontal line of diastole, or within a few hundredths of a second of this point. These relations are indicated in Fig. 8, which shows a simultaneous record of the electrical changes and the apical heart sounds.
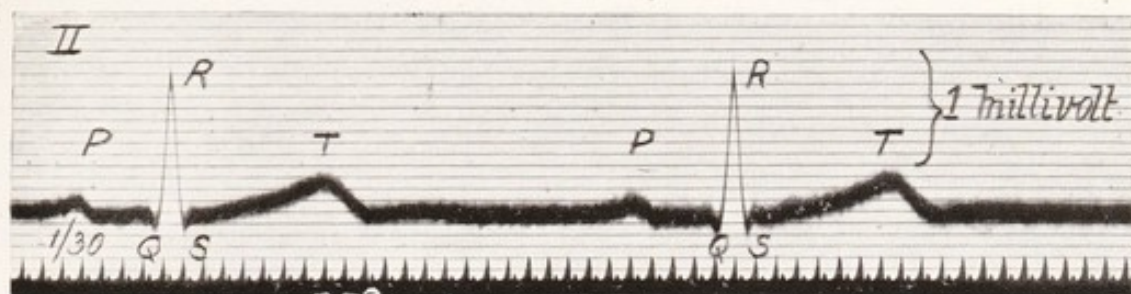
Fig. 7. An electrocardiogram from a normal subject, showing the auricular summit (*P*) and the ventricular deflections (*Q, R, S* and *T*).

In this and the remaining figures, the lead is marked in the upper left hand corner of the figure; the time, at the bottom, is in thirtieths of a second, the distances between the parallel horizontal lines represent tenths of a millivolt.
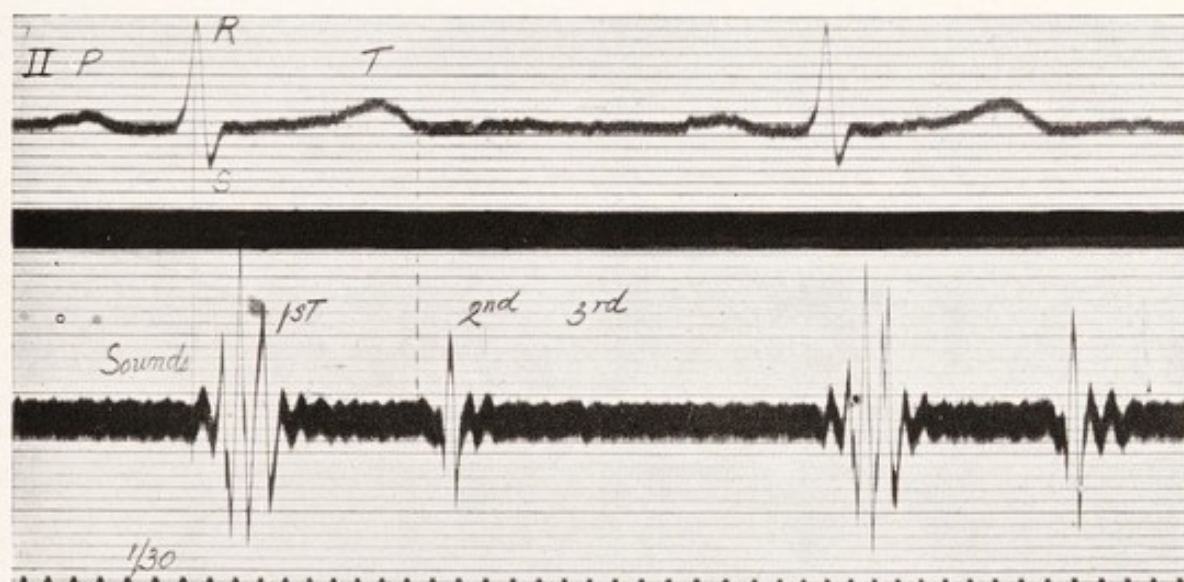

Fig. 8. Simultaneous electrocardiograms and heart sound curve from a normal subject. The figure shows the time-relations of the electrocardiogram to the beginnings of the 1st and 2nd heart sounds. All points on any vertical line are simultaneous.

Thus, the curves provide us with clear indications of the systoles of auricles and of ventricles, and enable us to establish, within very small limits of error, the time-relations of contraction in upper and lower chambers of any given heart.

The initial ventricular deflections (*Q R S*, or *R, S*, as the case may be) correspond to the spread of the excitation wave in the ventricle. This excitation wave is associated

with the wave of contraction. For the purposes of this book the wave of excitation and of contraction may be regarded as simultaneous and as following identical paths ; they are part and parcel of one process. The duration of the initial deflections may be taken as a measure of the time during which all parts of the ventricle are becoming active. The final deflection (T) is produced by subsidence of the ventricular activity, and finishes at the time when all parts of the ventricle first become quiescent.
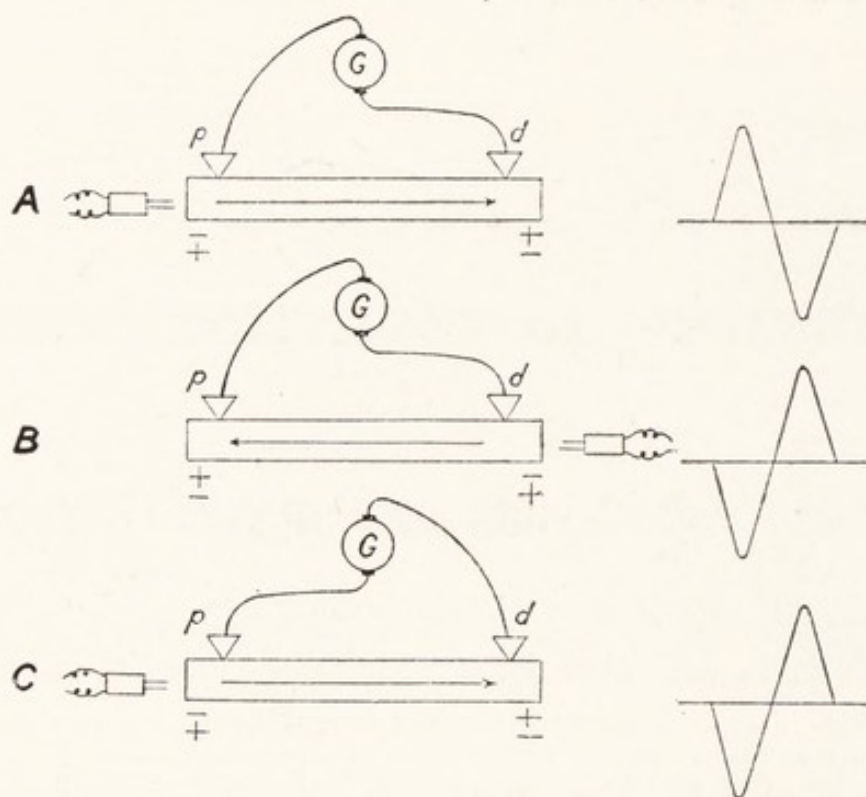
Fig. 9. A diagram illustrating in simplified form the theory of electrical curves. It represents a strip of muscle connected to the galvanometer and stimulated to contract from one or other end. The deflections, which are shown to the right, vary in direction according to the point at which the contraction originates and with the relation of the muscle to the electrodes.

Supposing that we take an isolated strip of muscle, the fibres of which are parallel, and connect its two ends, which we will term proximal (p) and distal (d), to the galvanometer. If this strip is stimulated by a single induction shock, say at its proximal end (Fig. 9A), then a wave of contraction flows

from p to d. When the muscle at p becomes active, it also becomes relatively negative to d, as shown by the swing of the galvanometric recorder ; the swing is in a definite and known direction, it is the same as that given when p is replaced by the zinc terminal of a copper-zinc couple. But the wave passes to d and after it reaches this point it subsides at p. The distal end becomes active and the proximal end resumes the quiescent state ; as an accompaniment of this change, d becomes relatively negative to p, and the swing of the recorder is reversed. Thus expressing the matter rather simply we may say that this electrical record consists of two deflections that are in opposite directions, a simple *diphasic* effect. Now if the stimulus is applied at the distal end (Fig. 9 B) the direction of contraction is reversed ; in these circumstances d first shows relative negativity and later p ; a diphasic effect is still obtained, but the directions of swing are the reverse of those seen when the contraction travels from p to d. It will be evident that the recorded curve will also show reversed phases if, stimulation being at the proximal end, the connections to the galvanometer are reversed (Fig. 9 C).

The meaning of individual deflections composing the electrocardiogram is necessarily a far more complicated matter ; though understood in great part, they cannot be explained in any detail in this handbook. I shall be content to formulate the chief conclusions reached but will defer these until the next chapter. Meanwhile certain fundamental principles emerge from our simple experiment. The shape of the electrical curve is controlled (1) by the path that the excitation wave takes through the tested muscle, and (2) by the lie of this muscle in relation to the leading off electrodes. *If the wave pursues an abnormal course through the heart, it will yield an abnormal electrocardiogram. If the points of contact,*

*which the instrument makes with the body, are altered, the curve
will alter correspondingly.*

Now it is known that all normal human hearts yield
from a given lead, electrocardiograms conforming to a type.
Although the curves taken from no two subjects are exactly
alike, yet by experience we learn the limits of variation
that are compatible with health.

The physiological auricular complex. P, the auricular
representative, is found as a summit* in all normal and
young adult subjects and in all leads ; it is small and rarely
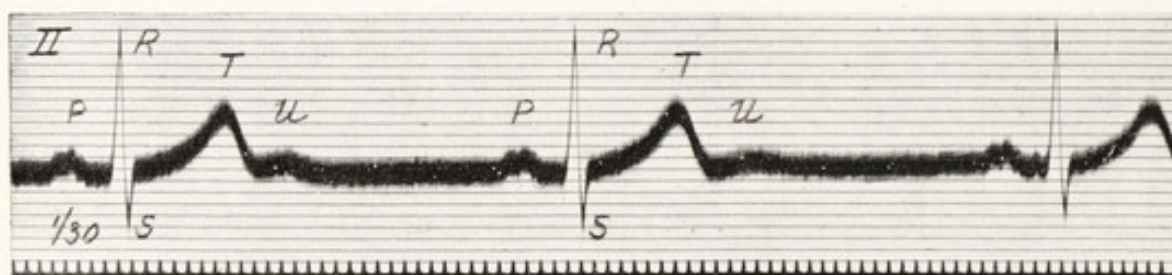exceeds 2 scale divisions in amplitude ; it is followed by a

Fig. 10.   A normal electrocardiogram, showing a divided P summit ; a
prominent U summit occurs at the beginning of diastole.

short line which is horizontal or dipping. Amongst the
variations in form, which P presents in the normal subject,
is occasional bifurcation (Fig. 10).   *Auricular complexes
of these forms are known to express the origin of the heart beat
at the normal site of impulse formation and the passage of
the wave through the auricular tissue along normal paths.*

The physiological ventricular complex.   The variations
that are found in the type of the physiological ventricular
complex are many.   R and T are always present ; Q and S

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* P and R are upward movements when the galvanometric connections
are so arranged that if the right arm contact is replaced by the zinc terminal
of a copper-zinc couple, and the left leg contact is replaced by the copper
terminal, an upward deflection results.

are often absent. All the deflections vary in amplitude and in shape, and in certain leads *T* may vary in its direction.

The *QRS* group of deflections is quick, having in normal subjects a total duration of no more than one-tenth of a second. The presence of a *QRS* group of rapid deflections, followed by a slow blunt *T* deflection, indicates that the ventricular contraction has been propagated from normal points. *It has been awakened by impulses that have traversed the auriculo-ventricular bundle, its main divisions and their arborisations.*

Certain variations in the form and amplitude of individual deflections require closer description, and it will be necessary to refer to the leads from which the curves are taken. We may confine ourselves to the features that are of chief clinical importance.

Curves from the three leads of a normal subject are shown in Fig. 11. It will be noticed that in all *R* is prominent, but that it is most prominent in lead *II*, and that *T* is directed upwards. On the other hand *Q* and *S* are inconstant in the leads. This series of curves may be regarded as of average type. In a series of over 50 young adult subjects the greatest variations presented by serial leads are depicted in Figs. 12 and 13. The importance of these extreme electrocardiograms will be better appreciated when we deal with pathological curves in the next chapter. At present it should be noticed that in Fig. 12 *R* is inconspicuous in lead *I* and most prominent in lead *III*, while *S* is most conspicuous in lead *I*. Precisely the reverse relations are shown in Fig. 13, where *R* is most prominent in lead *I* and inconspicuous in lead *III*, while *S* is deepest in lead *III*. These variations in amplitude may result from preponderance of the musculature in right (Fig. 12) and left (Fig. 13) ventricles respectively, and are

extreme examples of the pictures that are found in apparently healthy subjects.

Normal electrocardiograms occasionally exhibit notching of R or S (Fig. 13) ; and in lead *III* bizarre types of initial deflections (the QRS group) are not uncommon ; examples are shown in Fig. 14.

T is always upright in young and healthy individuals in lead *II* and under normal conditions, but it is often inverted
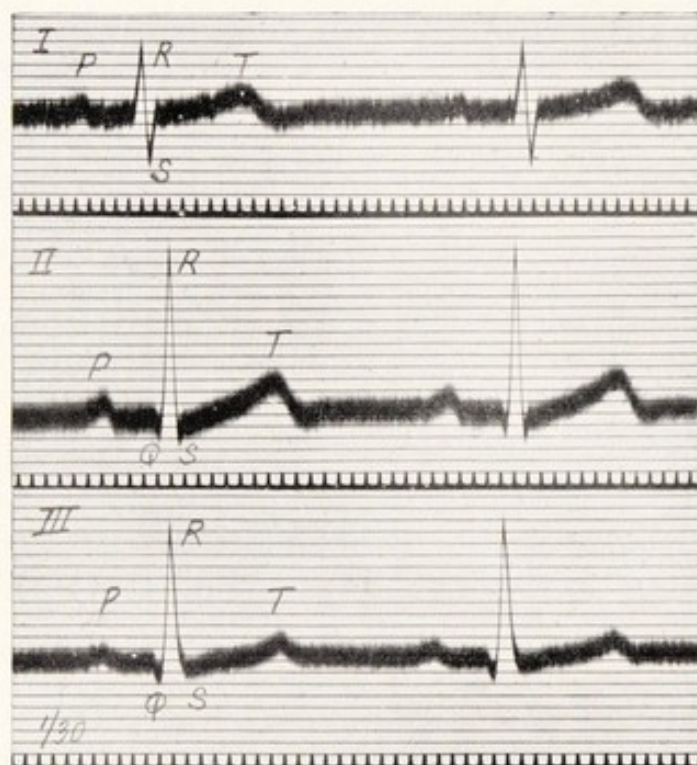

Fig. 11.   Electrocardiograms from the three leads in a normal subject. To illustrate the change in the type of curve with change of lead. ⁕ Note that R is tallest in lead *II*.

in lead *III* and may show partial inversion in lead *I*. Inversion in lead *III* is generally associated with the bizarre QRS group to which reference has been made already (Fig. 14), but may occur with the usual form of initial deflections (Fig. 12).

An additional deflection U is not uncommon and is occasionally prominent (Fig. 10). This deflection is related to the early events of diastole, but beyond this is not understood.
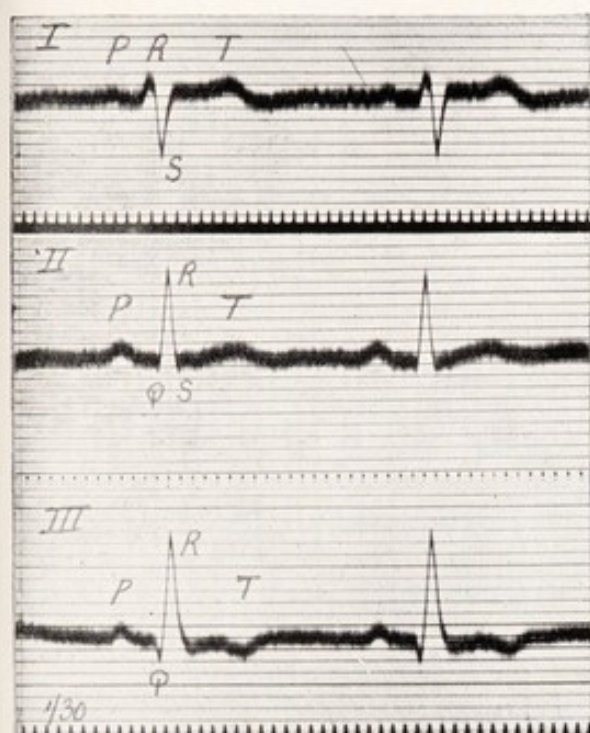
Fig. 12. Normal electrocardiograms, showing the tallest *R* in lead *III* and the shortest *R* and deepest *S* in lead *I*. For comparison with Fig. 21.
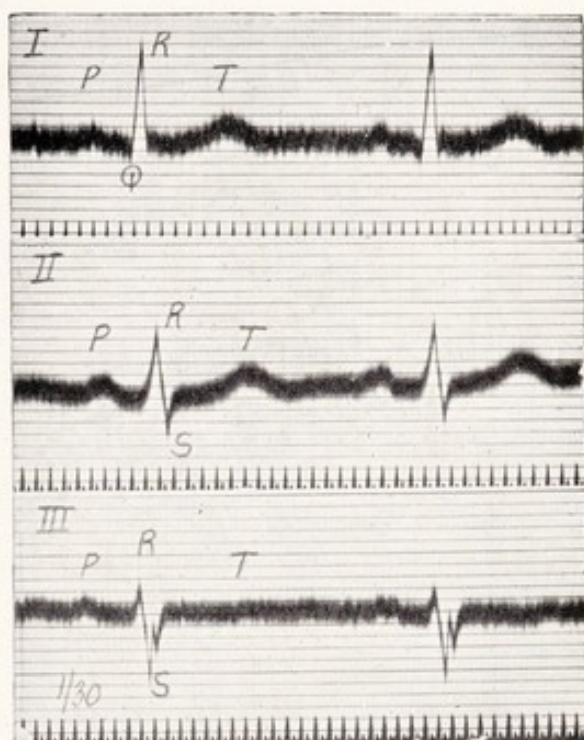
Fig. 13. Normal electrocardiograms, showing the tallest *R* in lead *I* and the shortest *R* and deepest *S* in lead *III*. For comparison with Fig. 20.
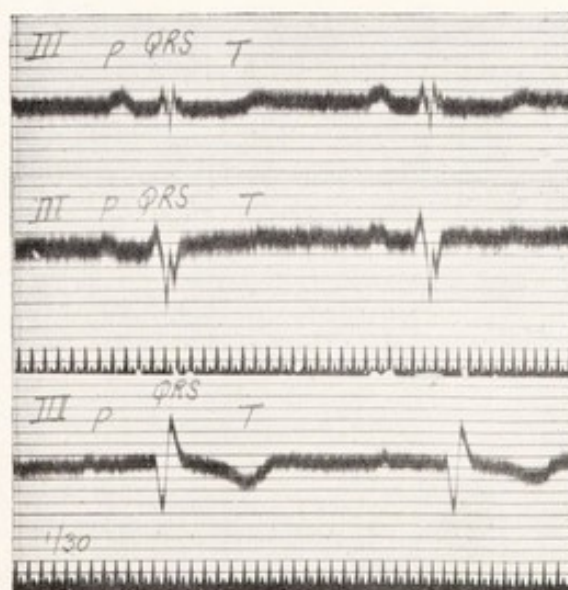

Fig. 14. Normal curves taken from lead *III* in three different subjects. Illustrating the curious initial deflections which are sometimes seen in this lead. They are often associated with inversion of *T*.

Standard curves from the same subject are almost identical in form and amplitude from time to time. The constancy is such, and the variations in distinct subjects are sufficiently definite, that electrocardiograms might be used to identify the individual. Considerable changes in the heights or directions of deflections in electrocardiograms, taken from time to time and under similar conditions, do not occur in health and rarely occur in chronic disease. An observed change is significant of a contemporary change in structure or function.

A certain familiarity with the limits between which the amplitudes of deflections vary in health is useful ; it is gained by experience. Some idea of their value may be obtained from a series of curves taken from a number of active students. There were in all 59 individuals from whom curves were taken. The limits of amplitude for the several deflections are given in the accompanying table, which includes measurements

		P	Q	R	S	T	U
				Lead I.			
Minimum		Trace	0	1·5	0	—0·5	0
Average ...	...	0·52	0·51	5·16	2·06	1·93	0·10
Maximum	...	1	2·0	12·0	6·0	5·5	Trace
				Lead II.			
Minimum	...	Trace	0	4·0	0	Trace	0
Average ...	...	1·16	0·73	10·32	2·23	2·46	0·16
Maximum	...	1·7	2·5	16·5	4·5	5·0	0·8
				Lead III.			
Minimum	...	Trace	0	2·0	0	—2·0	0
Average ...	...	0·81	0·86	6·61	1·73	0·61	0·06
Maximum	...	1·5	2·5	14·0	4·0	3·0	0·3

from 52 subjects. The measurements from seven subjects are not given ; they were rejected from the normal series for various reasons. Six manifested abnormal signs upon the ordinary physical examination, and, of these, *four* gave

electrocardiographic curves that diverged notably from those of the selected series. Only one student in whom no other physical signs were obtainable presented divergent electrocardiograms. Two subjects giving normal electrocardiograms were found to have, the one a tricuspid murmur, the other a slight extension of the left limit of heart dulness. These facts speak for themselves; if in any subject the electrocardiograms diverge from what is regarded as normal, it is probable that the heart is abnormal. But, on the contrary, if the electrocardiograms are normal in form, it does not follow that the heart is normal.

Electrocardiographic curves are modified by age; the chief change is a decrease in the amplitude of T as the subject becomes older. They are also influenced by displacement of the heart, such as occurs in different postures, or with deep breathing or where there is an accumulation of fluid in a pleural sac. They are modified by exercise, notably by an increase in the amplitude of T. But these factors materially influence the interpretation of pathological curves in exceptional cases only.

CHAPTER III.

————

RHYTHMIC BUT ANOMALOUS ELECTRO-CARDIOGRAMS.

In the last chapter electrical curves were shown to be controlled by the direction that the excitation wave takes in the muscle yielding the record. Clearly to appreciate this principle and to apply it constantly is the basis of electrocardiography. If the heart beat starts in an abnormal focus or if a wave, propagated from the normal focus, subsequently deviates from its accustomed paths, an anomalous electrocardiogram is the inevitable result. The systoles of auricle or of ventricle may be classified according to the forms of electrical curves that they produce.

Now, in the natural heart beat, the wave is propagated over auricle to ventricle ; the stimulus is conveyed from the first chamber to the last through the auriculo-ventricular bundle ; thence it spreads through the two main divisions of this bundle and is distributed to the right and left ventricle through the arborisations and basketworks of Purkinje cells. In these circumstances the ventricular systole yields an electrocardiogram conforming to a recognised type ; the familiar deflections, Q, R, S and T are produced. This type of electrocardiogram is only seen when the systole to which it corresponds is provoked by an impulse arising above the division of the A-V bundle—an impulse that I

distinguish as *supraventricular*—and it is limited to responses of the ventricle to supraventricular impulses that descend all the normal channels. Thus, if the ventricle responds to an impulse generated in its own walls, the electrocardiogram is abnormal. (*See* Chapter *V.* on *Premature Contractions.*) The curve is also abnormal if a supraventricular impulse is distributed to the ventricle in an unusual manner, as may happen. For in the first instance and in the second, the course taken by the resultant wave in the ventricular walls deviates from the normal.

The constitution of the ventricular complex.

When a supraventricular impulse (for example, the normal impulse), on passing to the ventricles, enters the normal field of reception, it reaches the two ventricles simultaneously. Each ventricle possesses a complete and separate system of distributing fibres (see Fig. 15, top diagram). Each ventricle yields its own electrical currents and each, while beating normally, yields a distinctive curve. Up to a point the forms of the curves distinguishing the normal systole of right and left ventricles, respectively, are known to us. It is for the student of electrocardiography to become thoroughly familiar with these types. In Fig. 15 is a diagram of the ventricles, seen in section, and of the auriculo-ventricular bundle and its ventricular connections. The septum of the ventricles forms a saddle across which the dividing bundle sits astride. If the right division of the bundle is transected below its origin (as at B^1) the normal impulse no longer travels through it, but passes solely through the left stem. The distribution of the impulse is faulty, but it is only faulty in so far as the right ventricle is concerned ; it is distributed to the left chamber in a perfectly normal fashion. The curves which this partial distribution yields are shown in the
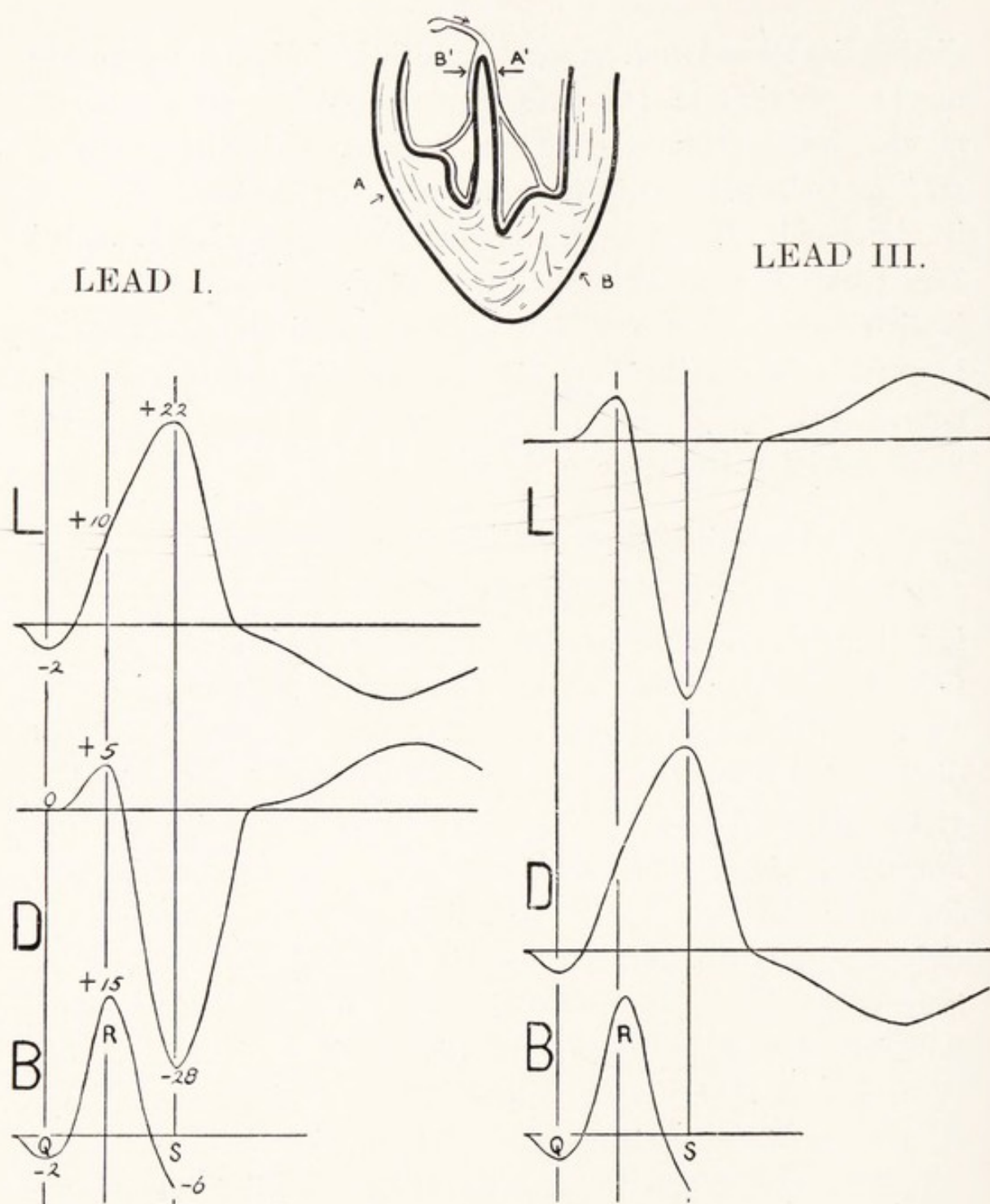
Fig. 15.   A diagram of the ventricles seen in section and of the auriculo-ventricular bundle and its branches.

A lesion of the bundle division at A^1 forces the natural impulse to descend solely through the right division, the supply of the right ventricle. The corresponding electrocardiograms are characteristic, and are not dissimilar in form to those obtained by stimulating the right ventricle at A. A lesion of the right bundle division at B^1 confines the descending impulse to the path of the left division. The corresponding curves are characteristic and are not dissimilar in form to those obtained by stimulating the left ventricle at B.

Below the diagram are two series of curves, arranged vertically and corresponding to leads *I* and *III*. The top curve in each series is the *levogram*, obtained when the right bundle division is damaged; the middle curve in each series is the *dextrogram*, obtained when the left bundle division is damaged. The bottom curves represent the initial phases of the normal electrocardiogram and the diagram is arranged to show that these initial phases are the algebraic product of the levogram and dextrogram. In the series from lead *I* I have inserted the amplitudes of the curves where they cross the common vertical lines, marking them as + or — quantities according as the deflection is up or down. Thus Q (-2) is the result of a similar dip (-2) in the levogram: R (15) is the product of the small spike (5) in the dextrogram and of the rising levogram (10).

diagram (L L), and these curves in their initial phases (while the excitation is confined to the left ventricle) represent the currents *normally* formed in the left ventricle. In lead I the curve comprises a small downward phase, a large upward phase and a final and sustained downward phase. In lead III the curve comprises a small upward, a large downward and a final and sustained upward phase. Because these curves correspond in their initial phases to the natural systole of the left ventricle, I call them *levograms*. If, on the other hand, the left bundle division is divided the impulse is conveyed solely to the right ventricle ; but this distribution, in so far as it affects the right ventricle, is again normal. The corresponding curves, which I term *dextrograms*, are shown in the diagram (D D). The curve of lead I comprises a small upward phase, a large downward phase and a final upward phase ; in lead III it comprises a small downward phase, a large upward phase and a final downward phase.

Now the levogram and dextrogram are not only of intrinsic clinical importance, but they also show us how the normal electrocardiogram is constituted. I have indicated that in so far as their initial phases are concerned they represent the normal events in the left and right ventricle respectively. Each ventricle forms its own currents and it is the combination of the two effects that constitutes the normal curve. In the left hand series of curves of Fig. 15 the levogram and dextrogram of lead I are charted. Summate the initial phases of these two curves—add them together algebraically—and the result is the initial phases Q, R and S of the normal heart beat. A similar summation of the levogram and dextrogram in lead III will yield the normal curve in lead III. If this diagram is studied it will become clear that in human electrocardiograms :—

In lead I " Q " is a left ventricular event.
       " R " is mainly a left ventricular event.
       " S " is a right ventricular event.
While in lead III " Q " is a right ventricular event.
   *" R " is mainly a right ventricular event.*
   *" S " is a left ventricular event.*

That these conclusions are important will become clear when the curves corresponding to hypertrophy of the ventricles are described.

Aberrant beats.

I term those beats of the ventricle *aberrant* that, propagated from supraventricular impulses, are distributed in a partial or faulty fashion ; these aberrant beats are frequently discovered in clinical electrocardiograms.   Just as the functions of the main stem of the *A-V* bundle are often defective in the human subject (see Chapter *IV*.), so also are those of its individual branches.   The types of electrocardiogram representing deficient conduction in the main divisions of the bundle are illustrated by actual examples in Figs. 16 and 17 (and have been in part described in reference to Fig. 15).   The natural auriculo-ventricular sequence is maintained, that is to say, each ventricular beat is preceded by an auricular one ;   the ventricular complexes are alone abnormal.   The common features of the ventricular curves are the exaggerated amplitude of excursion, an amplitude often much greater than that of the curves chosen for illustration, and the prolongation of the initial phases. The deflections that replace the usual QRS group (initial deflections) have a total duration exceeding a tenth of a second and comprise more than a third of the whole complex. When the *right* branch is at fault the first chief deflection is a broad summit R^1 in lead I, a deep and broad depression
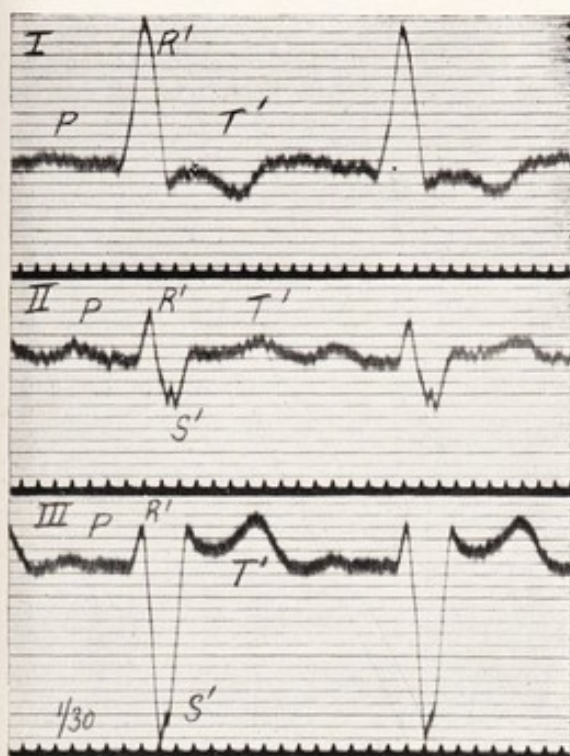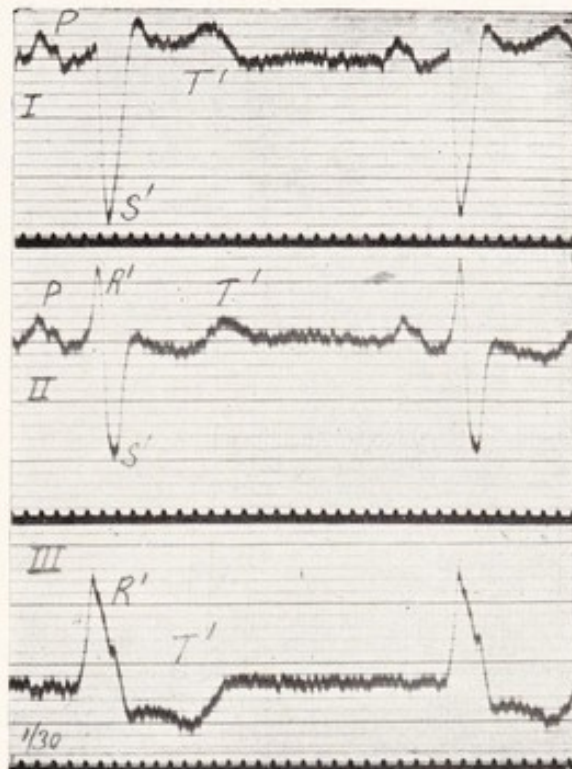
LEVOGRAMS.   DEXTROGRAMS.

Fig. 16. Curves from three leads, evidencing functional defect of the
right division of the auriculo-ventricular bundle. Note the long duration
of the initial phases and the large amplitude in leads *I* and *III*.

Fig. 17. Curves from the three leads, evidencing functional defect of the left
division of the auriculo-ventricular bundle. The directions of the
deflections are the reverse of those of the last figure, in leads *I* and *III*.

S^1 in lead *III* (Fig. 16). Each of these phases is followed
by a sustained and very prominent phase T^1, which
is of opposite sign to the first chief deflection in the
same lead; it is directed down in lead *I* and up in
lead *III*. When the left branch is at fault the pictures are
reversed (Fig. 17). A deep depression S^1 is followed by an
upright and prominent T^1 in lead *I*; a broad summit R^1
is followed by a downward directed T^1 in lead *III*. In
addition to these deflections, a dip (Q^1) may appear to a
variable extent in lead *I* of the levogram and in lead *III* of
the dextrogram, while a diminutive summit R^1 may open the
events in lead *III* of the levogram and in lead *I* of the
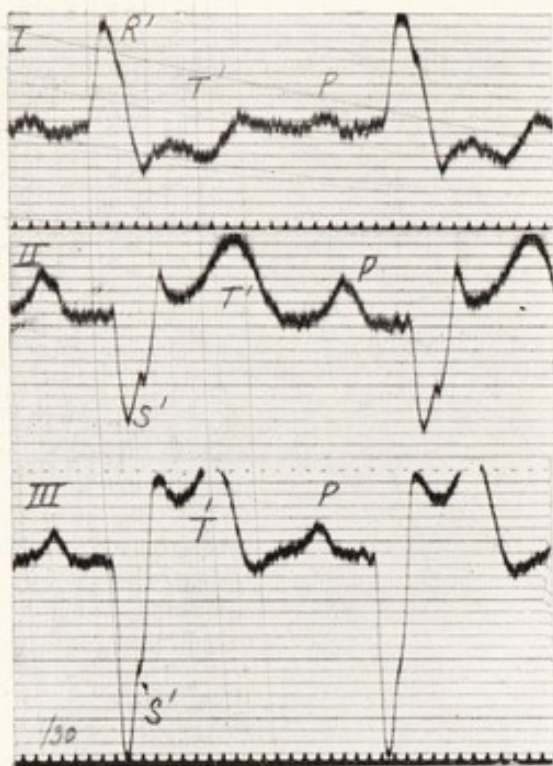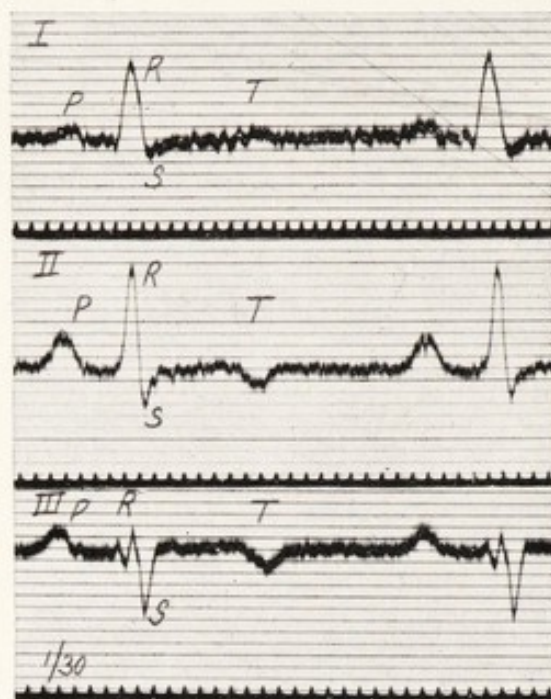
Fig. 18.   Fig. 19.

Fig. 18. Curves taken from the three leads in a case of aortic disease during
        a febrile attack. They show defective conduction along the right
        division of the auriculo-ventricular bundle (see Fig. 16).

Fig. 19. Curves from the same patient, taken a day later and during the
        subsidence of the fever. The ventricular portions of the curves have
        changed profoundly ; there is now no evidence of bundle defect, but
        rather of preponderance of the left ventricle.

dextrogram.   The curves of lead *II* are often similar to those
of lead *III* in lesions of the right division, but in lesions of
neither division are they so characteristic as in leads *I* and
III.

The value of these electrocardiograms as signs of disease
is great, for damage of the bundle divisions is to be identified
by them, and by them only.   A reduplication of the first
heart sound is a frequent but inconstant association.   The
pathology of the lesions is similar to that of auriculo-ventri-
cular heart-block, which is frequently coincident.   As a
permanent condition, damage of a bundle branch speaks for
a lesion in this situation, though the latter is usually but a

local expression of widespread mischief in more silent areas. As a temporary condition it results from an acute or subacute process, an invasion of the myocardium by an infective agent or poison. Such was the probable explanation in the case from which Figs. 18 and 19 were taken. Admitted to hospital he was found to have an aortic lesion ; the temperature was 100°, but there were no other clinical signs of infection. The electrocardiograms (Fig. 18) showed deficient conduction in the right branch of the bundle, a deficiency which passed away as the fever subsided (Fig. 19). The electrocardiogram provided the sole evidence of an acute myocardial involvement.

That the sign, as a persistent sign, is not infrequent in heart cases we know ; as a transient sign it is probably more frequent than we suspect. The right division is much the commoner to be affected, probably because it runs a much longer course as an isolated strand. Curiously, a defect of this division is often associated with aortic disease. The death rate amongst patients who exhibit it is extraordinarily high.

Preponderance of left or right ventricle.

Before discussing the electrocardiograms associated with hypertrophy of the ventricles it will be convenient to particularise in respect of terms. Normally the separated ventricles when weighed show a certain mass relation to each other ; the left ventricle in the average weighs approximately 1·8 times as much as the right. In instances of hypertrophy of the heart it is usual to discover an increase in the weight of both ventricles, pure unilateral hypertrophy as a manifestation of disease being rare ; but although there is this bilateral hypertrophy, one ventricle is often involved to a greater extent than the other and the normal weight ratio between the two ventricles is then disturbed. When the heart

is enlarged, such an altered ratio of weights changes the electrocardiogram to the forms now to be described.

To indicate a relative increase in the weight of a given heart chamber, I use the term preponderance of that chamber. We shall be dealing with instances of actual hypertrophy, confined may be to one chamber, but more usually affecting the two chambers unequally. Speaking of hypertrophy, there are certain preliminary considerations which I would impress. While purely mechanical influences undoubtedly change the distribution of the mass of ventricular muscle, yet hypertrophy often exists where there is no reason to suspect antecedent alterations of the fluid pressures in the chambers, and where the degree of hypertrophy in a given chamber cannot legitimately be ascribed to an increase in the burden which that chamber may be supposed to have borne. Unquestionably there are causes of ventricular hypertrophy, and of massive hypertrophy, that are still unknown to us ; I would even go so far as to say that the chief cause of hypertrophy is still unknown, and that we are unaware of the extent to which such unrecognised factors may be responsible for the hypertrophy associated with valvular disease. If in a case of enlarged heart electrocardio- grams are sometimes obtained that point to preponderance of one chamber, while the clinical signs display a valve lesion usually associated with hypertrophy of the other chamber, the electrocardiographic signs are not to be discarded on that account. Providing that the heart is not materially displaced, the interpretation of the electrical curves here given may still be accepted. If hypertrophy of the left ventricle is anticipated from the valve lesion and yet the curves of right preponderance are obtained, then the right ventricle is also hypertrophied and to an even greater extent than the left. Or if in instances of hypertrophy of the heart the curves

show no sign of left or of right preponderance, then both ventricles participate more or less equally in the enlargement.

The electrocardiograms associated with *preponderance of the left ventricle* are curves in which there is a predominance of the levogram; they are illustrated by Fig. 20. The amplitude of R is greatest in lead I and is least in lead III; S is greatest in lead III and is inconspicuous in lead I; Q is most prominent in lead I. Reference to Fig. 15 will
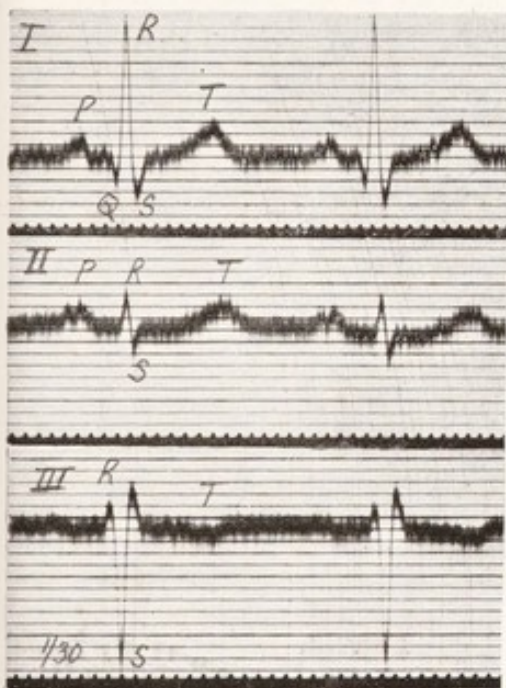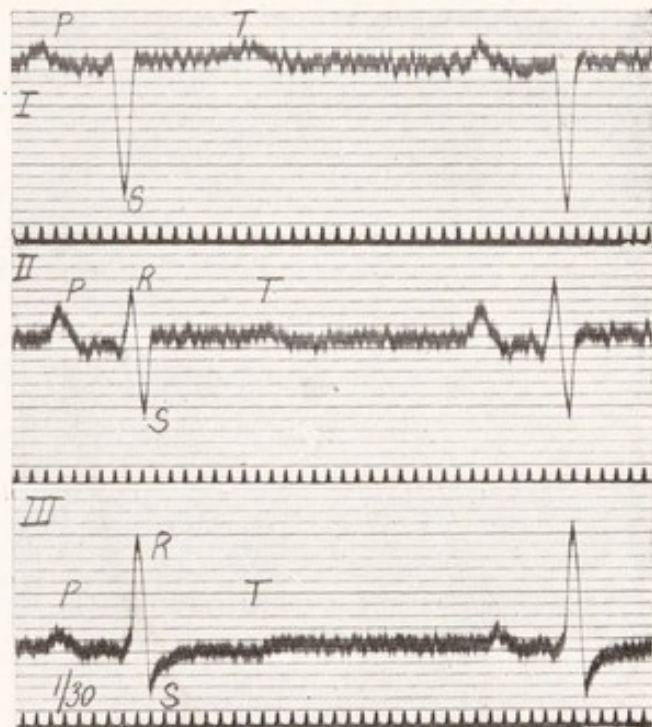
Fig. 20.   Fig. 21.

Fig. 20. Curves from the three leads in a case of aortic disease showing preponderance of the left ventricle. R is tallest in lead I and shortest in lead III, while S is deepest in lead III.

Fig. 21. Curves from the three leads showing preponderance of the right ventricle. Note that there is but a trace of R in lead I and that S is very deep; also that R is tallest in lead III, while S is small.

show why this should be, for the deflections that are exaggerated in left ventricular preponderance are the deflections derived entirely or in chief part from the levogram (see Table on page 28). As is to be expected, the curves of left preponderance are to be seen most frequently in

D

association with heightened blood pressure, aortic disease, and in the senile heart. In *preponderance of the right ventricle* (Fig. 21) the dextrogram predominates, *S* is exaggerated in lead *I* and is shortest in lead *III*, *R* has its greatest amplitude in lead *III* and is inconspicuous or absent in lead *I*, *Q* is most in evidence in lead *III*. The deflections that
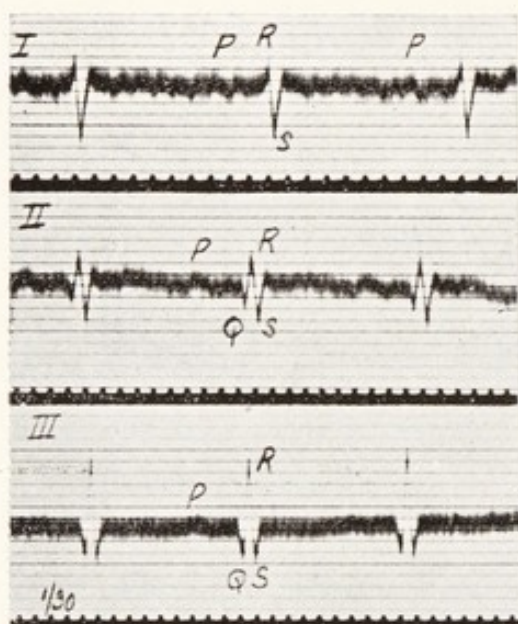
Fig. 22.
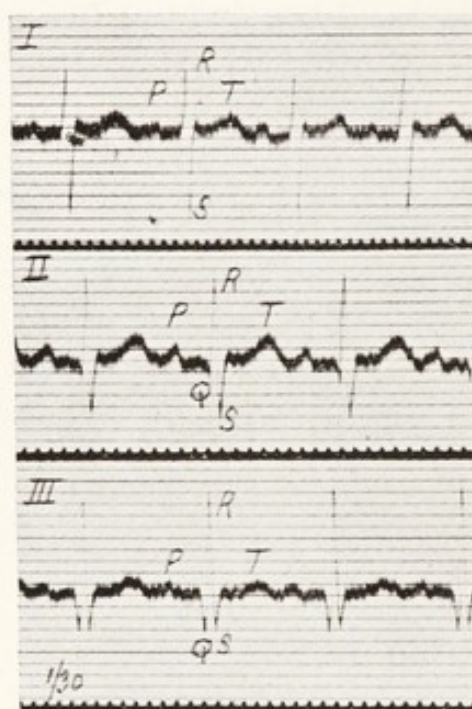
Fig. 23.

Fig. 22. Curves from a child two hours after birth. The relative heights and depths of the peaks is such as is expected where there is preponderance of the right ventricular muscle.

Fig. 23. Curves from the same child, but six weeks later. The right-sided preponderance is not evidenced by these curves to nearly the same extent.

are exaggerated are those derived from the dextrogram. The curves of right preponderance are obtained in an exaggerated form from cases of congenital pulmonary stenosis and they are obtained in nearly all uncomplicated cases of advanced mitral stenosis. The constancy with which they are obtained from the new-born child (Figs. 22 and 23) is most notable, for in the new-born child the right

ventricle is as heavy as is the left ; that is to say, the right ventricle preponderates, in comparison with the ratio in adults. In children the adult types of electrocardiogram become established between the ends of the second and third month of extrauterine life ; it is at the same period that the normal weight-ratio between right and left ventricles becomes established.

Between the curves of ventricular preponderance and the curves corresponding to lesions of the *A-V* bundle divisions there are naturally many points of similarity ; there are differences also. The directions of the chief initial phases are the same in the two groups ; but in instances of the ventricular hypertrophy, the *duration* of the initial deflections is in the average much less ; it does not usually reach a tenth of a second and does not comprise as much as a third of the whole complex. In exceptional instances of preponderance, however, where one ventricle is enormously increased as compared to the other, the initial deflections of the corresponding electrocardiograms are prolonged and may be indistinguishable in form from those seen in lesions of the bundle divisions. In such cases the distinguishing feature is discovered in the direction of the final deflections. In lesions of the bundle divisions, the final deflection T^1 in leads *I* and *III* is always of opposite sign to the chief initial deflection and is of unusual amplitude ; in cases of preponderance this rule does not hold good, moreover the final deflections *T* are not more pronounced in amplitude than are they in health. The two series of curves are well contrasted in Figs. 18 and 19, both series being taken from the same patient.

Although there is a close association between preponderance of one or other ventricle and the curves described, it has also been shown that these curves are obtained in

instances in which the relative weights of the ventricles fail to account for them. This is true chiefly of hearts of normal total weight, and it is thought that the unusual curves are due in these instances to variations in the position of the heart, since tilting up of the apex tends to produce curves resembling those of left preponderance, or to changed distribution of the excitation wave. In view of these exceptions it is inadvisable to interpret curves as displaying right or left ventricular preponderance in adults unless there are unmistakable clinical signs of cardiac enlargement.

Inversion of "*T*."

In young and healthy adults the final deflection T is always upright in lead II, almost always upright in lead I, but often inverted in lead III. Inversion of T in lead II may be provoked transiently by digitalis ; or by the ingestion of quantities of iced water, whereby the apical regions of the heart are cooled. As a persistent phenomenon it is seen from time to time in elderly subjects ; it is most commonly displayed by patients in whom there are abundant signs of grave myocardial disease (Figs. 19 and 96). Inversion in one or more leads is a usual sign after coronary artery thrombosis (*see p.* 117). Inversion in lead II when persistent is usually regarded as a sign of some prognostic consequence, being taken to indicate permanent myocardial damage. Although the death rate amongst groups of patients displaying it is heavy, sometimes it is found to persist for many years, while the health of the subject remains unchanged.

CHAPTER IV.

AURICULO-VENTRICULAR HEART-BLOCK, ETC.

Partial heart-block.

The chambers of the normal heart contract in orderly sequence ; each impulse is generated in the neighbourhood of the sino-auricular node. This is a special structure in the mammalian heart and situated at the opening of the superior vena cava. Because it controls the normal heart rhythm I term it the " pacemaker." The impulse, after leaving the node, courses through both auricles and, arriving at the auriculo-ventricular bundle, the specialised structure that unites auricles and ventricles, is transmitted to the ventricles, and these respond to it. There is an appreciable delay in this transmission ; the auricle, as we know, contracts before the ventricle ; and the interval between the beginning of systole in auricle and ventricle indicates the length of this transmission time. The electrocardiogram measures this interval more accurately than any other method which we possess. The interval may be gauged as accurately in the human subject to-day as in an experiment upon an animal in which auricle and ventricle are laid bare. The measurement is taken from the beginning of P, the auricular complex, to the beginning of the ventricular complex ; as a rule R is used for the sake of uniformity, but Q, which is often the first ventricular deflection, may be used if it is thought

desirable. The *P-R* interval, as it is termed, varies in normal subjects between 0·12 and 0·18 of a second in length. Prolongation beyond 0·2 of a second is never found in healthy subjects under normal conditions. Such prolongation represents the first stage of heart-block. Fig. 24 illustrates the condition described; in it the *P-R* interval measures 0·32 of a second. The sole disturbance, as shown by the electrocardiogram, is an increase of the interval named; individual
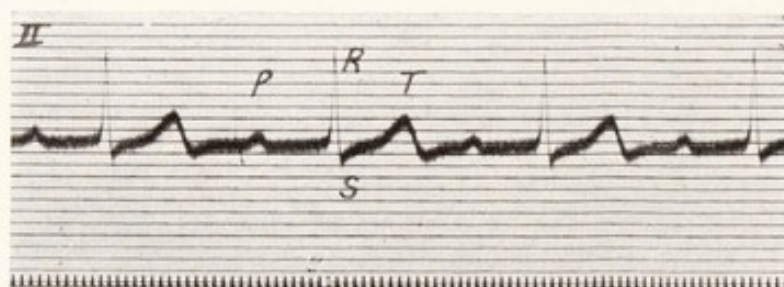
Fig. 24. An electrocardiogram from a case of subacute cystitis and a temporary affection of the heart, showing prolongation of the *P-R* interval to 0·32 of a second.
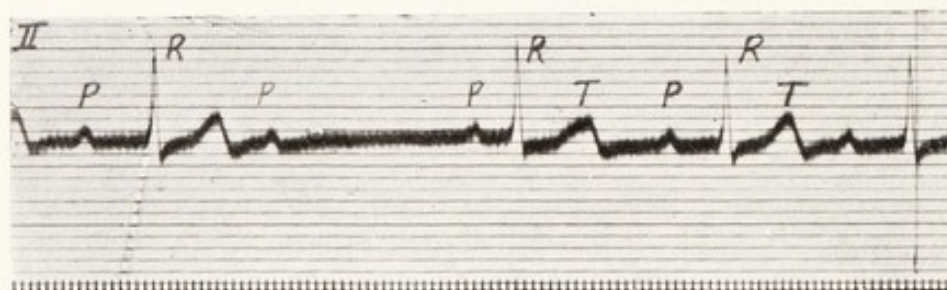
Fig. 25. From the same case, showing a single dropped beat.

auricular and ventricular summits are of normal outline. The heart that manifests this change beats regularly.

The next phase in the progress of heart-block is termed the stage of " dropped beats," where, from time to time, an auricular systole provokes no ventricular response. The electrocardiograms show perfectly regular and normal auricular summits *P*, and most of them are followed by ventricular complexes; but here and there a ventricular

complex is missed and the auricular peak stands isolated. This is one of the causes of so-called pulse "intermittence." A simple example of the condition is shown in Fig. 25 ; measurement will show that the auricular contractions are equidistant, while the intervals between the ventricular beats vary. An interval, of almost double the usual length, separates two of the ventricular contractions ; the long cycle is not exactly twice the length of the usual cycle, because, as the figure shows, the P-R interval that ends the long cycle is shorter than the remainder ; the ventricular beat that stands at the commencement of the restored rhythm has been moved a little to the left of its natural position, slightly shortening the cycle to the left of it and slightly widening the cycle to the right of it. All the remaining P-R intervals are of equal and increased length. This distribution of the ventricular beats is characteristic of the irregularity described. Figs. 27 and 28 exemplify the same phenomena. In Fig. 27 ventricular silences occur after each third or fourth auricular cycle. The analysis of such a curve is readily accomplished : consider the central group of three ventricular beats ; the first is preceded by an unmistakable auricular contraction (P^5), for it lies in a long diastole. A similar auricular contraction (P^9) is found at the end of the next long diastole. The remaining auricular contractions are discovered by attending closely to the shapes of the T waves. T^3, T^4 and T^5 have different forms ; the first and last are split ; T^4 is taller and more pointed. The T waves have different forms because P summits fall with them. If, in this curve, we take a distance of 6 time-marks from the commencement of an R summit and measure to the right, we always arrive at the apex of the corresponding T wave, for the length of systole is constant in the curve. That the remaining summits are

Fig. 26. An electrocardiogram showing prolongation of the *P-R* interval. *P* falls before the termination of *T* of the preceding ventricular cycle. From the same case as Fig. 27.

Fig. 27. Curve showing gradual prolongation of the *P-R* interval and the failure of response to each third or fourth auricular impulse. Partial heart-block of this grade is responsible for some instances of intermittent pulse.

Fig. 28. Curve showing gradual prolongation of the *P-R* interval, until *P* falls back to *S* of the preceding ventricular curve, with failure of response to each sixth or eighth auricular impulse.

Fig. 29. From a case of mitral stenosis (note the large and bifid *P* summit). Partial heart-block is present, the ratio of auricular to ventricular contractions being at first as 3 : 2, and later as 2 : 1.

auricular is proved by measuring the intervals between them ; P^5 and P^6 are separated by the same interval as P^8 and P^9. The distance between P^6 and P^8 is exactly twice as great, and the intermediate point lies on the summit of T^4. T^4 and P^7 are blended ; the two summits superimpose accurately, whence the unusual amplitude of this peak. A perfectly regular series of auricular summits, therefore, is present in the curve ; on the other hand the ventricle beats irregularly. Yet the contractions in upper and lower chamber are related, for the events are repeated. The fifth auricular contraction (P^5) yields the third ventricular response (R^3) ; the succeeding auricular contractions (P^6 and P^7) also stimulate the ventricle (R^4 and R^5), but after progressively greater intervals ; the last auricular contraction of the group (P^8), falling far back into ventricular systole as it does, stands isolated ; after the ventricular silence the events recur in precisely the same order. The shortening of the $P\text{-}R$ interval after the long diastole in this figure and in Fig. 25 results from rest and the consequent recovery of the conducting tissues ; subsequently, as impulse after impulse is transmitted, these tissues show greater and greater fatigue, until eventually a stimulus fails to pass. A beautiful example of similar events is portrayed in Fig. 28. The analysis proceeds along parallel lines to that just given and is portrayed by the diagram of auricular and ventricular contractions ruled upon the figure ; but the fatigue comes more slowly and only two ventricular silences are to be seen. Fig. 27 has a companion curve, namely Fig. 26. The two curves were taken from the same patient. Fig. 26 was obtained shortly after exercise ; it is noteworthy that all ventricular irregularity was abolished by this exercise, while the auricular rate was somewhat increased. The interpretation of this curve is not evident at first sight ; R and S are readily identified,

but T seems double. The first of the twin peaks represents an auricular systole in each cycle ; this is known by measuring the interval between R and the summits in this figure and in Fig. 27. The relations are the same as those shown by P^8 and T^5 of the second figure. Thus the P-R interval throughout Fig. 27 shows great prolongation ; the auricular systoles do not fall in presystole or even in early diastole, but they coincide with the preceding ventricular systoles. In human electrocardiography it is almost always safe to assume that, if T shows bifurcation, it is complicated by an auricular complex ; and this general statement applies equally to individual T's and to a series of T's so deformed. Fig. 29 also shows partial heart-block. Here there is no difficulty in picking out the auricular contractions, for they have the form that they so commonly assume in mitral stenosis. In the earliest part of the figure the response to each third auricular beat fails ; in the last part of the figure what is known as 2 : 1 heart-block, in which alternate auricular impulses alone yield responses, is present. The figure demonstrates the same variation in P-R intervals which has been referred to in discussing other figures. A long diastole is followed by a short P-R interval and a short diastole by a long one, according to the preceding period of rest.

Partial heart-block is of much clinical importance, though it is not within the scope of the present book to discuss this standpoint. This has already been done in *Clinical Disorders of the Heart Beat.* As a permanent condition it tells us of chronic myocardial damage ; occurring in patients who complain of symptoms, such as fainting attacks, breathlessness or sternal pain, which may or may not be consequent upon cardiac mischief, it helps materially in arriving at a correct conclusion. As a temporary event, it speaks of an acute heart lesion or poisoning. It will be

sufficient if I briefly relate the histories of the curves now presented. Figs. 24 and 25 were taken from a young boy admitted to hospital for subacute cystitis, resulting from an infection by an organism of the *coli* group. While lying in bed for observation he developed an occasional irregularity of the pulse to which little significance was attached. When specially examined, the heart-block was discovered, and it became clear that the heart was invaded, either by the organism itself, or by the products of its digestion. Appropriate treatment, applied to the bladder, was followed not only by subsidence of the cystitis, but eventually by a restored cardiac action.

Figs. 26 and 27 were taken from a young girl who had experienced an attack of acute rheumatism a year before. The girl sought advice for pains in the neck and chest, and a slight irregularity of the pulse was noticed at her preliminary examination, while she lay quiet. The special examination revealed the disordered heart action that has been described. What was its significance ? Partial heart-block appearing in cases of early rheumatic heart disease is one of our few reliable signs of invasion of the cardiac muscle. Chronic rheumatic heart disease, in its fully developed form, is not made in a day ; thickened mitral cusps and hypertrophic degenerate muscle take years to develop. They develop, to my mind, as a result of repeated infections, often slight and passing unnoticed. It is along these lines that we may read the curves (Figs. 26 and 27) ; they form evidence of a slight but acute cardiac muscle lesion, a lesion that is but one step in the course of what may become a chronic and incurable malady. The picture is not an uncommon one, and often it is combined with a clear source of infection ; the channel, may be the throat, is found and sealed.

The electrocardiogram in this instance took us further ; while the patient stood or gently exercised in the recumbent

position, the pulse was regular (Fig. 26), and there was no trace of diastolic murmur. Such was the condition when she was first seen. When the pulse was irregular a murmur was audible from time to time. The characteristic murmur of early stenosis of the mitral orifice was heard only when the heart's action was such that the auricular and ventricular systoles lay separate. When, as in Fig. 27, the contractions were simultaneous and the auricle forced no blood into the ventricle the murmur vanished. These electrocardiograms, therefore, not only explained the nature of the heart irregularity, its reaction to posture and exercise, which was at first so puzzling, but they prompted the search for the characteristic murmur during the irregular heart periods ; finally, they told clearly of an acute invasion of the cardiac muscle, in a child, up and about and otherwise seemingly almost well.

The curve shown in Fig. 29 was taken from a case of mitral stenosis in which digitalis was being administered. This drug frequently produces heart-block and that which is shown in the illustration resulted from poisoning. The electrocardiogram demonstrated that the patient was fully under the influence of the drug.

Complete heart-block.

Partial heart-block is an unstable condition ; it passes from one grade to another. In this it contrasts with complete heart-block, which is the next disorder to be considered. We have seen that partial heart-block may be present while the ventricular action is regular (Fig. 24) or irregular, and while it is rapid (Fig. 26) or slow (end of Fig. 29). Complete heart-block is generally associated with a perfectly regular and slow ventricular action. The auricles beat at their usual rates and regularly, the ventricles also beat
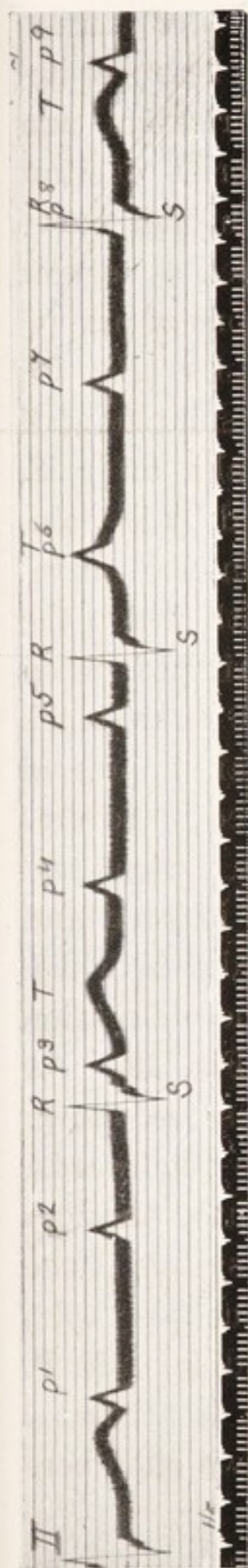
Fig. 30. A curve of complete heart-block, or dissociation of the auricular and ventricular rhythms. The auricles and the ventricles are beating regularly, but at the independent rates of 78 and 29, respectively. The time-marker shows fifths and thirtieths of a second. The exact algebraic summation of auricular and ventricular effects should be remarked.

Fig. 31. Showing complete heart-block in another case. The rates of auricle and ventricle are approximately 86 and 35, respectively. Note that each ventricular complex begins with Q, showing that the latter belongs to the ventricular systole.
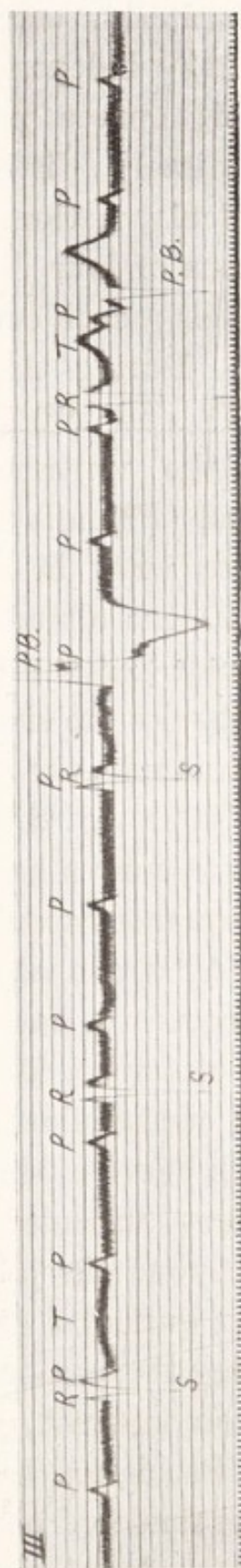
Fig. 32. From a case in which there was considerable enlargement of the heart towards the left side. The curve shows complete heart-block. The regular ventricular action is disturbed by two premature contractions (*P.B.*).

rhythmically, usually at a rate of about 30 per minute and quite independently of the auricles. The electrocardiographic curves portray the condition to perfection and immediately distinguish it from all other forms of slow ventricular action. Fig. 30 is an example ; in this curve portions of four ventricular beats are represented at regular intervals ; each consists of R, S and T deflections of normal form. During the diastoles evident P summits (P^1, P^2, P^4, P^5 and P^7) appear. The remaining P summits fall with the ventricular systoles. One (P^3) falls after S, another (P^6) falls upon T. One (P^8) is obscured by its coincidence with R. They occur equidistantly in the curve. Fig. 27 has already indicated the accuracy with which the auricular and ventricular electrical effects superimpose when the systoles are simultaneous ; it is in complete heart-block that this rule, the constancy of which will be again impressed from time to time, is so strikingly displayed. Several separate curves have been taken from the same patient and the separate ventricular complexes have been isolated and rearranged one above the other, not in the order in which they occurred, but in the order that displays the summation most clearly. The rearrangement is seen in Fig. 33. A single ventricular and two auricular complexes are shown in each curve of this figure. Traced from above downwards, the first auricular summits pass gradually into, through and beyond the opening phases of ventricular systole ; the second auricular summits of the curves continue the tale, showing the passage over and clear of the broad summit T. In each instance, where it occurs, summation is accurate. The dissociation between the rhythms of the two chambers is thus rendered obvious.

The electrical curves of complete heart-block teach us more than that the heart is generating two distinct rhythms.

The slower, or ventricular, rhythm is represented by curves of perfectly normal outline (Fig. 30). The contractions are awakened therefore through the normal channels. The channels that carry the normal impulses from auricle to ventricle also convey the impulses of the independent ventricular rhythm. The impulses are of supraventricular origin. Yet these impulses do not arise in the auricle, which is contracting independently under the influence of the sino-auricular node. The new ventricular impulses arise in tissue lying between the bundle lesion and the division of the bundle into its right and left branches.

Fig. 31 demonstrates that Q is a ventricular effect. The curve is again one of complete heart-block, the auricular and ventricular rhythms being independent; Q is constantly associated with the ventricular and not with the auricular contraction; the same fact is established by Fig. 28.

In complete heart-block deviations of the ventricular complexes from the normal outlines are to be interpreted according to the usual rules, as these apply to hearts beating with the normal sequence of

Fig. 33. A figure constructed from the electrocardiograms of the patient, whose curve is shown in Fig. 31. Single ventricular complexes have been re-arranged above each other so as to display their varying relation to the auricular complexes.

chamber contraction.   Fig. 32 illustrates the third lead in a
case of complete dissociation ; the ventricular curves indicate
preponderance of the left ventricle.   The same figure is com-
plicated by beats of the ventricle that occur before the
anticipated points.   They are premature beats (*P.B.*) or extra-
systoles, the nature of which will be described more fully in
the next chapter.   The auricular summits are traceable
throughout the whole curve, whether they fall with the supra-
ventricular or anomalous form of ventricular curve.

Slow action of the ventricle.

In partial heart-block when each second, third, or
fourth auricular systole stimulates the ventricle, and when,
too, the auricular rate is normal, the ventricular action is
necessarily slow.   The ventricular action is also slow, as
has been seen, in complete heart-block.

It is necessary that slow ventricular rhythms of these
types should be clearly distinguished from others in which
the heart's action is different.   To take the most familiar
examples first, when the ventricle beats slowly in jaundiced
patients or in those that are convalescent from acute
infections, the retardation results from a similar slow action
of the auricle.   The rhythm of all chambers is slow, but the
natural sequence of chamber beating is maintained.   Such
slowing may result from high vagal tone, and the heart then
quickens in response to effort or such drugs as atropine ; the
quickening is *gradual.*   To the same class belongs the slow
heart action of many healthy adults.   All these true brady-
cardias exhibit an electrocardiographic picture similar to that
shown in Fig. 34 ;   each ventricular beat is preceded by a
single auricular beat and both are of the normal type.

There is another, rare, and special form of slow action
that involves the whole heart and in which chamber sequence

is also undisturbed. On rare occasions patients are observed in whom the rate of the ventricle is persistently maintained at between 30 and 40 beats per minute while they rest. The hearts referred to react in a curious way to exercise ; the acceleration is not gradual, there is an abrupt passage of slow into fast rate from one heart cycle to the next. The curve that is shown was taken from an athlete ; the rate of contraction, while at rest, was usually 36 per minute ; the first effect of exercise was an abrupt and approximate doubling of rate ; further acceleration was gradual ; similar events, though ordered reversely, were observed at the cessation of effort. This action of the heart will be spoken of again in a later chapter ; it belongs to a series of phenomena that are at present classed under the term " sino-auricular block."

When the auricular action is slow and the ventricle is starved of its full quotient of impulses, the last chamber frequently develops the same automatic action as is to be observed in complete heart-block ; so that although there is no fault in conduction, yet two impulse centres, auricular and ventricular respectively, are active and give rise to responses when opportunity offers. The auricular action is generally the dominant one, but if its rate falls below a certain point or if the automatic rate of impulse formation in the ventricle rises above the same point, then the last named chamber beats spontaneously. Generally this event is recognisable at once in electrocardiograms ; an example is shown in Fig. 35. In the first four cycles of this figure, the chambers are beating sequentially and slowly at a rate of 50 per minute. The fifth cycle shows a ventricular complex of the usual form, but this beat is not a response to an auricular contraction, the *P-R* interval is curtailed and there is partial coincidence of the chamber contractions. This coincidence is more

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Chapter IV.
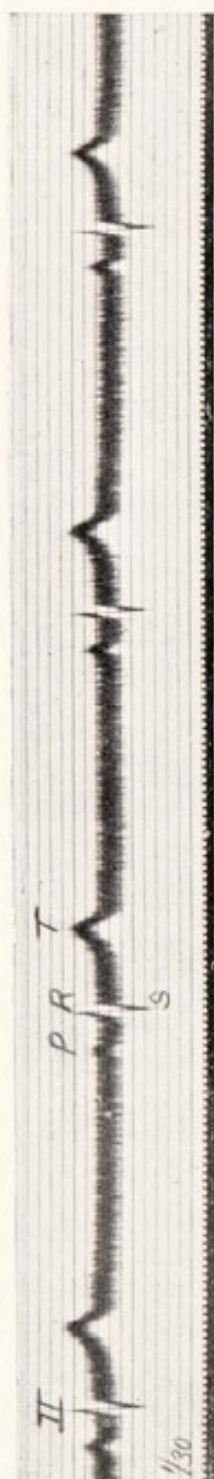
Fig. 34. An electrocardiogram taken from an athlete while at rest, and showing a regular action of the whole heart at 36 per minute. With moderate exercise the rate suddenly doubled. The original slow action probably resulted from sino-auricular block.
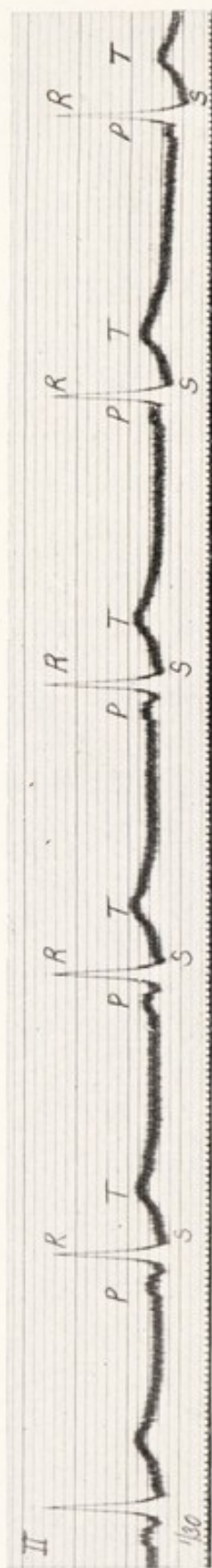
Fig. 35. A curve showing slow action of the whole heart, the rate being 50 per minute, with escape of the ventricle.
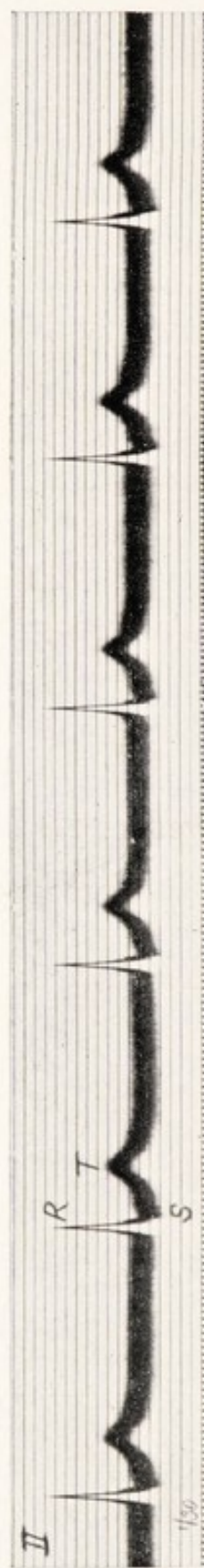
Fig. 36. A curve showing regular and slow action of the ventricle at 48 per minute. No trace of auricular complex is to be found and the line is perfectly smooth in diastole. These curves usually result from simultaneous contraction of auricle and ventricle (nodal rhythm).

pronounced in the last cycle of our illustration, where the
P-R interval is minute. Now each ventricular complex
is of normal outline ; so also is each auricular complex, so far
as it is visible ; the ventricular beats are therefore all of
supraventricular origin, while the auricular beats are all
derived from the natural pacemaker. Yet the ventricular
beats originate in distinct ways ; the first four are of supra-
ventricular origin in virtue of their response to auricle ; the
last two are of supraventricular origin in virtue of the impulses
formed in the junctional tissues, the usual site of ventricular
automatism. When the ventricle originates a rhythm of
its own it is this centre that is active ; the ventricular beats
are termed " escaped " ventricular contractions when they
are isolated or in short groups. The bundle is not the only
new centre that may come into play when the natural
pacemaker is sluggish ; other regions occasionally usurp the
function of the natural pacemaker and capture the lead ; the
centre that produces successive impulses most rapidly is
always responsible for the dominant rhythm. If the escape of
the new centre is continued, the rhythm of the whole heart
becomes controlled from that centre ; this has happened in
Fig. 36. The figure shows ventricular complexes only and a
regular action at 48 per minute. The auricular representa-
tives are not seen. Such electrocardiograms are found
when auricle and ventricle contract simultaneously in response
to impulses created in the neighbourhood of the auriculo-
ventricular node ; the mechanism is consequently termed
" nodal rhythm." When the ventricle contracts in response
to impulses derived from this centre, its electrical complexes
are naturally of the supraventricular type. On the other
hand, the auricular complexes are abnormal, for the auricular
contraction wave is propagated in an abnormal direction.
Having an abnormal form and falling at the same time

as the ventricular contractions, they cannot as a rule be identified in the curve, which therefore shows ventricular beats only. Another form of " nodal rhythm " is shown in Fig. 63, where there is a simple shortening of P-R interval and an inversion of the auricular complex.

Escaped beats of the ventricle or auriculo-ventricular node are without clinical significance ; they are nearly always dependent upon relatively slow action of the natural pacemaker, and it is to this that attention should be directed. Yet it is desirable that they should be recognised when seen, and they are generally clear in electrocardiograms.

CHAPTER V.

PREMATURE CONTRACTIONS OR EXTRASYSTOLES.

Premature contractions, or extrasystoles, are responsible for the majority of those pulse disturbances which are included in the term "intermittence." They are abnormal contractions of the heart, that generally spring from some region of the musculature other than the normal pacemaker or sino-auricular node. I distinguish beats that arise from abnormal foci by the adjective *ectopic*. A premature beat is characterised by appearing before the expected natural beat, which would continue the regular rhythm of the heart. There are two chief classes of such beats—those that arise in the ventricle and those that arise in the auricle. Both forms are readily identified in electrocardiograms. A third type arises in the tissues that lie between auricle and ventricle.

Premature contractions of ventricular origin.

When the rhythm of the heart is disturbed by premature ventricular contractions (or ventricular extrasystoles), the disturbance of rhythm is as a rule limited to the ventricle ; the auricle continues to contract at the expected instants. The premature ventricular beats are easily recognised. The electrical complexes representing them are known to be ventricular, because they have the same duration as the normal ventricular complexes in the same subject ; but they precede and usually replace the regular responses of the
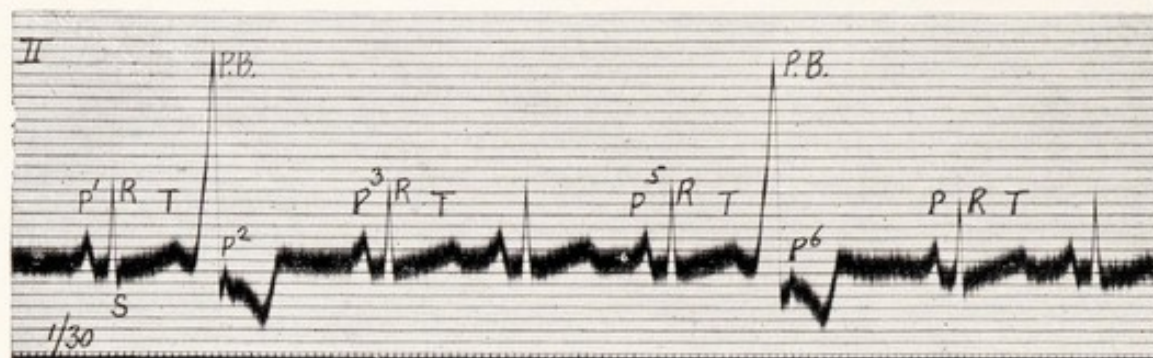
Fig. 37. An electrocardiogram showing two premature contractions, arising in the right ventricle. The curve shows two buried auricular complexes of normal outline. These premature contractions are responsible for the commonest forms of intermittent pulse (see Fig. 27).
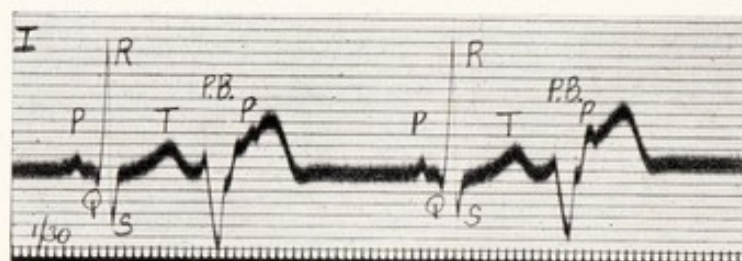
Fig. 38. Premature contractions arising in the ventricle, replacing the alternate normal ventricular beats and giving rise to one form of bigeminy of the ventricle and pulse. The figure illustrates the manner in which buried auricular complexes may be identified.
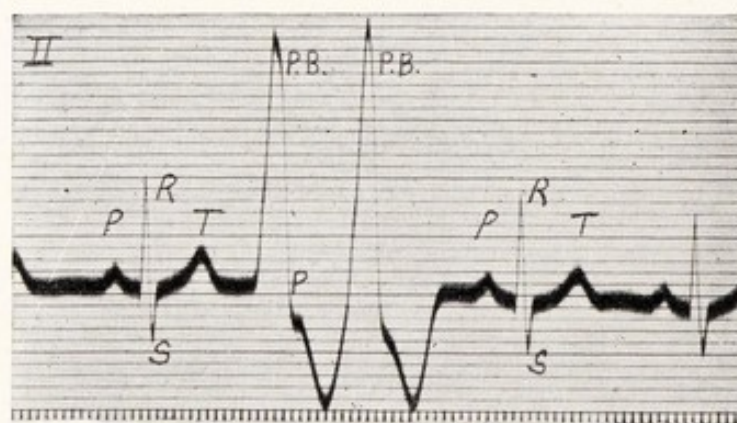
Fig. 39. Two premature contractions arising in the right ventricle. They occur together and replace a single normal ventricular contraction. The rhythmic auricular contraction falls with the first premature beat.
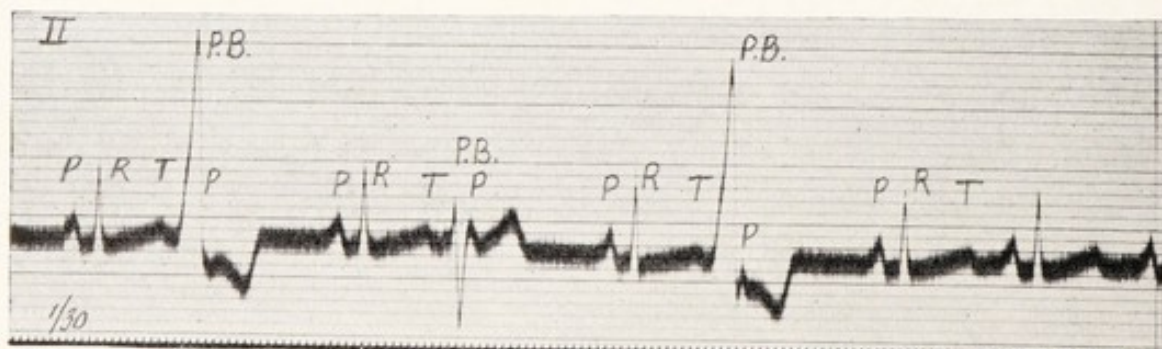
Fig. 40. Bigeminy of the ventricle, resulting from premature contractions of the ventricle. The premature beats arise from two separate foci.

ventricle, thereby disturbing the rhythmic action; their form is anomalous. These points are illustrated by Fig. 37. The figure begins with a perfectly normal heart cycle (P, R and T); this is followed by a tall and pointed summit ($P.B.$), which in turn is followed by a broad downward deflection. The total length of this anomalous complex is equal to that of R and T in the normal cycles. Succeeding the disturbance are three regular cycles and the disturbance is repeated. Now the premature beats of this figure are spontaneous and arise in the ventricle, for no auricular contractions precede them; they are *ectopic* in origin. The auricular rhythm is undisturbed; the distance between P^1 and P^3 is exactly equal to the distance between P^3 and P^5. Where then is P^2? It lies embedded in the complex of the premature beat. That this is so is ascertained by comparing the two premature beats of the figure; when the second occurs, the auricular rate has become a little slower and consequently the buried auricular complex (P^6) lies a little more to the right, relative to the corresponding premature beat, than does P^2. The two premature beats give complexes of exactly the same form, the only difference between the two curves is engendered by the auricular complexes that fall with them; this difference is slight, and is due to the time relations of the several events. The buried auricular beats being recognised, the curve as a whole may be analysed fully. The auricular systoles are represented throughout; they occur in regular sequence. After each premature beat, the ventricle is silent; it is waiting for the next auricular impulse, to which it will respond. The long diastole, which thus compensates for the prematurity of the abnormal ventricular contraction, is spoken of as the " compensatory pause." All the auricular complexes of this figure are of normal type, for all have arisen at the pace-

maker ; all but two of the ventricular complexes are of normal
type, for they have originated in impulses traversing the
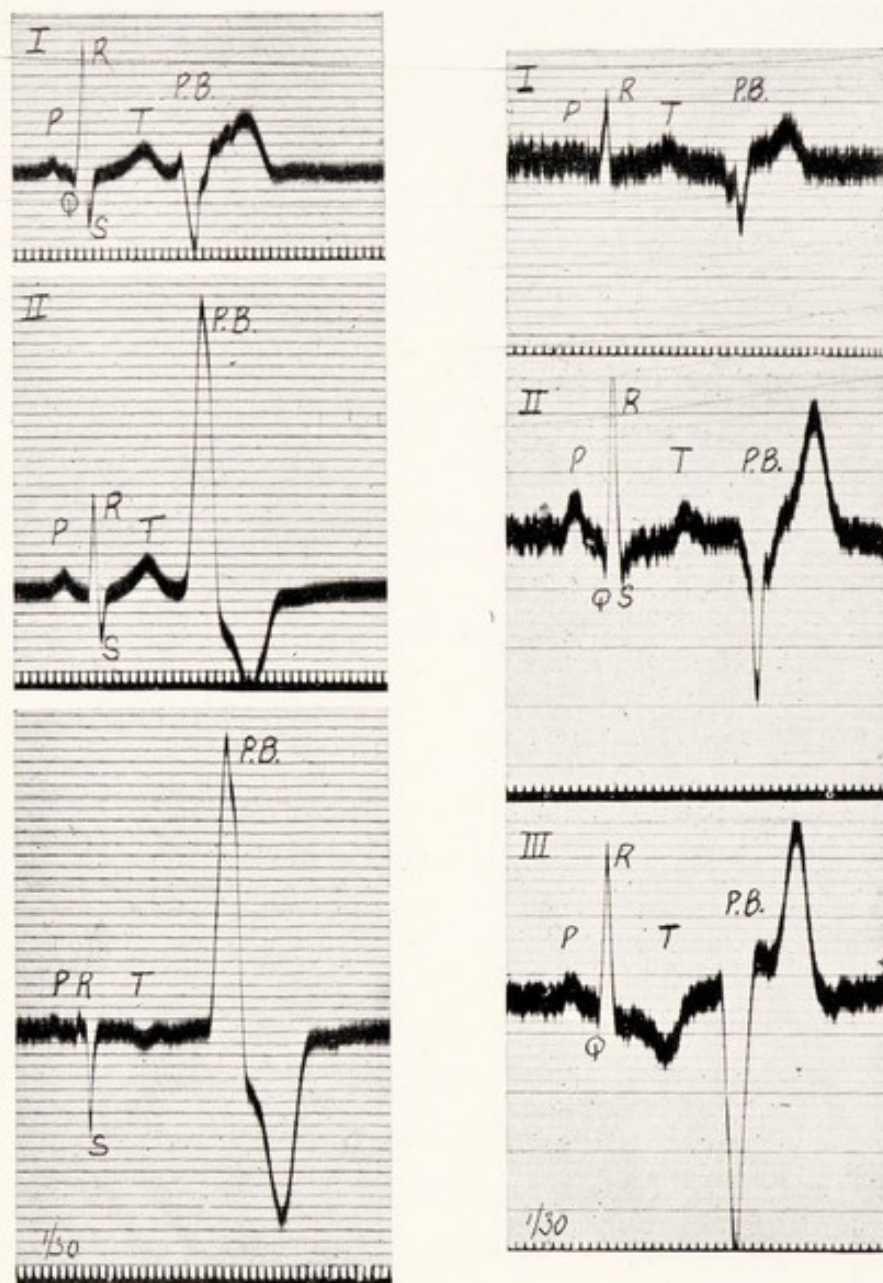
Figs. 41 and 42. Figures illustrating the two chief types of premature
  contractions of ventricular origin as they are portrayed in the separate
  leads. Fig. 41 shows a premature beat arising in the right and Fig. 42
  in the left ventricle.

normal paths. Two of the ventricular complexes are
anomalous in outline, showing that the corresponding
excitation waves ran abnormal courses ;   the course was

abnormal because the starting point of the wave was abnormal.

Premature ventricular contractions in different subjects yield electrical curves of very different forms independently of the lead from which the curves are derived. The pictures are also different in different leads in the same individual. In interpreting the curves they may not be treated pictorially, each should be analysed in detail and upon the general lines that I have here summarised. Another curve is shown (Fig. 38) illustrating the manner in which buried auricular contractions are discovered. The premature beats show two main deflections, one pointed and directed down and the other rounded and directed up. The shoulder of the rounded upstrokes is modified by a superimposed auricular representative (P) in each case, but it falls at slightly different points relative to the two premature beats. Two successive premature beats are shown in Fig. 39. They take the place of a single normal ventricular contraction. The single rhythmic auricular complex, which is buried, falls upon the downstroke of the first premature beat. If the two beats are compared, they will be found to differ chiefly in this respect. Premature beats from separate ventricular foci are shown in Fig. 40. Two are of the same form as those of Fig. 37; the third or central one consists of three deflections, a short spike, a deep and pointed depression and a rounded summit. The auricular contraction, which falls between the last two, is prominent and unmistakable.

There are two chief types of premature contractions of ventricular origin; one comes from the right ventricle (Fig. 41); the other comes from the left ventricle (Fig. 42; see Fig. 45 also). In any given case the shape of the electrical curves is usually very similar in leads *II* and *III*, but as a rule the direction of the deflections is reversed in lead *I*
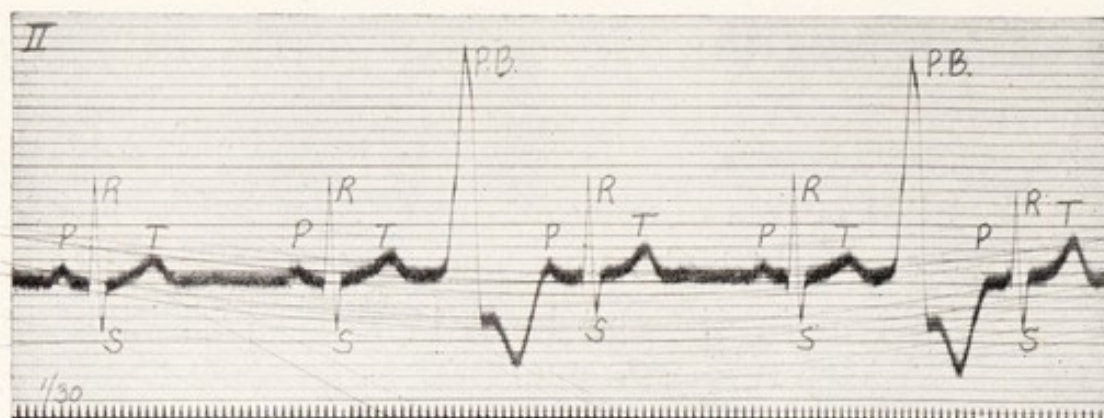
Fig. 43.   Premature contractions arising in the right ventricle and interpolated
between normal heart cycles.
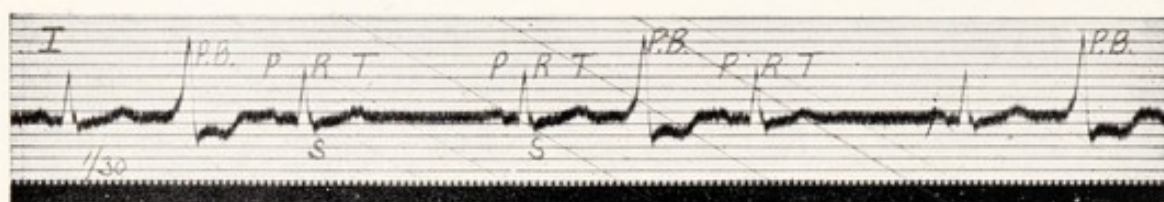
Fig. 44.   Interpolated premature contractions of ventricular origin.
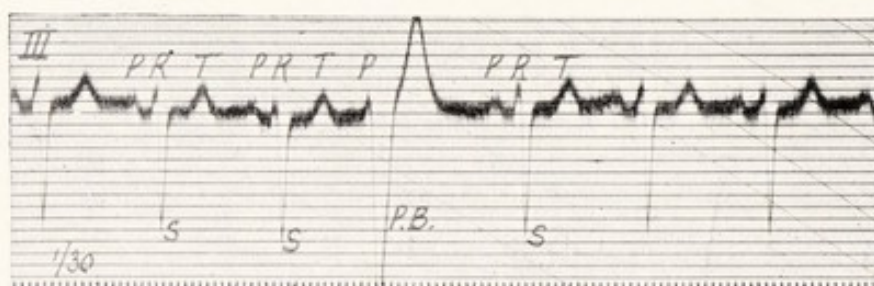
Fig. 45.   Premature contraction arising in the left ventricle.
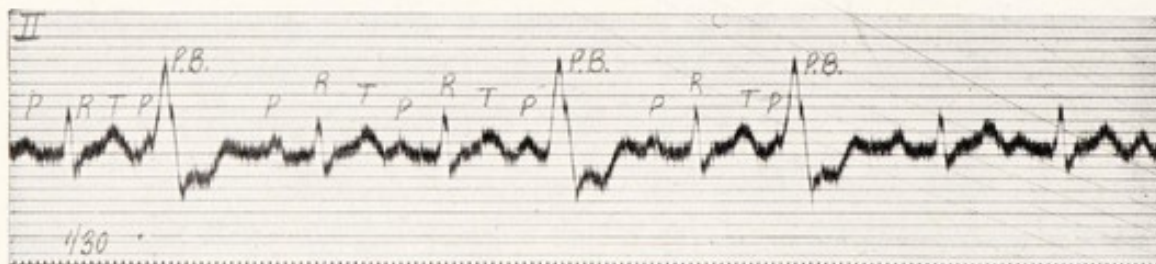
Fig. 46.   Premature contractions arising in the ventricle, late in diastole.
The auricular systole has begun, and in one instance is almost complete,
before the extrasystole of the ventricle begins.
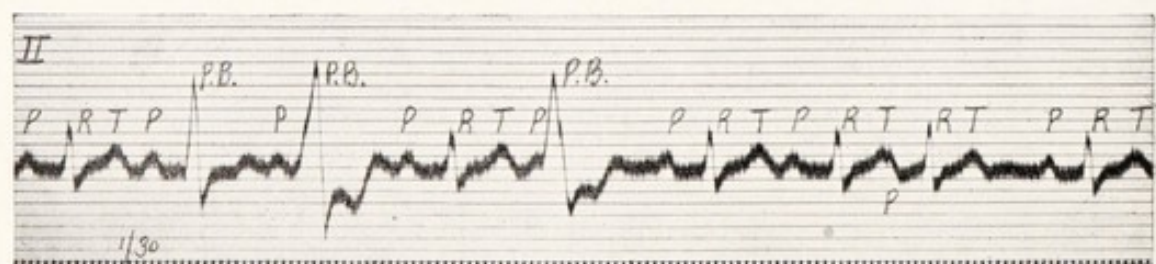
Fig. 47.   From the same patient as Fig. 46, showing interference between
two waves of contraction in the ventricle (see text).   A premature
contraction of auricular origin is seen towards the end of this figure.

(Fig. 41), though this is not invariably the case (Fig. 42). The complexes of extrasystoles arising in the ventricles are not dissimilar to those seen in the lesions of the bundle divisions. In experiment stimulation of the right ventricle (Fig. 15 at A) yields curves similar to a lesion of the bundle branch at A^1; that is natural since in both instances the wave is propagated from the same region of the heart. Similarly, stimulation of the left ventricle (Fig. 15, at B) yields curves which resemble those following a lesion of the bundle branch at B^1.

Although premature contractions of ventricular origin are generally followed by "compensatory pauses," this only happens when the rhythmic auricular impulse finds the ventricle already in contraction. The ventricle is then "refractory" to stimulation. This is the reason why there is no response to buried auricular contractions, such as are seen in Figs. 37 and 38. But if the heart is beating slowly and the extrasystolic beats of the ventricle are very premature, the extra ventricular beat may have terminated before the next auricular impulse arrives, and this yields its usual response. In such circumstances the premature beat does not replace a normal ventricular event, but is an added phenomenon. Such beats are called "interpolated extrasystoles" (Figs. 43 and 44).

When a premature contraction falls very late in diastole, the disturbance of ventricular rhythm is slight, for it happens at an instant close to that at which a rhythmic beat is expected. The auricle may even contract before the premature beat of the ventricle begins, in which case there is an appreciable though shortened interval between the auricular beat and the premature one. The origin of the latter is nevertheless clearly shown by the shape of its electrical complex. An example of this phenomenon is seen in Fig. 46. Supposing,

however, that the premature beat comes so late that an
auricular impulse is already well on its way to the ventricle,
then two waves of contraction, one propagated from the
normal source and the other propagated from the ventricular
source, travel over that chamber and meet somewhere in
its walls. The electrical complex of the ventricular contraction
will then be of transitional form. Figs. 46 and 47 were taken
from the same patient. A usual form of anomalous beat is
seen in Fig. 46. Each of the three premature contractions
of this figure produce similar electrical curves ; each falls late
and after the auricular contraction has started. The first
premature beat of Fig. 47 comes still later in diastole ; the
interval between it and the preceding auricular beat is just
perceptibly less than the normal *P-R* interval. The resultant
ventricular curve has a distinctive form, transitional between
the normal and the abnormal electrical complex. Such a
contraction results when simultaneous but independent
excitation waves meet in the ventricular walls.

Premature contractions of auricular origin.

When a new impulse arises in the auricle, the rhythm of
both auricle and ventricle is disturbed. Usually the premature
auricular contraction awakens a response in the ventricle.
A simple example of the auricular extrasystole is shown in
Fig. 48. The first and second cycles of the figure are normal,
consisting of *P, Q, R, S* and *T* deflections. The third cycle
is premature. Consider first the ventricular complex of
this premature beat. It has a perfectly normal outline. The
impulse responsible for it is known therefore to have travelled
along the normal channels ; that is to say, it has come down
through the auriculo-ventricular bundle and its branches.
It is a ventricular beat of supraventricular type. When
we search for the cause of this ventricular beat, we find

it in an auricular contraction that precedes it. But this auricular beat is of abnormal type, the electrical curve shows a depression and not a summit. To what is the inversion due? It results from the abnormal path taken by the excitation wave in the auricle. The impulse,
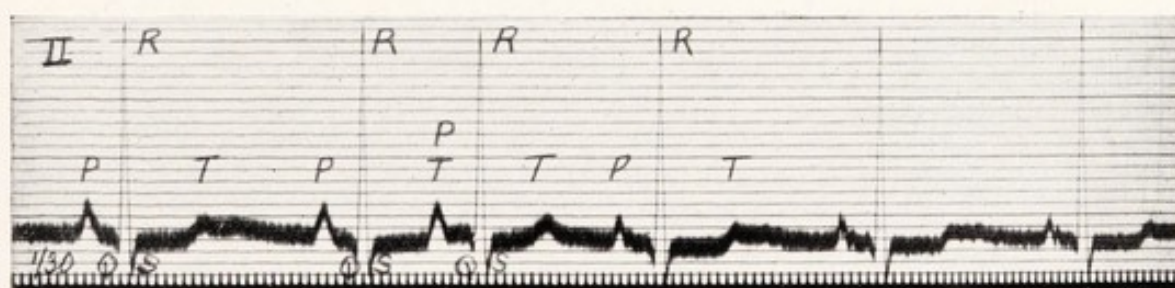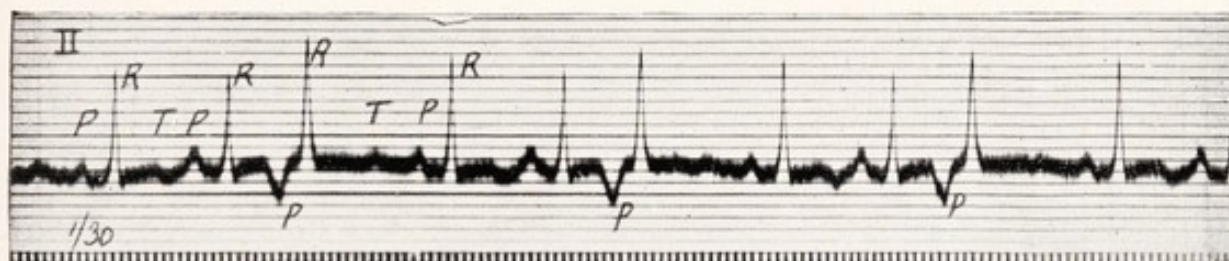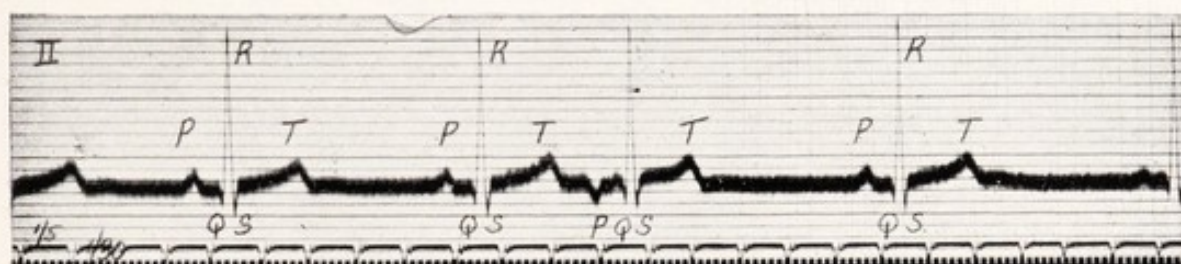
Fig. 48. A single premature contraction of auricular origin. The auricular complex is alone abnormal.

Fig. 49. Three isolated premature contractions, arising in the auricle. The corresponding auricular complexes are abnormal, and there is also slight distortion of the corresponding ventricular complexes (aberration).

Fig. 50. A single premature contraction, probably arising in the immediate neighbourhood of the sino-auricular node.

starting the whole premature heart cycle, has been generated in an abnormal auricular focus, *i.e.*, in a region lying at a distance from the pacemaker. It has arisen from an *ectopic* centre and the wave has consequently passed

through the auricle in a direction other than normal ; but
having arisen in the auricle, it travels to the ventricle along
the only path open to it, namely, that constituted by the
normal channels.   Usually, the diastole following a prema-
ture auricular contraction is not compensatory ; it is short
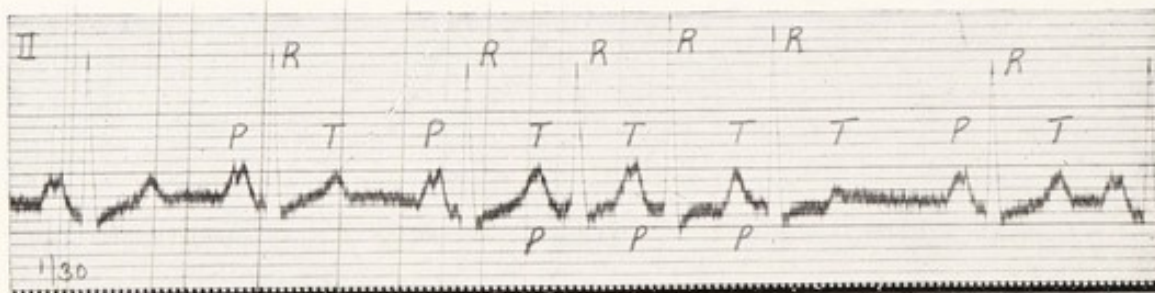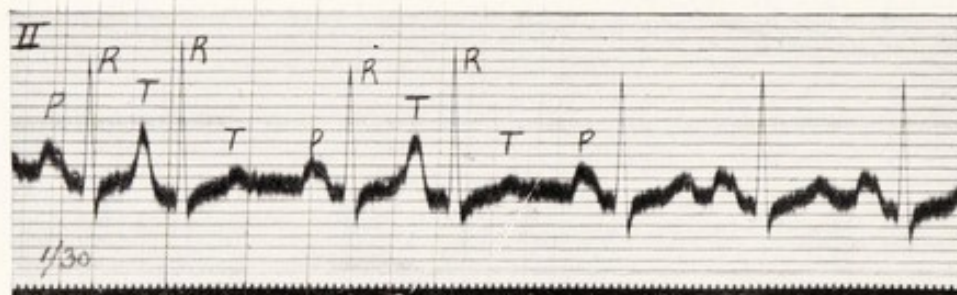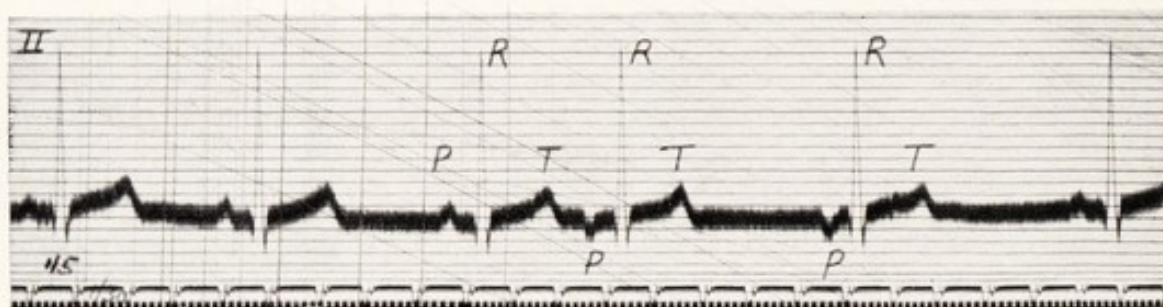
Fig. 51.   A single premature contraction of auricular origin.   The first
    beat after the pause originates in the same point as the premature one.

Fig. 52.   Two isolated premature contractions of auricular origin.   The
    premature auricular systole coincides with the preceding ventricular
    contraction ; P and T are thus superimposed.   Taken from a case of
    mitral stenosis.

Fig. 53.   Three successive premature contractions of auricular origin.   Taken
    from a case of mitral stenosis ; note the split P summits in this and the
    preceding figure.

of this, and consequently the fundamental rhythm of the
heart suffers disturbance.   A rare example of premature

auricular contractions, in which this diastole is almost if not quite compensatory, is shown in Fig. 49. The auricular complexes are inverted in this figure, as in Fig. 48. When a premature beat arises in or near the sino-auricular node, then the whole premature electrical complex is of normal form, as shown in Fig 50 ; the P summits are similar throughout, showing that the beats originate similarly ; moreover, the diastole that follows the premature beat is unduly shortened ; its length is either the same or somewhat shorter than that of the usual rhythmic beats.

On all but rare occasions the beat following a premature contraction is derived from the pacemaker ; the old heart rhythm is immediately restored (Fig. 48) ; but it sometimes happens that the first beat of the returning rhythm is also ectopic (Fig. 51) and springs from the same centre as the extrasystole. A not dissimilar disturbance, but of higher grade, is shown in Fig. 63.

When new impulses are created in the auricle, the premature auricular contraction may fall so early as to coincide with the preceding ventricular systole. P and T then fall together and summate. Summation of these summits is seen in Figs. 49 and 50, but is clearer in Fig. 52, where P and T are both prominent. The second and fourth beats of the last named figure are premature and arise at an ectopic auricular focus. The P summit of the premature beat coincides with the T summit of the preceding cycle and combines with it to form a tall blunt summit. Simple superimposition of P and T would not yield a united summit of the type depicted, for the rhythmic P is bifid in this case ; it is known, therefore, that the premature beats were ectopic in origin.

An example of premature auricular contractions, occurring in succession, is shown in Fig. 53. Each premature auricular

complex coincides with a preceding T summit. All told, there are three of them.

While it is the general rule that the ventricular complex of a premature auricular contraction is of normal outline,
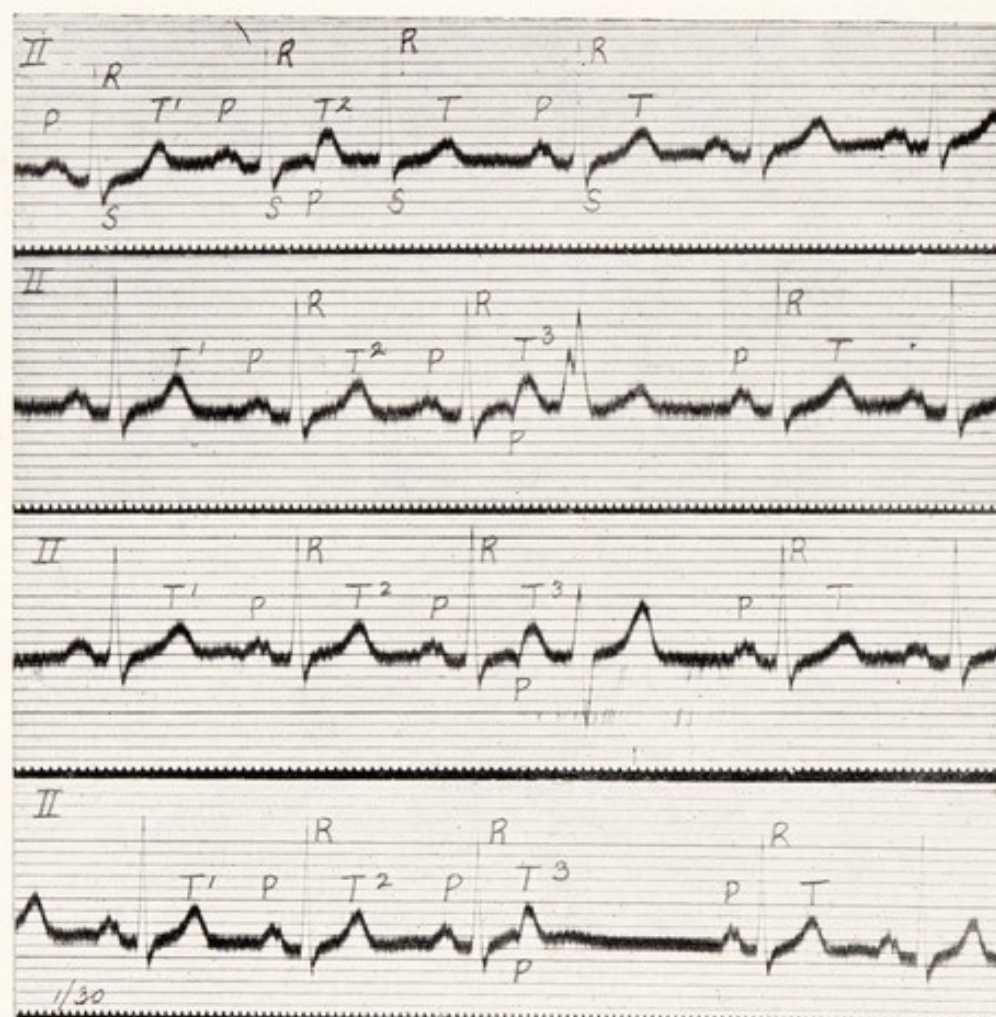
Fig. 54. Four curves from a single subject. Each shows a solitary premature auricular contraction. The premature auricular complex falls with the commencement of the preceding T and notches it. The corresponding ventricular complexes of the first three curves are of various forms; the central curves illustrate a phenomenon which is described under the term "aberration." In the last curve the premature auricular contraction is blocked.

yet its form often diverges; it may diverge so conspicuously as to be mistaken for a beat of ventricular origin. The premature R summits of Figs. 49 and 52 are taller than those of rhythmic beats; but the difference in general outline is but slight. Examples of the chief forms of conspicuous divergence

are illustrated in Fig. 54, which consists of four strips of curve taken from the same patient. In the first curve is a premature auricular contraction that conforms to the general rule ; all the ventricular complexes of this strip are similar ; the premature beat (the third in the curve) is evidently of supraventricular origin. The abnormal auricular complex notches the upstroke of the preceding T summit ; this is appreciated when T^1 and T^2 are compared ; they have not the same shape, and the difference is due to an inverted P falling with T^2. The second and third curves show precisely similar events, T^3 is notched by an abnormal auricular
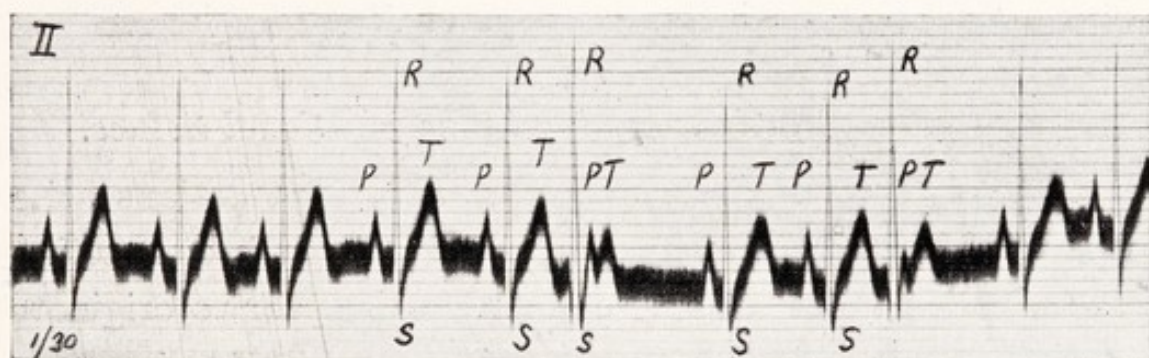
Fig. 55. Two premature contractions arising in the junctional tissues.

representative in both instances ; but in these curves the premature auricular contractions give rise to ventricular beats of anomalous type. In the second curve the complex consists of two upwardly directed summits, the first of which is bifid ; in the third curve upward and downward deflections of equal extent are followed by a rounded summit. Both the curious beats were of supraventricular origin, for each followed an auricular systole that is detectable in the curve ; the abnormality of the ventricular curves is attributed to deficient conduction along certain tracts of the junctional tissues, whereby the auricular impulse has pursued an abnormal course in passing to the ventricle ; the deficiency

F

has been confined to the premature beats because the rest preceding these has been brief. These electrical curves exemplify aberration (see page 28). Auricular extrasystoles, showing aberrant ventricular complexes, are almost confined to hearts in which conduction defects in the junctional tissues are demonstrable. The conduction intervals of the premature beats are prolonged in the second and third curves of the present figure; and in the fourth or last curve a premature auricular contraction is blocked; T^3 is again notched by an inverted P, but the auricular systole that the latter represents is not followed, as in the other curves, by a response of the ventricle. The last disturbance is often called a " blocked auricular extrasystole."

Premature contractions arising in the junctional tissues.

Although the majority of premature contractions arise in the auricle or in the ventricle, on occasion the conducting tissues, which unite these two chambers, appear to originate the disturbance.

There are two premature ventricular systoles in Fig. 55; and the time relations of the auricular and ventricular contractions in this figure are precisely the same as those seen when the premature beats are of ventricular origin. The pauses are compensatory, and there is no disturbance of the auricular rhythm. The auricular contractions fall regularly throughout the curve; each is represented by a normal P summit, though two of these summits are buried. The undisturbed auricular rhythm demonstrates that the premature beats have arisen below the auricle. Yet they have had their origin above the main division of the auriculo-ventricular bundle, for the ventricular complexes possess physiological outlines; the impulses have been supraventricular. The focus of disturbance, in such cases, is located in

the auriculo-ventricular node or in the main stem of the bundle.

Premature contractions of any form are always easy to recognise in electrocardiograms ; in other graphic records they often produce confusing pictures, and this is frequently the case when they appear in groups. The characters of the pulse may closely resemble those which are associated with grave disturbances such as auricular fibrillation, flutter and alternation. Premature contractions are relatively benign, and as electrocardiography may be the only certain method of differentiating this irregularity from more serious disturbances, it is to be recommended as a final means of deciding in doubtful instances.

CHAPTER VI.

SIMPLE PAROXYSMAL TACHYCARDIA.

Electrocardiography has explained many of the phenomena of paroxysmal tachycardia. In the first place it has proved that these transient attacks of cardiac accelera-tion are not the result of simple disturbances of innervation as was at one time thought. If a heart accelerates in response to exercise, emotion, fever or other such cause,
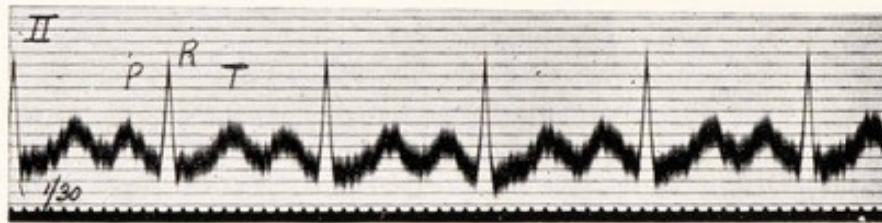
Fig. 56. An electrocardiogram from a case of exophthalmic goitre. The heart rate shown is 150 per minute. The curve indicates a simple acceleration of the normal rhythm.

then the electrocardiogram has in general a physiological out-line. Auricular and ventricular complexes retain their shapes, except for such minor changes as are the simple result of the increased heart rate. Fig. 56 was taken from a case of exophthalmic goitre, the ventricular rate being 150 per minute. The curve shows P, R and T summits; and these conform to the type of heart beat propagated from the natural pacemaker.

The curves of paroxysmal tachycardia are different; they show that the dominant centre of impulse formation has

moved at the onset of the attack, usually to some other part of the auricle. Figs. 57 and 58 are from a case of paroxysmal tachycardia ; each figure presents the three leads. The first (Fig. 57) was taken while the heart action was natural, its rate being 81 per minute. The shapes of the auricular and ventricular complexes in these curves should be noticed and compared with those of Fig. 58, which depicts a paroxysm in which the heart rate was 146 per minute. It is seen that the ventricular complexes of the paroxysm preserve their form and that they do so faithfully. Each ventricular
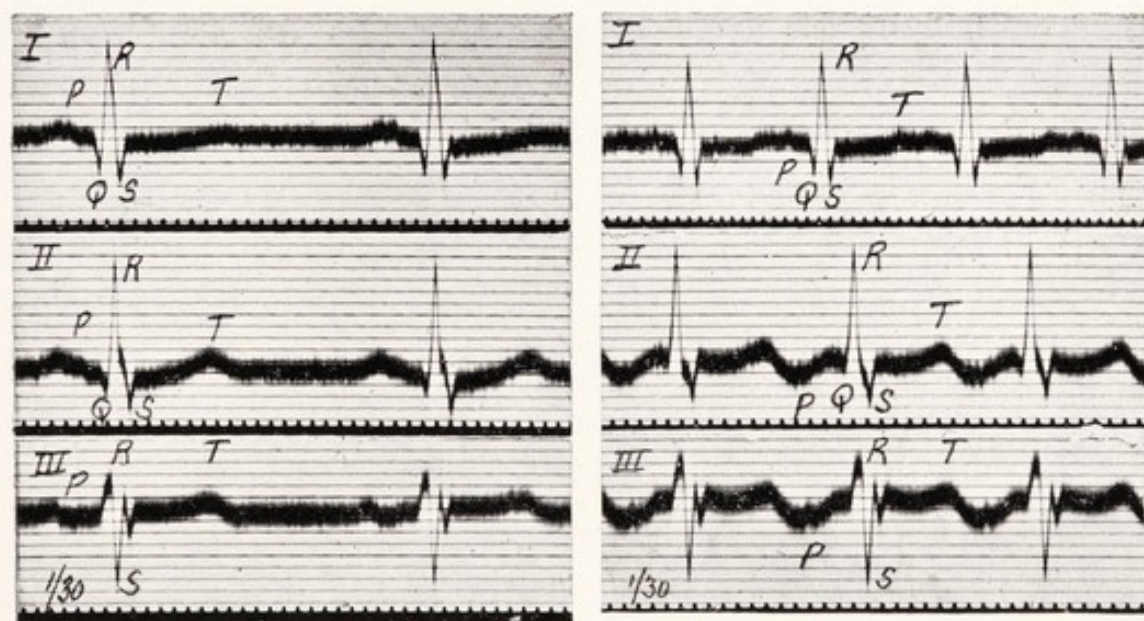
Figs. 57 and 58. Two sets of curves from a case of simple paroxysmal tachycardia. Fig. 57 was taken while the heart beat slowly ; Fig. 58 while it beat rapidly. The curves demonstrate the supraventricular origin of the paroxysm. The inversion of *P* in leads *II* and *III* of Fig. 58 indicates that it arose in an ectopic auricular focus.

summit or depression of Fig. 57 is repeated in Fig. 58. The similarity extends even to the minutiæ of the curves, even to the notch on the downstroke of *R* in lead *II* and the notch on the upstroke of *S* in lead *III*. The ventricular beats during the paroxysm were therefore of the same kind as those of the slow period ; in both phases the impulses pursued the same course ; that is to say, they travelled along the normal paths

of the junctional tissues. The paroxysm was therefore of supraventricular origin. When we examine the auricular complexes in the two sets of curves, it is seen that while the *P* summits of the slow period are upright, those of the paroxysmal period are inverted in leads *II* and *III* and are also different in lead *I*. Such changes are significant, for they tell us that when the paroxysm comes, the natural pacemaker is no longer the dominant impulse centre, but that some new auricular focus has gained an enhanced activity and that the old centre is outpaced. Not infrequently such a conclusion as to the nature of the paroxysm can be reached from its intrinsic beats alone. Yet it is always more justified when the curves of the two distinct phases of heart action can be taken, and when the similarities and dissimilarities that have been named are observed.

The similar nature of paroxysms of tachycardia and solitary premature beats is at once suggested when it is observed that the abnormal beats are of *ectopic* origin. There is in fact no essential difference between the individual beats of the one and the other. Premature beats are not always solitary ; they sometimes occur in short groups (Figs. 39 and 53) ; it is entirely a question of the length of these groups and the nature of our terminology as to whether we term them successive premature beats or short paroxysms of tachy-cardia. In one and the same case, isolated beats, short groups and longer paroxysms may be found, and the electrical curves usually show that all originate in the same focus (Fig. 59). Premature beats are characterised by the relatively short diastoles which precede them ; rapid impulse formation is expressed in the paroxysmal curves by the rate of the heart beat and by the prematurity of the first paroxysmal beat. That the two phenomena are essentially the same is also demonstrated by the manner in which
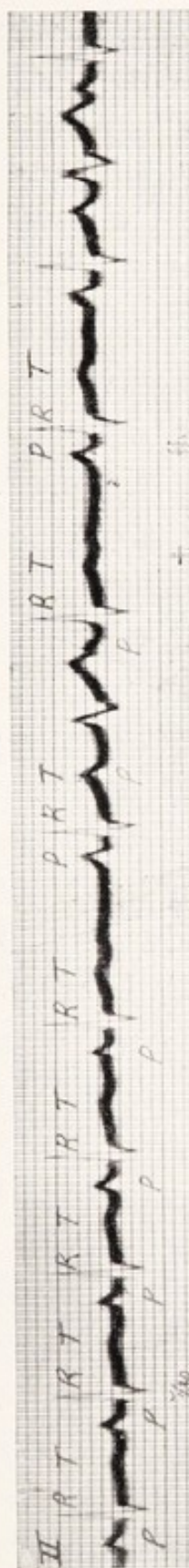
Fig. 59. The end of a paroxysm of tachycardia of auricular origin, and the commencement of the slow normal rhythm, interrupted by premature contractions. The last are auricular and two of them show ventricular complexes of aberrant type. The time marker in this curve rules vertical lines; a pair of lines occurs at each thirtieth of a second.
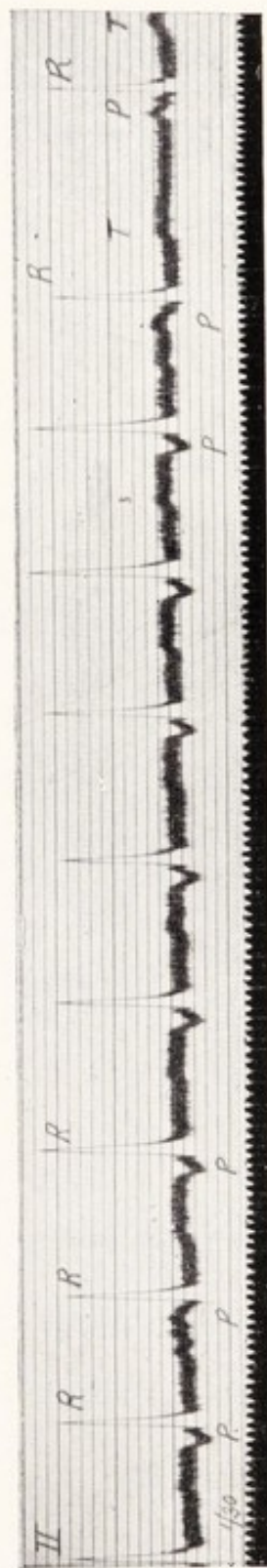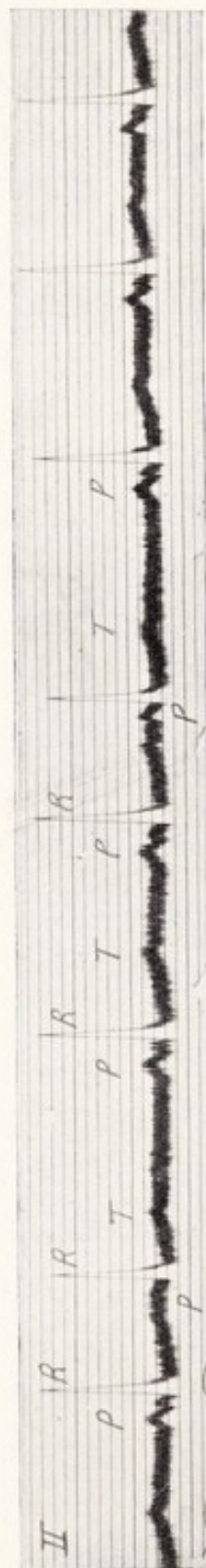
Figs. 60 and 61. Two curves from a case of simple paroxysmal tachycardia. Fig. 60 is from the period of slow and Fig. 61 is from the end of a period of rapid heart action. In Fig. 61 the first beat of the normal rhythm is shown at the very end of the curve. The curves show the ectopic origin of the paroxysm. Both slow and fast rhythms are interrupted by occasional premature beats having a common and auricular focus of origin.

each terminates. The solitary premature beat and the paroxysm are succeeded by a pause, which in a single case is of constant length, and there is an immediate return of the normal rhythm (Fig. 59). The change from one rhythm to the other in paroxysmal tachycardia is absolutely abrupt and in this way contrasts with the gradual slowing of a simple cardiac acceleration. Figs. 59, 61 and 62 are examples of curves that show the endings of paroxysms, and as these curves are published to illustrate special points they may be described in more detail.

In Fig. 59 the first five cycles are paroxysmal; these heart beats arise in a new auricular focus; this is ascertained by comparing the five paroxysmal beats with that which immediately succeeds them, the auricular summits are at first steep and small; with the return of the natural heart action they are larger and of a more customary form. The first normal cycle is succeeded by two premature contractions, each of which springs from the auricle and from the same focus as the paroxysmal

Fig. 62. The end of a simple paroxysm of tachycardia which arose in the auricle. There is delay in conduction during both slow and fast periods; during the latter the auricular contraction falls with the preceding ventricular systole. During the paroxysm the pulse shows alternation. The heights of *R* summits also alternate slightly, the *larger R* corresponding to the smaller pulse beat.

beats ; the first premature beat has a ventricular complex of aberrant type (see Fig. 54, second curve and explanation). The diastole following the two premature beats has the same length as the diastole following the paroxysm. Then come two normal cycles and, after the last, two premature auricular contractions again occur. The figure clearly shows the associations between paroxysmal and premature beats that have been discussed.

In Figs. 60 and 61 are two curves from one case. The first exhibits a normal rhythm disturbed by two premature auricular beats. The *P* summits that are premature are smaller than those of the rhythmic cycles. The second shows a paroxysm of tachycardia in the same patient ; the ventricular complexes are unaltered but the auricular ones are inverted. The second and last cycles of this paroxysm are premature and derived from the same source as those that disturb the normal rhythm in Fig. 60. A longer diastole terminates the paroxysm and the first beat of the returning normal rhythm is seen. Thus, in this patient, paroxysms of tachycardia, thrown in from an ectopic focus, disturb the normal rhythm ; and both the paroxysms and the normal rhythm are interrupted by single premature contractions from a second ectopic focus.

Fig. 62 exhibits an electrocardiogram and radial pulse curve covering the end of a paroxysm of tachycardia and the beginning of the slow rhythm. The long post-paroxysmal diastole is well displayed and there is a little quickening of the heart rate at the beginning of the slow rhythm, a not unusual event. If the ventricular complexes of the slow and fast periods are compared, they will be found to be alike, except that at its commencement T goes deeper during the paroxysmal stage. This change in the shape of T, which is semi-inverted while the heart rate is fast, is not incompatible

with acceleration alone. Had we simply the paroxysmal curves, or even the curves of the slow and fast periods to compare, then a complete analysis of the paroxysmal mechanism would not be feasible. The record of the transition from one form of heart action to the other provides us with the full data. If the last two cycles of the paroxysm are compared, it will be clear that T alters its shape ; the semi-inversion is not present in the last paroxysmal cycle ; it is due, in the other paroxysmal beats, to the superimposition of an inverted P upon T. The paroxysm has originated in a new auricular focus and each auricular beat has propagated a ventricular beat ; the rate is fast (128 per minute) and the P-R interval is long (0·33 second) ; consequently each auricular contraction falls with the preceding ventricular contraction. The last ventricular beat is necessarily uncomplicated in this respect, for the auricular paroxysm has terminated before the ventricle responds to the last paroxysmal impulse. The paroxysm ends in the usual long diastole and the natural pacemaker re-establishes its control ; but the prolongation of the P-R interval is maintained.

These illustrations show how valuable is a comparison of fast and slow periods in electrocardiograms. The curves of the paroxysmal phase are but rarely sufficient to provide a full analysis. The curves of both rapid and show heart action are also insufficient at times : curves showing the transition from fast to slow or from slow to fast are the most illuminating.

Paroxysms of tachycardia may also arise in one or other ventricle ; in these circumstances the electrical curves of individual beats are of the forms seen in Figs. 41 and 42. Tachycardia of ventricular origin is comparatively rare in records, a supraventricular origin is the general rule. Many

originate in the junctional tissues, but the resultant curves are often obscure and difficult fully to elucidate. Where, as in Fig. 61, the *P-R* interval is slightly reduced and *P* is inverted, it is customary to locate the origin of the paroxysm in the upper part of the *A-V* node. In other and commoner instances of nodal paroxysms, *P* and *R* fall together and the former cannot then be deciphered.

The chief value of electrocardiographic records in tachycardia is in distinguishing between simple acceleration, simple paroxysmal tachycardia and a third condition to be described in the next chapter. The natural history of the three conditions and their reactions to treatment are essentially different. Rapid heart action of obscure origin is frequent; the electrical method singles out those which are primarily of cardiac origin and distinguishes them from simple disturbances of innervation.

Dislocation of the pacemaker.

It may be useful here to contrast simple paroxysmal tachycardia with the disturbance of the heart's action that is shown in Fig. 63. This electrocardiogram opens with three cycles in which the heart beats

Fig. 63. From a patient who presented preponderance of the left ventricle. A regular rhythm of sinus origin is disturbed by a single premature beat arising in the auricle. A rhythm of ectopic origin follows this extrasystole; the P-R interval in these last three cycles is somewhat shortened; the new rhythm probably arose from the upper levels of the auriculo-ventricular node.

have been propagated from the natural pacemaker, as is evidenced by the shape of the complexes (S is deep because the curve was taken from a patient in whom there was a preponderance of the left ventricle ; the curve is from lead *III*). The fourth beat is a premature contraction of auricular origin. The usual pause succeeds this disturbance, and subsequently rhythmic heart action is resumed. The restored rhythm, however, arises from a new focus, as is shown by the shape of the corresponding auricular complexes. There is little or no difference in the rate of the heart before and after the disturbance, and in this respect the curve contrasts with those of paroxysmal tachycardia. The ectopic rhythm is developed in this instance from the same type of impulses as is the normal rhythm ; unlike the paroxysms of rapid heart action, it is not allied genetically to premature beats.

CHAPTER VII.

AURICULAR FLUTTER.

The term " auricular flutter " is used in this chapter to designate a regular and rapid action of the auricle due, so it is believed, to "circus movement " in the auricle. The disorder is not easy to diagnose by methods other than the electrocardiographic.

Circus movement is a continuous movement of the contraction wave around a ring of muscular tissue such as is provided by the mouths of the superior and inferior vena cava. Where circus movement has become established, there are no discrete impulses creating the separate beats of the heart, but each time that the wave completes the circuit of its chosen path, it spreads into the surrounding muscular tissue, which contracts in response. Thus, the revolving wave travelling in a ring of auricular tissue governs the movements of the whole auricle ; and the auricular movements, as usual, govern the beating of the ventricle.

The usual rate at which the auricle contracts in flutter is approximately 300 per minute ; in special circumstances it may be as low as 150, it may reach 360 per minute. The flutter comes abruptly and goes abruptly, as does a simple paroxysm of tachycardia. Rarely, flutter also occurs in short paroxysms, but in general it persists for months or years. In this and several other respects " flutter " is allied to another disorder, namely " auricular fibrillation," which is described in the next chapter.

The auricular rate is so rapid that the ventricle is rarely able to follow it ; so it happens that most patients who exhibit flutter also demonstrate heart-block, the grade of which is generally such that only the alternate auricular impulses stimulate the ventricle. Most patients who are the subjects of flutter have an enhanced ventricular rate, but the ventricular rate is only half the auricular. On the other hand, the ventricular rate may be slow ; any grade of block may be present.

The electrocardiograms.

The electrocardiograms are not difficult to recognise if carefully examined. It is often of advantage to have the
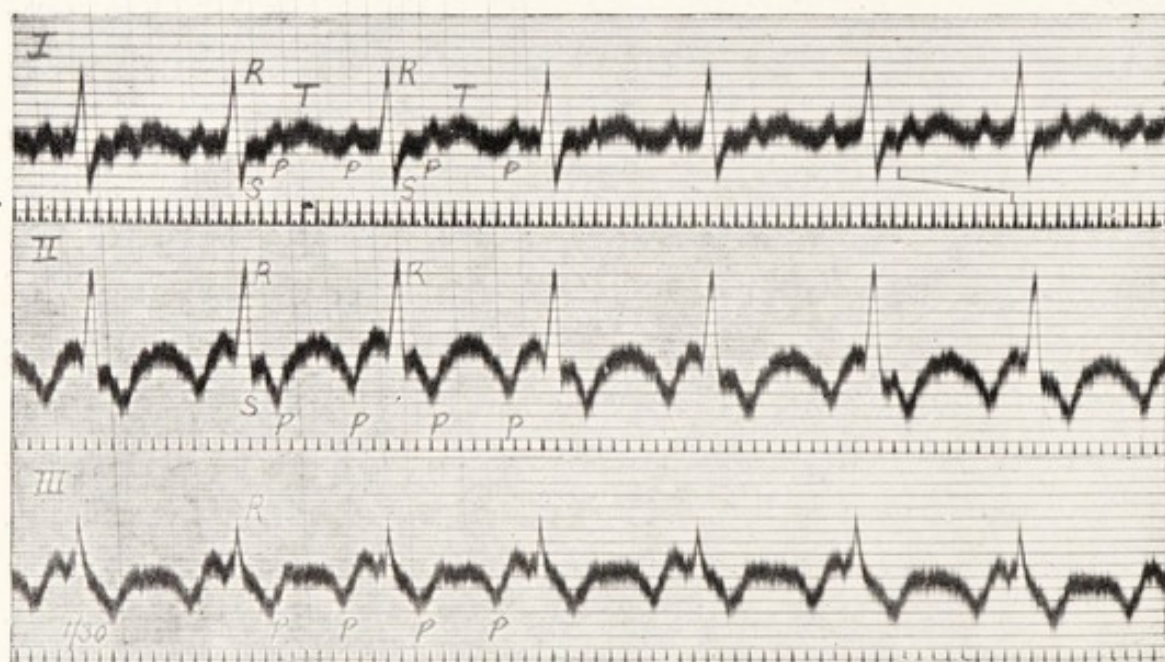
Fig. 64. Curves from the three leads in a case of auricular flutter. The auricular rate is 324, the ventricular rate is 162 per minute. In leads *II* and *III* the auricular complexes are contiguous.

curves from the three leads side by side, so that similar events may be compared in distinct leads. Fig. 64, which comprises all three leads, may be used to illustrate the chief points in the analysis. In lead *II* of this figure the most conspicuous peak is *R* ; it is followed by a small depression *S*. These are

the only certain indications of ventricular systole. The remainder of the curve may be described as consisting of two V-shaped depressions, one of which might seem to correspond to an inverted *T*, the other to an inverted *P*. The two depressions have an almost exactly similar shape. Now this is not inconsistent with their representing an inverted *P* and *T*, for similarity of the two deflections is not unknown. But it would be a strange coincidence if, with this similarity, *P* and *T* should be placed at exactly equal distances from each other throughout the whole curve. Moreover, even though *P* and *T* may resemble each other in a given lead, *they never do in all three leads*; yet in each of the curves of our illustration a uniform series of waves is present. In lead *III* they are of much the same form as in lead *II*; while in lead *I* they appear as small peaks. In all leads they lie at equal distances from adjacent summits of the same form; in all leads the deflections lie in orderly sequence. These facts demonstrate that the two waves have a similar origin; although only alternate waves lie in ventricular diastole, all are the result of auricular systoles. Fig. 64 therefore shows 2 : 1 heart-block, the rate of the auricle being 324 and that of the ventricle 162. In leads *II* and *III* the real *T* is scarcely distinguishable, but in lead *I* it is clear, having a broad form and falling between two *P* summits.

The electrical representatives of auricular contractions, when this chamber is in a state of flutter, are contiguous. In Fig. 64 this is clearest in lead *II*. For descriptive purposes, *P* may be said to commence in an upstroke and to be continued in a dome, which ends at the foot of the succeeding upstroke. Each of these complete complexes in reality represents a complete movement of the contraction wave around its circuit in the auricle and, as there is neither beginning nor end

to the movement, so there is no real beginning nor end of the auricular complex. Because one upstroke shortly precedes R, the dome into which it continues runs through R and S. The next dome commences during the ventricular systole and runs on into ventricular diastole. Each auricular contraction partially coincides with a ventricular contraction and it is to this that many flutter curves owe their obscurity. Other examples of curves are shown in Figs. 65, 66 and 67. Fig. 65
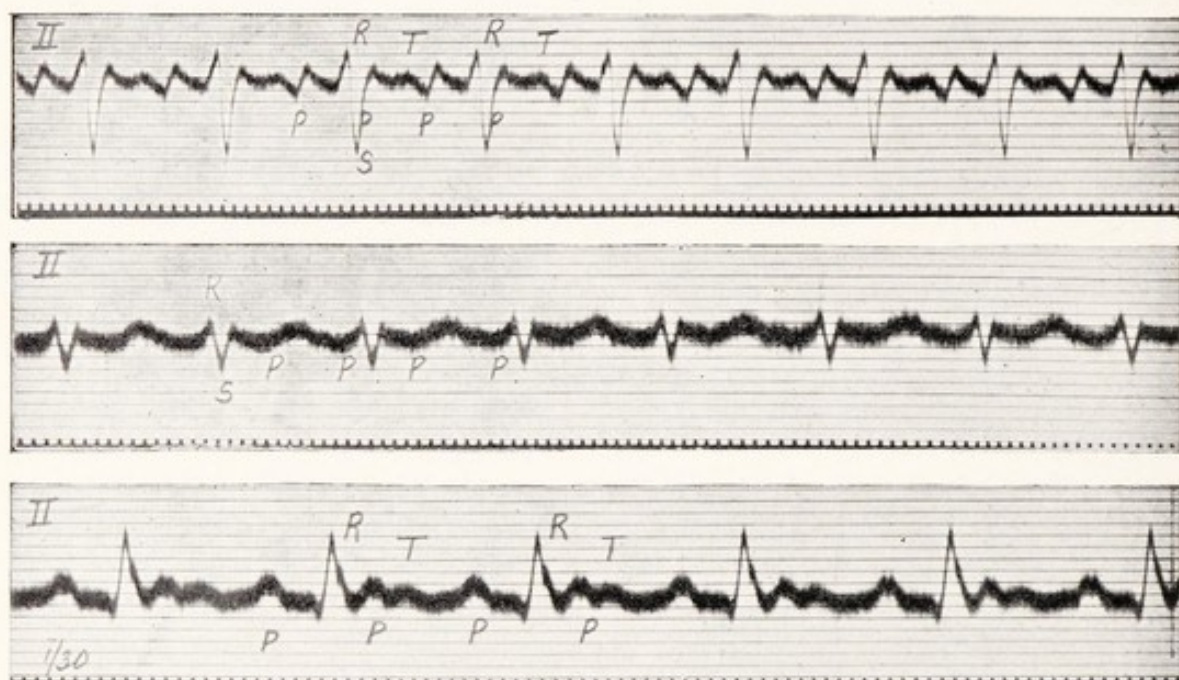
Figs. 65, 66 and 67. Examples of auricular flutter curves. In each 2 : 1 heart-block is present. The auricular rates are approximately 330, 280 and 228, respectively; the ventricular rates, 165, 140 and 114, respectively.

shows the common type of auricular curve. The auricular contractions are again twice as numerous as the ventricular. Each auricular complex may be described as commencing in an upstroke, but this upstroke is only clearly distinguished when it precedes R. It continues to a blunt summit and then the curve slopes away and becomes incorporated in the ventricular complex. The next upstroke coincides with the returning line of S; it comes to a point and slopes away again, but the gradual downstroke is interrupted by a small

T summit. Thus the auricular tracing as a whole is constituted by a zig-zag line of which the upstrokes are steep and the downstrokes are more gradual.

Fig. 66 exhibits a similar action. The ventricular complexes consist of R and S deflections. The auricular portion of the curve is traced as a uniform wavy line ; one convexity falls midway between R summits, the other falls with R. The general and uniform undulation is visible though disguised ; each convexity represents an auricular contraction ; 2 : 1 heart-block is present. The rates of auricle and ventricle are 280 and 140, respectively. The analysis of Fig. 67 is easier, for here the P's are more sharply defined. The auricular rate is 228 and the ventricular 114 per minute.

The analyses while 2 : 1 heart-block is present are difficult to those unfamiliar with the curves ; sometimes they are almost impossible at this stage. The true nature of the condition is at once revealed when a higher grade of heart-block becomes established. The first curve of Fig. 72 is not very dissimilar to that of Fig. 66. The auricular line is a wavy one and there are two convexities to each ventricular systole ; one falls midway between adjacent R summits, the other coincides with R ; 2 : 1 heart-block is present, the rates being 300 and 150. The same patient demonstrated a higher grade of block upon another day ; the auricular convexities then became clear, being less confused by ventricular summits ; the curve is shown in the second strip of Fig. 72 ; 4 : 1 block is present, the rates being 300 and 75. (Similar electrocardiograms are shown in the first two curves of Fig. 73, the top curve of which should be compared with Fig. 65.) The same 4 : 1 ratio is shown in Fig. 68, a curve that has been taken at a faster speed and with a more sensitive string. This curve has been marked

so as to reconstruct that portion of the auricular line which is obliterated by R and S; the regular relation of four auricular convexities to a ventricular systole is made more apparent in this way.

The constant activity of the auricle may be demonstrated in another fashion. If the vagi are pressed upon in the neck, the ventricular beating is often checked; but the auricle continues to contract at its former rate (Fig. 69). The continuously wavy line, each convexity of which stands for an auricular beat, is thus strikingly displayed. It is to be
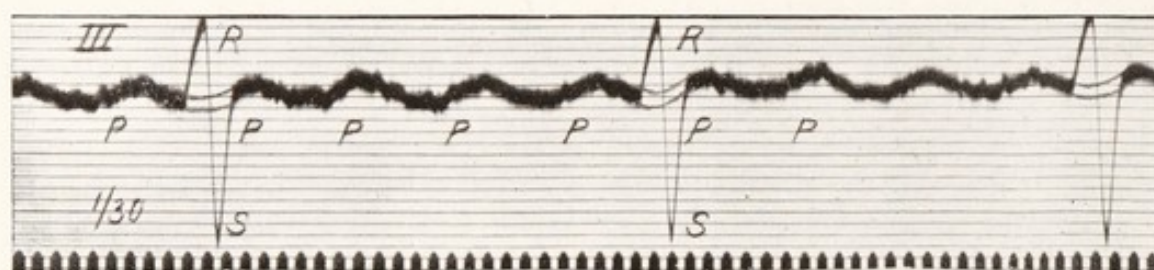
Fig. 68. From a case of flutter, showing 4 : 1 heart-block. The portions of the auricular complexes that have been obscured by the initial phases of the ventricular complexes have been indicated by reconstructing the auricular oscillations. The standard for this curve is 1·5 centimetres to the millivolt.

emphasised again that each of the waves so traced corresponds to a complete revolution of the contraction wave in the auricle; the sinuous type of curve well displays the fact that the revolutions are continuous, uninterrupted and regular.

In the case of simple paroxysms of tachycardia, in which the fast rhythms are developed from some ectopic centre, nerve influences, such as are induced by exercise, emotion, posture or pressure upon the carotid sheath are practically without influence upon the rate at which the impulses are generated. The same statements apply also to flutter. That is so because in flutter, as in paroxysmal tachycardia, the natural pacemaker is out of action, and nerve control of rate, which is normally exerted through this pacemaker, is
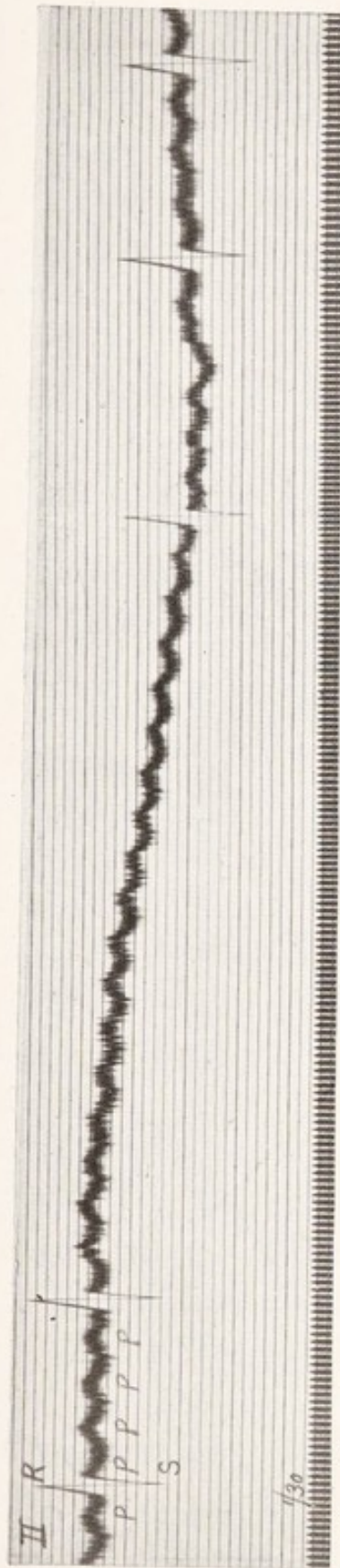
Fig. 69. From the same case, showing the effect of pressure on the right vagus during a period of 4 : 1 heart-block. The ventricle ceases to beat, the auricular action shows no abatement.
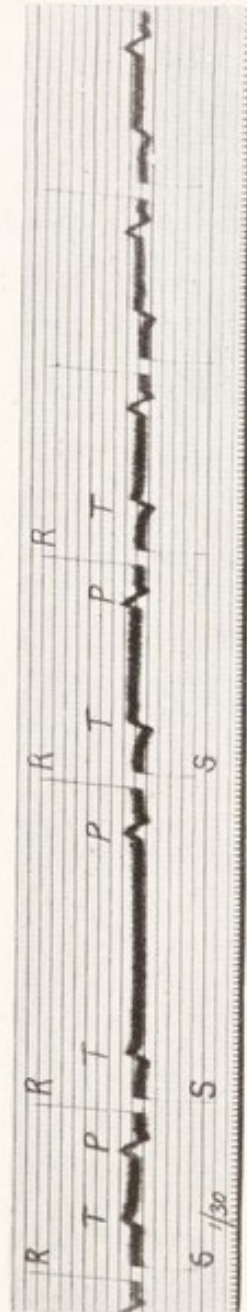
Fig. 70. From the same case after the heart's action had returned to the normal. Pressure on the vagus (right or left) now produced slowing of the whole heart. The left vagus was the one pressed upon in this instance.
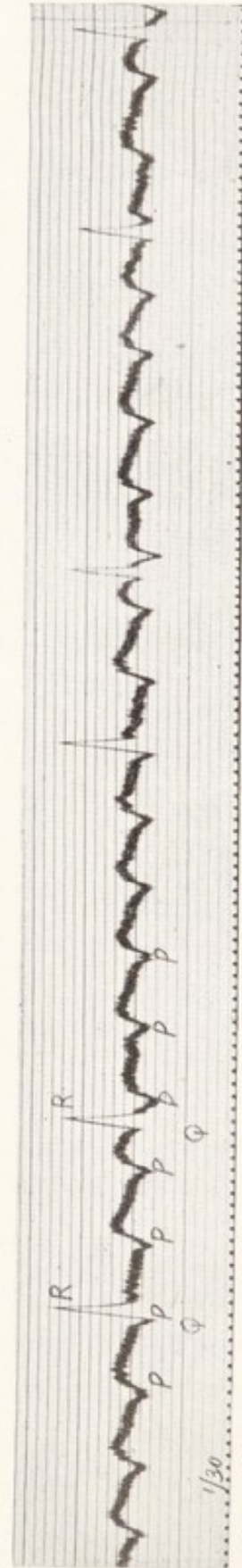
Fig. 71. From a case of flutter on digitalis, showing slow and irregular responses of the ventricle.

largely or wholly lost. Figs. 69 and 70 were taken from the same patient and show the effects of pressure on the carotid sheath, during the flutter stage and during the stage of normal heart action. In both cases the vagus has been stimulated, as is shown in the one instance by ventricular standstill and in the other by slowing of the whole heart with a slight prolongation of the *P-R* interval. Both curves afford evidence of heart-block as a result of pressure, but the essential difference lies in the action upon the auricle. The flutter is uninfluenced (Fig 69); the natural pacemaker reacts, the rate at which it builds up impulses being conspicuously decreased (Fig 70).

Auricular flutter occurs for the most part in elderly subjects and in those in whom there are evidences of cardiac enlargement, myocardial degeneration, and symptoms of an exhausted reserve. When it develops, it throws a burden upon the heart proportioned to the increase of ventricular rate. When the heart muscle is degenerate and the rate rapid, dilatation, engorgement of the veins, enlargement of the liver and dropsy appear. On the other hand, when it develops in a heart the muscle of which has considerable reserve, circulatory disturbances and embarrassment are not manifested ; but palpitation is common and the symptomatic response to effort is exaggerated. In this manner it disables even those in whom the heart muscle is strong. Fainting attacks are not uncommon in the condition, and are due to the ventricle suddenly responding to the full auricular rate.

Flutter may pass spontaneously into fibrillation, but more commonly the change is induced by digitalis adminis-tration. Digitalis and its allies are often very serviceable if given in full doses and the reaction is highly interesting. In my experience, the ventricular rate can always be reduced so that 2 : 1 block gives place to heart-block of higher grade.

Supposing that 2 : 1 heart-block is present originally, then irregularity develops, and this is due to responses at irregular intervals (Fig. 71). Later, if the drug is continued, the ventricle may become regular again, as a result of the develop-

Fig. 72. Four curves from a case of long standing auricular flutter, showing the effects of treatment. The first curve shows an auricular rate of 300 and a ventricular rate of 150. In the second curve the auricular rate is maintained, but the ventricular rate has been halved (4 : 1 block is present) as a result of digitalis administration. In the third curve auricular fibrillation is seen and it is accompanied by a slow and irregular action of the ventricle. In the last curve the normal rhythm, interrupted by occasional premature contractions of auricular origin, has been restored.

ment of 4 : 1 block (Figs. 72 and 73, 2nd curve of each). In these circumstances the ventricle may be beating at 75 while the auricles continue to beat at 300 per minute, and there

may be no evidence, except the electrocardiographic, of the
rapid auricular beats.   Finally, and in a large percentage of
cases, I find that fibrillation of the auricles appears under the
action of the drug (Figs. 72 and 73, 3rd curve of each) ; and
further that, if the digitalis is withdrawn at this stage, the
normal action of the heart is often restored (Figs. 72 and
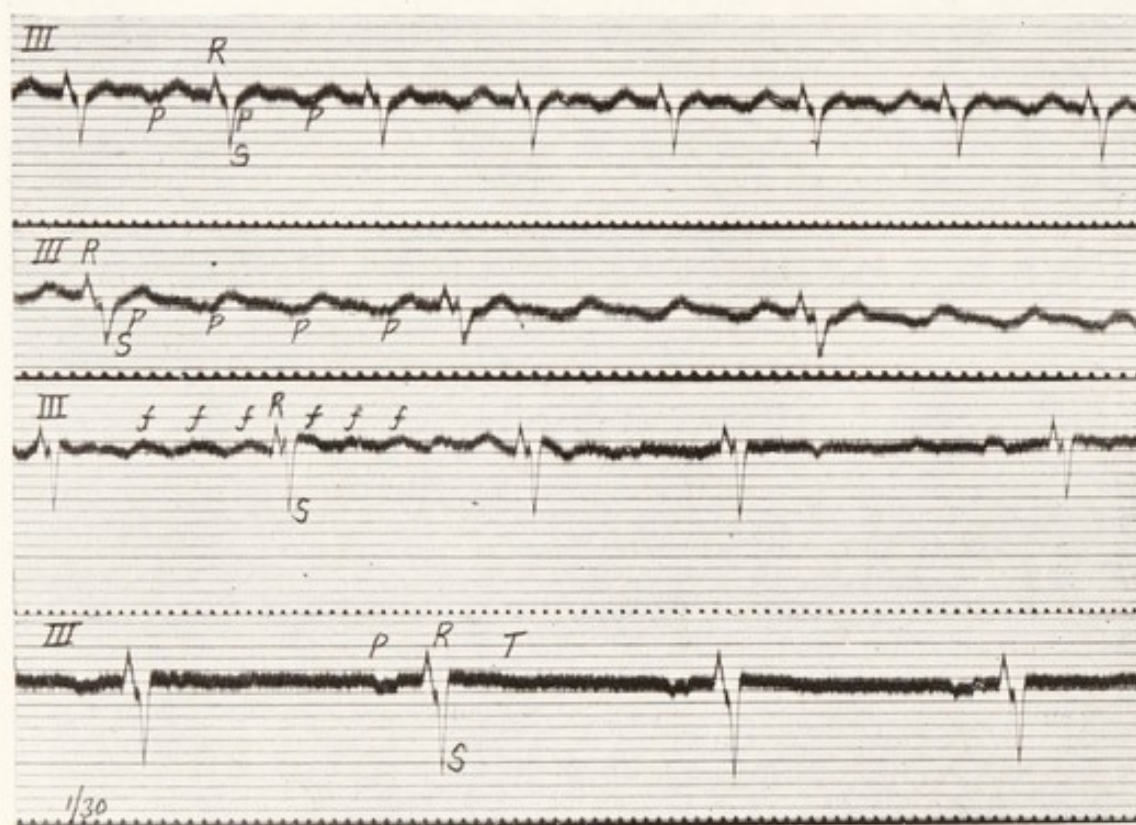
Fig. 73.   A similar series of curves to that shown in Fig. 72. They show the
   effects of digitalis administration upon long standing auricular flutter. In
   the first curve the auricular rate is 320 and the ventricular rate is 160. In
   the second the rates are 324 and 81 (4 : 1 heart-block). In the third, the
   auricles are fibrillating and the ventricle is responding irregularly at an
   approximate rate of 79 per minute. In the last curve a regular and
   normal rhythm is re-established. The inversion of *P* lead in *III* is not
   significant of an extopic origin of the heart beat in this instance.

73, 4th curve of each) and may persist. The treatment
of auricular flutter by digitalis is often most successful and
brings with it much improvement of the patient's general
condition.   As the rapid ventricular rate subsides, cyanosis,
engorgement, dropsy and other accompanying symptoms
vanish quickly.

CHAPTER VIII.

AURICULAR FIBRILLATION.

Fibrillation of the auricle is a variety of auricular flutter. In flutter, as has been described, the movement of the wave as it circulates in the auricle is regular ; that is to say, the same path is travelled over and over again in unvarying fashion. Consequently the beating of the auricle is regular, and the responses of the ventricle are arranged in relatively simple ratios. A circulating wave is also responsible for the condition termed fibrillation, but in this condition the path pursued is inconstant and the separate circuits are completed in varying times. Moreover the circuits are completed more rapidly than in simple flutter. The result is a much more disorderly movement of the auricle and a wholly irregularly response of the ventricle.

In fibrillation co-ordinate contraction may be said to have ceased in the auricles, the surface muscle of which exhibits constant minute twitching movements. The muscle, though extremely active, has relinquished its function of driving blood into the ventricles ; the normal impulses ordinarily transmitted to the ventricle are submerged and replaced by rapid impulses derived in a haphazard fashion from the irregularly and rapidly circulating wave in the auricle.

Our ability to recognise this disorder is a great asset because it is so frequent, because its effects are so profound, and because its reactions to treatment are almost peculiarly

its own.   The electrical curves of auricular fibrillation are characteristic and afford the only certain means of identifying it.   It is recognised by two groups of signs ; the first signs result from the changed functional state of the auricles, the second signs depend upon the character of the ventricular responses.   We will consider the last first.
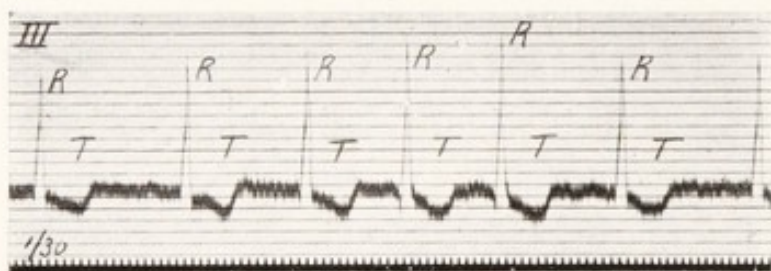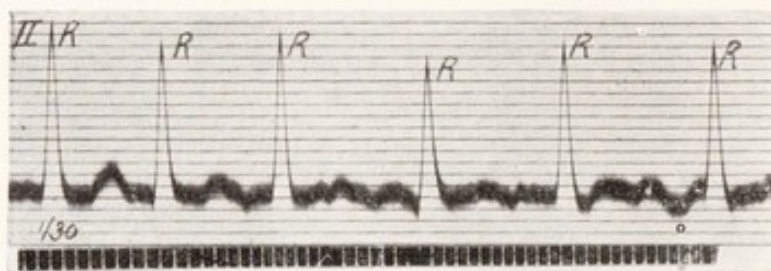
Fig. 74. A curve of auricular fibrillation showing rapid and irregular ventricular responses. The height of the peak *R* is variable and is not related to the length of the diastole that precedes it. The oscillations are obscured by the rapid ventricular action ; that they are present is evident from the changing shape of those parts of the curve which unite adjacent *R* summits. There are no *P* summits.

Fig. 75. A curve of auricular fibrillation, showing rapid and irregular ventricular response, great variation in the height of *R*, and inversion of *T*. Fibrillation oscillations are only just visible ; the very rapid and minute vibrations are from the body musculature. There are no *P* summits.

When the ventricle responds solely to a fibrillating auricle, its beats are necessarily of supraventricular origin ; they consequently present the normal outlines in electrical curves, *R* and *T*, or *Q*, *R*, *S* and *T* deflections are found. These ventricular complexes have all the same general outline though the heights of the *R* summits vary in many cases

from cycle to cycle. If the ventricular action is slow, then the R summits are almost uniform in height, but when it is rapid (Figs. 74 and 75) the excursions vary, and there is then no relation between their heights and the diastoles which precede them ; the individual complexes are placed at very irregular intervals.

The other group of signs is auricular. The R summits have no P summits before them, for, as there is no co-ordinate contraction of the auricle, so there is no presystolic auricular representative in the electrocardiogram ; but the quivering flesh of the auricle yields pronounced waves, oscillations that are clear and characteristic in the majority of the curves. The type of oscillation deserves study ; the average frequence is 450 per minute ; so, if the ventricle beats at 75, there will be about six oscillations to each cycle, or if it beats at 110, there will be about four oscillations to each cycle. Only the oscillations that occupy diastole are easily distinguished ; consequently, if the ventricle beats slowly, the oscillations are conspicuous ; but if it beats fast, they may be difficult to distinguish. Each oscillation corresponds to a complete circus movement in the auricle, as in flutter ; the difference being that the oscillations of fibrillation are more rapid than in flutter and irregular in length and form.

The oscillations of the first curve in Fig. 80 are very large, and it should be remarked that there is a tendency towards regular disposition, though the individual movements of the fibre *are never quite uniform as in flutter*. When T is prominent, as in this curve, it is deformed by oscillations that fall with it. The oscillations are continuous, but in one place (between the third and fourth R summits) their amplitude is diminished. This variation is always present, and if the mean amplitude is small, then the oscillations may vanish from place to place in the curves. In Fig. 81 (top curve) they are prominent
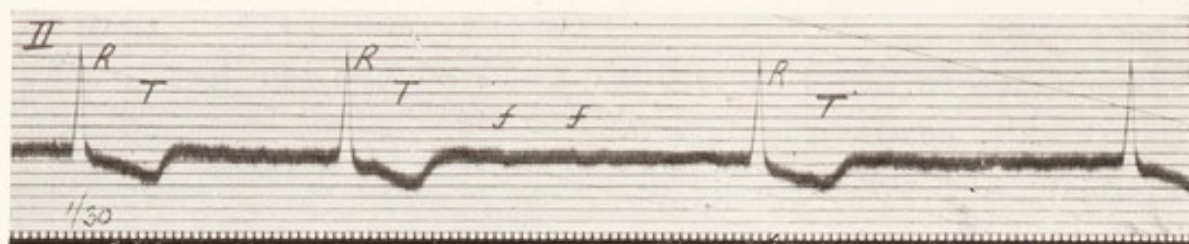
Fig. 76.   From a case of fibrillation under treatment with digitalis.   It
          shows irregularity of the ventricle and inversion of *T*.   The oscillations
          are small ; *P* does not appear.
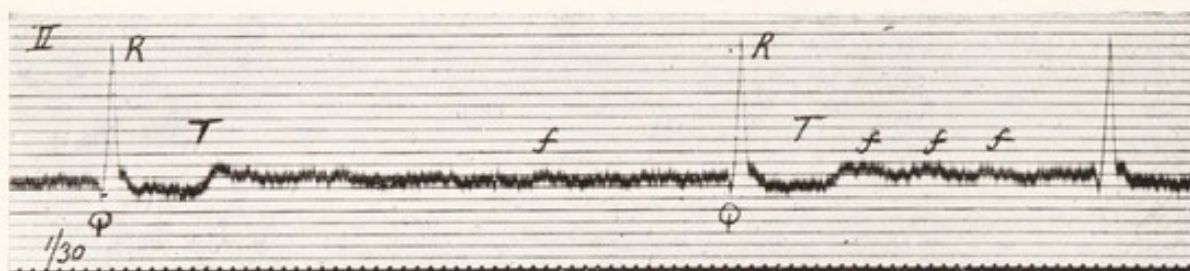
Fig. 77.   From a case of fibrillation under treatment with digitalis.   The
          ventricular action is irregular.   The diastolic portion of the curve shows
          two series of oscillations, the one (*f.f.*) due to the fibrillations of the
          auricle, the other resulting from tremor ; these last oscillations have a
          very high frequence.
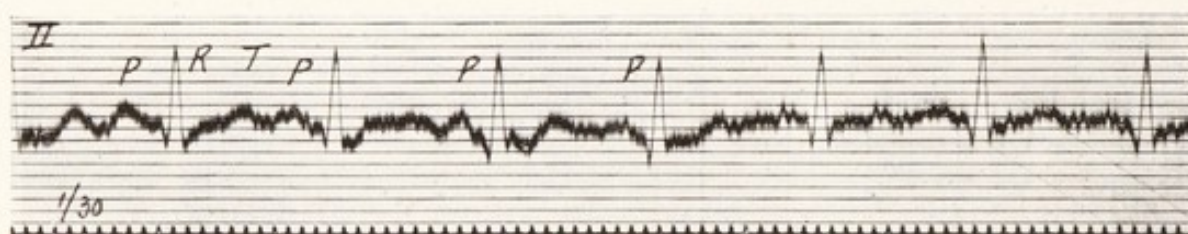
Fig. 78.   From a case of exophthalmic goitre, showing a regular heart action.
          Each ventricular complex is preceded by a summit *P*, but the whole
          curve is disturbed by oscillations resulting from tremor of the somatic
          musculature.
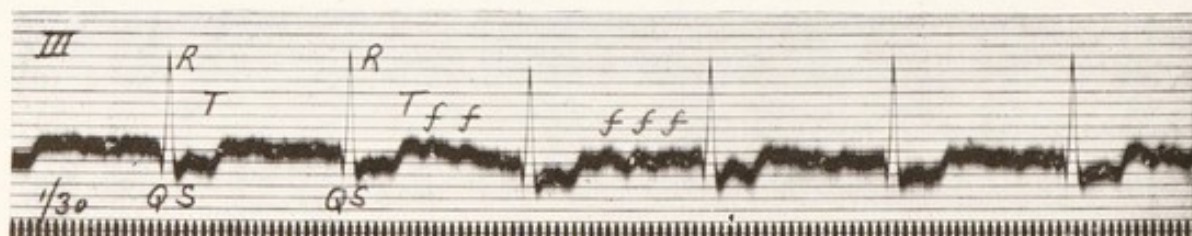
Fig. 79.   From a case of auricular fibrillation, under treatment with digitalis.
          The fibrillation is evidenced by the oscillations *f.f.* and by the dis-
          appearance of *P* summits.   The ventricular beats are placed regularly
          because complete heart-block was present.   The rate is exceptional for a
          ventricular rhythm, being approximately 90 per minute.

at the beginning, but towards the end they are barely perceptible. These changes in amplitude are due to gross change in the path taken by the circus movement. When the ventricle beats fast, as in Figs. 74 and 75, diastole is comparatively short and the oscillations are not clearly seen. Their presence is ascertained in such curves by scrutinising the lines that join the R summits ; these lines are differently configured from cycle to cycle. The oscillations are in reality always continuous, but, as in flutter curves (Figs. 65 and 66), fast ventricular action may obscure them ; in other instances, owing to change in the path taken by the wave, the lead becomes temporary unsuitable and will not display them. They are especially prominent in cases of mitral stenosis (Figs. 80 and 94). In other conditions they are often small ; Fig. 76 is an example of such a curve ; this electrocardiogram should immediately suggest fibrillation, for the ventricular complexes are all of the supraventricular type, and quite irregularly spaced ; furthermore, there are no P summits. There are deflections in Fig. 74 that at first suggest auricular contractions, but they are inconstant in form and position and should not mislead, being in reality individual and prominent oscillations that happen to fall in presystole. Fig. 77 shows another and distinct form of oscillation ; it is of very high frequence and due to tremor of the somatic muscles ; it is not to be ascribed to fibrillation, although the curve was actually from a case displaying this disorder. These minute vibrations are common in fibrillation cases, for the patients are often feeble or actually tremulous, but they may be present when no tremor is detected and may then confuse interpretations. It is wise to neglect them and to look to the general sweep of the curve. In Fig. 77 they are present throughout and distort the curve ; it is from a fibrillation case, as may be seen immediately from the shape
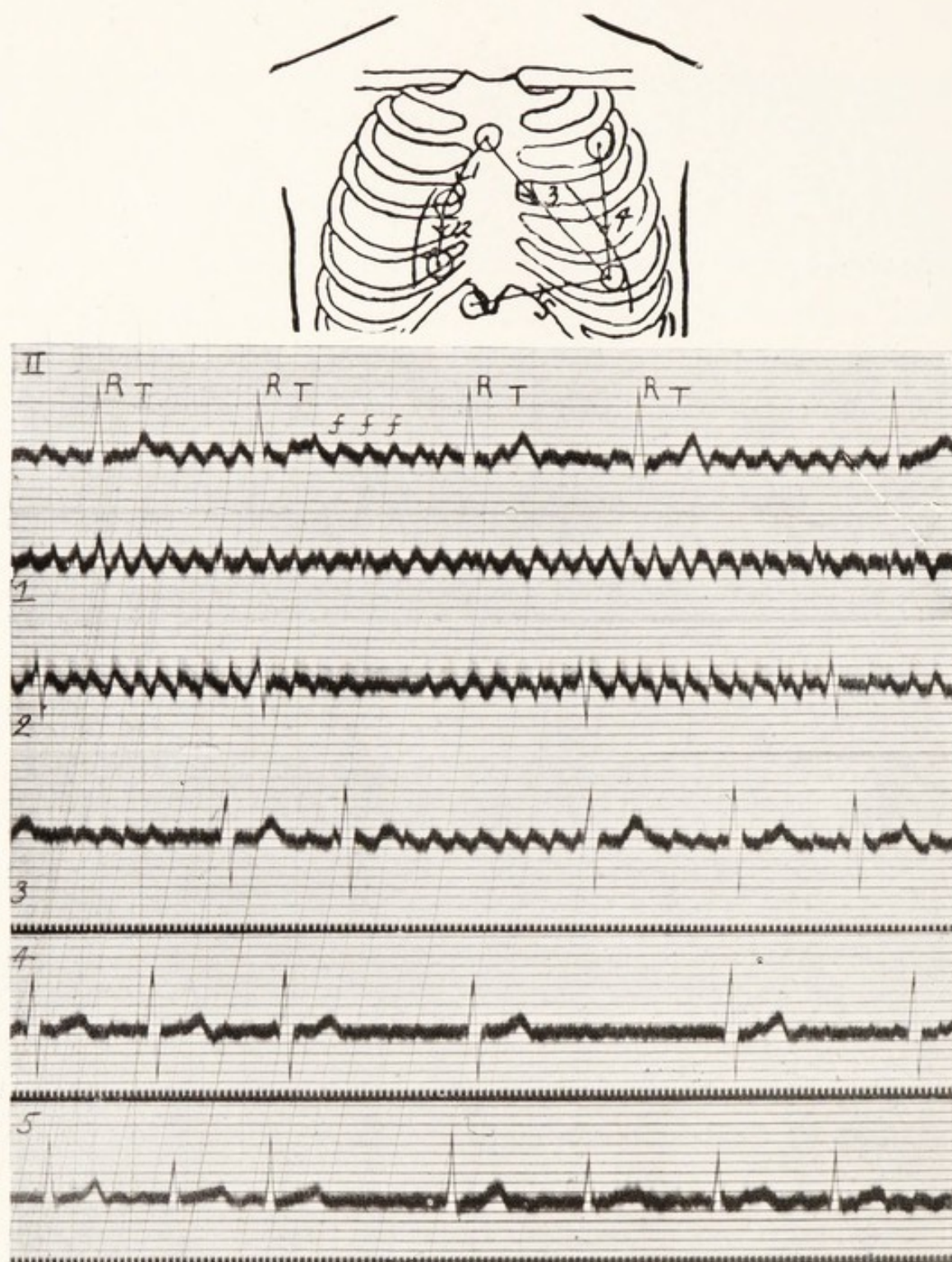
Fig. 80. A diagram of the chest wall showing the special leads (1 to 5) used in identifying the oscillations of auricular fibrillation; also six electrocardiograms. The first electrocardiogram is from lead *II*; it consists of irregularly placed ventricular complexes (*R*, *T*) and of large and continuous oscillations (*f.f*). The remaining five curves are from the chest wall. 1 and 2 were taken from the area overlying the right auricle; in these leads the oscillations are maximal and the ventricular complexes are minimal. 3 was taken from an oblique lead covering the whole heart, and it shows both oscillations and ventricular complexes. 4 and 5 were taken from leads along the margins of the ventricles; they show but little sign of the oscillations. From a case of mitral stenosis.

of the ventricular complexes, their arrangement and the absence of P summits ; if the curve is regarded with the eyelids almost closed, the fine tremor is no longer distinguished and the slower and inconspicuous oscillations of the fibrillating auricles then become clearer ; these are most prominent after the second ventricular beat and are of characteristic form. Fig. 78, a different type of curve, illustrates the same distinction ; it is from a case of exophthalmic goitre in which there was much tremor. The tremor oscillations are large and coarse, serrating the whole curve and obscuring P ; nevertheless P or some trace of it may be discovered in each cycle, despite the distortion ; it should be noted also that the ventricular beats are evenly distributed. The action of the heart from which this curve was taken was normal. Sometimes, when the somatic muscle tremor is coarse and the patient holds the limbs rigidly or unsteadily, the curves present difficulties and there may be doubt as to whether a given series of oscillations is of somatic or cardiac origin ; under these conditions special leads may profitably be adopted. Small metallic discs are fastened directly to the chest wall with a stiff paste of flour and salt. Convenient points of contact are depicted in the accompanying diagram, which illustrates the method (Fig. 80). When fibrillation is present and the electrodes lie in the vicinity of the right auricle (leads 1 and 2 of the diagram) the oscillations are maximal, and there is but a trace of the ventricular beats. When they lie in the long axis of the heart (lead 3) both the oscillations and the ventricular complexes are conspicuous. Finally, when they lie along the left or right ventricular border (leads 4 and 5) the ventricular complexes are clear cut while the oscillations are small or absent. The corresponding electrocardiograms are shown below the diagram, the first curve of which is from the customary lead *II* (right
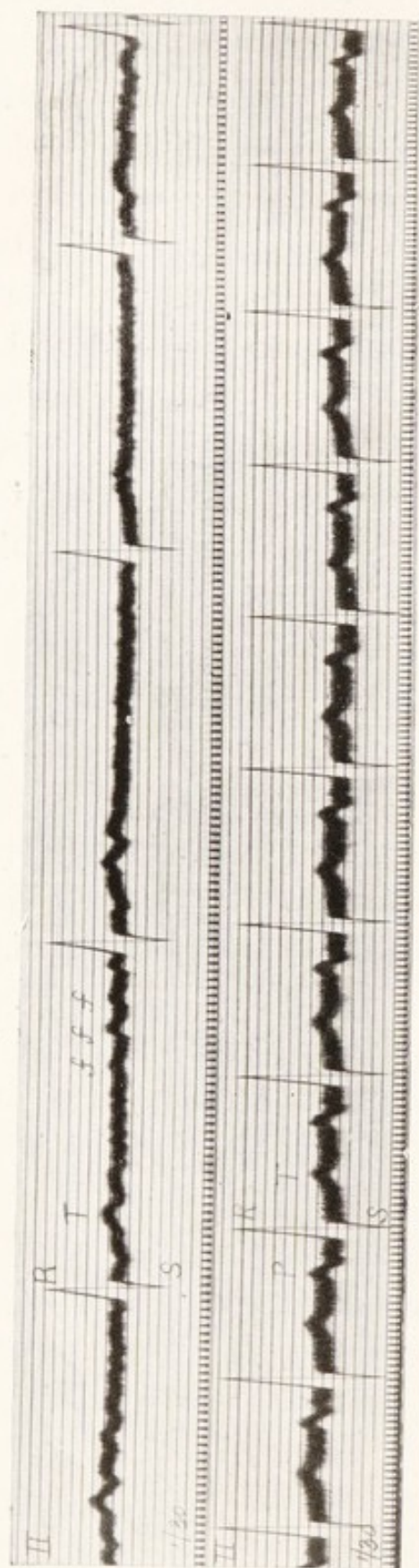
Fig. 81. Two curves from one case; the upper shows auricular fibrillation, the lower displays the normal rhythm. In the first the ventricular action is irregular, there are no *P* summits and oscillations are conspicuous; the patient was under digitalis when this curve was taken and the action of the ventricle was slow consequently. In the second the action is regular, *P* is prominent and there are no oscillations. Note the similarity of the ventricular complexes in the two curves.

arm to left leg). The special contacts analyse this axial lead, breaking it into its auricular and ventricular components. The oscillations of fibrillation are thus readily identified and their auricular origin is clearly indicated. In tremulous subjects, oscillations are not seen in leads from the chest unless the muscles of the chest are visibly twitching.

The chief features of the electrocardiograms in fibrillation of the auricle are strikingly displayed when the curves of this condition and those showing the normal rhythm can be compared in one and the same case. Fibrillation, though usually a chronic and persistent disorder, occasionally occurs in short paroxysms or, as we have seen, terminates a period of auricular flutter. In such patients the comparison is possible. The curves of Fig. 81 were taken from the same patient within a few days of each other. The ventricular complexes are of the same type in both; in one they are distributed regularly and are preceded by the usual P summits (second curve); in the other the spacing is irregular, no P summits are discovered, and the whole base line exhibits the oscillations of fibrillation (first curve).

Two groups of signs have been insisted upon, namely, the auricular and ventricular, respectively. In the great majority of curves, the signs of both groups are found. We may now discuss the exceptional curves. It has been said that the oscillations may almost fail; absolute failure in all leads, from limbs or chest, does not occur. More important, from the standpoint of diagnosis, is the association of auricular fibrillation with *regular* action of the ventricle. This combination is also very infrequent, but occurs when the auricular impulses no longer control the ventricle; in other words, when the last-named chamber beats independently. Being exceptional, it serves to emphasise the rule, that while auricular fibrillation is present, the ventricular movements
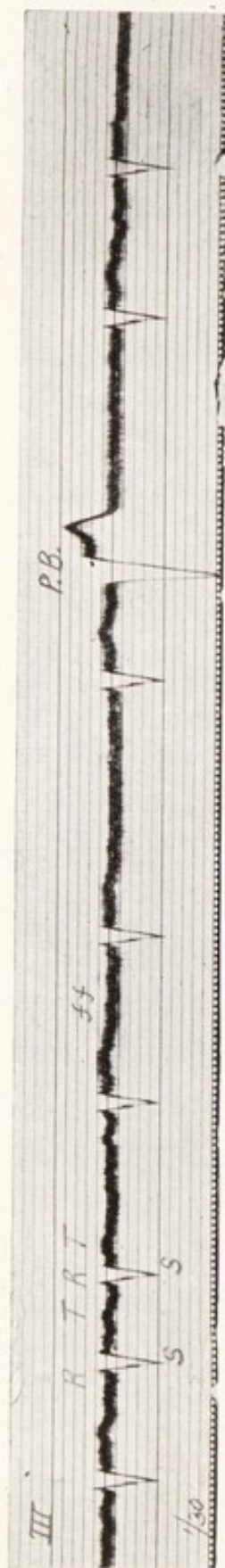
Fig. 82. From a case of auricular fibrillation. With a single exception, all the ventricular complexes are of the supraventricular type; the corresponding ventricular contractions are responses to the fibrillating auricle. The premature beat *P.B.* is of different origin ; it comes from the left ventricle.

Fig. 83. From a case of auricular fibrillation on large doses of digitalis. It shows the coupling of ventricular beats that results from over-dosage. The first complex in each couple is of the supraventricular type ; the second is of different form ; these last contractions (*P.B.*) are premature and originate in the ventricle. The pauses succeeding the couples are of equal length ; the drug had also produced complete heart-block in this instance.

are disorderly. Fig. 79 illustrates the condition ; it shows the usual type of oscillation, no auricular summits P are seen, but the ventricular beats have natural outlines and are in regular sequence. It is desirable to confirm the origin of the oscillations in these patients by means of the chest wall leads.

Fibrillation is the acme of auricular disturbance, consequently it is never complicated by other perversions of auricular rhythm. The heart exhibiting it may display other types of disorder at the same time, however. Thus there may be heart-block. The responses of the ventricle to a fibrillating auricle are naturally rapid and approach 200 per minute. A slower ventricular action indicates heart-block, and the degree of heart-block is gauged by the degree of slowing. It is by producing high grades of partial heart-block and by assuaging the original rapidity of the ventricular beats that digitalis and its allies produce their most notable clinical effect. Figs. 76 and 77 were taken from patients fully under the influence of these drugs ; the ventricular rates were 47 and 52 when the curves were taken ; before treatment they had been rapid. Digitalis heart-block may be complete (Fig. 79 is an example) and, when complete from this cause, the rate of the regular ventricle is relatively high. Heart-block resulting from bundle lesions produces slow ventricular action when the auricles fibrillate, and when complete, the ventricular rates are of the same order as those of uncomplicated heart-block. Thus, fibrillation may be accompanied by ventricular rates ranging from 30 to 200, according to the conducting power of the auriculo-ventricular bundle.

The ventricular complexes in fibrillation are, as has been said, of the supraventricular type, but T may show inversion (Fig. 76) and the aberrant forms of beat, described on page 28, may occur, when one or other bundle branch is damaged.

The comparative amplitudes of R and S in the three leads have the same significance as they have when the sequence is normal (see page 31 and Fig. 95).

Extrasystoles are seen from time to time; they spring from ventricular foci almost exclusively; they cannot originate in the auricle, but some appear to originate in the junctional tissues. An example of ventricular extrasystole is given in Fig. 82; this curve was taken from lead *III* and the diminutive R and deep S represent the supraventricular type in this lead; a single anomalous contraction, a premature beat, is seen towards the end of the figure, and its form in the curve is that of a beat arising in the left ventricle. These premature beats are recorded most often in patients who are taking digitalis or an allied drug; appearing in these circumstances, they suggest relaxation of the treatment. When more frequent, they constitute urgent messages that the drug be discontinued; the condition is exemplified by Fig. 83; it is known as "digitalis coupling." Each ventricular complex of the supraventricular type (R, T) is followed at a close and constant interval by a complex of anomalous form. In this instance, the form is that of beats coming from the right ventricle. The ventricular rate is slow; there are no P summits, but there are small oscillations during the diastolic periods. The auricle is fibrillating. A continuation of digitalis, when this slow coupling has declared itself, is culpable; it is followed only too often by sudden and avoidable disaster.

The recently discovered remedy for fibrillation of the auricle, namely, quinidine, is an alkaloid having a very powerful action upon the heart. It is a drug that is to be given only in selected cases; but in these it is capable of restoring the normal rhythm in a very high percentage of instances. The details of treatment and the necessary
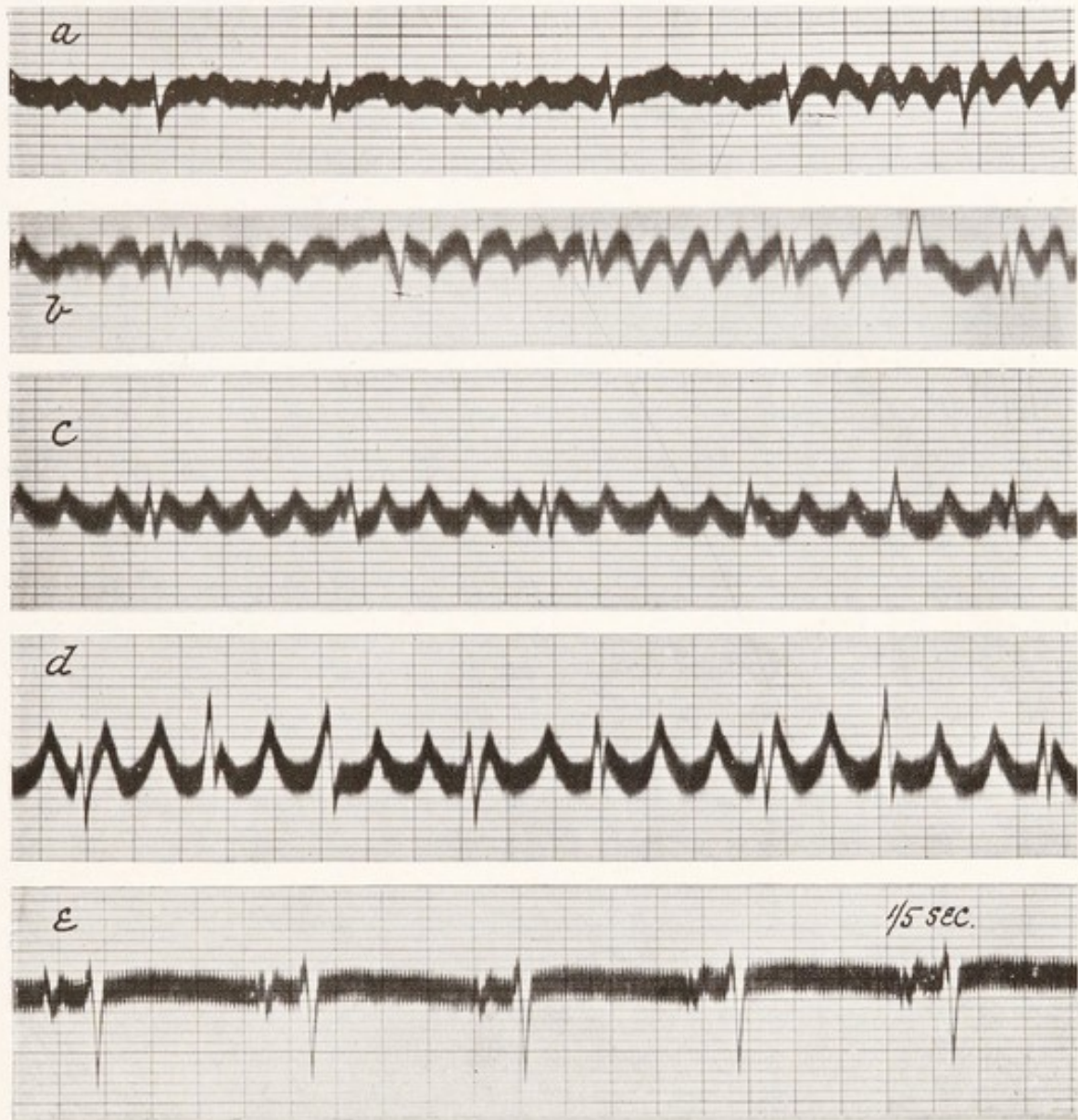
Fig. 84. Five curves taken from a pair of contacts arranged over the sternum
in a case of auricular fibrillation. The time marker is in fifths of a second
in all. (*a*) Before treatment; auricular oscillations at 450 per minute.
(*b*) After 1 dose (approximately 5 grains) of quinidine, the oscillation rate
has fallen to 350. (*c*) At the end of the first day's treatment, and after
3 doses of quinidine, the rate has fallen to 296. (*d*) At the end of the
second day's treatment, and after 6 doses of quinidine, the rate has fallen
to 247 and the oscillations have become almost regular. (*e*) Sixteen hours
later, and after a final dose (the seventh) of quinidine, the normal rhythm
has been resumed.

precautions to be observed do not properly belong to this small treatise, which is intended primarily as an aid to the interpretation of records ; they will be found in my book, " *Clinical Disorders of the Heart Beat.*"   But it is relevant to point out that the electrocardiograph is almost essential in following the progress of such cases under treatment and briefly to state why that is so.   The chief change seen is a slowing of the auricular oscillations ; the rate falls from its original level of 450 or thereabouts, to 300 or even 200 per minute, and the oscillations grow in amplitude and become more regular, until the final picture is similar to or identical with that given by simple flutter.   The lowering of auricular rate, hardly to be detected except electrocardiographically, is premonitory to the final and abrupt change to normal rhythm; whence its importance.   Dosage is largely regulated by the speed and extent of the fall.   Records are most conveniently taken by means of direct leads from the chest wall in the region of the right auricle and are illustrated by Fig. 84.

CHAPTER IX.

SINUS DISTURBANCES AND ALTERNATION.

Respiratory arrhythmia and allied irregularities.

Several closely related heart irregularities exist, which are due to variations in the rate at which the impulses are generated at the physiological pacemaker. A notable example is an irregularity of respiratory origin (Fig. 85) in which the heart beat gradually accelerates during the inspiratory phase and slows during the expiratory phase of respiration. In young subjects it is a normal phenomenon ; and during the earlier periods of life, allied irregularities, in which the whole heart participates, but in which there is no constant relation to the acts of breathing, are observed (Fig. 86). These irregularities, often grouped under the term " sinus arrhythmia," are brought about by changes of vagal tone. In electrocardiograms they are readily recognised. Each beat of the heart is propagated from the natural pacemaker; the electrical curve is consequently formed of physiological complexes, in which the usual auricular and ventricular summits are seen. The disturbance is confined to irregular disposition of the beats ; the normal sequence of chamber contraction is retained.
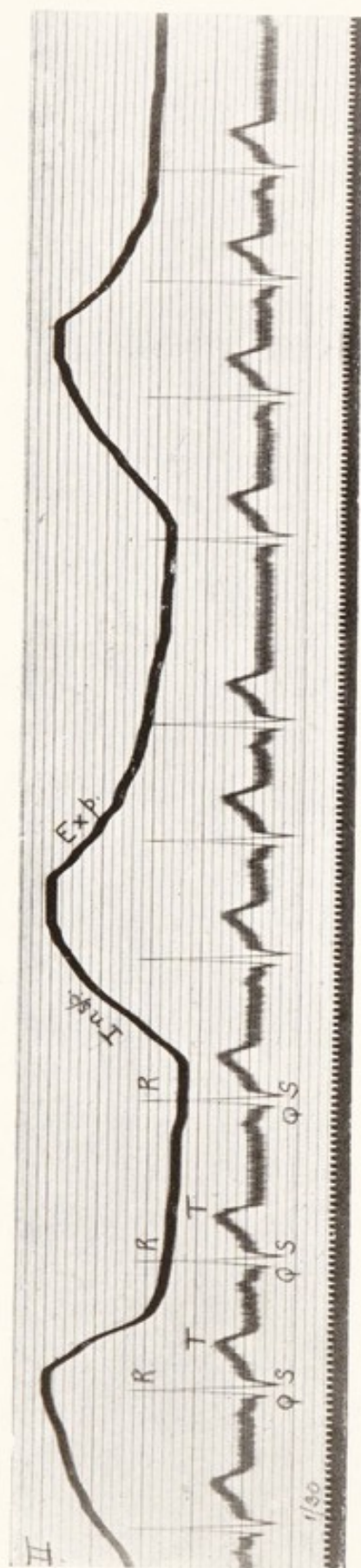
Fig. 85. Electrocardiogram and curve of respiration; showing a sinus arrhythmia. The heart quickens during the period when intrathoracic pressure is low. The whole heart is involved in the irregularity.
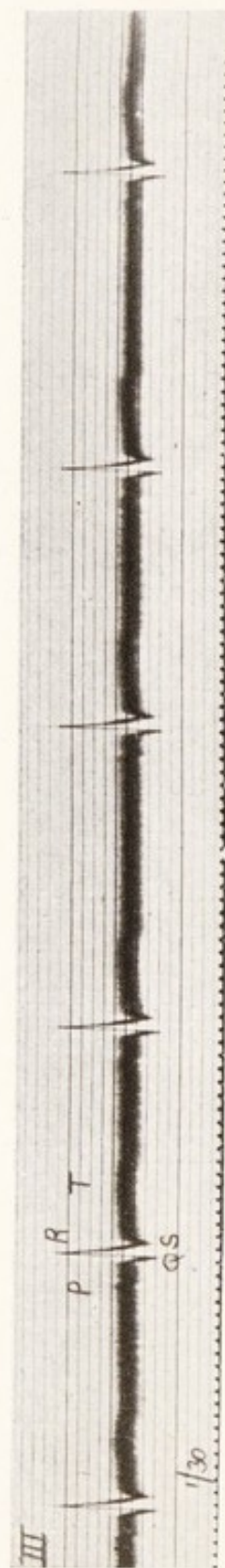
Fig. 86. A sinus irregularity, in which there was no relation to respiration. It was taken from a healthy child, a few hours after birth.

Sino-auricular heart-block.

Another though infrequent form of irregularity is related to those just considered. Provisionally it is termed " sino-auricular heart-block." This irregularity is but imperfectly understood and its significance, though apparently inconsiderable, is not fully known. In many patients it is of vagal origin ; not uncommonly it is associated with auriculo-ventricular heart-block and, like the latter, may appear during digitalis administration. It generally manifests itself in one of two ways, by producing intermittences of the whole heart, or by leading to steep falls of heart rate. When a single heart beat is lost (as in Fig. 87), the length of the longest cycles is usually somewhat shorter than two cycles of the natural rhythm. When the heart rate falls, the passage from one rate to the other is abrupt ; there is no transitional period (Fig. 88) ; although the slow rate may be almost exactly half the former rate (Fig. 88), yet more commonly halving of rate is hardly attained. Permanent slow action of the whole heart may be due to this disorder (Fig. 34), which supposedly is due to imperfect conduction between the natural pacemaker and the main mass of the auricular tissue.

The electrocardiographic curve of each heart beat presents auricular and ventricular complexes of forms known to be associated with heart beats of normal origin.

Alternation of the heart.

When alternation of the heart is present, and is displaying itself in the arterial pulse, the electrocardiograms do not always show it. If the heart is beating slowly and alternation of the pulse is conspicuous, the several ventricular summits and depressions may be of uniform amplitude from beat to
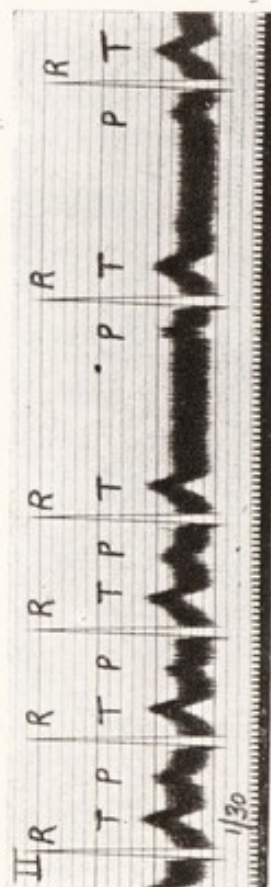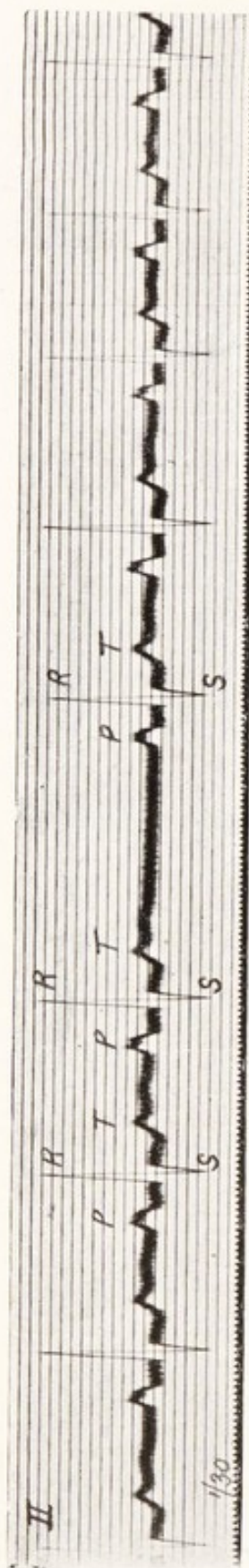
Fig. 87. "Sino-auricular block." An irregularity in which the whole heart is involved. A long pause interrupts what is otherwise an almost regular heart action. The long cycle is nearly, but not quite, twice the length of the short cycle. From a case of mitral stenosis (note the large split P summit). The irregularity resulted from digitalis.

Fig. 88. "Sino-auricular block" in a case of exophthalmic goitre. The rate of the whole heart is suddenly and almost exactly halved. Note the prolongation of the P-R interval in the last cycle of the fast rhythm.

beat (Fig. 89). Occasionally alternation is conspicuous
in the electrocardiogram ; it is seen to advantage in Fig. 63.
In this figure it is increased after the premature contraction.
When alternation accompanies paroxysms of tachycardia,
traces of alternation in the amplitudes of R and T are usual.
The left-hand portion of Fig. 62 serves as an illustration ; the
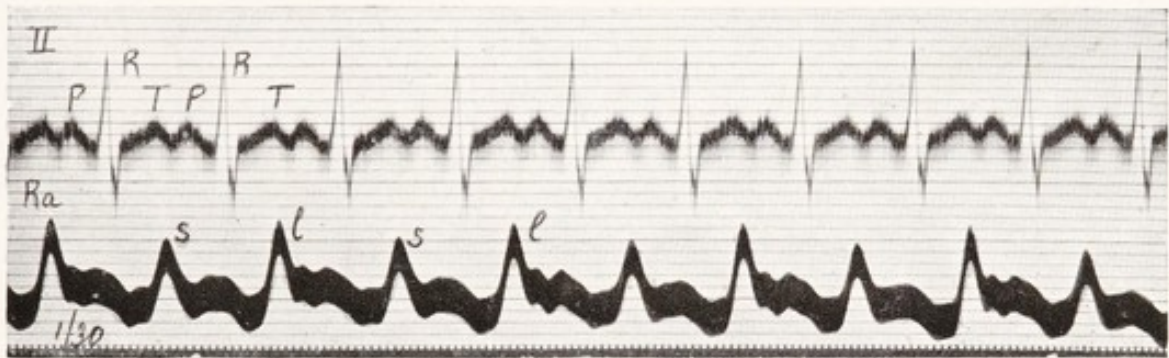
Fig. 89. Simultaneous electrocardiogram and arterial curve. The last
shows conspicuous alternation, but in the electrocardiogram it is not
perceptible (see Figs. 62 and 63).

alternation is confined to R and, though of minor degree,
is quite distinct. Curiously enough, the alternation in
electrocardiogram and pulse is not always parallel ; while
the small R summit may correspond to the small pulse beat,
yet quite as frequently (as in Fig. 62) the large R corresponds
to the small pulse beat. On occasion alternation of amplitude
may be present in the electrocardiogram when the pulse fails
to show it.

CHAPTER X.

SPECIAL CONDITIONS.

Electrocardiograms in valve lesions.

As a preliminary it should be stated emphatically that electrocardiography has little to do with valve lesions. The method is essentially one which investigates the muscle ; only, therefore, in so far as valve lesions affect the muscle do they influence the form of the electrical curves. Now the degree to which valve lesions upset the distribution of the mass of heart muscle is a question upon which we have no final evidence ; there is the primary difficulty that anything affecting the valves may also affect the contractile substance. The indiscriminate assignment of hypertrophy of this or that chamber to leakage at this or that valve as a casual agent is to be deprecated. However boldly it may be asserted in a given case that left- or right-sided hypertrophy is the result of purely mechanical defects, the conclusion in the individual case is often impossible of proof. We know that free aortic leakage may consort with a light or heavy musculature ; we know that, produced under strictly aseptic conditions, reflux at this valve is generally followed by but relatively little muscle change ; again, some of the largest human hearts are found where no sign of valve defect or other mechanical disadvantage, to which cardiac embarrassment
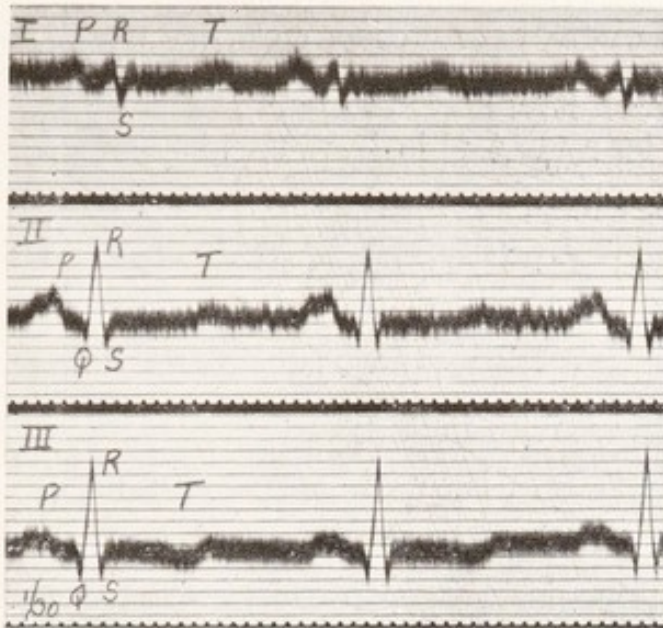
Fig. 90.
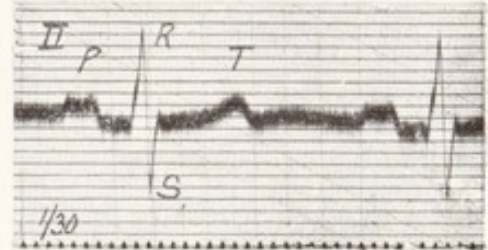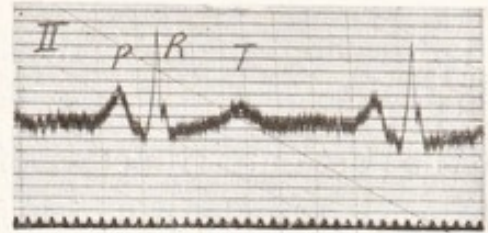
Figs. 91 and 92.

Fig. 90. From a case of mitral stenosis. The summit *P* is large, broad and notched in lead *II*. The initial ventricular peaks of lead *I* are small. *R* is tallest in lead *III*.

Fig. 91. From a case of mitral stenosis, showing a tall and pointed summit *P*.

Fig. 92. From a case of mitral stenosis, showing a broad and notched summit *P*.
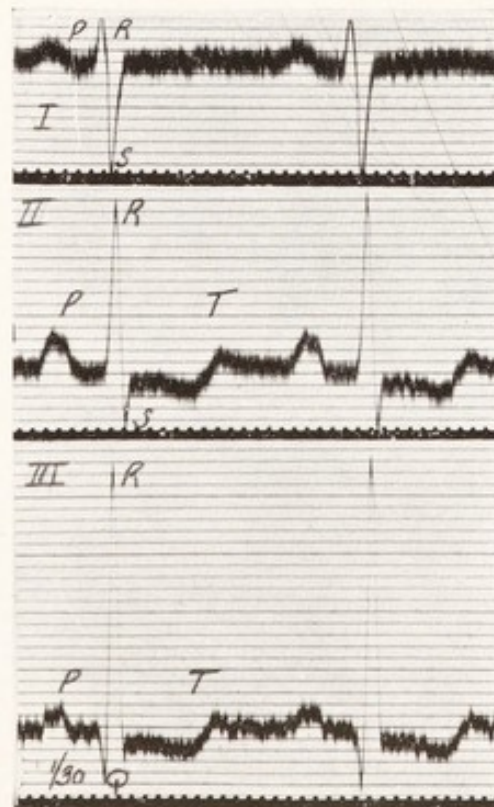
Fig. 93. From a case of mitral stenosis. The summit *P* is large, broad and notched in lead *II*. *R* is of least amplitude in lead *I* and of greatest amplitude in lead *III*; *S* is of greatest amplitude in lead *I*.

might be attributed, has ever been discovered. While
alteration in the muscle as a sequel to pressure changes
cannot be denied, yet it is clear that this hypothesis has in
the past outstripped the facts. These remarks are a necessary
prelude to those which follow ; for if we expect the electro-
cardiograms that are held to depict preponderance of the
right ventricle in all cases of mitral stenosis or those of
preponderance of the left ventricle in all cases of aortic
disease, our expectations will be disappointed. The sign
of left preponderance may appear in mitral stenosis and that
of right preponderance in aortic disease ; and the reason for
what may be regarded as a discrepancy—but which in
reality is not a discrepancy—is not obscure ; the left ventricle
may preponderate in mitral stenosis as the right *may* in
aortic disease.

Mitral stenosis. The electrocardiograms of mitral
stenosis are often so characteristic that the valve lesion may
be diagnosed from these curves alone. The summit P has an
exaggerated amplitude, amounting frequently to 2, 3 or even 4
scale divisions (Figs. 90, 91, and 92 and 93 ; and also Figs.
29, 52, 53 and 87) ; it is often broad, flattened and notched
in the centre (Figs. 90, 92 and 93). The ventricular complexes
are generally those associated with preponderance of the right
muscle. Often this change is shown conspicuously (Fig. 93),
S being exaggerated in lead I and R in lead III. Sometimes
the change is less evident ; small R and S deflections in lead I
(Fig. 90) are not uncommon. When the auricles are fibril-
lating the curves associated with right preponderance remain
(Fig. 95), and the exaggerated P summits are replaced by
exceptionally large oscillations (Figs. 74, 79, 80 and 95).
When new auricular rhythms, constituting paroxysms of
tachycardia, occur in mitral stenosis, the ventricular peaks
may suggest right preponderance, but the P summits no
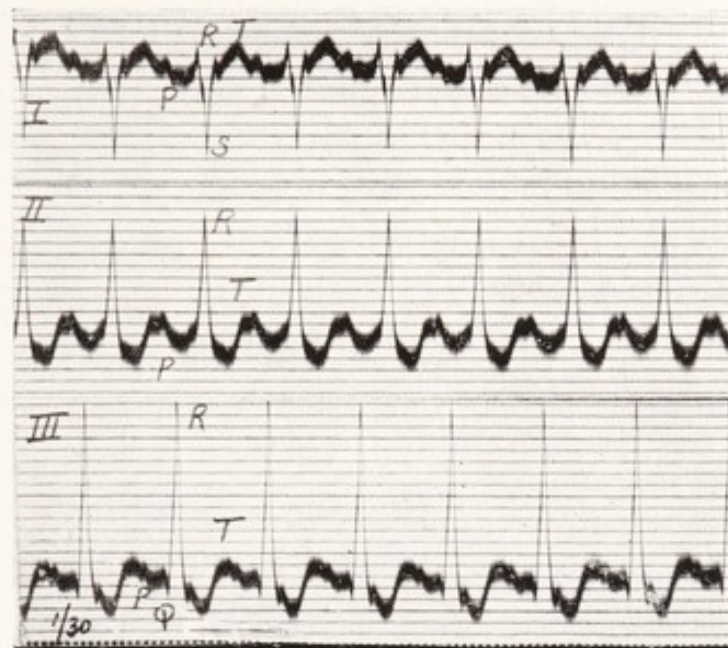
Fig. 94. From a paroxysm of tachycardia in a case of mitral stenosis. Preponderance of the right ventricle is indicated ; the *P* summits are small because the paroxysm arose in an ectopic auricular focus.
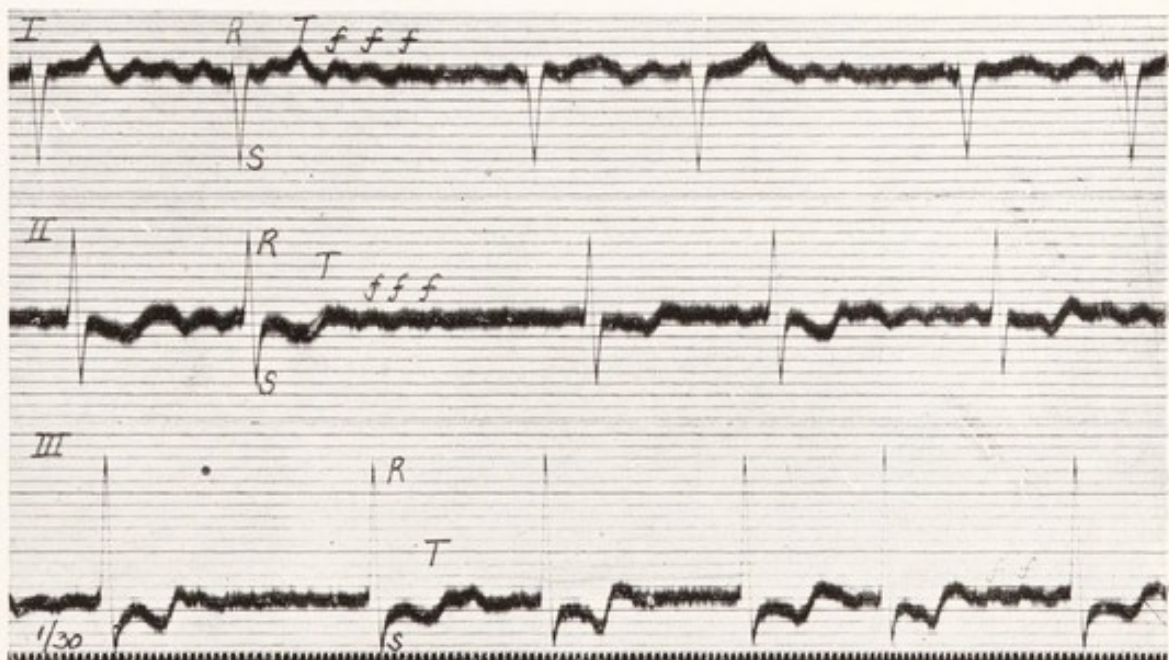
Fig. 95. From a case in which the auricles were fibrillating. Preponderance of the right ventricle is suggested by the ventricular complexes. The oscillations are prominent, as is usual in mitral stenosis.

The heart of this patient was subsequently obtained. Mitral stenosis was found. The left ventricle weighed 105 and the right ventricle 128 grammes.

longer occur in their usual forms (Fig. 94).   The signs
described may lead to a correct diagnosis of mitral stenosis,
which without the signs would be unrecognisable. Especially
is this so when, the auricles being in fibrillation, the presystolic
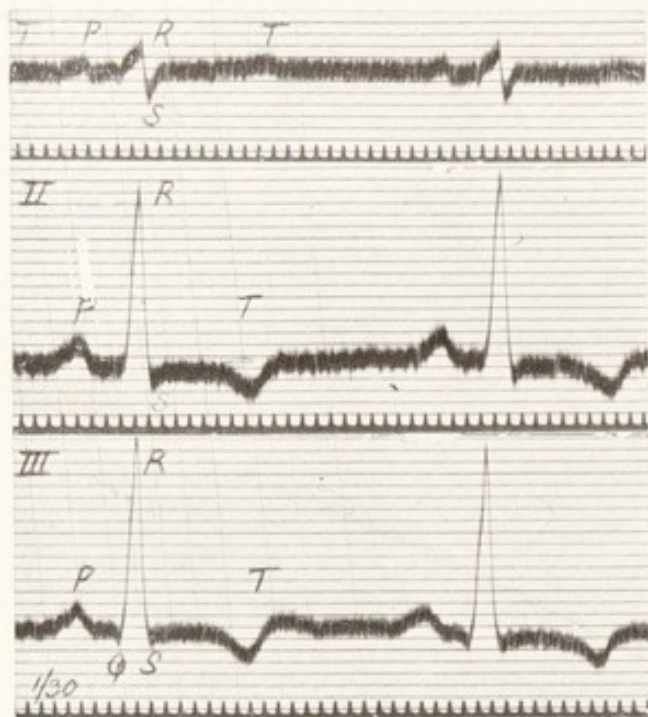
Fig. 96.   From a patient in whom aortic regurgitation was present, showing
inversion of *T* in lead *II*.   The curve is exceptional because it indicates
preponderance of the *right* ventricle.
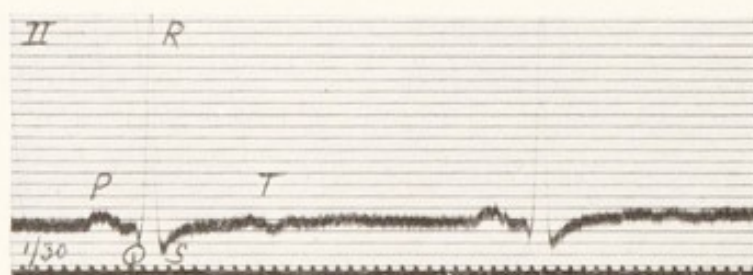
Fig. 97.   From a case of aortic regurgitation, showing the flattening of *T*
that is common in association with this valve lesion.

murmur fails or is replaced by a murmur falling in early
diastole. Similarly used, the records may be helpful also in
differentiating diastolic murmurs of aortic, pulmonary and
mitral origin (Steell's and Flint's murmur).

Aortic disease. The curves of aortic disease are varied in form ; the uncomplicated valve lesion produces little or no alteration. In a dog in which free regurgitation was produced, the electrocardiograms, taken before and some forty days after operation, showed no appreciable change ; such slight change as was exhibited was unstable from day to day. It is true that left ventricular preponderance is often incidated in the curves of the human subject (Figs. 19 and 20),
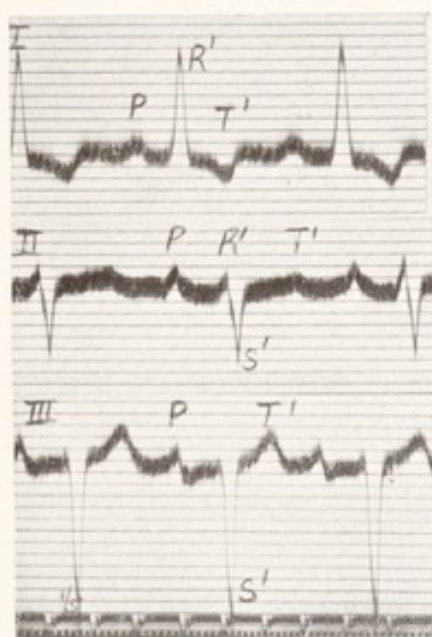
Fig. 98. From a patient in whom aortic regurgitation was present. It serves to emphasise the important distinctions between two series of curves, namely, those associated with left preponderance and those indicating a deficiency of the right branch of the auriculo-ventricular bundle (see Chapter III.).

but it is not indicated with constancy in this condition, and the signs are encountered more frequently when aortic disease is absent. The picture of right preponderance is also compatible with lesions of this valve, though the association is infrequent (Fig. 96). A large excursion of *R* in lead *II* (Figs. 96 and 97), smallness of *T* (Fig. 97) or inversion of the same summit in lead *II* (Fig. 96) are quite common. The variability of the electrocardiograms in aortic disease harmonises

with the findings at autopsy ; these are equally variable. If the muscle of the separate ventricles is *weighed*, an increase in the weight of the left chamber is almost invariably found, but so also is an increase in the weight of the right chamber. The *relative* weights of the ventricles, which influences the electrocardiogram, is normal in aortic disease almost as often as the natural ratio is disturbed by preponderance of the left ventricle.
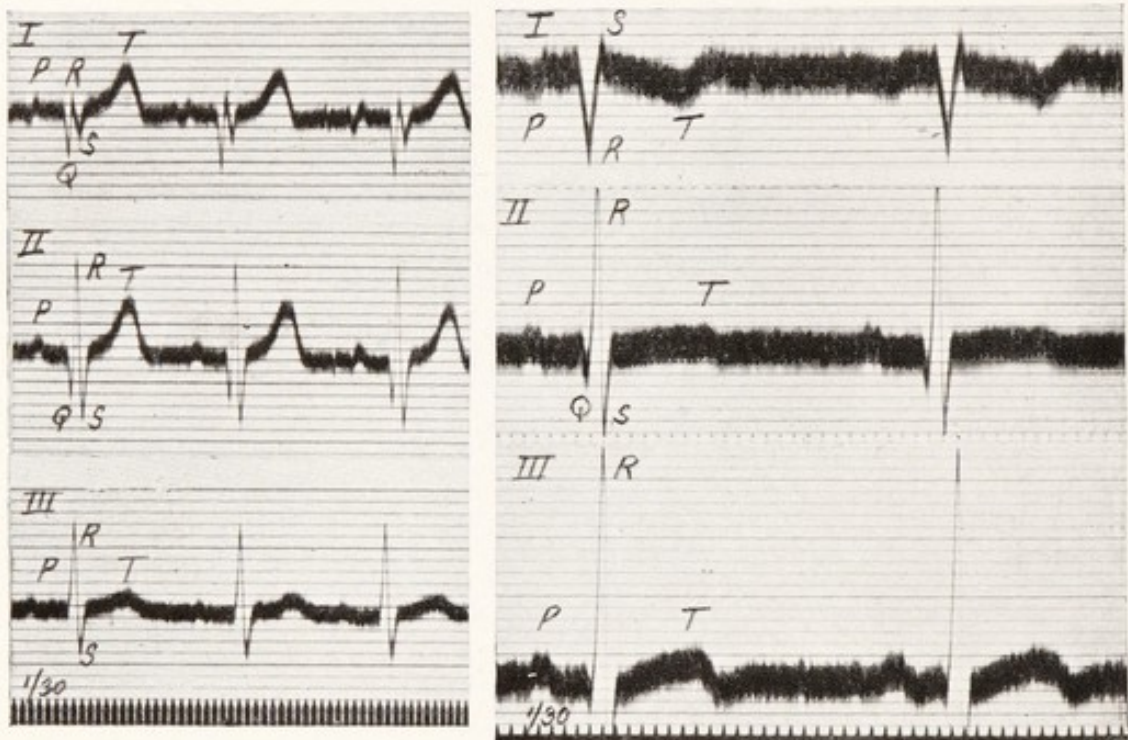
Fig. 99. From a case in which there was conspicuous displacement of the heart towards the right side.

Fig. 100. The curves of a transposed heart. All the summits are inverted in lead *I*.

The electrocardiograms that indicate deficient conduction through the right division of the bundle seem to be more frequent in aortic disease than coincidence would allow ; occurring in aortic disease they may be misinterpreted. Fig. 98 serves as an example. Now these curves were taken at a slow rate, and a cursory examination would suggest

preponderance of the left ventricle. But the initial phases of the ventricular complexes are long, they together exceed a tenth of a second in duration. The direction of T^1 in leads *I* and *III* and the notching of the downstroke of S^1 in lead *II* all point to a branch lesion (compare Figs. 18 and 19). A second example of the same defect in aortic disease has been given already in Fig. 18.
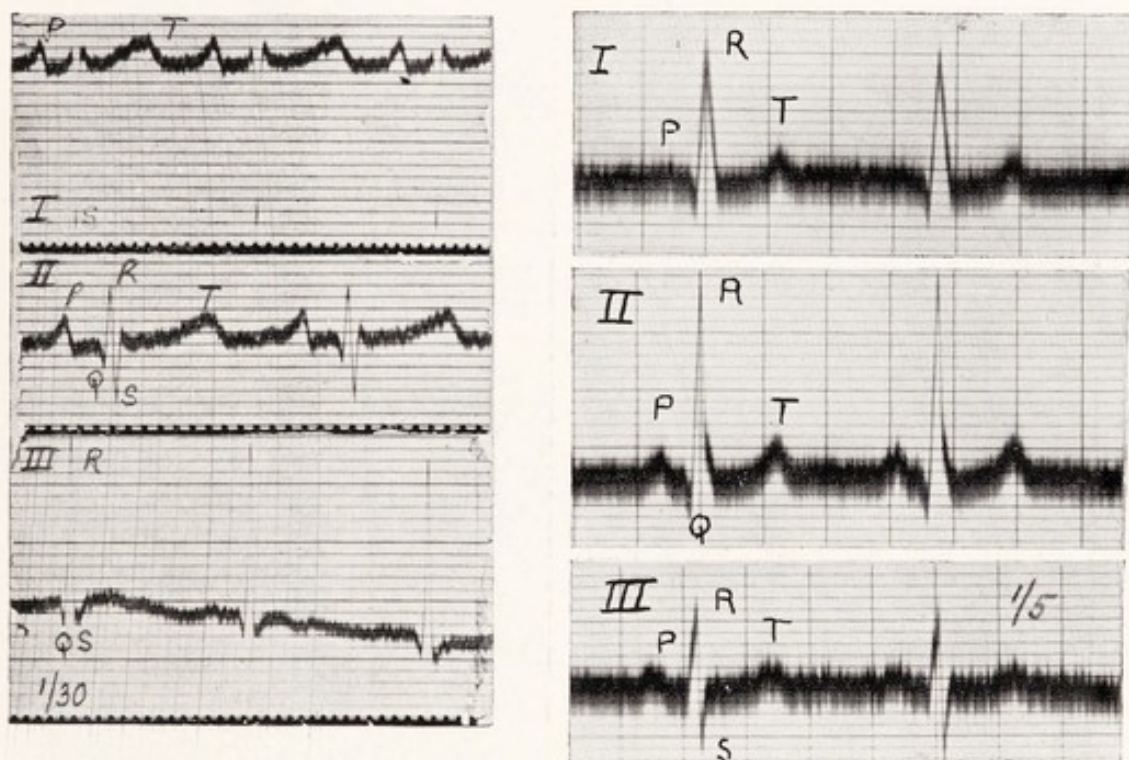
Fig. 101. From a patient in whom there were clear signs of congenital stenosis of the pulmonary artery. Right ventricular preponderance is indicated.

Fig. 102. From a patient aged 21, in whom the ductus arteriosus was subsequently found to be widely patent. The curves are normal.

Mitral regurgitation. The curves show no constant characters.

Congenital heart affections. Here the curves are often of value. The electrocardiogram, when the heart is transposed, offers the most distinctive sign of this abnormality that we
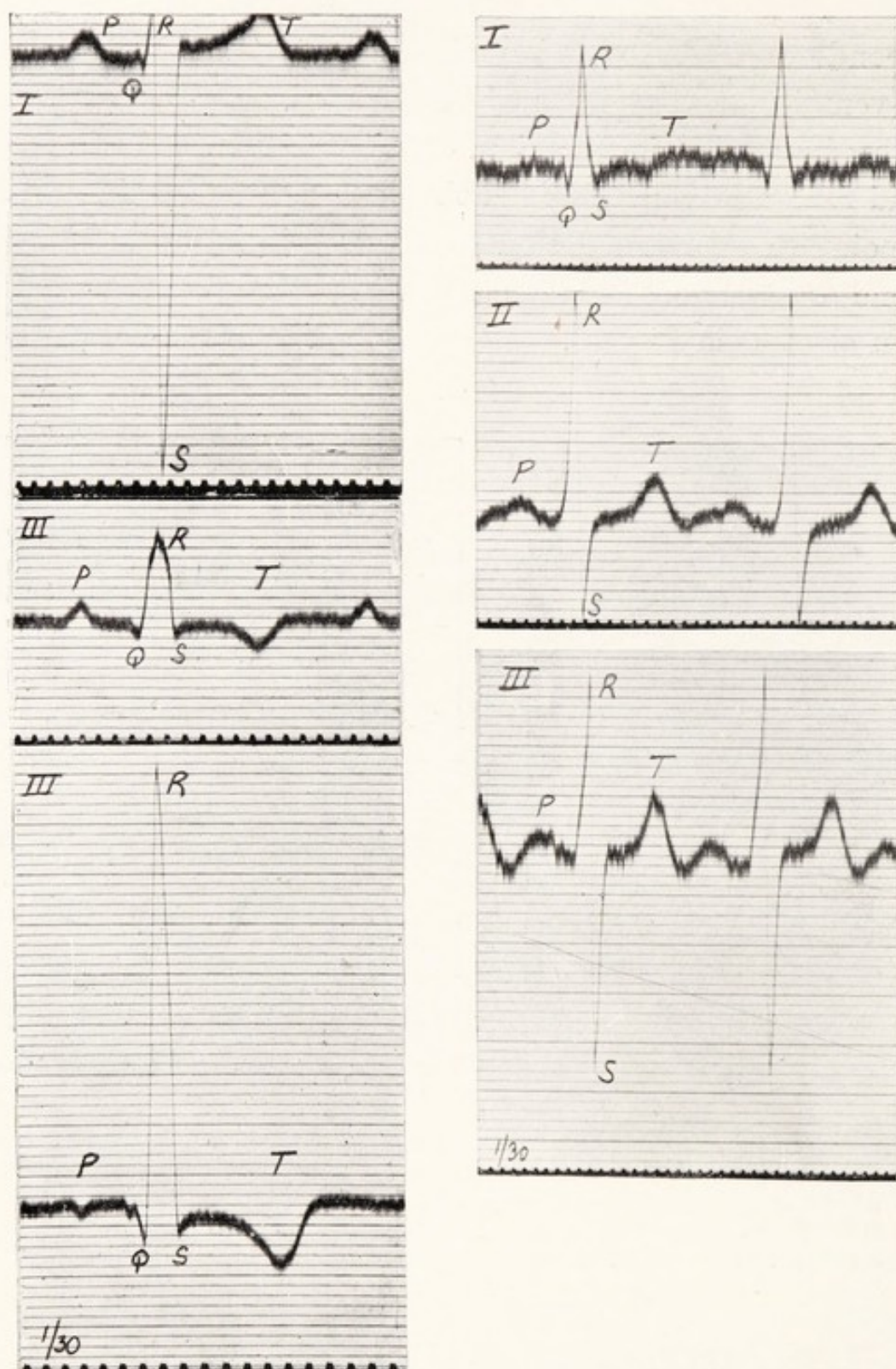
Fig. 103.   From a child in whom there were signs of congenital pulmonary
        stenosis.   Preponderance of the right ventricle is indicated in the curves.
        Note the exaggerated amplitude of excursion in leads *I* and *III*.   The
        heart of this child was subsequently obtained ; pulmonary stenosis was
        found ; the ventricle weighed 67 and the right ventricle 161 grammes.
        The middle curve is from lead *II* and not from lead *III*, as marked.

Fig. 104.   From a lad with a greatly enlarged heart and congenital mal-
        formations.   The chief signs were a systolic thrill, palpable over a wide
        area and maximal at the pulmonary cartilage ; a systolic murmur,
        and prolonged into and enforced in diastole, and maximal in the same
        region ; and a systolic apical murmur.   The right carotid artery crossed
        the trachea.   Note the increased excursion in leads *II* and *III*.

possess, not excepting the skiagram. All the summits of lead *I* are inverted (Fig. 100). A little consideration will make it clear how this change comes about. The lead is a symmetrical one, being from the right arm to the left arm. A lead from the *left arm to the right* inverts the picture in the normal subject, and the left arm stands to the normal subject as does the right to the subject of transposition. Neither of the other leads is symmetrical, and neither of the other leads shows inversion. For example, a lead from the right arm to the left leg in any subject gives almost precisely the same picture as does a lead from the right arm to the right leg. So it happens that a lead from the right arm to left leg (lead *II*) in a normal subject is almost equivalent to a lead from the left arm to the left leg (lead *III*) in the subject of transposition. In the subject of transposition, while *the curve from lead I is inverted, those of leads II and III replace each other.* The value of these curves will be evident ; displacement of the heart distorts electrocardiograms, but does not induce these changes. An example of extreme displacement is illustrated by Fig. 99. It was taken from a boy ; the heart's impulse was found well to the right of the sternum, and no reason for displacement was apparent. The question of transposition arose, but the electrocardiograms denied the presence of this anomaly, as did also the skiagram ; the chief distortion is in the opening ventricular events of lead *I*.

In those children in whom cardiac enlargement, cyanosis and harsh systolic thrills and murmurs, maximal over the pulmonary cartilage, are the chief diagnostic signs, the electrocardiograms generally indicate extreme degrees of right preponderance. These are instances of pulmonary stenosis. Fig. 101 exemplifies the curves of this condition; Fig. 103 is an exceptional example from a similar case and

illustrates another feature of congenital heart curves, namely, extreme amplitude of excursion. Exaggerated amplitude in several leads is in itself a valuable sign of congenital valve or septal defects. Another example, Fig. 104, shows this increase in the excursion. Such curves are obtained, so far as I know, only when there is reason to believe that congenital malformation is present. It is impossible to speak at all fully of these electrocardiograms at the present time ; we require more information regarding them, and especially a careful comparison with post-mortem material. If there is one congenital defect in which the electrical curves should be normal, it is in uncomplicated patency of the ductus arteriosus. Fig. 102 was taken from a young man who exhibited a continuous harsh murmur over the pulmonary area. Death occurred from cerebral tumour 7 years later and disclosed a widely patent ductus. The heart and vessels weighed 457 grammes. The curves present no abnormality.

Renal diseases and high blood pressure. The electrocardiograms of chronic renal disease are not distinctive. The curves may suggest preponderance of right or left ventricle, or the relative amplitudes of R and S in the several leads may be normal. However, curves of left preponderance are frequent in patients whose systolic blood pressure is high. These records are quite consistent with the ventricular weights in chronic renal disease ; these weights show the ventricles to be equally hypertrophied in the average. Some of the hearts show preponderance of the left ventricle, notably those that have pumped against high arterial pressures ; other hearts show right preponderance.

Exopthalmic goitre. It has been said that T is exaggerated in this condition. I have examined a number

of such patients and, apart from the enhanced rate of beat
and slight changes resulting therefrom, no definite alterations
have been seen (see Figs. 56, 78 and 88) in the shape of the
ventricular complex.

Coronary artery thrombosis.

These curves are similar to those obtained experimentally
by injuring a considerable area of muscle at the base or apex
of the heart. They vary much in form, being sometimes
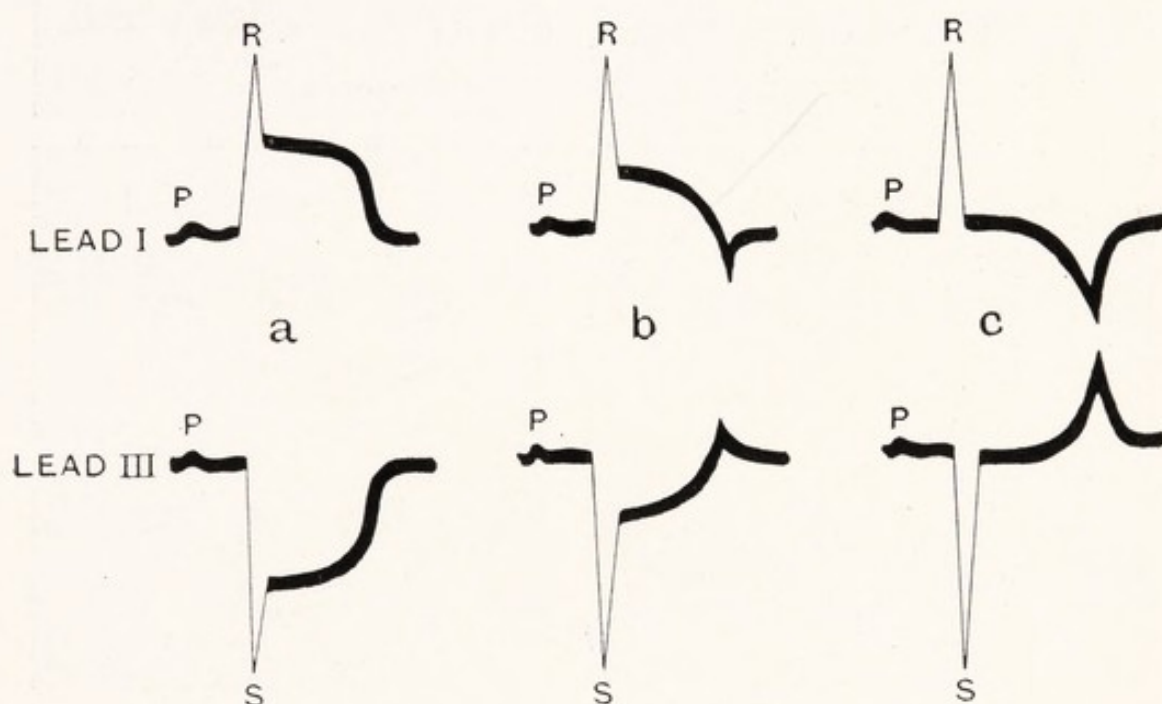
Fig. 105. A diagram after Parkinson and Bedford, illustrating a form of
electrocardiogram characteristic of coronary arterial thrombosis.
Immediately after the accident the form is that shown in *a*, but this
gives place by transition within a few days or weeks to curves (*b* and *c*)
in which *T* is inverted in lead *I* or *III*. Transpose the curves of *lead I*
and lead *III* in the diagram, and they remain equally representative
of coronary thrombosis.

quite characteristic, at others more obscure. The most
easily recognisable form (Fig. 105*a*) is that in which the des-
cent of the deflection *R* or the ascent of *S* is incomplete, the
curve running a more or less horizontal course on one or other
side of the zero position until it fuses with *T*. In other
words the usual return of the string to an isoelectric position

between the inscription of the initial deflections and the final
deflection (*T*) does not occur. This almost monophasic type of
ventricular complex may be directed upwards or downwards
in lead *I* (or in lead *III*); it is oppositely directed in leads *I* and
III (see Fig. 105*a*). It is unstable, disappearing within the
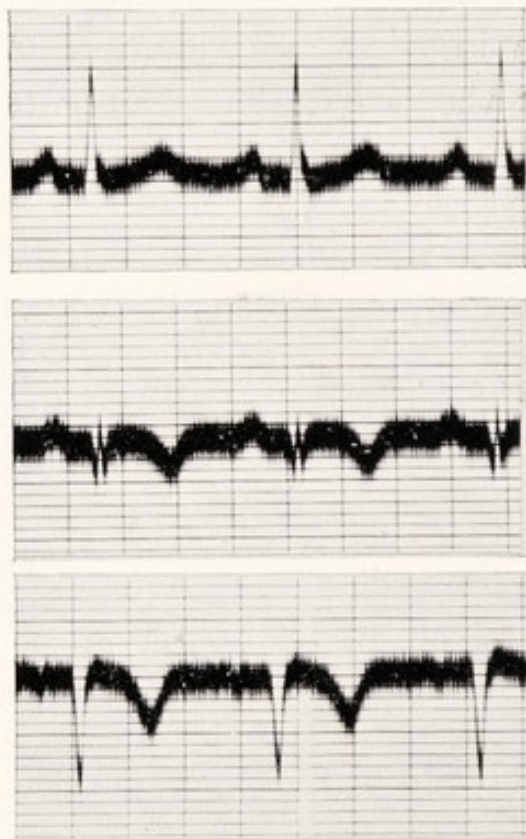
Fig. 106*a*.   Fig. 106*b*.

Fig. 106*a*. Curves taken from a man of 48 years 2 hours after the onset of
characteristic symptoms of coronary artery thrombosis. Starting with
anginal pain, fever followed on the next day and was associated with a
heavy leucocytosis. Curves taken 16 days later are shown in Fig. 106*b*
and display the usual changes in the *T* waves. I am indebted for these
and the next curves to Dr. H. M. Marvin.

course of a few days or weeks and giving place to curves in
which *T* is inverted in either lead *I* or lead *III*. In each
of these leads *T* then assumes a direction the reverse of the
original main deflection. This series of changes in the

electrocardiogram is well illustrated in the accompanying diagram (Fig. 105). The curves are often of rather low voltage (Fig. 107).

Less easy to recognise are instances in which the descent of R or ascent of S to the zero line is more nearly accomplished, so that the horizontal stretch of curve in the centre of the ventricular complex lies closer to the isoelectric position;
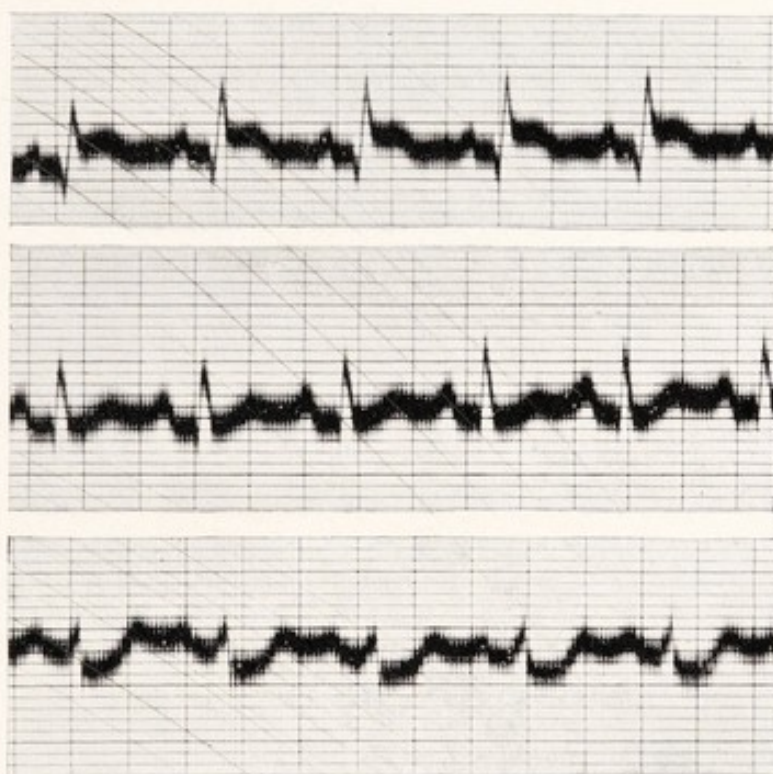
Fig. 107. From a man aged 55, 18 hours after violent epigastric and substernal pain. Subsequently fever, leucocytosis and pericardial friction developed. At the autopsy a fortnight later, complete closure of the anterior descending coronary artery with a large infarct at the apex of the heart was found.

so also are other instances in which the central portion of the complex lies away from the zero position but on the opposite side of it to the main initial deflection, R or S (see Fig. 106a). These types are also transient, passing over as previously described into curves in which T is inverted; here too the

direction taken by T is opposed to the original deviation of the central part of the complex from the zero position. These changes in T may be permanent or, after many weeks or months, the normal form of electrocardiogram may be restored.

These clinical curves, first described by H. E. B. Pardee, and recently in much detail by Parkinson and Bedford, are often crucial to the diagnosis of thrombosis of the coronary vessels.

INDEX.

INDEX.