

6121711

A RECENT METHOD OF STUDYING THE ACTION

OF THE HUMAN HEART, WITH SPECIAL REFERENCE

TO AURICULAR FIBRILLATION

-BY-

ALEC W. GILL M.B., Ch.B.,



During my term of Office as House Physician to Addenbrooke's Hospital, Cambridge, I have had an opportunity of studying and have paid special attention to many interesting diseases of the heart.

A large number of these cases have been associated with some irregularity of Rhythm.

Their study has been rendered more valuable by the fact that I have had access to tracings of each heart as shown by the Einthoven String Galvanometer, and in the majority of cases to tracings taken with Mackenzie's Polygraph.

During the last few months, I have examined several cases exhibiting the features of Auricular Fibrillation. The features of this disease have recently been clearly defined by several observers, notably Dr James Mackenzie and Dr Thomas Lewis, the former working with the Polygraph, the latter with the String Galvanometer.

The researches of these men have demonstrated that auricular fibrillation is quite a common occurrence in the human heart, constituting as it does about 50 per cent of the arrhythmias of the heart.

I propose in this paper to describe the usual characteristics of these cases with special reference to the String Galvanometer and the characteristic tracings obtained by its use.

The study of the irregularities of the heart has interested clinicians for many years, but it is only

since the introduction of methods of graphically recording the cardiac mechanism that any progress has been made in this direction. For many years the Sphygmograph was the only instrument in use, but as it recorded only the movements of the Radial pulse and therefore indirectly the action of the left ventricle, little advance was made.

Mackenzie, recognising the shortcomings of this instrument, devised the Polygraph, by which he was able to record the movements of the jugular pulse, Carotid or radial artery, and apex beat simultaneously. In this way, he could study the action of the other Cardiac chambers.

From a careful and systematic study of the arrhythmic cases under his care, he was able to classify them into certain well defined groups.

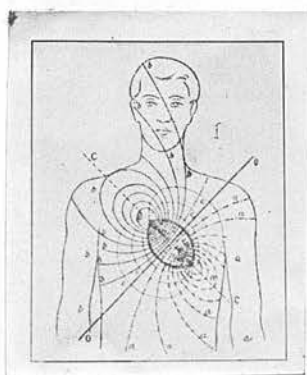
In one class of case, namely, that usually associated with an extreme disorder of rhythm, occurring particularly in old standing cases of Mitral Stenosis, he noticed that all signs of the normal auricular activity were absent.

In attempting to explain this absence he was guided by the conditions found at autopsy. He explains ⁽¹⁾ That in several cases where these features had been observed during life, the auricle was found at post mortem examination distended and thin walled. He therefore concluded that the absence of all signs of auricular systole was due to

the auricle having become atrophied and paralysed. This view was shortly afterwards withdrawn owing to the fact that similar cases showed that instead of the auricle being thin walled it was hypertrophied. In addition it was difficult to see how paralysis of the auricle accounted for the marked irregularity. He then suggested that the stimulus for contraction arose at a site lower down than usual and that the stimulus passing up to the auricle and down to the ventricle caused a simultaneous contraction of these chambers. Fixing on the auriculo-ventricular node as the possible site, he named the condition "Nodal Rhythm".

It is now known that neither of these explanations is the true one. The true solution of the problem only became apparent with the introduction of the StringGalvanometer.

The study of the Electrocardiogram is not new to physiologists as A.D. Waller in 1887 studied the changes of electric potential that occurred in the heart during its contraction. He even constructed a diagram showing the lines of diffusion of these changes of potential on the surface of the body. (Fig. 1)



These changes of potential are, however, very small measuring only anything up to .5 millivolt.

(2) Hirschfelder gives them as $\frac{1}{10,000}$ to $\frac{1}{3000}$ of a volt.

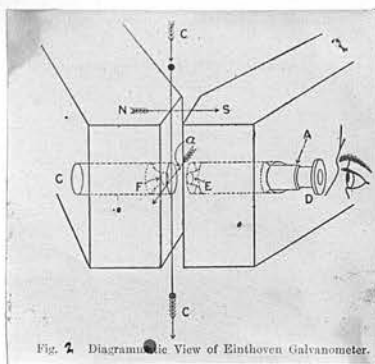
The capillary electrometer used by Waller at this time though sensitive was not quick enough in response to record these changes satisfactorily. In 1906, Einthoven succeeded in making a galvanometer sufficiently sensitive to record accurately the changes of potential of the heart. The records obtained by this new instrument are very accurate, but it is somewhat complicated and requires no little skill for its manipulation. The records used to illustrate this paper have been taken by Dr Shillington Scates, Physician in Charge of the Electrical Department at Addenbrooke's Hospital, who has kindly allowed me to use them for this purpose.

The principle of the String Galvanometer is simple. It depends upon the fact that a magnetic needle is deflected when an electric current is passed through a wire in its vicinity (Howell) ⁽³⁾

The needle will be deflected to the right or to the left according as the current passes in one direction or the other. The effect of the current upon the needle increases with the number of turns of wire and with the current. A similar result is obtained if a wire be suspended between the poles of an electromagnet on the principle of the D'Arsonval Galvanometer, the moving coil in this case being represented by the suspended wire.

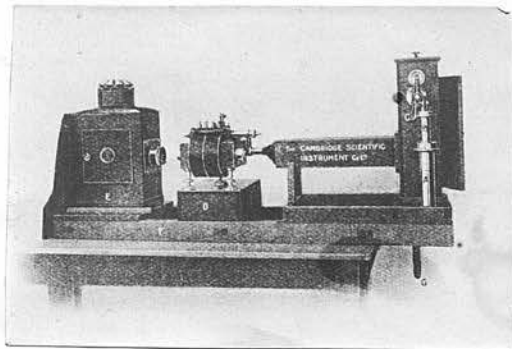
In order to make the instrument as sensitive as possible, Einthoven suspended a very fine quartz fibre, silvered to make it a conductor, between the poles of a powerful electromagnet. The quartz fibre is so fine as scarcely to be visible to the naked eye ⁽⁴⁾ and measures about 2.5 microns (James and Williams)

The fibre moves at right angles to the "field" and will vary in direction according to the direction of the current. (Fig. 2)

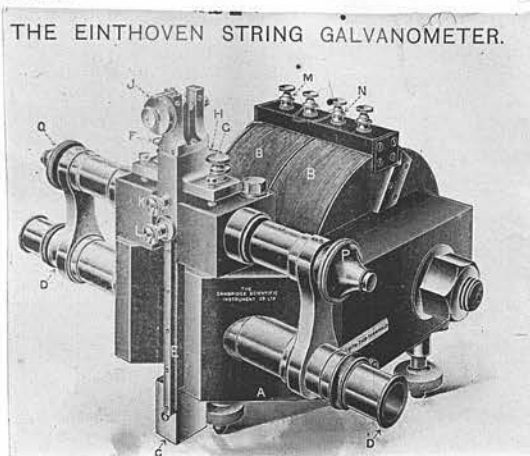


(N,S, = the poles of the magnet)
(cc = the string or fibre)

The poles of the magnet are perforated, the one containing a condensing system, the other a projection system. A powerful arc light acting in conjunction with these and through a cylindrical lens causes the shadow of the string to be magnified and focussed upon a sensitive photographic plate which moves in a vertical direction and so photographs the movements of the string. (Fig. 3)



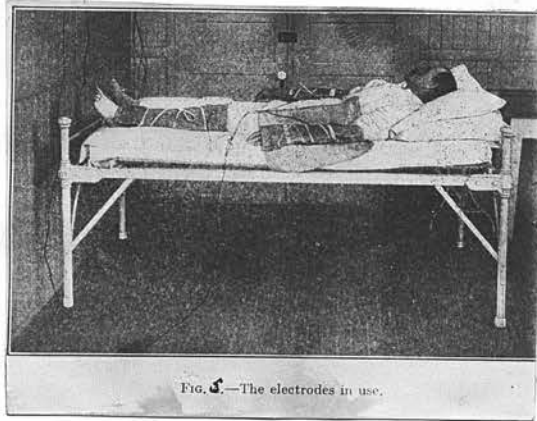
By means of a micrometer screw, the tension of the string may be altered so as to vary the sensitiveness of the galvanometer. The string is in connection with two terminals to which the wires are attached which convey the current generated in the patient to the galvanometer. Figure 4 represents a photograph of the galvanometer.



These wires are connected to the Patient by means of Electrodes. The latter may consist of vessels

containing solution into which the limbs are immersed and which act as non polarisable electrodes.

The electrodes which we have used at Addenbrooke's Hospital consist of thin, flexible sheets of Aluminium covered with thin felt. These are thoroughly soaked in normal saline solution and tied about the extremities with tapes. In this way dangerous cases need not be disturbed at all. (Fig.5)



We use this method because the Wards of Addenbrooke's Hospital are some considerable distance from the laboratories, and the electrodes are connected to the galvanometer by overhead wires. Certain other appliances are necessary to complete the apparatus.

The resistance of the body of the Patient and of the electrodes has to be equalized. This is accomplished by means of resistances and adjusted by a telephone in connection with a small faradic battery, or by means of a Wheatstone's Bridge.

In addition, we require some "Null" apparatus to compensate for the deflection of the string which occurs as the result of the skin current, Generally

assumed to be produced by the activities of the various skin glands etc.

Lastly, in order that records taken in this way may be easily compared, we require to have the string at a known degree of sensitivity. That originally proposed by Einthoven and still followed is, that the string should move one centimetre when a difference of potential of one millivolt is applied to the terminals. To attain this standard, some appliance for regulating the voltage or amperage is necessary.

When all these points have been attended to the Patient is ready for the photograph. The Patient must be warned to keep still as sudden muscular movements by producing great changes of potential might break the delicate fibre as well as prejudicially affect the tracing.

The whole process of actually recording usually takes from three to five minutes in expert hands.

In every case it is advisable to take photographs or electro cardiograms from three different "leads" or "derivations".

Those commonly used and known as Leads

I. II. III. are respectively:-

1. Right arm and left arm.
- II. Right arm and left leg.
- III. Left arm and left leg.

The electro cardiograms from these leads differ slightly from each other in certain important characters and are useful in forming a diagnosis. The records from Leads I. and II. usually give the best tracings.

In interpreting the electro cardiograms, certain facts must be borne in mind.

The surface of that portion of a muscle which is active becomes "negative" to the parts at rest. As the contraction spreads along the muscle, each part as it enters into contraction becomes electro negative to the parts at rest.

This wave of "negativity" or as it is better expressed, this change of electric potential is known as the Action current.

Fig. 6.

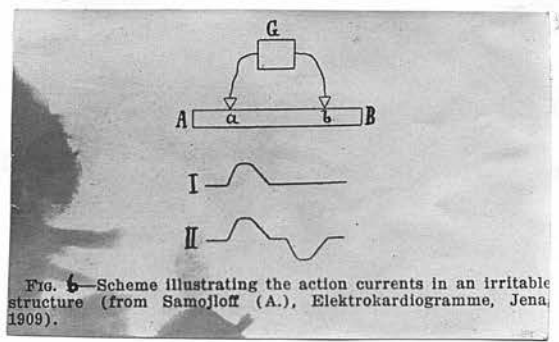
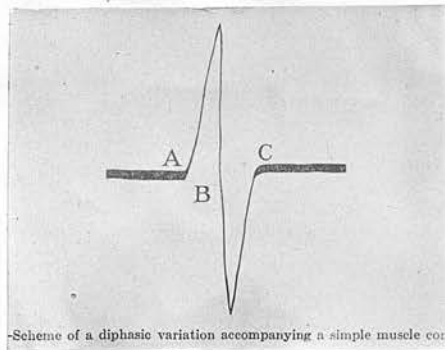


FIG. 6—Scheme illustrating the action currents in an irritable structure (from Samojloff (A.), *Elektrokardiogramme*, Jena, 1909).

If we take a strip of muscle A.B. apply electrodes at (a) and (b) and connect with a galvanometer, the contraction starting at A. will cause a deflection of the needle in a definite direction. As the contraction passes along the strip, the needle will return to its original position and when the contraction reaches B. the needle will deviate in the opposite

direction. The effect is therefore a diphasic one and may be represented diagrammatically thus. (Fig.7)



By photographing this deflection we can state where the contraction began.

The heart, however, is not a simple muscle strip like the example just chosen and in order to trace the path of the contraction wave through the heart, it is necessary to look at its development.

In an early stage of development, the heart appears as a simple tube. The posterior end of this tube receives the veins from the body into a pouch called the Sinus Venosus. This end of the tube appears to be the most excitable as the contraction commences here and passes along the whole length of the tube. As development proceeds this tube becomes bent upon itself and pouches appear upon it which ultimately become the auricles and ventricles. Still later the sinus venosus and the parts of the original tube which connect the auricles and ventricles become incorporated with the latter structures and lose their power of contraction. These portions which cease to exist as a tube are

believed to form a connecting band between the auricles and ventricles and in addition to play an important part in initiating the stimulus of contraction.

Recent observations have shown that remnants of these rudimentary structures still exist in the human heart.

Several definite nodes and bands have been discovered, some of which have very important functions and require mention. The most important of these are:-

1. The Sino-Auricular Node.

This node of tissue is found at the mouth of the Superior vena cava. It was first described by Keith and Flack and is often known by their names. This node consists of fine, delicate fibres faintly striated and is supposed to represent the remains of the original Sinus venosus.

2. The Auriculo-Ventricular Node.

This node is found near the mouth of the Coronary Sinus in the right auricle and was first described by Tawara. It is similar in structure to the Sino-Auricular node. From this node arises the third important structure namely,

3. The Auriculo-Ventricular Bundle.

Arising from the auriculo-ventricular node this band passed to the upper end of the interventricular septum and here divides into two main branches, one branch passes to each ventricle

and eventually breaks up into an extensive and complex subendo cardiac network of swollen, faintly striated cells known as the Fibres of Purkinje (5) (Lewis).

This bundle in the upper part of its course consists of cells of undifferentiated protoplasm with a large nucleus and slightly striated at the circumference. This bundle was first demonstrated by Stanley Kent and experiments by Kent, His and others tend to show that it conveys the stimulus from auricle to ventricle.

The actual starting point of the heart's contraction is still uncertain, but the Sino-auricular Node probably initiates the contraction. Other remnants of the Sinus venosus may be found, but the Sino Auricular Node is the only definite one known at present. From this node, the contraction is supposed to spread to the auricle and then passes by the auriculo ventricular node and bundle to the ventricle.

(6) and (7)
Experiments by Gotch tend to show that the contraction through the ventricles follows the direction of the original tube. He has demonstrated that an area at the root of the aorta is the last part of the heart to contract and represents the homologue of the Bulbus Arteriosus in the lower animals. These facts are of importance as they form perhaps the most satisfactory way of explaining the characters of the electro cardiogram, as will be seen later.

With these facts made clear, I shall now proceed to discuss the changes in and clinical features of Auricular Fibrillation.

The condition of fibrillation refers to that condition where the muscle fibres instead of contracting co-ordinately contract inco-ordinately and independently of one another.

These conditions may be produced experimentally by sending a rapid series of electric stimuli through the heart. The changes which occur are reported by Cushman (8) and by Cushman and Edmunds (9). They found that if the ventricle was sent into fibrillation, death rapidly ensued from failure of the circulation. On the other hand, when the auricle was experimented upon the circulation continued. They found that when Auricular fibrillation was produced in the dog, it was invariably associated with a rapid and very irregular arterial pulse, similar to that which had long been a recognised feature in the human being of old standing cases of Mitral Stenosis. It was therefore suggested by them that this was the cause of the irregularity in these cases. Several people were incredulous, the main objection being that auricular fibrillation could not go on for a long period of years as these cases of Mitral irregularity were known to do.

The String Galvanometer was then utilised and it was discovered that in each case, there was no trace of the normal auricular complex, but that there were numerous fine oscillations which were undoubtedly produced by the fibrillating muscle.

Lewis also proved that these oscillations were maximal when one electrode was placed over the right auricle.

Such a condition has never of course been actually seen in the human being, but a similar condition occurs in the horse. ⁽¹²⁾ Lewis has had the opportunity of seeing an auricle in fibrillation in a horse which had just been killed. The auricle was found distended and ballooned and no complete systole was accomplished, yet the wall of the chamber appeared to be in a constant state of movement. "Rapid, minute and constant twitchings or undulatory movements are observed in a multitude of small areas upon its surface" (Lewis).

There is no reason why this condition should not occur and persist for some time in the human heart. The auricles are really reservoirs which supply the ventricles and are not really essential to the circulation.

In addition, this condition fully explains the extreme disorder of rhythm which ensues.

Each of these vibrating areas would appear to be capable of originating a stimulus and exciting the ventricle to contract. Haphazard and irregular impulses pass to the ventricle as rapidly as they can be conducted along the auriculo ventricular bundle. The rate will depend upon the power of the bundle to contract and individual cases vary from 30 to 200 per minute.

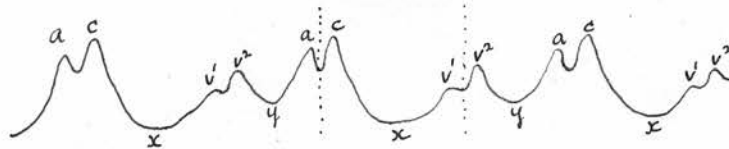
This condition is now known to be a very common affection. It is the usual cause of the irregularity in Mitral Stenosis and in Senile cases with Cardio Sclerosis. As regards the etiology it occurs most frequently after an old standing rheumatic affection of the heart. (13) Lea found that a history of Rheumatism or Chorea could be obtained in 56.6% of cases of Auricular Fibrillation. This fact will be emphasised in the special cases.

Although most common with Mitral Stenosis, it often occurs without valvular disease. The actual valvular disease is not the serious factor. It is the changes that are set up in the muscular wall that lead to fibrillation. The actual nature of these changes is not yet determined. In the majority of cases examined, changes of a chronic inflammatory nature were found with an increase of fibrous tissue and of nucleated cells. It is apparent, however, that Fibrosis itself is not the only cause as cases showing obvious fibrosis have frequently occurred without the occurrence of fibrillation.

Whatever be the real cause, it is a progressive one and leads sooner or later to heart failure. The heart is usually dilated, due no doubt to exhaustion, the result of the rapid and irregular stimulation.

Before discussing the clinical features which include the characteristic phlebograms and electrocardiogram, I shall review the normal records.

The Venous Pulse is sufficiently well known not to require a detailed explanation. On it are found several distinct waves of which the first one called (a) is due to the auricular systole. About $\frac{1}{5}$ second after (a) comes another wave due to transmitted pulsation from the carotid artery (c). This is followed by a depression (x) occurring during ventricular systole and then another wave (v) often marked off into two distinct waves (v^1) and (v^2). The notch between (v^1) and (v^2) marks the end of ventricular systole (see diagram)



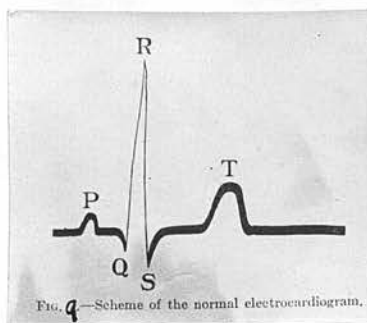
In cases of auricular fibrillation, as we have seen the notable feature is the total absence of the (a) wave due to auricular systole. This type of pulse has been designated by Mackenzie "ventricular" in contradistinction to the normal or "auricular" type. The extreme irregularity will also be apparent and if the heart is acting slowly, a series of fine oscillations is sometimes seen in the diastolic period. These are produced by the fibrillating auricle.

Examples of this type of venous pulse will be shown later.

The normal and abnormal electro cardiograms require more attention.

The normal electro cardiogram consists of three waves in an upward direction and two smaller waves in a downward direction these latter sometimes barely evident. Those directed upwards seem to indicate a relative negativity of the base to the apex; those with a downward direction have the opposite significance.

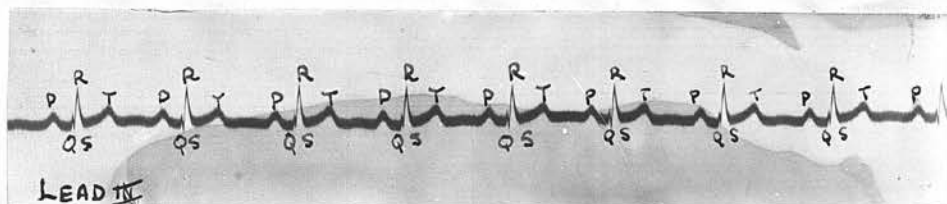
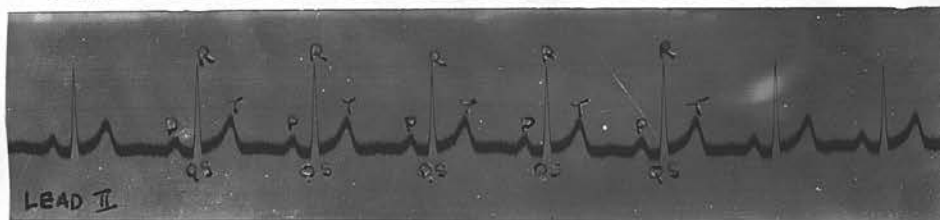
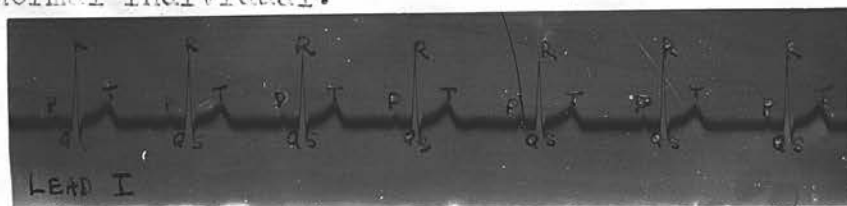
Figure 9 shows diagrammatically the characters of the normal electro cardiogram:-



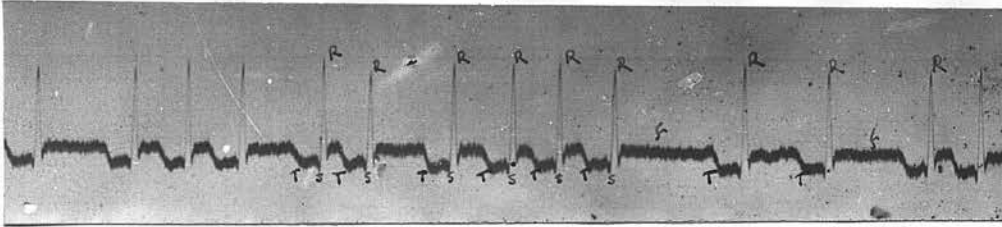
The waves are labelled for convenience P.Q.R.S.T. of which P.R.T. are directed upwards while Q.and S. are directed downwards. Of these waves P. is due to the contraction of the auricle. This has been proved both by actual observation and by its dissociation from the rest in cases of Heart Block. The other waves Q.R.S.T. are produced by the ventricle. Of these R. and T. are the most constant and most

important. Q. and S. are not always present and their significance is not quite understood.

P. is a small, rounded elevation. Between P. and Q. the string remains at zero and during this period, the stimulus is passing from auricle to ventricle through the auriculo ventricular bundle. R. is the tallest of the series and is clean cut and well defined. It corresponds to the initial contraction of the base of the heart and continues to rise till the contraction reaches the apex. Between S. and T. the string again remains at zero. This has been explained as being due to the fact that during this period the whole ventricle is contracting and all parts of the muscle are of equal potential. T. is a broad and somewhat prolonged wave and represents the contraction of that part of the ventricle at the base of the aorta which the experiments of Gotch have suggested is the last part to contract. Figure 10 shows the record in all these leads from a normal individual.



The characters of the electro cardiogram in auricular fibrillation show marked differences which are illustrated by the next figure.



(To be read from right to left)

Scattered throughout the tracing we see the clearly defined R. waves of the ventricular complexes. They occur at irregular intervals and bear no definite relation in height to the length of the preceding pause. The T. waves are also easily recognised but are somewhat obscured by the presence of fine oscillations upon them. The reversal of T. in this case is held to indicate some myocardial changes.

The most striking feature as in the phlebogram is the absence of the P. variation. Replacing P. is found a number of fine oscillations occurring throughout the whole cycle. These are the characteristic and pathognomonic oscillations referred to earlier in the paper. They occur in no other pathological condition and are only produced by the changes of potential occurring in a fibrillating auricle.

The characters of the oscillations vary in different tracings. In some they are very prominent and may be as large as a normal P. variation, in others they are less marked. They deform the T. wave but never affect the R. wave owing to the rapidity of the latter movement.

Also they bear no relationship to the extent of the myocarditis, for patients may have gross and obvious myocardial disease and yet not give these oscillations.

The presence of the oscillations throughout the whole cycle indicates the continuous activity of the auricle.

The following examples more fully illustrate the clinical features of cases of Auricular Fibrillation.

Case I. R.B. aged 38, labourer.

Was admitted to Addenbrooke's Hospital on July 20th 1911 with double Mitral Disease and Double Aortic disease, the result of Rheumatic Fever several years before.

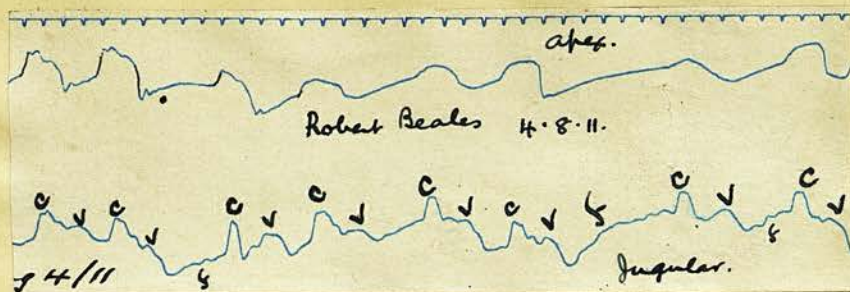
He complained of fluttering of the heart, was very short of breath and unfit for work. He had a troublesome dry cough, was somewhat cyanosed and lay propped up with pillows.

His urine was scanty and free from albumen. The veins of the neck were greatly distended. His pulse was very rapid, very irregular and impossible to count. His heart was slightly enlarged and also very irregular.

At the apex systolic and early diastolic bruits were heard, and over the aortic cartilage was a systolic and diastolic murmur.

The venous pulse was of the ventricular form

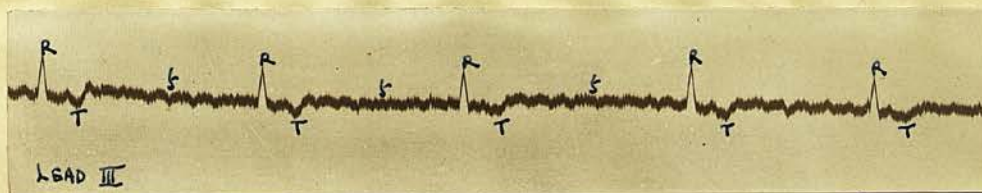
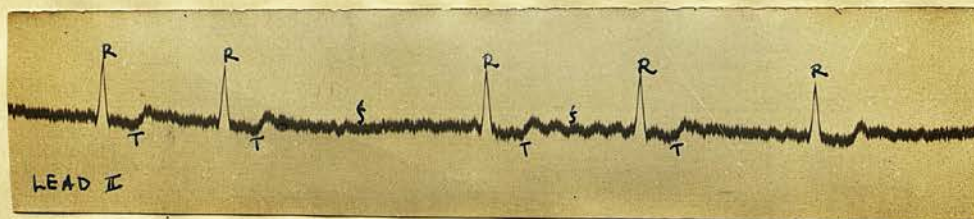
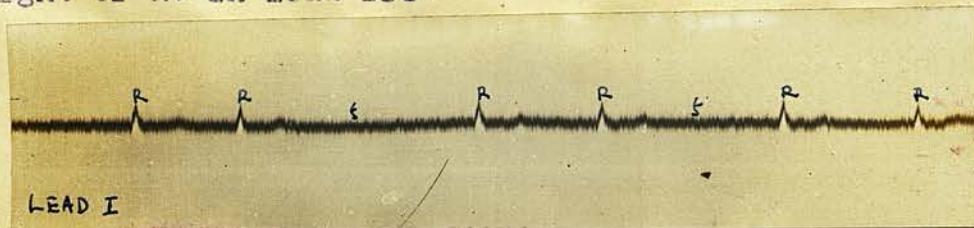
and showed all the typical features.



Note the complete irregularity, absence of the (a) waves and the waves in diastole due to the fibrillating auricle marked (f).

The electro cardiograms show the characters already described in the three leads.

Note the reversal of T. in lead III. which is supposed to indicate some change in the distribution of the conducting apparatus. It often occurs with myocarditis. There is also a slight tendency to Right Ventricular Hypertrophy as indicated by the height of R. in lead III.



Treatment. On admission, the patient was given Tincture of Digitalis 15 minims every 4 hours for three days. Marked improvement took place, the pulse became slower, more regular and stronger. The urine was increased and the patient became more comfortable. The Tincture of Digitalis was then reduced and the effect continued under 10 minims three times a day.

Patient was discharged in comparatively good health on August 15th. but was re-admitted on October 17th. with symptoms of heart failure brought on by hard labouring work.

On this occasion he received 10 minims of the Tincture every 4 hours at first but was later ordered Digitalin Granules (Nativelle) twice a day with similar good results.

He was discharged again on December 23rd. free from symptoms. Since then the patient has had another attack of Heart failure which proved fatal.

Case II. J.F. aged 48, Railway labourer.

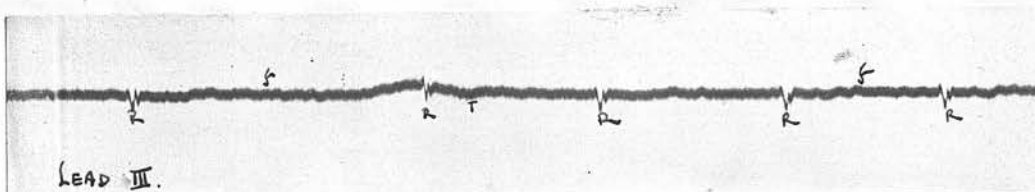
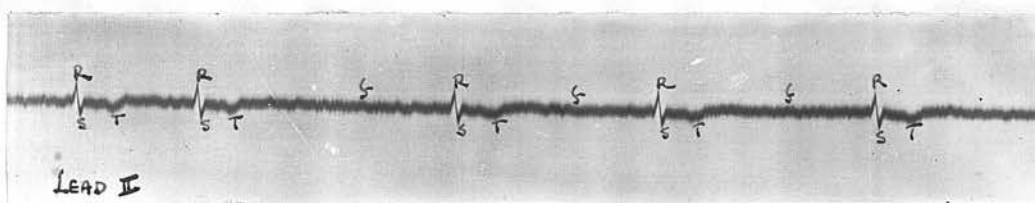
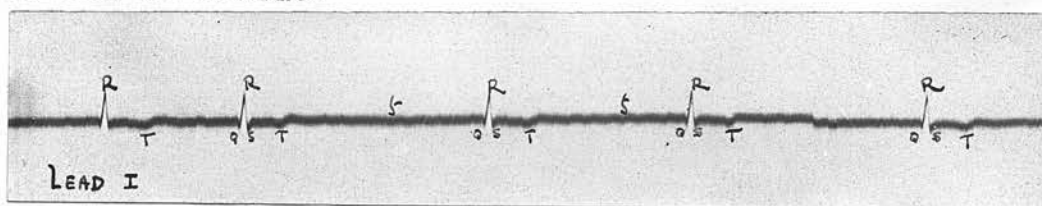
Was admitted to Addenbrooke's Hospital on August 4th 1911 with Mitral Incompetence. He had had three attacks of what he called Influenza but which were probably attacks of Subacute Rheumatism. He was admitted with all the signs of heart failure, namely, shortness of breath, cyanosis, cough, oedema of the legs, enlarged liver and scanty urine containing albumen and casts. There was some emphysema of the lungs and

oedema of the lung bases.

The pulse was rapid and continuously irregular, being difficult to count.

The Heart was enlarged and very irregular in action. There was a harsh systolic bruit at the apex.

The electro cardiograms show the typical characters namely, complete irregularity, absence of P. wave and the typical oscillations. The heart's action is feeble.



Note the reversal of the R. wave in lead III. which signifies a tendency to left ventricular hypertrophy.

Treatment. Patient was put under the influence of Tincture of Digitalis immediately. I began with 15 minims every four hours until the effect was produced and then reduced it gradually until I found that

the patient could be kept free from symptoms by taking 5 minims of the Tincture three times a day.

Marked slowing of the pulse occurred, the urine was increased and the patient was better in every way.

He was discharged on September 23rd. but continued to treat himself by taking Digitalin Granules. Case III. exhibits another feature which is of some importance.

In the early stages of Mitral Stenosis the presence of a presystolic bruit at the apex is usually detected. As the stenosis proceeds, another bruit becomes apparent occurring early in diastole immediately after the second sound. When auricular fibrillation sets in, a change is noticed. The presystolic bruit disappears while the diastolic remains.

The explanation of this is obvious. The presystolic bruit is produced by the systole of the auricle driving the blood through the contracted orifice. During fibrillation normal systole does not occur. The diastolic bruit persists as it is produced by the suction power of the ventricle during the diastole.

A diastolic bruit occurring in Mitral Stenosis usually indicates some considerable degree of contraction.

Case III. J.S. age 42.

Was admitted to Addenbrooke's Hospital on February 27th 1912 with Mitral Stenosis and Incompetence, following Rheumatic Fever 14 years ago.

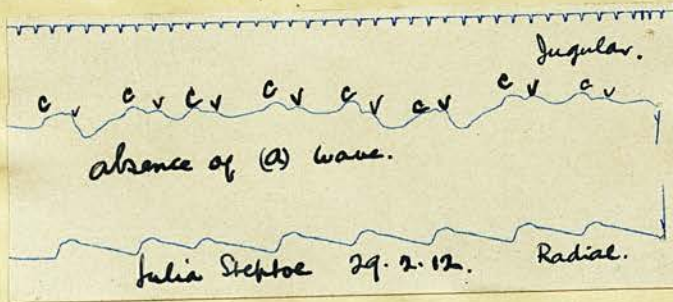
She had been an Outpatient for some years and her notes record in addition to the irregularity the

frequent presence of a presystolic murmur.

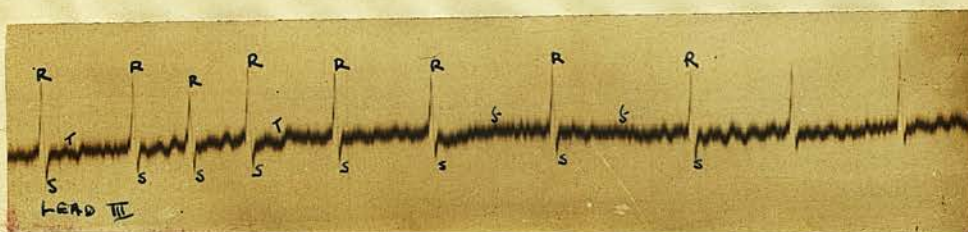
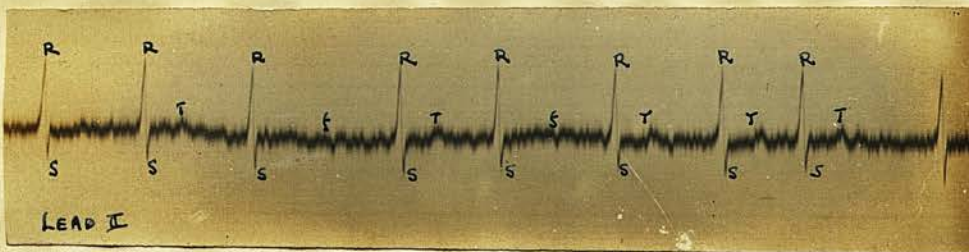
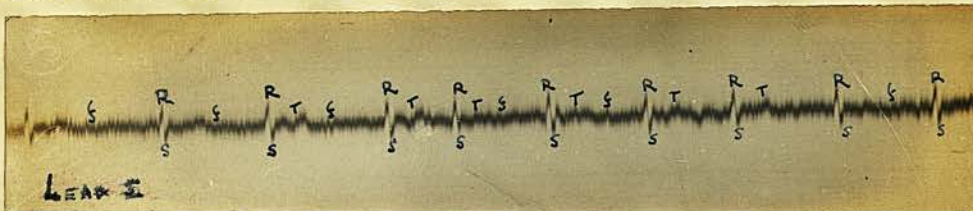
She was admitted with symptoms of heart failure. The pulse rate was 100 and the rhythm was irregular and disorderly.

The Heart was greatly enlarged, the apex beat being in the Anterior Axillary line. At the apex there was heard a systolic and an early diastolic bruit. No presystolic murmur was present. There was also a systolic bruit over the aortic cartilage.

The venous pulse is of the ventricular form.



The electro cardiograms show the usual features of auricular fibrillation.



Treatment. Marked improvement with digitalin granules.

These three cases serve to show the usual features of Auricular Fibrillation.

The chief points are the marked alteration in rate and rhythm, the increased size of the heart, and the reaction to digitalis.

The pulse itself is usually sufficient to diagnose the condition. The irregularity is of very disorderly kind, no two beats being of the same strength.

There is a constant intermingling of strong and weak beats and many beats do not reach the wrist.. Hering gave the name of Pulsus Irregularis Perpetuus to this form of pulse but this term has fallen into disuse.

Dilation of the heart occurs sooner or later due no doubt to exhaustion from the rapid and irregular stimulation of the ventricle and leads to symptoms of heart failure.

Although Auricular Fibrillation usually gives rise to this extreme irregularity of rhythm, it is occasionally the cause of attacks of Paroxysmal Tachycardia, a feature well illustrated by Case IV..

Case IV. A.W. age 18.

Was first admitted to Addenbrooke's Hospital in December 1905 with Mitral Stenosis and Incompetence following Chorea. She was treated with small doses of Tincture of Digitalis and was

discharged in good health in March 1906.

She was again an In-patient during July and August 1907. She gave a history of subacute rheumatism and frequent sore throat.

Her condition then showed signs of heart failure, shortness of breath, cyanosis, cough, and oedema of the legs being noted.

Presystolic and systolic murmurs were evident at the apex and the heart was considerably dilated.

The pulse was regular. The treatment again consisted of small doses of Tincture of Digitalis which gave relief.

Since then she has been a frequent In-patient, symptoms of heart failure coming on as soon as the Digitalis was stopped.

Her last Notes record:- The Patient is a thin pale, delicate looking girl with a slight malar flush. She suffers from considerable orthopnoea and is liable to severe attacks of palpitation and fainting. The pulse appears regular to the finger but sphygmographic tracings detect slight irregularity. The rate varies between 72 and 112 and the pulse is of the Water Hammer type.

There is marked bulging and pulsation over the praecordia. The heart is greatly dilated.

At the apex can be heard a systolic and an early diastolic bruit. At the aortic cartilage well marked systolic and diastolic bruits are apparent.

There is great pulsation in the neck and capillary pulsation can be obtained.

The Jugular veins are prominent and pulsate.

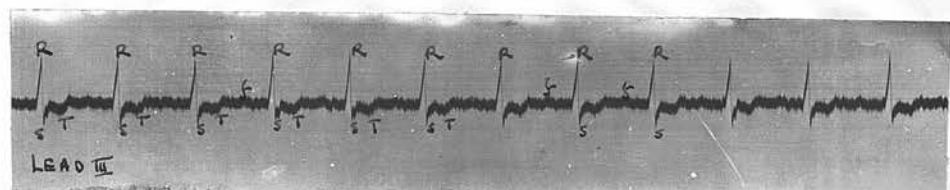
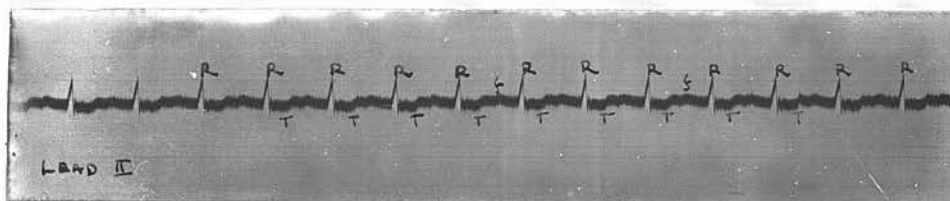
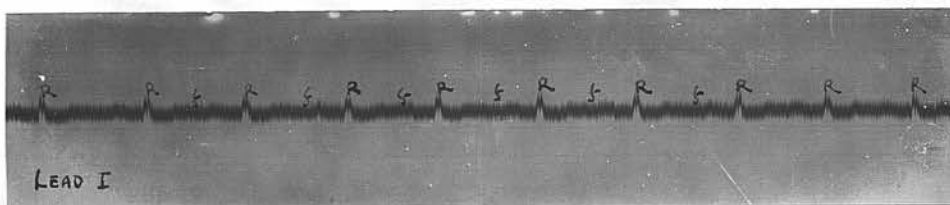
The Patient is liable to attacks of Tachycardia. These attacks come on suddenly, last a minute or two and pass off as quickly as they came on. During the attack the pulse develops a regular rate of about 200 per minute.

During the attack the Patient complains of a thumping sensation in the chest and gasps for breath.

The electro cardiograms show some variation in the strength of the ventricular complexes (lead III.).

There is some Hypertrophy of the Right Ventricle as shown by the increased height of R. in lead III. Reversal of T. indicates some Myocardial change.

The R. wave in lead I. is bifid, probably indicating some myocardial change.



Treatment by Digitalis in this patient acted miraculously.

I found that the Digitalin Granules of Nativelle gave the best results.

The pulse was slowed, the symptoms were relieved and absolute freedom from the distressing attacks of Tachycardia was attained.

After her discharge, she successfully treated herself with the granules, but succumbed eventually to a sharp attack of Lobar Pneumonia.

These cases have this in common, they all react well to Digitalis.

It is in cases of Auricular Fibrillation, especially those occurring after Rheumatism or Chorea in comparatively young persons, that digitalis produces the best results. In those cases occurring in elderly patients with Cardio Sclerosis, the results are more uncertain.

Its action is twofold, partly on the auriculo ventricular bundle and partly on the vagus.

The result is that a certain degree of Heart Block is produced which blocks the passage of many of the irregular stimuli to the ventricle and allows the latter to regulate its pace.

I have followed the lines of treatment laid down by Mackenzie and found them highly satisfactory.

He advises that the administration should in

the first instance be pushed until we get evidence of the physiological action of the drug. In adults it is advisable to begin treatment with 15 minims of the Tincture every 4 hours. If this should produce nausea and sickness, the granules of Digitalin may be used instead. Each granule is equal to about 15 minims of the tincture (Mackenzie) (14)

These large doses are continued until the pulse rate is about 70 per minute. The drug should then be stopped for a few days and as the rate begins to increase again, smaller doses will be found to answer. (15)
Price recommends that the dose be reduced until we find the "optimum" dose. This is one that usually keeps the heart rate about 70 per minute, but the best guide to this is the sensations of the patient.

Patients are readily taught to treat themselves with the drug and keep themselves in a comparatively fit condition.

The next case is one occurring in an elderly woman where digitalis failed to produce relief.

Case V. J.N. age 69.

Was admitted to Addenbrooke's Hospital on December 13th. 1911 suffering from Chronic Bronchitis, Emphysema and Mitral Incompetence. Her previous illnesses include Influenza 20 years ago and slight Rheumatic attacks.

When admitted she had great dyspnoea, cyanosis

frequent cough and had extensive oedema of the legs, back and Ascites.

The urine was scanty and contained albumen.

Her liver was enlarged. The veins of the neck were engorged and prominent.

The pulse was of slow rate and frequently irregular.

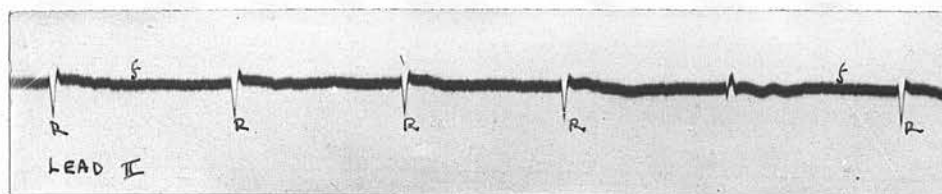
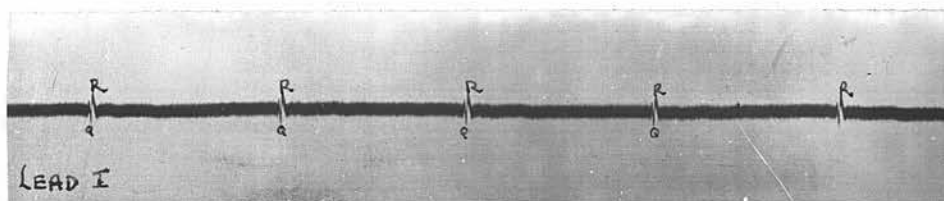
The Heart was dilated with a long systolic murmur at the apex.

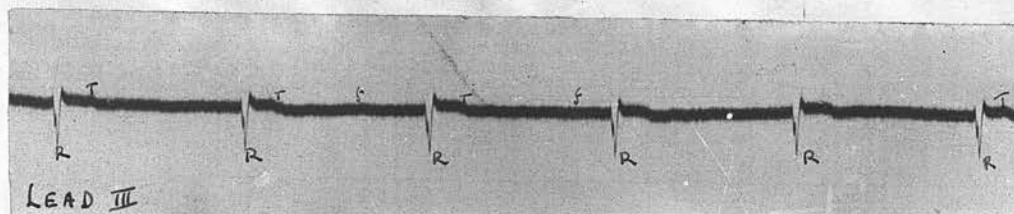
The electro cardiograms show only a slight tendency to auricular fibrillation, the oscillations being most marked in lead III.

In all the records, the beats are very small owing to the feeble action of the ventricle, and there is complete absence of P.

Note the long diastolic pause which probably means a certain degree of Heart Block.

There is a tendency to left ventricular hypertrophy as shewn by the downward direction of R.III. The "splintering" of R. in lead III. is believed by some to indicate a myocardial lesion (Barker) ⁽¹⁶⁾





Treatment. This Patient was treated on the usual lines with expectorants and digitalis but without effect. She pursued a downward course and died about 6 weeks later.

Some cases of Auricular Fibrillation are not always so easy of recognition.

Case VI. is that of an elderly man suffering from Chronic Bronchitis and Emphysema with Arterio Sclerosis and a dilated Heart. There was no valvular disease apparent. During the two months he was an In-patient, the rhythm on the whole was regular. On one or two occasions some arrhythmia was noticed.

Case VI. M.H. age 71, a labourer.

Was admitted to Addenbrooke's Hospital on October 26th. 1911 with Chronic Bronchitis and Emphysema and Chronic Nephritis. He was suffering from extensive oedema of the legs, back, scrotum etc. There was general anasarca of the body and free fluid in the peritoneal cavity.

He gave a history of several slight attacks of Rheumatism.

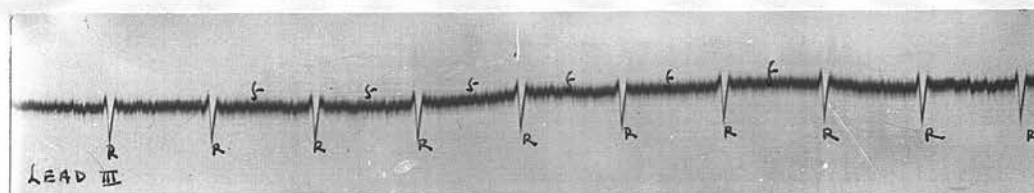
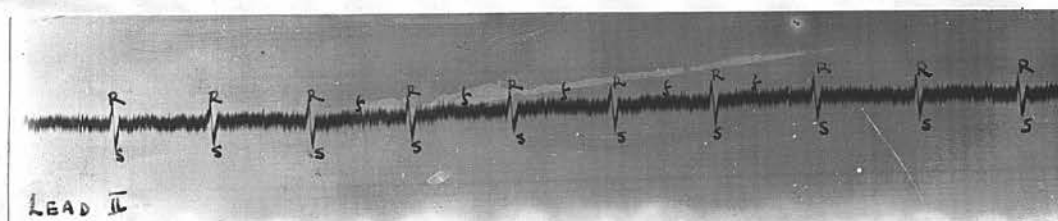
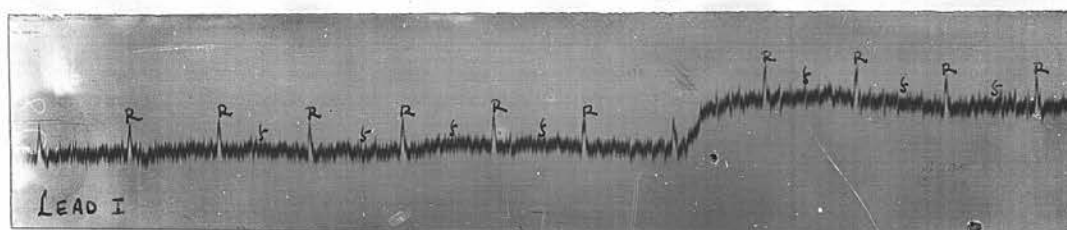
His pulse rate averaged 80 and except on one or two occasions was regular. The coats were thickened. The Heart was slightly enlarged and very feeble.

No bruits were audible.

The veins of the neck were distended. The urine contained albumen and casts.

I show the electro cardiograms from this case. They exhibit the usual features and show typical oscillations but there is no marked irregularity. T. is not well marked.

Reversal of R. in lead III. indicates left Ventricular Hypertrophy.



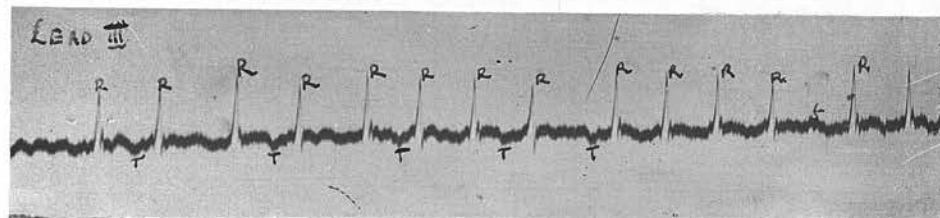
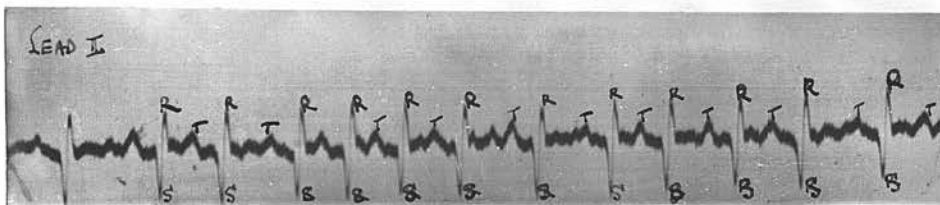
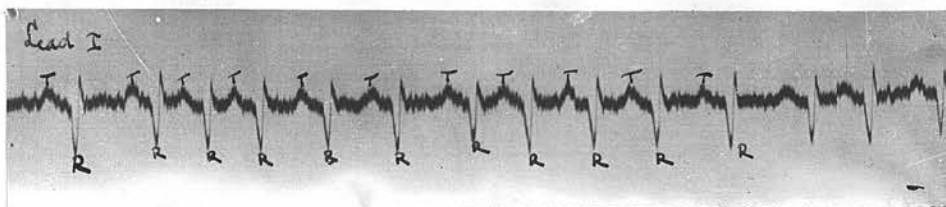
The following tracings are from a private patient who exhibited all the features of Auricular fibrillation.

The Patient was a rheumatic subject with Mitral Disease and suffered from symptoms of heart failure with an extremely irregular pulse.

The venous pulse was of the ventricular type and

the electro cardiograms show the usual characteristics.

This Patient was treated with Tincture of Digitalis in 15 minim doses twice or three times daily, but later on he was given Nativelle's Granules which were more pleasant to him.



(To be read from right to left)

In forming a prognosis in Auricular Fibrillation, it must be remembered that the degenerative conditions producing it are progressive and that the best we can do is to relieve symptoms.

The prognosis will vary in each case according to the manner in which the heart reacts to the altered rhythm. Some cases show auricular fibrillation without troublesome symptoms, and have been known to go on for years.

If the heart dilates greatly and the rate is rapid, then the prognosis is grave. If, however, there is not much dilation and the rate not above 90, or if over 90 but can be reduced by treatment, then the prognosis is favourable.

The Patient may go on for an indefinite number of years, sometimes in good health, often chequered by more or less severe attacks of temporary heart failure.

The length of time between the Rheumatic infection and the onset of fibrillation with the intervening history of the patient are important points in forming an opinion. The manner in which the patients are affected by work and exertion gives an important idea of the reserve power of the heart.

Senile cases are less favourable than cases occurring in younger persons.

Lastly, the reaction to treatment by Digitalis gives valuable information. Patients by keeping themselves under the influence of the drug can maintain a fair standard of health and can even undertake some light work.

I have attempted to show that the prognosis cannot be definitely stated on one point alone. It depends on many factors, including the duration of the condition, the extent of the changes, the age of the Patient, the reserve power of the heart and the reaction to treatment.

I have not attempted to give a complete account of the features of Auricular Fibrillation in this paper. I have endeavoured to demonstrate the commoner features of the disease and especially the steps that lead to its discovery. I have especially mentioned the characters of the string Galvanometer

and the tracings obtained by it as on account of the accuracy of the electro cardiograms, this method of analysing the Cardiac Mechanism is tending rapidly to supersede all other graphic methods.

37.



S U M M A R Y.

1. A description of the String Galvanometer.
Invented by Einthoven in 1906, it records the changes of electric potential which occur in the heart during its contraction and has been the means of proving the frequent occurrence of Auricular Fibrillation in the human heart.

2. Auricular Fibrillation refers to that condition of the auricle where the muscle fibres contract inco-ordinately and normal systole is never accomplished.

It occurs particularly in two groups of cases,

(a) Associated with old standing
Rheumatic disease.

(b) The senile cases with Cardio Sclerosis.

It occurs most commonly with Mitral Stenosis and is characterised in this disease with the disappearance of a presystolic bruit.

The clinical features are those described previously by Mackenzie under the term "Nodal Rhythm".

Of all cases examined after death, the only feature common to all was a fibrosis of the muscle.

3. The main clinical features are,

(a) A continuous irregularity of the heart.

(b) Absence of all signs of normal auricular activity.

In some cases it gives rise to attacks of Paroxysmal Tachycardia.

Sphygmographic Records.

2.

1. Rate usually increased.
2. Complete irregularity with beats of all shapes and sizes.
3. No fixed relation between the strength of a beat and the preceding pause.

Polygraph Records.

1. Absence of the (a) wave due to auricular systole - the ventricular type.
2. Presence during diastole when the heart is slow of rapid undulations of venous pressure.

Electrocardiograph Records.

1. Absence of auricular systolic variation P.
2. Ventricular peaks R. and T. occur but T. is often deformed by the presence of fine oscillations.
3. Occurrence of continuous oscillations generated in the auricle and occurring throughout the tracing.
4. Absence of fixed relationship between the height of peak R. and the length of the preceding heart pause.

Similar records are obtained from animals in which fibrillation of the auricle has been produced experimentally,

4. The prognosis in Auricular Fibrillation depends on a study of several factors in each case.
5. The reaction of these cases to digitalis is sometimes very remarkable. In certain forms, especially those of Rheumatic origin, one can regulate the rate at will by increasing or diminishing the amount of digitalis.

REFERENCES.

1. Mackenzie (James) "Auricular Fibrillation",
British Medical Journal, No: 2650. Oct.
1911, pp. 869-874.
2. Hirschfelder (Arthur D.) Recent Studies of
Cardio Vascular Diseases. St. Louis 1911
pp. 9-25.
3. Howell (Wm. H) Textbook of Physiology.
4. James (Walter B.) and Williams (Hoartio B.)
"The Electro cardiogram in Clinical
Medicine". Amer. Jour. Med. Science
Sept. 1911.
5. Lewis (Thomas) "The Mechanism of the Heart
Beat" 1911.
6. Gotch (F) "Capillary Electrometer records of the
Electrical changes during the natural
beat of the frog's heart" Proceed.
Roy. Soc. 1907 Vol. 79 pp. 323-328
7. Gotch (F) "The succession of Events in the con-
tracting ventricle as shewn by Electro-
meter Records - Tortoise and Rabbit"
Heart 1909-1910 Vol. I. pp. 235-261.
8. Cushney (A.R.) "Irregularity of the Heart and
Auric. Fibrill". Amer. Journ. of Med.
Sciences. June 1911 pp. 826-837.
9. Cushney (A.R.) and Edmunds (C.W.) "Paroxysmal
irregularity of the Heart and "Auricular
Fibrillation". Amer. Journ of Med.
Science 1907 Vol. cxxxiii. pp. 66-77.
10. Lewis (Thomas) "Origin of the Electric oscilla-
tions and the direction of contraction
of the ventricle in instances of complete
irregularity of the Heart". Quart.
Journ. Med. Vol. v. No: 17 Oct. 1911.
11. Lewis (Thomas) "Auricular Fibrillation and its
relationship to clinical irregularity of
the Heart". Heart vol. 1. No: 4 p. 306.
12. Lewis (Thomas) "Evidences of Auricular Fibrill-
ation treated historically" Brit. Med.
Journ. No: 2663 Jan..1912 pp. 57-100.
13. Lea (C.E.) "Some points in relation to the
Etiology of Auric. Fibrillation". Quart.
Journ. Med. Vol. 1V. No: 16 pp. 423-433.

14. Mackenzie (James) "Auricular Fibrill". Brit.
Med. Journ. No: 2651. Oct. 1911 pp. 969-974.
15. Price (Frederick W.) "Recent advances in Cardiac
Therapeutics". Brit. Med. Journal No:
2649 Oct. 1911 p. 810.
16. Barker (Lewellys) "On some of the clinical
methods of investigating Cardio-vascular
conditions". John Hopkins Hosp. Bulletin
Oct. 1909 page 307.