CARDIAC ARRHYTHMIA.

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The study of the pulse dates from earliest times. While the story of the circulation and composition of the circulating fluid were still the basis of fabulous conjecture, there was recognised by the early physicians the significance of the pulse.

Amid all the superstitious teaching of his day, we find the ancient Herophilus of Alexandria, about the year 300 B.C. making a study of the pulse in its relation to disease. And in our modern terminology we still trace his early work - the term "pulsus dicrotus" first appears in his classification of pulse variation.

At a much later date followed the Galenic period. Galen of Pergamon contributed largely to early medical literature, and has left sixteen treatises on the pulse. He believed that for each malady there were certain characteristics of the pulse peculiar to that disease; and his teaching was followed, more or less, for some 1400 years.

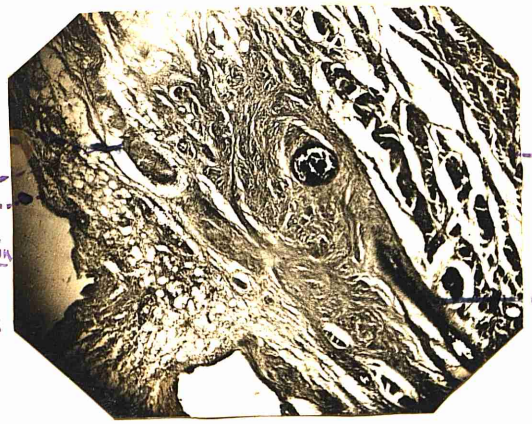
Towards the end of the 12th. century Aegidiensis Corboliensis wrote a series of verses "De Pulsibus" recognising and describing fifteen varieties of the pulse, including the irregular and unequal pulse.

The discovery of the circulation was made by William Harvey in 1616, and the first description of it published by him in 1628 in "De Motu Cordis".

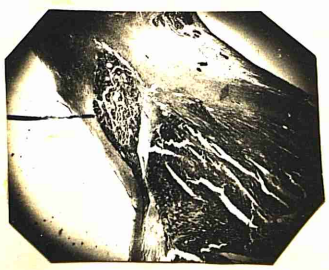
But the investigation of cardiac irregularity was only possible with the introduction of mechanical methods. Even in the time of Harvey, pulse-timing was not in general use, though several instruments had been used in his day. The ingenious watch described by Floyer in 1707 was barely known until his method was revived by Pierre-Charles Alexandre Louis. This was early in the 19th century but it was some years later that the first accurate instrument for recording the pulse was used by Marey. The venous pulse was also recorded by Marey and Bamberger in 1863. Several observers contributed to the investigation of the pulse, but Mackenzie's work stands foremost in the interpretation of cardiac irregularities.

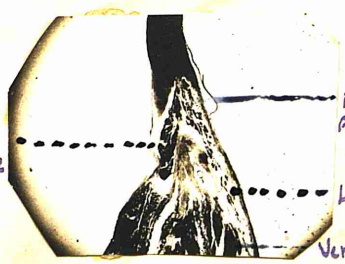
In 1902 "Study of the Pulse" was published. With the application of electro-physiology to cardiac contraction by A.D.Waller in 1889, a great advance was made. The capillary electrometer used by him was later replaced by the Einthoven String Galvanometer - a higher power instrument.

The interpretation of cardiac irregularity depends primarily on the knowledge of cardiac anatomy and physiology. The normal contraction begins at the sino-auricular node - a neuro-muscular structure and remnant of the primitive cardiac tube, situated at the junction of the superior vena cava and right auricular appendix.

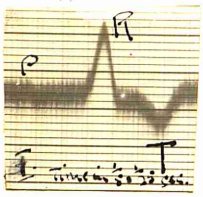


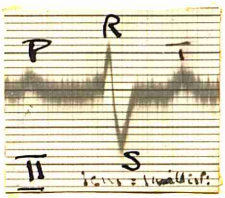
The nodal tissue appears as fine muscle fibres surrounding the central artery. To the right in the section is auricular muscle; in the lowest part is the commencement of the auricular appendix. This node is the point of primary negativity as tested electrically. The excitation wave spreads radially throughout the auricular musculature (R.and L. auricles) and along the auricular septum to the atrioventricular node. That the muscle of the auricle proper shows relative negativity before the A-V. node, has been proved. The contrary opinion held by Meek and Eyster that there is more rapid conduction of the excitation wave from the S-A node to the A-V node than to the other parts of the auricle, thus rendering the A-V node relatively negative before the muscle of the septum, has inconclusive evidence. From the A-V node the wave spreads along the A-V bundle, its right and left divisions, along the arborisation and network of their branches (the Purkinje system) and so to the ventricular muscle. The spread of the excitation wave in the ventricular musculature, therefore, takes place from within outwards. The actual conducting tissue resembles ordinary heart muscle but the fibres are finer and contain large amounts of glycogen.

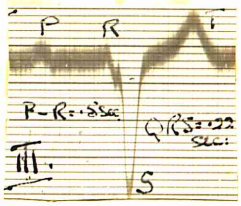




Anything interfering with the normal spread of the excitation wave will obviously give rise to abnormal cardiac contraction, and in the electrocardiogram will be seen an abnormal auricular or ventricular complex, or both combined. The degree of aberration will vary with the lesion present. The following photographs show a marked degree of interference with the normal spread in the ventricle - there being defective conduction in the A-V bundle and its right division.







The P-R interval in this case equals 0.5 of a second; the QRS complex exceeds the normal 0.1sec. by 0.12 sec.

There are many varieties of pulse irregularity. The Sinus Irregularity, frequent in the young and occasional in adults is produced by vagal stimulation either reflexly as by respiration and swallowing, or directly as by digitalis. There is present a degree of vagal instability. The irregularity is due to a variation in the length of the diastolic periods, e.g. fig.1

Fig. 1.

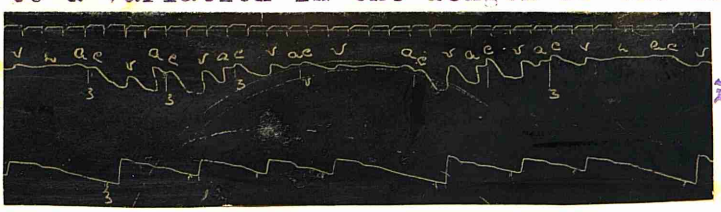
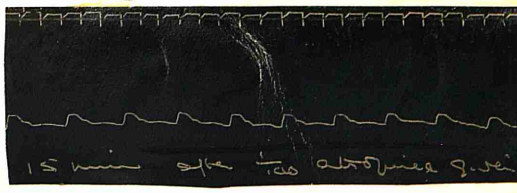


Fig. 2.



Anything which inhibits the vagi, inducing sympathetic predominance and consequently acceleration of rate, will abolish this irregularity, e.g. fig.2. Exercise has the same effect.

It is of no pathological significance . In fact its reappearance in the convalescent stage of acute febrile disease probably indicates that the heart is no longer acting under the abnormal stimulation of disease but is responding to physiological excitation.

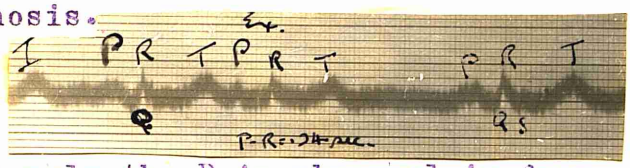
Extrasystolia is the most common form of irregularity. An extrasystole is defined as a premature contraction of abnormal origin. It is of lower pressure than the normal contraction. The force of the beat, as a rule, is diminished and so, in cases of valvular disease with murmurs and in which extrasystoles occur, the murmurs may not accompany the premature beat. The abnormal contraction fails to drive blood through the affected orifice. Whether or not this will occur will, of course, depend on the site

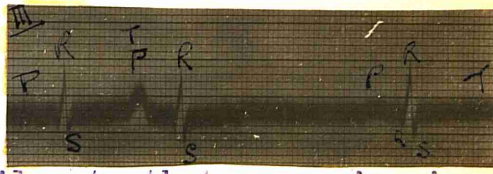
of origin of the abnormal contraction and on which orifice is affected. The nature of the valve lesion is also important in determining this result. This feature is very constantly present in a case of mitral incompetence and adhesive pericarditis with ventricular extrasystoles, at present under observation. A harsh systolic murmur is present with the ordinary contractions but the extrasystole is audible as two short sharp sounds.

Extrasystoles may be auricular, nodal, or ventricular in origin.

Auricular Extrasystoles are the least common variety, they are thought by many observers to arise high up in the A-V node. Some may do so but it is more than probable that many arise in other portions of the auricular muscle. When, in electrocardiograms the P wave is of normal form and the P-R interval approximately normal it is probable that the site of primary negativity is in close proximity to the S-A node. e.g. Fig.4 from a case of mitral stenosis.

Fig.4.

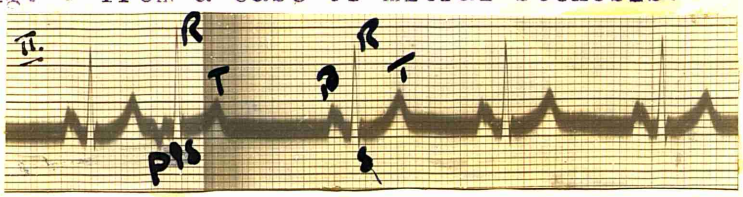




As a rule the P is abnormal in form, similar to that occurring in nodal extrasystoles. The ventricular complex is normal in form except in cases with concomitant conduction defect. Aberration of the ventricular complex is more likely to occur when the contraction appears very early in diastole. The length of the returning cycle is variable. Auricular extrasystoles are almost invariably indicative of some lesion of the auricular muscle and are frequently the forerunner of auricular flutter or fibrillation. In cases of paroxysmal flutter or fibrillation they frequently interrupt the periods of normal rhythm, as will be illustrated later.

Nodal Extrasystoles , likewise, possibly never occur apart from myocardial involvement. The auricles and ventricles contract prematurely and sometimes together; the auricular contraction may follow the ventricular or it may precede it at a reduced interval, e.g. Fig. 5 from a case of mitral stenosis.

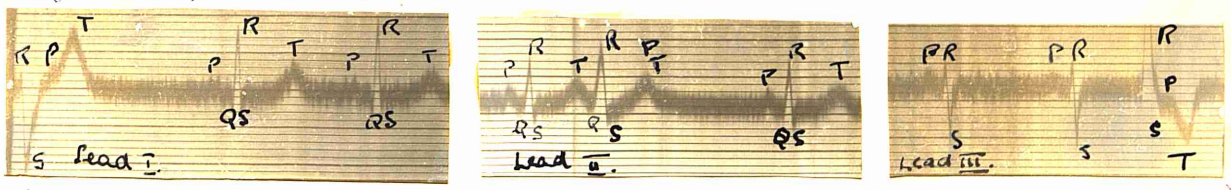
Fig. 5.



The P wave is inverted indicating an abnormal course of the excitation wave through the auricular muscle. The P-R interval is reduced from 0.12 sec. to 0.08 sec.

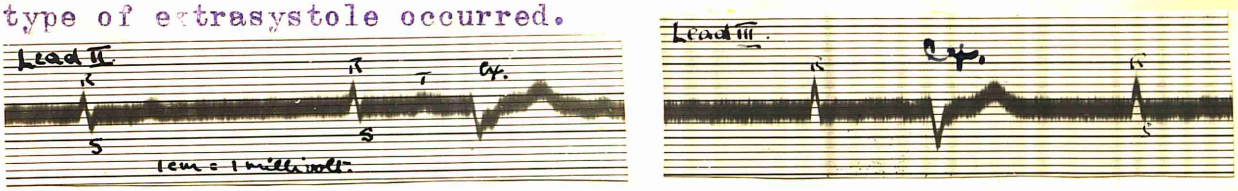
Ventricular Extrasystoles are the most common variety. They are recognised clinically by the compensatory pause - the refractory phase of the ventricle preventing response to the next auricular contraction. It must be remembered, however, that interpolated extrasystoles may occur. In electrocardiograms the P wave follows or is buried in the ventricular complex which is abnormal in form and varies according to the site of origin. Fig.6 illustrates right basal extrasystoles - taken from a case of high blood pressure (systolic, 280mm Hg; diastolic, 150mm Hg.) with no valve lesion.

Fig.6.



(Incidentally this tracing shows left ventricular preponderance.) The abnormal QRST complex resembles that occurring in defective conduction in the left division of the A-V bundle in which the dextro cardiogram, somewhat modified perhaps, results.
The left apical extrasystoles are the reverse of the above, e.g. Fig 7, from a case of auricular fibrillation in which this type of extrasystole occurred.

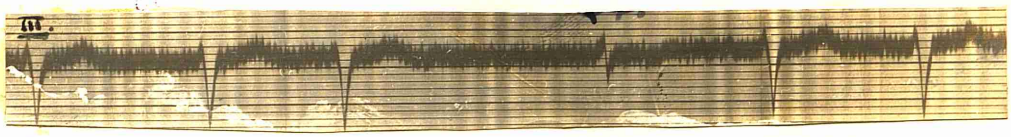
Fig.7.



The first two beats in lead II are supraventricular in type. It should be noted that the tracing shows slurring of the QRS complex indicative probably of severe myocardial involvement. The patient is now - three months later - in extremis.

Ventricular extrasystoles are to be distinguished from escaped beats of the ventricle which are not premature but in a given patient may terminate all diastoles of more than a given length, e.g. Fig.8. Case I.

Fig.8.

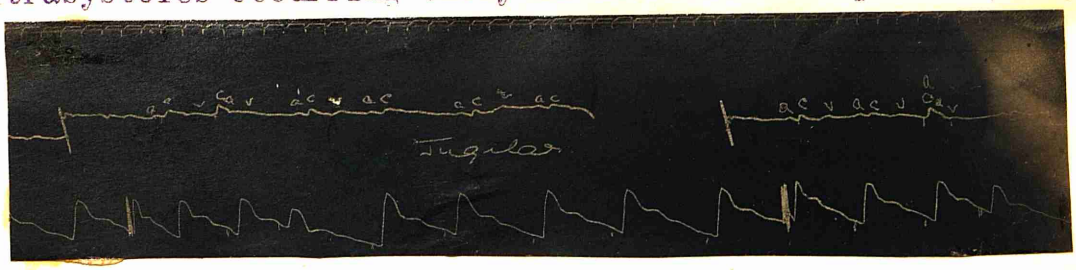


This condition is liable to result from vagal stimulation as in the above instance where it occurs from digitalis therapy. The cardiac condition was one of mitral incompetence with auricular fibrillation.

Case II. In one case of extrasystolia there was some variation in the time of their occurrence -which is rare. There was a sinus

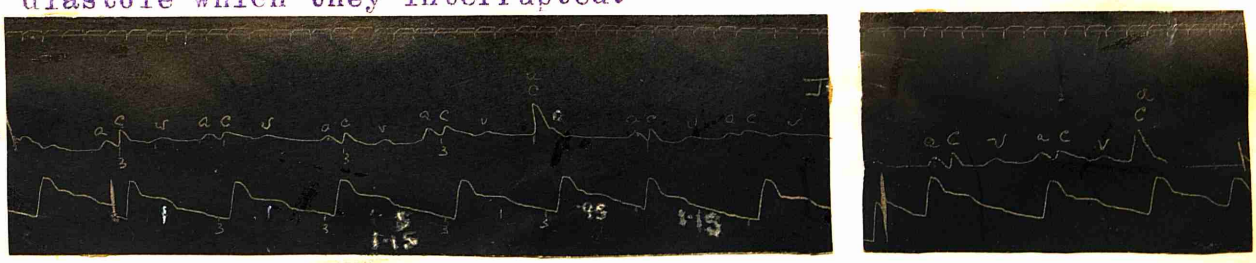
arrhythmia apparent in all the tracings. In some tracings ventricular extrasystoles occurring early in diastole were present, e.g. Fig.9.

Fig. 9.



In other tracings e.g. Fig.10 the abnormal contraction occurred late in diastole and the prematurity was barely recognisable on digital examination. They bore some resemblance to ventricular escape though their appearance was not constant in the length of diastole which they interrupted.

Fig. 10.



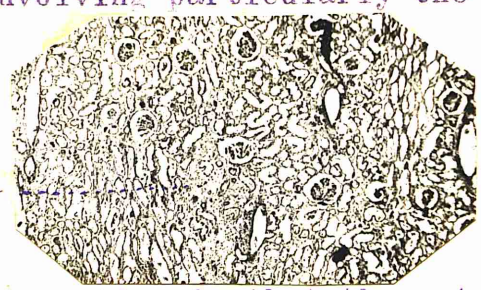
The premature contraction followed slowing of the sinus rhythm. It might be inferred that this depression of the S-A node was produced by vagal stimulation and the slowing allowed some irritable focus to initiate contraction. The former factor is usually the more important but in this case the irritability of the ventricular wall was, I think, the more essential feature. This was very suggestive during life by the actual occurrence of ventricular extrasystoles of the classical type as in Fig.9 and it was, I consider, confirmed at autopsy.

The patient, aet.36 yrs. demonstrated no sign of heart disease during life other than the occasional extrasystole. There were no symptoms referable to the heart and the physical signs were:- maximum impulse in fifth space 3½ inches from mid-sternum; area of cardiac dulness ~~was~~ 3½ by 3 inches; first sound at apex of poor tone; second sound closed at base; blood pressure normal. Urine examination nil. The patient had chronic phthisis affecting both upper lobes. He died within three days of acute pneumonic exacerbation of the disease. On post mortem examination of the heart, I found:- weight of heart 1 lb. The epicardium showed a thickened patch 1½ in. above the extreme apex. From this patch extended many fine fibrous tags which were probably at one time adherent to the pericardium. The adjacent myocardium was not

invaded. There was no dilatation of the heart. The valves were normal. Patchy atheroma was present in the first part of the aorta; it was most marked surrounding the orifice of the left coronary artery and behind the non-coronary cusp of the aortic valve. The left coronary artery showed several small atheromatous patches in its main portion. The patches were dull yellowish in colour and irregular in shape, about a sixth of an inch in their widest portion. The lesion affected the intima only. The right coronary artery was normal.

Microscopic examination:- serial sections were cut of the S-A node and this was found to be perfectly healthy. The innervation of the node was normal. A block was removed from the coronary sinus on the right including the central fibrous body, the pars membranaceae septi. This includes the A-V node, bundle and divisions. Sections were cut but no lesion was found. No change was found in the ventricular muscle.

The kidneys were firm and pale; the capsule stripped off readily but some increase of fibrous tissue was found on microscopic examination, involving particularly the cortex. viz:-

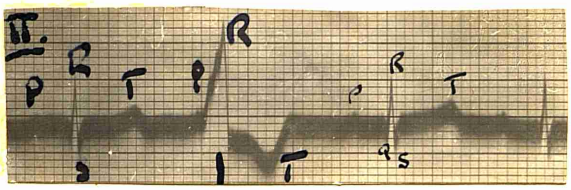


The case is interesting in that the extrasystoles were apparently the only indication of the early arterial disease and coronary involvement. That the absence of increased blood pressure could not be accounted for by the presence of tuberculous toxemia is, I think, clear to most. That the presence of high blood pressure is not excluded by the presence of active tuberculous disease is commom knowledge and in this case, until the acute exacerbation supervened, the toxemia was minimal as judged by pulse and temperature.

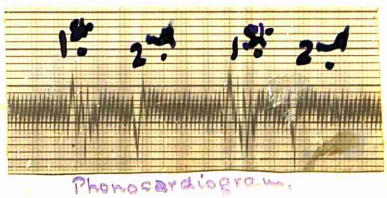
The significance of ventricular extrasystoles is still in dispute. That they are most common in hearts with definite organic disease is without doubt. They are most frequent in patients over 40 years of age when there is usually some change, even if early, in the arteries. There is a class of case frequently seen in which extrasystoles occur - best illustrated by a case which was under my observation some three months ago:- M.S. aet. 53 yrs.,

complaining of shortness of breath on exertion and sense of fatigue of three years' duration; also of pain in the region of the heart and left side of the neck of four months' duration. She had been conscious of occasional irregularity for some eight years. On examination, the pain was found localised to the praecordium and left side of the neck and tenderness of the skin was present in these areas. The sternomastoid and trapezius muscles were more sensitive to pinching than on the right side. Maximum impulse was 4 in. from mid-sternum. The area of cardiac dulness was slightly plus:- 5 in. by 5½. The first sound at the apex was not pure but no bruit was audible and none detected by the phonocardiogram. The second sound was closed at the base. There were occasional extrasystoles ventricular in origin.

Fig. 12. a.



b.



Phonocardiogram.

The systolic blood pressure was 150mm Hg., the diastolic 90mm Hg., i.e. a pulse pressure of 60mm Hg. was present. Urine showed a trace of albumen; Sp.Gr. 1016. The outstanding features in the heart examination were arterial hypertension, pain, and extrasystolia, the latter two being suggestive of muscular involvement as a result of arterial disease, with possibly some hypersensitiveness of the nervous system. The presence of albumen in the urine rendered the prognosis still less favourable. This case illustrates, probably, the next stage of the case last described.

That the arterial change is the full explanation of the condition is open to serious criticism, e.g. E.W. aet. 56 yrs., suffering from weakness and shortness of breath of one year's duration died in hospital 18. 2. 23. from progressive cardiac and renal failure. Systolic blood pressure was 235 - 250mm Hg., diastolic 150mm Hg.; pulse pressure varied from 85 - 100mm Hg. The arteries were thickened. There was no pain and no tenderness present - nothing to suggest hypersensitiveness of the nervous system. The pulse was perfectly regular to the end. At autopsy I found the heart enlarged and hypertrophied; slight atheroma involved the aortic cusps but the valve was competent. Atheromatous change was more marked in the first part of the aorta and involved the orifices of the right and left coronary arteries and their main portions. There was a slight degree of puckering. The kidneys showed chronic inflammatory change. The case was

striking in that the post mortem findings were almost identical with, though more advanced than, those in case II. Yet no irregularity was ever present.
That arterial disease predisposes to extrasystoles is without doubt but that there must be some exciting cause seems only reasonable. The exciting cause possibly varies, but would appear to be frequently neural in origin.

In some cases extrasystoles are induced by exercise; in other cases increasing the heart rate by exercise or stimulation will cause existing extrasystoles to disappear.

They may result and commonly do from toxic causes. Nicotine can induce extrasystoles but the action is complex. Nicotine is a cardiac stimulant having a direct action on the cells of the sympathetic ganglia belonging to the cardiac and vasomotor fibres. It also exerts a pressor effect by stimulation of the peripheral sympathetic ganglia and a combined pressor and cardiac stimulant effect through stimulation of adrenalin secretion. It is also common belief that nicotine exerts a direct toxic effect on the heart muscle. Thus the action of nicotine is due to sympathetic stimulation of an irritable muscle. Levy has shown that if the irritability of the muscle be further increased the irregularity becomes more marked and more complex. Tea may also induce extrasystoles; it too, is a cardiac stimulant and produces a pressor effect combined, possibly, with a direct irritant effect on the heart muscle. Several other drugs e.g. adrenalin - also a cardiac stimulant - and digitalis may produce them. Ventricular extrasystoles are commonly associated with chloroform anaesthesia e.g. J.W. aet. 6 yrs. operated on for tuberculous knee. Chloroform given. The pulse was regular during deep anaesthesia but half an hour after operation, when patient was not quite round from the anaesthetic, a bigeminal pulse alternating with a trigeminal pulse was noted. No tracing was possible as the child was too ill. The ventricular-extrasystolic nature of the irregularity was quite obvious from the auscultatory character of the beats. The irregularity passed off in 3-4hours. Much work has been done by Winterberg, Rothberger and Levy on the effect of chloroform on the heart. It has been found that the irregularities occur during secondary light anaesthesia. In the bigeminal pulse the premature contractions may arise in two different centres as recognised by electrocardiogram ; and this simple irregularity may give place to one more complex in which several

centres are active. The abnormal contractions may follow one another regularly and a rapid multiple tachycardia results. Under cardiac stimulation this tachycardia may pass into ventricular fibrillation.

Gastro-intestinal toxins appear to be capable of inducing extrasystoles. The action of these toxins is obscure; some would appear to be pressor in action. They probably have a direct effect on the muscle but too little is known of their nature to dogmatise about their mode of action.

Extrasystoles sometimes appear during acute febrile diseases and are probably an expression of the toxemia, but whether the toxins are acting on muscle alone, or on nerve and muscle, is in most cases, impossible to tell. When the irregularity persists it is supposed that some damage to the heart muscle has been done. The lesion may be quiescent.

Extrasystoles frequently arise in neurotic subjects but whether or not there is an underlying toxicity in these cases has not been investigated. That they occur under emotion, fear, anxiety, in certain individuals only, seems to support the view that nerve stimulation alone is not responsible. From experiment it seems clear that increased irritability of the muscle is a necessary factor before section of the vagi or sympathetic stimulation will produce them. In this connection it must be remembered that extrasystoles have occurred in the eviscerated and perfused heart.

The extrasystole is usually an isolated occurrence but it may occur rhythmically e.g. in coupled rhythm. The bigeminal pulse may result from sinus arrhythmia but this is rare. The second beat of the pair is usually extrasystolic, most frequently ventricular-extrasystolic, and the irregularity is most frequently associated with cardiac weakness where the heart rate has been greatly slowed by digitalis e.g. Fig. 13.

Fig. 13.

In this case the extrasystole has failed to reach the radial pulse; it is visible in the cervical pulse and was audible at the apex. Auricular fibrillation being present, the lengths of diastole vary.

The regularly recurring extrasystole is also very commonly associated with mitral incompetence or stenosis with a regular rhythm. The extrasystole may be auricular or nodal in origin e.g. Fig.14. from a case of mitral stenosis.

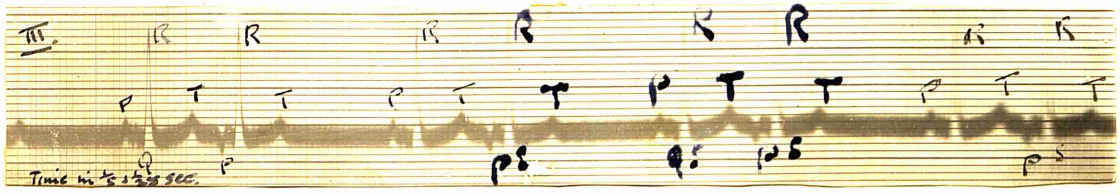
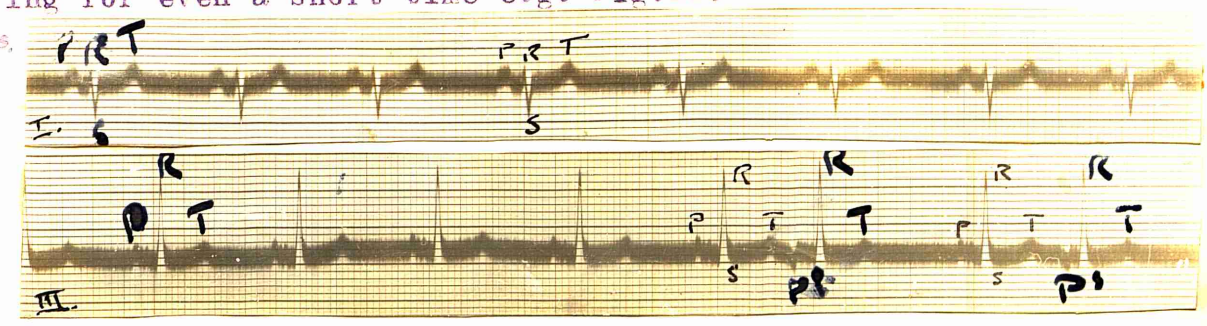


Fig. 14.

The tracing shows slight aberration of the QRS complex.
It has previously been pointed out that this form of irregularity is very liable to occur during chloroform anaesthesia, particularly when cardiac stimulation is present.
The bigeminal pulse has been produced by Levy, on the heart of the cat, by compressing the aorta and so inducing a high blood pressure or a sudden rise of pressure. The bigeminy in this experiment never proceeds to a more complex irregularity even under chloroform. Digitalis, by prolonging diastole, may induce over distension of the feeble heart and increased intraventricular pressure. This mechanical effect is not the principal feature of digitalis coupling for the digitalis bigeminy is not of the simple type above described and if the drug be continued rapid multiple tachycardia and later, ventricular fibrillation result. Digitalis has a direct irritant effect on the cardiac muscle.

The relation of over-distension to this form of arrhythmia is interesting. If the myocardium were healthy, increased distension during diastole would produce increased lengthening of the muscle fibres in direct proportion to which is the force of contraction. But when the muscle is unhealthy, over-distension occurs from prolongation of diastole; the contractility is diminished and systole is unable to empty the ventricle. During the following diastole early filling will occur with excitation of an irritable focus in the heart wall. The extrasystole can not be credited, in most cases, with relieving the distension as the force of the beat is small and in some cases may be insufficient to open the aortic valves. Thus the actual exciting factor is not dispersed but is, if anything, greater towards the end of diastole. It is conceivable however, that the refractory phase of the heart following the premature contraction prevents response to further stimulation until the next physiological impulse arrives. And so the process repeats itself.
It is found that the pulse in such cases may be perfectly regular

after exercise but coupling appears when the patient has been resting for even a short time e.g. Fig.15.



Coupling becomes apparent towards the end of lead III and continues while the patient is at rest. Exertion, by diminishing the inflow to the heart, abolishes the irregularity.

Extrasystoles may occur in series.

Paroxysmal tachycardia is now generally believed to be of essentially the same nature as the single premature beat. The paroxysm may comprise six or more beats; it may last for several minutes, days or weeks. During the paroxysm the heart is working at a disadvantage and if the attack be prolonged, symptoms of failing circulation arise.

The abnormal contractions may be auricular, nodal or ventricular in origin. During periods of normal rhythm single premature beats frequently appear and these are of the same origin as the beats of the paroxysm as demonstrated electrocardiographically. The first beat of the paroxysm is premature and there is a post-paroxysmal pause identical with the post-extrasystolic pause for that particular type of extrasystole. In experiment it has been found that if stimuli which produce extrasystoles be increased in intensity the paroxysm will result; and similarly, factors which suppress the one form of irregularity will have a similar effect on the other. The abnormal rhythm is apparently not under the control of the nervous system. Lewis however, has shewn that vagal stimulation will not affect the rate but may abolish the paroxysm. Paroxysmal tachycardia, like extrasystoles, may occur in apparently normal hearts but this is much rarer than in the case of the extrasystole. There is, as a rule, some indication of a pathological condition being present.

Auricular Fibrillation.

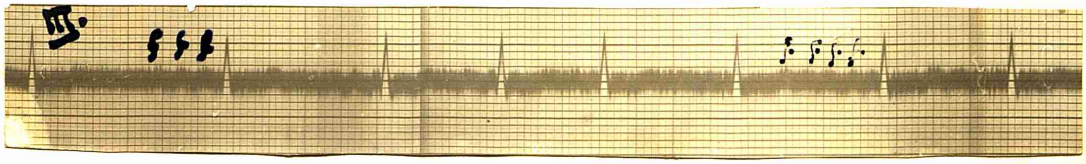
Auricular fibrillation is an exceedingly common condition. It is characterised clinically by a total irregularity of the arterial

pulse which becomes even more pronounced when the pulse rate quickens. For many years it was known as the "mitral pulse" or "delirium cordis". In 1887 MacWilliam produced fibrillar contraction of the dog's auricle by faradization of the auricular wall and in 1889 Cushny pointed out the resemblance of the so-called "mitral pulse" with that obtaining in the above experiment. The association of the two conditions was further suggested by Cushny and Edmunds in 1906. In 1902 Mackenzie pointed out the association of a totally irregular pulse with the ventricular form of venous pulse. The absence of signs of co-ordinate auricular activity led him to conclude that the auricle was paralysed. He later forsook this idea on finding post-mortem that in a percentage of cases the auricle was hypertrophied, and on finding from clinical experience that the condition might be paroxysmal. He then suggested that the auricles and ventricles were contracting simultaneously - a nodal rhythm being present.

Rothberger and Winterberg in 1909 proved that the condition was really due to auricular fibrillation, as suggested by Cushny and Edmunds, and similar evidence was forthcoming in the following year by Lewis and others.

The auricle no longer contracts as a whole but each fibre enters into independent contraction. In electrocardiograms the P wave is absent and irregular oscillations - auricular in origin - occur throughout the whole cardiac cycle e.g. Fig. 16.

Fig. 16.



The QRS complex escapes distortion because of the rapidity of the movement. The oscillations are best seen in leads II and III.

The ventricular beats are supraventricular in type but ventricular extrasystoles may occur as has been previously shewn. The irregularity of the ventricle in auricular fibrillation has been the subject of much discussion but it is now, I think, agreed that the irregularity is due to the conduction of impulses from the fibrillating chamber along the A-V bundle. The order may be reversed in ventricular fibrillation and a similar condition results when a portion of the auricle is made to fibrillate and its connection with the rest of the auricle reduced to a narrow band. If the bridge were wider then fibrillation itself would be conducted as pointed out by Garrey.

Auricular fibrillation may be paroxysmal or permanent - more commonly the latter. In the former case the onset and termination are sudden.

It is frequently seen in the senile heart especially when there is involvement of the coronary arteries e.g. J.C. aet. 75 yrs. complaining of cough and shortness of breath of nine months' duration. The shortness of breath has been progressive and for the past 8 or 9 weeks he has had attacks of difficult breathing and wheeziness in the early morning. These attacks are associated with sharp pain in the region of the heart. Past history was negative. Wassermann reaction was negative. The chest was emphysematous and the area of cardiac dulness much diminished. The heart sounds were distant, no bruit was audible, the second aortic sound was slightly accentuated. The heart action was very slightly irregular, its rate 90 per min.; no pulse deficit was present. The arteries were tortuous and thickened. Systolic blood pressure 198 mm Hg.; diastolic 103mm Hg. The urine examination was nil. A radiogram indicated globular enlargement of the heart and calcification of the aortic arch.
The electrocardiogram showed auricular fibrillation Fig. 17. There is slurring of the QRS complex in all the leads. Myocardial degeneration is obviously present.

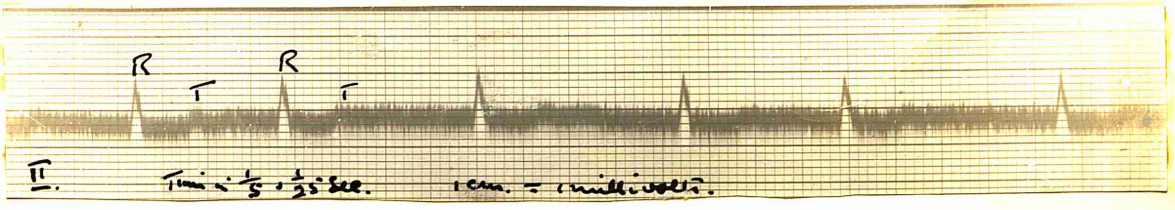


Fig. 17.

Arterial disease of syphilitic origin is accountable for some 15% of cases. Rheumatic infection is responsible for the greater proportion of cases. Fibrillation of this origin affects the sexes equally. It is most common in mitral disease particularly in mitral stenosis as in this condition the left auricle is subjected to great strain. It may occur in aortic incompetency but this is comparatively rare. It sometimes occurs in the terminal stages of exophthalmic goitre. It is very rarely seen in acute infections.

What actually precipitates the condition is still in dispute. Many hearts have now been examined post mortem and degenerative changes, involving the auricular muscle, are the rule. Fibrosis of the sinus node and interstitial myocardial involvement especially of the right auricle, as described by Dr. Cohn in 1918, would appear to be a not uncommon finding. In some cases the sinus node

escapes but the auricular muscle is involved; in other cases the sinus node only is affected. At the same time inflammation of the sinus node has been reported with the maintenance of a normal rhythm. In most cases, there is wide-spread involvement of the auricular wall and it is concluded that defective conductivity is the cause. This explanation was first suggested by Ritchie in 1912

It has been considered possible that auricular fibrillation is due to loss of control of the auricles by sinus block. From our anatomical knowledge of the sinus node and its intimate relationship with the auricular muscle it is impossible to imagine a lesion which would effectively block the impulses from the node and yet be so limited in extent as to involve no other structures. In the cases where sino-auricular block occurs it is probably of vagal origin.

Lewis at one time attributed auricular fibrillation to a number of foci in the auricular muscle each producing localised contractions. He later abandoned this theory and has now accepted the circus theory of Mines and Garrey. According to the authors, shortening of the refractory period of the muscle and delayed conduction are necessary for the appearance of circus movement. Under these circumstances, when a stimulus is applied at a point in the ring of muscle the contraction wave arrives back at the initial point of stimulation when the muscle there is again in a non-refractory phase; so the wave re-enters this path. This is the explanation offered of the mechanism in flutter; to explain fibrillation it is assumed that in this condition a number of blocks are present in the auricular muscle so that the wave becomes broken up and variable in direction.

The action of the cardiac nerves in relation to auricular fibrillation is interesting. Experimentally, Lewis has shewn that vagal stimulation may suppress it in very recent cases; but if fibrillation be of longer standing, vagal stimulation will establish it more firmly. In some cases, vagal stimulation may precipitate it - probably by depressing conductivity. It is possible that the action of digitalis in inducing fibrillation in certain cases is in part due to vagal stimulation. It is well known that vagal action is somewhat variable, apparently exerting a more profound influence on any function which is already depressed. It is quite conceivable, therefore, that in a heart in which conduction is somewhat defective stimulation of the vagi will so increase it as to induce fibrillation.

Fibrillation of the auricle resembles fibrillar contraction occurring in skeletal muscle from loss of nerve control. Section of a motor nerve will produce it; also nerve degeneration in certain chronic diseases of the nervous system e.g. progressive muscular atrophy. Even irritation of the nerve fibrils by inflammatory exudate or other toxins will produce it in skeletal muscle as is seen in peripheral neuritis.

An interesting case, in this connection, was under observation some few months ago.

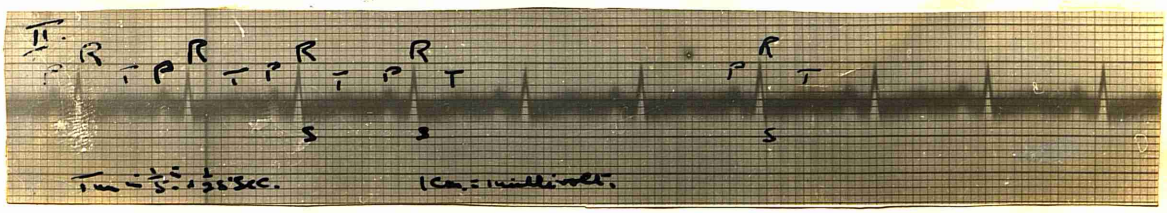
J.P. aet. 13yrs. Admitted to hospital 22.6.22. He complained of pain in the back, legs and knees. There was no swelling of the joints and no nodules, either subcutaneous or intramuscular. There was no oedema.

He had been under treatment in hospital for subacute rheumatism and disease of the heart at varying intervals during the preceding four years.

He was thin and pale - the blood examination indicated a secondary anaemia. The pupils were dilated.

The pulse was 84 per min., regular in rate and rhythm. The systolic blood pressure was 110mm Hg.: the diastolic, 78mm Hg. The electrocardiogram which I obtained showed a normal spread of the excitation wave. Fig. 18.

Fig. 18.



The venous pulse was ventricular in form indicating auricular engorgement.

There was systolic retraction of the epigastrium and Broadbent's sign was well marked. The maximum impulse was in the fifth space 4½ in. from the mid-sternum; area of cardiac dulness was 6in. by 7in.; the right cardiac border was 2in. to the right of the mid-sternum. A loud systolic bruit was audible at the apex and extended round to the left paravertebral groove. There was accent on the first sound. The second pulmonic sound was accentuated; the second aortic was closed.

The lungs were clear; the liver was not enlarged.

The temperature during the first fortnight was swinging in type - 98.8F. in morning to 99.8F in evening. The pains in joints and

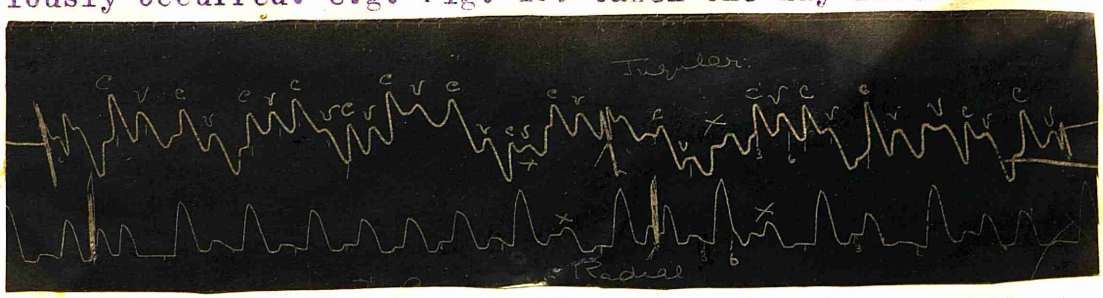
muscles were shifting in character. Gradually the temperature range increased, average swing being 97 - 100F during the third week. The average pulse rate was increased to 104 per min. There was no change in the physical signs.

23.7.22. Temp. 101.4F. Pulse 104 per min., regular. Patient complained of pain in the left front of the chest. Friction was audible in the third and fourth left interspaces close to the sternal margin. The pain passed off during the day.

24.7.22. He complained of pain along the right sternal edge and fine crepitant friction was audible there. The heart action was rapid - 116 per min. - and tumultuous. The systolic bruit was still very loud and harsh.

25. 7. 22. There were a few reversible crepitations - the "oedème à bascule" of the French - audible at the left base. Temp. 102.6F; pulse 118 per min.

27.7.22. At 2 a.m. patient complained of great discomfort in the chest. Breathing was embarrassed. The cardiac action was rather quieter but totally irregular. The ventricular rate was 145 - 160 per min. but difficult to count on account of the extreme irregularity. Pulse deficit was slight - the pulse rate was 140 - 145 per min. The systolic bruit was less loud. Auricular fibrillation had obviously occurred. e.g. Fig. 19. taken one day later.



The temperature subsided gradually; the pulse became rather steadier but the improvement was temporary. Slight recurrence of temperature, rheumatic pains and sore throat again upset the heart - there was slight increase of rate, averaging 118 per min., still irregular. Digitalis had no effect. Friction was still present along the right sternal edge. The area of cardiac dulness was increased particularly to the right - the right border was 4 in. from the mid-sternum. The area of cardiac dulness was 6 in. by 8½ in. Cardiac failure with general anasarca set in and the patient died 23.9.22.

Post mortem examination of the heart:-

The heart was greatly enlarged, weighing 2½ lb. The pericardium was much thickened and adherent to the pleura and chest wall.

There were a few ounces of fluid in the pericardial sac. The epicardium was thickened and shaggy. The thickening was general but most marked over the right heart where it averaged $\frac{1}{8}$ in. in thickness. It was most marked over the auriculo-ventricular junction.

The left ventricle was hypertrophied and dilated; the wall was $\frac{3}{4}$ in. thick at the base and the cavity 3 in. in transverse diameter. The left auricle was dilated. Its endocardium showed thickening and early puckering above the anterior cusp of the mitral valve, and extending on to the base of the cusp. There were thickening and shortening of the chordae tendinae and fibrosis of the tips of the papillary muscles. The accent on the first sound, audible during life, was obviously due to the impact of blood on the mitral valve in a condition of moderate fixation. The mitral orifice was not narrowed.

The aortic valve was competent. The left lateral and the non-coronary cusps of the aortic valve were adherent along their adjacent surfaces and an old vegetation was present. Serial sections were cut - well-formed fibrous tissue was found with fatty cells and crystalline debris in the centre of the vegetation. There were very few cells and no organisms found.

The right ventricle was not greatly enlarged. The wall was $\frac{1}{4}$ in. thick. The right auricle was slightly dilated. The tricuspid and pulmonic valves were normal.

Microscopic examination.

Several blocks were made from the ventricular muscle and examined. The sections showed great thickening of the pericardium but no definite extension of the inflammatory process into the myocardium e.g. Fig. 20. The muscle stained normally.

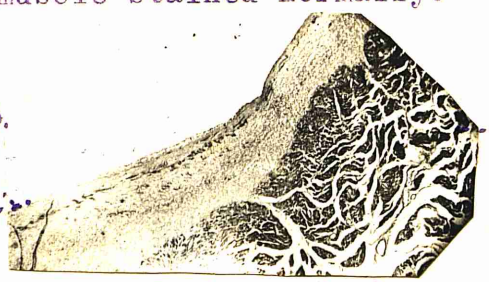
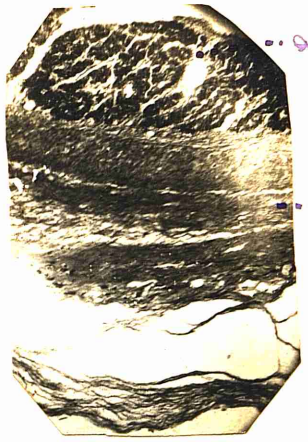


Fig. 20.
T.S. ventricular muscle (x10).
Stained haematoxylin-Van Gieson's
Reduction method.

Serial sections were cut of the auriculo-ventricular node, bundle and divisions and this tract was perfectly healthy. There was no ievolvement of the adjacent auricular or ventricular muscle.

Serial sections were cut of various portions of the auricular wall. As in the ventricle the pericardium was greatly thickened; the endocardium showed slight patchy involvement in sections of the left

auricle only. The auricular muscle was not invaded and stained normally. e.g. Fig. 21.



Section:. Auricular Wall. (x30).

Stained. Haematoxylin & Van Gieson. Blue filter used.

A block was next removed to include the sino-auricular junction with the sinus node. Sections were cut and I found marked cellular infiltration in this region. The infiltration commenced in the pericardium, peri-arterial in distribution and was small round-celled in type. e.g. Fig. 22.a.&b.

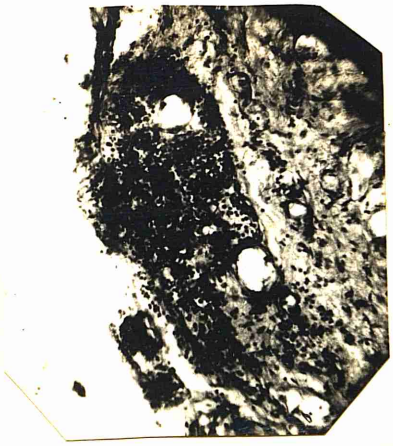


Fig. 22 a. (x60)

Fig. 22.b. (x60).

The vascularity of the pericardium was increased. The infiltration spread inwards along the arteries Fig. 22b and involved the numerous nerve branches in this region. e.g. Figs. 23&24.

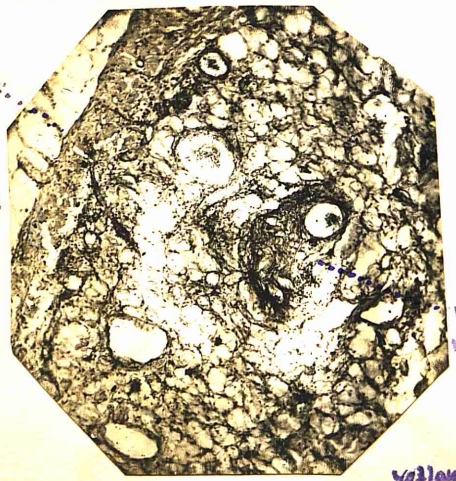


Fig. 23. (x30)

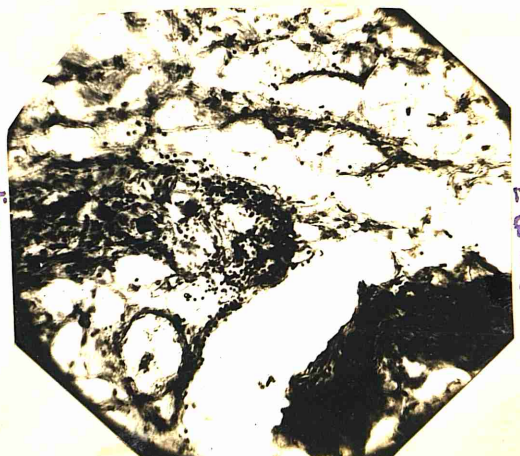


Fig. 24. (x60)

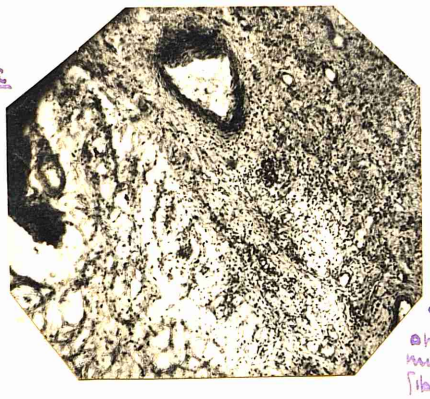
yellow filter used to contrast nuclei.

This involvement of the nerves was visible in nearly all the sections.

The sinus node showed localised involvement. Several of the sections showed no invasion of the nodal tissue; the muscle fibres showed striation and the nuclei stained well. In one or two sections, however, the node was found to be invaded by round-celled infiltration, e.g. Fig. 25.a.b.

a.

Nodal tissue (x.90.)



b.

Nodal tissue (x 60)

Showing localised round-cell infiltration

Stained haematoxylin & eosin

Yellow filter used to show up nuclei: muscle & connective tissue fibres are not differentiated.

There was chronic venous congestion of the lungs, liver, kidneys.

It seems clear in this case that the onset of fibrillation was due to inflammatory invasion of the sino-auricular junction interfering with the innervation of the node and to some slight degree involving the node itself. That the heart was already overtaxed and embarrassed by adhesions and the muscle probably ill-nourished would predispose to the onset. The most striking features of the case were the development of pain over the right heart, followed by auricular fibrillation, and the finding post-mortem of recent involvement of the sinus node and the nerves in the sino-auricular region.

It is also of interest that early degenerative changes were found in the thyroid gland. There were no signs to suggest the presence of this during life other than the dilated pupils but post-mortem I found a mixture of early adenomatous and colloid goitre. The degeneration was probably the result of the increased strain on metabolism resulting from the rheumatic infection. But once present it probably exerted an injurious influence.

Many facts suggest that there is in some cases a toxic basis of auricular fibrillation. The toxic muscle is defective in conductivity. e.g. J.R. aet. 54 yrs., of nervous temperament, complains of pains in the head, neck and praecordium and occasionally in the left arm. Auricular fibrillation has been present for 12 years. There is practically no enlargement of the heart; there

is no valvular defect; arterial hypertension is slight. Symptoms of gastro-intestinal toxemia have been present for many years and there is dilatation of the stomach and colon with ptosis. It would appear that in this case the gastro-intestinal toxemia was the primary cause of the cardiac condition, having a direct effect on the heart muscle.

The symptoms associated with auricular fibrillation depend on the integrity of the ventricular muscle and proper treatment. It is also important to note that in cases uncomplicated by valvular disease the efficiency of the heart is thereby increased. The benefit derived from digitalis is almost entirely due to the slowing of the heart which is due to a degree of heart-block produced mainly by a direct effect on the muscle of the A-V bundle, as shown by Cushny, and produced to a lesser degree, by stimulation of the vagi.

The symptoms actually referable to the fibrillating auricle are few. At the onset of fibrillation and in paroxysmal cases a certain number of patients complain of palpitation or discomfort in the chest.

A small proportion of cases complain of pain in the usual distribution of cardiac pain.

Auricular fibrillation would appear to be closely allied to auricular flutter which consists of rapid, regular, co-ordinate auricular contractions. The striking difference between fibrillation and flutter is the regularity in rhythm of both auricles and ventricles in the latter condition. Many points of connection, however, between fibrillation and flutter render it necessary to discuss the condition here.

Auricular Flutter was first described by MacWilliam in 1887. He produced it experimentally. In 1905 Ritchie described the first case. In 1909 Morison described a third case with audibility of the auricular contractions. Since then many cases have been reported but it is a much less common condition than auricular fibrillation.

Auricular contractions occur at the rate of 300 - 500 per min. They are remarkably constant in form and rate in any particular case. In electrocardiograms the P waves are diphasic.

The abnormal rhythm would appear to be free from nerve control as it is quite uninfluenced by the usual factors which induce inhibition or acceleration. In one case however, reported by Levine and Frothingham, the auricular rate was affected by deep respiration. A case reported by Ritchie is also of interest in that the

rate of the auricular contractions varies greatly; it was not, however, affected by pressure on the vagus. The P waves in this case were monophasic and of the same form as the physiological P waves. The ventricular rhythm is usually regular but if conduction be defective it will become irregular. The ventricles do not respond, as a rule, to every auricular contraction; a degree of heart-block being produced, possibly by the prolongation of the refractory phase of ventricular action as suggested by Heard and Strauss. That defective conduction is not the cause in most cases is proved by paroxysmal cases and post-mortem findings.

Auricular flutter most frequently occurs in later middle life, and is often associated with arterial disease. It may however occur at any age. A few cases have been reported as a result of rheumatic carditis in children.

As a rule it occurs in the course of chronic heart disease but may arise in apparently healthy hearts. The transient nature of the affection in otherwise healthy hearts and in some cases with no discoverable pathological lesion would point to their being a toxic factor in its production in certain cases. It may occur during chloroform anaesthesia but this is very rare. It has been recorded during acute infective illnesses and acute heart disease. It may result from hyperthyroidism and one case which I observed some 18 months ago is of interest:-

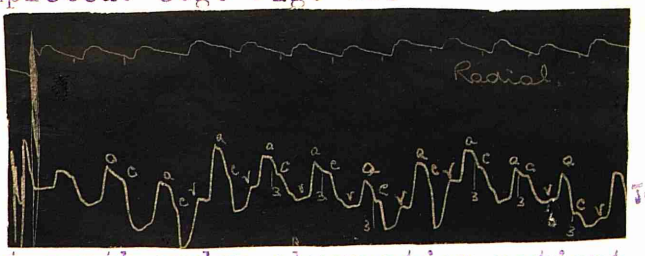
B.L. aet. 56 yrs. Admitted 9.3.22., complaining of shortness of breath, loss of weight and frequent sweating of some nine months' duration. During the three months preceding admission she had had frequent attacks of difficult breathing with wheezing in the early morning and had become nervous. She had suffered from palpitation and pain in the region of the heart for some 20 years and had been told that her heart was affected. She had been conscious of a swelling in her neck for at least 25 years but she thought it had become rather larger during the past two years.

Patient was very thin and her expression anxious; skin was moist; fine tremor of the hands was present; there was no definite exophthalmos. The thyroid gland was enlarged, particularly the left lobe and with a tendency to lobulation. It was not adherent to the surrounding structures. There was no thrill or bruit over the gland.

Pulse rate 92 - 112 per min., regular. Systolic blood pressure was 136mm Hg., diastolic blood pressure was high - 93mm Hg. - indicative of slight hypertension. The heart was enlarged - maximum impulse in fifth space 4½ in. from mid-sternum; area of

cardiac dulness was 5 in. by 4½ in. Systolic bruit was audible at the apex, traceable to the mid-sternum but not audible in the back. The second aortic sound was closed. Canter rhythm was present to the left of the sternum. The venous tracing showed a normal rhythm but the rapidity of the pulse interfered with the distinctness of the waves; the A waves occurring before the ventricular systole was completed. e.g. Fig. 26.

Fig.26.

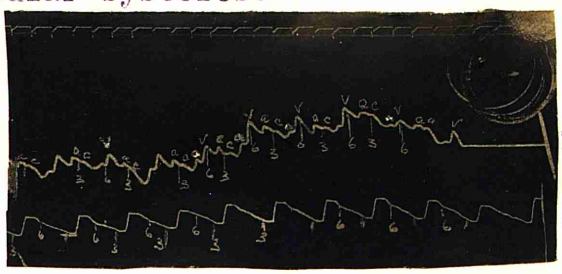


During the first month under observation patient continued to lose weight and at the end of that time began again to complain of wheeziness during the night. I saw her in several attacks of cardiac asthma in the early morning; they were associated with rapid pulse and canter rhythm.

7.5.22. Patient feels as usual - pulse rate 84 - 112 per min.

8.5.22. At 6 a.m. patient complained of severe palpitation. Pulse very rapid 156 - 160 per min. There was no engorgement or increased pulsation of the jugular veins. The systolic bruit disappeared. The tracing indicates the presence of auricular flutter - Fig. 27. There is a wave on the upstroke of "v" which occurs regularly at a constant interval from the "a" wave. It does not bear a constant relation to the "v" waves. It is auricular in origin. The auricular contractions may have been twice that of the ventricular or they may have been more frequent - the "a" waves are often obscured by the ventricular systoles.

Fig.27.



Patient was unfortunately unable to take digitalis or strophanthus.

10.5.22. The pulse continues at the same rate 160 per min. Pulsus alternans is present. The "acv" rhythm has quite disappeared and in some tracings two large waves, with smaller waves superimposed, are seen; in others there are found rapid minute waves occurring

throughout the whole tracing. (Care was taken to note that muscular twitching was not present). Fig. 28.

Fig. 28.

Unfortunately no electrocardiogram was available.
The blood pressure gradually fell to 108mm Hg., diastolic 76mm Hg. Patient felt very ill. She complained of a sensation of a lump in her throat which she could not swallow. Signs of failing circulation gradually set in.
14.5.22. Pulse rate fell to 108 per min. and symptoms ameliorated but her general condition was still very unstable. Pulsus alternans was still present. Blood pressure improved temporarily - systolic rose to 128mm Hg., diastolic to 90mm Hg. but on 23.5.22. a return of the high pulse rate occurred with a fall in systolic blood pressure to 110mm Hg. and in diastolic to 82mm Hg. The increased rapidity persisted with periods of irregular pulse. There was no definite onset of the irregularity - it would appear without any increase in the symptoms and would pass off quite as suddenly.
No tracing was available because of the extreme restlessness of the patient. The heart became more dilated - area of cardiac dulness was 7½ in. by 6 in. The liver became more enlarged. Cardiac failure increased steadily and patient died 6.6.22.

Post-mortem examination:-
The thyroid gland weighed 5 oz. It was enlarged, extending along the trachea into the thoracic cavity; it was firm and nodulated and on transverse section patches of colloid material were found with marked cystic and haemorrhagic degeneration. Calcareous deposits were present in the degenerated areas. On microscopic examination a mixture of colloid and adenomatous goitre with chronic inflammatory changes in the interstitial tissues, was found e.g. Fig. 29.a.b.

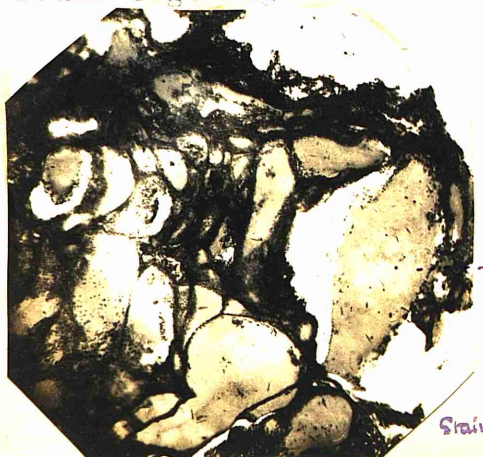


Fig. 29. a. (x10)

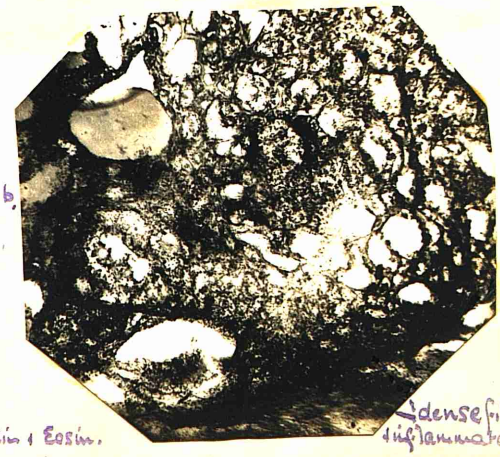


Fig. 29. b. (x10)

From the clinical picture - enlarged thyroid without thrill or bruit, delayed appearance of hyperthyroidism, absence of gastro-intestinal crises and exophthalmus, and presence of a degree of hypertension - and from the pathological findings, it was obvious that the patient had suffered from thyrotoxicosis as a result of adenomatous and colloid goitre.

The thymus was absent.

The heart weighed 1 lb 1½ oz. The pericardium and epicardium were normal. The right ventricle was dilated - its wall was ¼ in. thick at the base. The right auricle was much dilated and the musculi pectinati markedly separated. The valves of the right heart were normal.

The left auricle was slightly dilated as was also the left ventricle. The ventricular wall was a ¼ in. thick at the apex, ½ in. thick at the base. The aortic cusp of the mitral valve showed a few patches of atheroma. The aortic orifice was narrowed and the valve showed advanced calcareous deposit. The right lateral and medial cusps were firmly adherent along their adjacent surfaces and the edge of the cusps much thickened with calcareous masses. The left lateral cusp was puckered with calcareous nodules. The degenerative process had spread to the wall of the aortic sinuses but the orifices of the coronary arteries had escaped. The aorta showed patchy atheroma in its ascending portion. No lesion of the coronary arteries was found in their main portions or larger divisions. Microscopic examination of the ventricular muscle showed extensive fibrosis involving particularly the apex of the heart and the muscle immediately underlying the endocardium. There was also definite periarterial fibrosis. — Fig. 30.

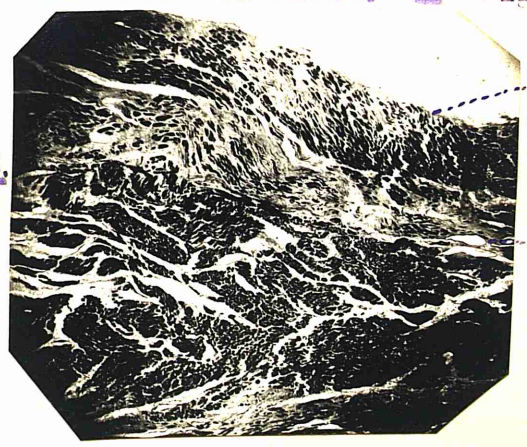


Fig. 30.
Section-Ventricular Muscle.
(×10).
Stained Haematoxylin & Van Gieson.

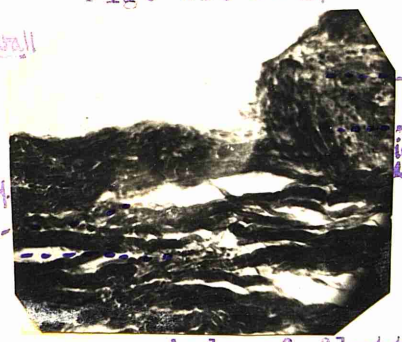
These findings were suggestive of some interference with the blood supply - the parts of the heart supplied by the finest divisions

of the coronary arteries being most involved; but in this case the quality of the blood must also have played an important part in determining the result.

The auricular muscle, particularly of the right auricle, showed extensive fibrosis. The muscle fibres were separated by well-formed fibrous tissue. Striation of the muscle fibres was present but the nuclei did not stain well. There was one small patch of lymphocytic infiltration found. The sinus node was not particularly involved.

Fig. 31. a. & b.

Section Auricular wall (X60)
Fig. 31. a.
Stains - Haematoxylin Van Gieson.
Fibrous t.



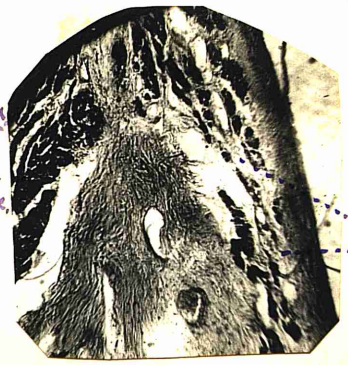
fibrous tissue
muscle fibres involved by fibrous tissue.

Fig. 31. b.

dense fibrous tissue.
fibrous tissue
muscle fibre.

In some cases periods of flutter will pass into fibrillation but in this case it is much more probable that the periods of irregularity were due to defective conduction in the auriculo-ventricular tract. I found the A-V node, bundle and right division healthy but the left division showed fibrosis e.g. Fig. 32.

Fig. 32.
Section of R. & L. Divisions A.V. Bundle (X30).
Stains - Haematoxylin & Van Gieson.
Fibre. Blue :, muscle fibres appear black.



R. division of A.V. Bundle.
L. division of A.V. bundle showing some increased fibrosis. Muscle fibres do not stain quite so well.

The degenerative change present would be quite sufficient to depress the conductivity of the muscle and this was still more likely to result when the auricular rate was greatly accelerated.

The liver, spleen and kidneys showed venous congestion.

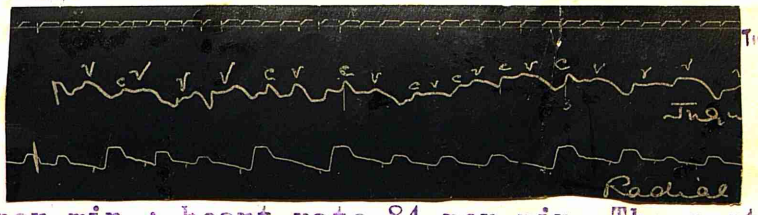
The onset of flutter was obviously associated with diffuse myocardial degeneration. The myocardial change was the result of thyroid intoxication in association with pre-existing heart disease.

In all cases in which a post-mortem examination has been made, disease of the auricular muscle has been present. It would appear

that depressed conduction in the auricular muscle is largely responsible for its occurrence. There is no evidence that it can be produced by nerve influences alone - section of the vagi or sympathetic stimulation. It would appear however that in certain cases there are nervous influences at work.
The associated symptoms of flutter depend on the ventricular rate.

Experimentally and clinically there would appear to be some connection between auricular extrasystoles, flutter and fibrillation. Experimentally it has been found that weak faradization of the auricular wall will produce a series of auricular extrasystoles; stronger stimulus will produce auricular flutter and if the strength of stimulus be still further increased auricular fibrillations will supervene. Vagal stimulation will convert flutter into fibrillation and this usually occurs when digitalis is administered in cases of flutter. The reverse order may occur when quinidine sulphate is given. The inter-relation of these three conditions is well illustrated in the following case:- M.C. aet. 49 yrs. was admitted 19.6.22. complaining of shortness of breath, palpitation and sharp pain in the region of the heart. The symptoms dated from an attack of acute lobar pneumonia from which the patient had suffered some four years previously. They had varied in intensity during that period but had become rather more distressing two weeks previous to admission.
Patient was well nourished. There was no oedema and his urine examination was nil. The pulse was totally irregular and venous tracings demonstrated auricular fibrillation with moderate venous engorgement e.g. Fig. 33.

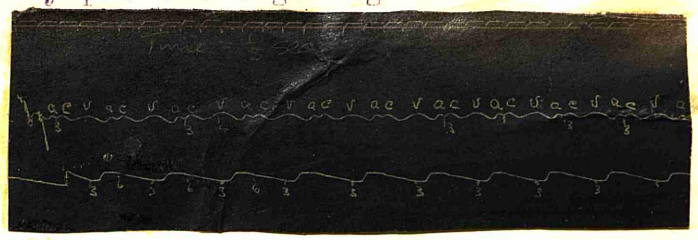
Fig. 33.



Pulse rate 72 per min.; heart rate 84 per min. The systolic blood pressure of the most forcible beats, was 135mm Hg; diastolic blood pressure 83mm Hg i.e. a pulse pressure of 52mm Hg was present. Maximum impulse was in the sixth space 7½ in. from the mid-sternum; Area of cardiac dulness was 5 in. by 7 in. By radioscopy the heart was found to be enlarged to the left.
There were some reversible crepitations at the base of the left lung.
The liver was not enlarged.

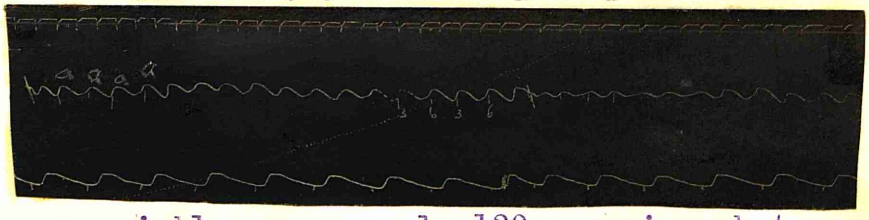
22.5.22. Quinidine sulphate grs. 6, t.i.d. given. After five doses the pulse was regular in rate and rhythm but the rapidity of the pulse was marked - 108 per min. The normal sequence of contraction was apparently present e.g. Fig. 34.

Fig.34.



This rate was maintained and the blood pressure gradually fell.
27.5.22. Quinidine stopped.
28.5.22. Patient complained of faintness. Systolic blood pressure 110mm Hg, diastolic 70mm Hg i.e. the pulse pressure was 40mm Hg. The polygraph tracing demonstrated regular action of the heart with disappearance of the normal sequence of contraction. Auricular flutter was apparently present e.g. Fig. 35.

Fig.35.

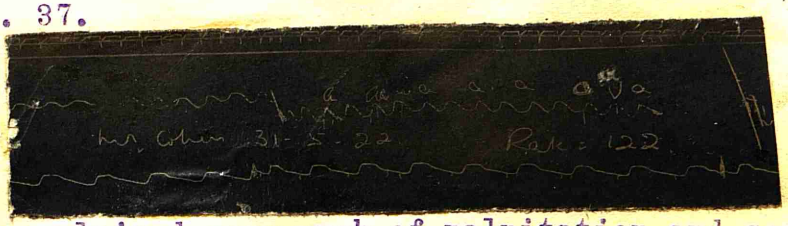


The rate was variable - as a rule 120 per min., but occasionally fell to half that rate which was a very significant feature.
No treatment was given.
30.5.22. Pulse irregular; a bigeminy was present in some tracings but the diastoles varied in length there was no "a" wave in the venous tracing e.g. Fig. 36.

Fig.36.

30.5.22. At 2 p.m. quinidine started t.i.d.
31.5.22. Pulse regular in rate and rhythm, 122 per min. The venous tracing was the same as that on 28.5.22. - flutter apparently present e.g. Fig. 37.

Fig.37.

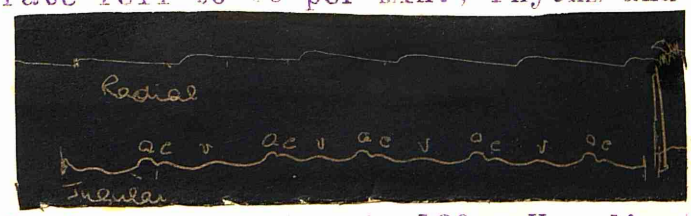


This continued.
4.6.22. Patient complained very much of palpitation and sense of

suffocation as he dropped off to sleep. Pulse rate 108 per min. and occasionally irregular e.g. Fig. 38.

In this tracing the ventricle responds as a rule to every second auricular beat but occasionally fails to respond to the usual auricular impulse and a ventricular contraction coincides with the next auricular impulse.

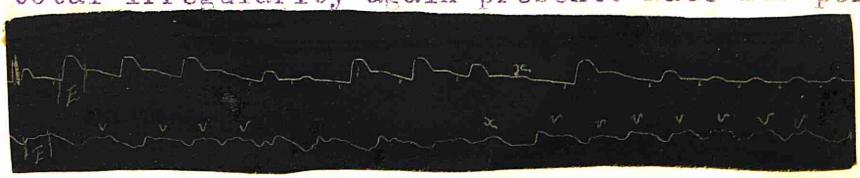
5.6.22. Pulse rate fell to 60 per min.; rhythm and rate regular e.g. Fig. 39.



The systolic blood pressure rose to 130mm Hg, diastolic to 85mm Hg A presystolic bruit was audible for the first time.
This rhythm was maintained during the following two months.
Patient was getting up 6 hours per day and feeling well.

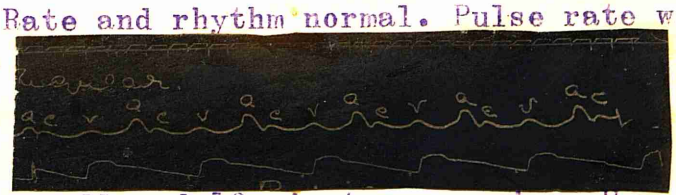
3.8.22. At 9 a.m. pulse normal in rate and rhythm.

At 11 a.m. total irregularity again present. Rate 100 per min. e.g. Fig. 40.

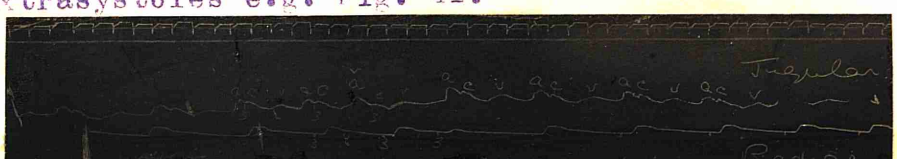


At 2 p.m. Quinidine grs. 6 given.

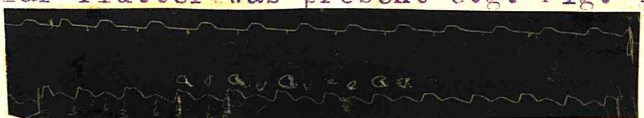
At 6 p.m. Rate and rhythm normal. Pulse rate was 75 per min. e.g. Fig. 41.



12.8.22. Patient was allowed 10 minutes exercise. He complained of palpitation following it and tracings demonstrated occasional auricular extrasystoles e.g. Fig. 42.



16.8.22. The condition again relapsed. Pulse rate was 132 - 140 per min. Systolic blood pressure fell to 112mm Hg, diastolic to 76mm Hg. Auricular flutter was present e.g. Fig. 43.



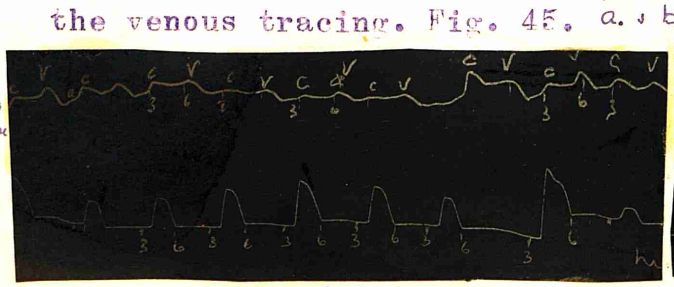
The patient complained greatly of weakness. He stated that he had to swallow to prevent fainting.
18.8.22 Tr. Digitalis min. 15 given t.i.d.
22.8.22. Auricular fibrillation present. Digitalis stopped e.g. Fig. 44.

Pulse rate 124 per min. Systolic blood pressure 124mm Hg, diastolic 86mm Hg. Quinidine sulph. grs. 6 t.i.d. started.
23.8.22. After three doses of quinidine sulphate the normal rhythm was restored. There was on the last two occasions apparently no intervening stage of flutter.

Polygraphic tracings are not always satisfactory in demonstrating flutter but in this case proof was adduced of the accuracy of the interpretation when the patient appeared at the out-patient department complaining of an attack similar to those in hospital. The pulse rate was 140 per min. Systolic blood pressure 105mm Hg. diastolic 70mm Hg. The presystolic murmur had disappeared. I took an electrocardiogram. v. opposite page.

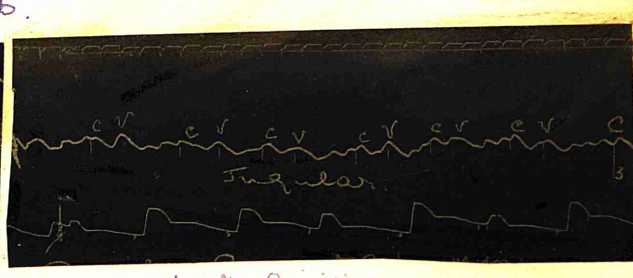
The interpretation of the tracings indicating the first appearance of the regular rhythm is open to question. The tracing could not be definitely said to indicate the normal rhythm which had a rate of 60- 70 per min. and was associated with the appearance of a presystolic murmur; nor did it resemble the tracings of flutter. What the nature of the rhythm was and what its origin - whether a normal, accelerated rhythm, an auricular tachycardia or flutter - can not be learnt from polygraphic tracings. As a rule in quinidine therapy the ventricular rate is accelerated but this occurs before the normal rhythm is restored; it occurs when the auricular oscillations are slowing and has been shown by experiment to be due mainly to the auricular slowing and in part to vagal paresis.

The above case illustrates both the action of quinidine sulphate and that of digitalis in auricular flutter.
The action of quinidine sulphate is of interest. I have observed nine cases. In two of the seven cases which did not respond to treatment, large oscillations, occurring in diastole appeared in the venous tracing. Fig. 45. a. & b.

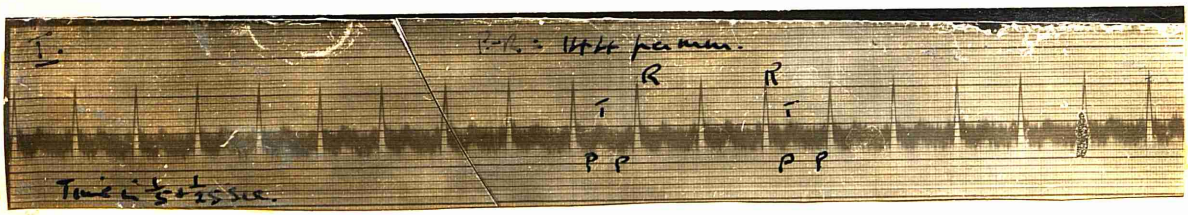


Time Racing Mitral because 95 p.m.

a. Before Quinidine given.



b. after Quinidine.



M.C. 11-12-22.

This change was associated with the complaint on the part of the patient of a fluttering sensation in the chest. These oscillations may have been indicative of transitions between fibrillation and flutter.

In one case only was the normal rhythm restored with any degree of permanency; the condition relapsed once within the following 18 months. In 4 cases in which definite organic disease was present from old rheumatic infection, quinidine was harmful; even when combined with digitalis no good resulted. In two cases no reaction was obtained. One case died suddenly after seven doses of gr. 6 had been given. The ventricular rate had increased from 84 to 100 per min.; otherwise no change had occurred. Sudden death from quinidine may be due to ventricular fibrillation. In some cases quinidine exerts a toxic action on the ventricular muscle; multiple ventricular extrasystoles may appear and are probably the forerunners of fibrillation. Embolism may follow restoration of the normal rhythm, particularly in long-standing cases.

The action of quinidine sulphate has been investigated experimentally and it has been found that it lengthens the refractory period of the muscle and depresses conduction. Applying these results to the circus theory Lewis concludes that the results from quinidine depend on which effect is the predominant - if prolongation of the refractory period be the important feature, then circus movement will cease as the contraction wave will be no longer able to re-enter its path because of the refractory auricular muscle; if depressed conduction be the principal result then the wave will travel less quickly but circus movement will persist. If this be the explanation of the action of quinidine then it is only to be expected that its use will be attended with very limited success. It exerts no influence on the pathological changes in the auricular muscle which are of prime importance.

The whole subject of cardiac arrhythmia is of intense interest. The more irregularities of the heart are studied the more obvious does it appear that extrasystoles, tachycardia, flutter and fibrillation are allied in nature. Experimental proof is not lacking, and has already been referred to, that in hearts with extrasystolic arrhythmia, the highest grade arrhythmia, namely fibrillation, may, under suitable conditions, be produced by cardiac stimulation. Unfortunately the same sequence has been seen clinically in treatment by quinidine and very occasionally by digitalis when the warnings by extrasystoles have been neglected.

The apparent difference between fibrillation of the ventricle and that of the auricle is merely a question of difference in structure of the two chambers. Faradic stimulation of the auricle will produce extrasystoles, flutter and fibrillation according to the strength of stimulus, and clinically it is a matter of common experience that auricular extrasystoles precede the onset of fibrillation.

The process underlying the single extrasystole would thus appear to be closely related to that underlying the grave disorders of cardiac rhythm. It has been said that extrasystoles may occur in normal hearts but to say that, is to go too far - all we can say is, that in a certain number of cases exhibiting extrasystoles we can find no lesion to account for them. In the greater number of cases there are some abnormal conditions, which are not a constant, which predispose to their occurrence, and in the case of the higher grades of arrhythmia these abnormal conditions are usually profound.

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I examined sections of the S.A. node in the human and other vertebrates' hearts, at the Royal College of Surgeons, London.

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