

FIBRILLAR CONTRACTION OF THE HEART. BY JOHN
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MANY years ago Ludwig and Hoffa¹ showed that the application of strong constant currents or faradic currents to the ventricles of the dog's heart causes an abolition of the normal beat. The ventricular muscle is thrown into a state of irregular arhythmic contraction, whilst there is a great fall in the arterial blood pressure. The ventricles become dilated with blood as the rapid quivering movement of their walls is insufficient to expel their contents; the muscular action partakes of the nature of a rapid incoordinated twitching of the muscular tissue. This condition persists for a very long time in the dog, and as Ludwig showed, it is possible to kill an animal in this way—by applying a faradic current to the ventricles. The auricles go on beating rhythmically; they do not participate in the irregular movement excited in the ventricles. These phenomena are familiar to all who have worked much with the mammalian heart; they have been designated by various names—Herz-delirium, Delirium cordis, Fibrillar contraction, Intervermiform movement, &c.

During the last two years I have performed a large number of experiments bearing upon this subject. My earlier investigations were pursued in the Physiological Laboratory of University College, London, and the more recent ones in the Physiological Laboratory of the University of Aberdeen. I have studied the phenomena in question in the hearts of the dog, cat, rabbit, rat, mouse, hedgehog and fowl; both in the young animal and in the adult.

The experiments were all conducted on completely anaesthetised animals; artificial respiration was carried on, a cannula being inserted in the trachea; the thorax was opened in many cases and the heart laid bare; the temperature of the animal was kept up by means of a warm pan.

¹ *Zeitschrift f. rat. Medicin*, 1850, vol. ix.

I shall briefly state the main facts in my investigation.

I. The state of arhythmic fibrillar contraction is essentially due to certain changes occurring within the ventricles themselves. It is not due to the passage of any abnormal nerve impulses to the ventricles from other parts, or to the interruption of any impulses normally transmitted to the ventricles and necessary for their normal co-ordinated action. The condition is not due to injury or irritation of the nerves that pass over the ventricles from the base of the heart.

The ventricles contain within themselves the entire mechanism necessary for the execution of regular co-ordinated beats. They are not dependent for this power on any nervous or mechanical connection with other parts. The continuity of the nerves that pass from the auricles to the ventricles is not at all essential for the execution of regular and effective beats by the ventricles; nor is the mechanical connection between those parts necessary. This is obvious from the fact that when a section is made through the auriculo-ventricular groove so as to separate the ventricles entirely from the auricles, the isolated ventricles can still exhibit their co-ordinated rhythmic contraction. Instead of cutting off the ventricles Wooldridge¹ and Tigerstedt² physiologically disconnected the ventricles from the auricles so as to destroy all vital connection between them while the parts were still kept *in situ* and the flow of blood through the cavities of the heart was allowed to go on; the ventricles went on beating in regular fashion though at a slower rate than before. I have frequently performed a similar experiment and have watched the ventricular action as it went on, strong and regular for prolonged periods. It is evident that neither the nervous, nor the mechanical connection between the auricles and the ventricles is necessary for the effective contraction of the latter. It is clear that a mere solution of the continuity of the nerves passing to the ventricles does not destroy the character of the ventricular beat; and it is plain, that such a solution of continuity cannot be the cause of a sudden replacement of the normal systole by the arhythmic fibrillar form of contraction.

Nor is the fibrillar contraction due to irritation of those ventricular nerve trunks. Many observers have noticed its occurrence when the nerve trunks on the surface of the ventricles were being stimulated. But such results appear to be due entirely to an escape of the exciting

¹ *Arch. f. Anat. u. Physiol.* 1883.

² *Arch. f. Anat. u. Physiol.* 1884.

current to the underlying ventricular substance. For when the nerve-trunk is isolated for some little distance and precautions are taken to prevent an escape of the current, I have never found the nerve stimulation to have any effect at all in inducing the fibrillar contraction. Moreover, an interrupted current readily brings about the arhythmic fibrillar condition when applied to regions of the ventricles where there are no nerve-trunks, e.g. to the very apex of the heart. Even mechanical or thermal stimulation applied to this region may lead to the same result.

The arhythmic fibrillar contraction is undoubtedly a phenomenon depending on changes within the ventricular substance; it can occur quite independently of any mechanical relation of the ventricles to the rest of the heart, and of any nervous relation of the ventricles to the rest of the heart or to the extra-cardiac nerves. The isolated ventricles whether in the quiescent state or beating rhythmically, can by the application of faradic currents be readily thrown into the characteristic fibrillar state, just like the ventricles of an intact heart. And in the intact heart the fibrillar contraction appears to be entirely uninfluenced by nerve excitation of any kind; stimulation of the vagus or any other nerve appears to produce no effect whatever.

Further, the fibrillar contraction can be propagated from one part of the ventricular substance to another quite independently of the nerve-trunks. For if a number of overlapping incisions be made across the long diameter of the ventricles so as to leave the apex attached to the rest of the ventricles by a zig-zag isthmus of tissue, it often occurs that fibrillar movement excited by faradisation in the apex travels along the zig-zag isthmus of connecting substance, and so comes to pervade the whole of the ventricular tissue.

II. The arhythmic fibrillar contraction is not necessarily dependent on the destruction or paralysis of a co-ordinating centre located in any particular part of the ventricles.

Kronecker and Schmey¹ succeeded in throwing the ventricles of the dog's heart into the state of fibrillar movement by piercing with a needle a certain limited part of the ventricular septum near the junction of its upper and middle thirds. This result these investigators attributed to the destruction of a centre located in that region, and normally presiding over the co ordination of the ventricular muscle in the execution of its regular beat.

¹ *Sitzungsber. d. Berliner Acad.* 1884.

There is conclusive evidence that all cases of fibrillar contraction of the ventricles cannot be explained by such a hypothesis—the destruction of a co-ordinating centre localised as indicated above. The fact that recovery may take place—that the ventricles may resume their co-ordinated rhythm, controverts the idea of the actual destruction of a centre essential for co-ordination. Such recovery I have witnessed in several instances in the dog's heart, and in a very large number of instances in the hearts of other animals (cat, rabbit, rat, mouse, hedgehog and fowl). Recovery occurs with different degrees of facility in different animals and in different conditions in the same animal. In the dog, recovery occurs with much difficulty and only after the fibrillar contraction has lasted for a considerable space of time; indeed there very frequently is no recovery apparent—the ventricles may not recommence beating after the inco-ordinated quivering movement has ceased. At times however a number of regular beats are seen after the termination of the fibrillar contraction. A depression of the excitability of the ventricular tissue often appears to favour recovery.

In most mammals recovery commonly occurs. Very often it is possible to induce the fibrillar movement again and again, complete recovery occurring in the intervals, when the normal systoles are seen. In young mammals, foetal or after birth, recovery appears to be the rule; the fibrillar movement is only a temporary condition, and soon gives place to normal beats.

In birds also I have frequently observed complete recovery. The fibrillar condition is readily induced by faradisation. The ventricles exhibit the characteristic quivering movement; they become dilated with blood. In consequence of the stagnation of blood in the ventricles the auricles also become gorged and may become so over-distended that they temporarily stop beating; asphyxial convulsions occur in the skeletal muscles. After a time however the fibrillar movement ceases, the ventricles remain quiescent for a little time, then give a regular co-ordinated beat and the action of the whole heart proceeds in the normal fashion. These phenomena can by the application of a current of the proper strength be induced again and again.

Further, in addition to the evidence afforded by the recovery of the ventricular beat, there is the fact that the arrhythmic fibrillar movement may very readily be induced by means that are not capable of destroying a deep-seated co-ordinating centre e.g., faradic, mechanical, or thermal stimulation of the surface of the ventricles even at the very apex.

Since it is certain that the arrhythmic fibrillar movement is not

necessarily due to the actual destruction of a co-ordinating centre, there next arises the question as to whether the fibrillar contraction may be due to the temporary paralysis of such a centre as that indicated by Kronecker—of the existence of which no histological evidence has, as far as I am aware, been advanced.

I shall at a later stage of this paper have to adduce some evidence regarding the action of certain poisons which when injected into the blood lead to the occurrence of fibrillar contraction of the ventricles. Such a result might be regarded as due to the paralysis of a hypothetical co-ordinating centre. And the fibrillar contraction caused by stimulation (electrical, mechanical &c.) of the ventricular surface might be explained in a somewhat similar fashion. For it is conceivable that such stimulation might give rise to strong abnormal afferent impulses with the result of deranging or paralysing the action of the co-ordinating centre; the paralysis might be a temporary one or might be permanent according to the particular circumstances in each case.

But there is strong evidence against the adoption of such a view—against the idea that the phenomena are due to the behaviour of a definite co-ordinating centre localised above the middle of the ventricular septum in the dog's heart. For the influence of such a centre does not appear to be at all essential for the production of co-ordinated and efficient beats. The amputated apex—the lower third or fourth of the ventricles—both in the dog and in all other mammals I have examined,—is capable of executing co-ordinated beats when it is entirely removed from all possible relation with any co-ordinating centre high up in the ventricular septum. This one can verify by the rough but conclusive experiment of tying the freshly removed apex of a vigorous heart upon a double cannula through which the cavity of the left ventricle can be filled with blood; the propulsion of fluid at each beat of the isolated apex can be readily observed. The visible character of the beat may also be noted, and the co-ordinated nature of the contraction causing a marked diminution of the cavity at each systole may be felt with the finger tip inserted into the cavity of the left ventricle. It is obvious then that the paralysis of a co-ordinating centre in the upper half of the ventricular septum would not necessarily cause a loss of co-ordination in the contraction of the whole of the ventricular muscle.

Further there is the fact that the apical portion of the ventricles—capable as it is of performing regular beats—can be thrown into a state of fibrillar contraction by the usual means, e.g. the application of a

faradic current. In the isolated apical part of the ventricles (in all the mammals I have examined) I have been able to excite the fibrillar contraction again and again, recovery occurring in the intervals, and co-ordinated beats being given in response to single stimuli applied during those intervals. It appears then that the behaviour of the intact ventricles and of the entire isolated ventricles both as regards co-ordinated single beats and as regards the fibrillar contraction can be reproduced in the isolated apical portion; and hence we may conclude that these phenomena are not necessarily dependent on the condition of any co-ordinating centre in the upper half of the ventricles.

III. The outstanding features of the arrhythmic fibrillar contraction are:—

- (1) The complexity of the movement.
- (2) Its persistence.
- (3) Its rapidity.

The complexity of the fibrillar movement appears to be in direct relation to the complex arrangement of the muscular fibres of the ventricular walls.

In the ventricles we have bundles of muscular fibres forming by their interlacement a texture of remarkable complexity. It appears that the complex quivering movement depends on the passage of rapidly repeated waves of contraction along the complexly arranged muscular bundles which are enclosed by connective tissue and joined to one another by cross-branches. It is readily conceivable that contractions simply conducted along the muscular fibres should be transmitted with unequal rapidity along the ventricular walls and should reach the same part of the ventricular wall at different points of time. Some bundles of fibres are in a state of contraction while neighbouring bundles are relaxed and so instead of a co-ordinated contraction causing a definite and (in the case of the left ventricle) concentric narrowing of the ventricular cavity, there occurs an irregular and complicated arrhythmic oscillation of the ventricular walls which remain in a position of diastole.

That the complexity of the fibrillar movement in the grown animal depends on the character of the muscular structure is illustrated by the appearances presented by the corresponding movement in the hearts of foetal and young animals. In these as long as the structure of the ventricles is simple the rapid movement excited by faradization is of a simple character. And just as the complexity of the muscular structure

increases in the growing animal so does the complexity of the movement obtained. There can be observed a complete gradation from the simple movement excited by faradisation in the ventricles of the mammalian foetus or of the chick (a movement much resembling that seen in similar circumstances in the comparatively simple ventricles of cold blooded animals) to the very characteristic and striking complexity of the fibrillar contraction in the adult mammal or bird. It is obvious that the nature of the muscular structure is a cardinal feature, and it is not very evident why such should be the case if the condition is due to derangement of a nervous mechanism causing it to discharge irregularly; for a deranged nervous mechanism discharging irregularly might cause an equally irregular movement whether the muscular arrangement is simple or complex.

The simpler character of the movement excited by faradisation in the auricles of warm-blooded animals is probably due to the simpler histological structure of the auricular walls and the simpler mode of propagation of the normal contraction.

The persistence of the fibrillar contraction appears to depend on the high excitability of the ventricular tissue.

When the fibrillar contraction has been brought about by stimulation of the ventricles, the prolonged continuance of the movement, after the cessation of the exciting cause is a striking feature. It appears to be a result of the excitation of a highly excitable, and probably highly rhythmic tissue. The duration of the movement, varies in each instance with the excitability of the ventricular muscle. It can easily be shown, that in certain depressed conditions of the ventricular tissue, the duration of the fibrillar movement, induced by stimulation is much diminished, and when the ventricular excitability is very much lowered, (by gradual cooling, exhaustion etc.) it frequently occurs that the fibrillar contraction does not persist after the stimulating current is discontinued; it simply occurs during the passage of faradic current and passes off at the cessation of that current. Indeed, in some instances it may be found that the fibrillar contraction cannot be excited at all by faradisation, whilst the ventricles are still capable of executing single beats. A certain degree of excitability is necessary for the production of the fibrillar contraction in response to stimulation.

Similar facts with reference to the duration of movement, after the discontinuance of the exciting cause, may be seen in the hearts of cold-blooded animals. In the heart of the eel, for example, where there are a number of parts possessed of different degrees of excitability and

rhythmic power, very marked differences are to be observed in the behaviour of the several parts after a stimulating current has been temporarily applied. The sinus with the basal wall, and the canalis auricularis, the auricle and the ventricle, form a descending series as far as rhythmic power is concerned, and they present similar differences as regards the after effects of stimulation. In the ventricle a short period of moderate stimulation excites a movement, which usually terminates immediately or very soon after the end of the stimulation; the precise period at which the movement terminates, varies according to the strength of the exciting current and the excitability of the ventricle; in a very excitable ventricle (in situ with the normal circulation intact) the movement may persist for some little time after the stimulation has ended. In the auricle the movement usually persists longer, and in the sinus a great deal longer still. Indeed, in the sinus a single stimulation can often lead to a series of beats, whereas in the case of the auricle, and still more in the ventricle a single stimulation excites but a single contraction. Moderate heating of the tissue causing a rise in its excitability usually leads to a marked increase in the persistence of the movement excited by a short period of stimulation.

Similarly in the mammalian heart the duration of the fibrillar movement after the end of the period of excitation varies. In the foetal heart it lasts but a short time, and in adult hearts that have been much depressed by exhaustion and by gradual cooling the fibrillar movement usually passes away very much earlier than it does in a more excitable heart.

The mechanism of the movement, as will be subsequently stated, appears to be such as to involve its continuance as long as the excitability of the ventricular tissue is sufficiently high.

The cause of the great rapidity of the series of contractions that course over the ventricular fibres during the state of fibrillar contraction will be considered later on.

IV. The arhythmic fibrillar contraction is in one class of cases a phenomenon of irritation induced by the action of various recognised stimulants.

The state of excitement generated in the muscular tissue appears to resemble in some respects the state of excitement obtaining in the nerve cells of the cortex cerebri during an attack of epileptiform convulsions induced by strong stimulation.

It has been stated that the duration of the fibrillar contraction

depends on the excitability of the ventricular tissue. In like manner the readiness with which the fibrillar contraction can be excited by stimulation, is in close relation with the ventricular irritability. In a depressed heart it is frequently very difficult to produce the phenomenon in question by stimulation; very powerful currents are necessary.

On the other hand when the excitability is heightened, it is easy to induce the fibrillar contraction. The occurrence of this phenomenon in response to stimulation is retarded and its duration shortened by conditions that depress the excitability of the cardiac muscle; its occurrence is favoured and its duration prolonged by causes that augment the cardiac irritability. In an exhausted heart it can frequently be seen that faradisation of the right ventricle leads to the occurrence of the fibrillar contraction in both ventricles, when such a result has ceased to be obtained by faradisation of the left ventricle. The difference in the behaviour of the ventricles, in this respect appears to be due to the greater persistence of the excitability in the right ventricle as compared with the left.

When the fibrillar contraction has been excited by stimulation it can often be arrested by the cautious application of depressant measures calculated to diminish the excitability of the ventricular tissue, e. g. deprivation of blood supply and cooling.

The readiness with which the ventricles are thrown into the fibrillar condition varies remarkably in different conditions of the cardiac tissues. In a normally-contracting and vigorous heart it usually requires a faradic current of considerable strength to produce the result in question. And it is not easy in these circumstances to induce the fibrillar contraction by mechanical or thermal stimulation. But in certain changed conditions of the organ it becomes extremely easy to throw the ventricles into the fibrillar movement. An exceedingly weak faradic current, a touch with a hot wire, a mere scratch with the point of a pin, slight friction of the ventricles against the cut end of a rib, or even slight pressure with the finger, are each of them sufficient at such times to excite the fibrillar contraction. The precise conditions in which there is such a remarkable sensitiveness to certain forms of stimulation are difficult to define; I have frequently observed such a sensitiveness when the action of the heart has been deranged or impaired by various causes—among others by a temporary arrest of the respiration or by a great fall in the blood-pressure leading to anaemia of the cardiac tissues &c.; the phase of increased sensitiveness seems to be a transitory one.

The frequent occurrence in the ventricles of such phases of extreme

readiness to assume the fibrillar form of contraction appears to me to be of great importance with regard to the question of electrical stimulation of the heart in man during sudden cardiac failure (syncope during the administration of anaesthetics, &c.). It is obvious that the use of faradic currents of any strength is attended with grave danger in such cases. For although Von Ziemssen and others have applied the induced current to the human heart without any serious results, the conditions were different in such cases. They experimented with normally-beating hearts, the tendency of which to assume the fibrillar form of contraction is strikingly less than what frequently obtains in hearts placed in abnormal circumstances—necessarily present in those cases where the faradic current is employed clinically.

But although the exposed heart in the opened thorax may be readily thrown into the arhythmic fibrillar contraction by faradisation, it may be urged that possibly the normally-beating heart in the intact thorax, is not similarly affected. I have on several occasions introduced a fine platinum wire electrode through the chest wall so as to come in contact with the ventricles, and have then faradised, the other electrode being applied to the outside of the chest wall; the fibrillar contraction was at once induced.

By the use of single induction shocks I have never seen the fibrillar contraction excited either when the shock is passed through the thoracic walls or when it is applied to the exposed heart. The single induction shock seems to be free from the dangers accompanying the use of the faradic current. Hence I have urged its superiority as a means of cardiac stimulation, in a paper to be read at the Ninth International Medical Congress at Washington.

The extreme readiness with which in certain circumstances the ventricles are thrown into the fibrillar contraction by any form of irritation, mechanical as well as electrical, renders it apparent that the experiment of puncturing the heart in order to destroy a certain part is attended with many difficulties. For very frequently the mere mechanical irritation would be amply sufficient to produce all the phenomena usually resulting from faradisation. And this condition of increased sensitiveness to irritation and increased tendency to assume the fibrillar mode of contraction appears to occur with special frequency and to a very marked degree in the heart of the dog.

V. In another class of cases the fibrillar contraction is induced by the more or less sudden action of certain influences of a depressing nature.

The injection of certain salts (e.g. bromide of potassium in strong solution) into the blood appears to induce the fibrillar condition in a very short space of time (frequently within one minute). A dose of about 0.1 gramme is sufficient in the hedgehog.

When such an injection is made (cat and hedgehog) there is almost immediately a marked change in the character of the systole. The origin and course of the contraction become very apparent both in the auricles and in the ventricles. In the former it passes forwards from the entrance of the great veins; in the latter it sweeps from the base of the heart towards the apex; on the front of the heart the contraction can be most distinctly seen beginning at the conus arteriosus and passing downwards. The ventricles become dilated with blood; the contractions are evidently unable to empty the cavities. When the heart is in a depressed state no further important change may be observed; the contractions gradually become weaker and slower until they cease altogether. But in the case of a vigorous heart there usually occurs a striking change—a short time after the injection of the bromide. The ventricles go into the state of fibrillar contraction with its usual features.

I have not as yet seen any complete recovery from the incoordinated condition produced in this way. The ventricles do not seem to recover their power of giving regular beats. Single contractions may occur after the rapid quivering movement has ceased but they appear to be fibrillar in their nature. And any contractions excited by single induction shocks in such circumstances appear to be of the same character.

After the injection of a solution of atropin I have observed somewhat similar phenomena; here however the fibrillar movement was arrested by the injection of pilocarpin, and complete recovery of the ventricular beat took place.

I have on some occasions observed phenomena of the same kind when an animal (cat) was suddenly and powerfully cooled by the application of a mixture of ice and salt to the surface of the skin and the insertion of an ice bag into the abdominal cavity. After the cooling had gone on for a time, the ventricles suddenly passed into the state of fibrillar contraction.

See and others have described the occurrence of a similar fibrillar movement in the dog's ventricles as one of the results of sudden occlusion of the coronary arteries.

VI. The arhythmic fibrillar contraction is fundamentally different from a rapid series of normal contractions. Its

genesis probably assumes in all cases one or other of two forms.

It is probable that the normally contracting ventricles possess within themselves certain co-ordinating arrangements in virtue of which the muscular contraction constituting a normal beat rapidly traverses the whole of the ventricular substances, causing a uniform or nearly uniform contraction of all the fibres of the ventricular walls thus leading to a concentric narrowing of the ventricular cavity and a consequent expulsion of its contents. The co-ordinating arrangements appear to exist in the lower portion of the ventricles as well as in the upper portion; for it has been seen that the apical part can execute co-ordinated beats when severed from the rest of the heart.

A normal co-ordinated contraction appears to be essentially different from the individual beats that may be seen after poisoning with bromide of potassium and occasionally in other conditions. In the latter case the contraction is obviously of a peristaltic nature; the contraction wave can be seen passing over the ventricular surface in definite directions. The contraction may be caused to start at any part in the ventricular substance by the application of a single direct stimulus; the contraction begins in the stimulated area and hence spreads over the rest of the ventricles; a phenomenon precisely similar to what one sees in the hearts of cold-blooded animals.

The peristaltic contraction evidently passes over the various interlacing bundles at different points of time, so that the whole thickness of the ventricular wall at any part is never uniformly contracted. Hence there is a wiry feel distinctly perceptible when the ventricles are held between the fingers as the peristaltic contraction is passing through its substance; certain fibres are hardened by the presence of contraction in them while neighbouring fibres are relaxed and soft. Such peristaltic contraction appears to be incapable of emptying the ventricular cavities of their contents; it appears to be essentially different from a co-ordinated beat however slow the latter may be. A co-ordinated beat never presents a wiry feel to the finger; it gives the sensation of a steady and uniform hardening of the muscle substance—of precisely the same nature as the hardening one feels in a skeletal muscle during its contraction. The contraction seems to involve as a whole the complicated interlacement of fibres forming the ventricular wall.

It appears then that the ventricles are capable of executing two forms of beat. One is the co-ordinated contraction seen in the normal

heart and capable of being excited by artificial stimulation (e.g. by single induction shocks) either in an intact heart, or in the fresh and vigorous excised ventricles or ventricle-apex. The other form of beat is the inco-ordinated or simple peristaltic contraction, such as may be seen after poisoning with bromide of potassium and in certain other conditions.

VII. The state of arhythmic fibrillar contraction (*delirium cordis* &c.) appears to be constituted by a rapid succession of inco-ordinated peristaltic contractions—a condition that can be brought about either (1) by the influence of certain depressing or paralysing agents upon the ventricular tissue, or (2) by the application of certain forms of stimulation to the ventricular tissue.

In the first class of cases the depressing influences alluded to probably throw out of gear the co-ordinating arrangements while they leave the muscular irritability intact—or it may be even augmented largely. Then the excitable (and probably highly rhythmic) muscle contracts, but its excitation instead of assuming the form of a normal beat becomes a peristaltic contraction wave along the complexly arranged and inter-communicating muscular bundles. And if the ventricular muscle is in an excitable state there would naturally occur a rapid series of such inco-ordinated peristaltic contractions. For apart from the possibility of rapid spontaneous discharges of energy by the muscular fibres, there seems to be another probable cause of continued and rapid movement. The peristaltic contraction travelling along such a structure as that of the ventricular wall must reach adjacent muscle bundles at different points of time, and since these bundles are connected with one another by anastomosing branches the contraction would naturally be propagated from one contracting fibre to another over which the contraction wave had already passed. Hence if the fibres are sufficiently excitable and ready to respond to contraction waves reaching them there would evidently be a more or less rapid series of contractions in each muscular bundle in consequence of the successive contraction waves reaching that bundle from different directions along its fibres of anastomosis with other bundles. Hence the movement would tend to go on until the excitability of the muscular tissue had been lowered, so that it failed to respond with a rapid series of contractions. Then there might be some isolated peristaltic contractions, such as I have often seen after the cessation of the fibrillar movement.

In the second class of cases—when the fibrillar contraction is excited by stimulation (e. g. faradisation of the surface of the ventricles) there appears to be a condition of violent excitement set up in the muscular tissue. The excitation of the muscular fibres travels peristaltically producing the characteristic movement; the inco-ordinated contraction of the various fibres may be most distinctly realised when the ventricles are held between the forefinger and thumb; there is a sort of wriggling sensation to be felt as the individual muscular bundles become hard and wiry while the contraction is passing over them in succession. The co-ordinating arrangements of the ventricles are powerless to regulate and guide the contractions; those co-ordinating arrangements are very possibly not paralysed nor rendered incapable of action, but they are temporarily superseded and rendered inoperative by the excessive state of excitement which pervades the muscular fibres—just as the cerebro-spinal co-ordinating mechanism might be rendered impotent by strong local stimulation of the skeletal muscles. When the fibrillar movement having become less rapid has at length stopped its duration depending on the excitability of the muscle—there ensues a pause.

Then there may be a recovery of the normal co-ordinated beat provided the fibrillar condition (and consequent blood stasis) has not lasted so long as to involve a paralysis or death of the co-ordinating mechanism.

When the last mentioned change has taken place, any beats that may occur are of the fibrillar character.

VIII. The phenomena resulting from faradic stimulation of the auricles differ in various respects from those seen in the ventricles.

The application of the current sets the auricles into a rapid flutter, the rapidity of which largely depends upon the excitability of the auricular tissue and the strength of current employed. The movements are regular; they seem to consist of a series of contractions originating in the stimulated area and thence spreading over the rest of the tissue. The movement does not show any distinct sign of inco-ordination; it looks like a rapid series of contraction waves passing over the auricular walls. The difference between this appearance and that seen in the ventricles probably depends on the simpler structure and arrangements obtaining in the auricles.

The persistence of the movement after the discontinuance of the stimulating current varies according to the excitability of the auricular tissue and strength of current employed. In very excitable conditions

the rapid movement lasts for a considerable time ; in depressed states the movement ceases almost immediately after the stimulation has ended. The persistence after the use of a strong current is, *cæteris paribus*, usually very much greater than when a weak current has been employed to excite the fluttering action.

IX. The movements excited by faradisation in the auricles and ventricles differ very markedly in their relation to the inhibitory influence of the vagus nerve. The fibrillar movement in the ventricles appears to be entirely unaffected by vagus stimulation; the fluttering movement of the auricles can be checked or arrested by the influence of the vagus.

Sometimes, when the auricles are very excitable the fluttering movement is entirely suspended during vagus stimulation only to reappear when the inhibitory influence has passed away. The vagus influence appears to act by weakening the individual contractions to the point of invisibility. At other times the contractions are markedly weakened without being rendered invisible. Often the movement is entirely arrested and does not recur; the normal action of the auricles goes on after the period of inhibition has passed.

The relation of the vagus nerve to the auricular muscle seems to be entirely different from the relation of that nerve to the ventricular muscle.