glacier in which unusually powerful forces may be supposed to be at work. Moseley's argument, however, seems to be decisive against the belief that the ordinary comparatively undisturbed descent of a glacier along a moderately sloping bed takes place by fracture and regelation. Moseley's value of the shearing strength of ice, which has been shown to be enormously too great as a measure of the resistance of ice to slow shearing, would appear on the other hand to be an inferior limit to the resistance to the shearing fracture which must precede regelation. Moseley has at any rate done good service by calling attention to the comparatively small intensity of the shearing force of gravity in the ordinary descent of a glacier. It would hardly have occurred to one fresh from the study of Forbes to look for evidence of the viscosity of ice in hand specimens exposed to moderate shearing forces in a laboratory.

II. "On the Structure and Rhythm of the Heart in Fishes, with especial reference to the Heart of the Eel." By J. A. McWilliam, M.D., Demonstrator of Physiology in University College, London. (From the Physiological Laboratory, University College, London.) Communicated by Professor Schäfer, F.R.S. Received January 14, 1885.

I. *On the General Arrangement and Structure of the Eel's Heart.*

The pulsation of the eel's heart can easily be seen externally on the ventral surface of the body a short way behind the pectoral fins. There are no rigid structures of any kind between the integument and the heart. When the very tough and resistant skin is cut through and the great lateral muscles are separated from each other, the pericardium is seen, loosely adherent to the surrounding tissues. The pericardial cavity being laid open, the various parts of the heart, abundantly lubricated with fluid, come into view. The organ is not freely suspended in the pericardial chamber, but is attached to the walls of that chamber by numerous and considerable bands, which vary in size and arrangement. The bands connected with the ventricle pass chiefly to the lateral and dorsal aspects of that part; they generally communicate with one another, forming a plexiform arrangement, and they tend materially to restrict the locomotion of the organ during systole. These bands are for the most part fibrous; they convey, however, large blood-vessels to the ventricle. These blood-vessels come from the dorsal part of the pericardial chamber and climb up in the fibrous bands on to the moving ventricle to

ramify on its surface. The partial fixation of the ventricle by the bands mentioned is probably in relation with this peculiar mode of blood-supply. There are generally two such vessels passing on to each side of the ventricle. These vessels are arterial; they are distributed to the *outer* part of the ventricular wall; they do not supply the whole thickness of the ventricular muscle. The greater part of the ventricular substance is spongy (like the ventricle of the frog) and is permeated by the venous blood passing through the heart; the outer part, however, is much more dense and compact in structure, and is supplied with arterial blood by the special system of vessels mentioned above. Veins are also seen upon the surface of the ventricle; they run upwards and backwards to terminate near the mitral orifice, opening there into the tubular vessel which connects the ventricle with the remaining parts of the heart.

The ventricle is also attached to the bulbus arteriosus, to the auricle, and to the sinus, by means of fibrous bands, which vary considerably in number and arrangement. A series of such connexions between the auricle and the ventricle exists around the mitral orifice.

The auricle is attached dorsally near the middle line by several short thick bands, which pass to it from the dorsal aspect of the pericardial chamber; there are also some slender threads, which pass to its ventral and lateral surfaces. The auricle is similarly connected to a slight extent to the sinus, and the sinus has some connexions with the lateral parts of the parietal pericardium.

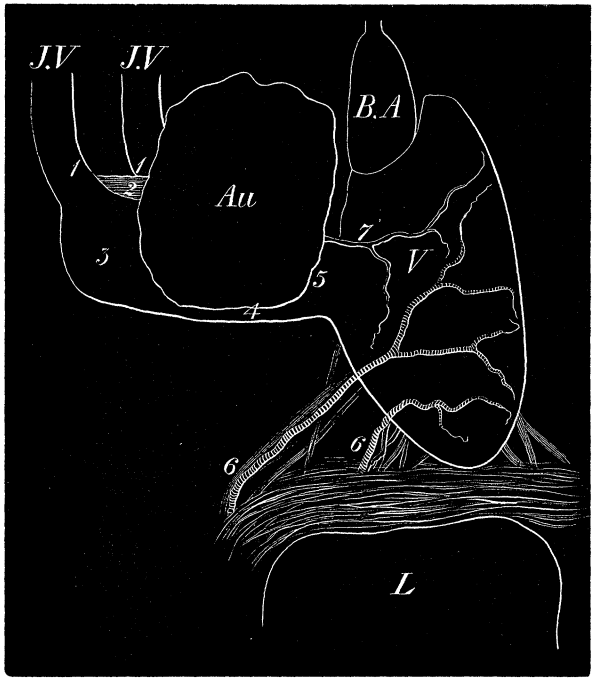
The sinus is placed dorsally, and is almost concealed by the overlying auricle. It receives, besides other vessels, the right and left jugular veins, which enter it from above.

Between the terminations of the two jugular veins, there is a part of the sinus marked off from the rest by a slight fold—best distinguished when the heart is well filled with blood. This part is physiologically distinct; it may be termed the "interjugular part of the sinus." It becomes continuous with the dorsal wall of the auricle.

The ventral wall of the sinus passes on to be directly attached to the ventricle at the mitral orifice. The proper auricular tissue does not form a complete chamber; it forms the lateral and dorsal parietes of a chamber, the floor of which is chiefly made up by the prolonged ventral wall of the sinus. (This prolonged ventral wall of the sinus corresponds to the "basal wall" described by Gaskell in the tortoise heart.) The auricle appears as a sort of appendage or diverticulum bulging laterally and dorsally from the prolonged ventral wall of the sinus. The auricle is not directly contiguous to the ventricle, but is separated from the latter by a short intervening tubular vessel, somewhat resembling the canalis auricularis of the mammalian fœtus.

This tubular vessel becomes continuous with the ventricle at its mitral orifice, which is placed a little below the middle of the dorsal aspect of the ventricle.

EEL'S HEART (diagrammatic).



J.V. Jugular Veins (right and left).
Au. Auricle.
V. Ventricle.
B.A. Bulbus Arteriosus.
L. Liver.
1. Ostial parts of Sinus.
2. Interjugular part of Sinus.
3. Main part of Sinus.
4. Basal wall of Auricular Chamber continuous with ventral wall of Sinus.
5. Tubular communication between Auricle and Ventricle (canalis auricularis).
6. 6. Arteries passing to Ventricle.
7. Veins on surface of Ventricle.

When the circulation is going on this disposition of the parts of the heart is rendered extremely obvious by the differences in colour presented by the various portions. The sinus is of a blue colour; the auricle is dark red; the ventricle is of a lighter red tint. When the ventricle is turned upwards, the blue ventral prolongation of the sinus

forming the floor of the auricular chamber is very apparent; and when the fibrous threads attaching the external surface of the auricle to that of the ventricle around the mitral orifice are divided, the blue intervening tubular vessel is plainly seen. The connexion of this vessel (canalis auricularis) with the ventricle is seen, under the microscope, to be chiefly established by connective tissue. There is, however, muscular continuity as well; for the ventricular substance and the muscular wall of the above-mentioned vessel (canalis auricularis) are connected by an extremely narrow and prolonged isthmus of muscular tissue.

II. *On the Spontaneous Rhythm of the Heart as a whole and of its Various Parts.*

The normal contraction of the heart begins by a distinct simultaneous beat in the right and left jugular veins, near their termination in the sinus. This spot may be denominated the "ostial part" of the sinus. Thence the contraction passes along the remaining part of the sinus to the auricle, and then to the ventricle. The mitral orifice being placed, not at the upper end of the ventricle, but a little above the middle of its dorsal aspect, the contraction of the ventricle begins at this part, and affects the middle portion of the ventricle before it passes over the upper and lower ends.

The normal order of contraction seen in the heart can easily be reversed by direct stimulation of the ventricle. Contraction then begins at the stimulated point and spreads over the whole heart; the ventricle first contracts, then the auricle, and lastly the sinus. This reversed mode of contraction usually ceases to be seen whenever the stimulation of the ventricle is discontinued; in some instances, however, it persists for a short time longer.

The passage of a constant current through the ventricle is sometimes able to cause that part to contract first, and so to lead to a reversal of the normal order of contraction of the parts of the heart.

During the continuance of the reversed mode of cardiac contraction, the circulation becomes much obstructed; the auricle and sinus are distended with blood. For the auricular beat occurs before the ventricular beat has ended, and the auricle is unable by its systole to force the blood into a contracted ventricle.

A regular contraction of the auricle leads very readily to a beat of the ventricle. A partial contraction affecting only the part of the auricle remote from the ventricle is not followed by a ventricular contraction; a partial contraction affecting the part of the auricle adjacent to the ventricle is usually followed by a ventricular beat, just as if a regular beat of the whole auricle had occurred.

But apart from the occurrence of a preceding auricular beat, a

ventricular contraction may occur in sequence to a sinus beat without the intervention of a contraction of the proper auricular tissue at all. This is evidenced by the state of matters observable in a mode of cardiac action which frequently presents itself as a result of certain conditions—especially nervous influences—to be afterwards described. The phase to which I refer is that in which contraction of the sinus, followed by contraction of the ventricle, occurs once or many times without any auricular contraction at all. Here the contraction is first seen in the sinus; after an appreciable pause it passes over the ventricle, the whole auricle meanwhile remaining perfectly motionless. Moreover, if the ventricle is made to contract first (*e.g.*, by direct stimulation), the contraction is after a short interval propagated to the sinus, the auricle being perfectly quiescent as before. And the presence of the proper auricular tissue is not at all necessary for the transmission of the beat between sinus and ventricle. For the whole of the auricle proper can be removed without interfering with the propagation of the contraction from the sinus to the ventricle—provided the direct anatomical connexion of the sinus and ventricle be left intact—that connexion which has been described as a prolongation of the ventral wall of the sinus to the ventricle (basal wall). In the absence of all the auricular tissue the ventricle continues to respond regularly to each beat of the sinus. That the ventricular beats here observed are really consequent on the preceding sinus beats—that the action of the ventricle is not an independent automatism, can be readily shown by detaching the ventricle from all connexion with the sinus tissue. When such is done the ventricle either stands still or goes on beating at a much slower rate than the sinus, and with a rhythm independent of that of the sinus.

It is obvious then that contraction may readily pass from sinus to ventricle, or from ventricle to sinus, without the intervention of any of the proper auricular tissue. And this condition seems to obtain not only in the heart of the eel but in that of many other fishes as well, *e.g.*, salmon, carp.

On the other hand, contraction can be readily propagated from the sinus to the ventricle, and *vice versâ*, through the auricular tissue, without the presence of the direct anatomical connexion (basal wall) between sinus and ventricle. For if this connexion be completely divided it will be found that the contraction can pass with the greatest regularity from the sinus over the intact auricle to the ventricle. And partial section of the auricle shows that a very slender strip of auricular tissue is sufficient to allow the transmission of the contraction to take place. A "blocking" of the contraction can easily be brought about by further section or by carefully applied pressure—just as Gaskell has found to be the case in the heart of the tortoise. And many of the conditions which Gaskell has described with refer-

ence to blocking in the tortoise heart seem to obtain in the eel's heart as well. If the means employed to bring about blocking have been kept within certain limits—if the section has not been carried too far or if the pressure applied has not been too great—the blocked condition usually passes off after a time, and the normal propagation of the contraction is again evident. The recovery of the tissue from the blocked condition can be materially accelerated by the application of the normal salt solution, and a similar beneficial effect is often apparent after the cardiac action has been arrested for a time by stimulation of the vagus nerve.

The normal sequence of the events constituting a cardiac beat is often in the course of prolonged experiments seen to become interrupted. The change which presents itself with the greatest frequency is a failure of the ventricle to respond to each beat of the other parts of the heart. Such failure often occurs without any loss of excitability in the ventricular tissue: the condition often seems to be one in which the propagation of the contraction from the auricle to the ventricle is interrupted. The ventricle then seems to remain quiescent, not because it is incapable of contracting, but because the contraction is not transmitted to it from the auricle and sinus. The failure of transmission seems to occur at the junction of the ventricle with the rest of the heart—at the mitral orifice. This fact is of interest when considered in relation with the peculiar character of the muscular connexion between the ventricle and the rest of the heart —a connexion which is established by means of an exceedingly narrow and prolonged isthmus of muscle substance. Whether or not this peculiarity in structural arrangement is the cause of the frequent failure of conduction at this part is a question on which it would be premature to make a decided statement.

A similar failure of the ventricular sequence can usually be brought about by repeatedly heating the whole heart or the sinus and auricle alone. Heat causes a great acceleration of the beat with a simultaneous enfeeblement. When the contractions of the auricle and sinus have thus been rendered extremely weak, a complete suspension of the ventricular action is commonly seen. When the auricular beats begin to recover their strength, the ventricle again begins to respond to each auricular beat, even though the auricular rate be still very rapid; the ventricle contracts in sequence to each auricular beat if the auricular beats are tolerably strong. The strength of the auricular beats seems to be a very important factor in regard to the question of the transmission of the contraction to the ventricle. And this statement is borne out by a number of facts—among others by a result which is often seen when the auricular beats are rendered excessively weak in consequence of their being elicited in rapid succession by artificial stimulation. In

such circumstances the ventricular action is often completely suspended until such time as the auricular contractions are slower and consequently stronger.

When failure of the ventricular sequence has occurred, the normal order of events can often be restored for a time—(1) by application of salt solution to the junction of the ventricle with the rest of the heart; (2) by passage of an interrupted current through the same part; or (3) by stimulation of the vagus nerve, leading to cardiac standstill, followed by a phase during which the normal ventricular sequence is restored. Whether this result of vagus stimulation is due to a direct beneficial effect on the tissues or to the rest which has been afforded to the various parts, is not at present clear.

Failure of the ventricular sequence seems to be at times associated with a depressed excitability of the ventricle itself.

All the parts of the heart when isolated from one another can manifest the property of automatic rhythmical action, though this property is possessed by the different portions of the organ in very different degrees. The excised ostial parts of the sinus go on beating at the ordinary rate of the heart's rhythm. The interjugular part when isolated exhibits (after a short pause) an independent rhythm which is slower than that of the ostial parts. The prolongation of the sinus to the ventricle comes next with regard to rhythmic power; then the auricle; and finally the ventricle. As regards the property of independent rhythmic contraction then, the various parts of the heart form a descending series, the highest term of which is the ostial part of the sinus; the lowest term is the ventricle.

The high rhythmic power possessed by the sinus constitutes it the leader in the series of events that make up the cardiac beat. Each contraction originating in the sinus of a normal intact heart spreads over the remaining parts of the organ, and thus leads to a rate of action in these parts identical with that in the sinus—a rate of action very much more rapid than they could exhibit in virtue of their own independent rhythmic power.

III. *Some Points in the Behaviour of the Heart with regard to the Results of Direct Stimulation.*

I shall here deal chiefly with the phenomena exhibited by the ventricle, reserving the consideration of the results obtained in the case of the auricle until the influence of the cardiac nerves has been discussed.

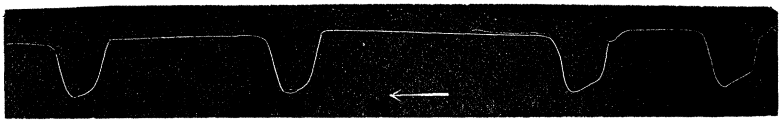
A single stimulation applied to the ventricle is followed by a single responsive beat. If the ventricle experimented on forms part of an intact heart, the result of a single excitation is to cause a reversed beat of the heart, the ventricle contracting first. And if the

circulation is going on, a peculiar dark bulging of the ventricular wall occurs at the stimulated part; this bulging occurs towards the latter part of the systole which results from the excitation. This phenomenon occurs with great constancy and in well-marked form in all cases where the circulation is intact and the ventricle is well filled with blood. It is not seen in the bloodless ventricle. The momentary or continued application of pressure may cause the same result, even though the pressure be too weak to act as a direct stimulus leading to contraction of the part where it is applied; the bulging may occur at the spot where pressure has been applied, even though the contraction of the heart occur in the normal fashion. The *rationale* of the phenomenon in question seems to be that in the area where a direct stimulus or even pressure insufficient to act as a direct stimulus has been applied, the muscular contraction is much impaired in efficacy, and as a result that area is unable to resist the high intra-ventricular pressure which occurs during systole. The area in question, therefore, becomes dilated with blood forced into it by the vigorous contraction of the rest of the ventricle; hence the dark bulging apparent on the surface. A somewhat similar bulging is occasionally to be seen in the ventricle of the frog as a result of strong direct stimulation.

The application of a strong direct stimulus (*e.g.*, an induction shock) to the ventricle is able to elicit a forced beat at almost any phase of the cardiac cycle. In a spontaneously-acting heart the occurrence of a forced beat from ventricular stimulation is usually followed by a prolonged diastolic interval before the appearance of the next spontaneous beat. In an intact and normally acting heart this diastolic prolongation observable on a ventricular tracing is not due directly to the properties of the ventricle; it is due to the conduct of the leading part of the heart—the sinus. For the rate of contraction of the ventricle of an intact heart is determined by the sinus—in which the contraction of the heart originates. The property of exhibiting a prolonged diastolic interval after a forced beat is not, however, peculiar to the sinus. It is equally well seen in the case of an isolated automatically-acting auricle or ventricle. A similar phenomenon has been described in the case of the entire frog-heart, and has there been attributed by some observers to an influence of the nervous mechanism.

The contraction which occurs after the prolonged diastolic interval is frequently of markedly increased size.

When the heart is intact and the circulation going on, the form of curve yielded by an artificially excited ventricular beat (resulting from direct stimulation of the apex of the ventricle) is quite distinct from that traced by a beat which occurs in normal sequence to an auricular beat; and the naked-eye characters of the beats obtained in the two instances are quite different.



Tracing shows difference in curves of normal and artificially-excited (reversed) ventricular beats. The two right-hand beats are spontaneous ones; the two left-hand beats were caused by direct stimulation of the ventricle with single induction shocks.

The muscular tissue of the isolated ventricle presents some characters that are similar to those of the frog's ventricle, and some that are markedly different.

I shall proceed to refer briefly to some points in the behaviour of the eel's ventricle with regard to

(1.) Single stimulations, electrical and mechanical.
(2.) Faradisation with strong and weak currents.
(3.) The constant current.

With regard to the effects of single stimulations on the quiescent ventricle, minimal stimulation is at the same time maximal—as in the frog's ventricle. Induction shocks obtained with one Daniell's cell in the primary circuit, when the secondary coil of the Du Bois Reymond's induction machine completely covers the primary coil, give the same strength of contraction as do shocks obtained when the secondary coil is 10 cm. removed from the first-mentioned position.

When the isolated quiescent ventricle is made to contract regularly by induction shocks at certain definite intervals (*e.g.*, 10 seconds) there is, as a rule, no progressive augmentation of the contraction force up to a maximum—no staircase of beats ("aufsteigende Treppe" of Bowditch), like that which is so conspicuously seen in the ventricle of the frog in similar circumstances. In the quiescent ventricle of the eel's heart the maximum beat is almost invariably obtained at once;

Tracing showing beats obtained from an isolated quiescent ventricle by stimulating it at intervals of 20 seconds with single induction shocks.

the beats elicited at regular intervals by a long consecutive series of shocks are almost always of exactly the same size, provided the interval between the successive shocks is sufficiently long to allow full recovery

of the muscular tissue from the effects of the preceding contraction. If the shocks follow each other too rapidly, the curves obtained assume the character of a descending series, resembling in the main a fatigue trace of an ordinary voluntary frog-muscle—at least as far as the progressive diminution in the size of the beats is concerned. When the ventricle has thus been reduced to a condition of fatigue, if the interval between the shocks be lengthened, the beats will for a time show a progressive increase in force, as more time is now allowed for the recovery of the tissue from the effects of contraction. Whenever the recovery from the fatigued condition is complete, the beats remain of fixed strength, showing no alteration whatever as long as the conditions under which the experiment is conducted remain constant.

Mechanical stimulation of the quiescent ventricle gives the same results.

Only in one ventricle (amongst a large number examined) have I seen any trace of the "beneficial effect of contraction." And in that case the ventricle had sustained considerable mechanical injury before the experiment was begun.

In order to test further the properties of the ventricular muscle with regard to the production of a staircase of beats in response to a regular series of stimulations, I have many times repeated the experiment on a quiescent ventricle in the uninjured state filled with normal blood. For this purpose the ventricle of a normally acting heart was rendered quiescent by stimulation of the vagus nerve—a proceeding which, by arresting the action of the other parts of the heart, leads to ventricular standstill without (as will be explained in the following pages) influencing in any way its contraction force. The standstill of the ventricle occurs simply as a result of the quiescence of the other parts of the heart from which the rhythmical contractions are normally propagated to the ventricle. An uninjured ventricle that has been rendered quiescent in this way is presumably in a more normal state than an isolated ventricle artificially fed outside the body.

Such a ventricle, excited at regular intervals by a series of mechanical or electrical stimulations, gives a series of beats of precisely equal strength, the commencing beats of the series being maximal, and differing in no respect from the subsequent ones.

With regard to the effects of faradisation upon the ventricular tissue, some remarkable results are obtained.

A surprising influence is found to be exerted by the *rate of interruption* in the faradising current.

It is well known that in the case of ordinary voluntary muscle when a slowly interrupted current is applied, the muscle contracts at the rate at which the shocks are sent in. And when a more rapidly interrupted current is employed, the muscle exhibits a more rapid

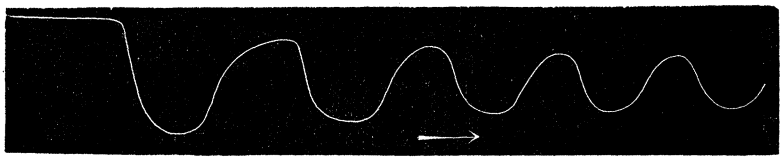
succession of contractions—in response to the more rapid series of shocks sent into it. In short, the rate of contraction depends on the rate at which the stimulations are applied; the quicker the series of shocks sent in, the quicker is the series of responsive contractions.

In the eel's ventricle the phenomena are of a strikingly different nature. For a slowly interrupted current is found to cause a much more rapid series of contractions than does a rapidly interrupted current of precisely equal strength. When a rapidly interrupted current is sent through the quiescent ventricle (of an eel's heart), the rate of contraction induced varies within certain limits according to the strength of the current employed. When a rapidly interrupted current (*e.g.*, sixty shocks per second) is used of such a strength as to cause a *slow* series of ventricular beats, it is found that a diminution in the rate of interruptions (*e.g.*, to six per second), at once leads to the appearance of a much *more rapid* series of ventricular beats. The number of contractions resulting from the application of the slowly interrupted current is much greater than that caused by a rapidly interrupted current; the influence, moreover, of the former is much more lasting than the influence of the latter. A much closer approach to a tetanic condition is induced by a slowly interrupted current than by a rapidly interrupted one. And this statement holds with regard to (1) the ventricle of an intact heart; (2) the isolated quiescent ventricle; and (3) the isolated automatically-contracting ventricle. It holds also with regard to various rates of interruption in the stimulating currents. A current interrupted ten times a second is much more effective than one interrupted fifty times a second. The rule holds, then, that—within certain limits—a slowly interrupted current, whether galvanic or faradic, produces much more striking effects than a rapidly interrupted one, the influence of the latter being much less powerful, and, at the same time, of a less enduring character. It is not necessary when dealing with a slowly interrupted current that the shocks be sent in at any particular phases of the cardiac beat; a series of shocks is simply sent in without regard to the state of the organ at the moment when each shock is sent in.

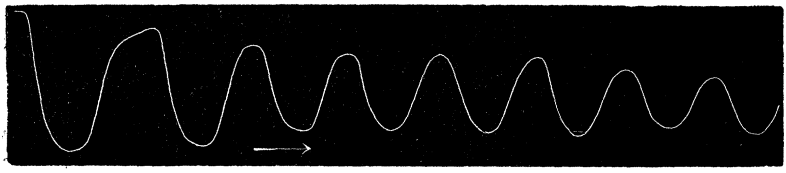
A very striking difference is also evident in the results obtained by the continued application of a weak interrupted current to the ventricle as compared with the *repeated temporary* application of the same current. The latter is commonly able to induce a fairly rapid series of ventricular contractions when the former has no apparent effect at all or merely causes a *very* slow succession of beats.

These remarkable differences in the effects of interrupted currents are manifested not only in the case of the ventricle, but in the auricle and sinus as well—in all the portions of the cardiac muscle.

As a result of the application of *weak* interrupted currents, I have observed no marked effect upon the ventricle of the eel's heart. There



Tracing shows the contractions excited in an isolated quiescent ventricle by stimulating it with a rapidly interrupted current (50 interruptions per second).



Tracing shows the result of stimulating the same ventricle with a slowly interrupted current (5 interruptions per second) of precisely the same strength.

is no evidence whatever of its having the slightest effect in weakening the contractions—a result which has been shown to follow the application of a weak interrupted current to certain cardiac tissues, *e.g.*, the auricle of the tortoise and the ventricle of the frog.

In the case of the intact heart with the normal circulation going on, the passage of a constant current through the apex of the ventricle has been seen to be accompanied by a reversed order of contraction of the parts of the heart, the apex of the ventricle—through which the current is passing—being the portion which initiates the systole of the whole organ.

IV. *Action of the Cardiac Nerves.*

All the nerve impulses affecting the heart appear to pass along the vagus nerve trunks.

Stimulation of the peripheral end of a cut vagus nerve exerts upon the heart an inhibitory influence of a very powerful nature. The inhibitory phase is often followed by a phase during which the heart's rhythm is accelerated. Such an accelerating *after* effect of vagus stimulation is, however, slight in degree and variable in occurrence.

Stimulation of either the right or the left vagus is effective.

When continued stimulation of one vagus fails at length to keep the heart quiescent any longer, stimulation of the other vagus usually causes a further inhibition of the cardiac action.

The latent period of vagus stimulation appears to be short.

The vagus has a very low minimal stimulation; a *very* weak interrupted current such as is insufficient (when applied to the peripheral

end of a cut vagus nerve) to cause contraction of the œsophagus, leads readily to cardiac standstill.

Single induction shocks to the vagus nerve have no distinct effect on the heart's action unless the shocks are very powerful.

When arrested by vagus stimulation, the heart stands still in a state of diastole, and—when the circulation is going on—the whole heart, but more especially the auricle and sinus, become greatly distended with blood, if the inhibition be of any considerable duration. The great veins also become much gorged; they are very capacious, whilst the presence of dilatations upon the jugular veins (jugular sinuses) allows of the accommodation of a large amount of blood.

The effects of cardiac standstill on the circulation can readily be observed by fixing the transparent part of the animal's tail under a microscope, and then inducing inhibition of the heart by stimulation of the vagus nerve, or simply by pressing on the gill—a proceeding which causes reflex cardiac arrest. When the heart stands still, there is a very gradual slowing of the blood-current which at length comes to a state of complete stagnation; this result is not arrived at until the cardiac action has been completely stopped for a period of from one to two minutes. When inhibition has passed away, and the heart's action has recommenced, the blood-flow is very speedily restored; the change is a much more rapid and abrupt one than the change observed when the heart was brought to a standstill; the first recommencing beat causes a distinct movement of blood in the capillaries of the tail.

High intra-ventricular systolic pressure and distension of the heart with blood (caused by clamping the branchial artery) do not obviate the inhibitory effects of vagus stimulation.

The eel's heart presents some striking peculiarities as regards its mode of recommencing action after it has been arrested through the medium of the inhibitory nerves.

It will be remembered that in the case of the frog, toad, rabbit, dog, when the heart's action goes on after an inhibitory standstill, the various parts of the heart recommence action in the fashion in which they normally beat. Thus in the frog, when the cardiac action recommences after a period of inhibition, the heart beats present their usual characters as regards the order of succession; the contraction first affects the sinus, and then passes over the auricles and ventricle successively. In the eel the mode of recommencing is, after *slight* inhibition, similar as a rule to that seen in the other animals mentioned,—the contraction begins in the sinus (ostial part) and passes over the rest of the heart in the normal fashion. But after *profound* inhibition the renewal of cardiac action presents features of an entirely different and very peculiar character. For

when the heart recommences after a prolonged standstill, the order of contraction of the different part is, as a rule, markedly changed, and moreover, the contraction is for the first few beats restricted to certain parts of the organ. The remaining parts of the heart remain quiescent for a time, and then come to participate in its activity; the order of contraction generally remains modified, and it is only after a considerable though variable period that the ordinary succession of events again obtains in the contracting heart.

The part in which spontaneous contractile activity usually reappears after profound inhibition is not the ostial part of the sinus (which ordinarily leads the rhythm of the heart), but the interjugular part of the sinus, which ordinarily contracts second in the series, *i.e.*, in succession to the leading ostial part. The recommencing contraction is commonly—at least after powerful inhibition—confined for one or more beats to the interjugular parts; it often extends, however, at the first beat from the interjugular part to the ventricle. The beating of the interjugular part, soon at least accompanied by responsive ventricle beats, goes on for a short though variable time without the slightest movement being perceptible in the auricle or in the ostial part of the sinus. After a time the contraction originating in the interjugular part of the sinus spreads over the ostial part of the sinus, and soon afterwards over the auricle as well as over the ventricle, so that the whole heart is now in action, though the order of contraction of its various parts is not identical with that normally present. For instead of the systole being initiated by the ostial beat, it is now initiated by the interjugular beat. Soon, however, the ostial beat regains the precedence and the normal order of events is restored.

These phenomena seem to depend (1) on the unequal influence exerted by vagal stimulation on the different parts of the heart; (2) on the high inherent rhythmic power of the interjugular part; and (3) on the existence of the anatomical and physiological connexion between the sinus and ventricle already mentioned. The ventricle is not directly affected at all by vagus stimulation; its excitability and contraction force remain quite unimpaired. The interjugular part and the path between it and the ventricle are less profoundly affected than are the auricle and ostial parts; the former parts recover more readily from the inhibitory influence than do the latter. Early released from inhibitory control, the interjugular part, in virtue of its high rhythmic power, begins to beat, and the contraction is soon propagated to the excitable ventricle. Hence the appearance of the peculiar form of recommencement seen.

In various fishes besides the eel (*e.g.*, carp) I have observed a somewhat similar mode of recommencing action after inhibition. The sinus and ventricle usually contract once or several times before

any movement was perceptible in the auricle. I have not seen a distinct division of the sinus into ostial and interjugular parts in any fish except the eel.

During the inhibitory standstill the condition of the various parts of the heart, as evidenced by the results of direct stimulation, is strikingly different from that observed in the hearts of those animals in which cardiac inhibition has been chiefly studied.

In the frog-heart, for example, a single direct excitation applied to the heart during the inhibited phase produces a single beat; the contraction begins at the stimulated point and extends over the whole organ. And this result is obtained whether auricle or ventricle is stimulated. In the eel's heart the state of matters is more complex. The ventricle resembles the frog-heart in giving a single beat in response to a single excitation; its irritability and contraction force seem to be quite unaffected. The interjugular part, except when the heart is most powerfully inhibited, remains excitable to direct stimulation. And contraction excited in the interjugular part is usually propagated to the ventricle, whilst the auricle remains perfectly quiescent; contraction excited in the ventricle is commonly propagated in a similar fashion to the interjugular part. The ostial part of the sinus is, during strong inhibition, quite inexcitable to direct stimuli of all kinds, and so is the auricle. The result of nerve stimulation has evidently in this instance been the peculiar one of temporarily abolishing the irritability of the muscular tissue. Such a result is quite incompatible with the old conception of vagal action, viz., that the vagus inhibited the motor discharges from the cardiac ganglia, and so brought the muscular tissue into a state of quiescence. If such were the case the muscle ought of course to respond readily to direct stimulation. And this the auricular muscle signally fails to do.

It has been mentioned that during the inhibited state direct stimulation of the ventricle readily causes contraction of that part; the contraction originates at the stimulated point and spreads over the rest of the ventricle. It does not pass over the auricle. The non-participation of the auricle in the contraction cannot be explained by the assumption of a block between the auricle and ventricle preventing the contraction being propagated. For if such were the case, if the auricle remained quiescent merely because the contraction failed to reach it, the auricular muscle ought to respond readily to direct stimulation. But it has been shown that the auricular muscle is quite inexcitable, and that it is in such a state as to be quite incapable of responding to any contraction impulses that might reach it.

Effects of Vagal Stimulation on the Properties of the Muscular Tissue of the different Parts of the Heart.

In the investigation of these effects the cardiac action was registered by means of a simple arrangement of the graphic method. Simultaneous tracings of the auricular and ventricular action were obtained by means of two writing levers suspended in the horizontal position by slender elastic bands; these levers were brought into connexion with the auricle and ventricle respectively by threads. In order to prevent locomotion of the heart it was sometimes necessary to afford a fixed point by holding in a clamp the bands passing into the dorsal aspect of the auricle.

At times a simultaneous record of the contractions of the auricle and ventricle was obtained by a single writing lever: (1) by attaching the threads from the auricle and ventricle to the same lever, or (2) by connecting the lever by the auricle alone (by means of a thread), and then firmly fixing the apex of the ventricle. The auricular and ventricular beats are then recorded on a single tracing.

I shall take each part of the heart in succession, and briefly consider the effects observed in each.

(1.) *Effects of Vagus Stimulation on the Ventricular Muscle.*

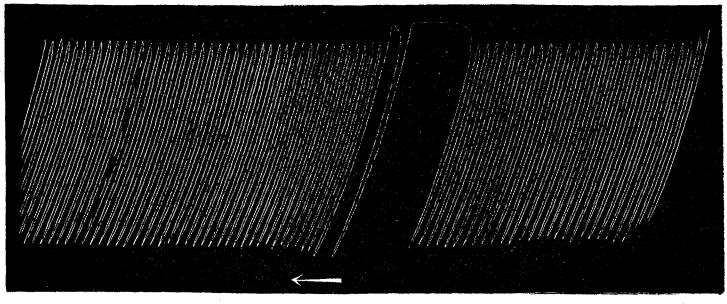
A large number of experiments have clearly shown that the ventricle is not directly affected by stimulation of the vagus nerve.

As has been already mentioned, the automatic rhythmic power of the ventricle is low, and the ordinary rate of action of this part in the intact heart results not from the spontaneous rhythm of the ventricle itself, but in consequence of the rapid action of the sinus and auricle, from which parts the contraction is propagated to the ventricle. The ventricular contraction is started off by the other parts of the heart; its inherent rhythmic power remains latent. It is obvious then that an arrest of the action of the other parts of the heart would at once lead to a suspension of the ventricular activity. The ventricle, deprived of the impulses which ordinarily start off its contraction, must necessarily stand still until such time as the starting off impulses come again into play, or until its latent automatic rhythm begins to manifest itself—an event which, in ordinary circumstances, would not occur for a very considerable length of time. So that with regard to the suspension of the ventricular rhythm caused by vagus stimulation, the removal of the normally efficient cause of that rhythm is the reason of the ventricular standstill, *i.e.*, the ventricle stands still because the other parts of the heart have stopped action.

The excitability of the ventricular muscle during inhibition of the

heart seems (from a comparison of the results obtained by direct stimulation in the normal and inhibited states) to be unchanged. A contraction can be elicited by a direct stimulus applied during the inhibitory standstill quite as readily as in the normal condition of the heart.

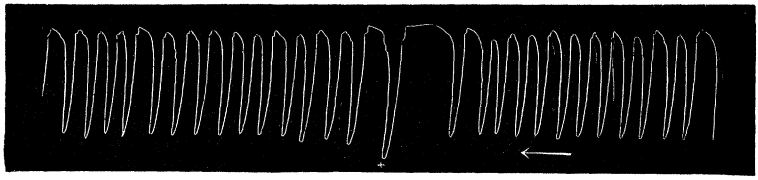
Upon the contraction force of the ventricular muscle, stimulation of the vagus nerve seems to be entirely without effect.



Ventricle tracing showing inhibition resulting from stimulation of vagus nerve. The recommencing beats are seen to be large.

When a heart which is beating at a fairly rapid rate is slowed by very gentle vagal stimulation, the ventricular beats are larger proportionately to the slowing—up to a certain maximum. The increase in the force of the beats is dependent on the slowing; it occurs when the heart is slowed by means other than by vagal influence, *e.g.*, by the application of cold to the sinus. It is the converse of what occurs during rapid action of the heart, such as may result from the application of heat to the sinus, or may follow vagal inhibition as an after effect. During rapid action of the heart the ventricular beats become smaller in proportion to the increased rates, until at length, if the acceleration is excessive, the recording lever writes a mere wavy line on the smoked paper.

When the heart is arrested by vagus stimulation, and the ventricle is made to contract by a direct stimulus, the force of the beat so elicited is, even during the most powerful inhibition, quite as large as the ordinary beats occurring spontaneously before the standstill. Indeed an artificial beat elicited during inhibition is generally larger than the spontaneous beats occurring before inhibition, just as the beats occurring during a very slow cardiac rhythm are larger than those seen during a quicker rhythm. A rapid artificial rhythm induced in the ventricle during inhibition of the heart shows a diminution in the size of the rapidly recurring beats, corresponding to what is seen in a rapid natural rhythm.



Ventricular tracing showing an inhibitory standstill—a result of vagus stimulation. The beat marked + was elicited by direct stimulation of the ventricle during the period of inhibition.

(2.) *Effects of Vagal Stimulation on the Auricle.*

The auricle, though possessing an inherent rhythmic power of its own, has this independent rhythm masked and rendered latent (like the lower rhythmic power of the ventricle) by the more rapid rhythmical lead of the sinus. The auricle then is, in consequence of the rapid action of the sinus, made to beat at a considerably quicker rate than it would otherwise do in virtue of its own inherent rhythm.

When the vagus is strongly stimulated, and the whole heart brought to a standstill, the auricle becomes relaxed, and a marked diminution of its ordinary tone is evident. It stands still not merely in the way the ventricle does because of the arrest of the dominant part which normally starts off its contraction, but because of a profound influence directly exerted on its muscular tissue. Its inherent rhythmic power is markedly depressed, for vagal stimulation can keep the auricle in a motionless state much longer than would be required for the exhibition of its independent rhythmic power. If the auricle merely stopped in consequence of the stoppage of the sinus, and if the inherent auricular rhythmic power was intact, an independent action would very soon be exhibited by the auricular tissue. It is obvious that this inherent automatic rhythmic property is held in abeyance by vagal stimulation.

Moreover, there occurs in the auricle a striking depression of the excitability of the muscular tissue as tested by direct stimulation. The auricular excitability is, during strong inhibition, annulled for the time being. Such a loss of excitability does not depend on over-distension of the auricle with blood during the cardiac standstill; it occurs in the bloodless auricle as well. As the inhibitory phase is passing away direct stimulation of the auricle leads to localised contraction in the stimulated area. These contractions seem to be localised in consequence of the great depression of excitability which still pervades the auricular tissue, and prevents the contraction from spreading over the whole auricle as it would do in the normal state. Very weak stimulation of the vagus nerve causes a distinct depres-

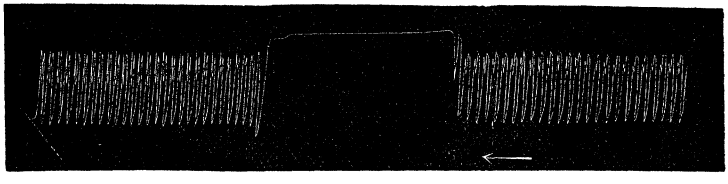
sion of the auricular excitability without any absolute suspension of that excitability. Very weak vagal stimulation is often able to cause the auricle to beat more slowly than the rest of the heart, or to stop action for a time while the other parts of the organ go on beating.

At high temperatures (29—33° C., &c.) the auricular excitability to direct stimulation does not seem to be depressed during cardiac standstill brought about by vagus stimulation.

Tracing of auricle showing vagal inhibition. The beats marked + were elicited by direct stimulation (single induction shocks) applied to the auricle before its action had recommenced. It will be seen that the auricular beats continue to be of diminished size for some time after the spontaneous action has recommenced.

Vagal stimulation is also able to depress very markedly the contraction force of the auricular muscle. Any beats that can be elicited by strong direct stimulation (when the inhibitory phase is passing away) are much diminished in size, and the beats with which the auricular action recommences are as a rule very small ones; they gradually increase till they regain the normal size. The depressing effect on the contraction force accompanying vagal inhibition occurs both in the intact heart and in the bloodless auricle; it is not due to over-distension during the inhibitory period.

The conduction power of the auricle is, during inhibition, temporarily abolished.



Tracing showing the contractions of both auricle and ventricle. The large beats are those of the ventricle; the smaller ones between the large beats are those of the auricle. The effects of vagal inhibition are shown. The recommencing auricular beats are much diminished in size; the recommencing ventricular beats are large.

These primary depressing effects of vagal stimulation on the auricular tissue are by far the most constant and powerful ones; the secondary accelerating effects are slight and variable. The increase

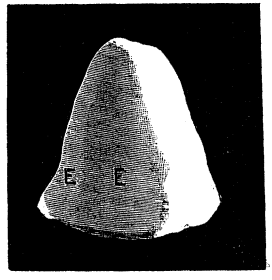
in the rate of the auricular action, which is at times seen as a secondary effect of vagal stimulation, does not depend on the auricle itself, the inherent auricular rhythm being latent; the acceleration depends on the increased rate of action of the sinus which leads the cardiac beat.

I shall here mention some results obtained by the application of electricity to the auricular tissue.

Single induction shocks (as well as mechanical or thermal stimuli) applied to the normal auricle during its diastole cause a single contraction, accompanied as in the ventricle by a bulging of the auricular wall at the point of stimulation.

The passage of a weak interrupted current produces some peculiar effects in the auricle. For when an interrupted current of suitable strength is sent through any part of the auricle, that part immediately stands still, becoming distended with blood, while the rest of the auricle goes on beating as before. By gradually increasing the strength of the current a wider and wider area can be inhibited, until at length the whole auricle becomes arrested, while the sinus and ventricle may still go on beating. Any escape of the current to the sinus will, however, cause a stoppage of the whole heart.

When local inhibition is brought about in the auricle in the way mentioned, there is a marked change in the excitability of the inhibited area, as well as a suspension of its rhythmic activity. Its excitability to direct stimulation is greatly depressed or temporarily annulled; the application of a direct stimulus commonly fails to induce any contraction. The inhibited tissue seems to be in a state similar to that into which the whole auricle is thrown by stimulation of the vagus nerve. The action of the inhibited area is not usually resumed for some little time after the discontinuing of the inhibitory current.



When a projecting corner of the auricle is dealt with, and the electrodes are applied at the points indicated by the letters E and E, only part of the projecting piece of auricle is, as a rule, thrown out of action. The shaded part becomes arrested, while the remainder goes on contracting in unison with the rest of the auricle.

Poisoning with curare (a minimal dose) prevents the occurrence of local inhibition in the auricle as a result of the passage of a weak interrupted current. And when an isolated piece of auricle exhibiting an *independent* rhythm is subjected to the influence of a weak interrupted current, its behaviour differs markedly from that of the same piece of tissue whilst it formed part of an intact auricle, for in the case of an *automatically contracting* isolated piece of auricle the application of a weak interrupted current produces *no* inhibitory effect. When the current is strengthened a depression of the contraction *force* is induced, but the rhythm remains unchecked, unless, perhaps, by *very strong* currents. The rhythm may even be accelerated, and when *very strong* currents cause an apparent standstill, it is doubtful whether this standstill is due to a true inhibition of rhythm.

(3.) *Effects of Vagus Stimulation on the Sinus.*

A. *On the Interjugular Part.*

During the inhibitory standstill of the heart the interjugular part is directly affected by nerve influence; it does not merely stop action because the leading parts have stopped. During vagus stimulation there is a marked depression of the inherent rhythmic tendency. The interjugular part can, by vagus stimulation, be kept quiescent for a very much longer time than would be required for the manifestation of the independent rhythm of the part; there is evidently an inhibition of the inherent rhythmic property. This depression of rhythmic power seems to pass off from the interjugular part earlier than it does from the neighbouring ostial parts.

As a result of vagal stimulation the excitability of the interjugular part (as well as its inherent rhythmic power) is depressed. During moderate excitation of the vagus nerve the interjugular part responds less readily to a direct stimulus, and during powerful inhibition the interjugular part fails to contract at all on the application of a direct excitation. Moreover, when recommencing action begins after a prolonged inhibitory standstill, the contraction is often seen to begin at the dorsal aspect of the interjugular part, and to be for a time limited to a small area of the tissue; at each succeeding beat the contraction extends, and soon involves the whole interjugular part. Such facts indicate the occurrence during inhibition of a marked depression of the excitability of the part.

As a secondary effect of vagus stimulation, there is sometimes a distinct increase in the rhythmic power, and probably also in the excitability of the interjugular part.

The passage of an interrupted current through the interjugular part very readily brings about an arrest of the whole heart. A very weak current is sufficient for this purpose; frequently a current too

weak to be perceived on the tongue causes prolonged cardiac stand-still.

The passage of a constant current through the interjugular part appears to cause an acceleration in the rhythm.

B. *Effects of Vagus Stimulation on the Ostial Parts of the Sinus.*

When either the right or left vagus nerve is stimulated the rhythmic power of both the right and left ostial parts is completely suspended for a time. The excitability to direct stimulation also is temporarily annulled; no contraction is caused by the application of even a powerful direct excitation.

As a secondary effect of vagus stimulation there often occurs a marked heightening in the rhythmic power of the ostial parts, and a consequent acceleration of the rate of the heart's action. Probably there is also in some instances an increase in the excitability of the ostial parts.

Weak or strong interrupted currents applied to the ostial part cause an immediate arrest of the heart's action, with the usual features of the inhibited state. Such a result is obviated by the administration of curare.

III. "On the Structure and Development of the Skull in the Mammalia. Part III. Insectivora." By W. K. PARKER, F.R.S. Received January 15, 1885.

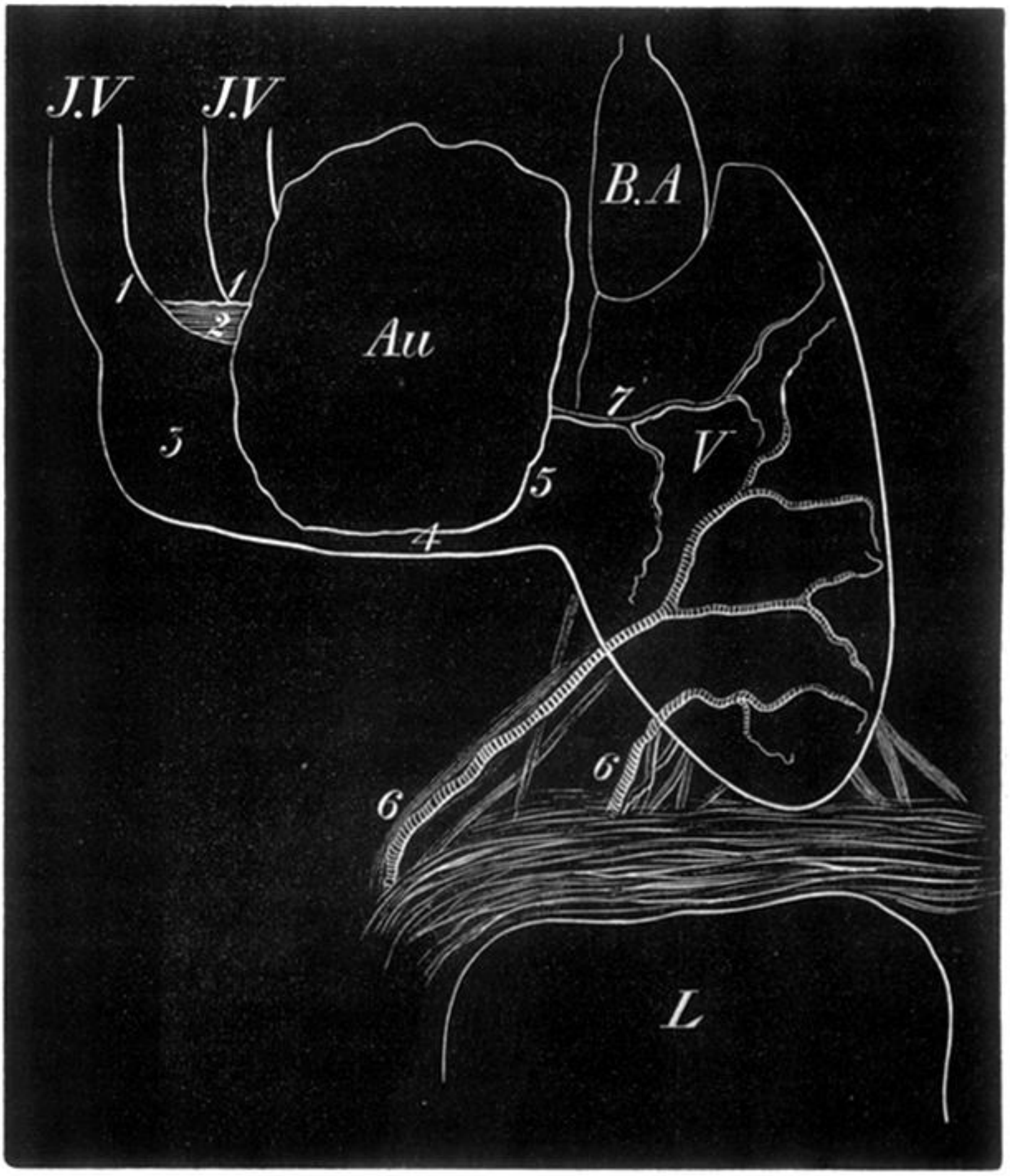
(Abstract.)

Although this paper is confessedly only a fraction of what is necessary to be done in this polymorphic order, it shows at least how difficult a group it is to handle. For the Insectivora are set in the midst of the other Mammalia—low and high. They might be called the Biological stepping-stones from the Metatheria to the Eutheria.

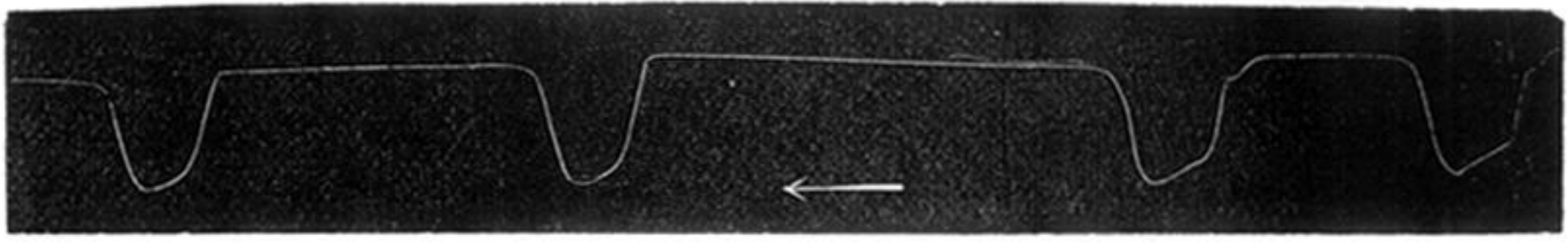
One thing can be done, even now, with our present fragmentary knowledge of the structure and development of the Insectivorous types —we can assure ourselves that these types are immediately above the Marsupials, that they have the Bats (Chiroptera) obliquely above them, that their nearest relations must be sought for amongst extinct Eocene forms, and that, lowly as they are, and arrested and often dwarfed to the uttermost (so that nature could not safely go further in that direction), they are rich in prophetic characters that have come to perfection in larger and nobler types.

I think it will not be denied that in the ascent of the types the Chiroptera are above the Insectivora, and, as it were, a sort of special

Eel's Heart (diagrammatic).

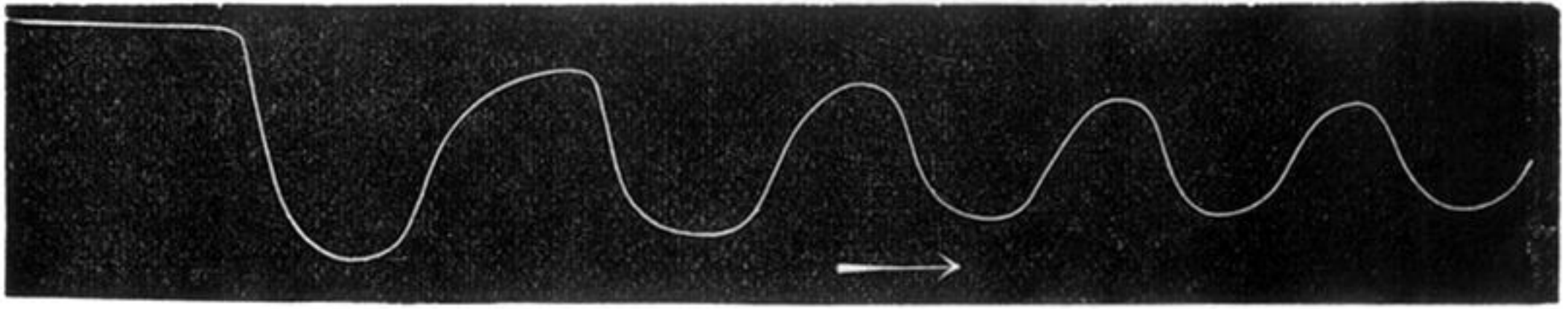


J.V. Jugular Veins (right and left).
Au. Auricle.
V. Ventricle.
B.A. Bulbus Arteriosus.
L. Liver.
1. Ostial parts of Sinus.
2. Interjugular part of Sinus.
3. Main part of Sinus.
4. Basal wall of Auricular Chamber continuous with ventral wall of Sinus.
5. Tubular communication between Auricle and Ventricle (canalis auricularis).
6. 6. Arteries passing to Ventricle.
7. Veins on surface of Ventricle.

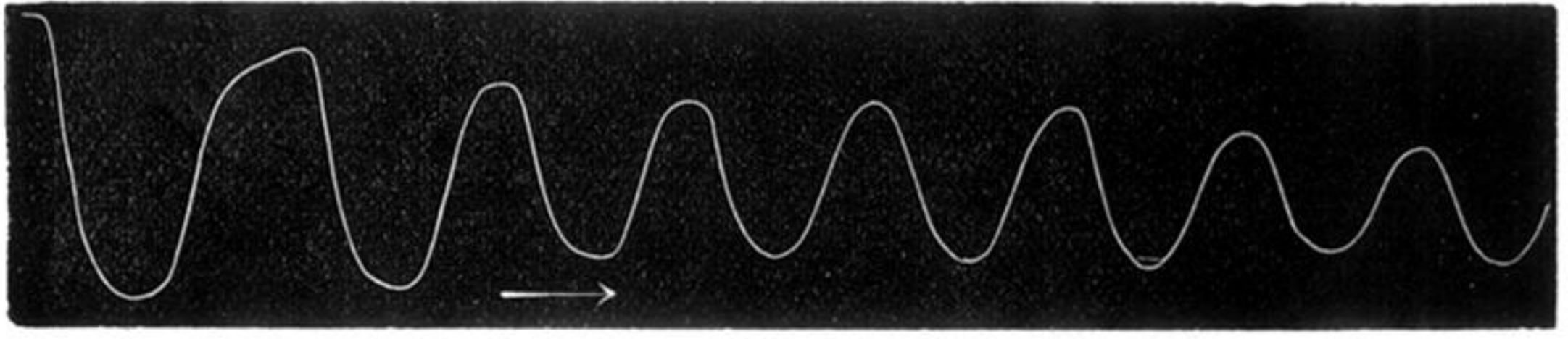


Tracing shows difference in curves of normal and artificially-excited (reversed) ventricular beats. The two right-hand beats are spontaneous ones; the two left-hand beats were caused by direct stimulation of the ventricle with single induction shocks.

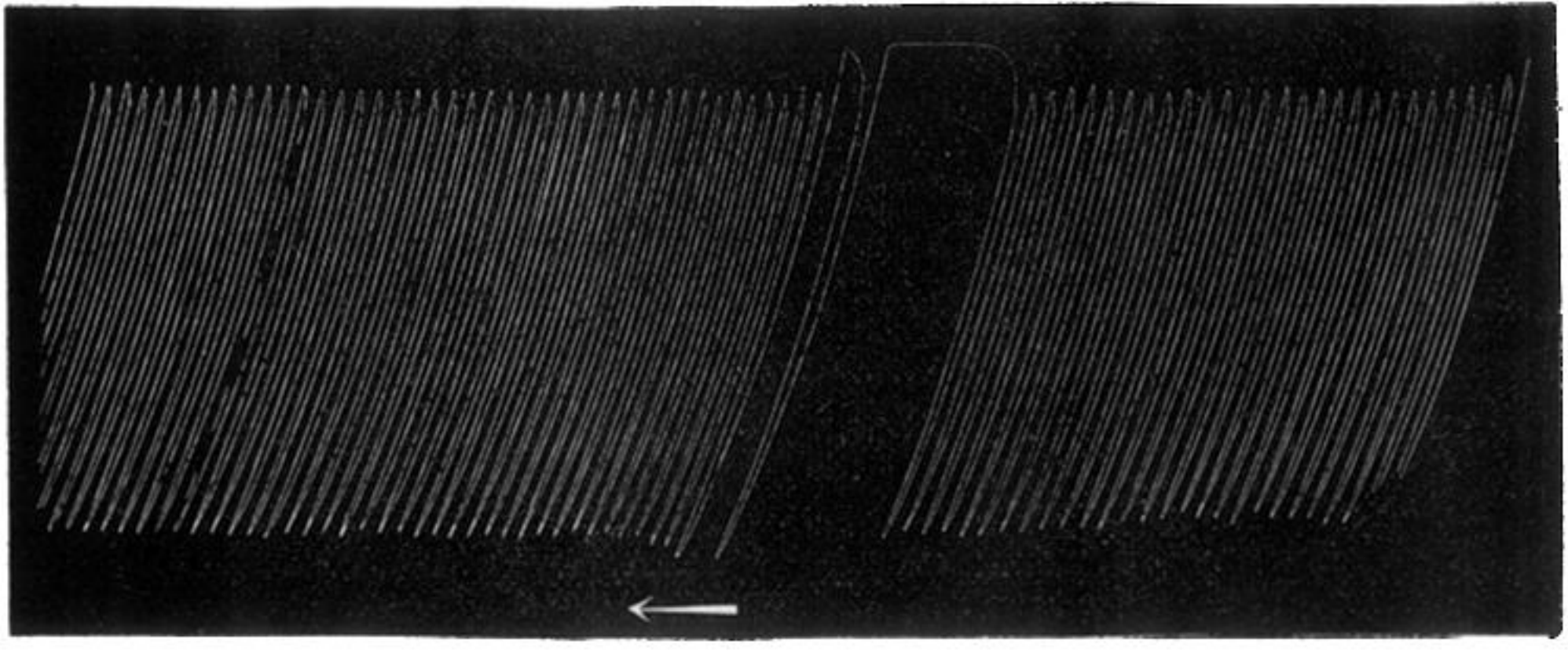
Tracing showing beats obtained from an isolated quiescent ventricle by stimulating it at intervals of 20 seconds with single induction shocks.



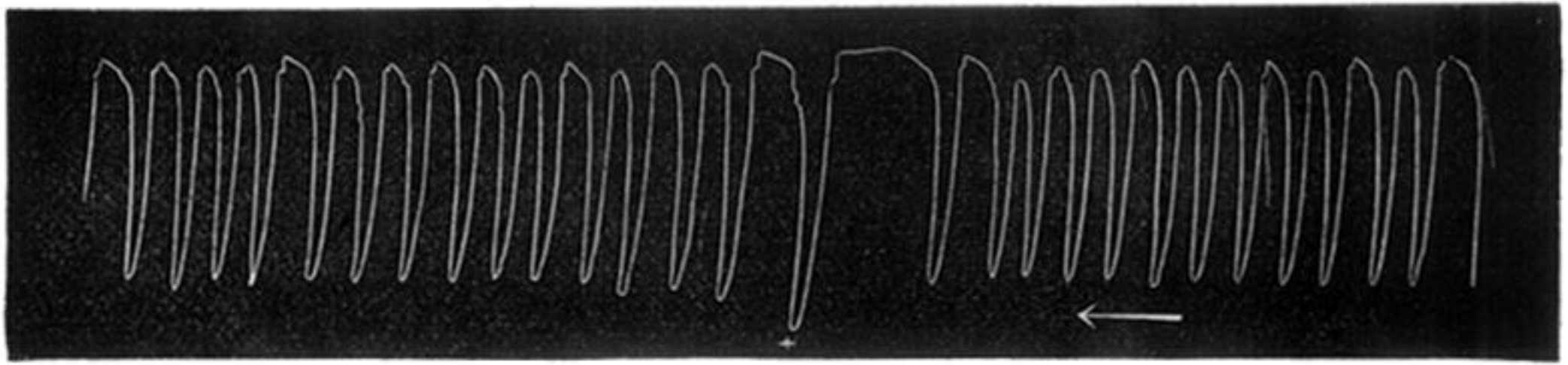
Tracing shows the contractions excited in an isolated quiescent ventricle by stimulating it with a rapidly interrupted current (50 interruptions per second).



Tracing shows the result of stimulating the same ventricle with a slowly interrupted current (5 interruptions per second) of precisely the same strength.

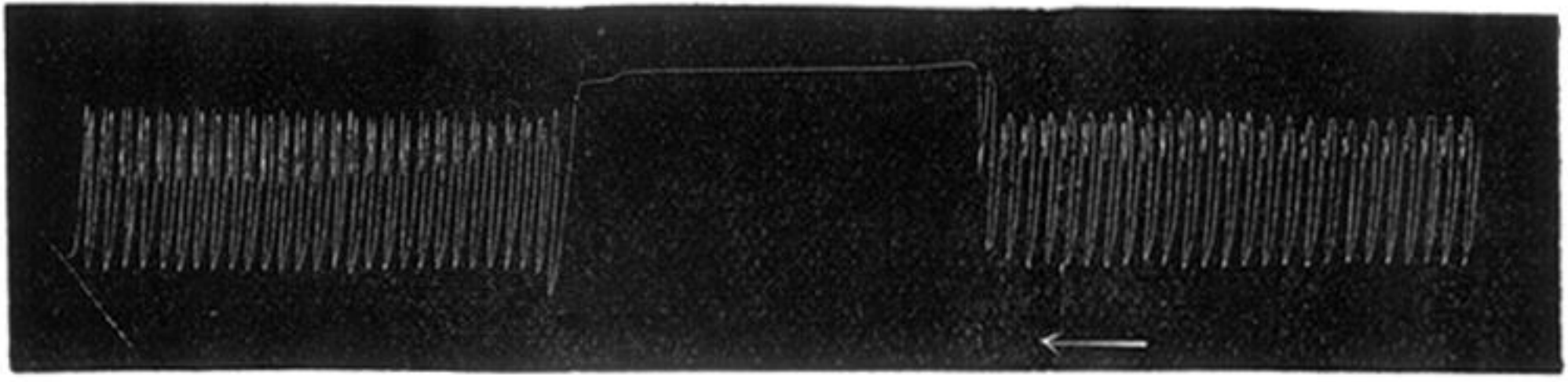


Ventricle tracing showing inhibition resulting from stimulation of vagus nerve. The recommencing beats are seen to be large.



Ventricular tracing showing an inhibitory standstill—a result of vagus stimulation. The beat marked + was elicited by direct stimulation of the ventricle during the period of inhibition.

Tracing of auricle showing vagal inhibition. The beats marked + were elicited by direct stimulation (single induction shocks) applied to the auricle before its action had recommenced. It will be seen that the auricular beats continue to be of diminished size for some time after the spontaneous action has recommenced.



Tracing showing the contractions of both auricle and ventricle. The large beats are those of the ventricle; the smaller ones between the large beats are those of the auricle. The effects of vagal inhibition are shown. The recommencing auricular beats are much diminished in size; the recommencing ventricular beats are large.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