Croonian Lecture.—*Upon the Motion of the Mammalian Heart.*

By Thomas Lewis.

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In these days when Europe is ablaze from end to end and our armies are fighting to regain that freedom and peace which a few years back enabled us undisturbed to pursue our search for knowledge, I wish that this lecture could deal with some problem which affects the health of our troops. But though engaged for some while with my fellow workers upon problems of this kind, I am unable to deliver the lecture in this form. Were I to attempt it with the material at my disposal, it would not be compatible with the traditions of this lectureship to which you have done me the honour of appointing me. As an alternative permit me to review a chapter of physiology recently brought to completion and one which we may fairly claim to have been compiled in chief part by workers in this country.

Three hundred years have passed since William Harvey, our fellow countryman, preached the doctrine by which his name has been immortalised. In his book 'De Motu Cordis,' that famous model of unclouded thought and of scientific reasoning, he wrote of the mammalian heart in these words:—

"First of all, the auricle contracts, and in the course of its contraction throws the blood into the ventricle, which being filled, the heart raises itself straightway, makes all its fibres tense, contracts the ventricles, and performs a beat, by which beat it immediately sends the blood supplied to it by the auricle into the arteries; these two motions, one of the ventricles, another of the auricles, take place consecutively, but in such a manner that there is a kind of harmony or rhythm preserved between them."

It is that sequence of movement, it is that harmony or rhythm of which Harvey wrote, that forms the subject of this lecture.

Our knowledge of the heart's movements progressed but slowly from the time of Harvey's discoveries. In the days of Albrecht Haller, the Swiss, the movement of the heart was described as peristaltic and was likened to that seen in portions of the alimentary tract. Guided by observations upon cold-blooded vertebrates, physiologists regarded the movement as a muscular wave, originating in the neighbourhood of the sinus venosus and passing over the chambers of the heart in regular order. But when it became known that the system of the ventricular contractions may in certain circumstances become independent of the auricular contractions this view was largely abandoned.

In the middle of the last century collections of ganglionic nerve cells were

discovered in the auricular wall of the amphibious heart; the functions of these cells were not understood, they were for many years wrongly interpreted. The incorrect interpretation obtained a powerful hold upon men's minds, colouring their thoughts and their observations. The cells were regarded as central regulating stations, from which the rhythm of the heart was propagated, from which the sequence of chamber contraction was ordered. It is to this period that the observations of Stannius belong. Though doubts arose at a later date, it was not until W. H. Gaskell published his observations (9), that the hypothesis of a neurogenic control of sequence in the sense in which it was then employed was laid finally to rest. The discovery of Gaskell which now concerns us followed and was prompted by the work of that distinguished English scientist Romanes, upon the contractile bell of jelly fishes. Romanes (20) proved that the contraction is propagated as a wave through the bell and that the direction of its spread is governed by continuity of the tissue. The wave of contraction could be diverted by systematic incisions, zig-zag or spiral as the case might be, and would follow regularly the path devised for it, however unusual such a path. Gaskell demonstrated the same fact in the muscular wall of the auricle, proving beyond cavil that contraction travels as a wave, and that the contraction of one segment of an isolated strip is provoked by the passage of the contraction wave into it from a neighbouring segment. He showed, as Romanes had done for the umbrella of Medusa, that the sequence of contraction in the elements of a strip of heart muscle is independent of the natural order in which these elements contract, that continuity alone guides the flow of the wave. He showed that the muscle of the heart may be so cut as to disorganise the supposed system of governing nerve fibres united to a central nerve station without interrupting the passage of the wave. He clearly enunciated his conclusion that the passage of a contraction wave from one mass of heart muscle to another depends upon the bridging of the gap between them by muscle tissue and upon the functional integrity of the bridge. He demonstrated that when one mass of heart muscle contracts in sequence to another, the stimulus which promotes the contraction of the former is derived from the activity displayed by the latter. If the functional integrity of the bridge is impaired by such an experimental procedure as pressure upon it, then the response in the distal mass is delayed, or interrupted, according to the degree of damage. These and other ingenious experiments overthrew those hypotheses which held the nerve cells in high relief, and gave the lead to the work which followed; for it was assumed, in the light of his experiments, that the contraction travels throughout the heart as a wave from one chamber to the next and that it is carried across the gaps by means of muscular

bridges. As a sequel the attention of workers gradually became focussed to discover (1) the precise point at which the contraction originates and (2) the precise paths followed by the natural wave as it travels over the heart.

I do not propose to follow in detail the growth of knowledge from the time of Gaskell's experiments; I shall be content to enumerate the chief steps as they were taken; neither shall I follow those steps in their exact chronological order. Gaskell worked upon the relatively simple hearts of the frog and the tortoise; my own object as a clinician has been an understanding of the motion of the mammalian organ; to this, therefore, I shall now in the main confine myself.

For several reasons, investigation of the mammalian heart is more difficult than that of the cold-blooded heart. The mammalian organ is more viable, it is more complex, its movements are far more rapid. The blood stream through the mammalian heart has to be maintained, the heart insists upon respectful treatment, otherwise the natural beat is not maintained. I lay particular emphasis upon the fact that those methods of investigating the mammalian heart which subject the organ to the least manipulation and damage are the methods which are most successful in elucidating the nature of its beating. The rapidity with which the contraction wave passes over the tissues of the mammalian heart necessitates the use of delicate apparatus in its study. Our methods of registering the movement in different heart chambers have improved very rapidly of recent years; systems of recording levers have become ever lighter and quicker in their movement, but mechanical contrivances, much as they have been employed for the purpose, have so far proved inadequate. For accurate observation, electrical methods have almost wholly replaced them. In 1878 the first records of the heart beat were taken by Burdon-Sanderson and Page (21), using the capillary electrometer; their work upon the tortoise heart may be regarded as the real starting point of modern electrocardiography. Five years later Waller (24) showed that the beat of the heart may be recorded in mammals (including the human subject), without exposing the organ, without damaging the animal in any way. In 1892 the mammalian heart was the subject of special study by Bayliss and Starling (1). These early galvanometric studies, while throwing little actual light upon the course of the contraction wave, are, nevertheless, to be regarded as essential steps; they opened up a new pathway, which later workers have pursued; in this sense the workers were pioneers. It was in the present century that Einthoven (4), in Holland, perfected an instrument which, on account of the facility of its working and the precision of its movements, has enabled us to unveil much which was formerly mysterious.

This instrument is the string galvanometer, and its construction must rank as a chief milestone in the study of the questions which we are considering. It has been known for very many years that, when muscle becomes active, that is to say, when it passes to a state immediately premonitory to the actual contraction, it becomes relatively negative to inactive muscle, in the sense that the zinc element of a copper-zinc couple is relatively negative to the copper element. When a wave of contraction passes through a strip of muscle, it travels in the immediate wake of what is termed the excitation wave, a wave of electrical change whose crest is a crest of relative negativity. Each part of the tissue, as it is about to become involved in the contraction process, shows this electrical change; the moment at which each part of the tissue is about to contract can be signalled by a sufficiently delicate instrument. It was this fact and Einthoven's instrument of which I took advantage when I commenced my studies of the origin and propagation of the contraction wave throughout the heart.

From this general historical introduction, we may proceed to examine the chief problems which presented themselves, and which have now been solved.

The Pacemaker of the Mammalian Heart and the Spread of the Excitation Wave in the Auricle.

In the cold-blooded heart the contraction wave has been recognised long since to start in the region of the mouths of the great veins which enter the sinus venosus, for in the frog and the tortoise the sinus contracts first, and is followed by contraction of the auricle. This sequence may be witnessed in the frog or tortoise with the unaided senses; it is disturbed if a clamp is placed upon the sinus, for then, while the mouths of the veins continue to beat at their old rate, the whole of the auricle and ventricle remains quiescent.

In the mammalian heart an anatomical sinus does not exist, sinus and auricle have become closely incorporated; but morphological reasoning directed attention to the mouths of the great veins, to the superior and inferior vena cava, as the probable starting point of the contraction wave. A number of early experiments, conducted for the most part upon the dying heart or upon the heart mutilated by fatal incisions, purported to demonstrate that the heart beat starts in the region of the great veins; this localisation lacked both precision and certainty. A chief step (12) was taken when Keith and Flack (1907), in their search for sinus remnants, lighted upon the highly differentiated mass of neuro-muscular tissue, which has since been termed the *sino-auricular node*. This mass of tissue lies in the dog immediately to the caval side of the *sulcus terminalis*, a line bounding

the mouths of the two cavæ; it extends along the ventral border of the superior cava for some distance. This discovery was significant and prompted the experiment which I am about to describe. Simultaneously with the observations of my laboratory, Wybauw, in Liége, instituted independent observations (14 and 26). The results of these investigations were in perfect harmony; we speedily found that the region of the sino-auricular node becomes relatively negative before any other point on the surface of the auricle. The method first adopted was relatively crude; we sought the direction of the first flow of current produced by the action of the auricle through the galvanometer, when leading off from pairs of chosen points on the auricular surface. It was shown that, providing one contact lies over the sino-auricular node, it is a matter of indifference where the second contact is placed, the *S-A* contact is always negative at the start of the electric change. These observations have since been abundantly confirmed by similar observations and by the method of cooling. The last method merits further description. A withdrawal of heat from the tissues is well known to depress their functions, and in experiments upon the frog's heart, cooling of the sinus region was known to decrease the rate of the heart's beating. It does so because it depresses the function of the tissue elements in which the heart's rhythm has its origin. MacWilliam (19) and Flack (7) were responsible for some of the earliest experiments on cooling in the mammalian heart. The rate of the mammalian heart beat is lowered when the area containing the sino-auricular node is cooled, and this reduction of rate is provoked by cooling no other region of the heart (8). This experiment has confirmed in a striking manner what we already knew, namely, that the wave of contraction starts in this locality, but it has shown further that the impulses which promote the rhythm are born there.

At a later date a method (16) was devised in my laboratory whereby the time at which the excitation wave reaches any given point on the surface or lining of the auricle can be determined with a maximal error of 0·002 second. This method allowed us to map out in a precise fashion the wave of excitation in the auricular musculature over the greater part of its course. Our conclusions may be briefly summarised. The excitation wave starts in the head or swollen part of the sino-auricular node; it spreads from this node at an average rate of 1000 mm. per second into the surrounding auricle along lines radiating in every direction from the node. It is noteworthy that the surrounding muscle is arranged in bands which converge in this region of the heart as though arranged to speed the spread. I have likened the spread in the auricle to the spread of a viscous fluid poured from a funnel upon an almost flat surface; the margin of the fluid invades the plate as an ever

widening circle. The spread in the auricle differs in this respect only, that it is confined to the muscle bands. The wave spreads up the superior cava against the blood stream; when it reaches the mouths of the inferior cava and of the pulmonary veins, it similarly spreads up them; it spreads from the base to the apex of each auricular appendage; it spreads down the septum towards the ventricle. There are no special paths of conduction; the spread is uniform and from one muscle element to contiguous ones, and involves the two auricles as though they composed a single sheet of muscle.* The spread in the auricle is ordered upon a simple plan.

Spread from Auricle to Ventricle.

Between the contraction of the auricle and the ventricle there is considerable delay; the delay is very pronounced in the frog and tortoise. It was Gaskell who showed, by placing a clamp upon the groove which separates these two chambers and by gradually tightening it, that precisely the same disturbance of sequence may be induced as when a clamp is applied to an isolated strip of auricular muscle. By clamping, the passage of the wave is at first hindered, later the ventricle fails to respond after occasional auricular contractions, later still as pressure is increased the responses of the ventricle become fewer until there is no response at all. These experiments led Gaskell to the conclusion that the sequence of the ventricular upon the auricular beat can be explained without the intervention of any special nervous mechanism, that the passage of the impulse over the groove is of the same nature as its passage over a bridge of tissue in the incised auricle. He showed also that this sequence is not disturbed by removal of Bidder's ganglia. In the frog the junction between auricle and ventricle is formed by a ring of tissue, and composed in the main of muscle fibres of relatively primitive type. This primitive muscle formed, in Gaskell's view, the functional bridge between the two chambers. Following Gaskell's studies came the early experiments of Tigerstedt (23), and of Wooldridge (25) and MacWilliam (19) in this country. Their experiments determined that conduction across the *A-V* groove in the mammal is subject to the same disturbances as is conduction in the frog, and showed that the impulse received by the ventricle is not the filling with blood by the auricle, and that the impulse is not conveyed through the superficial cardiac nerves. But at this time and for many years afterwards the auricle and ventricle of the mammalian heart were not known to be joined by a bridge of muscle

* In this conclusion we differ from Eyster and Meek (6), who believe that there is specially rapid conduction to the *A-V* node. Our criticisms of their methods have been published (16).

tissue, though nerves pass freely over the auriculo-ventricular groove. The musculatures of the two chambers were, in fact, widely believed to be disunited. It seemed as if either Gaskell's conclusions for the cold-blooded heart lacked finality, or that a fundamental difference exists between the mechanism in frog and mammal. The obvious discrepancy was removed from the minds of English physiologists by Kent's description of a muscle union between the two chambers in the mammal in 1892 (13). A year later, His described the muscle bridge more distinctly, and made the first experiments upon it (11). The final experiments were made by the Americans, Erlanger (5) and Cohn (3), and others (10). It has been proved beyond all reasonable doubt that this muscle bridge, which runs from the auricular to the ventricular septum, is in the mammalian heart the sole path by which impulses are conveyed from auricle to ventricle. That pressure upon or cooling of this bundle hinders the passage of the impulse, that division of this bundle completely dissociates the movements of auricle and ventricle, is now recognised; these effects are regularly obtained in a number of experimental laboratories.

Distribution of the Wave in the Ventricle.

I pass over the earliest experiments upon the spread of the contraction wave through the ventricle. The observations were electrical, and were inaugurated by Burdon Sanderson; they were contradictory because the complexity of the spread was not appreciated.

In respect of the spread in the mammalian ventricle a great step in the progress of knowledge came with the anatomical discovery of Tawara (22), the Japanese. Many years before Purkinje had described a network of highly differentiated cells lying beneath the endocardium in mammals. The functions of these cells were in his time quite unknown. Tawara demonstrated that they compose in each ventricle a striking basketwork, lining each chamber. He traced the ending of the muscular bundle which unites auricle and ventricle into a right and a left division, each of which by a few chief strands unites with the basketwork lining the corresponding ventricle. He concluded, and rightly concluded as it transpires, that the bundle forms with its branches and arborisations the channels by which the impulse to the mammalian ventricle is distributed. The experimental proof that this is so is now forthcoming.

The ventricle is the chamber which accomplishes the heart's real work; it is powerful muscle, arranged around the blood-containing cavities, and forming to these cavities a thick wall. The muscle fibres are arranged in an intricate fashion, largely in the form of broad spiral bands, some of

which encircle both cavities, while others limit themselves to a single cavity. The chief spiral bands start at the bases of the ventricles and pass to the apices of the ventricles where, turning sharply, they form vortices and pass up to constitute the papillary muscles. The arrangement is such that these layers may be dissected off one by one, and as each new layer is reached, the inclination of the fibres alters.

Our first efforts (17) to unravel the course of the excitation wave took account of these muscle bands, for we naturally supposed the course of the wave to be controlled by them. But the readings which we obtained demonstrated to us clearly that the times at which the excitation wave appears at different points of given superficial bands are incompatible with this view. We explored the whole superficies of the ventricle, and constructed maps of the ventricle in a number of animals which showed the precise surface distribution of the excitation wave in relation to time. These maps portrayed a number of new facts. It was to be seen that the system of distribution is tolerably uniform from animal to animal; it was shown, considering each ventricle separately or the two ventricles together, that points widely apart are activated simultaneously. A simple form of distribution, the passage of the wave from one muscular element to the next, a radial spread from a given point on each ventricle or a given area of each ventricle would not suffice; we were compelled to recognise that the spread occurs to a large number of surface points simultaneously; no other hypothesis could explain the rapidity of the spread or the order of it; we were forced to the conclusion that the excitation wave is distributed by a number of separate and distinct channels. This conclusion was at variance with the conclusions of workers who had previously used the electrical method: these had all assumed a simple form of spread; but it harmonised in a general way with the recent anatomical discovery of the arborisations of the A-V bundle. Attention was consequently directed to this system, and experiments specially devised to test the matter proved its importance.

If after mapping out the distribution of the wave to the ventral surface of the two ventricles, the right division of the auriculo-ventricular bundle, which breaks up into the network of the right ventricle, is divided, the spread becomes altered (17 and 18). Over the left ventricle it remains unchanged, over the right ventricle it is delayed; moreover, after such a lesion, the order in which the surface is activated is found changed; the wave now starts at the margin of the right ventricle where it borders the left ventricle and travels over the ventricle away from this margin. Briefly, when the right division of the bundle is cut, the excitation wave at first spreads only to the left ventricle; later, when the left ventricle is

completely involved, the right ventricle receives the wave through the muscle which unites the two chambers; thus, after section of the right stem of the bundle the right ventricle is activated in a high abnormal manner.

Another experiment explores the network itself. The natural spread of the excitation wave over the conus of the right ventricle is away from the chamber of the ventricle and towards the outlet of the pulmonary artery. A cut traversing the conus at right angles to the general direction of spread and penetrating the muscle fibres for a considerable depth, fails to delay the appearance of the excitation wave above the incision, while a shallow incision or even a scratch in a similar plane, but applied to the inner or endocardial surface, produces a profound delay. Normal conduction is therefore through the branches of the auriculo-ventricular bundle and ultimately through the arborisation and network.

But this demonstration still leaves several questions unsolved. If the impulse which descends the bundle from the auricle is distributed to the networks lining the cavity of the heart, by what paths is it conveyed to the surface of the heart? It is conveyed, as the following experiment clearly shows, by direct penetration of all the muscle layers along paths radiating from the cavity. The muscle of the left ventricle is very thick, and can be deeply incised with impunity; if a point is chosen on the surface, and the time at which the excitation wave arrives at it is determined, this time is uninfluenced by deep incisions which completely surround the point in question. But if an incision is made which undercuts the point examined, the wave is greatly delayed in its passage. This simple experiment shows that the wave is carried from within outward from one muscle band to the overlying one, and that it travels in a plane which is at right angles to the direction of the fibres. In support of the same conclusion are time readings taken from the cavity of the heart; if corresponding points on the inside and outside of the heart are tested, it is found that the excitation wave always reaches the lining before it reaches the surface of the heart.

Now the facts which I have so far related point steadily to one conclusion, namely, that the distribution of the excitation wave in the mammalian heart is accomplished through the branches of Tawara's tree. Arriving at the bundle the impulse travels through the main branches, through the smaller branches and twigs, until the whole lining of the cavity becomes implicated; from the network which lines the cavities it spreads directly into the ventricular wall, penetrating each layer of it in succession. This general account appears a simple and satisfactory explanation of the facts so

far as I have related them, but the problems which arose during the researches were not so simple; an observation, of the soundness of which we were convinced, for a long while mystified us. Our surface maps showed that the earliest parts of the surface to be activated are the extreme apex of the left ventricle and an area of the right ventricle lying a little above its apex. So far as the latter was concerned there was no great difficulty, for in the dog, upon which our experiments were conducted, this region of the right ventricle is directly supplied by large branches of the arborisation which pass directly to it. No such direct strands pass to the apex of the left ventricle; moreover the activation of the surface of the left ventricle at a little distance from the apex, although this surface lies nearer to the trunk of the tree than does the actual apex, is activated almost as late as any part of the heart's surface. This discrepancy is one of many observed; the surface distribution is not to be explained solely by the length of conducting fibres which pass to underlying points of the lining; there is some other and chief factor influencing the surface distribution. This factor was discovered when the rates of conduction of the excitation wave propagated by electrical stimulation were investigated. If two points, one of which lies on the surface and the other of which lies on a corresponding part of the lining, are tested and readings are obtained from them while the heart is beating naturally, the point on the lining is the first to show activity; this is natural, seeing that the impulse has its starting point within. But if the same points are tested when the excitation wave is artifically excited from a *point on the surface* at some little distance from the tested points, *the same phenomenon is witnessed.* The point on the lining becomes active first, and by the same time interval, as it does when the heart is beating naturally. The excitation wave reaches the point on the lining first, although that point is further away from the point of stimulation than is the point on the heart's surface. It now began to be evident that conduction along the lining is more rapid than is conduction along the surface. That the rapidity of conduction in the lining is due to the Purkinje network is shown by scratching the lining between the points tested and the stimulating electrodes; after this interference the point on the lining is no longer activated first. On the other hand a deep incision into the surface muscle, though almost penetrating to the cavity, does not affect the time relations. Further investigations showed that the interval which elapses between the arrival of the wave at the points tested varies within certain limits according to their distance from the point of stimulation. If this distance is reduced sufficiently, then the excitation wave arrives at both points simultaneously. By suitably arranged experiments and measurements of the network and thickness of the muscle walls it is possible to calculate

the ratio between the rate of conduction through the muscle and through Purkinje tissue. The proportion is approximately as 1 is to 10.

Again, if the rate of conduction between two points on the surface of the heart is examined and these points lie on the wall of the ventricle where this is thickest, the conduction rate is approximately 300–500 mm. per second; a similar rate of propagation is ascertained during the natural passage of the wave from within outwards through the muscle wall; but if the rate of conduction is tested similarly over thin portions of the ventricular wall, it is found to be as high as 1500–2000 mm. per second. In the last-named circumstances the wave is short-circuited through the Purkinje network for the longest part of its course. After consideration of all the circumstances we have concluded that the rate of conduction through ventricular muscle lies between 300–500 mm. per second, while that through straight paths of Purkinje tissue lies between 3000–5000 mm. per second.

This conclusion clears away the discrepancies in surface readings to which allusion has been made. The surface points which are activated earliest are those points which overlie the thinnest parts of the ventricular walls. There is an unmistakeable relation between any surface reading and the thickness of the muscle at the point tested.

The surface readings are controlled by two factors: first by the length of the Purkinje strands and secondly by the thickness of the muscle wall. If a number of surface points are tested, and the heart fixed; if measurements are made upon the lining and on the basis of the estimated conduction rate over the lining the corresponding times are calculated; and if measurements are made of the thickness of the ventricular wall and on the basis of the estimated conduction rate in ventricular muscle the corresponding times are calculated and added to those of the first series; then a complete series of calculated readings is obtained which harmonises with the readings won from the naturally beating heart.

A General Summary.

We are now in possession of a very detailed knowledge of the course taken by the contraction wave through the mammalian heart. It behoves us, in so far as we are able, to review the mechanism of the heart beat in the light of this new knowledge. The mammalian heart has onerous duties to perform, but the first plan of its construction is the plan of a simple tube contracting peristaltically from end to end without interruption. During phylogenetic and embryological development the work of the heart increases, the shock of its movements increases. The heart divides in two, an upper and lower chamber, the first frail and thin-walled, the second sturdy and

massive. Why are these two chambers developed in the heart? The function of the auricle is not to fill the ventricle; the blood pumped from auricle to ventricle at the end of the heart's diastole is but a fraction of its full content. The function of the auricle is to save the veins from over-distension. During a third of the ventricular cycle, during the period when the ventricle is pouring blood into the body, the circulation is completely obstructed at the auriculo-ventricular orifices. Yet the blood flow in the veins continues; the flow goes on into the expanding auricles. The mechanical function of the auricle is that of a reservoir, to catch and hold the content of the stream when a dam is thrown across it. It wants little strength for this task; its walls must stretch easily; they have to pump against a low resistance; the muscle layer is therefore thin.

The first sign of contraction is in the region of the opening of the superior cava; it quickly spreads to the mouth of the inferior cava. The mouths of these veins tend to close before the main mass of auricular muscle is involved, thus hindering or preventing regurgitation. The spread of the contraction wave to the auricular muscle as a whole is expedited by the central position of the node from which the wave starts, by the architecture of the muscle bands which radiate from it in all directions, and, lastly, by the relatively high rate of conduction which auricular muscle possesses (1000 mm. per second). In the mammalian auricle the plan of distribution is primitive.

The wave travels through the auricle to the first part of that bridge which joins the auricle and ventricle. Here there is delay and long delay, while the auricle is emptying itself and the auriculo-ventricular valves float up preparatory to closing. This delay occurs, so it is believed, in the auriculo-ventricular node, a structure of very small muscle fibres (15). Leaving the node, the wave passes to the bundle. The need of this structure and of its branches and twigs has developed with the division of the ventricle into two cavities. This special system of fibres is endowed with the highest order of conducting power; it is arranged that both ventricles and all their parts may be thrown into contraction with the closest approach to simultaneity; in that lies a mechanical advantage which the ventricle as a driving muscle demands for itself. The wave does not travel along the ventricular wall: were that so and were its progress as rapid as in the wall of the auricle, its course, nevertheless, would be slow. It penetrates the wall; its course in muscle is short, and therefore a high rate of conduction in the muscle is not essential; we actually find it to be of a low order.

The system of distribution in the thick-walled ventricle possesses a further virtue in that it protects the laminated tissues from disruption.

A contraction wave following the spiral muscle bands, bands arranged to wring the blood powerfully from the heart, would tear them asunder, would rupture the fragile vessels penetrating all at right angles.

Gaskell, with that acuity of perception which was his pronounced quality, thought of the muscles of the sinus and of auriculo-ventricular ring as primitive remnants, highly endowed as is the primitive cardiac tube with the function of rhythmicity, poorly endowed with the function of conductivity; he associated the fine structure of the tissue of the rings with peculiarity of function. A similar but more complex relation between structure and function is to be demonstrated in the striated tissues of the mammalian heart. Rhythmicity as a function is most highly developed in the sino-auricular and auriculo-ventricular nodes, structures which closely resemble each other in the fine details of their construction, structures which are now both held to represent remnants of, or developments of, the original sino-auricular ring. Conductivity is a function held in varying degree by the mammalian tissues. The Purkinje cells are the largest striated cells, and possess the highest content of the carbohydrate glycogen; they conduct most rapidly. The fibres of the auriculo-ventricular node, the smallest to be found in the heart, conduct most slowly (15); they are almost devoid of glycogen. Between these two extremes are the fibres which compose the walls of the auricle and ventricle. These fibres are of intermediate size, the content of glycogen is intermediate, the power of conduction is intermediate. The ventricular fibres are said to contain less glycogen than the auricular (2), they also conduct less rapidly. Thus, the rate at which the wave flows is controlled by the structure and chemical constitution of the tissues through which it passes; the musculature in its various parts is so differentiated that from its appearance the manner of its working may be known.

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On the Influence of Vibrations upon the Form of Certain Sponge-Spicules.

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It has been pointed out recently* by one of us that the development of the remarkable chessman-spicule or discorhabd in the genus *Latrunculia* is a somewhat complicated process depending upon several factors. The protorhabd or axial thread appears first as a slender rod capable of independent growth. With these protorhabds two kinds of silica-secreting cells appear to be associated, viz., formative cells which are responsible for the actual deposition of the silica upon the protorhabd, and accessory silicoblasts which are supposed to collect supplies of silica and bring them to the formative cells to be used in the process of spicule-formation. The spicule in this case consists of an elongated axis with whorls of flattened lobes arranged at more or less definite intervals along its length, and it was suggested that the

* A. Dendy, Presidential Address to the Quekett Microscopical Club, 'Journ. Q.M.C., Ser. II, vol. 13, p. 231 (1917).