A NEW OUTLOOK IN CARDIOLOGY.

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(Continued from page 5.)

THE SINO-AURICULAR NODE.

IN the lower vertebrate animals the part concerned in starting the heart beat is spread diffusely through the sinus venosus. In the mammalian heart the sinus disappears and the parts concerned in starting the heart beat are, to a great extent, accumulated in a well defined collection of cells—the sino-auricular node, which is situated at the mouth of the superior vena cava. There are other portions in the auricle capable of initiating the contraction, for the auricle continues to beat regularly and effectively when the specialized tissue of the sino-auricular node is destroyed. Although not clearly defined, there is every reason for assuming that all the parts concerned in the starting of the heart beat act harmoniously, and probably cease to act at the same time.

The node consists of a number of filiform cells. How these are connected with the muscle cells of the auricle or of other structures that are concerned in the transmission of impulses is not known. There is no doubt that the "control" influence of the sino-auricular node extends beyond the auricle—certainly to the auriculo-ventricular node.

The Rhythmic Activity of the Sino-auricular Node.

The heart when freed from all its nerves, and even when removed from the body, continues to beat in a regular and normal manner. This is due to the sino-auricular node, which in consequence has been said to possess a "rhythmogenic function."

In describing the nature of cell activity, it was shown that a cell recovers after it discharges its function. As the recovery proceeds the cell becomes more and more susceptible to stimulation. This is what happens to the cells of the sino-auricular node, and they are peculiar in the sense that they are particularly sensitive to stimulation. It may be asked, Whence come the stimuli which excite the cells of the A-V node to discharge their function? If it be true that all living cells throw out impulses, there can be no want of adequate stimuli. Moreover, as the restoration of the energy proceeds, the susceptibility to stimulation becomes greatly increased, and it is possible that the temperature alone may be sufficient to cause a discharge. Such seems a more reasonable explanation than endowing the cells with the quality of automaticity, or a rhythmogenic function.

PARALYSIS OF THE SINO-AURICULAR NODE.

(*Paralysis of the Auricle: Auricular Fibrillation and Flutter.*)

In the history of medical progress it is of interest to note that some discoveries are recognized at once and an exalted view taken of their possibilities, and it may take many years' experience before they are seen in their proper perspective, while other discoveries are not recognized or appreciated till long years after, when experience gradually reveals their significance. The condition which at one time has passed under such terms as "paralysis of the auricle," "nodal rhythm," and "auricular fibrillation" belongs to the latter group. When the full significance of the discovery of this condition comes to be recognized, it will be looked upon as one of the most important discoveries, not in the heart alone, but in medicine. While its study has thrown a flood of light upon so much that was hidden and obscure in affections of the heart, it has revealed principles which govern vital processes applicable to every field of medicine. Some of these are as yet dimly perceived, but only time and experience are needed for them to be fully recognized.

A brief account of the history of this condition serves to illustrate how research in clinical medicine may be

carried out. For more than a hundred years clinical observers were dimly conscious of a peculiar condition of the heart which occurred in certain people in ill health. The description by Withering⁶ of the powerful effect of digitalis upon certain hearts shows that he was dimly aware of some peculiarity which we can now recognize. When physicians came to identify mitral stenosis some recognized a peculiar pulse which frequently was associated with this condition, and was called the "mitral pulse." As long as forty or fifty years ago some physicians recognized that in mitral stenosis the presystolic murmur sometimes disappeared. In carrying out an inquiry into irregularities of the pulse I recognized that the pulse gave information only of what the left ventricle was doing, and I endeavoured to find out what the auricles were doing. Over thirty years ago I noted that in certain conditions the auricle gave no evidence of its presence, but it was not till five years later (1898) that I discovered that in certain patients all evidences of auricular activity suddenly disappeared. The first case in which I clearly recognized this was a patient I had watched for seventeen years. She had shown for some years a presystolic murmur at both the mitral and tricuspid orifices. She had also shown pulsation in the jugular veins and in the liver, due to the auricular systole. Suddenly all these symptoms disappeared, and the heart became irregular. Following the custom of the day, I assumed that with the disappearance of auricular activity the auricle had become paralysed. At this date little was known of the processes concerned in regulating the heart beat. The heart beat was supposed to start somewhere in the auricle, and I assumed that as the auricle ceased to beat the ventricle started on its own. When later the auriculo-ventricular node and bundle were discovered and it was shown that the ventricular contraction was dependent upon the A-V node, I suggested that the contraction started here, and called the condition "nodal rhythm."

In 1906 Cushny and Edmunds drew attention to the similarity of the irregularity of the pulse of a dog in whom they had produced auricular fibrillation to that of a patient with an irregularly acting heart, and they suggested that auricular fibrillation was the cause of the patient's abnormal rhythm. This view was confirmed by Winterberg and Rothberger and Lewis in 1909. The demonstration was so convincing that, although ignorant of its nature, I, with others, accepted the discovery as settling the problem.

The conception that auricular fibrillation was the cause started investigations upon a new line, which one must now recognize has been of little profit. In reading the recent literature upon this subject it is astonishing to find how obsessed clinical observers are by laboratory discoveries. A number of writers reckon to give the history of this discovery, and they start with the time when it was labelled "auricular fibrillation," evidently unaware that all the signs by which it is diagnosed, the basis of prognosis, and the indications for treatment were all recognized before the term "auricular fibrillation" was applied to it. As a matter of fact, the recognition that the auricle was fibrillating has not added one single fact to the clinical aspect of the subject.

As years went on I became convinced that there was a great deal more in the matter than a fibrillating auricle. I recognized, for instance, that with the onset of this condition the heart acted, in many ways, in a manner different from that when the normal rhythm was present. The pulse became quite distinctive, having a character of its own. The rate of the pulse could be influenced in a manner also distinctive. Sometimes with the onset, the rate would rise to over 150 beats per minute; in some patients, when at rest, the rate would fall below the normal, to about 50 beats, but with the slightest effort might rise to over 150 beats. Frequently when at rest the rate would vary in a most extraordinary manner. Equally striking was the reaction to digitalis. Patients upon whom digitalis would have little effect when the rhythm was normal became extraordinarily susceptible to the drug when fibrillation set in. After a few drachms of the tincture the rate would be reduced from 130 or 150 down to 50 beats and under per minute. Moreover, as far as can

be seen, sudden death from digitalis only occurs in this or its allied condition, auricular flutter. Probably the most striking feature of all was the form of heart failure that set in sometimes immediately on the onset of this condition. Furthermore, patients who might be suffering from all the symptoms of heart failure which is characterized by angina pectoris would suddenly lose all the symptoms of angina and would suffer from the symptoms peculiar to auricular fibrillation.

The Application of the Principle of the Reflex Arc.

I recognized these features, and looked hopefully to experiment and to the electro-cardiograph to throw light on the subject, but very little has resulted from their employment. It became evident that no further progress could be made unless some other method of studying the subject were employed. When we recognized disturbed reflexes as a cause of many symptoms we applied the principles of the reflex arc to the interpretation of cardiac phenomena, and at once an unexpected field of investigation was opened up. This principle requires for its application that we should go back step by step from the organ whose disturbed activity produces the symptom, inquiring into the part played by each element of the arc, as was done in interpreting Figs. 3 and 4. The reason for this, as already described, is that the process which regulates the function of an organ is governed by a series of elements which constitute the arc. In the arc the higher element governs the lower, so that when we find a disturbance in the lower element we must continue the inquiry into the elements that are above it to see if they are acting in a normal manner.

For an illustration, let the tracing in Fig. 5 be taken—it shows an irregular pulse with a venous pulse of the type which we know is characteristic of auricular fibrillation—and the facts which we recognize in such a tracing be represented in a diagram (Fig. 5A). In this diagram there are four spaces as in the diagram of Fig. 3.

The lower space represents the ventricular systoles, which are identical with the beats of the radial. The downstrokes represent the ventricular beats and their force varies, as shown by the size of the pulse beats. Manifestly, the ventricle is disturbed. Is this disturbance limited to the ventricle, or does it come from a higher level? The electro-cardiogram has revealed that in such forms of ventricular activity the impulse for contraction comes from a higher level—that is, from the auriculo-ventricular node. Fig. 6 demonstrates this. The radial pulse which is given here, though somewhat unclear, is nevertheless quite typical of auricular fibrillation. The character of the electro-cardiogram shows that the ventricular complex

is of that kind due to the stimulus for contraction which enters the ventricle by the normal paths—that is, by the auriculo-ventricular bundle. We know that when the stimulus is produced in the bundle (as in complete heart-block) the rate is very slow, so that we can infer that the stimulus for the ventricular contraction in Fig. 5 arose in the auriculo-ventricular node. This node is represented in the diagrams Fig. 5A and Fig. 6A (space A.V.N.), and shows it varying greatly in its activity. Still applying the principle, we look higher to see what the auricle is doing, and we find it in the condition of fibrillation. This is represented in the spaces *As* by a series of small strokes

which represent a shower of impulses which the auricle throws out. Going still higher, we look at the space of the sino-auricular node, and discover that there are no signs or evidence of its function. This raises the question, What is the sino-auricular node doing?

It has been assumed that the fibrillating

auricle has thrown it out of action; but, on the other hand, there are good grounds for assuming that its going out of action is the reason for the auricle fibrillating. As yet the grounds for this reason cannot be directly demonstrated, but by a process of reasoning it can be assumed with fair certainty.

PARALYSIS.

The cessation of activity raises the question of paralysis of the sino-auricular node, but when we ask what is meant by paralysis we find no clear answer. It seems to be a subject, like a great many others in medicine, which is supposed to be so well understood that nobody ever thinks of investigating it. One or two illustrations will show what is meant. The action of atropine produces an increased heart rate, dilatation of the pupil, and delirium. At first sight one might say that atropine stimulates the heart, stimulates the dilator muscle of the pupil, and stimulates the brain. But a more intimate knowledge shows that atropine acts upon the terminal fibrils of the vagus nerve and abolishes their function, so that impulses by the vagus fail to reach the sino-auricular node. In

a similar way atropine acts upon the terminal fibres of the nerve that supplies the constrictor muscle of the pupil, so that impulses by this nerve do not reach the muscle and it ceases to act, or, in the words commonly used, it is paralysed. It is reasonable to assume that the drug has an identical action on the terminal fibrils of certain neurones of the brain, so that impulses from a higher centre are blocked, and the result is delirium. Delirium, then, is no more due to stimulation of the brain than the resultant increased heart rate is due to stimulation of the heart.

I put the matter in this way to indicate the lines of

FIG. 5.—Simultaneous records of the jugular and radial pulses. The radial pulse is irregular, of a kind typical of auricular fibrillation, inasmuch as there is no relation between the length of the preceding pause and the size of the beat. The waves (v, c'v) in the jugular tracing are due to the ventricle, and are characteristic of auricular fibrillation in that they show no auricular wave. At times there is no evidence of a beat in the radial, but there is one v wave in the jugular, showing that the ventricle contracted, but so feebly that the wave in the radial was not sufficient to raise the lever of the sphygmograph. (See diagram Fig. 5A.)

FIG. 5A.—A diagram representing the events in Fig. 5. In the sino-auricular node space there is no sign of activity. The short strokes in the space *As* represent the impulses from the fibrillating auricle. The space A.V.N. represents the auriculo-ventricular node and shows its varying activity, and the irregularity of the pulse is seen to be due to the varying periods of recovery of this node. The downstrokes in the space *Vs* represent the ventricular systoles as shown in the radial pulse. These systoles vary in size and in force. The length of the strokes represent the output of the heart as shown in the radial tracing.

FIG. 6.—An electro-cardiogram of the heart taken at the same time as a tracing of the radial pulse from a case of auricular fibrillation. The complex of the electro-cardiogram is indicative of that given by the ventricle when the stimulus for contraction comes from the auriculo-ventricular node. The radial tracing is typical of the small imperfect beats which occur in auricular fibrillation, and resemble those in Fig. 5. (J. Parkinson.)

FIG. 6A.—A diagram of the events in Fig. 6.

investigation we pursue in using the reflex arc, and to show that the excitability of the part below the disturbance is not an indication that such excited part (as the fibrillating auricle) is the original seat of the disturbance.

The next point to which attention should be directed is, What happens to the organ or tissue which is said to be paralysed—that is, when impulses from a higher level are blocked, as when the nerve to a voluntary muscle is cut? In the description of the cell it was shown that the cell possesses the faculty, when supplied with appropriate material, of assimilating nutrition, of storing energy, and of discharging it. When a motor nerve is cut the muscle cell is still supplied with nourishment, and it is still capable of discharging energy, and of renewing its store, so long as appropriate material is supplied to it. Moreover, such cells are stimulable from other sources than through the nerve fibres. It would be expected that such muscle cells would show some form of activity, and, as a matter of fact, they pass into fibrillation, as Langley has shown, and even into a condition which resembles flutter. I put forward the view that the same thing happens in the auricle, and that the fibrillation is the result of the loss of control of the sino-auricular node. But some of my friends have raised the objection that the fibrillation in the two instances is not the same; I am not quite sure that this objection is justified. At the recent meeting of the Physiological Congress in Edinburgh, two Japanese physiologists, Ishihara and Nomura, demonstrated that when branches and terminal ramifications of the auriculo-ventricular bundle were extirpated and placed in warm oxygen-saturated Ringer's solution, they continued to beat for a long time. In a small piece of heart muscle placed in this solution, examined with a low power of the microscope, I saw minute rapid movements exactly like that of the fibrillating auricle. So far as one can see at present the same law, therefore, holds good in regard to the muscle of the heart and the voluntary muscle.

A suggestive indication of another sort can be obtained. In a conversation with my friend Professor J. H. Ashworth, he drew my attention to the work of Mayer on the medusae. I had long ago accepted the teaching of Gaskell, who pointed out that Romanes had demonstrated that the stimulus for contraction of the bell of the medusa was conveyed by the muscle cells. This was supposed to be proved by the impulses being conveyed along zigzag strips of the bell, and it was used as an argument in favour of the myogenic doctrine. Mayer has shown that the conduction is not by the muscle, but by the network of the nerves which are spread all over the body. In his experiments Mayer cut out a ring of the subumbrella tissue, and immersed it in seawater, and applied a stimulus to this ring of muscle, and thus set up a contraction that passed round the ring, one contraction following another, so that for weeks this wave of contraction went round and round this ring of tissue. If, however, the ring is not entirely separated from the other part of the bell, but there is left a very narrow bridge of tissue connecting the ring to the rest of the body, this "circuit" movement in the ring could be stopped at once by an impulse set up by touching one of the sensory bodies at the margin of the bell—in other words, when the ring was free from the control of the marginal sensory bodies it would contract indefinitely, but as soon as these marginal bodies exercised their function the ring came at once under control, and the "circuit" movement ceased.

The suggestion I put forward is that the sino-auricular node has the same sort of control over the muscle fibres of the auricle as the marginal bodies in the medusa have over its musculature, and with the loss of this control the muscle fibres contract in response to impulses from another source.

Further suggestive evidence in favour of the cessation of activity of the sino-auricular node being the cause of auricular fibrillation is found in the consideration of the relation of organs to disease. There is no organ of the body exempt from disease, and there is no reason for assuming that this node is exempt. One of the great causes for the production of impaired function of organs is the impaired blood supply either from obliteration of the capillaries or disease of the arteries. The former condition is an accompaniment of advancing years; indeed, it is this factor which produces so many of the senile changes. Evidence of it can be found

in every organ, affecting in some people one part, and in other people other parts. This is the chief reason of the changes in the valves of the heart so common in old age. This is the reason of the occurrence of heart-block late in life, when a diminution of the capillary field leads to those fibrotic changes which destroy the auriculo-ventricular node and bundle. It is amongst the elderly that auricular fibrillation is so common. We would expect also that diseases apart from senile changes would have a similar destructive effect upon the node, just as we find them affecting the auriculo-ventricular node. Damage of the auriculo-ventricular node, for instance, is fairly common in the rheumatic heart; it is also in the rheumatic heart that we find auricular fibrillation, frequently in the young.

In an experiment to which reference is made later Gaskell demonstrates that the auriculo-ventricular ring, which is probably the antecedent of the auriculo-ventricular node, becomes easily stimulated after the sinus is cut away. I may remind the reader of Hughlings Jackson's doctrine already mentioned: "that destructive lesions do not produce positive signs, but a negative condition in which positive signs appear." This is manifestly applicable to the condition when the sino-auricular node ceases to function.

Finally, when the great principle that underlies control, and the effects which follow the loss of control are understood, the probabilities are that the cessation of the functions of the sino-auricular node will be found to be but one of a number of similar processes which result in the disturbance of the harmonious and effective activity of organs.

An objection which has been raised to this view is that a stimulus like an electric discharge applied to the auricle sets it into fibrillation. In reply to this it is necessary to point out that it is seldom or never possible to recognize the real effect of an experiment. In applying a very gross stimulus of a kind Nature never employs some effect may result; but we do not know what effect such gross stimulation has had upon fine structures such as the junction between the sino-auricular node and the fibres of the auricle. It is quite possible that this junction is put out of action.

While I do not contend that these reasons are sufficient to prove that the condition that we now call auricular fibrillation is due to the cessation of activity or paralysis of the sino-auricular node, they are sufficient to call for a reconsideration of the matter, for the supposition throws light upon the nature of a number of phenomena which has hitherto baffled explanation.

THE AURICULO-VENTRICULAR NODE.

Before dealing further with the condition of auricular fibrillation it is necessary to consider some features of the auriculo-ventricular node. This structure is a collection of cells similar to those of the sino-auricular node, but differs in that it is continuous with a bundle of fibres which passes from it to the ventricle, so that while we cannot yet tell how the impulses are conveyed from the sino-auricular node to the auricle, we know how they are conveyed from the auriculo-ventricular node to the ventricle.

As in so many other instances, in normal circumstances the part played by one element of a complex process, as the auriculo-ventricular node in the heart beat, cannot be differentiated in the harmonious action of the whole complex. In the various disturbances of rhythm the functions peculiar to each element of the regulating process may be revealed.

The function of this node is similar to that of the sino-auricular node in that it discharges impulses which, being conveyed to the muscle cells of the ventricles, cause their contraction. There has been a tendency to look upon the auriculo-ventricular node as being mainly a conductor of impulses, acting in this respect like a nerve. But there is a very great difference. In the conduction by a nerve, the impulse is received by fibrils and conveyed by the axon process to its connexion with other cells, without the cell body taking part in the process. The impulses, for instance, in the afferent nerve in Figs. 1 and 2 can be received and conveyed to the centre when the cell body is cut away.

The cells of the auriculo-ventricular node are not conducting structures in this sense. They are cells actually discharging impulses in the same way as the cells of the sino-

auricular node discharge impulses. After they have discharged impulses they are incapable of being stimulated until their store of energy has been replenished. They are then susceptible to stimulation, and when stimulated they discharge impulses.

Just as the sino-auricular node controls the auricular contractions, so does the auriculo-ventricular node control ventricular contractions. The impulses which the latter discharge are conveyed to the ventricle, so that the node controls and regulates ventricular contraction. We can therefore interpret the functional activity of the auriculo-ventricular node from the ventricular contractions.

This view has to be kept in mind, for we must recognize that the disturbances shown by the ventricle have, in most cases, their origin in the auriculo-ventricular node. When the ventricle is spoken of as being refractory to stimulation it is the auriculo-ventricular node that is refractory, and not the ventricle.

In the course of this investigation there are found contractions of the ventricle where only a portion of the fibres of the ventricle participate in the contraction, and these imperfect contractions can be traced to the auriculo-ventricular node and it can be inferred that only a few cells of the node threw out impulses at that time.

This view of the auriculo-ventricular node and its relation to the ventricular muscle brings it into harmony with the mode of action of other muscular organs. All other muscular organs, both voluntary and involuntary, show great variations in their activity. Some contractions are slight and due to the participation of only a few fibres, others

stronger when more fibres participate in the contraction, while there are contractions in which most, if not all, fibres participate. These variations in the number of fibres taking part in the contraction are dependent upon impulses reaching the organ from nerve centres, and in these nerve centres there is the same variation in the number of cells taking part in discharging impulses. This principle is applicable to the relation between the auriculo-ventricular node and the ventricle.

THE CONTROL EXERCISED BY THE SINO-AURICULAR NODE ON THE AURICULO-VENTRICULAR NODE.

There is a curious and intimate relation between the sino-auricular node and the auriculo-ventricular node—the agents which influence the one influence the other so long as the sino-auricular node is active and dominant. They are both supplied by the sympathetic and vagal nerves (Fig. 2), and both are responsive to the influences of these nerves. When the sino-auricular node speeds up, the auriculo-ventricular node also increases its activity. When the sino-auricular node slows down, the auriculo-ventricular node also slows down. But there are instances when the auriculo-ventricular node may become affected by agents which do not affect the sino-auricular node, and then we obtain evidence of its presence. These changes may be very slight and give rise to little or no distinctive sign, or they may be so marked that the rhythm of the ventricle is affected. The simplest sign of an affection of the auriculo-ventricular node is a slight delay in the ventricular contraction after the auricular. This is best seen in a tracing of the jugular

pulse. The normal interval between the appearance of the *a* and *c* waves is one-fifth of a second. In a slight affection of the auriculo-ventricular node this interval is increased and frequently there results a failure of the node to respond to the impulses from the sino-auricular node. This is shown by the dropping out of a ventricular beat. A diagram (Fig. 7A) on the principle of the reflex arc brings out clearly where the disturbance arises which produces the irregular pulse in Fig. 7. It will be seen from this diagram that the ventricular beats (*Vs*) are irregular, and as these are due to the auriculo-ventricular node, it is shown also to be irregular. But when we look at what the auricle is doing it is seen to be acting quite regularly, so we can therefore recognize that the seat of disturbance is in the auriculo-ventricular node. The shading shows that the auriculo-ventricular node recovers its functional activity more slowly than the sino-auricular node, with the result that the impulses not only take longer to pass, but every now and again the auriculo-ventricular node does not respond to the auricular stimulus and a ventricular contraction drops out. (Compare with Fig. 4A.)

THE AURICLE IN PARALYSIS OF THE SINO-AURICULAR NODE.

When the sino-auricular node ceases to exercise its

influence on the auricle, the auricle passes into fibrillation or flutter. Looking at the auricle while fibrillating, it is seen to stand still as a whole, but the wall of the chamber is in incessant movement, due to rapid and repeated contractions of individual fibres. No sooner does a fibre cease to contract than it at once passes again into contraction—each one distinct and in-

dependent of its neighbour. The result is that, notwithstanding the incessant movement, there is no contraction of the chamber as a whole.

In place of this disorderly activity, some muscle cells may contract simultaneously, so that a rapid movement of the whole auricle occurs, to which MacWilliam gives the name of "flutter." The rate of this movement is about 300 per minute. There is good reason for suspecting that all the fibres do not participate in each of these movements, but I do not propose to deal with that matter here. Fibrillation and flutter are closely allied, and the one condition may pass into the other.

Lying in the auricular wall and exposed to the impulses from these muscle cells is that sensitive structure, the auriculo-ventricular node.

THE AURICULO-VENTRICULAR NODE IN PARALYSIS OF THE SINO-AURICULAR NODE.

When the sino-auricular node ceases to act a number of changes take place: the auricle passes into fibrillation; the normal impulses to which the auriculo-ventricular node is accustomed are no longer present, and instead there is a shower of impulses thrown out by the fibrillating auricle. Great numbers of these are without effect, and the auriculo-ventricular node only responds when sufficient time has elapsed after it has discharged its function to render it susceptible to such impulses. If we assume that these impulses are constant in number—and there are good reasons for this assumption—the peculiar variations in the activity of the ventricle can only be due to variations in the

FIG. 7.—A simultaneous tracing of the pulsation in the neck—from the jugular veins (waves *a*) and of the carotid pulse (waves *c*)—and of the radial in partial heart-block. The radial and carotid pulses are irregular, but the auricular waves (*a*) are quite regular in time; the reason of this is shown in Fig. 7A. The waves *a* will be seen to vary in height, and this is due to the fact that the auricle contracts at times during the diastole of the ventricle and produces the small waves, and during the systole of the ventricle, when they increase in height. This is shown by the gradual approach of the *c* wave to the auricular wave in the tracing from the neck.

FIG. 7A.—Diagram of tracing in Fig. 7, showing where the disturbance took place which caused the irregular pulse. The sino-auricular node causes an auricular beat at regular intervals (space *As*). The auriculo-ventricular node (space A.V.N.) is seen to vary in its periods of recovery, so that occasionally an auricular beat falls out, when the node is not susceptible. In consequence the ventricular systole drops out and the pulse becomes irregular.

excitability of the auriculo-ventricular node. We have thus an extraordinary opportunity of studying a series of vital processes, and the manner in which they can be affected by a variety of circumstances, including the influence of nerves, of the agents of disease, and of remedies.

There is something peculiar about the auriculo-ventricular node. As I have said, while it is under the dominance of the sino-auricular node its peculiarities are hidden. I remember that when first I read Tawara's account of this node I was reminded of Gaskell's description of the primitive cardiac tube.⁸ In the canalis auricularis of the tortoise there is a ring of muscular fibres of a peculiarly sensitive kind. Gaskell describes the manner of exploring this ring as follows:

"Slit up one auricle and the ventricle, and pin back so as to expose the septum; the two vagi nerves on the septum, with their terminations in Bidder's ganglia, are then most easily seen, as well as the muscular ring between auricles and ventricle, and the whole field can be explored, under a lens, with the point of a needle before and after removal of the septum and Bidder's ganglia. Now, cut away the sinus, the whole preparation remains quiescent; touch the outlying auricular or ventricular tissue with the needle point, each time a single contraction takes place, and no more; touch either of Bidder's ganglia, not a single contraction takes place; pierce it again and again, and put it about, no sign of contraction. *Touch the auriculo-ventricular ring of muscle with the slightest stimulus, immediately a series of rhythmical contractions occurs. It is most striking to see, after removal of the septum, how every portion of auricular and ventricular tissue can be explored up to the very edge of the ring, without obtaining more than a single contraction, while immediately the needle touches the muscular ring a series of rapid contractions results.* Conversely, the two Bidder's ganglia can be cut away, and yet stimulation of the ring will produce a rhythmical result."

I have assumed that this muscular ring is the progenitor of the auriculo-ventricular node, and I was reminded of this fact when I investigated the behaviour of the auriculo-ventricular node in auricular fibrillation. In this condition the auriculo-ventricular node becomes extraordinarily susceptible to stimulation. In some cases, on the onset of fibrillation of the auricle, the rate of the ventricle may reach a speed of 150 to 160 beats a minute. This rate may be kept up for days and weeks; usually it slows down, sometimes very considerably, but when it has slowed down, even below the normal rate, the slightest effort may cause an increase in rate from 50 to 160 a minute, an increase of rate far greater than that which occurs with the normal rhythm.

Striking as is this excitability, just as striking is the manner in which this excitability becomes diminished. Agents which have an effect in depressing or inhibiting this activity have far more effect than upon either the sino-auricular node or the auriculo-ventricular node with the normal rhythm. It has been shown that bodily effort, by setting up impulses which are conveyed to the heart by the sympathetic nerves, increase the rate, while the cessation of effort causes a fall in rate. This fall in rate may become greater in auricular fibrillation than in the normal rhythm, so that bodily rest may be accompanied by a remarkable slowing of the pulse. Digitalis, which usually has little or no effect in slowing the heart when the sino-auricular node is active, readily acts upon the auriculo-ventricular node in auricular fibrillation—to such an extent that the rate of the pulse may fall as low as 40 or 30 beats a minute.

This susceptibility to digitalis is brought strikingly out in those cases in which the normal rhythm is restored while the heart is under digitalis. During auricular fibrillation the ventricular rate may be 40 a minute, but as soon as the normal rhythm is established the rate may rise to 60 or 70 beats a minute, and although digitalis may be continued in large quantities no effect may be produced in the pulse rate.

THE LOSS OF CONTROL.

These results demonstrate that something more has happened to the heart than the onset of auricular fibrillation. The susceptibility of the node has become enormously increased, both in a positive and negative sense. The question arises, What is the reason for this curious alteration? It was the recognition of this which led me to suggest that

the higher levels of the regulating processes have an influence beyond merely conveying impulses which stimulate the cells into activity. There is some influence which keeps them from responding to other stimulation, and restrains and regulates their activity so that they all work harmoniously.

There are several instances in nature which suggest this influence. When the muscles of the body are removed from the influence of the cortex, as in the decerebrate animal, the muscles tend to pass into the state of rigidity. This is due to their having become much more sensitive to impulses which reach them from other parts of the body.

It was because of this increased susceptibility to stimulation that Sherrington employed the decerebrate animal for his investigations into muscle reflexes. With the brain intact he found the muscles so restrained that he could not get the same satisfactory results.

We have seen in the description of the experiments of the medusa that the sensitive bodies at the margin exercise a controlling influence which is lost when a ring of muscle is separated from the body.

It is to be noted in Gaskell's description that before he demonstrated the excitability of the auriculo-ventricular muscular ring he removed it from the influence of the sinus, which he cut away. This excitability of the auriculo-ventricular node is only shown when it is likewise removed from the influence of the sinus—that is, the sino-auricular node.

REFERENCES.

- ⁶ Withering, W.: *Account of the Foxglove*, 1785.
⁷ Mayer, A. G.: *Rhythmical Pulsation in Scyphomedusae*. Papers from the Tortugas Laboratory. Carnegie Institution of Washington, publication No. 102, vol. i.
⁸ Gaskell, W. H.: The Contraction of Heart Muscle. Article in Schafer's *Textbook of Physiology*.

(To be continued.)

Memoranda : MEDICAL, SURGICAL, OBSTETRICAL.

A NEW SKIN FORCEPS.

I VENTURE to submit to surgeons a novel idea in forceps for skin suture, illustrated in the accompanying figures. The object is to secure full eversion of the edges of the skin in closing a wound. The forceps have strong blades, deeply serrated on the inner surface near the ends, to secure a firm hold on the skin and prevent slipping. The ends of the forceps, as shown in the figure, are bent at an angle to the blades, and on the under edge are three strong teeth. On applying the forceps fairly close to the edges of the

skin, one on each side, and pressing the blades firmly together, the edges themselves are evenly everted, and an assistant can run either a straight or curved needle through both lips of the wound, and tie the suture, while the surgeon holds them in the everted position; or the assistant can apply the forceps and the surgeon, if he prefers it, pass the needle. The second figure illustrates the forceps in operation. They have stood the test of experience, and furnish a very ready means of closing a wound without any inversion of its edges, and without having to pick up each edge separately.*

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* The forceps have been made for me by Messrs. Down Bros.