Observations

ON

THE PROCESS WHICH RESULTS IN AURICULAR FIBRILLATION.

BY

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It is just twenty-five years since the condition now called "auricular fibrillation" was recognized as a clinical entity. Its study in the human subject threw a new light upon a great number of heart problems. It was found, for instance, that this abnormal state resulted in the greatly increased susceptibility of the heart to stimulation not only by agents which increase the rate of the ventricle but also by agents which diminish the rate. It was found also that the heart reacted to drugs of the digitalis group with a greater readiness than does the normal acting heart. Heart failure is more readily induced, and the character of the heart failure is peculiar, differing greatly from most of the forms of heart failure which occur when the rhythm is normal.

Distinctive as these features are, the condition is of importance from another point of view as it represents a form of abnormal activity which is common to other organs and which holds out the promise of revealing the nature of abnormal states which have hitherto baffled inquiry. This aspect of the subject has been obscured by the unfortunate use of the term "fibrillation," for a better understanding of the subject shows that fibrillation of the auricle is but one of a number of incidents that follow the inception of the new condition. In this respect the use of the term has but followed the traditional method in medicine of calling the diseased condition by its more prominent symptom. In this case the use of the term has hampered progress, as it gives the impression that fibrillation is the cause of the condition, and so has led to attention being concentrated on what is but one incident.

The Loss of Control.

There seems to be some law or principle which governs the functions of the organs of the body by which they are controlled by higher agencies. When this control is lost the functions of the organs become disturbed and their activities greatly modified. The phenomena produced by this disturbance vary with the organ affected. In an article recently published in this JOURNAL I suggested that the sino-auricular node and the conducting system exercised control over the muscles of the auricle and ventricles, and that the condition called auricular fibrillation was due to a loss of this control. Further research on these lines supports this view.

Retrospect.

In order to present the subject comprehensively, I give here a short review of the manner in which this condition was brought to light. It is now nearly forty years since I began an endeavour to solve the problem of pulse irregularity. Before I undertook this work many attempts had been made, with very little success. For a time I could make no progress, having no definite principle to guide me. After some years I recognized that in the study of irregularities, whether by the unaided senses or by the sphygmograph, it was only the left ventricle that was being studied. It occurred to me to inquire what the auricles were doing when the ventricle was irregular. The answer to this simple question revealed the principle which guided a research which has been successful in differentiating the great majority of irregularities. Means were devised by which the movements of the auricle could be registered at the same time as the radial pulse, so that it was possible to obtain simultaneous movements of auricle and ventricle. In this way a method was found by which we were able to differentiate most forms of irregularity.

During this time I was also watching coincident changes in the heart and their relation to heart failure. One patient with mitral stenosis I had been watching for about seventeen years. One day in 1897 her whole circulatory system suddenly underwent a change. The heart, which was perfectly regular before, became rapid and very irregular, pulsations in the veins and in the liver, due to the contraction of the auricle, disappeared, as well as the presystolic murmur, which had been unceasingly present for a great many years. At the same time there were signs of extreme heart failure—great breathlessness, blueness of the face and lips, swelling of the ankles, and enlargement of the liver. On account of the disappearance of all evidences of auricular activity I assumed that the ventricle had taken on the rhythm of the heart, and that the auricle had become paralysed, and in a book on the pulse published in 1902 I described this condition as "auricular paralysis."

I found among my patients others in a similar way, and so I came to recognize the condition as a distinct clinical entity.

I watched the progress of these cases with particular reference to heart failure, and sought for the best methods of treating them, and in the course of this investigation I discovered the peculiar susceptibility of these hearts to the action of digitalis.

At the *post-mortem* examinations of the first few cases the walls of the auricle were so enormously distended and thinned that my idea of auricular paralysis seemed confirmed. But ten years later I had a *post-mortem* examination of a patient whom I had attended when the heart was normal in its action, and I witnessed the onset of this auricular "paralysis," and watched her progress seven years afterwards till she died. At the *post-mortem* examination the auricles were not dilated and atrophied, but seemed to be somewhat hypertrophied. I therefore concluded that if the auricle were hypertrophied it could not have been paralysed. This altered my conception, and I reasoned that the heart's contraction may have started at the auriculo-ventricular node, and that both auricle and ventricle had contracted together, and so called the condition "nodal rhythm."

In 1906 Cushny and Edmonds described the resemblance of the irregularity of the pulse in this condition with that of a dog's in whom they had produced auricular fibrillation, and suggested that both irregularities were due to the same cause. This view was confirmed in 1909 by Lewis, and from that time the term "auricular fibrillation" has been applied to this condition.

Continuing my observations on this condition I became gradually conscious that there was more in the matter than the auricle passing into fibrillation; but although I had studied a very great number of cases I could not see my way clearly to make any further progress until we began our systematic research in St. Andrews. Our methods of investigation led us to inquire into the mechanism of symptoms, and when we looked at the peculiar group of symptoms which resulted from this condition we were led to inquire into the nature of the stimulus which started the heart's contraction.

We considered the manner in which the stimulus for contraction arose in the normal heart; Keith and Flack had shown that it arose in a tissue specially developed for that purpose—the sino-auricular node, which is probably evolved from the sinus venosus of the lower animals. Accepting this view, we recognized that in auricular fibrillation this node is out of action and the stimulus for the contraction of the heart is produced in the fibres of the auricular muscle. Here, each fibre in its contraction stimulates its neighbours, and in this way an incessant movement is carried on by these fibres, and from this movement impulses are discharged which affect the auriculo-ventricular node. This node is in close relation to the auricle and from it a stimulus for contraction passes to the ventricle by the auriculo-ventricular bundle (see Fig. 1).

It will be seen that the whole fibrillating auricle is the source of the stimulus in this condition, while in the normal acting heart a specially evolved tissue of a minute size is the source of the stimulus. We can reasonably infer that the nature of the stimulus must be different in the two cases, and

FIG. 1.—Diagram representing the two main systems concerned in the heart beat. (1) The conducting system, represented by the sino-auricular node (S.A.N.) and the auriculo-ventricular node (A.V.N.) and bundle (A.V.B.). In both auricles and ventricles are also Purkinje fibres which belong to this system, represented by the scattered cells (P) in the auricle. The vagus (vag.) and sympathetic (sy.) also belong to this system. (2) The contracting system, represented by the muscle cells of the auricle (A) and ventricle (V).

we find evidences of this difference in the response of the ventricle to contraction.

Looking at the matter in this way, it seemed that the fibrillation of the auricle was the result of a process and not the process itself. The contraction of the auricle and ventricle are normally under the control of the sino-auricular node and other portions of the conducting system, and it seemed that the auricle passing into fibrillation was due to a loss of this control. The cause of the condition thus was not to be found in the study of the fibrillating auricle but either in the sino auricular node or its connexion with the auricle.

Langley's Observations on the Denervated Muscle.

One of the principles which guide us in our research is that when we find certain phenomena produced by one organ the mechanism of which is obscure we turn to the examination of other organs to see if similar phenomena are exhibited by them. We accordingly turned our attention to other organs of the body. In this search we recognized that while the mechanism which produced the phenomena would be the same, the phenomena themselves would be different, and would correspond to the function of the organ disturbed. A number of examples were revealed which seemed to favour this view, and to these I shall refer later. It was while on this quest I met Sir Edward Sharpey Schafer and discussed the problem with him, and he called my attention to some experiments by Langley which I had overlooked. Langley¹ had found that on the division of the motor nerve of a voluntary muscle the paralysed muscle which resulted therefrom was not an inert body, but that sooner or later the fibrils of the muscle showed constant movement. In other words, fibrillation of the muscle resulted when it was uncontrolled by the motor nerve.

There would thus seem to be a law which governs the activity of muscles, in that so long as a muscle is under control of the nervous system fibrillation does not occur. In the case of the ordinary muscles it is the nerves which exercise the control. The same law holds good for the heart, but in the case of the heart muscle it is the sino-auricular node and conducting system which exercise the control.

Difference between a Fibrillating Voluntary Muscle and a Fibrillating Auricle.

The voluntary muscle is not connected with any other organ, consequently no evidence of the fibrillation can be detected unless the muscle is exposed to view. With the auricles, however, the condition is different. They are hollow muscles and contain fluid, the slight movements of the wall are communicated to the fluid, and it is for this reason we can sometimes detect fibrillary waves in the jugular vein during auricular fibrillation. Moreover, the auricles are in direct communication with the very sensitive auriculo-ventricular node (Fig. 1), and therefore the impulses which arise from their movement act upon this node, which, being stimulated, sends impulses to the ventricle, and there results that peculiar irregularity of the ventricle which is characteristic of this condition.

Rhythmical Movements in the Fibrillating Muscle.

The minute study of fibrillation in the denervated muscle reveals that at times there are rhythmical movements which resemble flutter or the more rapid movements that are sometimes present in fibrillation of the auricle. Langley, in describing fibrillation in the voluntary muscle, states: "In close examination it is seen that the contraction of any one spot—as a rule, at any rate—has a perfectly regular rhythm, and that the apparently continuous movement of the surface as a whole is mainly due to the rhythm in adjoining fibres being different. The rate of movement and the intensity of contraction vary considerably in different parts of the muscle."

The Results on the Heart of Loss of Control.

When this loss of control occurs the heart becomes a different organ in respect of the way in which it maintains the circulation and of the manner in which it responds to outside stimulation and to remedies. The auriculo-ventricular node and the bundle become much more sensitive to stimulation, while the ventricle contracts in a manner which it never does when under the control of the conducting system. I have already dwelt upon the fact that the varying size of the pulse beats which occur so frequently in auricular fibrillation would seem to imply that all the fibres of the ventricle do not participate in the smaller contractions.

I had reasoned that this is probably due to the difference in the stimulus, but it is quite possible that it may be due to an altered condition of the ventricle when it is removed from the control of the sino-auricular node.

Circumstances that cause Loss of Control.

These views are mainly suggestive, and intended to indicate the lines on which further investigation should be pursued. The investigation of this subject should be devoted to the consideration of the conditions which give rise to it. The change manifestly takes place in the sino-auricular node or in its connexion with the auricle. This is brought out particularly well from the consideration of some of the circumstances which induce this loss of control.

Many years ago I drew attention to the fact that digitalis acted upon the auriculo-ventricular node and bundle, causing sometimes the blocking of the stimulus from the auricle, so that ventricular beats dropped out. In 1906 I pointed out that this was due to the action on the vagus. This evidence was obtained from a man in whom digitalis caused a dropping out of ventricular beats. When the effect of the digitalis was passing off this action disappeared, but the patient could bring it back at once by the act of swallowing, a procedure which could only produce an effect by acting through the vagus.

I have recorded an account² of another patient in whom digitalis showed an instructive series of responses. The digitalis caused slowing of the whole heart for short periods—that is, the drug acted on the sino-auricular node, and the auricle and ventricle contracted at a slow rate. During some of these periods the standstill was so long that the ventricle contracted independently of the auricle. At another period the auricle passed into fibrillation—in other words, under the vagal influence the sino-auricular node ceased for a time to act, so that the ventricle contracted independently, and at another time the auricle, escaping from the control of the sino-auricular node, passed into fibrillation.

This observation throws a light upon certain experiments of Lewis and his colleagues. In one experiment³ they stimulated the vagus nerve and took an electrocardiograph record of the auricular contraction. The record shows at first a great slowing or standstill of the auricle, followed by a sudden and enormous increase of the auricular rate up to 1,200 beats a minute. It seems almost certain that this great increase in rate was due to the auricle passing into fibrillation or into some other abnormal state in consequence of the suspension of the action of the sino-auricular node, just as happened in the patient under digitalis. It should be borne in mind that in stimulating the vagus nerve the slowing of the heart is due to the action of the nerve upon this node, and that as the node ceases to act a condition equivalent to cutting the nerve to a muscle results, and the auricle passes into fibrillation. Lewis, therefore, is scarcely justified in drawing the conclusion that the action of the vagus is to re-excite the auricular muscle and produce a shortening of the refractory period.

The importance of looking to the loss of control of the sino-auricular node as the state in which auricular fibrillation occurs directs attention to the immediate cause of the condition. It is manifestly vain to seek for the cause of paralysis of a muscle whose nerve is cut in the muscle itself, and it is therefore equally vain to seek for the cause of auricular fibrillation in the study of the movements of the fibrillating muscle. The mere fact that in auricular fibrillation there is a short refractory period does not justify the assumption that the shortened refractory period is the cause of auricular fibrillation. Trying to stop fibrillation by attempting to lengthen the refractory period is manifestly a procedure which is bound to fail, and is due to a misconception of the nature of the condition.

The Loss of Control of the Ventricle.

What happens when the auricle escapes from the control of the sino-auricular node also happens when the ventricle escapes from the control of the conducting system. This part of the subject has so far been very imperfectly investigated. At least two peculiar forms of ventricular contraction occur—one shown by a series of small beats apparently of the same origin as ventricular extra-systoles, another when the ventricle fibrillates and death occurs.

The Loss of Control in other Organs.

In the search for a similar condition in other organs I looked at the muscular system, and I find some very suggestive comparisons. Recently, while watching an operation for the removal of a small tumour from the vocal cords

by my friend Dr. Douglas Guthrie, I was struck by a peculiar movement in the neck. The patient had been for a short time under chloroform, and I observed what seemed to be the pulsation of a vein, which extended from the left clavicle near its inner end half-way up the neck. The movement was perfectly regular at 60 beats a minute. When I felt the pulse and watched the veins in the neck I discovered that there was no relation between this curious beating and the arterial and venous pulse, the latter beating about 80 a minute, with frequent intermissions due to extra-systoles. Looking more closely at the movement and feeling it with the finger, it became evident that it was due to a few fibres of the platysma muscle. These fibres were about an eighth of an inch across and appeared to be attached to the skin over the clavicle at the lower end, and they extended up the neck for three or four inches and caused a movement of the skin at the level of the thyroid cartilage. There was also a smaller movement of the same kind at the lower end, about an inch behind the larger movement. These movements continued for fully an hour—in fact, during the whole time the patient was under chloroform. As soon as he came out of the chloroform the movement ceased.

It is very likely that the paralysis of certain muscles, as in diphtheritic paralysis, is really due to this loss of control. Dr. Maitland Ramsay tells me that the ciliary muscle in certain cases of errors of refraction may suddenly cease to act, and it occurs to him that probably the same condition is present here. It is well known that in dangerous *post-partum* flooding the uterine muscle is in a state of extraordinary laxity. Dr. Orr has suggested that when this happens the uterine muscle is probably in a similar condition. The circumstances certainly favour its occurrence, for experience has shown that a common factor in the production of auricular fibrillation is great bodily effort; and the observation of Dr. Ramsay points the same way, in that the ciliary muscle ceases to act after the strain in attempting to correct the error of refraction, and the uterus has just ceased from a severe strain shortly before flooding occurs.

That other organs may show the same peculiarity seems reasonable. Loss of control results in the cells taking on an individual action and stimulating one another. Professor Herring has called my attention to the fact that when the nerve to the salivary gland is cut the gland is not inactive but continues to secrete, and this secretion is spoken of as "paralytic."

Probably the condition of epilepsy affords another illustration. It is manifest that in this state there must be the same direct cell to cell stimulation as occurs in the fibrillating muscle. As we watch the onset of an attack of epilepsy we observe that the finer movements of expression or of the fingers are first affected, and from here the attack spreads to all the muscles of the body. It is evident that no other cause but the disturbance of the cellular elements in a localized area of the brain can produce these effects, and it is likely that here also the condition is due to a loss of control of some higher centre. The same explanation probably accounts for migraine attacks.

These views are not put forward as if they were established, but merely to provide a guide for further investigation. They are sufficient to justify the suggestion that we have here an abnormal process which is common to a great many organs, and that the aim of the investigator should be to find out the nature of the process rather than to study its result.

Terminology.

Though the term "auricular fibrillation" is misleading, it has gained such a wide acceptance that for the present it is better it should be retained. It would be more appropriate to describe the condition by the process which produces it, and in a sense the original term "auricular paralysis" might be employed, seeing that it resembles the paralysed muscle. But as that term does not clearly describe the process the change of term would avail little. The important consideration is that the fibrillating auricle is but one result of a vital process, as yet not fully determined.

REFERENCES.

- 1 "Fibrillation noticed during a Research on the Action of Physostigmine on Denervated Skeletal Muscle," Langley and Kato, *Journ. of Physiol.*, 1914-15, vol. 48, p. 425 et seq. "Observations on Denervated Muscle," J. N. Langley, *Ibid.*, 1915-16, vol. 50, pp. 335-342. "Observations on Denervated and on Regenerating Muscle," J. N. Langley, *Ibid.*, 1917, vol. 51, pp. 390-393. "Observations on the Atrophy of Degenerated Muscle," Langley and Hashimoto, *Ibid.*, 1918-19, vol. 52, pp. 43-45 and 62 et seq.

² Mackenzie, J.: *Diseases of the Heart*, p. 465, third edition.

³ Lewis, Drury, and Iliescu: *Heart*, vol. viii, p. 331, Fig. 5.

Remarks

ON

BONE GRAFTING AS AN AID IN TREATMENT OF TUBERCULOUS SPINAL CARIES.

[With Special Plate.]

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THE use of bone grafting as an aid in the treatment of tuberculous spinal caries is of comparatively recent origin. It was introduced by Hibbs and Albee, and naturally, in view of the reputation of the originators, was tried at once by those who sought more satisfactory methods of treating Pott's disease than were previously known. As is usual in such cases, strong opposition has been provoked in many quarters, and it is therefore not surprising that a distorted conception of the value of the treatment exists at present in the profession. We have to attempt to sift out the truth from the apparently contradictory views expressed on the subject. When, from reports by reputable observers and from my own experience, the facts become established that cases of Pott's disease of the spine are enabled to return to a normal mode of life in a shorter period of time after this than after any other previously adopted method of treatment, and that such cases maintain their capacity without recurrence of the trouble over periods of many years, I am forced to believe that this operation has a useful place in the therapy of spinal caries and to seek to define the cases in which it may be applied with good prospects of success.

GENERAL PRINCIPLES IN THE TREATMENT OF TUBERCULOUS DISEASE.

The present-day conception of the rational treatment of tuberculous disease of any tissue may be briefly stated as follows:

If there is reasonable prospect of attaining complete eradication of the disease without maiming the patient most surgeons prefer to operate in order to procure this end, in spite of the fact that radio-therapists and other advocates of conservative treatment claim excellent results from their treatment. The help of the radio-therapist, however, is usually also invoked by surgeons in order to make cure as certain as possible—that is, such cases are treated on the same plan as cases of accessible cancer. If tuberculous disease cannot be eradicated surgically without interfering materially with the capacity of the patient to lead a healthy, active life, conservative methods of treatment are tried, and are often successful. The chief drawback to such forms of treatment is that they take a long time when compared with surgical ablation of the disease. In young children this is not of the same importance as in older children and adults. Generally speaking, however, the best remedy is that which most rapidly brings the disease to a standstill and is followed by cure. No form of treatment apparently can ensure that the disease will not break out at any time in other parts of the body.

Whether "conservative" or surgical methods of local treatment are used, all are agreed that general treatment, providing suitable diet, good hygienic surroundings, open air, sun baths, possibly vaccine or other inoculations, etc., is also of the greatest importance.

Local treatment aims at producing rest of the affected part. When bones or joints are concerned, fixation in the best possible way is established. Fixation is essential to success. The sooner all such measures are carried out in an efficient manner the greater is the prospect of cure.

APPLICATION OF GENERAL PRINCIPLES TO THE TREATMENT OF SPINAL CARIES.

It is obvious that disease of the bodies of the vertebrae cannot be attacked surgically in the satisfactory way in which a focus, for example, in the tibia can be dealt with. At best the occasional removal of sequestra is all that has been attained, coupled, perhaps, with more or less inadequate scraping of the surrounding affected bone; even such measures are attended with considerable risk to the patient. It is evident therefore that Pott's disease must be treated expectantly so far as the actual *focus mali* is concerned, and