tubercle bacillus which is best described by the old-fashioned phrase "the tendency to consumption." In what way and to what extent this blood defect may be salutary as regards tuberculous lung disease, we can only conjecture. My own idea is that it may be by inducing an air hunger, and so stimulating lung function and nutrition. Anyhow, when we reflect that the quality of the blood is quite good, we can understand that treatment to improve the health generally rather than the blood specially is what is required.

THE AUTOMATIC RHYTHM OF THE HEART.

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THE prolonged controversy during the last quarter of a century as to the origin of the automatic power of the heart to beat rhythmically has received marked stimulation during the last few years. Leaving aside the earlier hypotheses, one of which attributed the beats to impulses sent from the central nervous system, while another regarded distension of a cardiac chamber by blood as the exciting cause of its systole, there are left two opposing theories, both of which recognize that the automatic power of the heart lies in itself, and is quite apart from the central nervous system or the circulation of the blood. The researches of Gaskell, Engelmann, and others have given us certain facts which it is the province of the theories to explain, although these facts are sometimes lost sight of in the heat of discussion. These facts are based, of course, on the examination of vertebrate hearts in general; and it is the discovery that they are also true of human hearts which constitutes the important advance in clinical knowledge that has followed the application of physiological methods by Mackenzie¹ and Wenckebach² in this particular branch of practical medicine. The theoretical explanation of these facts is relatively unimportant although it is of great interest.

In considering any theory it is necessary to assume that the heart possesses certain properties in order to account for the phenomenon of its rhythmical beat, but many of these properties are also possessed by the undifferentiated protoplasm of the lowest forms of life. In the first place it must respond to stimulation, a property which is really divisible into two: (1) excitability, and (2) in this muscular organ, contractility. Since the heart is capable of beating when separated from all external stimuli, it must possess its own stimulus which excites it to contract. Experimentally it has been found that an artificial stimulus is conducted in all directions in the heart from the point stimulated. Consequently the heart owns the further property of conductivity. Like all muscle it also exhibits tone, and during life is never completely flaccid. Thus the heart has these five properties: a stimulus of its own, excitability, conductivity, contractility and tonicity. The point at which theoretical controversy arises is over the question, "In what anatomical components of the heart are these various properties seated?" In the heart we find a special kind of muscular tissue, nerve cells and fibres and connective tissue with blood vessels, etc. It is evident, therefore, that the foregoing properties must be situated either in the nervous or the muscular elements. Some observers attribute them almost entirely to the first, whilst others attribute them absolutely to the second. It is, of course, possible that they may be divided between the two tissues, and it is necessary, therefore, to consider each property individually and to weigh the evidence before assigning it to the one or the other.

Contractility, or the power of contracting, is evidently a function of the muscle fibres, and does not need further discussion. Muscle fibres do not contract unless they are stimulated, and in order to receive the stimulus they must be excitable. Ordinary striped muscle may be excited through its nerve fibres, and it has been contended that both it and the cardiac muscle fibres can only be excited through nerve fibres. The cardiac muscle cells are surrounded by a very close network of non-medullated nerve fibres, as can be demonstrated by modern methods of staining.³ We now know also, contrary to what was previously believed, that, in mammals at any rate, a quiescent heart may be made to respond with a contraction to each stimulus sent to it by the accelerator nerve.⁴

It has been found impossible to eliminate by means of poisons the possible action of the accelerator nerve endings in cardiac muscle without seriously injuring the muscle itself. On the other hand, nerve fibres, when separated from their cells of origin, degenerate and in time become functionless, and there is no reason to suppose that the cardiac nerve fibres differ from others. Although new methods of staining have shown that ganglion cells are more numerous in the heart than was at one time supposed, yet no ganglion cells have been found in the apex of the frog's ventricle; and if this part be separated from the rest of the heart, the nerve fibres may be assumed to degenerate in a few days and become functionless. It has been found possible to keep such an apex preparation for twenty-one days or more in a condition in which it will respond by a contraction to a direct stimulus. Further, it has been found that certain stimuli, such as ammonia or weak acids, excite ordinary skeletal muscular tissue, but not nerves; while others, such as glycerine, excite nerves, but not muscle. The apex of the frog's heart is excited by ammonia and weak acids, but not by glycerine.⁵ It is thus evident that the cardiac muscle possesses the property of excitability as well as contractility, quite independently of the nerves with which it is surrounded.

It has already been pointed out that if the heart be stimulated artificially at any point, the stimulus is conducted in all directions from the point stimulated. This may be explained in three ways. It was at one time suggested that the stimulus excited nerve endings which transmitted it to the ganglion cells, which in their turn excited the muscle tissue, but there is no convincing evidence of any such reflex action, and the regular progression of the contraction wave from the spot stimulated weighs strongly against any such hypothesis. A direct conduction of the stimulus may occur through the muscle tissue or by means of the all pervading plexus of nerve fibres. Although nerves have the power of conducting stimuli in both directions, the terminations of nerves are not the same at each end. It is only one end which is connected with a responsive tissue. Practically, therefore, effective stimuli which produce tangible results only pass along nerve fibres in one direction. In the heart, however, stimuli pass in all directions—as readily from ventricle to auricle as from auricle to ventricle, a phenomenon which is somewhat difficult to explain if the conduction be through nerve fibres. In favour of the neurogenic theory it is contended that facts derived from the study of isolated nerve fibres are not applicable to a nerve plexus, for a nerve plexus is a very complex structure whose properties are little known. Engelmann⁶ attempted to solve the problem by the study of the rate of conduction in the heart, which he found to be 300 times less than the rate of conduction in the frog's motor nerves and to be remarkably influenced by bleeding, which has but small influence on the rate of conduction in nerve fibres. Here, again, it has been contended that arguments drawn from isolated nerve fibres cannot be applied to the nerve plexus in the heart; and Carlson,⁷ working with the heart of *Limulus*, where the heart nerve fibres can be isolated, found the rate of conduction in them eight to ten times less than in the motor nerves of the same animal.* A stronger argument against the neurogenic theory is afforded by Engelmann's⁸ zigzag experiments, which showed that a stimulus was conducted from end to end of one piece of cardiac muscle tissue, which had been cut into an irregular zigzag shape and in which every nerve fibre had probably been cut across at least once.

During a contraction the heart is refractory to further stimulation, and this refractory period has been shown to be due to the destruction, or at any rate the great diminution, of its contractility, excitability, and conductivity. It is the contraction that brings about this change, since a stimulus that is ineffective in producing a contraction does not alter these properties. After a contraction it can be shown that contractility, excitability, and

* It is to be noted that in this experiment the nerve fibres were separated from the ganglion, and that in another place (*Amer. Journ. of Physiol.*, xlii, 1905, p. 217) Carlson states that separation from the ganglion results in depression of conductivity. The evidence of depression relied on, however, is that after removal of the ganglion a local stimulation of the heart is often followed only by a local contraction not spreading all over the heart. The local character of this contraction might be explained by the depression of excitability and contractility which, as Carlson has shown, follows removal of the ganglion.

conductivity are slowly recovered. It seems unlikely that this remarkable effect would be produced on these properties by the contraction of a muscle fibre if the properties were situated outside that muscle fibre. A similar inference may be drawn from the influence of alterations of tone on conductivity. Tone may be dependent on the nervous system, but is essentially a property or condition of the muscle fibres. In the early part of 1906,⁹ I suggested, largely on theoretical grounds, that increase of tone had the effect of diminishing the other properties of the cardiac tissue, and about the same time Porter¹⁰ demonstrated experimentally that increase of tone directly diminishes conductivity. One would not expect this result unless conductivity were located inside, not outside, the muscle fibres. Moreover, just as with contractility and excitability, there is direct evidence that conductivity is possessed by the muscle apart from the nerves. In the separated apex of the frog's ventricle a contraction can still be obtained on stimulation even after the lapse of twenty-one days, when all the nerve fibres should be degenerated, and the stimulus is conducted to all parts of the preparation.

There is thus conclusive evidence that the muscle fibres possess the properties of contractility, excitability and conductivity; and the various phenomena of the active heart can be explained by assuming that the stimulus is conducted through muscle, and that the muscle is excited directly without the intervention of nerves. This, of course, is no proof that under normal circumstances the conduction and stimulation do not take place through nerves, but since the muscular channel is sufficient, the proof of the wholly unnecessary nervous interference should come from those who support the neurogenic theory.

The question of the origin of the intrinsic stimulus is, however, in an entirely different category. We have no direct evidence that muscle fibres elsewhere can contract except under the influence of an external stimulus. It seems probable that the peristaltic contraction of the ureter, contrary to Engelmann's opinion,¹¹ depends on the nervous system, while it is almost certain that the intestinal movements depend on the intrinsic nervous mechanism. It is true that muscular tissue will contract rhythmically under the influence of salt solution when separated from nerve cells (for example, the apex of the frog's ventricle or a piece of striped muscle), but under these circumstances the salt solution in all probability acts as a constant stimulus to the muscle. Carlson¹² has recently shown that in the heart of one animal at least—*Limulus*, an invertebrate—the power of automatically beating depends on the nerve cells in a ganglion. In this animal the ganglion is quite separate from the heart muscle, as are also the larger nerve fibres. On removal of the ganglion the heart ceases to beat although it responds by a single contraction to a direct stimulus. There are also inhibitory and accelerator nerves which apparently act on the cardiac muscle through the ganglion cells. It seems also certain that the lymph hearts found in frogs and certain other animals depend for their automatic action on their connexion with the nervous system.¹³ If we are to suppose that all these similar organs act in exactly the same way the question as to the neurogenic origin of the heart stimulus is settled. The application of facts, drawn from the study of the invertebrates, to the vertebrates has, however, several times proved to be fallacious, so that a further difference between them is quite possible. Moreover, on carefully examining the phenomena exhibited by the heart of *Limulus*, it is found that in several respects it does not correspond with the vertebrate heart. The heart of *Limulus* is a large organ 15 to 20 cm. long and 2½ cm. broad, and yet under ordinary circumstances it appears to contract simultaneously in all its parts.¹⁴ It is true that, with tired hearts, the contraction can be seen to start at the posterior end and to travel to the anterior. Further, this heart consists only of a single chamber. The character of its beat differs very strikingly from the orderly procession of contraction of the great veins, auricles, and ventricles, which we can see in the vertebrate heart. Such a beat as that of the heart of *Limulus* could not possibly be co-ordinated and conducted by the muscle tissue alone, and, as a matter of fact, Carlson shows that the conduction and co-ordination of the normal beat takes place in the ganglionic cord. Cross-section of the heart itself does not interfere either with the conduction of the stimulus or the

co-ordination of the heart beat, whereas section of the appropriate nerves or of the ganglion cord destroys conduction and co-ordination. This peculiar action of the heart, no doubt, is suited to the requirements of the animal, and it is not unreasonable to suppose a special mechanism is available to bring it about. The refractory period, which is such a marked feature of the muscular contraction of the vertebrate heart, is less conspicuous in the heart of *Limulus*, so that this heart can easily be tetanized. This also applies to the lymph hearts. In the vertebrate heart the contraction does not depend on the strength of the stimulus, but provided that the stimulus is sufficient to produce a contraction at all, then the contraction is the largest one of which the heart is at that moment capable, and this is also true of an isolated apex preparation. This "all or nothing" law does not apply to lymph hearts, where the contraction varies directly with the strength of the stimulus,¹⁵ as in ordinary striped muscle, and only applies to a limited extent to the heart of *Limulus*, and then only when the ganglion is intact. On stimulation of the ventricle of a mammalian heart, an extra-systole can be produced which is followed by a pause—the compensatory pause—which is due to the ventricle having to await the next stimulus from the veins before it again contracts. Extra-systoles can be produced by direct stimulation in lymph hearts, but they are not followed by any compensatory pause.¹⁶ This aspect of the extra-systole does not yet seem to have been worked out in *Limulus*, but in the invertebrate *Palimurus*,¹⁷ the extra-systole, even when the stimulus occurs late in diastole, has apparently no compensatory pause, while other invertebrates, such as *Heliotis* and *Aplysia*, show a well-marked compensatory pause.*

These remarkable differences justify one in not accepting the proved neurogenic origin of the heart beat in *Limulus* as necessarily involving a similar neurogenic origin for the beat of the vertebrate heart, although, of course, a strong argument is afforded in favour of this view.

Engelmann¹⁸ showed that the isolated ventricle of the frog's heart beats rhythmically under the influence of a constant stimulus, a contraction taking place whenever the excitability had sufficiently recovered after the previous beat. In such a ventricle preparation an extra-systole may be produced by applying another momentary stimulus, stronger than the constant stimulus, during the quiescent period. Such an extra-systole is not followed by a compensatory pause. But if an intermittent stimulus be used to cause the apex preparation to beat rhythmically, an extra-systole is followed by a compensatory pause, the explanation being that the first stimulus after the extra-systole finds the muscle in the refractory stage, so that it is not until the second regularly occurring stimulus that a beat is produced. From this experiment it follows that the stimulus to the ventricles in the intact heart is intermittent, but it does not necessarily follow that the stimulus to the heart as a whole is intermittent. It is clear that the beat of the heart starts in mammals in the great veins, and spreads thence to the auricles and then to the ventricles. If we assume that the conduction of the stimulus is through the muscle, and that the muscle is directly excited without nervous intervention, the stimulus to auricles and ventricles must be intermittent, since both conductivity and excitability are destroyed by each contraction, and are only slowly recovered after a contraction. The stimulus to the muscle tissue in the great veins, however, might well be constant, only taking effect when the excitability there had sufficiently recovered.

Under these circumstances the rhythm of the heart would depend on the rate at which the excitability was recovered in the great veins, and on any variation in the strength of the supposed constant stimulus. The condition of excitability in the auricles and ventricles would be immaterial. A stimulus applied to the ventricles would produce an extra-systole destroying excitability and conductivity, and this extra-systole would spread up to the great veins and destroy excitability there. The next contraction of the heart could not occur until the excitability at the great veins had sufficiently recovered, and this would be at an appreciable interval after the recovery of excitability in ventricles and auricles according

* It is interesting that Straub (*Pflüger's Archiv*, vol. lxxxvi, 1901, p. 504) regarded the heart of *Aplysia* as showing strong evidence for the myogenic theory, owing to the supposed absence of nerve cells and fibres.

to the time taken in the conduction of the extra stimulus to the great veins from its point of application. Thus a compensatory pause would follow an extra-systole of ventricular or auricular origin. If the extra stimulus were applied to the ventricle, it is possible that it would not arrive at the great veins before the normal contraction had started, but this normal contraction would be confined to the great veins, as auricle and ventricle would be refractory from the extra-systole. Thus the next complete heart beat would not occur until the next normal contraction started from the great veins at the usual interval after this ineffective one, and so, under these circumstances, there would be a full compensatory pause (see Figs. 1 and 2). A constant stimulus to the muscle cells of the great veins would thus afford a sufficient explanation of the phenomena so far described. Such a constant stimulus might be—and, indeed, probably would be—derived from sources outside the muscle fibres them-

Fig. 1 shows extra-systoles starting from the auricle. The time-marker shows fifths of a second, and the normal interval between two pulse waves is 78 tenths of a second. After every third beat an extra-systole occurs too early, and is followed by the compensatory pause, which is longer than the normal interval. For instance, the pause before the first extra-systole is only 62 tenths, while that following it is 84 tenths. These two together are not equal to the double of the normal interval, so that the compensatory pause is incomplete.

Fig. 2 shows extra-systoles arising from the ventricle. Every other beat is followed by a long pause, in which the small extra-systole *x* can be seen: The normal interval is approximately 7 tenths, although there is no time-marker, while the long pause endures for 14 tenths, or exactly double the normal interval. Here, then, there is a complete compensatory pause. In the tracing from the jugular vein, which is given above this radial tracing, small waves, *a* and *c*, are shown, due to the auricle and carotid respectively, the relation of which to the radial waves is obtained by measuring off from the uprights, which exactly correspond. Where the extra-systole, *x*, occurs in the radial a large single wave, *d*, is found in the jugular. The reason of this is that the ventricle here has contracted before the auricle, and when the auricle comes to contract it finds the ventricle in systole, and so sends its blood back into the veins, instead of into the ventricle. The auricles are beating quite regularly and take no part in the ventricular irregularity.

selves—for example, from the ganglion cells situated in the great veins. It is more probable that nerve cells discharge their energy intermittently rather than constantly, but the recovery after discharge is probably very rapid. Such an intermittent discharge with a rapid recovery would act in much the same way as the constant stimulus that has been described.

Engelmann considered that the stimulus was intermittent, was situated in the muscle fibres, and destroyed by each contraction. Obviously his theory gives a wider scope of variation of rhythm than that just considered. One point, upon which all observers are agreed, is that the stimulus and excitability are independent of one another. It is, for instance, possible to stop the heart experimentally without affecting its excitability, as shown by its response to direct stimulation. In other words, the stimulus may be removed while excitability remains unaltered.¹⁹ This, of course, is explicable on either theory. Engelmann¹⁰ has shown that variations in the stimulus and variations in excitability may occur independently of one another. The experiments are not, however, conclusive in showing that the variations of rhythm, etc., produced are not explicable with a constant external stimulus, just as they are with an intermittent internal stimulus of the muscle fibres, but the wideness

and extent of the variations are certainly in favour of Engelmann's view.

Some experimenters²¹ have come to the conclusion that the excitability may be recovered to its full extent some time before a contraction takes place. This weighs strongly in favour of Engelmann's view, but it is to be remembered that the experiments dealt with the excitability of auricle and ventricle, and not of the great veins.

Certain indirect arguments have been applied to the solution of the problem from time to time. For instance, Yandell Henderson,²² from experiments on the volume of the heart, considered that a neurogenic origin of the beat was the more probable. His results are, however, not incompatible with the myogenic theory. Gibson, in his address this year to the Medical Society of London, brought forward some arguments from the human side. The possibility of a block between the veins and auricles seems definitely proved.²³ In human beings the

Fig. 3.

Fig. 4.

Figs. 3 and 4 show the juvenile type of irregularity. They are both taken from boys aged 11 years, one suffering from epilepsy, the other convalescent from enteric fever. The intervals between the pulse waves are approximately 8, 7.5, 11, 9, 8.5, 11.5, 10.5 tenths of a second in the first, and 12, 8.5, 8.5, 12, 9.5, 8, 12 in the second. In spite of the inequality of the intervals the individual pulse waves do not differ much in size.

probable starting point of the heart beat is at the root of the superior vena cava, and this point is connected with the auricles by numerous muscle fibres,²⁴ a condition of things not favourable for the occurrence of a block. It is, of course, true that a stimulus is much more likely to be blocked in a narrow channel of communication than in a wide one, but a block in the latter is by no means impossible. Conductivity in a particular group of muscle fibres may be depressed in two ways: (1) By pressure from the outside, which is obviously more likely to affect a narrow strand; or (2) by its internal condition, which may be altered by nervous influences, poisons in the blood, fatigue, etc. This second cause of depressed conductivity would be likely to act equally on all muscle fibres, though it is quite possible that it might cause a block in a narrow channel sooner than in a wide one. Where, however, conductivity is gravely depressed in all the muscle fibres, the stimulus would never be conducted from its point of application. In other words, there would be an appearance of a block close to the point of application of the stimulus, which in a normally beating heart would be between the superior vena cava and the right auricle. A fibrosis might very well convert the wide channel between vein and auricle into a narrow one, in which a block might easily be induced, or communication between vein and auricle might be abolished by disease. Gibson also instances the juvenile type (see Figs. 3 and 4) of heart irregularity—when the beats are equal in size but are separated by unequal intervals—as being impossible of explanation on the myogenic theory, but it is almost equally difficult to offer any satisfactory neurogenic explanation of this irregularity.

The arguments adduced hitherto are, on the whole, rather in favour of the nervous origin of the stimulus, but they are certainly not incompatible with a muscular source. We have, therefore, to turn to more direct evidence in attempting to find a solution for the problem. Here we are met by a flat contradiction, one side affirming

and the other denying, that the muscle can be seen to beat when separated from nerve cells. The contention that every cardiac muscle fibre is capable of supplying its own stimulus and beating independently may be taken as disproved. All the later evidence goes to show that the ganglion free apex of the frog's heart is incapable of beating unless stimulated. Experiments on mammalian hearts also tend to prove that only certain parts of the heart are capable of starting an independent rhythm, though observers do not exactly agree with one another. For instance, Hering,²⁵ by making extensive cuts through the hearts of dogs and other mammals, found that a section cutting off the great veins stopped the heart for a time, and that later the heart started beating again; further cuts removing other parts of the heart showed that certain regions—such as the left auricle, the right auricular appendage, and the ventricular apex—never started a rhythm of their own. All the parts capable of starting a rhythm, such as the great veins, the greater part of the right auricle, and the upper parts of the ventricles, contain numerous nerve cells, while the auricular appendages and the ventricular apex contain no nerve cells. Langendorff and Lehmann²⁶ had previously come to the conclusion that the auricular appendages were incapable of starting an independent rhythm, but Erlanger and Blackman,²⁷ while generally confirming Hering's results, state that the whole of the right auricle, including the appendage, exhibits the property of rhythmicity. Assuming, however, that Hering is correct in his view, that only those parts of the heart exhibit rhythmicity which contain numerous nerve cells, this does not absolutely prove that the nerves supply the stimulus, though, of course, it affords strong testimony in favour of that view. If only certain of the muscle fibres possess the power of rhythmically contracting, we should expect these important fibres to be specially well provided with blood vessels and nerves.

A difference of structure would be expected in association with a difference of function. In respect of this point there is some evidence in favour of the myogenic theory; Gaskell had already²⁸ remarked that the muscle in the sinus venosus of the frog, which exhibits the greatest rhythmicity, was of a more embryonic type than the rest of the cardiac muscle, and recently Keith and Flack²⁹ have shown that a similar difference of muscle exists in mammals and man. They point out that the remnants of the sinus venosus are found chiefly in the right auricle in the muscle round the vena cava superior, coronary sinus, and remnants of the venous valves, although it is possible that some remnants may also exist in the left auricle round the openings of the pulmonary veins. The muscle in these situations belongs to the pale variety of striped muscle, as does also the muscle in the A-V bundle, whereas the rest of the cardiac muscle is red. The A-V bundle may be regarded as the remnant of the auricular canal, the appendage forming the auricle proper. Thus a special muscle tissue is found in those situations where a rhythmical beat may start, and is found nowhere else, with the possible exception of the muscle round the opening of the pulmonary veins in the left auricle. At two points or "knots" the muscle differs still more markedly from the general cardiac muscle. One knot forms the commencement of the auricular ventricular bundle just above the septal cusp of the tricuspid valve, and the other is at the root of the superior cava. The second of these receives a specially large blood supply, and has numerous nerve cells, and is possibly the spot from which the normal cardiac contraction starts.

There is also direct evidence in favour of the stimulus being of muscular origin. The first and most important is afforded by Engelmann's experiments on the great veins of frogs,³⁰ in which he found that these veins were capable not only of rhythmically contracting as a whole but that even very small pieces of them, hardly a cubic millimetre in size, rhythmically contracted also. In many of these tiny pieces Engelmann was quite unable to find any nerve cells although the muscle was very thin and the cells should have been easy to find. It is true that modern methods of staining show nerve cells to be numerous over the great veins, and it would be well if Engelmann's observations were repeated; but until they are disproved it seems to me that they must be accepted, and the possibility of the muscle beating rhythmically without the intervention of nerve cells acknowledged.

Support is lent to this conclusion by the fact that the embryonic heart in birds and fishes starts beating before there are any nerves or nerve cells in it.³¹ It is true that it also starts beating before the muscle fibres have developed, but the cells which start this rhythmic pulsation are mesoblastic cells from which muscle fibres are subsequently developed, while the epiblast, from which, so far as we know, all nerve cells are derived, has no part in the causation of the beat.

In conclusion, it may be emphasized that the myogenic theory is not incompatible with any of the known phenomena of the heart beat, while it affords the best explanation of many of them. It is certain that the muscle fibres possess the properties of contractility, excitability, conductivity, and tonicity, and it is probable that these are exercised during the normal beat of the heart without the intervention of the nervous tissue. It is also certain that all the muscle fibres are not capable of building up a stimulus for themselves, but there is evidence to show that certain fibres of peculiar structure possess this property. While certain facts seem to find their readiest explanation in the neurogenic theory, especially the response of the quiescent heart to the stimulation of the accelerator nerve, there are others pointing as strongly to the hypothesis that the heart beat is purely muscular; and in the face of Engelmann's direct observations on the great veins in frogs and the occurrence of pulsation in the embryo heart before development of nervous elements, it seems to me that the complete myogenic theory has the more weighty evidence in its favour, and should be accepted until stronger arguments are brought against it. The conception that the rhythmic beat is purely muscular in its origin does not preclude the possibility of the beat being influenced by outside nervous impulses; this would give a sufficient explanation of the large nerve supply to the heart.

Attributing the rhythmic power of contraction to the muscle is, of course, only a very partial explanation of the cardiac beat, the cause of which lies deeper in physico-chemical changes in the cells. Much suggestive work has been done on these lines by Loeb,³² Howell,³³ and others, but a discussion of this is outside the province of the present article.

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LOCOMOTOR ATAXIA: ITS EARLY RECOGNITION AND GENERAL MANAGEMENT.

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IN considering the nature of the lesion, a sclerotic change in the posterior columns of the cord, in the posterior nerve roots, and in the peripheral nerves, we must bear in mind that although acute and subacute attacks do occasionally occur, the disease is essentially chronic and progressive, and that from a strictly curative point of view little can be expected. The course of the disease is usually very long, and may extend through a period of twenty or thirty years. Our aim, therefore, in treating the malady should be chiefly to relieve the symptoms, and as far as possible to arrest or retard the degenerative process. The cause of this degeneration is by most authorities considered to be syphilis. Erb, in his investigations on this subject, found that there was a history of syphilis in 90 per cent. of cases of tabes; Moebius