ANTHONY FEILING

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MODERN TRENDS

IN

NEUROLOGY

Edited by

ANTHONY FEILING

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PREFACE

It is generally agreed that the present-day output of medical literature throughout the world has become so vast that it is virtually impossible for any busy worker, even the specialist, to keep abreast with all the latest developments.

The aim of this book has been to help the neurologist and the postgraduate student by condensing herein some at least of the more interesting and valuable additions to the science and practice of neurology which recent years have produced.

The Editor has been fortunate in obtaining the collaboration of many distinguished authors to deal with a variety of neurological subjects in which real advances have been achieved.

Advance has been the main criterion for selection, but obviously progress has been more rapid in some subjects than in others: thus the first chapter on the Conduction of the Nervous Impulse introduces a subject which is hardly ever mentioned in the ordinary text-book of neurology and yet is one of the greatest scientific interest. The lengthy discussion of the Frontal Lobes and their Functions not only includes a full and up-to-date account of previous work and opinions but also new and original matter on a subject of vital importance and particular interest at the present time.

It has been deemed appropriate to include a chapter on neuroradiology, since this has not only become a very specialized branch of radiodiagnosis but is indeed an essential part of the neurologist's diagnostic armamentarium.

The articles on Abscess of the Brain and Intracranial Tumours afford convincing evidence of the strides made by the neurosurgeon in the practical fields of diagnosis and treatment.

It is believed that neurologists will find this book a valuable and even an indispensable addition to their library.

Finally the Editor would take this opportunity of expressing his cordial thanks to all the contributors who have combined to make *Modern Trends in Neurology* a volume worthy of its subject.

February, 1951. ANTHONY FEILING

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CHAPTER 1

CONDUCTION OF THE NERVOUS IMPULSE

W. A. H. RUSHTON

IS THE ELECTRICAL THEORY OF CONDUCTION STILL VALID ?

THE IDEA that nerve conduction is an electrical process is of some antiquity and arose naturally from two observations. In the first place, nerves are extremely irritable to electric shocks and they may be repeatedly excited some hundreds of times per second with little expenditure of energy or the appearance of nerve damage: this suggested that an electrical stimulus is the " natural " stimulus. In the second place the only change which could be demonstrated to pass down the nerve was electrical. Upon this basis Bernstein and Lillie developed the theory that when a nerve was active at any point it produced a local electric circuit which excited the neighbouring points and made them in turn active.

The theory, supported by a wealth of congruent facts, is so plausible and so well entrenched in the text-books that it is apt to lose its vigour and to be slow to rally at the onslaught of rival views based upon, say, the properties of acetylcholine, orientated monolayers or other modern notions. Actually, the work of the last few years has fortified the Bernstein-Lillie position to such an extent that it is now virtually impregnable. Let us view the layout of the defences by making a mock attack, and then try in earnest to force the weakest place.

A nerve can be excited if it is suddenly heated; a nerve impulse is accompanied by the minutest evolution of heat. Heat, therefore, is formally in the same causal relation to propagation as is electricity. Why should not heat rather than electricity be the mechanism of transmission? The idea is of course ridiculous. Consider the temperature rise necessary to excite, and the almost undetectable temperature change during, nerve propagation. The discrepancy is fantastic. But it must be admitted that there is a distinct discrepancy also in the electrical analogue. The quantity of electricity which flows during the passage of a nerve impulse is a small fraction of that which must be passed from an induction coil in order to excite. Of course, only a fraction of the action current is directly measured by the recording system, and again only a fraction of the applied shock enters the part of the nerve where excitation occurs, so these two losses together *might* account for the discrepancy—but do they? This is the crux of the electric theory. If they do not, the action current is not strong enough to excite, and some other mechanism must be invoked. But if the action current can be shown to be strong enough to excite, then any other *independent* mechanism would be superfluous, since transmission would certainly occur without it.

The question was settled by Hodgkin (1937), who showed that in a frog's nerve the action current is certainly strong enough to excite. The principle of the experiment was as follows. The nerve was arranged as shown in Fig. 1. It could be excited by a shock applied at S_1 and the success of conduction to the other end of the nerve was indicated by recording thence the action potential amplified and observed upon a cathode-ray tube. In the middle of the stretch the impulse was progressively blocked either by cooling the nerve or by compressing it. Consider the instant when the transmission has just been blocked. Then the impulse

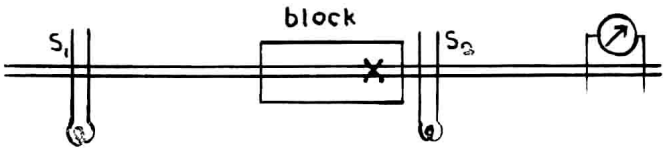

FIG. 1.—Frog's nerve showing region of conduction block by cooling or pressure; S_1 and S_2 are stimulating electrodes. Circle containing arrow represents system for recording action potential.

is propagated as far as, say, the cross (x), and is there extinguished. But the action current (like any passive applied current) can still spread forward through the saline medium into the region outside the block, and if this current is in normal cases sufficient to excite, it should even now appreciably lower the threshold in the region S_2. This was tested, and in the most favourable case as much as 90 per cent lowering of threshold occurred. So it is certain that *something* spreads forward from the blocked impulse capable of lowering the threshold. The following proves that what spreads is the action current.

As the electrodes S_2 are moved farther away from the block, the threshold lowering was found to be less and less, and a curve was plotted to show how the " spread " attenuated with distance. Does the action current fall away along the same curve? This was tested by using the electrodes S_2 not to stimulate but to record the spread of the action current from the blocked impulse. As S_2 moved away from the block the action current was found to attenuate along the same curve. But is this spread the purely passive flow of current, or is there a spread of some other influence which is associated both with threshold lowering and current flow? To test this, the electrodes S_1 were moved to the position in the block marked by the cross. Then a just sub-threshold shock (made about the same shape as the action potential wave) sent in through S_1 caused a spread of current and of threshold lowering which also attenuated with distance from the block along the same curve.

This experiment, taken in conjunction with many controls which cannot here be discussed, proves that the blocked impulse causes a lowering of threshold to spread in front of it, having the same time course and space course as the observed spread of action current, and the same as the effects of an external current similarly applied. Hence the action current certainly contributes appreciably to excitation. And since this contribution may be as great as 90 per cent some little way from an extinguished impulse, it is clear that it would be at least 100 per cent in the

vicinity of a normal impulse. It is therefore certain that the action current excites the next region of nerve, and so propagates.

It follows from the electrical theory of conduction that the propagation velocity should depend, amongst other things, upon the electrical conductivity both of the axis cylinder and the external return circuit. When the conductivity is high *ceteris paribus*, so will be the velocity. In keeping with this is the well-known fact that fibres with large diameter conduct fastest, and that the nerves of marine invertebrates with a high salt concentration in the body fluids conduct faster than similar nerves of fresh-water animals. But direct comparison with theoretical expectation is complicated by differences in myelination, nodes of Ranvier, and so forth. With regard to the influence of external conductivity, however, the prediction of the electrical theory admits of a neat verification in non-medullated nerve, again due to Hodgkin (1939).

The single giant fibre of a squid was placed in moist air on an arrangement consisting of stimulating and recording electrodes (Fig. 2) and rested between upon a grid of platinum strips insulated from each other except for contact with the

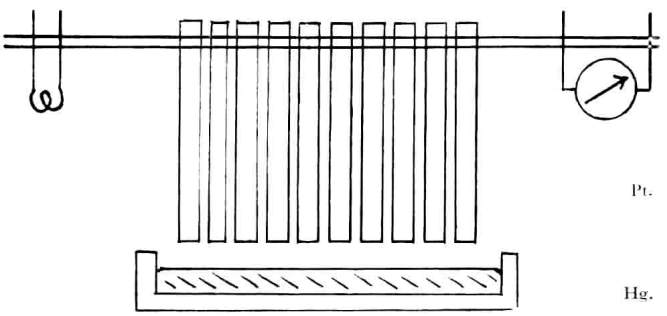

FIG. 2.—Single giant nerve fibre in contact with a grid of platinum strips, the lower ends of which may all be electrically connected by raising the mercury trough.

nerve. A little trough of mercury could be raised to immerse the lower ends of the strips, thereby short-circuiting them and increasing the external conductivity of the nerve. The effect upon the recorded action potential was dramatic, for the picture on the cathode-ray tube suddenly jumped to a position of earlier arrival, and as instantly jumped back to its old place when the mercury trough was dropped.

The importance of this result is that it shows so clearly that the conduction velocity may be markedly increased by a procedure which does not affect the nerve in any way except to increase the path through which an electric current may flow. It follows that any other factor which is claimed to be responsible for propagation cannot be independent. It must operate through the generation, the transmission or the action of this electric current. It may therefore be of interest to consider these processes a little more closely.

HOW IS THE ACTION CURRENT GENERATED ?

There is a store of electric energy built up in the nerve and a little of this is expended in the propagation of each impulse. The store of energy is seen in the *resting potential*, which is the potential difference between the inside and outside

of a nerve fibre. It may be demonstrated most directly in the giant nerve fibre of the squid, for this is so large (1 millimetre in diameter) that a fine electrode can actually be passed down the axis cylinder and can record the potential there. The technique is difficult, for if the nerve sheath is touched during this procedure the nerve will be destroyed. Nevertheless, Hodgkin and Huxley (1939; 1945) and Curtis and Cole (1942) have succeeded in recording both resting potentials and action potentials from healthy nerves. Fig. 3 shows such a record from Hodgkin and Huxley, led off between the internal electrode and the sea-water outside. In

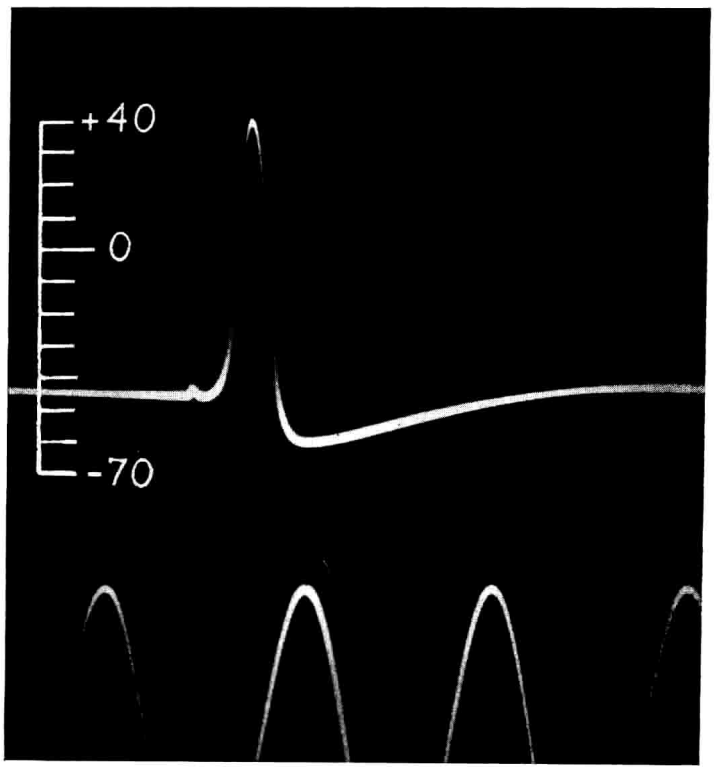

FIG. 3.—Resting and action potential of giant nerve fibre recorded directly between the sea-water outside (taken as zero) and a fine wire passed down the axis cylinder. The scale indicates the value of the internal potential in millivolts. There is an interval of 2 milliseconds between successive peaks of the time tracing. (*By courtesy of J. Physiol.*)

the resting state the inside is seen to be some 50 volts negative to the outside, but at the peak of activity this polarity is reversed, and the inside becomes positive.

Earlier workers could not measure directly the internal potential, and had to deduce its value from other measurements. They concluded that, in activity, the resting potential was simply abolished, not reversed. Reversal, however, certainly occurs in all cases in which it has been possible to measure the internal potential directly—namely, in several kinds of invertebrate nerve and vertebrate muscle.

HOW IS THE ACTION CURRENT GENERATED?

The resting potential itself is correlated with the chemical composition of the axoplasm and with the permeability of the cell membrane to diffusible constituents. The most striking constituent is the potassium ion, since it is the principal ion present in axoplasm and must therefore be chiefly responsible for the transport of electricity through the greater part of the nerve substance. Its concentration in nerve is 40 times that in plasma, and to account for this one's first idea, perhaps, would be that the cell membrane must be impermeable to potassium, for otherwise it would diffuse out. But this cannot be the explanation. Boyle and Conway (1941) working on muscle (which behaves in the same way but is easier to analyse) have shown that when potassium is increased in the external fluid bathing the muscle, this ion actually enters the muscle against the stiff diffusion gradient—as was proved by the direct analysis of pairs of muscles with high and with normal external potassium.

Even better evidence is given by radioactive potassium (*see* review by Ussing, 1949). If the membrane is normally impermeable to potassium, then a normal nerve placed in Ringer's fluid containing radioactive potassium instead of common potassium will not allow any of this substance to enter the cell. Actually radioactive potassium enters quite rapidly, showing that it can easily pass through the membrane. But if it can, why does it remain concentrated within instead of leaking out? The explanation appears to be that most of the anions which neutralize the potassium ions in muscle and nerve are non-diffusible, and so realize the condition of the Donnan equilibrium.

This simply means that the potassium ions, which on their own could easily sail in and out, are anchored to the fixed anions. Thus potassium ions can only begin to diffuse out; they are then checked by the pull from the anions, and the strength of this (electrical) pull exactly neutralizes the tendency to diffuse. The resting potential is the electrical pull which keeps the highly concentrated potassium ions in equilibrium in the axoplasm.

It is sodium, however, which is the important substance in the process of excitation. The axoplasm contains very little sodium, so that in the resting state these ions tend to enter the cell not only because of the diffusion gradient, but because the electric field which keeps the potassium ions inside will also tend to force the sodium ions in. The fact that sodium does not enter and replace potassium in these conditions must be due to one of two things. Either the membrane is quite impermeable to sodium, or else there is a vigorous excretory mechanism which pumps the sodium out as fast as it leaks in. The study of radioactive sodium distinguishes clearly between these alternatives. If the cell membrane is quite impermeable, clearly no external radioactive sodium can diffuse in. But it is found that radioactive sodium starts to enter rather rapidly, replacing the small amount of sodium already there. The total amount of sodium in the cell is not increased and equilibrium is soon attained. It follows that sodium must be removed from the cell just as rapidly as it enters, and it is probable that an appreciable fraction of the entire resting metabolism is directed to its excretion.

What happens, now, when the nerve becomes active? It has long been supposed that the membrane increases in permeability so that things can pass through more easily. Recent work fully confirms this idea. The electric resistance of the sheath gives a measure of the ease of passage of ions, and Cole and Curtis (1939) showed

that in non-medullated nerve this resistance falls nearly to zero as the action current flows across the sheath. But to what substance does the sheath become permeable? Not to potassium, for that was in equilibrium all the time and could not be affected simply by a change of permeability. Obviously, sodium with concentration gradient and electrical field both tending to drive the ions inward is just the substance we look for. In the resting state there is a leak which is made good by vigorous pumping, but if in activity the whole membrane becomes permeable, equilibrium will be overwhelmed by an inundation of sodium ions.

In 1902 Overton (quoted by Hodgkin and Katz, 1949) found that, with the exception of sodium alone, all the constituents of the fluid external to a nerve may be replaced by an indifferent substance without rapidly abolishing the action potential wave. Hodgkin and Katz (1949) have used the squid giant nerve fibre to make a beautiful study of the dependence of action potential upon sodium concentration, showing how accurately the results fit the expectations of the sodium permeability theory. The reversal of the sign of the resting potential during activity follows as a consequence of this theory, and a direct confirmation of the increased sodium permeability during activity has also been obtained by Keynes (1949), using radioactive sodium.

In the resting state potassium is held concentrated in the axoplasm by the electric field across the membrane. When this field is reversed during activity, potassium would be expected no longer to be withheld, and so to flow out. This too has been shown to occur (Keynes, 1948), by the use of radioactive potassium.

This outline of the way the action potential is generated may be summarized as follows.

Summary

In the *resting state* the nerve membrane is freely permeable to potassium, which is in equilibrium across it. It is less permeable to sodium which is not at all in equilibrium, but is excreted as fast as it leaks in. The anions of the axoplasm are mainly non-diffusible, so that the potassium equilibrium is of the Donnan type, the negative potential of the inside of the cell precisely counteracting the tendency of potassium to diffuse out owing to its high internal concentration.

In *activity* the membrane suddenly becomes freely permeable to sodium, so that this ion enters under the action of both electric and diffusion forces and reverses the sign of the resting potential, permitting the escape of some potassium.

WHY DOES AN ELECTRIC SHOCK EXCITE?

We have considered the energy stored in the nerve as the resting potential, and the change in the membrane permeability to sodium which generates the action potential wave. Since an applied shock can excite a nerve, it must in some way bring about this permeability change. At first sight the mechanism might seem to resemble the way that insulation breaks down in a condenser when too high a voltage is applied. But this analogy will not do at all in the case of nerve, since the polarity is the wrong way round. The shock stimulates not where it increases the resting voltage (anode) but where it neutralizes it (cathode). And so we are led to the picture of a crowd which cannot leave a building owing to the very press and urge to escape: ease the pressure and the crowd pours forth readily.

Various more or less likely physico-chemical schemes may be suggested which

will allow this fancy to be worked out quantitatively with encouraging results. According to them, an electric shock excites because it depolarizes, and hence releases the strain which holds back the sodium. The sodium current which now begins to flow further depolarizes the membrane, and so an " explosive " wave of depolarization develops which, however, is self-limiting and self-terminating. The theory can probably account quantitatively for the restoration of the resting potential, the shape of the action potential wave, and the appearance of the refractory period, but it is rather complicated and still too hypothetical for inclusion here.

HOW DOES THE ACTION CURRENT SPREAD ?

The action current is in essence the inrush of sodium ions when the permeability of the membrane is suddenly increased. This makes the axoplasm less negative, and allows the potassium to come out. Since it is the potassium coming out which excites, it is important to consider where this occurs. If, for instance all the potassium came out just where the sodium went in, electrical propagation could not take place, since no new region would be excited. An essential feature of the electrical theory is that the current spreads away from the place where it was generated, and activates a fresh region.

Lord Kelvin worked out the appropriate formula for the spread of electricity in the submarine telegraph cable. It is true that the electric conductivity of axoplasm is minute compared to stout copper, and so the spread in nerve is only a few millimetres instead of hundreds of miles. But if the proper physical values for nerve are put into Kelvin's formula, it is found to describe fairly satisfactorily the passive spread of current in the nerve structure. As would be expected, the potassium current leaving the nerve is greatest quite close to the active region where sodium enters. The attenuation with distance was mentioned in describing Hodgkin's experiment with conduction block. In non-medullated nerve, then, propagation occurs by the continuous spread of the excitability process from point to point, like the combustion of a cigarette in smoking.

Lillie, with a remarkably good model, demonstrated the process by placing a steel piano wire in strong nitric acid. The " passive " state of the iron could be reduced by an electric shock, and a wave of reduction and re-oxidation travelled rapidly down the wire, as could be seen by the local evolution of gas which accompanied the activity. The transmission, moreover, was accompanied by an "action potential", and followed by a "refractory period". Lillie (1925) found the effect of turning his model into a medullated nerve by threading on to the steel wire several short lengths of glass tubing to represent the myelin sheath with nodes of Ranvier. The result was that conduction changed altogether in type. Instead of proceeding continuously along the wire, the active process only occurred in the regions opposite the gaps between the glass tubes, so that propagation seemed to jump from node to node. This mode of transmission appears to have two advantages, for the speed was much greater than by the continuous (non-medullated) process, and since only a few regions of the wire were active, the total energy expended in transmission is presumably greatly reduced. It is certainly the case that medullated nerves conduct much faster and with smaller heat evolution than do non-medullated nerves of comparable size.

This leads to the question as to whether in fact medullated nerves behave as in Lillie's model. Erlanger and Blair (1934) found that when nerve transmission is blocked by the anode of a polarizing current, the blocking occurs at a node of Ranvier, and Tasaki (1939) showed that electric excitation and narcotic action took effect there. But a full analysis of how propagation is actually conducted has only recently been achieved by Huxley and Stämpfli (1949).

A single fibre from a frog's nerve was dissected free, tied to a fine nylon thread and drawn through a 40μ hole in the partition separating two troughs A and B (Fig. 4) containing Ringer's fluid. With this arrangement, all the current passing down the axis cylinder of the fibre in the region of the partition must return through the space left between the fibre and the walls of the 40μ hole. The resistance of

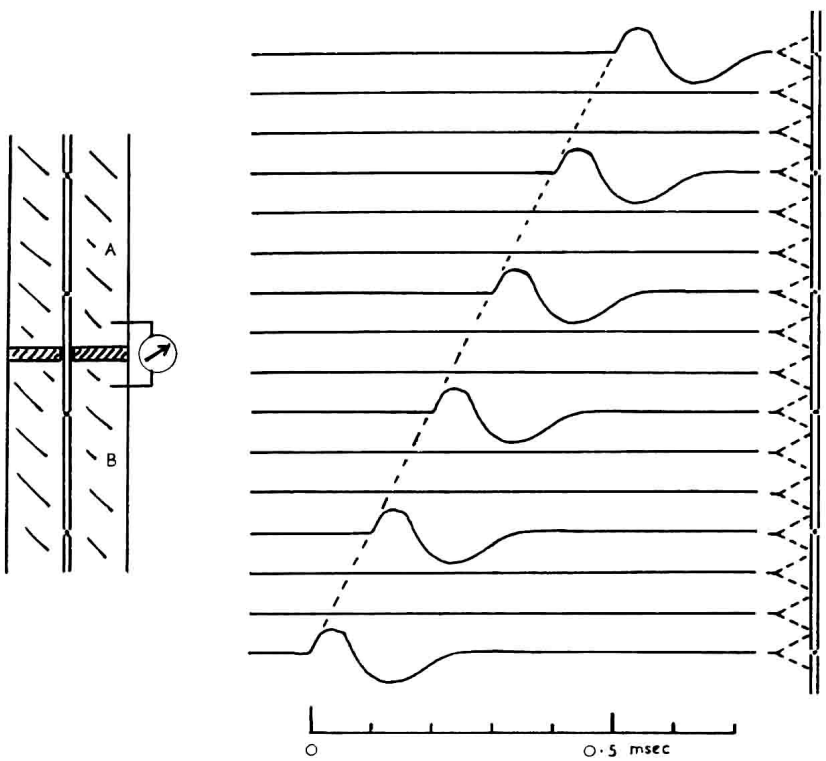

FIG. 4.—Single medullated fibre threaded through a 40μ hole in the partition separating the trough into regions A and B. Potential recorded between fluids in A and B.

FIG. 5.—The nerve fibre is drawn little by little through the partition of Fig. 4 and in each position records are taken. The *difference* between successive records is plotted here. The two corresponding locations of the partition are indicated by the dotted lines at the right of each record. Only when a node lies between the two locations is a wave obtained. Distance between nodes about 2 millimetres; conduction velocity about 20 m. per sec.

this tiny gap was half a megohm, so any current flowing would develop an appreciable potential difference between A and B. This was recorded by electrodes placed in the two troughs, thus measuring directly the current in the axis cylinder at the level of the partition. The nerve fibre could be drawn through the hole and the records for different positions compared.

The results are shown diagrammatically in Fig. 5. On the right is shown the nerve fibre and on the left the *difference* between the records obtained when the partition was at each of the two points indicated by the dotted lines on the right. It is seen that there is no difference unless a node lies between the two points measured. his of course means that current only leaves and enters the axis cylinder at the nodes. In the internodal region, the current in the axis cylinder is the same at all points, and thus the difference is zero. The nature of the membrane current at the nodes is first an outward flow, presumably of potassium, which excites, followed by a larger reversed flow, presumably from the inrush of sodium. The inflow at one node occurs at the same instant as the outflow of the next node, showing that the circuit is chiefly through adjacent nodes, and determines in this way the velocity of conduction. In detail the records are not quite so simple as here represented because, although the internodal stretch is passive, the myelin is not a perfect insulator.

We have seen (Fig. 2) Hodgkin's proof that in non-medullated nerve the flow of electricity is essential for conduction; Huxley and Stämpfli have an elegant demonstration that this is also the case in medullated nerve. Fig. 6 shows the arrangement (in very distorted proportion). Two glass microscope slides, wet with Ringer's fluid, are separated by an air gap of 1–2 millimetres. This is bridged

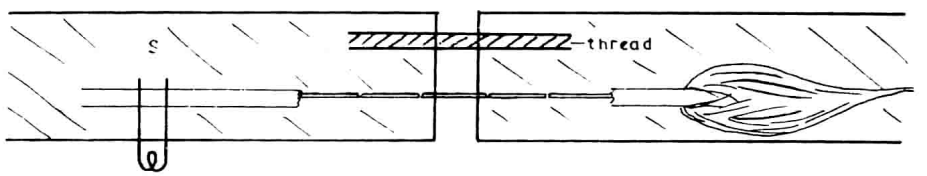

FIG. 6.—Two wet microscope slides separated by an air gap bridged by a single fibre with no node in the gap. The stimulus at S causes a twitch of the motor unit only if a return circuit for the action current is provided across gap, such as by a moist thread.

by a single fibre with no node in the air gap. The nerve may be excited at S, and the conduction is indicated by the twitch of the motor unit connected to the single fibre. It was found that conduction was impossible unless a return circuit was provided, for instance by placing a moist thread across the air gap. In the absence of the thread no current could pass down the axis cylinder bridging the air gap, for practically no fluid adhered to the outside of the fibre, and there was no other pathway for the returning current. The nerve fibre itself had not dried up and ceased to conduct, because joining the two wet slides by a moist thread which did not touch the fibre at once allowed conduction. Since the only contribution the thread can make is to complete such a circuit, it is clear that the electric current is essential for conduction.

It has often been stated that the tracts of the spinal cord are myelinated but do not exhibit the regular nodal constrictions of peripheral nerve. If this is true, it raises some rather difficult questions in the mechanism of conduction there. But recent evidence suggests that the peculiarity of the white matter lies not so much in the absence of "nodes" as in the difficulty of finding a technique suitable for revealing their presence. Hess and Young (1949) have injected methylene blue and examined fibres of various sizes from teased portions of white matter. They find that the dye penetrates the myelin at regular intervals separated by distances of roughly a hundred times the fibre diameter.

Some years ago Gasser and Graham (1933) showed that a fibre in the dorsal column of the cord conducted for some centimetres at about the same speed as did the peripheral branch of this same neurone. So, on the whole, it seems likely that the mechanism of conduction in the two anatomical regions is not very different.

HUMAN NERVES

All the work so far considered has been done upon the excised nerves of animals, in which conditions may be accurately controlled. The difficulties of recording from nerves in the normal human subject are so formidable that it has not often been attempted. Dawson and Scott (1949) have succeeded in recording through the skin the action potential of a volley of synchronous impulses in the ulnar nerve excited by a shock. But the equipment required has to be exceptionally free from noise at the highest magnification. In view of the difficulties of recording from human nerves *in situ*, we may well ask what information may be expected from such records and what alternative means are available for getting this information.

The information can answer three questions. (1) Is the impulse transmitted? (2) How fast does it travel? (3) What is the shape of the action potential wave?

(1) If the electric wave is recorded, it proves that the nerve impulse has been transmitted as far as the recording site even though it never reaches, say, the muscle. But the result can only be demonstrated for a group of synchronous impulses and it may sometimes be hard to prove what kind of nerve fibres are involved. The latter information, of course, is given much better by the usual tests in neurological examination, in which fibre function and peripheral localization can be rather well determined.

The muscle twitch has been used as an index of nerve activity for a century and a half. It still has a very important place in human investigation, since it may fairly easily give information about single motor nerve fibres in normal function. A single nerve fibre innervates a hundred or more muscle fibres which form a group all discharging together. A needle electrode constructed to detect only the electric events occurring near the tip, if thrust into the midst of this "motor unit" of muscle fibres, will therefore record this activity and hence the activity of the single motor fibre which supplied them. Two forms of needle electrode are commonly employed in this work. One is a sharp sewing needle (steel, silver or platinum) insulated all over by good varnish. The varnish always withdraws from the extreme tip, leaving a minute conducting area which will record the potential of the point. A moist pad placed somewhere on the patient constitutes a return electrode. This type of electrode is preferred by Jasper and Ballem (1949), who discuss the technique before proceeding to the investigation of denervated muscle. The other