Section of Physical Medicine

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SAMUEL HYDE MEMORIAL LECTURE
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On the Fringes of Electrodiagnosis

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IN this lecture my point of departure must be a definition of the operative word in the title "electrodiagnosis". To me this means the application of electrology to the study of deviations from the normal behaviour of the motor unit and their interpretation to diagnostic ends. In this context motor units must be regarded as individuals in societies which, anatomically speaking, go to make up the anterior horns in the cord or the nuclei in the brain, the nerves and the muscles. Nor should their behaviour, normal or abnormal, be considered except in the light of this membership and of their share in the activities of this society. Again, the word "behaviour" of the motor unit is here intended to include not only the manner in which it conducts itself at rest and on volition, but its mode of responding to stimuli, either reflex or artificial, or to the action of certain drugs.

By and large when we speak of a clinical examination we imply that observations are made by the direct use of four of our senses—sight, hearing, touch and smell. Some might hold a brief for the imponderable sixth sense. Because of the limitation of our senses, some investigations require complex equipment, frequently demanding specialized knowledge and training. Yet in every case the intricate instrumentation is merely a convenience calculated to extend the range of our perception. As with specialized investigations in other fields, electrodiagnosis is a complementary one—generally to a neurological examination from which it varies only in the difference in approach to a more restricted problem. An electrical stimulator and an electric probe replace the patellar hammer and pairs of searching eyes, ears and hands.

For many, the intricacies of the electrodiagnostic equipment have a fascination which acts as an impediment to the appreciation of what it aims at and accomplishes. For this reason, I shall ask you to forget the complex machinery and indulge with me in a little imaginary adventure which I might well have entitled "20,000 Microns Below the Skin".

As a prelude to these mental exercises I shall ask you to imagine that your body shrinks to the point of being capable of being enclosed in a microscopic capsule, itself embedded in the muscle to be examined. From this vantage point, the workings of the muscle could be observed at first hand.

At rest the normal muscle would be a silent world. During volitional activity there would first occur an electrical disturbance produced by the propagated action potentials of the various muscle fibres composing the nearest active motor unit. If all these potentials started at the same time from their individual motor end plates towards you, provided their velocities were the same, they would summate and approach you as a sort of electrical tidal wave. After this had died down there would follow the mechanical manifestations related to the actual muscular contraction. Should your host suffer with some condition which causes the muscle fibres in the motor unit to be excited in a staggered fashion instead of simultaneously, then a series of small electrical waves would replace the large tidal wave.

As an encapsulated observer in a muscle you might well make some interesting observations and deductions in respect of the temporal dispersion of the action potentials propagated along the muscle fibre of a motor unit. Some of you will here object that by no extension of your senses would you be able to detect these electrical disturbances. This, of course, would be wrong. Electricity does affect us although we cannot lay claim to having been provided with special sense organs for this purpose, and if for electromyographic examinations we prefer to display amplified action potentials on the screen of a cathode ray tube or from a loudspeaker rather than in the shape of electric shocks, it is only because it is more comfortable, more dignified and scientifically more acceptable to do so.

FIG. 1. J. Dewe

There is something to be learned from Fig. 1 which is of importance. It will be noted that the unfortunate gentleman has just suffered a severe shock in his left leg through the unwelcome attention of a compact shoal of electric eels. Little does he realize that he is about to undergo a similar, but less unpleasant experience in his right leg, for a second shoal in a less tight formation is approaching that. The fact that the fishes are scattered means that at the most he will endure a series of minor shocks spread over a longer period. It is interesting to note that while the first shock will not be substantially affected by the position of the leg in the shoal, in the case of the second shock the pattern of the sensation will be affected by the position of the right leg in the midst of the shoal. Similarly, a change in the location of your exploratory capsule would substantially modify the pattern of the electrical disturbance only when the action potentials are staggered.

During volitional activity of a normal muscle you would come under the influence not only of the nearest active motor unit, but also that of more remote ones, and would thus be

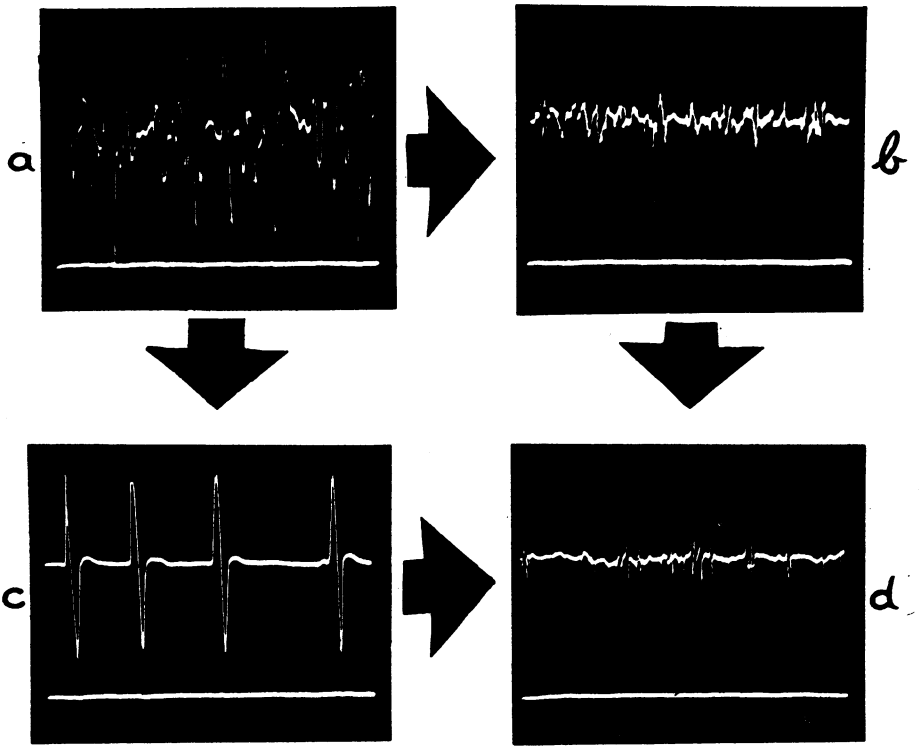


FIG. 2.—Shows the modifications that would result in the EMG tracing of a muscle (A, normal pattern) if a large proportion of the muscle fibres became inactive within the motor unit (B, myopathic pattern) as distinct from a numerical decrease of active motor units in the muscle (C, discrete pattern). In the presence of axon degeneration fibrillation might be observed at rest (D, profuse fibrillation).

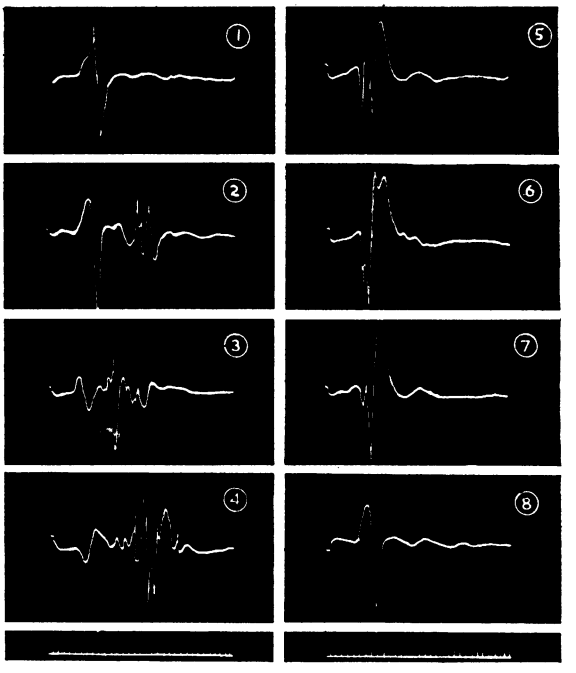


FIG. 3.—EMG tracings recorded on shock stimulation of the radial nerve with eight different positions of the needle electrode in extensor digitorum communis demonstrating temporal scatter of the activity in different motor units in polyneuritis. (Time scale in msec.)

subjected to a veritable electric storm, in form similar to the well-known electromyographic interference pattern on full volition. A little imagination will help you to conjure up the difference in the picture which would result if the electric storm abated by a progressively increasing percentage of idle muscle fibres in the motor units on the one hand, and a progressively increasing percentage of inactive whole motor units on the other hand.

In the first case the picture would pass from the normal interference pattern (Fig. 2A) to the myopathic pattern (Fig. 2B); in the second, from the normal interference pattern to a grossly decimated pattern (Fig. 2C) before you found yourself back in the silent world. In the presence of some denervated muscle fibres, fibrillation potentials might, of course, be detected at rest (Fig. 2D).

It would be tedious to describe all the other manifestations of pathology of the neuromuscular system for with very little further call on your imagination, you will be able to extend the analogy in which we have indulged, to myasthenia before and after Prostigmin or Tensilon exhibition; to myotonia; and to affections of the cord with their giant potentials and their fasciculation potentials.

What would happen if instead of volitional activity, nerve stimulation were substituted? With an adequate stimulus all the nerve fibres would be excited simultaneously at virtually the same level. The nerve impulses would then race down the fibres and the first to reach its group of muscle end plates would activate that motor unit which would be the first to break the latency. This would, of course, be the impulse set up in the fibre of largest calibre. Others would be propagated at a slightly reduced rate along fibres of lesser cross-section. All the same, the electric storm to which you would be subjected would be a violent, albeit a short-lived one—a matter of 10 msec. If for some reason a great discrepancy existed between the velocity of the winner, that of the loser and that of the intervening competitors, then you, as an intramuscular implant, would be tempest-tossed for some time, perhaps 20–30 msec. Of course, the longer the racing track, the longer you would have to wait for the stragglers.

That under certain conditions motor units respond in a staggered fashion although their nerve fibres have been stimulated simultaneously, can be seen from this series of eight action potentials, all from the same muscle in a patient with polyneuritis (Fig. 3). They were recorded electromyographically by means of a needle electrode inserted into the extensor digitorum communis while the radial nerve was being stimulated. The position of the needle was moved a few millimetres between stimulating pulses. It will be noted that the latency for the motor unit nearest the tip of the needle varies from 6 msec. for the first motor unit to fire off to 23 msec. for some of the late arrivals.

Again, when the stimulus applied to a nerve is progressively increased so that the fibres which are most easily stimulated are excited first, then successive records show the order in

which different motor units come into play. The final record is a kind of panorama of these sequential responses. Fig. 4 shows a sequence of four electromyographic responses picked up at the same time by means of a needle electrode (upper tracing) inserted in the extensor digitorum brevis, and a bipolar surface electrode (lower tracing) applied over it. The stimulus was increased between records.

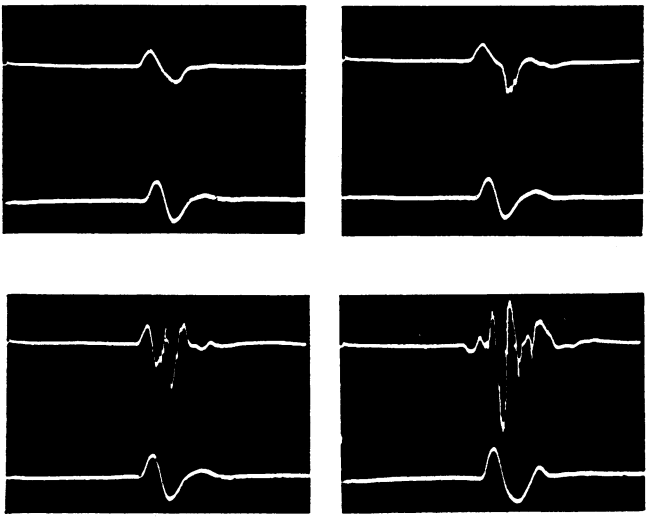
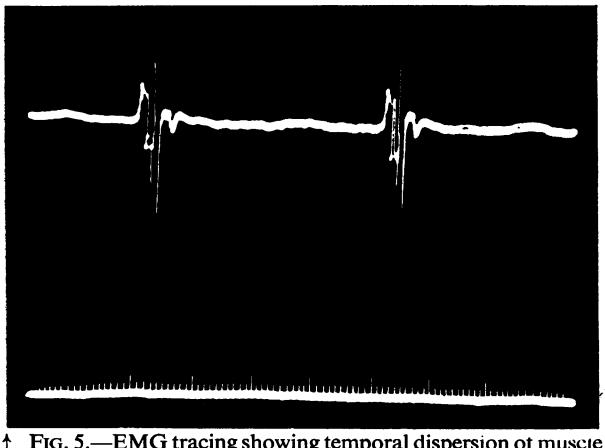


FIG. 4.—EMG tracings simultaneously recorded by means of a needle electrode (upper tracing) inserted in extensor digitorum brevis, and of surface electrodes (lower tracing) applied to the skin over this muscle during progressively increased stimulation of the anterior tibial nerve.

I should like to seize this opportunity of pointing out that although the surface electrode picks up the summated potentials, a filtering out of the high frequency components occurs. As the detection and analysis of this particular component is of value from a diagnostic angle, and is unfortunately obliterated by the use of surface electrodes, it follows that these electrodes cannot supplant needle electrodes for analytical purposes. However, just because of their power of picking up gross potentials of motor unit origin while rejecting finer potentials of muscle fibre origin, they possess a kind of coarse frequency discriminating faculty. For instance, the surface electrodes will readily pick up the discrete giant potentials encountered in a weak muscle where this is due to a motor neuron disease, but it will fail to pick up anv-



↑ FIG. 5.—EMG tracing showing temporal dispersion of muscle fibre activity within a motor unit on volition. (Time scale in msec.)

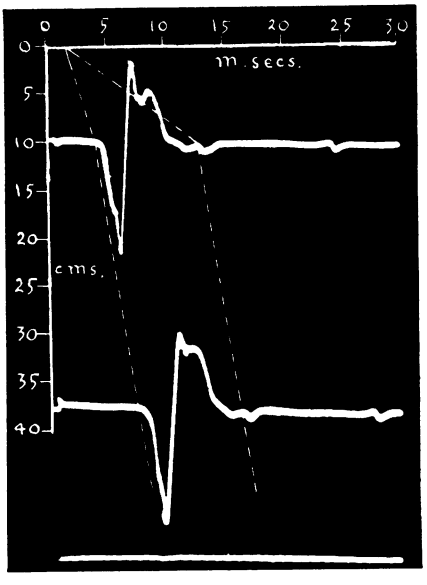


FIG. 6.—EMG tracing of responses in the first dorsal interosseus on stimulation of the ulnar nerve at the wrist and elbow.→ (Distances of points of stimulation from the pick-up electrode along ordinates in cm. Time relationships along abscissa in msec.)

thing in a muscle of similar weakness where this is due to a myopathy. In this sense surface electrodes could be regarded as a frequency discriminating device.

The question to be answered now is whether the temporal dispersion is of any significance. I must here remind you that two distinct types of temporal dispersion can occur. Temporal dispersion within a motor unit (Fig. 5), which can only be due either to unequal delay in neuromuscular transmission or—and this is more likely—to a differential velocity of conduction along the branches of the neuron distally, occurs mainly in the polyneuromyositis group of affections and in nerve regeneration, but it can also be observed at some stages in certain motor neuron affections of myelopathic nature. It is more readily detected during voluntary activity than on nerve stimulation.

The temporal dispersion of motor unit activity within a muscle of which I have already spoken on the other hand can be explained on the basis of differential velocity of conduction of nerve fibres, *qua* nerve fibres as distinct from axon branches. It is encountered in conditions where a motor nerve is subjected to chronic compression—as in the carpal tunnel syndrome. It is not easily observed on volition but is readily demonstrated on nerve stimulation. There appears to be no reason why in some conditions the two types of dispersion should not co-exist.

The cross-section of a nerve fibre is known to determine the velocity of the impulse propagation along it. A diminution of the calibre of a fibre reduces the speed and consequently increases the latency. Hodes *et al.* (1948), Lambert (1956), Simpson (1956) and Gilliatt and Thomas (1957) have drawn attention to gross increases in latency in some pathological conditions of the lower motor neuron and in nerve compression syndromes. They have shown how in the latter the pressure point may be located by stimulation at two points along the nerve.

Fig. 6 shows the electromyographic responses obtained in the first dorsal interosseus on stimulation of the ulnar nerve at the level of the wrist and the elbow. The two resulting electromyograms have been projected horizontally on to a graph in which the ordinates represent distances in centimetres from the point at which the needle electrode is embedded in the muscle while the abscissa is graduated in milliseconds. The position along the vertical occupied by each record shows at what distance stimulation was effected from the point of pick-up. In this particular case, the stimulation at the wrist and elbow was carried out approximately 10 cm. and 38 cm. from the point of the needle electrode respectively. The slope of the lines joining the beginnings and the ends of the potentials obtained is a measure of the velocity of conduction. It is interesting to note that those two lines are almost parallel, which would indicate that the velocities of conduction for all the neurons are approximately the same over the stretch between the elbow and the wrist.

If 2 msec. is taken as a generous estimate of the delay at the neuromuscular junction, the

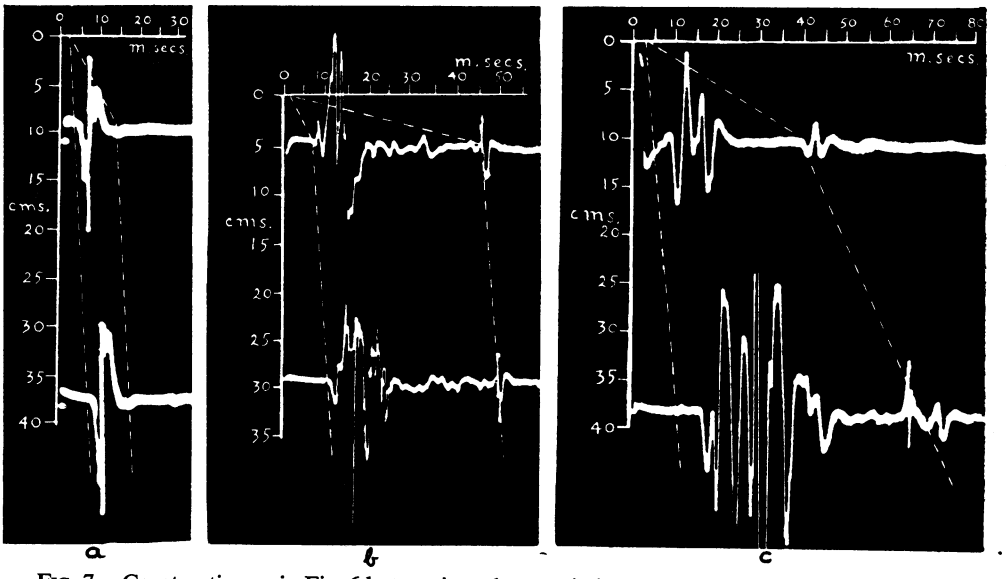


FIG. 7.—Construction as in Fig. 6 but tracings show variation according to situation of the lesion. A, normal; B, median nerve compression in carpal tunnel; C, ulnar nerve involvement in the epicondylar groove. (Time scale in msec.)

fact that both lines alter their slope to reach this point would indicate that not only does slowing down take place but that in the case of the motor units which account for the latter part of the response, the decrease in velocity is considerably greater (a reduction from 70 metres per second to 50 metres per second for the faster fibres, and from 70 metres per second to 9 metres per second for the slower ones).

Fig. 7 shows how three different conditions can affect the responses obtained on stimulation of a nerve at two levels in each case. The points to note in comparing these three sets of responses are:

(1) The latencies.

(2) The spread of the phenomenon on stimulation at the wrist.

(3) The spread on stimulation at the elbow.

Many of the perplexing phenomena encountered in electrodiagnosis appear to find their solution in the work of Paul Weiss. Briefly, he confirmed the hypothesis of Cook and Gerard (1931) and of Parker and Paine (1934) who postulated that the endoneural axoplasm of a motor neuron was formed in the anterior horn cell and was gradually propelled down the axon and that along the whole course of the axon a kind of metabolic consumption occurred.

Fig. 8 is an illustration from Weiss's paper (1956) on "The Life History of the Neuron"

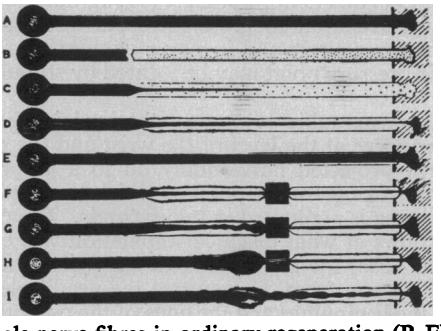


FIG. 8.—Diagrams of single nerve fibres in ordinary regeneration (B–E) and in regeneration combined with constriction (F–I), in the last case (I) followed by later release of the constriction. (From Weiss, P. (1956) The Life History of the Neuron, *J. chron. Dis.*, 3, 340, by kind permission.)

which graphically epitomizes his contentions. It is the result of six years' study involving experiments and observations on more than 100,000 nerve fibres. I can do no better than quote him. He says:

"You see here nine mature neurons, a normal one on top, the others in various stages of ordinary or modified regeneration. The nucleated cell bodies are on the left, the periphery is on the right. Lines B to E recapitulate the regenerative events following the crushing of a fibre. The proximal stump issues a thin filamentous outgrowth, which advances through the distal degenerated portion into the periphery (as seen in C), where it makes connexion with an old degenerated end-organ, as in D. The new sprout is then still very thin. Only gradually does it enlarge in width, eventually approximating full calibre. To achieve this, it must grow in protoplasmic mass about a hundredfold.

"It was this increase in girth which formed the subject of our investigations. By placing a constriction around a nerve, we can reduce the calibre of each constituent nerve fibre locally, as shown in lines F, G and H. If now we crush the fibre proximally to the constriction, a thin filament again regenerates through the constriction (in F). But as this filament keeps enlarging and attains the diameter of the constricted portion, a marked asymmetry begins to appear between the portions of the fibre lying proximally and those distally of the constriction, as you can see in G and H.

"The distal portion remains thin, and permanently so; while at the proximal end of the constriction, surplus neuroplasm piles up."

He concludes by saying:

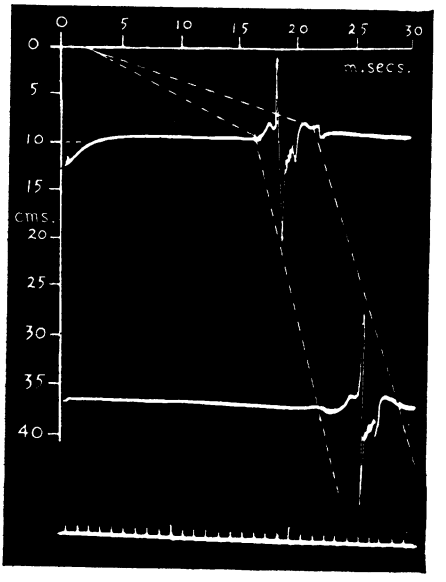
"The growth of the axon evidently occurs solely from its base, and the constriction, acting as a bottleneck, has throttled the supply of growing mass to the distal portion."

It is likely that when the position is such that the axon remains of small cross-section the velocity of conduction remains grossly reduced and the excitability low. The situation diagrammatically depicted here is probably very pertinent to that found in carpal tunnel compression, lesions of the ulnar nerve at the level of the elbow and in the palm of the hand, neurofibromata, and facial paralysis. In these conditions an increase in latency can almost invariably be demonstrated in some, if not all the motor units. In addition a considerable rise in the threshold may be observed distally to the lesion.

When dealing with a system like the one envisaged by Weiss where a cytoplasmic substance is produced at one end, propelled down a shaft and consumed at the other end, obstruction is not the only factor which will cause a depletion at the periphery. Deficient production and leakage along the conveyor or even excessive consumption would produce the same result.

Work carried out by Wohlfart (1957), Coërs (1955), and Woolf and Till (1955) has drawn attention to the dramatic phenomenon of sprouting of normal axons in the presence of denervated muscle and virtually effecting a collateral innervation of these with the formation of new neuromuscular junctions. The increase in the number of muscle fibres supplied by one motor neuron through this process must create gigantic motor units, and the caricature of the normal electromyographic pattern which we frequently observe may well be the result of this expansion coupled with temporal dispersion.

Fig. 9 shows the degree to which slowing down of impulse conduction can descend. These records were taken from a patient's first dorsal interosseus. He had a ganglion compressing the deep palmar branch of his ulnar nerve. The only motor units to respond showed a latency of 17 msec. on stimulation at the wrist, and 22 msec. on stimulation at the elbow. The reduction in velocity in this case is from 60 metres per second down to 7 and 5 metres per second distally to the lesion.



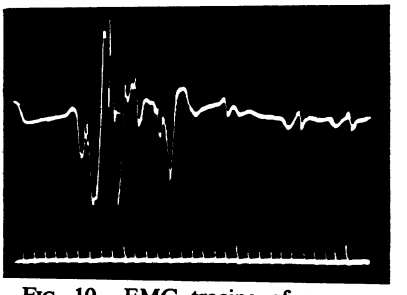


FIG. 10.—EMG tracing of response obtained in tibialis anticus on stimulation of the lateral popliteal nerve showing gross temporal scatter of the motor unit activity response.

← FIG. 9.—Construction as in Figs. 6 and 7. EMG records show gross decrease in velocity of conduction along the deep palmar branch of the ulnar nerve due to compression by a ganglion. (Time scale in msec.)

Fig. 10 shows gross dispersion in the tibialis anticus on stimulation of the lateral popliteal nerve at the level of the knee-joint. The damage in this case resulted from ischæmia caused by a tight plaster.

Electrodiagnosis is not merely a technical procedure whereby an investigator, elaborately equipped, manages to manifest audibly and visually a sort of accepted hall-mark of a pathological condition. On the contrary it is in every sense a form of exploratory operation on a community of simple systems of which the individual and collective behaviour may have some diagnostic significance.

I use the word "exploratory" because the process of electromyography is not a static one. To extract the maximum information from it some precepts must be heeded. The most important of these is that the investigator should in mind withdraw from his surroundings and project himself on to the tip of the needle electrode, or under the surface electrode. He should be conscious of the machinery around him only in so far as it tells him of the electrical conditions prevailing there, and in so far as it is capable of making some permanent records or taking samples—like relevant snaps and films in the course of some quest.

The queries which have to be and can be answered as a result of this are many, and I can list only the more important, When the needle electrode is plunged into a muscle, insertion potentials may be noted. These may themselves suggest denervation or myotonia. After this, spontaneous activity may be registered in the shape of fibrillation, positive potentials or fasciculation. To increase the chances of finding out whether such phenomena are present it

is essential to move the needle electrode about with the object of tracking down any delinquent muscle fibre or eccentric motor unit. It must be remembered that a single fibrillating muscle fibre means that at least one muscle fibre is denervated, and this may be accounted valuable evidence.

At the inception of volitional activity the electromyographic pattern should present with small amplitude potentials, and on a progressive increase in activity larger and larger potentials should make their appearance until the normal full interference pattern is obtained. A deficiency of active motor units betrays itself either by gaps in the pattern which reveal the base line of the cathode ray trace or by the presence of an easily identifiable potential which dominates the picture. If an attempt is made at bearing down on the motor unit responsible for its presence, the potential may be found to be a very large one—so large that it may necessitate cutting down the amplification. From the point of view of population census, these tactics are invaluable. What you are really doing is locating a recognizable motor unit and finding out how near or how far from it other active motor units are on full volition. There may be no others within pick-up range of your electrode, in which case a discrete motor unit activity pattern results. This identifiable motor unit may have some interesting characteristics. It may show signs of temporal dispersion or give the impression that through accretion of muscle fibres, not originally part of it, it has become enlarged. Gentle transfixion of it during activity may reveal it to be a giant. At this point you may become conscious of an inconsistency. You may remember that when you stimulated the nerve supplying this particular muscle you obtained a response which justified your anticipating a far fuller pattern on volition than that obtained. This discrepancy between what could be demonstrated on stimulation and on volition is almost diagnostic of a myelopathic lesion just "north" of the anterior horn cells. I have coined the term "motor unit apraxia" to denote this set of conditions.

On the other hand, the electromyographic pattern on volition may be full and disturb the cathode ray base line throughout, but this may be made up of low amplitude short duration potentials. Such a picture spells an internal derangement of the motor unit and may be due to a myopathy, a distal neuronitis, a myositis or a myasthenia. The use of an *agent provocateur* may now be indicated. An intravenous injection of Prostigmin or Tensilon may dramatically improve the pattern if the deficiency is due to myasthenia gravis, or it may induce a denervated muscle fibre to fibrillate and thus act as an informer.

The time may now have come to stimulate the nerve and pick up the response electromyographically. I have already dwelt at some length on this, but I should like to draw attention to the fact that latency which has been given much prominence is not the only feature of importance. The motor unit which breaks the latency may well be one with an intact motor neuron. Far more importance attaches to the "also-rans". Indeed the stragglers may well be the ones on which a diagnosis has to be based.

Stimulation of the nerve by single shocks is not the only technique employed. Tetanizing currents can be made use of to demonstrate early fatigue in myasthenia or to precipitate a protracted myotonic contraction. Again, a defective power of accommodation such as is encountered in tetany can be brought to light by applying a progressively increasing direct current over the ulnar nerve at the elbow. Action potentials may then be picked up in the intrinsic muscles of the hand with quite low values of current. Moreover, these potentials frequently show signs of rapid repetition—doublets, triplets and quadruplets are frequently noted.

Exactly ten years ago (Bauwens, 1948) I expressed the belief that the clinical application of electrophysiology had immense potentialities, but that the value of the contribution it would ultimately bring to neurology would depend in a large measure on how well it was supported during its developmental stages by the tripod made of the neurologist, the neurosurgeon and the morbid pathologist. The intervening decade has done nothing to make me alter this belief except in so far as I can now say that this support together with a great deal of encouragement has been forthcoming.

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