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ELECTRODIAGNOSIS AND ELECTROMYOGRAPHY IN CLINICAL PRACTICE*

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One of the problems confronting a clinician at the present time is the steady increase in the number of laboratory tests which he may have to use; thus the situation arises that the clinician to-day is often in the position of asking for an investigation which he only dimly understands and which requires interpretation by somebody else. The development of clinical electromyography has created just such a situation, and I should like to take the opportunity presented by this lecture to try to explain in simple terms what the electromyographer is doing and how much reliance can be placed on the results of his tests. A recent bibliography collected by Rosenfalck (1961) listed nearly a thousand articles and monographs devoted to human electromyography alone, and last year we witnessed the first International Conference of Electromyography, which was held in the University of Pavia and attended by more than a hundred delegates. It seems, therefore, that we must all accept electromyography as one of these thrusting, expanding, subspecialties, which is inevitably going to continue to grow in technical complexity, and which will also increase its demands for recognition and facilities in the future.

Before beginning to review this field I should perhaps make one point in relation to terminology. The term "electrodiagnosis" is an old and venerable one which has been used for many years to describe the electrical stimulation of muscle when this is being done as a clinical aid to diagnosis. Electromyography could be strictly interpreted to mean the recording of electrical activity from muscle either at rest or during voluntary activity, but in fact it is often used in a much wider sense. One of the recent and interesting trends in this field has been the development of techniques for stimulating motor-nerve trunks and for recording the response of muscles when they are activated artificially in this way; there is also great interest at the moment in the reflex responses of muscle to various forms of sensory stimulation. Some workers have not been satisfied with the simple recording of the electrical activity of muscle but have measured actual twitch tension and compared this with the electrical output of a muscle under various conditions. All these newer procedures can justifiably be included under the general heading of electro-Recently, however, several laboratories myography. have begun to record directly from sensory-nerve fibres and thus to obtain information which really has nothing to do with muscle at all. One continues to refer to this as a part of electromyography for lack of a better name, but it may be asked whether one should not go back

to the old term electrodiagnosis, to include all these broadly similar methods of investigating the peripheral nervous system.

The Detection of Denervation

The oldest and simplest electrodiagnostic test that is still in routine use to-day is the examination of the effects of faradic and galvanic currents on muscle. I think that this very simple test still has a legitimate place in the diagnosis of denervation provided that its limitations are understood. The faradic stimulus consists of a train of short pulses, the duration of each pulse delivered by an ordinary induction coil with a trembler being something of the order of 1 millisecond, whereas a galvanic stimulus is a continuous pulse which may be as long as a second in duration. When the motor point of a normal muscle is stimulated the intramuscular nerve fibres respond as well to the short pulses of faradism as to the long square wave of galvanism, but in denervated muscle the current must produce direct excitation of the muscle fibres themselves, and these have a much higher threshold to short pulses than to long ones. Thus, provided the test is carefully carried out with stimulation over the anatomical point of entry of the motor nerve into the muscle, loss of the response to faradism with preservation of the response to galvanism is legitimate evidence of denervation. However, the reverse is not true, as it is well known that completely denervated muscles do sometimes respond to faradic stimulation; this is particularly likely to occur if the patient has a high pain threshold so that he is able to tolerate strong shocks.

The relation between the duration of a stimulus and the intensity necessary to excite is best described by plotting a full intensity-duration or strength-duration curve. This requires a stimulator capable of delivering pulses of pre-set duration which can be varied from relatively long ones at the galvanic end of the scale to pulses of short duration, even shorter than those produced by the old-fashioned faradic coil. One then finds the relationship illustrated in Fig. 1, that the shorter the duration of a stimulus the higher must be its intensity in order to excite. While this general relationship is true for all excitable tissues, the actual values of intensity and duration are quite different for nerve and muscle. This is clearly shown by Fig. 1, for which I am indebted to Professor Ritchie. His patient had a complete radial-nerve lesion requiring suture, and the upper curves are from abductor pollicis longus on the affected side. At the bottom is a normal curve from abductor pollicis longus in the opposite arm. At the

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top right is the curve for the paralysed muscle at the time of nerve suture, and it can be seen that the muscle did not respond to the shorter duration pulses, whereas in the normal muscle shown at the bottom the intramuscular nerve fibres were almost as easily excited by short duration pulses as by long ones. Further curves were plotted for the paralysed muscle during reinnervation, one at 16 weeks, one at 20 weeks, and a third at 24 weeks. It will be noticed that they show kinks or discontinuities, suggesting that they are in fact composite curves combining features of both the normal and the denervated type of response.

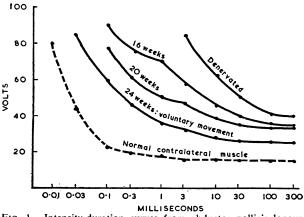


FIG. 1.—Intensity-duration curves from abductor pollicis longus in a patient with a radial-nerve lesion in the upper arm, showing recovery after nerve suture. (From Ritchie, 1>54.)

Strength-duration curves were introduced into clinical neurology by Lord Adrian during the first world war (Adrian, 1916, 1917), and since that time they have had an accepted place in clinical work, perhaps their greatest value being in the assessment of traumatic peripheral nerve lesions. To obtain stimulating pulses of known duration Adrian used the Keith Lucas pendulum, but to-day there are many electronic stimulators available for this purpose. This should make the technique very easy, but in fact it still has certain limitations: not only is the procedure time-consuming but it is also subject to observer error. Each point on a strength-duration curve is obtained by setting the pulse duration on the stimulator and then gradually increasing the stimulus strength until a faint twitch of the muscle under the The stimulating voltage or electrodes can be seen. current strength is then read from a meter on the stimulator. The obvious source of error here is in detecting the minimal muscle twitch, and considerable practice is needed for consistent results to be obtained. It was for this reason that the development of electromyography during the second world war seemed likely to provide a simpler and more objective method of detecting denervation, and we must now consider whether this has proved to be the case.

Electromyography involves the insertion of a recording needle into a muscle in order to observe its electrical activity under various conditions. For this purpose most workers use the concentric or co-axial needle (Adrian and Bronk, 1929) in which the core is insulated from the shaft of the needle but bared at the tip; this makes it possible to record activity from a relatively small area deep in the muscle. After appropriate amplification the potentials are displayed on a cathode-ray tube so that the characteristic features of muscle-fibre or motor-unit activity can be recognized. The term motor unit was first used by Sherrington

(1929) to describe the functional unit of normal muscular activity, which is one anterior horn cell, its axon, and the bunch of muscle fibres which it innervates. The actual number of muscle fibres in a motor unit varies in different muscles, but for the limb muscles of man the figure is probably several hundred (Feinstein, Lindegaard, Nyman, and Wohlfart, 1955), so that on the electromyograph motor-unit action potentials are easily distinguished from those of single muscle fibres. Fig. 2 shows a motor unit discharging at low frequency

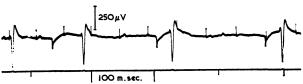


FIG. 2.—Tracing from a partly denervated muscle during minimal voluntary contraction. In addition to a single motor-unit potential repeating at low frequency, the record shows much smaller and briefer potentials due to repetitive discharge of a single muscle-fibre. On three occasions "positive sharp waves" (Kugelberg and Petersén, 1949) can be seen to cause a downward deflection of the trace.

during minimal voluntary contraction, and it also shows the very much smaller and briefer potentials due to a single muscle fibre appearing on the same trace. Muscle fibres with an intact nerve supply do not normally discharge except in conjunction with the other fibres of their own motor unit, and this particular record was taken from a partially denervated muscle in which some of the motor-nerve fibres and end-plates had already degenerated, releasing the muscle fibres from neural control and producing this tendency to isolated musclefibre activity which is called fibrillation.

Since fibrillation is easily recognizable in an electromyographic tracing and is something which is never seen in normal muscle, it might be thought that this would be the answer to the diagnostic problem of detecting partial denervation, and that strength-duration curves are no longer necessary. Unfortunately this is not true; although fibrillation is undoubtedly a valuable sign of denervation, it has important limitations which must be considered. The first of these is that spontaneous fibrillation does not appear until the terminal part of the motor axon and the motor end-plate have degenerated, so that a period of perhaps three or even four weeks may elapse after an acute peripheralnerve injury before it can be detected (Weddell, Feinstein, and Pattle, 1944). On the other hand, the electrical threshold of the degenerating nerve fibre begins to change after a much shorter period, such as three or four days (Landau, 1953; Bowden, 1954), so that stimulation will show that Wallerian degeneration is occurring long before fibrillation appears.

A further danger in relying too much on fibrillation as a diagnostic aid arises from the fact that in some cases of partial denervation fibrillary activity is not seen. In a series of about 200 cases collected by Richardson (1951), in which the presence of partial denervation was established by other means, fibrillation was absent in approximately 20%, whereas abnormal strengthduration curves were present in nearly all of them. The reason for this is by no means clear. The presence or absence of fibrillation does not seem to be related to the severity of denervation, and it is not an uncommon experience to sample a muscle with only one or two surviving motor units and yet fail to find fibrillation. It is probably true that fibrillation is least likely to be seen in partial denervation of long standing, and in such cases it has been suggested that lateral sprouting of nerve fibres from the terminal parts of surviving motor nerves may result in the incorporation of denervated muscle fibres into surviving motor units.

This theory has been used to explain both the frequent absence of fibrillation in chronic partial denervation and also the fact that the surviving motor-unit potentials are often of much greater amplitude and duration than normal (Wohlfart, 1958). Both of these facts could be explained by the incorporation of extra muscle fibres into surviving units, but I think that the situation is probably For example, we quite more complicated than this. often see denervated muscle fibres whose mechanical excitability has increased so that insertion of the exploring needle is sufficient to produce showers of fibrillation potentials. This is usually called insertion activity, and when it is profuse and long-lasting one must assume that it is due to denervated muscle fibres as it is never seen to this degree in normal innervated muscle (Kugelberg and Petersén, 1949). In such cases, however, fibrillary activity does not persist indefinitely in the absence of mechanical stimulation and the muscle gradually becomes silent until the needle is moved again. In some cases of chronic partial denervation only a few short-lived fibrillation potentials are seen on needle movement, in others none at all; so there appears to be a continuous gradation in the excitability of the denervated muscle fibre, ranging from a condition in which it discharges spontaneously at rest, through an intermediate state in which it is liable to discharge if distorted mechanically, to the other end of the scale, at which it shows an excitability little if at all greater than that of a normal innervated fibre.

It was originally suggested by Denny-Brown and Pennybacker (1938) that the spontaneous contraction of denervated fibres was due to an exaggerated sensitivity to the small amount of acetylcholine present in the muscle, but I doubt whether this can be regarded as an adequate explanation of the phenomenon, and certainly attempts to increase the amount of fibrillation in denervated muscle by systemic injection of either neostigmine or edrophonium (" tensilon ") have proved disappointing. One much more promising approach to this problem is to use an external pulse of long duration in order to evoke fibrillation; in other words, to use a galvanic stimulus to the muscle and to record the The first person to fibrillation during the stimulus. suggest that this might be clinically useful was Landau (1951), who wrote: "The evanescent and slight effect of prostigmine in our experiments compared with the effectiveness of direct current stimulation suggests that the latter may be useful in the clinical situation where it is important to demonstrate fibrillary activity.'

The difficulty here is a purely technical one—namely, that one is attempting to record potentials of only a few millionths of a volt from a muscle while at the same time applying to it an external stimulus which may be ten or a hundred volts in amplitude. Conventional stimulators are not suitable for this purpose, but in 1958 Humbert and his colleagues at the Salpétrière described a method of evoking fibrillation which they considered sufficiently reliable for use as a routine test. Profiting by their experience, Thomas and Morton (1962) have recently developed a technique at the National Hospital, Queen Square, which takes matters a step further, in that they can now carry out strength-duration curves and actually record the muscle response on the cathode-

ray tube instead of merely observing the muscle twitch by eye. Since motor-unit potentials are easily distinguished from fibrillation on the cathode-ray tube, one can therefore record two separate strength-duration curves from a partially denervated muscle, one curve being the threshold curve for muscle fibres and the other the curve for surviving motor units. This should prove to be a much more certain and accurate process than the plotting of conventional curves in which the muscle response to stimulation is detected with the naked eve.

Electromyography in Muscle Disease

In muscular dystrophy or in polymyositis the role of the electromyograph might appear at first sight to be a relatively simple one. In these primary diseases of muscle there is a diffuse loss of active muscle fibres, so that, although the number of motor units will not be significantly reduced, at any rate in the early stages, each unit may be expected to contain a smaller number of muscle fibres than normal. This leads to the classical electromyographic sign of muscle disease—namely, the appearance of motor-unit potentials of small amplitude, with a brief duration and with a rather ragged polyphasic outline which may be contrasted with the smooth and relatively simple contour of a normal motor unit potential.

Fig. 3 (top) shows a single motor unit repeating at low frequency, the record being taken from a healthy muscle during minimal voluntary contraction; it demonstrates the typical amplitude and duration of a normal motor unit and also its simple triphasic wave-Fig. 3 (bottom) is taken from a patient with form. muscular dystrophy, and it can be seen that although motor units are present during voluntary contraction in normal numbers each of these is a small sharp potential suggesting that very few muscle fibres are left out of the several hundred which go to make up the normal motor unit. In such a case the electromyographic findings are quite clear-cut and have obvious diagnostic value, and there is no doubt that the more advanced the condition the easier it is to distinguish between these small myopathic or myositic potentials and the large surviving motor units of chronic partial denervation.

In polymyositis and dermatomyositis the situation is complicated by the fact that spontaneous fibrillation sometimes occurs and could lead to an incorrect diagnosis of denervation, so that in these two conditions

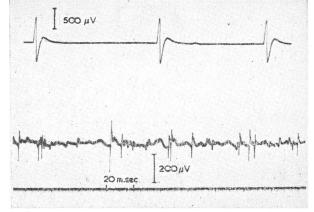


FIG. 3.—Top: A normal motor unit in bieeps during minimal voluntary contraction. Bottom: Myopathic motor units in a patient with muscular dystrophy. (From G.Iliatt, 1957.)

it becomes particularly important to study the appearances of the motor units themselves and to recognize the small brief or "broken-up" potentials of primary muscle disease. In mild or early cases, however, the recognition of slight changes in motor-unit size and duration becomes a matter of considerable difficulty. In theory there is no reason why a large number of motor units in a given muscle should not be examined in turn and each one displayed on the oscilloscope and photographed so that its amplitude and duration can be measured (Kugelberg, 1949; Pinelli and Buchthal, 1953). A careful study of this sort was carried out by Sanderson and Adey (1952), who recorded 80 separate motor-unit potentials from the triceps muscle of a patient with thyrotoxic myopathy, and then repeated their investigation at intervals during the course of treatment with methylthiouracil (Fig. 4).

Their patient was a man aged 68 admitted with an 18-month history of loss of several stones in weight and a six-month history of cramps and weakness of the limbs. Examination in May, 1951, revealed diffuse enlargement of the thyroid, with tachycardia and gross wasting and weakness of the proximal muscles of the limbs. His B.M.R. was +30%. On methylthiouracil the patient gained four stones (25.4 kg.) in five months, with equally rapid improvement in muscle power. His B.M.R. fell to -4% and his tachycardia disappeared.

The normal mean duration of motor-unit potentials in the triceps muscle is approximately seven milliseconds, but it can be seen from Fig. 4 that before treatment most of the potentials in this patient were less than four milliseconds in duration; after four months' treatment there had been an obvious shift to the right, the mean duration returning to a normal figure of about seven milliseconds.

Unfortunately the analysis of individual motor-unit potentials on this scale is such a laborious procedure and R. TRICEPS MUSCLE

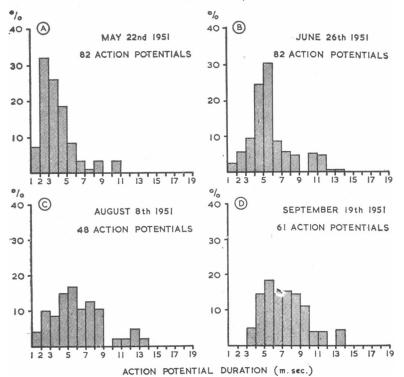


FIG. 4.—Histograms of motor-vnit action potential duration in a patient with thyrotoxic myopathy before (A) and during treatment (B-D). (From Sanderson and Adey, 1952.)

so painful for the patient that it is rarely used in routine work. All that an electromyographic report usually provides is some sort of judgment based on a visual impression of motor-unit potentials as they appear on the oscilloscope, and it is not uncommon for two different examiners to form opposite opinions regarding the presence or absence of a myopathic change. While this may be forgiven in a routine report, it certainly should not be tolerated in published work, and nothing does greater disservice to electromyography than to publish this sort of clinical impression in such a form that it appears to be an incontrovertible scientific fact.

As an example of this I may quote a recent paper on polymyositis in which it was stated that 80% of a large series of patients showed electromyographic evidence of myositis in one or more muscles; nowhere in that paper was it stated how many motor-unit potentials were measured for each muscle, or what the range of normal variation might have been by the technique used. The authors may have been perfectly correct in their conclusion, but surely a problem of this importance requires much more precise and detailed treatment; it should have been possible to display, photograph, and measure a sufficient number of motor-unit potentials to build up a statistically significant picture of the muscles under investigation. The results would be influenced by the size and shape of the recording needle and by the physical characteristics of the amplifying system, in particular by its input impedance and frequency reponse (Buchthal, Guld, and Rosenfalck, 1954), so that this information must also be given in any published paper if the results are to be of value to workers in other laboratories.

However, when all this has been said it must be admitted that even in the best hands the measurement of individual motor-unit potentials is a procedure which

is inevitably both time-consuming to carry out and exacting for the patient. For this reason some form of automatic frequency analysis may eventually prove preferable, so that the general sharpness of contour of the trace can be expressed quantitatively without the necessity of visualizing single motor-unit potentials in isolation from each other. An audiofrequency spectrometer was first used for this purpose by Walton in 1952, and although there may be certain theoretical objections to frequency analysers in electromyography it is rather disappointing that since Walton's paper was published ten years ago there has been so little in the way of further development. This is a problem for the electronic engineer as much as for the doctor, and all that need be said here is that further work in this field is urgently required.

Motor- and Sensory-nerve Conduction

I should now like to turn to another aspect of electrodiagnosis—namely, the study of nerve conduction. This is essentially different from the techniques which have been considered so far, in that we are no longer so interested in the muscles themselves but more concerned with the excitability and conduction velocity of the motor and sensory fibres of the peripheral-nerve trunks in the limbs. This is a field which has been the subject of several recent reviews (Lambert, 1960; Thomas, 1961; Gilliatt, 1961), and I shall restrict myself to considering the type of information it may provide in certain specified clinical situations.

At the Middlesex Hospital the commonest clinical condition requiring nerve-conduction tests has been the carpal-tunnel syndrome. In the majority of these patients slowing of conduction can be demonstrated between the wrist and the thenar muscles with relatively normal conduction velocity in the same nerve fibres at a higher level in the arm (Simpson, 1956; Thomas, 1960). For examining the segment of nerve distal to the wrist the standard stimulating and recording arrangement shown in Fig. 5 is used, and the degree of nerve damage is classified as mild, moderate, or severe according to the increase in latency which is found. This is shown in Fig. 6, in which a normal response is contrasted with delayed responses of varying severity

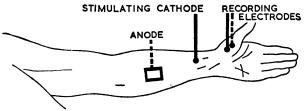
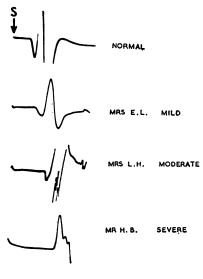


FIG. 5.—Position of stimulating and recording electrodes for estimation of motor latency.

from carpal-tunnel patients. We find the latency of the muscle response to be a voluable objective criterion of the severity of median-nerve damage, and one which enables us to assess the long-term effects of different forms of treatment. In Fig. 7, for example, are the recovery curves from two groups of patients. One group was treated by surgical division of the flexor retinaculum, the operations being carried out by Mr.



200 c/s FIG. 6.-Records from abductor pollicis brevis with median-nerve stimulation just above the wrist. Stimulus (S) at com-mencement of trace in each case. Top record shows a normal response; lower records show mild, moderate, and severe conduction delays in patients with the carpal-tunnel syndrome. (Form Good-

man and Gilliatt, 1961.)

Valentine Logue. The other group received local steroid therapy, three injections of prednisolone into the carpal tunnel being given in each case by Dr. Β. 1 Foster. Patients in the two groups were classified as mild, having moderate, or severe lesions according to the conduction delay before treatment, and in Fig. 7 subsequent latencies plotted are as percentages of the pretreatment figure. From these results it can be seen that whereas in the surgical cases the latencies

fell rapidly after operation and stayed down, the prednisolone cases showed a tendency to relapse towards the end of the first year.

In the cases shown in Fig. 7 the results of surgery were uniformly satisfactory, latencies falling abruptly in the first few months after operation and finally reaching a value within the normal range in every case (Goodman and Gilliatt, 1961). In Fig. 8, however, are the results from a patient with a carpal-tunnel syndrome who had been operated on without clinical improvement, and in whom our measurements showed that nerve conduction was also unaffected by the operation. On inquiring further, it appeared that the flexor retinaculum had not been divided under direct vision but by a blind technique through a small and rather high skin incision. After the usual diplomatic exchanges which occur in such cases, the median nerve was re-explored by Mr. Valentine Logue through a much larger incision, and it was found that the distal fibres of the flexor retinaculum had been left intact. It can be seen from Fig. 8 that there was prompt recovery of normal latency after the second operation.

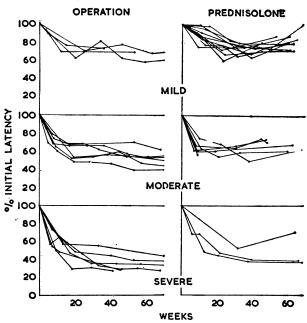


FIG. 7.-Results of prednisolone injection compared with the rig. 7.—Results of predinscione injection compared w results of surgery in patients with mild, moderate, or conduction delays due to the carpal-tunnel syndrome. La shown as percentages of initial figure in each case. Goodman and Foster, 1962.) Latencies (From

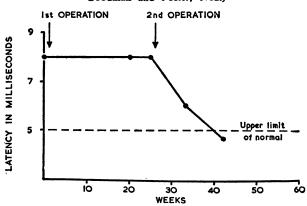


FIG. 8.—A patient with the carpal-tunnel syndrome requiring two operations. Motor latency unchanged by the first operation. but falls to normal after the second. (From Goodman and Gilliatt, 1961.)

In patients with ulnar-nerve lesions in the hand it is possible to demonstrate slowing of conduction in the motor fibres of the ulnar nerve below the wrist, with preservation of relatively normal conduction velocity above this level (Simpson, 1956; Ebeling, Gilliatt, and Thomas, 1960). In the case of ulnar-nerve lesions at the elbow, however, the situation is rather different. There may be slowing of nerve conduction confined to that part of the nerve in the region of the elbow (Simpson, 1956) or, alternatively, conduction may be slowed throughout the length of nerve distal to the lesion (Gilliatt and Thomas, 1960). Such findings are most likely to be present in severely affected patients, and in milder cases motor-conduction velocity is often normal. In this situation it may be helpful to stimulate the sensory fibres and to record the ascending volley directly from the nerve trunk itself. This can be done at several different levels in the arm; for example, the digital nerve fibres may be stimulated through small ring electrodes wrapped round the fifth finger, and in this case the recording electrodes are placed over the ulnar-nerve trunk at the level of the wrist. To examine conduction at a higher level the ulnar nerve is stimulated at the wrist and the action potential of the ascending volley can then be recorded through electrodes placed a few centimetres above the elbow. Finally, one may stimulate the nerve just above the elbow and record the ascending action potential from the axilla itself.

Fig. 9 shows the results of these procedures in a patient with an ulnar-nerve lesion at the elbow. Records were made from both the median and the ulnar nerve, and it can be seen that a well-formed action potential was recorded from the median nerve at all three levels ; the ulnar potential, however, was almost absent when the stimulus was put in below the elbow, whereas a normal potential was recorded when both the stimulating and the recording electrodes were placed above elbow level. This was not in fact a patient with a severe ulnar-nerve lesion but one in whom there was very little wasting of the intrinsic muscles and no thickening of the nerve in the ulnar groove, so that this type of examination can make a real contribution to the diagnosis of mildly affected patients. The importance of this needs no emphasis, as it is in these mild early

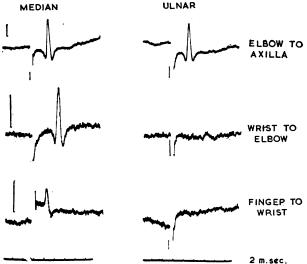
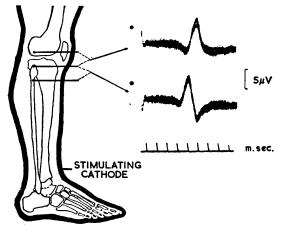


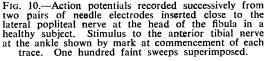
FIG. 9.—Median- and ulnar-nerve action potentials in a patient with a mild ulnar-nerve lesion at the elbow. Stimulus shown by artifact in each trace. Calibration 25 μ V. (From Gilliatt, 1961.)

cases that either decompression or transposition of the nerve gives the best results.

In addition to the investigation of localized peripheralnerve lesions, conduction tests have an important place in the diagnosis of generalized peripheral neuritis (Henriksen, 1956; Johnson, Guyton, and Olsen, 1960). In such cases it may be an advantage to study the lower limbs; for example, it is possible to measure conduction velocity in the motor fibres to extensor digitorum brevis by stimulation of the peroneal nerve at the head of the fibula and of the anterior tibial nerve at the ankle, the recording electrodes being placed over the muscle on the dorsum of the foot. To study sensory conduction we stimulate the anterior tibial nerve at the ankle, and record the action potential of the ascending volley from the peroneal nerve at the head of the fibula (Gilliatt, Goodman, and Willison, 1961); Fig. 10 shows a normal result. Even in mild cases of peripheral neuritis these tests may show significant abnormalities. The accompanying Table, for example, shows the results in five diabetic patients with sensory disturbances in the feet but without clinical evidence of muscle wasting or weakness. It can be seen that nerve action potentials were reduced or absent in all of them, and that in two patients there was a substantial reduction in motorconduction velocity although no clinical weakness had been detected.

There is at least one possible source of error in this technique which must be faced if it is to be used as a reliable clinical tool, and this is the problem of the nerve threshold. In these patients the threshold of nerve fibres to electrical pulses is usually raised; indeed, it is sometimes raised to such an extent that it is not possible to activate motor units by electrical stimulation through the skin although the patient himself is able to do so





Conduction in Lateral Popliteal Nerve in Patients with Diabetic Neuropathy (From Gilliatt and Willison, 1962)

Case	Motor Velocity (m/sec.)	Nerve Action Potential	
		Velocity to Peak (m/sec.)	Amplitude (µV)
1 2 3 4 5	33·0 39·0 34·0 18·5 15·2	Absent 33 (approx.) Absent ,,	Absent 1.0 Absent
ormal range	35.6-63.5	33.8-51.8	2-15-5

on voluntary effort. In the case of sensory fibres, this means that failure to record a nerve action potential need not be due to failure of conduction or to dispersion of the volley, but merely to the fact that the stimulus was not adequate to excite the fibres in the nerve trunk. Downie and Newell (1961) failed to find nerve action potentials in a number of diabetic patients, many of them without signs of diabetic neuropathy. This is not entirely in keeping with our own experience, and from their paper it seems likely that they were using a stimulus which was only just adequate for nerve fibres of normal threshold, and that in a proportion of their diabetic patients they were merely finding a raised threshold rather than demonstrating a defect of propagation of nerve impulses. It is clear that a lot more attention will have to be paid to the design of stimulators and to the standardization of stimulating pulses if these newer tests are to give comparable results in different laboratories.

This brings me to the final point which I should like to make in this lecture. Electromyography to-day is being carried out by a variety of workers with widely differing backgrounds and clinical interests. No special qualifications are required other than an interest in this type of work. Apparatus also varies from clinic to clinic; it may be home-made or supplied by one of several different commercial firms. It is therefore hardly surprising that there are differences of opinion and interpretation such as have been mentioned earlier in this lecture. While this may be accepted in a subject which is advancing rapidly, there is no doubt that there must come a time for consolidation and for clinical exploitation of what has been discovered. If patients are to be referred in increasing numbers for electromyography, the value of the investigation will be greatly increased if the reports can be based on standardized techniques with uniform terminology and accepted criteria of abnormality. We are thus facing certain basic problems of organization in this field: Who should be doing these tests ?; What sort of training should they have ?; What sort of apparatus should they use ? These questions are not easily answered, but they require our urgent attention and thought if the full potentialities of this type of investigation are to be realized.

Summarv

Some electrical methods which exist for the detection of denervation have been reviewed. In the case of partial denervation the advent of electromyography should not be regarded as having diminished the usefulness of strength-duration curves, and in a doubtful case both techniques may be required.

In the diagnosis of primary muscle disease electromyography still has limitations which should be recognized by those referring patients for examination.

Nerve-conduction tests constitute a recent addition to the repertory of the electrodiagnostic laboratory. They are likely to be particularly helpful to the clinician in the case of patients with localized peripheral-nerve lesions or with polyneuritis. In this rapidly expanding field there is urgent need for standardization of both equipment and terminology.

I am grateful for permission to reproduce figures and tables from the following publications: Annals of Physical Medicine; Journal of Neurology, Neurosurgery and Psychiatry; M.R.C. Report on Peripheral Nerve Injuries;

Modern Trends in Neurology; Electrodiagnosis and Electromyography.

REFERENCES

- Adrian, E. D (1916). Brain, **39**, 1. (1917). Arch. Radiol. Electrother., **21**, 379. and Bronk, D. W. (1929). J. Physiol. (Lond.), **67**, 119. Bowden, R. E. M. (1954). In M.R.C. Report on Peripheral Nerve Injuries. H.M.S.O., London. Buchthal, F., Guld, C., and Rosenfalck, P. (1954). Acta physiol. scand., **32**, 200. Dennyk Revers D. and Pennyhacker, J. B. (1938). Brain, **61**, 311.
- Denny-Brown, D., and Pennybacker, J. B. (1938). Brain, 61, 311. Downie, A. W., and Newell, D. J. (1961). Neurology, 11, 876. Ebeling, P., Gilliatt, R. W., and Thomas, P. K. (1960). J. Neurol.

- Ebeling, P., Gilliatt, K. w., and House, *Psychiat.*, 23, 1.
 Feinstein, B., Lindegaard, B., Nyman, E., and Wohlfart, G. (1955). Acta anat. (Basel), 23, 127.
 Gilliatt, R. W. (1957). In Modern Trends in Neurology, edited by D. Williams, 2nd series. Butterworth, London.
 (1961). In Electrodiagnosis and Electromyography, edited by S. H. Licht, 2nd edition. Licht, New Haven, Conn.
 Goodman, H. V., and Willison, R. G. (1961). J. Neurol. Psychiat. 24, 305. Goodman, H. V., and Willison, R. G. (1961). J. Neurol. Psychiat., 24, 305.
 and Thomas, P. K. (1960). Ibid., 23, 312.
 and Willison, R. G. (1962). Ibid., 25, 11.
 Goodman, H. V., and Gilliatt, R. W. (1961). Ann. phys. Med., 6, 137

- 6, 13
- and Foster, J. B. (1962). Ibid., 6, 287. Henriksen, J. D. (1956). M.S. (Phys. Med.) Thesis. University of

- Henriksen, J. D. (1956). M.S. (Phys. Med.) Thesis. University of Minnesota.
 Humbert, R., Dehouve, A., and Laget, P. (1958). J. Radiol. Electrol., 39, 737.
 Johnson, E. W., Guyton, J. D., and Olsen, K. J. (1960). Arch. phys. Med., 41, 185.
 Kugelberg, E. (1949). J. Neurol. Psychiat., 12, 129.
 and Petersén, I. (1949). Ibid., 12, 268.
 Lambert, E. H. (1960). Res. Publ. Ass. nerv ment. Dis., 38, 247.
 Landau, W. M. (1951). Electroenceph. clin. Neurophysiol., 3, 169.
- (1953). J. Neurosurg., 10, 64. Pinelli, P., and Buchthal, F. (1953). Neurology (Minneap.), 3, 347.
- 347.
 Richardson, A. T. (1951). Proc. roy. Soc. Med., 44, 992.
 Ritchie, A. E. (1954). In M.R.C. Report on Peripheral Nerve Injuries. H.M.S.O., London.
 Rosenfalck, P. (1961). Bibliography on Electromyography. Disa, Herlev, Denmark.
 Sanderson, K. V., and Adey, W. R. (1952). J. Neurol. Psychiat., 15, 200.

- 15, 200. Sherrington, C. S. (1929). Proc. roy. Soc. B, 105, 332. Simpson, J. A. (1956). J. Neurol. Psychiat., 19, 275. Thomas, P. K. (1960). Neurology (Minneap.), 10, 1045. (1961). Postgrad. med. J., 37, 377. and Morton, H. B. (1962). Proc. roy. Soc. Med., 55, 406. Walton, J. N. (1952). J. Neurol. Psychiat., 15, 219. Weddell, G., Feinstein, B., and Pattle, R. E. (1944). Brain, 67, 129

Wohlfart, G. (1958). Neurology (Minneap.), 8, 175.

At the recent conference of the National Society for Clean Air at Harrogate progress towards clean air in Europe was discussed in papers from France, Holland, West Germany, and the Republic of Ireland, with contributions from the United Kingdom on progress in medical research and activities now beginning in Northern Ireland. The chairman, Dr. J. L. BURN, said that a Common Market for the discussion of Europe's air pollution problems was being established, and it was hoped that this would lead before long to some form of federal organization between the bodies working for clean air. Air-and the pollution in the air-recognized no frontiers or customs barriers. From Holland came a report of a scientifically controlled investigation on the pollution of one particular area, Geleen; and from France a paper on the considerable progress being made in that country in investigations, research, and organization. West Germany had sent in two reports, one on progress in countering the pollution arising from the new processes in steel manufacture, and the other on the directives that have been made, in detail, for standards or limits of pollution from different sources. The report from Dublin concerned the new legislation for clean air that was being enacted, while the Northern Ireland report, from the honorary secretary of the Society's new division there, was concerned with the first steps to mobilize public opinion towards a law similar to the Clean Air Act, which applied only to England, Scotland, and Wales.