Electrodiagnosis of Peripheral Nerve Lesions and Infantile Paralysis

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Among the other advances in the field of physical therapeutics, the Great War has brought opportunities for the observation and treatment of peripheral nerve injuries on a very large scale. Many important investigations as to methods of electrodiagnosis and of the phenomena of electrophysiology and nerve regeneration were carried on, apparatus for diagnosis and electrotherapy was greatly improved, and neuro-surgeons and other medical men became convinced for the first time that advanced physical methods of treatment are fully as important as the preceding surgery.

It remains for us now to apply the results of wartime experience to the manifold injuries of civil life. The relentless warfare of industrial activities alone brings a great number of peripheral nerve injuries and, owing to the comparative simplicity of their origin and the usual lack of complications, there ought to be no delay in the early recognition of such injuries. If recognized early, appropriate surgery, supporting treatment and effective methods of physical therapy can be employed at once, and thus the splendid results of the war hospitals duplicated. Unrecognized and unskillfully treated nerve injuries are the greatest contributors towards permanent maiming of individuals, and for this reason all medical men should be familiar with their early recognition and the possibilities of treatment.

During the past three years the following cases of peripheral nerve injuries have come for the purpose of electrodiagnosis or treatment to the Physiotherapy Department of the Reconstruction Hospital:

Nineteen further cases, which do not belong to this classification, but which came under our observation and treatment, were: *Myopathies:* two cases of Volkman's contracture of forearm; one case of progressive muscular distrophy. *Traumatic neuroses:* one case of upper extremity, one case of pseudoparaplegia, one case of occupational neurosis of hand. One case of myelitis (post pneumonia), one case of amyotrophic lateral sclerosis, eleven cases of hemiplegia.

All cases of paralysis due to poliomyelitis were included in this series, as this form of paralysis is closely related to other nerve injuries, being also a lesion of the lower motor neuron, the anterior horn cell, while the former are due to injuries of the peripheral nerve fibre. Both types of lesions show the same kind of degeneration, and are therefore diagnostically and therapeutically uniform. The best proof of this statement is the fact that any modern clinic with its superior equipment for physical therapy, will draw a large number of cases of paralysis following poliomyelitis, our own institution being evidence of this experience.

OBJECTS OF ELECTRODIAGNOSIS

The object of electrodiagnosis in peripheral nerve lesions and infantile paralysis is:

1. To amplify or to corroborate clinical findings by electrical ones, affording objective evidence in place of subjective signs, and to assist in differentiating a central lesion or malingering.

[1] Read before the Thirty-fourth Annual Meeting of the American Electrotherapeutic Association in New York City, September 10, 1924.

Cases of nerve injuries accompanied by paralysis

CASE NUMBER	DIAGNOSIS	LOCATION	ETIOLOGY
1	Brachial plexus injury, contusion	L.	Dislocated shoulder
2	Brachial plexus injury, contusion	R., L.	Pressure of straight jacket (alcoholism)
3	Brachial plexus injury, compression	R.	Cervical rib operation
4	Brachial plexus injury, laceration	R.	Cervical rib operation
5	Brachial plexus injury, compression	L.	Fractured humerus and clavicle
6*	Brachial plexus injury, division	R.	Gun shot wound
7	Brachial plexus injury, contusion	L.	Erb's palsy, birth injury
8	Brachial plexus, toxic degeneration	L.	Lead poisoning
9	Circumflex nerve, compression	R.	Dislocated shoulder
10	Circumflex nerve, contusion	R.	Sprain of shoulder
11	Musculospiral nerve, toxic degeneration	R.	Osteomyelitis after typhoid fever
12*	Musculospiral nerve, division	L.	Fractured humerus
13*	Musculospiral nerve, division	R.	Wood chopping
14	Musculospiral nerve, contusion	L.	Fluoroscopic examination
15	Musculospiral nerve, contusion	R.	Heavy lifting
16	Musculospiral nerve, contusion	R.	Fall over arm
17	Musculospiral and ulnar nerves, compression	R.	Sleep with arm behind head: alcoholism
18	Ulnar and median nerves, compression	L.	Fractured humerus, radius and ulna
19	Ulnar and median nerves, compression	R.	Fractured radius and ulna
20	Ulnar and median nerves, toxic degeneration	L.	Sepsis of forearm
21	Ulnar nerve, division	L.	Glass cut at wrist
22	Ulnar nerve, laceration	R.	Nail in elbow
23	Ulnar nerve, compression	L.	Fractured ulna
24*	Sciatic nerve, division	R.	Gun shot wound
25*	Common peroneal nerve, division	L.	Fall
26	Sciatic nerve, toxic degeneration	L.	Alcohol injection for pain
27	Sciatic nerve, contusion	R.	Fall on ice
28	Sciatic nerve, toxic degeneration	L.	Lumbar myositis
29	Common peroneal nerve, contusion	R.	Gun shot wound
30	Deep peroneal nerve, division	L.	Ice chopping
31	Deep peroneal, compression	L.	Pressure of cast
32	Common peroneal, compression	L.	Automobile accident
33	Deep peroneal, compression	R.	Fall
34	Deep peroneal, compression	L.	Fracture of femur
35	Deep peroneal, contusion	L.	Fracture of femur
36	Deep peroneal, toxic degeneration	R.	Myelitis, lumbar
37	Facial nerve, infections, degeneration	L.	Exposure
38	Facial nerve, infections, degeneration	R.	Exposure
39–48	Monoplegia of lower extremities		Poliomyelitis
49–51	Diplegia of both lower extremities		Poliomyelitis
52–63	Paraplegia		Poliomyelitis
64	Circumflex nerve, contusion	L.	Hit by a plank, dislocation of acromioclavicular joint

*Denotes nerve suture done.

2. To determine the severity and the site of a lesion, to prove or disprove complete loss of conductivity in a peripheral nerve lesion, or full degeneration of a muscle in infantile paralysis.

3. To be a guide in the prognosis: and

4. To enable us to outline the most efficient plan of therapy.

ELECTRICAL CHANGES IN NERVE INJURIES

When an injury occurs to a peripheral nerve trunk at any place below its spinal origin, the following may result:

1. Complete division.
2. Partial division (laceration).
3. Compression (by scar tissue or callus).
4. Contusion (bruising).

Modern researches in nerve degeneration and nerve regeneration have given a complete picture of the events following either one of these possibilities, clinically and histologically. After any injury producing severance of continuity in a peripheral nerve trunk, the entire length of the nerve fibres distal to the point of injury inevitably degenerates—not even immediate nerve suture can prevent it. Regeneration is effected by descent of new axis cylinders from the intact central end at a rate of one to two millimetres a day. Restoration of the nerve function takes place much later than the anatomical regeneration. It is also known that toxic injury (lead, tobacco, alcoholic poisoning or exposure) might cause similar damage (toxic degeneration) to a peripheral nerve. Even if the continuity of a nerve is not destroyed, the function of the axones as to conduction may be lost, and therefore, clinically, the immediate result may be a total paralysis of the nerve and its muscular distribution, and we are called upon to assist in the differentiation of the condition on the basis of our muscle and nerve tests.

Normal muscle or motor nerve, stimulated by faradic current (alternating, asymmetrical, high tension current), will remain in continuous contraction (tetanus) as long as the current flows. Normal muscle or motor nerve stimulated by a galvanic (constant) current will respond by a sharp, brisk contraction at the moment when the current commences to flow (closure of switch); no contraction occurs while the current is flowing; when the current ceases to flow (opening of switch) a second twitch occurs. The response to the galvanic current is most marked when the active electrode is the negative (kathode) and in all of our nerve testing and therapeutic work we always use the negative pole as the active one.

If conduction through a peripheral nerve has ceased (either by gross anatomical or finer molecular disarrangement of the nerve trunk, or by a lesion in the spinal cord), within ten days certain well known changes in the electric reaction occur—known as the reaction of degeneration or, abbreviated, RD. The nerve no longer responds to either faradic or galvanic stimulation; the muscle does not contract on faradic stimulation, and responds to galvanic stimulation by a slow, sluggish contraction, instead of a brisk one.

In normal conditions each muscle responds best to electricity, if stimulated over its "motor point"—the surface marking of the spot where the motor nerve enters the muscle—usually over the middle of its body. In case of RD the motor point disappears and the distal end of the muscle is the most effective point of electrical stimulation (longitudinal reaction).

In partial RD the muscle still responds feebly to the faradic current and gives a sluggish response to the galvanic current, the explanation being that some of the fibres of the nerve trunk have remained unimpaired by the injury or disease.

Absolute RD—a term introduced by Turrell (2)—signifies total lack of response to any electrical stimulation of the muscle or nerve. This implies that the continuity of the nerve has been completely destroyed and that the muscle tissue has been replaced by a mass of fibrous and fatty tissue. Prognosis for recovery of the muscles affected is hopeless in case of absolute RD. Before rendering this verdict one always has to consider that high skin resistance, much oedema of the tissues, spreading of the galvanic current to neighboring muscles, might at times temporarily prevent a galvanic response; therefore in any doubtful case repeated and careful examination is required.

The classical test of RD enables any physician skilled in the elements of electrodiagnosis to give a verdict, most important from the standpoint of diagnosis, prognosis, and therapy, within ten days after an injury or disease followed by paralysis. The muscle ceases to respond to faradism between the fourth and seventh day after the division of the nerve, and the sluggish response to galvanism begins about the tenth day.

The importance of the RD in prognosis lies in the fact that, when present, there are changes in nerve and muscle substance that will take considerable time to recover—therefore RD by no means indicates an irreparable damage. If present in a case of a penetrating injury with other clinical evidence of nerve division, it indicates the necessity of surgical interference, nerve suture, followed by many months of appropriate physical therapy. If on the other hand, ten days after an injury accompanied by paralysis, there is no RD present, our diagnosis is "contusion" of the nerve with only temporary disturbance of nerve conduction, and a prognosis of an early recovery can be made. The same significance prevails in cases of "rheumatic" facial paralysis with no RD present, as compared with facial paralysis with RD due to compression or severe degeneration of the nerve.

The following case histories will serve to illustrate the above:

Case 33. Injury of left deep peroneal nerve, contusion, full recovery. C. T., letter carrier, admitted in January, 1922. Slipped from a step two months ago and strained left foot; slipped again seven weeks later, and since last injury has moderate drop foot with diminished sensation in foot. Nerve and muscle test, taken eight days after last injury, shows partial RD in deep peroneal distribution (tibialis anterior and extensors.) Patient received whirlpool bath to left foot, followed by massage and slow sinusoidal to affected muscles. Four weeks later nerve test showed normal response, and by January 16, two and a half months after injury, patient has fully recovered power of leg.

Case 16. Injury of right musculospiral nerve, contusion, full recovery. B. G., a clerk, admitted June 29, 1923, fell five days before with right wrist doubling beneath his body; immediately could not extend his wrist. Complete wrist drop present; flaccidity of extensor muscles of right forearm, with some anesthesia over dorsal aspect of right forearm. Nerve test on July 5 showed normal response of all muscles. Spade splint applied with wrist and fingers in full extension. Radiant light and heat, Bristow coil, massage to extensors ordered. Two weeks later patient showed returning power; active and passive exercises were added. By August 15 or six weeks after injury, patient had fully recovered.

Case 9. Injury of right musculospiral nerve, compression, full RD, recovery. F. F., a man aged forty-two, suffered a subglenoid dislocation of the right humerus, December 15, 1922, which was promptly reduced and the shoulder immobilized for two weeks. By February 21 the patient still had marked limitation in abduction, flexion, and extension of the shoulder in spite of active physiotherapy, consisting of radiant light, massage and mechanotherapy. He was suspected of being a malingerer, when electrical testing showed full RD of the right deltoid muscle. Interrupted galvanism and later sinusoidal was added to the treatment, and the shoulder improved steadily. By June 30 the function was almost complete, and by November 12 full normal electrical reactions had returned. The patient resumed work as a pianist.

A case very similar to the above came under observation very recently.

Case 64, a laborer, was hit by a plank over the right shoulder on July 10, 1924. A diagnosis of dislocation of the acromioclavicular and the sternoclavicular joint, and of fracture of the greater tuberosity without displacement was made. The dislocation was reduced under gas and the arm put up in plaster at right angle to the body with the elbow in 90° flexion. On August 16 the plaster was removed, and on August 25 the patient had only 10° abduction and flexion in right shoulder. On September 6 he had 30° abduction and was referred for physiotherapy, consisting of radiant light and heat, massage, and active and passive exercises of shoulder. He was tested electrically and full RD of deltoid, teres minor and infraspinatus found. Consequently diagnosis of injury of right circumflex nerve with paralysis following was made, and prognosis and therapy accordingly changed.

EXPLANATION OF THE RD IN VIEW OF MODERN RESEARCH

The explanation of the RD and its variations by the various textbooks is either hazy or unnecessarily complicated. Researches in electrophysiology during the past few years have made the reasons for these phenomena much more lucid and thus opened the way for great progress in both finer diagnosis and better therapeusis.

It has been found by the investigations of Adrian, Lapicque, and others that, if a muscle or nerve is stimulated by means of an electric current, the current must reach a certain intensity before any contraction will occur. But, in addition, this minimal current must last for a certain period in order to produce a response. If the minimum duration during which a current will produce a response is reduced, no contraction will result, unless the strength of the current is increased; and, as the duration is decreased, the strength has to be increased in a rapidly increasing ratio.

Using weak currents, it has been found that a normal nerve responds with a contraction of its muscle to a current lasting $\frac{1}{1500}$ of a second or less; that normal muscle responds to current lasting $\frac{1}{1000}$ of a second; and finally, muscle degenerated after being separated from its trophic center will require currents lasting from $\frac{1}{100}$ to $\frac{1}{10}$ of a second to contract.

This explains very clearly why degenerated muscles in peripheral nerve injuries do not respond to faradism, but contract under the impulse of the make of the galvanic current. The essential difference between the faradic and galvanic current is that the faradic flows intermittently for short periods of time, the

ordinary faradic coil imparting stimuli of about $\frac{1}{1000}$ second duration, while the oncome of flow of the galvanic current can be lengthened at will. Muscles whose nerve supply is degenerated do not respond to the ordinary faradic current, because the duration of each faradic impulse is shorter than the minimum duration of the current necessary to elicit a contraction. It is possible, however, to get a response in a denervated muscle, even with a current of very brief duration, if the strength of the stimulus is greatly increased, by using the high voltage of a very powerful induction coil or of static sparks; but the patient's lack of endurance of such painful stimuli prevents their clinical application.

Modern investigators (1, 2, 9, 11) also agree that the time honored formula of polar inversion, whereby in RD better response is elicited by the positive pole (anode) of the galvanic current, is no longer considered essential. It has been found that this polar reversal is by no means constant, and it was shown also that it is only at the kathode that at make (closing) a stimulus is effective, and the contraction which occurs on stimulating with the anode is explained by the occurrence of "virtual kathodes" on the deep surface of the muscle.

THE CONDENSER METHOD OF TESTING

The classical method of faradic and galvanic testing continues to be the most important one for the recognition of gross changes in the electrical response of peripheral nerves and muscles and also for the differentiation of functional from organic lesions. But when it comes to exact measurements of the degree of change and to the use of definite figures instead of subjective observations for the measuring of progress or further degeneration, the insufficiency of the simple faradic and galvanic test at once becomes evident. The strength of the ordinary faradic current is not measurable at all, the length of the impulse varying in different coils. Faradism moreover has no prognostic value, because in a paralyzed muscle voluntary power returns as a rule before the response to faradism. The strength of the galvanic current can be measured, but its duration of flow cannot be measured. Both the faradic and galvanic tests are very often decidedly painful, because as a rule much too strong and much too long currents are used for testing.

Among the number of new and more accurate nerve and muscle tests, the condenser method of testing by Lewis Jones seems most rational and practical. The method was presented by Capt. A. B. Hirsh (4), then stationed at Walter Reed Army Hospital, at the 1919 meeting of our Association. The principle of the condenser set testing is to use electrical impulses of uniform strength, gradually varying the duration for the measure of response. With the American Army modification of the original apparatus in use a set of condensers, varying in capacity from 0.01 microfarad to 2.00 microfarads, is charged at 100 volts. The discharge time of the smallest condenser lasts about $\frac{1}{24000}$ of a second, and the largest $\frac{1}{50}$ of a second. A large indifferent electrode and a small active electrode are used, the latter being placed over or near the motor point and starting with the lowest number; the smallest microfarad capacity at which a distinct contraction occurs is then charted. The response of the same area on the healthy side is charted for comparison. To visualize the working of the condenser set, one can aptly compare it with a set of springs of gradually increasing size, kept at even tension. As each spring is released it will oscillate according to its length and furnish an impulse, the duration of which will depend on the length of the spring. Thus the largest spring, representing the largest condenser capacity, will furnish the longest duration of stimulation. The expression "farad" has nothing to do with faradism, but is an arbitrary measure (like the volt or the ampere) of capacity, i.e., the measure of the quantity of electricity that a conductor is capable of receiving. A microfarad is a millionth of a farad.

According to Cumberbatch (1), it may be taken as a rule that, if muscles with reactions of the normal type require condensers of capacities from 0.01 to 0.09 microfarads, according to the degree of departure from the normal, then muscles with a partial RD require condensers of 0.10 to 0.9 microfarad capacity; muscles with complete RD require condensers of one or more microfarads. Instead, therefore, of three types of reaction, normal, partial RD, and complete RD, we have any number of degrees of departure from normal, each being represented numerically. Having decided to give the condenser test a thorough trial in our work at the Reconstruction Hospital, we adopted a test chart similar to the one used at Walter Reed, one for the lower and two for the upper extremity, with a column for faradic testing and condenser testing, and with space for four subsequent examinations. After having used the condenser set on all of our cases for the past two years, we have modified this chart by adding another column to record response to the ordinary interrupted

THE RECONSTRUCTION HOSPITAL UPPER EXTREMITY—II.

MUSCLE AND NERVE TEST Median and Ulnar Distribution

Name..... Number..... Diagnosis .....

		Date			Date			Date		
N. MEDIANUS		Farad.	Condens.	Galv.	Farad.	Condens.	Galv.	Farad.	Condens.	Galv.
Pronator Radii Teres	R.									
	L.									
Flexor Carpi Radialis	R.									
	L.									
Palmaris Longus	R.									
	L.									
Flexor Sublimis Digit.	R.									
	L.									
Flexor Longus Pollicis	R.									
	L.									
Flex. Profundus Digit.	R.									
	L.									
Abductor Pollicis	R.									
	L.									
Opponens Pollicis	R.									
	L.									
Flexor Brevis Pollicis	R.									
	L.									
Lumbricales I. et II.	R.									
	L.									
N. ULNARIS										
Flexor Carpi Ulnaris	R.									
	L.									
Flexor Profundus Digit.	R.									
	L.									
Palmaris Brevis	R.									
	L.									
Abductor Minimi Digiti	R.									
	L.									
Flex. Minimi Digiti	R.									
	L.									
Opponens Minimi Digiti	R.									
	L.									
Lumbricales III. et IV.	R.									
	L.									
Interossei I.-II.	R.									
	L.									
Interossei III.-IV.	R.									
	L.									

galvanism (see charts on accompanying pages). We very soon found out that a negative condenser set result in itself was unreliable, that in muscles in both advanced or less advanced stages of degeneration a response was easily elicited by ordinary galvanic stimulation, whereas none was elicited by the condenser set. The explanation of this is partly that even the largest condenser does not provide an impulse long enough for many muscles with full RD, and, partly that, as the condenser discharges, its intensity decreases gradually and a portion of each discharge is below the minimum strength of current necessary for stimulation and, therefore, is ineffectual. We gave, therefore, a standing order to test for galvanic response in every case of negative condenser response.

THE RECONSTRUCTION HOSPITAL UPPER EXTREMITY—I.

MUSCLE AND NERVE TEST Shoulder and Musculo Spiral Distribution

Name..... Number..... Diagnosis .....

		Date			Date			Date		
		Farad.	Condens.	Galv.	Farad.	Condens.	Galv.	Farad.	Condens.	Galv.
N. ACCESSORIUS AND PLEXUS BRACHIALIS										
Trapezius	R.									
	L.									
PLEXUS BRACHIALIS										
Serratus anterior	R.									
	L.									
Pectoralis major (n. thorac. ant. extern)	R.									
	L.									
Rhomboideus (n. post. scapularis)	R.									
	L.									
Supra spinatus (n. supra scapularis)	R.									
	L.									
Infra spinatus (n. supra scapularis)	R.									
	L.									
Latiss. dorsi (n. sub scapularis)	R.									
	L.									
Teres major (n. sub scapularis)	R.									
	L.									
Deltoideus (n. circumflexus)	R.									
N. MUSCULO CUTANEUS	L.									
Biceps	R.									
	L.									
Brachialis	R.									
N. MUSCULO SPIRALIS	L.									
Triceps	R.									
	L.									
Brachio Radialis (Supin. long.)	R.									
	L.									
Ext. Carpi Radii Longior	R.									
	L.									
Ext. Carpi Radii Brevior	R.									
	L.									
Supinator	R.									
	L.									
Ext. Carpi Ulnaris	R.									
	L.									
Ext. Communis Digitorum	R.									
	L.									
Ext. Minimi Digiti	R.									
	L.									
Ext. Indicis	R.									
	L.									
Ext. Oss. Metacarp. Poll.	R.									
	L.									
Ext. Brevis Pollicis	R.									
	L.									
Ext. Longus Pollicis	R.									
	L.									

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Briefly stated, our conclusions after using the condenser method of testing are as follows:

Its advantages are:

1. It provides a method of precision to chart the degree of excitability of individual muscles, and enables us to measure progress in figures.

2. It is more painless in many cases than the ordinary faradic and galvanic test, as proved in most of our cases of infantile paralysis.

The disadvantages of the condenser testing are:

1. It takes considerably more time than the ordinary methods of testing.

2. It often elicits no response at all in cases of full RD, and used alone would be misleading.

3. It has in some cases an unpleasant tendency (in using larger condenser capacities) to spread to antagonistic muscles in the vicinity

THE RECONSTRUCTION HOSPITAL LOWER EXTREMITY

MUSCLE AND NERVE TEST

Name..... Number..... Diagnosis .....

		Date			Date			Date		
		Farad	Condens.	Galv.	Farad.	Condens.	Galv.	Farad.	Condens.	Galv.
N. FEMORALIS										
Sartorius	R.									
	L.									
Rectus Femoris	R.									
	L.									
Vastus Internus	R.									
	L.									
Vastus Externus	R.									
	L.									
Pectineus	R.									
	L.									
Adductor Longus	R.									
N. SCIATICUS	L.									
Gluteus Maximus	R.									
	L.									
Gluteus Medius	R.									
	L.									
Biceps Femoris	R.									
	L.									
Semitendinosus	R.									
	L.									
Semimembranosus	R.									
	L.									
N. PERONEUS (Ext. Poplit)										
N. PERONEUS PROFUNDUS										
Tibialis Anterior	R.									
	L.									
Extens. Longus Digit	R.									
	L.									
Extens. Longus Hallucis	R.									
	L.									
Extens. Brevis Digitorum	R.									
	L.									
N. PERONEUS SUPERFICIALIS										
Peroneus Longus	R.									
	L.									
Peroneus Brevis	R.									
	L.									
N. TIBIALIS (Int. Popliteal)										
Gastrocnemius	R.									
	L.									
Soleus	R.									
	L.									
Flexor Longus Digitorum	R.									
	L.									
Flexor Hallucis Longus	R.									
	L.									
Tibialis Posterior	R.									
	L.									

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and their contraction interferes with the response of the paralyzed group.

These findings correspond with those of English investigators (1, 2, 12) and with Sampson's experience (6). While some men see no advantage at all in the condenser testing, in our experience the condenser set, combined with the faradic and galvanic test, gives much more accurate information than the old methods alone. The progressive improvement or retrogression of a lesion can be charted and compared by different examiners and at various times, provided that a uniform method of testing is used.

TECHNIC OF MUSCLE AND NERVE TESTING

In cases of muscle and nerve testing, whether with the faradic, galvanic, or with the condenser set, the following elementary rules of technic have to be observed:

1. Preliminary to testing, the parts to be tested are warmed by fifteen minutes' exposure to radiant light and heat or by a whirlpool bath.

2. The muscles to be tested and their antagonists have to be carefully relaxed.

3. The electrodes are moistened with warm saline (about two per cent); the indifferent electrode placed, in case of upper extremities on the back or chest, in case of lower extremities over the sacrum, but always in good contact. The active electrode has a metal disc about 3–4 to one inch in diameter, well padded with gauze and with a make and break key.

4. The operator picks out the individual motor points with the minimum strength of current to which they will respond, always comparing the healthy side as to the strength of current needed and the kind of contraction elicited—brisk or sluggish. Easily available charts with the motor points marked thereon are of great assistance to beginners (5). It requires a well trained and experienced operator to take a reliable muscle and nerve test. First the faradic, then the galvanic current is used, and all findings are accurately charted. Our routine examination always includes the condenser set testing described previously, and only in cases of rapid elimination of malingering or of a central lesion are we content with the simple galvanic and faradic test.

In case of very weak muscles in which the testing current "spreads" to the strong antagonists, we apply the galvanic and condenser test by the "bipolar" method. This is carried out by placing two active electrodes of equal size directly over the muscle. The concentration of current thus obtained often elicits a response when the normal monopolar method fails.

We repeat the muscle and nerve tests every four to eight weeks on an average in cases under active treatment, in order to check up progress. It is very gratifying to see in some instances how, from a faint flicker of response to the strong interrupted galvanic current at first, there is only a response to the higher values of the condenser set, while later a weak normal response occurs on the same set, gradually turning to normal.

SIGNIFICANCE OF ELECTRICAL TESTS IN DIFFERENTIAL DIAGNOSIS

Using the electrical reactions of nerves and muscles in our diagnosis, we must bear in mind that these tests are only part of the evidence, the other part being supplied by careful clinical examination of the muscular power, the reflexes, and of the sensory condition of the region affected. To illustrate this in the very type of cases we are dealing with, in both anterior poliomyelitis and peripheral nerve lesion a flaccid paralysis and rapid muscle wasting occurs and the typical reaction of degeneration develops. But there is no sensory change in poliomyelitis because there only the motor cells of the anterior horn are out of function, while in peripheral nerve lesions, the sensory fibres being included in the common nerve cord, there is always a definite area of sensory change. Furthermore, the distribution of paralysis in poliomyelitis is irregularly located in the various muscles (with some well known sites of predilection) and does not correspond with any particular peripheral nerve. Another example of the difficulty of making a diagnosis on the basis of nerve tests alone are cases of multiple injuries of the extremities especially of the forearm. Extensive scar formations and adhesions, due to longitudinal incisions or multiple infectious processes, result in a loss of response of several muscles, thus imitating a nerve lesion. A nerve lesion might also be aggravated or appear more extensive from the coexistence of tendons bound down by adhesions. The results of electrical testing without a thorough clinical examination are not sufficient in case of a paralysis of traumatic origin to determine whether the paralysis is caused by the original trauma or secondarily by contracting scars or pressure of callus.

The occurrence of the RD proves a lesion of the lower motor neuron of the nervous system, consisting of the anterior horn cell, anterior root and nerve plexuses, and peripheral nerve. Clinically, such lesion is always accompanied by flaccid paralysis and loss of tendon reflexes.

1. Lesions of the motor cells of the spinal cord are caused commonly by poliomyelitis, by progressive spinal muscular atrophy, or by traumatism (hematomyelia).

2. Lesions of the anterior roots are caused by traumatism (compression) or inflammatory process.

3. Lesions of the peripheral nerves or nerve plexuses are caused by traumatism (division,

laceration, compression or contusions) or degeneration (alcoholic, postdiphtheritic, lead, arsenical or other peripheral neuritis).

Functional, hysterical paralyses and paralyses of cerebral origin or resulting from lesions of the pyramidal tract (upper motor neuron) are, according to Tinel (7), never accompanied by important disturbances of the electrical reactions. At most, there is slight electrical hyperexcitability, while in the later stages there is usually hypoexcitability from muscular disuse. Clinically these cases are always accompanied by spastic paralysis, increased reflexes, lack of sensory changes, and lack of muscle wasting Electrical muscle and nerve tests in spastic paralysis therefore are of no special diagnostic value. In hysterical and other functional paralyses, however, the normal electrical reactions, together with the sensory and motor changes which in no way correspond to any form of nerve distribution, form a most important diagnostic evidence.

In myopathies, for the observation of which we had but scant material, there is never any real RD present, although both the galvanic and faradic response will show alteration. According to Tinel (7) and Reading (12), we find in weakened muscles and oftener during muscular regeneration a condition known as the "myasthenic reaction." The muscles at first contract normally to faradism, but quickly become exhausted and cease to respond. A short interval of rest will restore faradic excitability. Galvanic stimulation produces a continuous contraction, which does not relax if the stimulation is prolonged.

The "myotonic reaction" is characteristic of Thomsen's disease (congenital myotonia). There is present galvanic hyperexcitability of the muscles, characterized by a tonic, lasting contraction after the cessation of galvanic excitation. Faradic reaction may be normal or also increased.

In pseudohypertrophic muscular paralysis there is greatly diminished faradic and galvanic excitability but no RD.

According to Tinel, there is only one affection accompanied by electrical disturbances as profound and rapid as those of the peripheral nerve lesions; this is ischemic paralysis from arterial obliteration. Still, the syndrome of fibrous transformation of the muscles is seen, rather than the true RD, inasmuch as inexcitability of the muscles comes on earlier and is more marked than the inexcitability of the nerve controlling it.

CONCLUSIONS

1. The early diagnosis of peripheral nerve injuries in civil life is of great importance for instituting appropriate and successful therapeutic measures.

2. The classical galvanic and faradic test is still the most expeditious way of electrodiagnosis, and it will show any nerve involvement within ten days from the date of injury.

3. The condenser set of electrical nerve testing in conjunction with the above is valuable for charting findings in definite figures and thus provides the means of measuring progress accurately.

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DISCUSSION

DR. MASSEY: The importance of this subject is very well presented, particularly the diagnosis of disabling injuries of the secondary motor centers and the external motor neurons, and their relations to the compensation bill. It seems to me that no industrial surgeon could do his work well without applying these methods so well described by the writer of the paper. I have been working on this subject for thirty-six years. In 1888 I tested out the exact amount of galvanic current required to move certain small muscles of the face and forearm. These tests were made on students of the Women's Medical College, and I am reminded of the work now by occasionally seeing a middle aged woman physician, who was one of the subjects. The most important point is to convince the physician that this examination is not difficult.

When we think of the interrupted electrical current, we must think of it as a wave of kathodic electrons. I don't know whether Dr. Kovacs made that point clear, but we may confine our consideration to kathodic closure as the essential part of the test. The impulse of kathodic closure is like a wave of sea water; it is strongest when it is most sudden and when it has its greatest bulk. The more sudden the impact, the less the bulk need be to get a result. Conversely, the greater the bulk the less sudden the impact need be. As for the anodic opening contraction, the next strongest to kathodic closure, this is doubtless a kathodic wave also, striking through the body from the indifferent pad, and weaker because the nerve or muscle is struck by a wave that is more diffused.

All over the country the courts are disposing of cases which need our assistance to clear up. The courts in Philadelphia are overwhelmed with cases against the traction company, and a large proportion of them claim injury to the external motor neurons, which can only be decided properly, for or against the claimant, by such an examination. Electrodiagnoses will enable you to tell the truth, no matter which side you are on.

DR. WADDINGTON: Dr. Kovacs has directed attention to a most important subject and one which is considered quite difficult by the general practitioner. Dr. Kovacs is to be commended for the illuminatingly simple manner in which he has presented the subject, whereby he has shown that the difficulties of diagnosis exist more in anticipation than in the realization of practical application. I wish to draw your attention to the subject of Bourguignon's work in France. He is at the head of the Electro-Radiothérapie department of La Sapêtrière in Paris, and considered the foremost authority on electrotherapy in France. He has recently written a thesis entitled "La Chronaxie chez l'homme," in which, instead of stressing du Bois-Raymond's law, which enunciated that muscular contraction is governed by the strength and suddenness of the current, he has followed out the conclusions first brought out by Lapicque and has accomplished a vast amount of research along these lines. Lapicque showed that a better and more appreciable method of measuring the reaction of degeneration was to test the healthy muscle first and note the amount of current (called the chronaxie) necessary to cause the merest flicker of a response. He then doubled this current and applied the electrode over the corresponding affected muscle, noting how quickly or slowly the muscle responded; this he termed the rheobase. The negative is always used as the differentiating electrode, and Bourguignon has developed a most complicated apparatus of innumerable switches and rheostats, all installed upon a long narrow table, behind which his technician instantaneously makes adjustments as the professor tests out the patient. With this apparatus of his he has so adjusted voltage to amperage in any desired ratio that his tests are exceedingly delicate.

DR. BROOKE: Dr. Kovacs has brought out many important points. I have had many such cases under observation and treatment in the government hospital clinics. In this connection I would like to emphasize the importance of accurate localization of motor points for muscle stimulation. Attention is called to the sequence of application of treatment, beginning with interrupted galvanism. Here a word of caution: do not overtreat paralysed muscles, because muscle fatigue is worse than no treatment at all. This application calls for a small amount of current with only a few contractions at first, to be stepped up gradually at subsequent treatments. It is best to record at each application the amount of current used and likewise the number of contractions given over each motor point. Often in the early stage of treatment the muscle contraction is scarcely visible; in order to determine the presence of a slight contraction, you must develop a sense of touch by lightly palpating over the muscle or its tendon distribution. The interrupted galvanism is given until a good response is obtained. The next step in the treatment is the application of slow

sinusoidal to the motor points, given with same technic as the interrupted galvanism until a good contraction is obtained. In addition, the slow sinusoidal may be given to affect groups of muscles, producing mass muscle contraction. The last step of treatment calls for faradism to the motor points to induce muscle contractions, until a normal muscle action is produced. During the process of electrical stimulation to affected muscles avoid the use of strong currents, which often cause the antagonistic muscles to contract and produce a deformity. Following faradism application the patient is ready to assume suitable occupational therapy in order to reeducate muscle activity, so as to develop muscle power and normal range of motion.

The adherence to this sequence of treatment is important for the achievement of best results. In preparation of the extremity for treatment, I prefer the use of the hot whirlpool bath for twenty minutes, to produce a hyperemia and relaxation of the musculature. This application definitely increases the conductivity of electrical currents as compared with a cold unprepared extremity. In conjunction with the electrical stimulation, massage and light exercises are valuable adjuncts. During the course of treatment operative procedures are indicated, such as neurolysis, etc., to insure best results. Adequate supportive treatment with braces and splints is always indicated. In this connection it is essential to give treatment before operation, as well as after operation. In closing, I want to emphasize three points: accurate localization of motor points, carefully recording strength of current and number of contractions at each treatment, and the employment of the physical modalities outlined.

DR. WALSH, Syracuse: I believe it a very important procedure during treatment to keep the paralyzed muscle or group of muscles and the antagonistic group in a state of equilibrium by properly adjusted splints. This will tend to prevent any overaction of the antagonistic group with resultant deformities.

This procedure is too often neglected while the patient is under treatment, and it invariably retards and oftentimes entirely prevents recovery, in spite of otherwise well directed treatment.

DR. YATES: This subject has been a puzzling one to me. I have not had time to study it sufficiently, but it is an important one to every one doing this work. It is particularly important in regard to industrial compensation cases. The paper, on this account, is extremely valuable, and I hope that Dr. Snow will see an early opportunity to publish this paper. I would like Dr. Kovacs to give some description of the apparatus.

DR. REA: There was one case of pseudoparalysis. I should like to hear Dr. Kovacs say something about that in closing the discussion.

DR. RICHARD KOVACS, in closing, said: I thank Dr. Massey for aiding me in his discussion in my efforts to present this problem as clearly and simply as possible. Even excellent textbooks, like Cumberbatch's, which contains a chapter of thirty-two pages on nerve testing alone, make it very complicated for the beginner to comprehend the main facts clearly. As a matter of fact, it ought to be easy for anyone having an outfit with faradic and galvanic current to start practicing on himself, and learn to recognize the various motor points. With a patient we always can start on the healthy side for comparison. With a little practice we quickly learn to get accurate results and thus, when testifying in court on any case of paralysis, we are on safe ground as experts.

Dr. Waddington's report of Bourguignon's work is quite comprehensible to experienced workers in this field, but for the average physician it has only a theoretical value, like a number of the ingenious methods which are more or less of the nature of a complicated laboratory test. Dr. Brooke mentioned valuable points in regard to the treatment of paralysis, a subject which I reserved for a later paper. The whirlpool bath is an excellent modality to warm up and relax an extremity for both diagnostic and therapeutic procedures. The avoidance of fatigue is very important, and also the development of the sense of touch to discover slight contractions. As to the comparative value of interrupted galvanism and the slow sinusoidal, this is explained by the physics of the two modalities. Interrupted galvanism furnishes a longer flow at a higher and suddenly increased voltage, while the slow sinusoidal delivers a slowly oncoming stimulus of gradually increasing strength, and it very often happens that, in case of advanced degeneration, neither the duration nor the strength of the stimulus is sufficient to cause the remaining fibres to contract. As long as we believe that it is beneficial under all circumstances to cause contractions in de-

generated muscles, interrupted galvanism is indicated when there is no response to sinusoidal.

As regards splinting, healthy muscles have two properties, one of contractility and the other of elasticity. We maintain contractility in paralyzed muscles by our electrical methods, while elasticity is maintained by proper splinting.

If paralyzed muscles are left to be overstretched by their antagonists they lose their elasticity and when the normal motor impulse returns, it finds a muscle over-stretched like a rubber band. Splinting, however, should not be over-done and should not prevent neighboring joints from being exercised at regular intervals to prevent ankylosis.

As to Dr. Yate's remarks, the subject of paralysis has been neglected in our recent American literature and therefore an early publication might serve a good purpose.

The case of pseudo-paralysis of the lower extremities was a traumatic neurosis in which all nerve tests proved negative.