THE VALUE OF ELECTROMYOGRAPHY IN NEUROLOGY AND NEUROSURGERY*

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It is the purpose of this paper to survey the increasing value of electromyography as an aid in the field of neurologic diagnosis. In 1943 Weddell, Feinstein and Pattle\textsuperscript{27} presented some of the clinical applications of electromyography. Since then much valuable investigative work has been done.\textsuperscript{1–28} It would seem timely, therefore, to present a systematic review of how the electromyograph can be of practical use to the clinical neurologist and the neurosurgeon. This practical value largely depends on detecting the presence or absence of lower motor neuron disease which evidences itself by denervation fibrillation potentials accurately recorded both visually and acoustically by the electromyograph. It should be emphasized, however, that electromyography, like any other laboratory procedure, is in no way a substitute for a careful anamnesis and meticulous neurologic examination.

It is now generally accepted that for accurate electromyography it is necessary to use a needle electrode placed directly into the muscle to be tested, rather than the older method using a percutaneous electrode. The authors prefer the Meditron Clinical Electromyograph, Model 201, which utilizes a monopolar needle electrode, a cathode ray oscilloscope, a sensitive sound amplifier and a myoscope.

We have used this instrument to examine patients with the usual wide variety of disorders seen in the practice of clinical neurology and neurosurgery. We have been surprised by the amount of useful diagnostic information obtained. Many puzzling problems that could not be accurately diagnosed by ordinary clinical means fell, logically, into their proper classification with the information given us by the electromyograph. We shall attempt to present the bioelectric findings in diseases of the nervous system, and to point out features that have proved useful in differential diagnosis.

A detailed description of the Meditron Clinical Electromyograph, and the methods used in its clinical application, have been recently published.\textsuperscript{22} In addition, a myoscope is now available which permanently records, by means of a magnetic tape, electromyographic patterns. This tape can be played back through the instrument and faithfully reproduces, visually and acoustically, the muscle action potentials as recorded at the time of the original examination.

CLINICAL OBSERVATIONS

Those diseases of the spinal cord that affect the anterior horn cells often are not only diagnostically confusing, but difficult to detect in their early stages. Since one of the causes of denervation fibrillation is anterior horn cell damage, it is often possible, with the electromyograph, to detect minimal or early involvement where the classical clinical signs are absent. We have found this of particular value in cases in which the patient has been diagnosed as having a "functional" rather than an organic disorder. In other words, no true objective neurological abnormalities could be found, yet the patient's symptoms were corroborated as organic by the EMG findings of scattered lower motor neuron disease. Case 1 illustrates this point:

Case 1. Mrs. P., a 60-year-old white female, entered Mercy Hospital with the chief complaint of increasing weakness and numbness of both legs over a period of 6 months. She stated that these symptoms had come on gradually after the death of her husband, accompanied by nervousness and depression. The disability had progressed to the point where she could no longer climb stairs.

Neurological examination revealed only absent abdominal reflexes, diminished ankle jerks, and generalized weakness of the lower extremities without fasciculations or focal atrophies. The plantar responses were flexor. Vibratory and position sense were slightly diminished. No true loss of pain, touch or temperature sensation could be demonstrated. Ethyl iodophenylundecylate myelography was negative. A psychiatric diagnosis of psychoneurosis, conversion hysteria, was made.

The EMG, however, revealed evidence of diffuse lower motor neuron disease of all four extremities, more marked in the lower. A diagnosis of arteriosclerotic myelopathy was suggested. Subsequently the disease progressed to almost complete spastic paraplegia with bilateral extensor plantar responses.

DEGENERATIVE DISEASES OF THE SPINAL CORD

The EMG achieves one of its most important functions in the diagnosis of degenerative diseases of the spinal cord. For example, in early amyotrophic lateral sclerosis clinical examination may reveal evidences of anterior horn cell damage only in the intrinsic muscles of the hands, whereas electromyographic examination demonstrates denervation fibrillation and fasciculation voltages in the lower extremities as well. Later in the disease the EMG continues to reveal this widespread anterior horn cell damage, even though it is impossible clinically to demonstrate it in the lower extremities because of the marked pyramidal tract involvement producing a spastic paraplegia. It should be emphasized that careful multiple needle insertions must be made in the various muscles in all four extremities in order to demonstrate these abnormal electrical impulses. This is necessary because the frequency of the diagnostic wave forms varies from muscle to muscle. Thus, multiple sampling becomes an indispensable feature of accurate electromyography. We have all seen patients who are suspected of having either amyotrophic lateral sclerosis or a cervical cord tumor. If denervation fibrillation and fasciculation voltages in such a patient are found in the lower extremities, the possibility of a cervical cord tumor is automatically ruled out.
The protean nature of multiple sclerosis makes the early diagnosis of this disease obviously difficult. Therefore, any corroborative evidence of diffuse central nervous system disease is of particular importance. The EMG, in patients suspected of having multiple sclerosis, often reveals diffuse lower motor neuron disease which cannot be demonstrated by clinical diagnostic methods. It should be emphasized, however, that electromyographic findings of diffuse lower motor neuron disease are in no way diagnostic of multiple sclerosis because, obviously, they may be found in any degenerative disease of the anterior horn cells. We have noticed, however, that in multiple sclerosis fasciculation voltages are never encountered whereas they are a characteristic finding in other chronic degenerative diseases of the spinal cord. Thus, in a young patient, with only a few signs and symptoms suggesting multiple sclerosis (possibly urinary urgency, questionable nystagmus and absent abdominal reflexes) the finding of diffuse denervation fibrillation without fasciculation voltages is very suggestive of the disease.

Similarly, in subacute combined cord degeneration, early diffuse lower motor neuron disease may be demonstrated despite meager clinical evidence. Again, this finding should be interpreted as suggestive but not conclusive.

Progressive muscular atrophy (whether the spinal form of Aran-Duchenne, the juvenile type of Werdnig-Hoffmann, the scapulohumeral type, or the bulbar form of Fazio-Londe) and amyotrophic lateral sclerosis are probably different manifestations of the same degenerative disease process. Electrically, certainly, the EMG findings would tend to confirm this concept since all of these forms, whether or not pyramidal tract involvement is present, show diffuse denervation fibrillation and fasciculation voltages indicating anterior horn cell involvement.

The value of the electromyograph in diagnosing progressive muscular atrophy is indicated by Case 2:

Case 2. A 46-year-old white male was referred by an insurance company because of weakness and atrophy of the right arm and hand. The patient stated that this disability first appeared after an auto accident in which he was purported to have injured his neck and back.

Neurological examination revealed atrophy of the intrinsic muscles of the right hand without sensory or reflex changes. There were no abnormalities of the cranial nerves. In particular no fasciculations of the tongue were noted. The remainder of the findings were negative, including flexor plantar responses bilaterally.

Widespread sampling of the muscles of all four extremities revealed diffuse denervation fibrillation and fasciculation voltages, most pronounced in the visibly atrophied muscles of the right hand. The insurance company was advised to withhold compensation in this case because the patient most probably was suffering from a degenerative disease of the spinal cord which could have no relationship to the alleged trauma. Over the period of the next year there developed the typical findings of progressive muscular atrophy (spinal form of Aran-Duchenne).

INFECTIOUS AND INFLAMMATORY DISEASES OF THE SPINAL CORD

In infectious and inflammatory diseases of the spinal cord the EMG proves a useful diagnostic adjunct by indicating the extent of the disease
process and also by providing important prognostic information. For example, in *acute anterior poliomyelitis* the electromyographer can determine accurately the specific muscles affected, and also the amount of denervation of any particular muscle. It can readily be seen that this is important because it channels physical therapeutic efforts toward those muscles that are capable of regaining function. For instance if, in any given muscle, denervation fibrillation voltages are elicited in 100 per cent of all areas tested and with voluntary effort no discrete motor unit contractions are seen, then this muscle can be said to be hopelessly denervated and physical therapy useless. This, then, obviates the long, costly hours of physical therapy so often wasted on a particular muscle that can never regain function, and allows early orthopedic surgery without unnecessary delay. Conversely, oftentimes an apparently paralyzed muscle will be found to be only slightly denervated but rendered useless by psychologic inhibition. The patient, by watching and listening to the EMG, can readily be reeducated to contract this muscle once he, himself, recognizes that it is capable of contracting. Another important concept is that of Hipps, who has pointed out that there is often a segmental destruction of portions of the poliomyelitic muscle as seen on the operating table. We have confirmed this electromyographically, thus providing the surgeon with an opportunity to utilize the viable segments that remain.

Other inflammatory and infectious diseases of the spinal cord in which we have used the EMG to similar advantage are the *Guillain-Barré syndrome*, *herpes zoster*, *bacterial infections*, either diffuse or focal (intra- and extra-medullary abscesses), and *lytic infections*, particularly in *tabes dorsalis*. In all of these diseases the presence and extent of the lower motor neuron involvement can be accurately determined and consequently an early and precise prognosis of the degree of return of function in any specific muscle can be made.

**TRAUMA TO THE SPINAL CORD**

Trauma to the spinal cord often produces both upper and lower motor neuron injury. It is of practical importance to know the degree and extent of this injury. For example, in a paraplegic who has suffered a crush injury of the conus medullaris at the level of the first lumbar vertebra, it is of extreme prognostic value to determine whether or not there has been a complete transection or whether there are viable nerve pathways remaining that cannot be demonstrated clinically. If such pathways can be demonstrated, every effort can be concentrated on increasing the power of the partially innervated muscles. As in poliomyelitis, the psychologic effect is invaluable if it can be demonstrated, by having the patient watch and listen to the EMG, that he still has some remaining muscle function, even though at the time the muscle is apparently totally paralyzed.

**VASCULAR DISEASES OF THE SPINAL CORD**

Our investigations with the electromyogram in vascular diseases of the spinal cord have revealed a surprising amount of damage to the motor cells
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of the cord in elderly patients whose only systemic disease is diffuse arteriosclerosis. Many of these patients present symptoms that seem incompatible with the meager neurological findings. As we have mentioned (Case 1), these symptoms are often termed "functional". Also we are finding an increasing number of elderly patients who, apparently presenting only the neurologic picture of a pure corticospinal disease ("primary lateral sclerosis"), actually, on EMG examination, show a diffuse lower motor neuron disturbance which is masked, clinically, by the pyramidal tract signs. Thus we can confirm the concept that an arteriosclerotic myelopathy, probably on the basis of a circulatory insufficiency, can and does involve both levels of the motor system of the spinal cord. Therefore, in patients in the upper age group, presenting a confusing neurologic picture, we can often rule out functional disorders and establish a diagnosis of a true organic syndrome.

TUMORS OF THE SPINAL CORD

Tumors of the spinal cord may frequently present an obscure neurologic picture which the EMG can often clarify. These tumors, other than intramedullary, almost always involve the spinal nerve roots and, therefore, produce localized denervation fibrillation in the muscles supplied by the involved roots.

The differential diagnosis between a high extramedullary cervical cord tumor and a degenerative cord disease without involvement of the cranial nerves (as frequently occurs in amyotrophic lateral sclerosis), is often difficult. This difficulty arises because in amyotrophic lateral sclerosis, for example, the upper motor neuron signs in the lower extremities obscure the lower motor neuron component. Utilizing the EMG, however, we find denervation fibrillation voltages widespread throughout the skeletal musculature in the degenerative disease (the muscles of the face are always tested), but in the high cervical cord tumor we find them confined only to the distribution of the involved cervical nerve roots.

Case 3. A 36-year-old white female entered with the chief complaints of difficulty in walking, weakness, wasting of the upper extremities, and pain in the occipitocervical region. These symptoms had been slowly progressive over a period of 4 years.

Examination showed no involvement of the cranial nerves. There was atrophy of the intrinsic muscles of the hands and of the arms with occasional fasciculations confined to these extremities. The lower extremities showed a spastic paraparesis with increased deep reflexes and bilateral extensor plantar responses. The abdominal reflexes were absent. There was localized tenderness on palpation of the upper cervical spinous processes and a persistent hypalgesia over the occiput. There were no other sensory disturbances. Cervical x-rays were negative. Pantopaque myelography was refused.

Electromyography revealed marked denervation fibrillation voltages in the muscles of all four extremities and the face, thus establishing the diagnosis of a diffuse degenerative disease rather than a cervical cord tumor. Recently obvious fasciculations of the tongue and lower extremities and typical bulbar speech have developed in this patient.
An extradural tumor in the cervical region may present the same symptoms and signs as a protruded cervical disc or an anterior scalene syndrome, namely, pain in the cervical region radiating to one or both upper extremities, accompanied by numbness, tingling and weakness. Sensory, motor and reflex changes may or may not be demonstrated. The extradural tumor may be differentiated by the EMG from the anterior scalene syndrome because the tumor will involve the nerve roots and, consequently, will cause denervation fibrillation of the posterior cervical muscles since they are innervated by the posterior primary divisions of the cervical nerves (emerging proximal to the brachial plexus). Similarly, a protruded disc will also result in denervation over the posterior primary divisions. On the other hand, pressure on the brachial plexus by an anterior scalene muscle cannot involve these posterior primary divisions; thus, a clearcut differential between an extradural spinal cord tumor (or a cervical disc) and an anterior scalene syndrome can be established by the EMG.

To differentiate an extradural tumor and a cervical disc electromyographically, we depend upon the fact that the cervical disc involves only one nerve root, almost invariably unilaterally. The tumor, however, can be shown to involve more than one root, often bilaterally, even though there are no clinical indications of this.

As in all of electromyography, caution must be exercised to sample the muscles extensively and to always bear in mind the possibility of the occasional anomalous innervation.

Case 4. A 63-year-old Greek male presented himself with the complaints of pain and weakness of both upper extremities for about 5 months. Past history was significant in that prostatic resection 5 years previously had revealed the presence of a malignant adenoma of that gland.

Examination demonstrated mild tenderness of the lower cervical spine, vague hypalgesia over the 4th, 5th and 6th cervical dermatomes, without reflex or motor changes. The pain in the upper extremities was not made worse by coughing, sneezing or straining nor by jugular compression. X-rays of the cervical spine revealed a partial collapse of the 3rd cervical vertebra without displacement and no other abnormalities. Pantopaque myelography, up to the foramen magnum, was completely negative without evidence of narrowing of the cervical canal.

Electromyography showed bilateral involvement of the muscles innervated by the 2nd through the 6th cervical nerve roots including the posterior primary divisions, thereby placing the lesion at or proximal to these divisions. With the above evidence, an extradural tumor was suspected. This was confirmed at operation when a metastatic prostatic adenoma was found extending extradurally from the 2nd to the 7th cervical vertebrae.

THE SPINAL NERVE ROOT COMPRESSION SYNDROME

In conjunction with neurologic examination and pantopaque myelography, the electromyogram is proving a valuable aid in diagnosing the spinal nerve root compression syndrome. We wish to re-emphasize that the basis of the electromyographic localization of a single nerve root lesion is the finding of denervation fibrillation in those muscles supplied by that specific-
cally involved nerve root and in no other muscles. In other words, although an isolated muscle may have multiple nerve root innervation, it will show denervation fibrillation in common only with those other muscles that are innervated by the same compressed nerve root. To illustrate how a lesion compressing, for example, the 7th cervical nerve root may be localized by a process of elimination, the needle electrode is first placed in several areas of the deltoid and biceps muscles, innervated by the 4th, 5th and 6th cervical nerve roots. No denervation fibrillation voltages are found. Next, the electrode is inserted successively in the extensor carpi radialis, extensor communis digitorum, extensor carpi ulnaris, and the flexor carpi ulnaris (innervated by the 6th, 7th and 8th cervical nerve roots). In these muscles a maximum of denervation fibrillation voltages are noted. The next muscle tested is the abductor digiti quinti, innervated by the 8th cervical and 1st dorsal nerve roots. As in the first instance, no evidence of denervation is found in the muscle. The lesion, therefore, since we have demonstrated that the 5th, 6th and 8th nerve roots are not involved, must be compressing the 7th cervical nerve root. Examination of the erector colli musculature reveals segmental denervation fibrillation which confirms the fact that the lesion must involve the posterior primary division. In other words, the nerve root must be involved.

Using the same deductive method, a specific lumbar nerve root lesion can be diagnosed. To illustrate this, let us assume that the 1st sacral nerve root is compressed by a herniated nucleus pulposus. As in the case of the cervical nerve root lesion, a definite sequence of muscle testing is followed. The needle electrode is placed successively into several areas of the quadriceps femoris (L-2, L-3 and L-4) and no evidence of denervation is found. Progressing, the tibialis anticus (L-4 and L-5) is thoroughly sampled and, again, no denervation fibrillation voltages are picked up. However, in the extensor hallucis longus, extensor digitorum longus, and peroneus longus (L-5 and S-1) a considerable amount of denervation fibrillation voltages is demonstrated. Similarly, in the gastrocnemius (L-5, S-1 and S-2) and the soleus (S-1 and S-2) the EMG reveals evidences of denervation. Since we have demonstrated that the muscles supplied by the 4th and 5th lumbar nerve roots are not involved, whereas the muscles partially innervated by S-1 are, the lesion must be compressing this root. Finally, denervation is found in the erector spinae group which is segmentally innervated by the posterior primary divisions, thus proving that the root and not a portion of the lumbosacral plexus is implicated.

In our hands this method of localizing an isolated nerve root lesion has been used successfully in diagnosing involvement, from whatever cause, of the 6th, 7th and 8th cervical nerve roots and the 5th lumbar and 1st sacral nerve roots. Further work is being done to extend and perfect this method.

THE NERVE PLEXUSES

Lesions of the brachial plexus need to be differentiated, most commonly, from those of the cervical nerve roots, or those of peripheral nerves. Pressure
on the brachial plexus, for instance, by the anterior scalene muscle or a cervical rib, is almost invariably accompanied by EMG evidence of diffuse denervation of the upper extremity, whereas a cervical nerve root lesion shows segmental denervation only in those muscles served by that specific root. Another point of differentiation is that in brachial plexus lesions the posterior primary divisions are never involved and, therefore, denervation fibrillation voltages are never found on the erector muscles of the neck such as are found in cervical nerve root lesions. For example, we all see the patient who complains of radiating pain down the arm. Neurologic examination may reveal neither the classical signs of an anterior scalene syndrome or those of a cervical disc. Here the EMG can rule out the cervical nerve root lesion by demonstrating, first, diffuse denervation of the arm and, second, absence of fibrillation voltages in the posterior neck muscles. This evidence is present in spite of the lack of specific reflex, motor or sensory findings on clinical examination. We hope, with further experience, to confirm the fact that brachial plexus lesions can be differentiated from those occurring in the axilla by finding denervation fibrillation voltages in the infra-spinatus muscle (supplied by the subscapular nerve, which usually arises well above the axilla).

The lumbosacral plexus is involved by many conditions, either intra- or extrapelvically, without definite neurological signs differentiating the lesion from involvement of the spinal nerve roots. Again, the important EMG finding is diffuse denervation of the leg without segmental denervation of the erector spinae group. Lumbosacral plexus involvement can be distinguished from peripheral nerve injuries because in the former the denervation is usually found to be diffuse in the lower extremity whereas in the latter it is found only in the muscles supplied by the specific nerve. An important point in this respect is the differentiation of a pure lumbar plexus lesion from a femoral nerve injury, or a sacral plexus injury from a sciatic nerve injury. In both instances the plexus lesion produces denervation of the gluteus muscles which is absent in the peripheral nerve injuries.

PERIPHERAL NERVE INJURIES

The electromyograph reaches its highest degree of accuracy in the diagnosis and prognosis of peripheral nerve injuries. A completely severed peripheral nerve will show, approximately 18 days after injury (in the human), denervation fibrillation voltages from all areas of the muscles tested. In addition, no discrete motor unit voltages are produced from any portions of the affected muscles by voluntary contractions. The only bio-electrical sign seen during such voluntary activity is a disturbance of the cathode ray base-line, which is a result of motor unit contractions arising from adjacent muscles not innervated by the same nerve, and with experience this can be clearly recognized. Thus, a confident opinion can be expressed by the electromyographer that complete interruption of the nerve exists.

Immediately, therefore, the neurosurgeon can reapproximate the severed
ends without undue delay. This is of paramount importance since there is an inverse relationship between a prolonged interval before suture and the effectiveness of the results.

After a nerve suture has been done, and following an appropriate waiting period for nerve regeneration to occur, beginning motor unit contractions are seen in the paralyzed muscles. The frequency and amplitude of the denervation fibrillation voltages gradually diminishes and the cathode ray baseline disturbance is replaced at first by complex (or polyphasic) motor unit contractions of very low amplitude and frequency and, finally, simple motor unit contractions begin to appear. These electromyographic phenomena can be seen long before clinical evidence of motion occurs and assures the surgeon that his nerve suture is intact and reinnervation is occurring.

Using a mean rate of 2.5 mm. a day for neurotization, and 1.7 mm. per day for maturation (complete myelinization) a table has been constructed which makes it possible to determine the approximate time interval before EMG evidence of regeneration appears. If, after this appropriate waiting period, which is markedly shorter than the usual period of clinical waiting, no EMG evidence of regeneration of the nerves is demonstrated, immediate reexploration should be carried out. Again the neurosurgeon is afforded the earliest opportunity to establish the continuity of the nerve and, thus, forestall further atrophy of the affected nerve pathways and muscles.

In partial nerve injuries, despite the clinical evidence of complete paralysis, the EMG enables the examiner, on finding motor unit contractions, to state that intact nerve fibers exist and, therefore, the chances of spontaneous regeneration are good. The importance of this to the neurosurgeon is obvious.

In peripheral neuritis the EMG reveals far more widespread denervation fibrillation than can be detected on neurologic examination. This is often of diagnostic value in ruling out purely localized lesions or "functional" states.

CRANIAL NERVES

The most frequent use of the EMG in testing the motor function of cranial nerves is in lesions of the facial nerve. Of these, Bell's palsy is the most common. If, after three weeks (the time necessary for denervation fibrillation to commence in human muscle) denervation fibrillation voltages are found in all areas of the facial musculature and if, with voluntary effort, no discrete motor unit contraction waves occur, a diagnosis of complete interruption of the facial nerve may be made. Thus, if, after an appropriate waiting period, there is still no EMG evidence of return, the neurosurgeon can undertake early surgical intervention before irreversible changes have occurred in the muscle. Conversely, if voluntary motor unit voltages are elicited from any part of the affected muscles, no matter of how small amplitude, then, despite the clinical evidence of complete paralysis, it is possible to state that at least a portion of the nerve is still intact and to predict that spontaneous recovery should occur.

Another instance in which the electromyographic examination of the
facial muscles is of diagnostic importance is in differentiating compressive lesions of the high cervical cord (cord tumors, Arnold-Chiari deformity, etc.) from widespread degenerative disease of the nervous system. In the latter, subclinical denervation of the facial muscles can be demonstrated, whereas in the former none is found.

We hope, with further experience, to demonstrate the value of electromyography in other disorders of the cranial motor system. An example of this might be the testing of the lateral rectus muscle in traumatic lesions of the 6th nerve in order to determine whether or not the lateral rectus muscle is completely denervated so that the ophthalmological surgeon could proceed with surgical corrective measures before irreversible muscle changes could occur.

PRIMARY DISORDERS OF THE MUSCULAR SYSTEM

The most dramatic and pathognomonic electromyographic picture is seen in the myotonias. When the needle electrode is inserted into the muscles of the patient suffering from myotonia congenita (Thomsen’s disease), myotonia dystrophica (atrophica), and myotonia acquisita (Talma’s disease), there is a tremendous burst of what are apparently spontaneous, involuntary, simple motor unit discharges. These wave forms are of high amplitude, rapid in frequency, and are accompanied by a high-pitched howling noise over the amplifier. Both the characteristic wave forms and sound from the hyper-irritable muscle reach an immediate crescendo, then wax and wane to reach electrical silence within a few seconds. The slightest touch on the implanted needle electrode sets off an immediate repetition of this phenomenon. No denervation fibrillation voltages are seen, placing the myotonias in the category of a primary muscular disorder. Voluntary contraction produces normal diphasic wave forms which persist for a period after the patient attempts to relax the muscle.

In the muscular dystrophies, whether pseudo-hypertrophic, atrophic, or a combination of both, there is no evidence of lower motor neuron disease and no characteristic wave forms appear, such as are seen in the myotonias. However, with voluntary contraction, the amplitude and frequency of the normal diphasic wave forms are diminished as compared with those found in the normal skeletal muscle. With the EMG an early differential diagnosis between a pure muscular dystrophy and progressive muscular atrophy can be established.

Electrically, myasthenia gravis shows no evidence of lower motor neuron disease of the skeletal musculature. With voluntary contraction, however, as might be expected, there is a gradual diminution in the amplitude and frequency of the diphasic discharges progressing, in some instances, to a complete iso-electric base-line. After a period of rest, there is a gradual reappearance of normal contraction potentials.

Often a patient may be suspected, because of gross fascicular twitchings of the muscles of the extremities, of suffering from a degenerative disease of
the anterior horn cells such as amyotrophic lateral sclerosis. If these fasciculations are found to be, electromyographically, of a simple diphasic type, and if no denervation fibrillation voltages are seen, a myokymia may be suspected and the more serious disorder ruled out.

MEDICOLEGAL ASPECTS OF ELECTROMYOGRAPHY

The EMG assumes an important position in forensic medicine, because it offers indisputable and objective evidence of the presence or absence of lower motor neuron disease. This evidence has already been accepted as bona fide in some courts. The particular value of the EMG lies in its ability to determine more accurately than purely clinical examinations whether or not the patient is suffering from an organic disease of the lower motor neuron system. In addition, the disease process can be accurately localized in many cases, so that in the psychoneurotic or malingerer a true evaluation can be reached. With the myoscopic tape record it is possible for the physician to have in his possession a reproducible permanent record of the patient's original condition and subsequent course. Evidence of this nature is of great legal importance in this era of the litigious-minded patient.

SOURCES OF ERROR IN ELECTROMYOGRAPHY

Electromyography, depending upon a mechanical diagnostic instrument, is subject to errors arising, first, from lack of skill or thoroughness on the part of the examiner; second, from incorrect interpretation of the electromyogram; and third, from the fact that there are anatomic variations of motor nerve supply just as are seen in the sensory distribution.

As we have previously emphasized, the technique of multiple electrode insertions and extensive sampling of the skeletal muscles must invariably be carried out. There is often a tendency on the part of the electromyographer to limit his efforts because of the discomfort to the patient, thus failing to discover significant evidences of lower motor neuron disease.

Considerable experience is required on the part of the electromyographer in interpreting certain aberrant wave forms which may arise at the time of insertion or motion of the needle electrodes, from outside electrical interference, or from the normal contraction potentials of certain small muscles such as those of the face, which may be confused with denervation fibrillation voltages.

A major source of confusion arises in surveying certain patients who demonstrate widespread lower motor neuron disease in spite of the fact that they clinically have only a localized lesion. We feel that these patients may have suffered in the past from some unrecognized disease of the nervous system such as subclinical poliomyelitis, and thus retain evidences of lower motor neuron disease. In older patients this confusion also arises occasionally because of circulatory insufficiency which results in damage to the anterior horn cells. In these cases the electromyographer must utilize his clinical knowledge in interpreting the EMG findings.
Finally, it should be re-emphasized that there must be an approximate waiting period of 18 days after interruption of the nerve supply to a human skeletal muscle before denervation fibrillation voltages appear and any accurate conclusions can be reached.

CONCLUSIONS

1. The electromyograph, utilizing a monopolar needle electrode, cathode ray oscilloscope, sensitive sound amplifier, and a magnetic tape recorder, is a diagnostic instrument with a high degree of accuracy in diagnosing certain diseases of the neuromuscular system.

2. In diseases of the spinal cord affecting the anterior horn cells, the EMG offers early diagnostic and prognostic information that often cannot be obtained by any other means.

3. The differential diagnosis between certain spinal cord tumors (i.e. high cervical) and degenerative spinal cord disease is often established by electromyography.

4. Specific spinal nerve root involvement can be diagnosed accurately with the EMG and differentiated from plexus or peripheral nerve lesions.

5. In peripheral nerve lesions the EMG is of great value in determining (a) the presence of complete or partial interruption; (b) the earliest possible evidences of reinnervation following nerve suture; (c) the shortest period of delay before re-exploration can be carried out if no evidences of reinnervation have occurred.

6. The EMG can accurately separate the diseases of the anterior horn cells of the spinal cord from those affecting the skeletal musculature alone.

7. Medicolegally the EMG is of great importance because the finding of denervation fibrillation voltages is indisputable objective evidence of the presence of lower motor neuron disease.

8. The accuracy of electromyography depends first on knowledge of neuromyology; second, on proper techniques of examination; and third, on recognition of fundamental sources of error.

9. The improved clinical electromyograph is opening up new investigative fields in the physiology and pathology of the nervous and muscular systems.

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