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REVIEW ARTICLES

Advances in Electrodiagnosis in Zimbabwe. Part I: peripheral nerve conduction studies and electromyography

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SUMMARY

This paper reviews the two electrodiagnostic techniques – nerve conduction measurements and electromyography. A brief description of each technique is given followed by their clinical applications. Finally the types of abnormalities observed are discussed and where possible, illustrated.

INTRODUCTION

Electrodiagnosis is the use of electrophysiological techniques to diagnose disorders of the central and peripheral nervous system. The three basic electrophysiological techniques currently used are:

A. Peripheral nerve conduction measurements.
B. Electromyography.
C. Evoked potentials.

These techniques have recently been reintroduced to Zimbabwe.

The objective of this paper and its sequel is to review the physiological basis of these techniques and, more importantly, to discuss their relevance and use in clinical practice to diagnose or aid in the diagnosis of various

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neurological disorders. This paper (Part I) will deal specifically with *peripheral nerve conduction measurements* and *electromyography*. These are used concurrently and complement each other. The sequel (Part II) will review *evoked potentials* and their clinical applications.

PERIPHERAL NERVE CONDUCTION STUDIES.

A brief description of the techniques used will be given followed by the type of abnormalities encountered. In these studies the rate of propagation of impulses along peripheral nerves, both motor and sensory is measured. Conduction velocity is indicative of the integrity of the nerve.

Motor conduction: The peripheral nerve trunk is stimulated with surface electrodes placed on the skin overlying the nerve trunk (see Figure I). Supramaximal stimulation is applied to at least two sites (proximal and distal). The response is recorded from the muscle innervated by that nerve using surface electrodes and is displayed on a cathode ray oscilloscope. This electrical activity recorded from the muscle in response to nerve stimulation is the evoked muscle action potential (EMAP) and represents the composite depolarization of individual muscle fibres within the muscle mass. It has a characteristic wave form. The conduction velocity of the fastest fibres can be calculated from the difference between the latencies (proximal – distal), and the distance between the proximal and distal stimulating sites. Other clinically relevant parameters are the distal versus proximal latencies, the amplitude (peak to peak) of the EMAP and its shape.

Sensory conduction: To measure sensory nerve conduction a peripheral nerve is stimulated by surface electrodes and a sensory nerve compound action potential (SNAP) is recorded also with surface electrodes – although needle electrodes may be used. The sensory nerve action potential as compared with the EMAP has a much smaller amplitude and averaging techniques may have to be used to detect it. The recording electrodes may be placed distally or proximally to the stimulating electrodes such that the impulse may be propagated orthodromically or antidromically. Conduction velocity is the same irrespective of the method used. For median and ulnar nerve the index and little finger are stimulated respectively and the response is recorded by electrodes placed on the appropriate regions of the wrist.

Figure I: The set up for eliciting evoked muscle action potentials. The median nerve is stimu-lated at the elbow (proximal) and at the wrist (distal) and the response is recorded from the thenar muscle.

The latency to peak of the SNAP is measured from the oscilloscope and the conduction velocity can be calculated after the distance between the stimulating and recording electrodes is measured. The other parameter of note is the amplitude of the response.

In some neuropathies the proximal sections of the nerves are affected more than the distal segments and the following two techniques are useful in assessing the functional status of these particular segments.

The H reflex: The H reflex was discovered by Hoffman in 1918[1]. It is elicited in muscles innervated by S1 roots (ie calf muscles) in response to stimulation of the tibial nerve. The methodology involves electrically stimulating the tibial nerve in the popliteal fossa and recording the evoked muscle action potential from the gastrocnemius/soleus muscle. Two evoked muscle action potentials are seen – the short latency response (M response) which is due to direct stimulation of the motor nerves supplying the gastrocnemius/soleus and a later response (H reflex) which appears to be due to stimulation of the sensory components of the tibial nerve (probably the Ia afferents). Although the neurophysiology of the H reflex is still disputed most investigators consider it to be a monosynaptic reflex. The mean latency of the H reflex is 30 ms and is highly correlated with age and leg length such that the latency in an individual can be predicted from nomograms. The limitation of the H reflex is that it can only be used to test the proximal segments or nerves exiting in S1 roots.

The F wave: The F wave was first observed by Magladery in the early fifties.[2] It is recorded from muscles in response to motor nerve stimulation. Its long latency (25 – 30 ms) is due to the nerve impulse first travelling antidromically to the anterior horn, rebounding off the anterior horn cells and the propagating orthodromically towards the muscle. Hence it is a wave rather than a reflex since synaptic transmission does not occur. It is much smaller than the EMAP and although it cannot be used to measure motor nerve conduction velocity it is nevertheless invaluable in assessing conduction in the proximal parts of a motor nerve.

Types of nerve conduction abnormalities: These may be found in single nerves, may be generalised or affecting segments of nerves. There are two major classes of abnormalities – those due to axonal loss and those due to demyelination.

In diseases in which there is loss of large diameter myelinated fibres (i.e. axonal loss) conduction velocity is generally only mildly to moderately reduced. However, the amplitude of the EMAP is decreased but its duration is little affected.

In disorders of myelin resulting in demyelination of the nerve conduction velocity is moderately to markedly reduced and temporal dispersion of the EMAP is apparent (manifested as an increase in duration). In diseases in which both axonal loss and demyelination occurs a combination of the above findings is found.

Both axonal and demyelinating diseases may affect only sections of the nerve, either proximal or distal or the whole length of the nerve. The H reflex (if appropriate) and the F wave are useful in determining whether proximal sections of the nerve are involved.

Similar changes are also observed in the sensory nerve action potential – axonal degeneration causing a reduced SNAP, and demyelination a reduction in conduction velocity and temporal dispersion.

ELECTROMYOGRAPHY.

In electromyography the electrical activity of muscle is recorded usually by needle electrodes inserted into the muscle and the resulting electromyogram (EMG) is displayed on a cathode ray oscilloscope. The following describes the types of "activity" and are relevant to the electromygrapher.

During an EMG examination the electrical activity recorded as the needle is inserted into the muscle is observed. This is the "insertional activity". It is due to injury potentials generated as the electrode mechanically damages muscle fibre membranes in its traverse through the muscle. In a normal muscle this activity ceases as soon as the electrode is brought to rest. The patient is then asked to relax the muscle under examination. Normal muscle is quiescent and the EMG is "flat".

On voluntary contraction of the muscle "spikes" appear on the EMG. As strength of contraction increases the spikes become more numerous until with maximum contraction spike activity is so numerous that individual spikes can no longer be identified. The spikes are "motor unit action potentials" (MUAPs) and they represent the composite depolarization of muscle fibre membranes. A motor unit comprises the anterior horn cell (motor neurones), its axon and all the muscle fibres that are innervated by the terminal branches of the axon. As voluntary contraction is increased more and more motor neurones are recruited giving rise to an increasing number of MUAPs.

Of particular interest to the electromyographer is the shape of the MUAPs, normal MUAPs have a triphasic wave form, and there is a range of amplitudes and duration that are considered normal. The other parameter of relevance is the pattern observed as motor neurones are being recruited and is known as the "interference pattern". An EMG is displayed not only as a visual signal but also as an audio signal and much information can be gleaned from the sound emitted by the MUAPs.

Types of EMG abnomalities:

Fasciculation Potentials.

These are spikes recorded in a relaxed muscle. Generally they are large, complex MUAPs characterised by a slow firing rate (<5/s) which is irregular. Their origin remains unclear. Fasciculation potentials may be recorded in healthy individuals and certainly those recorded in the absence of any other EMG abnormality are considered benign.

Fibrillation Potentials.

These can also be recorded from a relaxed muscle. They differ from fasciculation potentials in that they have a low amplitude, are of short duration and discharge regularly. Although fibrillation potentials are seen in a variety of neuromuscular disorders they are not characteristic of any. It was thought that fibrillation potentials could only be recorded from denervated muscle and were due to increased sensitivity of the muscle membrane to acetylcholine. Now it appears more likely that these potentials are due to an unstable muscle membrane[3] which can be caused by various myopathies such as inflammatory conditions, altered electrolyte states (periodic paralyses) as well as in denervated muscle.

Insertional Activity.

In denervated muscle the insertional activity is often prolonged and characterised by the so called "positive sharp waves". These waves are regarded as the early indicators of denervation.

Volitional Activity.

In neurogenic lesions a decrease in the number of recruited motor units is observed. In severe muscle weakness, even with maximal innervation, only one or a few motor units which are close to the needle, discharge. Since a weak muscle has fewer motor units available for graded contraction than a normal muscle, it uses the remaining units, prematurely at a higher frequency. The interference pattern will be abnormal.

Shape, Duration and Amplitude of MUAPs.

MUAPs are normally triphasic (ie -\/-), and polyphasic potentials (-\M\-) account for less than 3 pc of all potentials in normal muscles. In peripheral nerve dysfunction and myopathic conditions polyphasicity is increased. The duration of the MUAP is increased in a variety of neurogenic disorders such as anterior horn cell disease, polyneuropathy and following traumatic lesions of the motor unit. The mean amplitude of the MUAP which represents the number of muscle fibres within the motor unit is increased in conditions in which reinnervation of muscle fibres by axons from still functioning motor neurones has occurred.

Clinical applications of nerve conduction measurements and electromyography.

Nerve conduction studies aid the clinician in several ways:

1. To distinguish between a generalised disorder (peripheral neuropathy, polyneuropathy) and local lesions (compressive syndromes).
2. To establish the site of compression in single nerve lesions (ulnar nerve at the elbow, median nerve at the carpal tunnel).
3. To document pre- and post-operative velocities in nerve release procedures.
4. To establish the type of peripheral neuropathy.
5. To establish the *absence* of a neuropathy and serve to reassure the patient suffering from "burning feet" and "tingling hands".

The EMG is clinically useful to:

1. Identify a myopathy versus a neuropathy – although there is overlap in the EMG pictures the overall impression is usually accurate.
2. Determine the degree of denervation and hence prognosis in for example, Guillain Barre syndrome, myelopathies and spinal cord injury.
3. Diagnose motor neurone disease.

A brief description of the pathological conditions in which nerve conduction studies and electromyography have proved invaluable will be given below, together with the types of abnormalities encountered.

Neurogenic disorders.

Peripheral neuropathies.

Peripheral neuropathies are commonly encountered neurological disorders. They can be divided into the following categories:

1. Idiopathic, eg Guillain Barré, chronic inflammatory polyneuropathy.
2. Hereditary, eg Charcot-Marie-Tooth disease.
3. Toxic, eg alcohol, heavy metals, drugs and organic compounds.
4. Nutritional, eg vitamin B12 deficiency.
5. Neuropathies associated with diseases, eg *Diabetes mellitus,* HIV.

Sensory neuropathies tend to give rise to paraesthesia and pain and are much more common than motor neuropathies which give rise to muscle weakness.

Electrical responses from patients suffering from Guillain Barre syndrome, and chronic inflammatory polyradiculopathy and *Diabetes mellitus* are shown in Figure II B, D and F respectively. Note the delayed and temporally dispersed response evoked by stimulating the peroneal nerve at the ankle in the patient with Guillain Barre syndrome indicating demyelination. In chronic inflammatory polyradiculopathy the F wave is delayed (2E) indicating that the proximal segment of the ulnar nerve is also involved. In the patient with *Diabetes mellitus* the sensory nerve action potential is barely discernible (2F). The patient complained of paraethesia. Figure III A and B is a comparison of the muscle action potential evoked by stimulating the ulnar nerve at the elbow in a healthy volunteer (A) and in a patient with HIV infection who complained of muscle weakness in the arms. The EMAP in the latter is delayed, reduced and temporally dispersed indicating both axonal loss and demyelination.

The responses illustrated in these figures are characteristic of these clinical conditions. Other electrical abnormalities were also encountered in these patients but are not illustrated here.

Figure II: A—B are EMAPs evoked by stimulating the peroneal nerve at the ankle of a healthy individual (A) and from a patient with Guillain Barre syndrome (B). The F wave response evoked by stimulating the ulnar nerve in a healthy individual is illustrated in C and in a patient with chronic inflammatory polyradiculopathy is shown in D. E and F are median sensory nerve action potentials recorded in a normal volunteer (E) and from a patient with Diabetes mellitus (F) Calibration marks are shown.
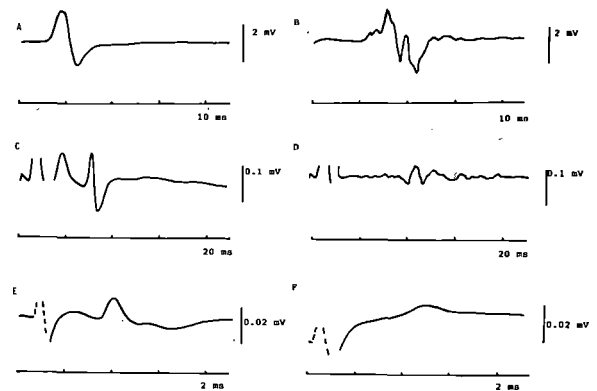
Entrapment syndromes.

Peripheral entrapment neuropathies are common and represent a specific type of pressure neuropathy in which a nerve is compressed by some other anatomical structure. EMG and nerve conduction studies are invaluable in their diagnoses. Nerve fibres exhibit two types of pathological response to injury – demyelination and axonal degeneration. The electrodiagnostic picture seen depends on the proportion and degree of various fibre changes present and the time elapsed since the injury. A useful conceptual framework for analysing the changes in EMAPs is to classify the changes into the two categories – neurapraxia and axonotmesis.

Neurapraxia is the mildest level of injury. It is a temporary segmental block in conduction. Recovery

Figure III: A and B are EMAPs evoked by stimulating the ulnar nerve at the elbow in a healthy individual (A) and in a person infected with HIV (B). C and D are the EMAPs evoked by stimulating the radial nerve in the left arm (C) and in the right arm (D) of a patient with radial nerve palsy. E and F are the Hoffman reflex recorded from a healthy individual (E) and from a patient with S1 radiculopathy (F). Calibration marks are shown.
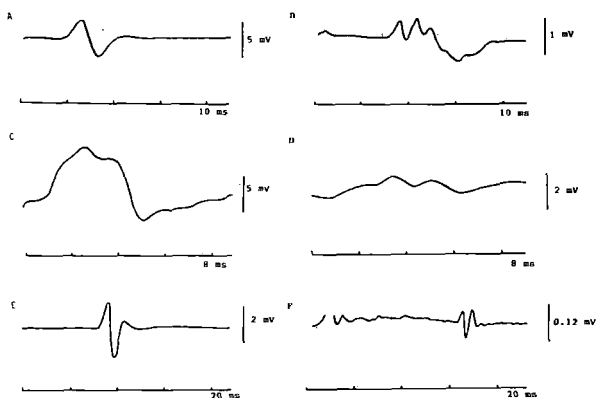
can be rapid and complete if the source of injury is removed. The EMAP is absent when the neurapraxic axon is stimulated proximally to the lesion but is normal following distal stimulation. In incomplete neurapraxia a proximal EMAP is present but reduced in amplitude and the conduction velocity is slowed. Figure III C (left arm) and D (right arm) illustrate the muscle action potential evoked by stimulating the radial nerve in a patient who presented with wrist drop in the right limb. The radial nerve was stimulated above the lesion and the presence of an EMAP, albeit much reduced, suggest that the patient had an incomplete neurapraxia. In a chronic entrapment syndrome such as carpal tunnel and tardy ulnar palsy the distal EMAP is normal and the proximal EMAP is reduced in size and temporally dispersed.

Axonotmesis results from injury sufficient to cause Wallerian degeneration with dissolution of the distal segment of the axon. Stimulation at any point along such an axon produces no EMAP. EMG changes such as positive sharp waves and fibrillation potentials in the denervated muscle will develop 5 – 28 days post injury.

Anterior horn cell diseases.

Many different types of disorders affect the anterior horn cell.

Amyotrophic lateral sclerosis.

This is the most common disorder affecting motor neurones and is commonly referred to as "motor neurone disease". Its aetiology remains a mystery. It is a progressive disease usually starting as a painless asymmetric weakness. It proceeds at a variable tempo; death ensues usually from respiratory paralysis or aspiration after bulbar palsy. The classic electrodiagnostic picture in this disease is positive sharp waves, fibrillation potentials, MUAPs of increased amplitude, duration and phasicity and a decreased number of recruited MUAPs per strength of contraction. Sensory and motor conduction velocities are essentially normal. EMG abnormalities may also be seen in clinically unaffected muscles and are invaluable in confirming the diagnosis. Repeated EMG examinations throughout the course of the disease are helpful in following the progression of the disease and may be useful in predicting outcome.

Poliomyelitis.

The EMG in poliomyelitis shows large complex MUAPs together with positive sharp waves and fibrillation potentials. The latter may still be present 20 years post infection.

Myelopathies.

Disorders affecting the spinal cord as a whole may have their primary effect on the anterior horn. Infections, lesions from direct trauma, pressure or infiltration from tumours and vascular insults may all have a relatively selective effect on the anterior horn producing an EMG picture similar to that of motor neurone disease.

Radiculopathies.

Radiculopathies are caused by compression of either posterior or anterior spinal roots or both. They commonly occur in the cervical and lumbar region. In acute nerve root compression there is a sequence of EMG abnormalities. During the initial few days following onset tendon reflex latencies are prolonged due to conduction block. In a S1 radiculopathy the H reflex is delayed (see Figure III F). Prolongation of the F wave is present when other roots are involved. After three days or so the EMG shows an increase in polyphasicity in muscles innervated by the root, after about a week positive sharp waves are seen in the paraspinal muscles and then fibrillation potentials appear in the paraspinals and in muscles supplied by the anterior rami. The number of MUAPs per strength of contraction is also decreased. Other neuromuscular disorders may give a similar picture. However, the hallmark of a radiculopathy is the finding of positive sharp waves and fibrillation potentials in the paraspinal muscles.

Disorders affecting neuromuscular transmission.
Myasthenia gravis.

Myasthenia gravis is characterised by muscle weakness after effort. Since muscle fatigues easily in this condition the definitive electrophysiological test is recording EMAPs in response to repetitive (2 – 3 /s) stimulation of a peripheral nerve. In a healthy individual the amplitudes of each successive EMAPs are more or less constant but in the myasthenic patient the EMAP diminishes in amplitude with each successive contraction. Several nerve muscle combinations should be tested since not all muscles are affected to the same extent in myasthenia.

Myogenic disorders.
Duchenne muscular dystrophy.

The EMG is characterised by MUAPs of diminished duration and amplitude and increased polyphasicity. These changes are due to muscle fibre degeneration. Regenerating muscle fibres complicate the EMG picture since superimposed on the small MUAPs may be short duration, high amplitude spikes recorded from nearby hypertrophied fibres. Fibrillation potentials and positive sharp waves can be recorded in up to half the patients with this type of dystrophy.

Polymyositis and dermatomyositis.

These conditions typically present with symmetrical proximal limb weakness often accompanied by muscle pain, tenderness and swelling. Histologically there is degeneration and phagocytosis of muscle fibre together with regeneration. Fibrillation potentials and positive sharp waves are recorded. On voluntary contraction polyphasic MUAPs of diminished amplitude and duration are apparent.

CONCLUSIONS

The neurophysiological basis of nerve conduction measurements and electromyography have been reviewed and a few clinical situations in which nerve conduction studies and EMG are helpful have been presented. They are of course merely adjuncts to good history taking and careful examination but may aid the diagnostic process, as well as allowing clinicians to critically assess the state of the peripheral nervous system.

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