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## Nerve conduction study. An approach to clinical application

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### ABSTRACT

The electrical activity of a discreet region of an accessible muscle can be registered by means of the insertion of an electrode and needle. The pattern of electrical activity in the muscle (electromyogram) in rest and during the activity has determined itself, and the abnormality has been correlated with disorder at different levels from motor unit. Studies of motor nerve conduction. These studies practice by means of the registry of the electrical answer of a muscle to the stimulation of their nerve motor two or but points throughout their passage, which determines the speed of fast conduction but in motor fibers of conduction between the stimulation points

**KEY WORDS:** Latency of nerve conduction, F-wave, motor multifocal neuropathy, cranial nerve testing.

### RESUMEN

*La actividad eléctrica de una región discreta de un músculo accesible se puede registrar mediante la inserción de un electrodo y aguja. Se ha determinado el patrón de actividad eléctrica en el músculo (electromiograma) en reposo y durante la actividad, y las anomalías se han correlacionado con trastornos a diferentes niveles de unidad motora. Estudios de conducción de nervios motores. Estos estudios se practican mediante el registro de la respuesta eléctrica de un músculo a la estimulación de su nervio motores dos o más puntos a lo largo de su trayecto, lo que determina la velocidad de conducción más rápida en las fibras motoras de conducción entre los puntos de estimulación.*

**PALABRAS CLAVE:** Velocidad de conducción nerviosa, onda -F, neuropatía multifocal motora, examen de pares craneales.

### INTRODUCTION

The injuries can affect the muscular and neural component or of the motor unit, or the neuromuscular junction. When the neural component is affected, the pathological process can be at cells level of the spinal cord, horn previous or in some point throughout axon to the passage by a nervous system root, plexus of the extremity and a peripheral nerve. Before final ending fibers itself in its terminal muscle (white organ).

The electromyography detects disorder of the motor units and indicates the site of the underlying injury. The technique also allows detecting the neuromuscular disorder when the clinical examination is not productive because the disease is in an early stage (or due to poor man cooperation on the part of the patient or the presence of

other symptoms as pain makes difficult the clinical evaluation). Those electrographic findings do not allow, by it, to obtain the etiology or diagnosis, and the electrophysiology is due to correlate with the clinical discoveries and permit to compare the results of other studies of laboratory.

The discoveries electromyography can provide a guide for the prognosis; for example. A patient with an acute upheaval of a peripheral or cranial nerve (for example, a paralysis by pressure of the radial, medium nerve [carpal tunnel syndrome or a Bell palsy] of the face nerve -VII cranial nerve-, or the trigeminal nerve -V cranial nerve-, the electromyography's data of desnerivation imply what the prognosis is poor for the recovery when denervation has not happened.

## 1) Short and Long Pathways<sup>1</sup>

In the evaluation of a focal lesion, studies of a longer segment tends to lower the sensitivity of the test because the inclusion of the unaffected segments in calculation dilutes the effect of slowing at the site of lesion. In contrast, studying a shorter segment helps isolate a localized abnormality and provides better resolution of restricted lesions that may otherwise escape detection.

For example, patients with the carpal tunnel syndrome show a sharply localized latency increase, averaging 0.8 ms across a 1 cm segment. This, compared to a normal value ranging from 0.16 to 0.21 ms, clearly indicates a focal abnormality. An abrupt change in waveform of the recorded response provides an additional, and perhaps more convincing.

Finding that nearly always accompanies a latency increase across the site of compression. In fact, waveform analysis often localizes a focal lesion unequivocally even in the absence of an abnormal latency prolongation. This technique suits not only in assessing a possible compressive lesion but also in characterizing focal nature of some widespread abnormalities such as multifocal motor neuropathies.

Despite the traditional use of conduction studies across a relatively short distal portion of the peripheral nerves, a longer segment may provide a better result in assessing a more diffuse or multi-segmental process such as polyneuropathies. A longer path has an advantage in accumulating all the segmental abnormalities, which individually might not show a clear deviation from the normal range.

Thus, in general, the longer the segment under study, the more evident the conduction delays for a diffuse process. A number of neurophysiologic methods supplement the conventional techniques for the assessments of longer pathways. The selection of such techniques necessarily reflects the special orientation of each laboratory. Those of general interest include the F wave and the H reflex. Assume a nerve impulse conducting at a rate of 0.2 ms/cm (50 m/s). A 20 percent delay for a 10 cm segment is only 0.4 ms, whereas the same change for a 100 cm segment amounts to 4.0 ms. an obvious increase for easy detection.

Evaluating a longer, as compared to shorter, segment also improves the overall accuracy because the same absolute error leads to a smaller percentage of change in measuring either the latency or the distance. In routine practice, a surface measurement of a 10 cm nerve segment may yield an estimated distance of 9.5 to 10.5 cm. A 1cm difference constitutes a 10 percent error, or a calculated conduction velocity between 50 m/s and 55 m/s.

The same 1 cm error in a 100 cm segment represents only 1 percent error, or a conduction velocity between 50 m/s and 50.5 m/s.

The same argument holds in determining the effect of possible error in latency measurement. Thus, studying a longer path offers a better sensitivity and accuracy as well as improved reproducibility in serial studies.

## 2) Reproducibility of Various Measures<sup>2</sup>

We conducted a multicenter analysis on intertrial variability of nerve conduction studies in preparation for future drug assessments in diabetic polyneuropathy. All measurements were repeated twice at a time interval of 1-4 weeks by the same examiners, who underwent a hands-on workshop to standardize the method. In all, 32 centers participated in the study of 132 healthy subjects (63 men) and 65 centers in the evaluation of 172 patients with diabetic polyneuropathy (99 men). The protocol consisted:

- 1) Motor nerve conduction studies of the left median and tibial nerves for measurement of amplitude, terminal latency, and minimal F-wave latency, and calculation of motor conduction velocity and F-wave conduction velocity
- 2) antidromic sensory nerve conduction studies of the left median and sural nerves for recording of amplitude and distal latency, and calculation of sensory conduction velocities. In both the healthy subjects and patients with diabetic neuropathy, amplitude varied most. Followed by the terminal latency, and motor and sensory conduction velocities.

The minimal F-wave latency showed the least change, with the range of variability of only 10 percent for the median nerve and 11 percent for the tibial nerve in normals. The corresponding values were 12 percent and 14 percent, respectively.

In patients with diabetic polyneuropathy. These results support the hypothesis that the minimal F-wave latency serves as the most reliable measure of nerve conduction for a sequential study in the same subjects.

When evaluating individual patients against a normal range established in a group of subjects, however, F-wave conduction velocity suits better, because it minimizes the effect of limb length. Alternatively, some prefer the use of a nomogram plotting the latency against the height as a simple, albeit indirect, measure of limb length.

## 3) Clinical Consideration<sup>3</sup>

Our data indicate that the length of the nerve segment under study dictates the accuracy and sensitivity of measurement. Although studies of shorter or longer segment pose technical merits and demerits. The choice seems to depend entirely on the pattern of the conduction abnormalities. In summary, short distances magnify focal conduction abnormalities despite increased measurement error, and long distances, though insensitive to focal lesions, provide better yields and reliability for a diffuse or multisegmental process. These findings also underscore the importance of choosing nerve stimulation techniques best suited for detecting the clinically suspected lesion. Thus, electrophysiological studies serve ell only hen conducted as an extension of the history and physical examination, which provide an overall orientation for subsequent physiologic evaluation.

## Multifocal Motor Neuropathies

Multifocal motor neuropathy (MMN), a variant of CIDP, deserves special mention to distinguish this potentially treatable condition from amyotrophic lateral sclerosis (ALS) and other motor neuron syndromes. These patients develop chronic asymmetric predominantly motor neuropathy with multifocal conduction delay and persistent conduction block. Although MMN typically causes distal upper limb weakness and atrophy, proximal muscles, biceps brachii in particular, may show hypertrophy possibly associated with continuous motor unit activity. Similar to earlier reported cases with sensory and motor involvements the long-lasting conduction block suggests chronic demyelization as the pathological basis.

The patient often has normal or occasionally even increased stretch reflexes with a normal or only slightly elevated CSF protein. Some patients develop cranial nerve involvement and others, central demyelization. These features make it difficult to diagnose the condition solely on the basis of clinical examination. Conduction blocks typically involve unusual sites such as the median nerve in the forearm or brachial plexus rather than the common sites of compression seen in multiple entrapment neuropathies. Most patients have selective involvement of motor fibers with normal sensory conduction through the sites of motor conduction block.

Both motor conduction block and abnormally increased threshold probably reflect a chronic focal demyelization lesion, which for yet undetermined reason becomes persistent without repair. Some patients with features indistinguishable from ALS have multifocal motor nerve conduction abnormalities. In one series 17 of 169 patients clinically diagnosed as motor neuron disease had some abnormalities in motor nerve studies including 10 with conduction block.

Demonstration of motor conduction block at multiple sites differentiates this potentially treatable clinical entity from the small subgroup of ALS patients with only lower motor neuron involvement.

Electrophysiologic studies must confirm the diagnosis before initiating therapeutic trials using. For example, immunosuppressant such as cyclophosphamide. Several authors have documented a successful treatment by intravenous immunoglobulin. Outcomes of therapy by either immunosuppressants or immunoglobulin vary considerably among different reported cases. Some patients improve but do not return to normal, others stabilize, some require long term therapy and still others become refractory to any form of treatment. Most studies suggest more favorable results after cyclophosphamide or human immune globulin therapy compared to prednisone or plasmapheresis.

In our series, two cases of MMN had focal conduction block involving motor but not sensory fibers at the site of nerve swelling.

A nerve biopsy, taken adjacent to the enlargement in one patient revealed subperineurial edema and slight thickening of the perineurium under low-power light micrographs.

The perivascular area at the center contained scattered large-diameter axons almost devoid of myelin or with very thin myelin. These thinly-myelinated axons usually had small onion bulbs. The presence of cytoplasmic processes covered with basement membrane suggested their Schwann cell origin. A nerve biopsy specimen from another patient also revealed perivascular area containing scattered demyelinated axons surrounded by small onion bulbs. Morphometric studies using high-power light micrographs showed a fiber density of 6458 fibers/mm<sup>2</sup>, as compared to 7906 fibers/mm<sup>2</sup> in the control. Axonal diameter and myelin thickness showed a linear relationship in the normal subjects.

By contrast, the patient had numerous large-diameter axons with thinner myelin, although some normally-myelinated large axons remained. The underlying pathogenic mechanism centers on elevated titers of anti-GM1 antibodies found in a wide variety of neuromuscular conditions, but more commonly in some lower motor neuron disorders and MMN. Antibodies may have predilection to the GM1 component of motor fibers, which have a longer carbon chain than in sensory fibers.

Auto antibodies may exert their effect, in part, by binding to GM1 on the surface of motor neurons. Anti-GM1 antibodies may or may not cause motor dysfunction by binding to the nodal and paranodal regions. Sera of patients with MMN but not with progressive spinal muscular atrophy induced conduction block in rat's tibial nerve despite similar elevation of anti-GM1 titers in both categories.

These antibodies however, may not have a causal relationship with MMN, as evidenced by many patients without raised levels. Surface-bound antibodies directed against major axoplasmic antigen may act interfering with remyelination rather than causing demyelization. In some cases, nerve ischemia may play a role in the pathogenesis.

In an extraordinary case, a patient had duck embryo rabies vaccine three months before the onset of her motor neuron disorder. She had multifocal conduction block, elevated levels of anti-GM1 IgM antibodies and deposits of IgM at nodes of Ranvier. Aside from attacking motor neurons guided by the abundant GM1 on the cell-surface, anti-GM1 antibodies may cause conduction block in peripheral nerves by binding to the node of Ranvier. An autopsy study in another patient showed findings consistent with both ALS and MMN. It is necessary to clarify the exact pathogenesis underlying these findings to properly classify the motor neuron disease and MMN.

## Cranial Nerve Testing

Isolated cranial nerve palsies may result from lesions of the respective nerves along their extra-axial courses or as the sole manifestation of brainstem lesions. Cranial nerves most commonly assessed in an electromyography laboratory include the facial and accessory nerves. They both travel superficially to allow easy access to electrical stimulation from the surface. They also innervate the muscles readily approachable by needle or disk electrodes for recording.

## 1) FACIAL NERVE<sup>4</sup>

Bell's palsy affects the facial nerve sporadically in an isolated incidence. Although the exact etiology remains unknown accumulating evidence suggests that herpes simplex virus type I (HSV-1) reactivation causes Bell's palsy in some, but not all patients, giving a rational for antiviral therapy with acyclovir. The same principles apply to the electromyography examination of facial and limb muscles. In the face, however, physiologically small motor unit potentials may mimic fibrillation potentials, and signs of denervation appear early in less than three weeks following injury presumably because of the short nerve length. Serial electro diagnostic studies help delineate the course of the illness.

The amplitude of the direct response elicited by stimulation of the facial nerve provides the best means for prognosis after the fourth to fifth day of onset. An amplitude greater than one half the control value on the normal side indicates a good prognosis, although late degeneration can still occur. Preservation or return of R1 or R2 of the blink reflex also serves as a reliable measure in predicting a satisfactory recovery, providing a reasonable assurance that the remaining axons will survive. Diabetic patients who develop a facial palsy also tend to have a more severe paresis and the evidence of substantial denervation. Acoustic neuroma strategically located at the cerebellopontine angle; May compress not only the facial nerve, but also the trigeminal nerve and the pons. i.e., the efferent. Afferent and central arcs of the blink reflex. Peripheral facial palsy may herald other symptoms of multiple sclerosis in young adults. In these cases, blind reflex studies usually show an absent or delayed R1. indicating demyelization of the central reflex arc, which includes the intrapontine portion of the facial nerve. Myokymic discharges, although characteristic of this disorder may also appear in other conditions such as pontine glioma, and subarachnoid hemorrhage.

## 2) TRIGEMINAL NERVE<sup>5</sup>

Trigeminal sensory neuropathy characteristically evolves with unilateral or bilateral facial numbness sometimes accompanied by pain, paresthesia and disturbed taste. This type of neuropathy may accompany systemic sclerosis or mixed connective tissue disease. Patients with trigeminal neuralgia have altered coetaneous sensation not only in the affected but also unaffected adjacent divisions, suggesting combined peripheral and central pathology. A mandibular fracture may result in an isolated lesion of the mandibular nerve.

Demyelinating lesions affecting pontine trigeminal pathways may cause trigeminal neuralgia in patients with multiple sclerosis. Exposure to trichloroethylene causes a cranial neuropathy with peculiar predilection to the

trigeminal root damage. Facial numbness may herald other symptoms of an expanding tumor involving the trigeminal nerve. Other causes of trigeminal nerve lesion include perineural spread of carcinoma. The blink reflex help establish abnormalities of the trigeminal nerve. Other techniques of interest include conduction studies of the trigeminal motor nerve and of the mandibular nerve.

## 3) ACCESSORY NERVE<sup>6</sup>

Pressure from a tumor or surgical procedures of the posterior triangle can damage the spinal accessory nerve. Other causes include stretch induced injury, cargo loading, coronary artery bypass, carotid endarterectomy, and ligature injury during surgical exploration. In trapezius palsies following injury of the accessory nerve, the upper vertebral border of the scapula moves away from the spinal vertebrae. With the lower angle of the scapula relatively fixed by muscles supplied by the C-3 and C-4 roots through the cervical plexus, the whole scapula slips downward and the inferior angle rotates internally or clockwise for the right, and counter clockwise for the left scapula as viewed from the back. This type of winging tends to worsen by abduction of the arm to the horizontal plane that displaces the superior angle further laterally. The paralysis of the sternocleidomastoid causes weakness in rotating the face toward the opposite shoulder in proportion to the degree of muscle atrophy. Bilateral involvement of the muscles makes the flexion of the neck difficult. In a sequential study of patients with trapezius palsy, nerve conduction changes revealed the evidence of spontaneous regeneration after complete axonal degeneration.

## 4) OTHER CRANIAL NERVES

Hypoglossal nerve palsy may result from compression by kinking of the vertebral or aneurysm, or as a complication in approximately 5 percent of endarterectomies

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