**Cercetări Experimentale & Medico-Chirurgicale**
Anul XIII • Nr.1/2006 • Pag. 41-44

Cercetări experimentale & medico-chirurgicale

# CLINICAL UTILITY OF ELECTROMYOGRAPHIC EXAMINATION IN NEUROPATHIES

**Claudia Borza**[1]

***SUMMARY:***

*The aim of this study was to evaluate the utility of EMG examination in prognostic evaluation of neuropathies.*
*Results: the motor deficit and the decrease of muscular force were the main clinical signs in these diseases. In 65% of the patiens, the cause of neuropathy was the appearance of the melitus diabetes. The nerve conduction velocity (NCV) value is significantly decreased in patients with melitus diabetes who have the disease more than one year. An inversely proportional relationship exists between the NCV value and the duration of disease (between 1-25 years).*
*The NCV determination together with EMG exam are useful for asymptomatic latent neuropathy diagnosis in patients with a short duration of disease (1-2 years). The EMG examination revealed the denervation signs and was correlated with the decrease of NCV by a prolonged proximal and distal latency. On EMG recording during maximal contraction the aspect depending on the type of lesion is: normal in minor axonal lesion, in segmental demyelination without conduction block and in secondary axonal degenerations. The aspect is modified in severe secondary degenerations and in demyelinations with conduction block when the decrease of potentials amplitude appears. During the disease evolution, the reinervation potentials appear.*
*On EMG recording, neurogenic type is characterized by a spontaneous activity in resting state and during contraction by a low recrutation pattern and high amplitude and duration potentials were record.*
*Conclusion: The changes of peripherical nerves function depend on the site of primary disease. These studies of EMG aspect and NCV determination are useful for diagnosis of the disease site. The dynamic studies establish the regeneration potential, prognosis and the therapy efficiency.*

Received for publication: 15.12.2004
Revised: 01.02.2005

*1 - Department of Physiology, University of Medicine and Pharmacy "Victor Babeş" Timişoara*

## Introduction

The modifications of peripherical nerves functions can be classified depending on the site of the primary disease. The diseases which produce the death of neuron with the loss of the neuronal soma and axon are defined as neuropathies. The selective damages of the axons are defined as axonopathies. The axonopathies can be focalized or generalized. The axonal regeneration represents 1-2 mm/day.

Other neuropathies affect primarily the myelin layer direct or in relationship with the functions of Schwann cells; the result is a selective demyelination with a relative axonal integrity. This effect can appear to Ranvier nodes (paranodal demyelination) or to the internodal segment (segmental demyelination) with corresponding conduction block.

In other neuropathies the nervous fibers can be secondary damaged by processes which affect primarily the nervous connective tissue or vasa vasorum. Frequently a combination between demyelination and axonal loss appears (1, 2, 3).

Classification of the neuropathies - in table no 1.

***Correspondence to:*** Claudia Borza (claudia_borza@yahoo.com), Department of Physiology, University of Medicine and Pharmacy « V. Babeş » Timişoara, str. Col. Martir Ioan Uţă 2, 300041 Timişoara

**Table 1.** *Classification of the neuropathies*

| Neuropathy type | Diseases |
|---|---|
| **Toxic neuropathies** | • Toxic neuropathies<br>• Polyneuropathies by deficiencies<br>• Dismetabolic polyneuropathies<br>• Hereditary polyneuropathies<br>• Nevralgic amyotrophy |
| **Myelinopathies** | • Toxic neuropathies<br>• Infectious neuropathies<br>• Dismetabolic polyneuropathies<br>• Hereditary polyneuropathies |
| **Mixed neuropathies (axonal and with demyelination)** | • Toxic neuropathies<br>• Autoimmune neuropathies<br>• Dismetabolic polyneuropathies<br>• Paraneoplasic neuropathies<br>• Infectious neuropathies |

### Clinical symptoms

The decrease of the muscular force and paresthesia are produced by the motor conduction block or axonal degeneration. The conduction block is associated with demyelination with axonal integrity (neuropraxia). If axonal damage appears, the axonal degeneration appears beyond the interruption site. The motor deficit is associated with atrophy and denervation EMG signs. An important mechanism in regeneration, when a partial denervation exists, is the reinervation of these fibers through the collaterals of the normal neurons.

In symmetrical, generalized neuropathies the motor deficit and atrophies are peripheral and appear primarily to the legs (step walk). In the arms, the motor deficit and the atrophy appear to the small muscles and the decrease of muscle force appears primarily to the finger extensors and then to the flexors. In other neuropathies a symmetrical damage of proximal muscles of limbs appears (in Guillan-Barre syndrome or in porphyritic neuropathy). The absence of osteotendinous reflexes (frequently achilean reflex) is associated with peripherical neuropathies.

The sensibility disturbances, distal localized are associated with symmetrical neuropathies. Other complications of peripherical neuropathies can be the trophic lesions (4, 5).

### Positive diagnosis

The anamnesis and clinical exam can prove the modification of peripherical nerves functions. The conduction velocity study in motor and sensitive fibers is important in localized and generalized neuropathies.

In myelinopathies, the decrease of conduction velocity, characteristic for segmental demyelination. The decrease of velocity conduction can be diffuse or localized for a nerve segment (in compression neuropathies). The value is useful for the site of compression diagnosis and for the surgical therapy. The EMG aspect in peripherical motor neuron lesions depends on the inervation ratio. These examinations are useful in subclinical stage when only subjective signs exist.

## Material and method

The aim of this study is to evaluate the EMG modifications in 28 patiens with neuropathies in Laboratory of Functional Exploration of Clinical Hospital Timisoara. In study group 50% of patiens were in 50-59 years old group and 71% of patients were women.

The classification of the diseases - table no 2.

**Table 2**. *Classification depending on the neuropathy type*

| Neuropathy type | No. Cases | % |
|---|---|---|
| **I. Axonal** | | |
| Vitamins deficit (alcohol consumption) | 2 | 7 |
| **II. Myelinopathies** | | |
| Dismetabolic neuropathies (melitus diabetes) | 18 | 65 |
| Hereditary neuropathies (Charcot-Marie-Tooth disease) | 1 | 3.5 |
| **III. Mixed neuropathies** | | |
| Autoimmune disease | 2 | 7 |
| By compression (carpian tunnel syndrome) | 4 | 14 |
| Toxic (saturnism) | 1 | 3.5 |

**Table 3.** *NCV evaluation in patients with diabetic neuropathy*

| NCV value | % cases |
|---|---|
| Normal (45-65 m/s) | 56.40 |
| Decreased (36-41 m/s) | 43.60 |

## Results

### Clinical examination

The pain and the decrease of muscular force and motor deficit were the most important clinical signs.

### Paraclinic examination

Determination of nerve velocity conduction (NCV)

Determination of NCV represents an important method for the peripherical neuropathy diagnosis. An electro-myograph with neurostimulator was used. The normal value NCV in peripherical motor nerves is 45-65 m/s.

The decrease of NCV was correlated with the glycemia value which varied between 130-300 mg%. The NCV value significantly decreased in patients who have diabetes mellitus for more than one year. An inversely proportional relationship exists between the NCV value and the duration of disease which varied between 1-25 years.

The NCV determination together with EMG exam are useful for asymptomatic latent neuropathy diagnosis in patients with a short duration of disease (1-2 years). The EMG examination revealed the denervation signs and was correlated with the decrease of NCV by a prolonged proximal and distal latency (table 3).

Electromyographic examination

The EMG recording analysis consists of the amplitude, duration and the form of motor unit potentials (MUP) evaluation (table 4, 5, 6, 7, 8).

## Discussions

The EMG neurogenic modifications vary and depend on the site of lesion in the segment of the peripherical motor neuron: (pericarion, roots, plexus and the nervous branches). Lesion can be diffuse or localized. The lesion depends on the cause of the disease: hereditary, toxic, degenerative, compression, autoimmune dismetabolic mechanism. The classification of peripherical motor neuron deficits depends on the site of the lession in neuro- pathies, axonopathies and myelinopathies. Frequently the peripherical neuropathies are mixed: axonopathies and myelinopathies. In axonopathies the conduction velocity is normal or decreased with 10-30%. In axono- pathies in early stage the decrease of the amplitude of evoked muscle potentials appears. This represents an early electrophysiologic sign which is useful for the diagnosis of lesion type when the nerve conduction is normal.

In axonal degeneration during resting state, fibrilation potentials and positive sharp waves after 2-3 weeks from the beginning of the disease were observed. Their appearance represent a severe form of the disease. The duration of the disease is 7-8 months and in forms without fibrilation, the reinnervation potentials after 3-4 weeks appears (9, 10).

On EMG recording during maximal contraction the aspects depend on the type of lesion: normal aspect in

**Table 4.** *MUP amplitude*

| Amplitude potentials ( V) | % cases |
|---|---|
| Normal (400-600 V) | 20 |
| Increased | 80 |

**Table 5.** *MUP duration*

| Duration potentials | % cases |
|---|---|
| Normal | 22 |
| Increased | 78 |

**Table 6.** *MUP form*

| Form potentials | % cases |
|---|---|
| Monophasic | 40 |
| Polyphasic | 60 |

**Table 7.** *EMG aspect during resting state*

| Spontaneous activity | % |
|---|---|
| Positive | 12 |
| Negative | 88 |

**Table 8.** *EMG aspect depending on the muscle contraction intensity*

| Recording type | Contraction intensity | % |
|---|---|---|
| Simple | Medium | 60 |
| Intermediary | Maximal | 40 |

minor axonal lesion, in segmental demyelination without conduction block and in secondary axonal degenerations. The aspect is modified in severe secondary degenerations and in demyelinations with conduction block when the decrease of potentials amplitude appears. During the disease evolution, the reninervation potentials appear. Depending on the value of motor conduction velocity, the patients were divided into two study groups:

- I-st group: patients with objective signs of motor-sensitive polyneuropathy
- II-nd group: patients with subjective signs.

In group I and II the motor conduction velocity was significantly decreased.

The velocity in group I was:

- 36 $\pm$ 2 m/s in sciatic nerve (normal – 49.78 $\pm$ 0.98 m/s) ;
- 43 $\pm$ 3 m/s in median nerve (normal – 57.2 $\pm$ 0.93 m/s);
- 44 $\pm$ 5 m/s in ulnar nerve (normal – 57.33 $\pm$ 0.91 m/s);

The velocity in group II was:

- 42 $\pm$ 1.85 m/s in sciatic nerve
- 49 $\pm$ 2 m/s in median nerve;
- 50 $\pm$ 2 m/s in ulnar nerve.

In group I in 50% of patients the reinnervation potentials appear and in group II in 74% of patients. This aspect expressed that the regeneration potential is increased in minor forms of the disease. The EMG modifications appear before the alteration of the nerve conduction velocity. These studies of EMG aspect and NCV determination are useful for diagnosis of the disease site. The dynamic studies establish the regeneration potential, prognostic and the efficiency of therapy .

## Conclusions

The neuropathies prevalence is increased in male patients

Clinical signs associated with these diseases are: the decrease of muscle force, motor deficit, paresthesias.

The neurogenic EMG recording is characterized by a spontaneous activity ( fibrilation potentials) in patients with a short duration of disease

The bioelectric potentials during contraction are poliphasic, with a high duration and amplitude. Frequently a correlation between contraction intensity and type of recording does not exist (low recrutation pattern)

Determination of NCV represents an useful test for diagnosis of diabetic neuropathy. The diagnosis of asymptomatic latent neuropathy was exclusively establish based on the the NCV determination and EMG recording

The decrease of NCV was present in a high ratio in patients with non-insulin-dependent melitus diabetes with diabetic neuropathy

The value of NCV was correlated with the glycemia value and the duration of disease (inversely proportional relationship)

In axonal neuropathies the decrease of NCV is correlated with the denervation EMG aspect. In severe segmental demyelination the decrease of amplitude and duration of potentials appears with a low value of NCV.

**References:**


1. **Dawson, D.M., Hallett, M. and Millender, L.H.** Entrapment neuropthies. (2nd edn). *Little, Brown and Co, Boston*, 1990.
2. **Dyck, P.J., Thomas, P.K., Ashbury, A.K., Winegrad, A.I. and Porte, D.** Diabetic neuropathy. *W.B. Saunders, Philadelphia*, 1987.
3. **Lundborg, G.** Nerve injury and repair. *Churchill Livingstone. Edinburgh*, 1988.
4. **Schaumburg, H.H., Berger, A.R. and Thomas, P.K.** Disorders of peripheral nerves (2nd ed). F.A. Davis, Philadelphia, 1991
5. **Stewart, J.D.** Focal peripheral neuropathies. *Elsevier. New York*, 1987.
6. **Chu - Andrews J, Johnson R.** - Electrodiagnosis - An Anatomical and Clinical Approach, *JB., Lippincott Company*, 1986.
7. **Dyck, P.J., Thomas, P.K., Griffin, J.C., Low, P.A. and Podulso. J.** Peripheral neuropathy, (3rd ed). W.B. Saunders, Philadelphia, 1993.
8. **Suter, U. and Patel, E.I.** Genetic basis of peripheral neuropathies. In:*Human Mutation*, 3,95- 102, 1994.
9. **Kimura J.** - Electrodiagnosis in Diseses of Nerve and Muscle: Principles and Practice. *Philadelphia, FA Davis*,1983
10. **Stomatoiu I, Aşgian B., Vasilescu C.** – Clinical electromyography, *Med Ed*., 1981.