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Research Article

Interest of Electroneuromyography in the Diagnosis of Peripheral Neuropathy in the Neurology Department of the Ignace Deen National Hospital

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ABSTRACT

Background: Peripheral nerve involvement during disease states is extremely common and remains a major cause of disability. The aim of our study was to determine the role of electroneuromyography (ENMG) in the diagnosis of peripheral neuropathy. A

Methods: We conducted a descriptive cross-sectional study over a period of three (3) months at the neurosensory functional exploration unit of the neurology department of the Ignace Deen national hospital.

Results: ENMG was performed in 104 patients; 62 men (60%) and 42 women (40%), i.e. a sex ratio of 1.5. The mean age was 50 ± 16 years. ENMG results were pathological in the majority of our patients (93.3%). Axonal damage was the most common type of neuropathy (85.6%). Truncal involvement was found in 88.7% of patients. The external popliteal sciatic nerve (EPS) and internal popliteal sciatic nerve (IPS) were the most affected, with 76.3% and 47.4% respectively. The most common diagnosis was multiple mononeuropathy (47.4%), followed by polyneuropathy (32%).

Conclusion: A long-term study of other aspects of electroneuromyography would allow better exploration of the different types of peripheral neuropathy.

Keywords

Contribution, Electroneuromyogram, Peripheral neuropathy, Conakry-Guinea.

Background

Peripheral neuropathies (PN) are defined as the clinical, electrical, biological and histological manifestations of peripheral neuron damage [1]. Peripheral nerve damage during disease states is extremely common and remains a major cause of disability [2]. Peripheral polyneuropathy (PN) is one of the most common neuromuscular disorders, with recent studies indicating a prevalence of around 4% in the general population [3]. In Africa, recent studies have reported an overall prevalence of 6.9% in Benin and 3.2% in Mali [4,5]. In the clinical context of a suspected peripheral neuropathy, the history and neurological examination provide information on the general characteristics but cannot define the nature of the neuropathy; only electrodiagnostic tests allow a more detailed characterisation of a neuropathy [6]. ENMG, in addition to the clinical examination, confirms the peripheral origin, specifies the mechanism and site of the lesion, and the severity of the damage, on which the prognosis for recovery depends [7]. The aim of our study was to determine the role of electroneuromyography (ENMG) in the diagnosis of peripheral neuropathy.

Methods

This was a cross-sectional, prospective, descriptive study conducted over a period of three (3) months in the Neurology Department of the Ignace Deen National Hospital. All patients admitted for consultation or hospitalised for suspected NP in whom the ENMG was performed were included in this study. Patients admitted for consultation or hospitalised for suspected NP who did not undergo ENMG or who were intolerant of ENMG examination were excluded. Our sampling was exhaustive and our variables were socio-demographic, clinical, paraclinical and diagnostic.

All ENMGs were performed and interpreted by an experienced clinical neurophysiologist with at least one year of postgraduate training in clinical neurophysiology. ENMGs were performed on patients in a sitting or lying position in an examination bed bilaterally, symmetrically and comparatively on unclothed limbs; from distal to proximal extremity. The average duration was 30 minutes (exploration of 2 limbs) to 1 hour (exploration of 4 limbs). The examination itself comprised 2 successive key stages: stimulus-detection and detection.

The diagnosis of NPs was made on the basis of a clinical examination and the results of the ENMG (mononeuropathies, multiple mononeuropathies, polyneuropathies, polyradiculopathies, polyradiculoneuropathies, plexopathies). Data were entered and analysed using IBM SPSS statistics 21.0 software, and word processing and graphics were produced using software from the Microsoft Office 2019 suite.

Table 1: Breakdown of patients by type of disease.

Type of neuropathy	Workforce (n=97)	Proportion
Axonal damage	**83**	**85,6**
Demyelinating disease	10	10,3

Table 2: Breakdown of patients by lesion site.

Lesion site	Workforce (n=97)	Proportion
Radicular damage	41	42,3
Truncal damage	86	88,7
Plexus damage	4	4,1
Ductal syndrome	15	15,5

Table 3: Breakdown of patients according to fibres affected.

Type of lesion	Fibres	Workforce (n=97)	Proportion
Axonal Ductal syndrome	Sensitive fibres	3	3,1
	Motor fibres	24	24,7
	Fibres sensitivomotor	56	57,7
	Motor fibres	7	7,2
	Fibres sensitivomotor	8	8,2

Electro-Neuromyographic Data

Analysis of the electrical activity of the peripheral nerves and muscles in the patients highlighted (Tables 4,5,6,7).

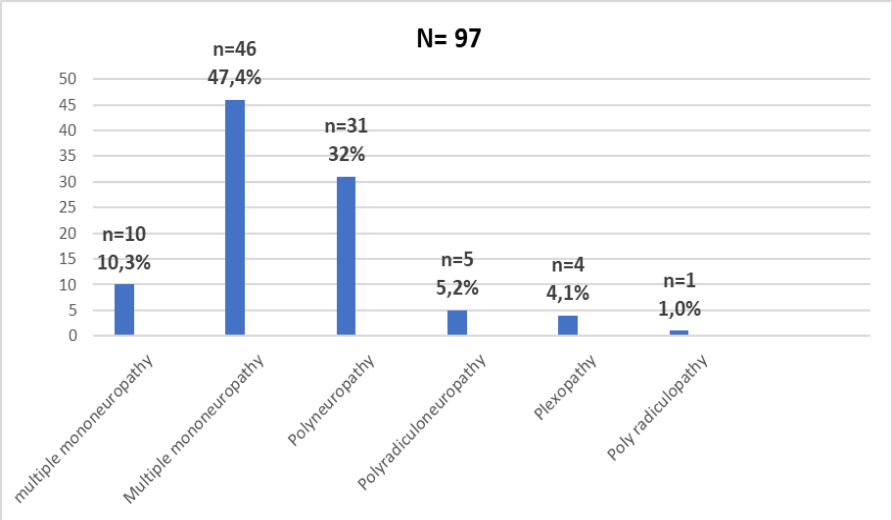

Figure 1: Breakdown of patients by ENMG diagnosis

Table 4: Study of pathological motor nerve conduction.

VCM								
Nerve	Latency (ms)	Amplitude (mV)	Area mv.ms	Durée Ms	Distance mm	Vitesse (m/s)	Rap. Surface %	Ondes F Ms
Médian Moteur Droit								
Palm - CAP | CAP	--	--	--	--				36.0
Wrist-Palm | CAP	3.54	1.91	4.7	3.9		--	--	
Elbow-Wrist | CAP	8.79	1.96	3.7	3.4	300	57.1	-21.3	
Axillary-elbow crease | CAP	11.0	1.88	5.7	6.2	115	52.0	54.1	
Médian Moteur Gauche								
Palm - CAP | CAP	--	--	--	--				41.3
Wrist-Palm | CAP	4.60	1.67	5.7	6.1		--	--	
Elbow-Wrist | CAP	11.0	1.89	6.4	6.1	295	46.1	12.3	
Axillary-elbow crease | CAP	13.5	2.5	7.7	5.7	130	52.0	20.3	
SPE Pédieux Moteur Droit								
Ankle - Pedals | Pedals	--	--	--	--				
Sous Col- Ankle | Pedals	15.7	0.31	1.08	6.2		--	--	
Sus Col-Sous Col | Pedals	19.2	0.89	2.7	4.4	85.0	24.3	150	
SPE Pédieux Moteur Gauche								
Ankle - Pedals | Pedals	--	--	--	--				
Sous Col- Ankle | Pedals	--	--	--	--		--	--	
Sus Col-Sous Col | Pedals	--	--	--	--		--	--	
SPI Moteur Droit								
Dowel - CFGO | CFGO	4.60	0.22	0.92	6.0				28.2
SPI Moteur Gauche								
Dowel - CFGO | CFGO	7.71	0.76	1.25	2.9				25.5
Ulnaire Moteur Droit								
Wrist - Add V | Add V	3.23	2.1	5.4	4.6				29.5
Under Elbow-Wrist | Add V	6.04	2.2	8.5	7.6	270	96.1	57.4	
Sus Elbow-Sous Elbow | Add V	10.4	1.31	4.2	5.2	130	29.8	-50.6	
Axillary Crevice-Over Elbow | Add V	12.0	1.58	6.0	6.3	100	62.5	42.9	
Ulnaire Moteur Gauche								
Wrist - Add V | Add V	3.54	1.80	7.3	6.1				34.7
Under Elbow-Wrist | Add V	8.71	2.5	9.5	6.0	267	51.6	30.1	
Sus Elbow-Sous Elbow | Add V	11.3	2.5	9.4	6.1	120	46.3	-1.05	
Axillary Crevice-Over Elbow | Add V	12.9	2.5	8.9	6.0	135	84.4	-5.3	

Upper limbs

- A lengthening of the distal motor latencies of the 2 medians at the wrist, predominantly on the left;
- A decrease in the amplitude of the motor evoked responses of the 2 medians and the 2 ulnaries;
- A lengthening of the central latency of the F wave of the 2 medians.

In the lower limbs

- A lengthening of the distal motor latency of the left SPI;
- Collapsed amplitude of the motor evoked responses of the 2 SPEs and the 2 SPIs;
- The central latency of the F wave was not recorded.

Table 5: Study of pathological sensory nerve conduction.

VCS					
Nerve	Latency (ms)	Vitesse (m/s)	Amplitude (µV)	Area ms.µv	Distance Mm
Median Orthodromic Sensitive Straight					
Dig I – Wrist	1.61	55.9	4.4	3.1	90.0
Dig III – Wrist	2.26	57.5	2.5	1.46	130
Wrist V – Wrist	2.03	49.3	2.7	2.1	100
Dig I - Wrist | Wrist		55.9			90.0
Dig III - Wrist | Wrist		57.5			130
Wrist V - Wrist | Wrist		49.3			100
Dig III-Dig I | Wrist		--	-43.2	-52.9	
Wrist V-Dig III | Wrist		--	8.0	43.8	
Median Orthodromic Sensitive left					
Dig I – Wrist	2.42	39.3	3.4	2.3	95.0
Dig III – Wrist	3.03	42.9	2.2	1.61	130
Wrist V – Wrist	2.87	34.8	1.56	1.07	100
Dig I - Wrist | Wrist		39.3			95.0
Dig III - Wrist | Wrist		42.9			130
Wrist V - Wrist | Wrist		34.8			100
Dig III-Dig I | Wrist		--	-35.3	-30.0	
Wrist V-Dig III | Wrist		--	-29.1	-33.5	
Musculocutaneous (MI) Sensory Right					
Leg - Kick	1.80	52.8	3.8	4.9	95.0
Musculocutaneous (MI) Sensory left					
Leg - Kick	1.50	66.7	6.0	3.8	100
Radial Sensitive Right					
Avt Bras – Wrist	1.30	53.8	12.3	7.3	70.0
Avt Bras - Wrist | Wrist		53.8			70.0
Radial Sensitive left					
Avt Bras – Wrist	1.60	53.1	7.8	7.8	85.0
Avt Bras - Wrist | Wrist		53.1			85.0
Sural (short saphenous vein) Sensory Right					
Mid-leg - Malleolus	2.07	43.5	1.35	0.76	90.0
Sural (short saphenous vein) Sensory Left					
Mid-leg - Malleolus	2.18	34.4	1.81	1.05	75.0

Demonstrating

In the upper limbs

- A decrease in the amplitude of the sensory action potentials of the 2 medians and the 2 radials;
- A slowdown in the speed of sensory conduction in the 2 medians.

In the lower limbs

- A decrease in the amplitude of the 2 sural and 2 musculocutaneous sensory action potentials;
- Sensory conduction velocities of the 2 sural muscles and the 2 musculocutaneous muscles within normal limits.

Table 6: Numerical values for the study of normal motor nerve conduction in the 4 limbs of Mrs Y.

VCM								
Nerve	Latency (ms)	Amplitude (mV)	Area mv.ms	Durée Ms	Distance mm	Vitesse (m/s)	Rap. Surface %	Ondes F ms
Median Engine Right								
Palm - CAP | CAP	1.15	8.5	17.7	4.1				
Wrist - CAP | CAP	2.59	14.5	38.0	4.9				24.9
Wrist-Palm | CAP	2.59	14.5	38.0	4.9	50.0	34.7	115	
Elbow-Wrist | CAP	7.00	9.4	25.7	5.1	270	61.2	-32.4	
Median Engine Left								
Palm - CAP | CAP	1.34	8.4	18.9	3.9				
Wrist - CAP | CAP	2.39	13.2	36.3	5.4				24.2
Wrist-Palm | CAP	2.39	13.2	36.3	5.4	60.0	57.1	92.1	
Elbow-Wrist | CAP	6.85	9.3	32.2	5.4	270	60.5	-11.3	
SPE Pedestrian Motor Right								
Ankle - Pedals | Pedals	2.30	6.0	14.0	4.0				42.3
Sous Col- Dowel | Pedals	9.48	4.6	11.0	4.4	390	54.3	-21.4	
Sus Col-Sous Col | Pedals	10.7	4.6	11.7	5.2	90.0	73.8	6.4	
SPE Pedestrian Motor Left								
Ankle - Pedals | Pedals	4.17	2.8	5.4	3.6				44.9
Sous Col- Dowel | Pedals	11.0	2.5	4.6	3.6	350	51.2	-14.8	
Sus Col-Sous Col | Pedals	12.5	2.5	6.7	5.6	95.0	63.3	45.7	
SPI Right motor								
Dowel - CFGO | CFGO	3.85	7.6	14.5	4.5				46.5
SPI Lift motor								
Dowel - CFGO | CFGO	5.69	9.6	15.4	2.9				49.4
Ulnar Right motor								
Wrist - Add V | Add V	2.68	7.5	18.5	4.4				25.7
under elbow - wrist | Add V	6.42	7.8	18.8	4.5	280	74.9	1.62	
Sus elbow- under elbow | Add V	8.42	7.3	17.5	4.5	150	75.0	-6.9	
Ulnar Lift motor								
wrist - Add V | Add V	2. 30	6.8	22.2	5.7				24. 6
under elbow - wrist | Add V	5.81	6.5	18.9	5.2	280	79.8	-14.9	
Sus elbow- under elbow | Add V	7.96	5.8	15.8	5.0	140	65.1	-16.4	

This table shows

In the upper limbs: Normal distal motor latencies, motor evoked responses and motor conduction velocities of the 2 medians and 2 radials;

In the lower limbs: Distal motor latencies, motor evoked responses and motor conduction velocities of the 2 SPEs and 2 SPIs within normal limits;

Central F wave latencies were normal in all 4 limbs.

Table 7: Numerical values for the study of normal sensory nerve conduction in the 4 limbs of Mrs Y.

VCS					
Nerve	Latency (ms)	speed (m/s)	Amplitude (µV)	area ms.µv	Distance mm
Median Orthodromic Sensitive Right					
Dig I – Wrist	1.77	62.1	28.2	12.5	110
Dig III – Wrist	1.88	74.5	26.8	11.2	140
Dig IV – Wrist	1.81	77.3	19. 7	8.4	140
Paume – Wrist	1.81	66.3	15.0	6.6	120
Dig III - Wrist | Wrist		74.5			140
Dig IV - Wrist | Wrist		77.3			140
Palm - Wrist | Wrist		66.3			120
Palm-Dig III | Wrist		--	-44.0	-41.1	
Median Orthodromic Sensitive Left					
Dig I – Wrist	1.76	62.5	18.2	8.5	110
Dig III – Wrist	1.88	74.5	21.9	8.8	140
Dig IV – Wrist	1.73	80.9	10.2	4.5	140
Paume – Wrist	1.73	66.5	15.9	6.3	115
Dig III - Wrist		74.5			140
Dig IV - Wrist | Wrist		80.9			140
Palm - Wrist | Wrist		66.5			115
Palm-Dig III | Wrist		--	-27.4	-28.4	
Musculocutaneous (MI) Sensitive Right					
Leg - Kick	1.63	61.3	22.4	12.7	100
Musculocutaneous (MI) Sensitive Left					
Leg - Kick	1.84	54.3	15.4	9.5	100
Radial Sensitive Right					
Avt Bras – Wrist	1.42	70.4	41.1	19.6	100
Avt Bras - Wrist | Wrist		70.4			100
Radial Sensitive Left					
Avt Bras – Wrist	1.06	84.9	51. 4	28.2	90.0
Avt Bras - Wrist | Wrist		84. 9			90.0
Sural (Long saphenous vein) Sensory Right					
Mid-leg - Malleolus	1.46	61.6	19.8	10.9	90.0
Sural (Long saphenous vein) Sensory Right					
Mid-leg - Malleolus	1.49	67.1	22.6	11.4	100

This table shows
In the upper limbs: Sensory action potentials and sensory conduction velocities of the 2 medians and the 2 radials were normal;
In the lower limbs: Sensory action potentials and sensory conduction velocities of the 2 sural muscles and the 2 musculocutaneous muscles were normal.

Discussion

In this study, the age group most affected was between 41 and 60 years, i.e. 43.3% with an average age of 50 ± 16 years. This proportion is similar to that of Djibril S et al. [5] but differs from the data in the literature according to which NP are more likely to occur in people over 65 years of age [8]. Indeed, the over-65 age group is not negligible in our series, i.e. 28.9% behind those aged 40 to 60.

Males predominated (60%) with a sex ratio of 1.5 in our series, whereas Djibril S et al. reported a female predominance of 56.6% [5]. No link between sex and LOC has been found in the literature.

In our series, the majority of patients (92.3%) were referred from the neurology department. This can be explained on the one hand by the fact that in the neurology department, the ENMG is routinely performed in cases of suspected LOC, and on the other hand by the fact that it remains an examination with which healthcare professionals are not very familiar.

In our study, diabetes was the most common risk factor (26.9%). A study of 212 diabetic patients found 79.4% of peripheral neuropathy on ENMG [9]. This confirms the data in the literature according to which diabetes remains the most important risk factor and the most common cause of PN. The low rate of diabetic

patients found in our series could be explained by the absence of systematic screening for peripheral neuropathy in our context. Arterial hypertension was the second most common risk factor (15.4%). In the Malian study, it was found in 31.6% of patients. The association between hypertension and NP is a risk factor that remains controversial in the literature. Some studies have found that there is no association between hypertension and the development of LOC [10]. However, Gnonlonfou et al. reported in their study that diabetes and hypertension are factors associated with peripheral neuropathy [4]. Peripheral nervous system (PNS) involvement is often suggested by sensory and motor deficits, most often distal. The most common sensory signs in our study were paresthesia and pain in 88.5% and 75.9% of patients respectively. Sy et al. in their study found that pain was the main symptom in 86.7% of the patients surveyed [6]. Paraesthesia and neuropathic pain are recognised in the literature as positive signs of peripheral neuropathy [4,9]. Motor signs are dominated by muscle cramps (65.4%), muscle weakness (50%) and fasciculations (21.2%). These results are also in line with the literature [11-13]. ENMG results were pathological in the majority of our patients (93.3%). This reflects and confirms the importance of ENMG in the diagnosis of NP. Indeed, it is well known that this examination is the reference tool for positive, topographical diagnosis. In a Greek study evaluating the diagnostic accuracy of ENMG and muscle biopsy in patients with neurogenic disorders, ENMG was found to be particularly sensitive and specific (>90%) in these patients [13]. The challenges of ENMG are manifold: to confirm the existence of NP and the clinical hypothesis of its pattern, or to propose an alternative pattern, or even the association with other neuromuscular pathologies; to approach its lesion mechanism by providing arguments for a pathology of the axon, myelin, motor neuron damage (motor neuron pathology), sensory neuron damage (ganglionopathy), to assess the severity of NP (and therefore its prognosis); and provide arguments to define its chronicity and progressive nature, all of which will affect the speed with which the diagnostic investigation, treatment and monitoring are implemented, and the prognosis of the disease [11,13]. These ENMG examinations enabled us to observe that axonal involvement was the most frequent lesion mechanism (79.8%) among these patients suffering from NP. Truncal involvement was the most common lesion site. This distinction between axonal neuropathies and demyelinating neuropathies will help to guide future investigations aimed at aetiology. The ENMG examination of these patients also made it possible to assess the type of fibres affected and the severity of the damage. Whatever the lesion mechanism, the sensitivomotor fibres were the most affected, with 31.3% showing very severe damage. This high proportion of patients with very severe damage could be explained by the fact that patients consult us at a chronic stage of the disease. The information provided by the ENMG enabled us to diagnose the different types of NP suffered by our patients. Multiple mononeuropathies and polyneuropathies are the most common diagnoses.

In the studies by Gnonlonfou et al., multiple mononeuropathies represented only 0.6% of the sample studied. This high frequency of multiple mononeuropathies (47.4%) in our series may be due to the fact that the majority of patients are referred from the neurology department, where vascular pathologies are most frequently found. Indeed, among the causes of multiple mononeuropathies, vascular pathologies are the most frequently incriminated [14]. At the end of this study, we were able to report the value of ENMG in the diagnosis of PN, as reported in previous studies [3,15]. It allowed us to confirm the suspicion of peripheral neuropathy in the patients, to specify the lesion mechanisms, the fibres affected and to assess the prognosis.

Conclusion

In our series of studies, the results of ENMG were marked by a predominance of multineuritis, which could be overestimated in our study because almost all the cases came from the neurology department of the HNID, followed by polyneuritis, mononeuritis, polyradiculoneuritis, plexopathy and polyradiculopathy. However, a study over a long period and on other aspects of electroneuromyography would allow a good exploration of the different types of peripheral neuropathies.

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