

Incidence and nature of damage to the peripheral nervous system in patients with multiple sclerosis

Dominik Siutka¹, Katarzyna Siutka², Małgorzata Fudala¹, Waldemar Broła³ 

¹Department of Neurology with Stroke Unit, Regional Hospital, Skarżysko-Kamienna, Poland

²Department of Neurology, Regional Hospital, Kielce, Poland

³Department of Neurology, *Collegium Medicum*, Jan Kochanowski University, Kielce, Poland

Medical Studies 2025; 41 (2): 89–95

DOI: <https://doi.org/10.5114/ms.2025.152528>

Key words: incidence, peripheral nervous system, multiple sclerosis, type of damage.

Abstract

Introduction: Multiple sclerosis was considered a disease of the central nervous system only, but more and more evidence show the involvement of the peripheral nervous system (PNS).

Aim of the research: The aim of this study was to estimate the prevalence of PNS damage in patients with multiple sclerosis (PwMS), compared to the general population, and to evaluate selected electrophysiological parameters in these patients.

Material and methods: Ninety-three individuals (38 men and 55 women) aged between 18 and 65 years diagnosed with MS according to the 2017 modified McDonald criteria were included in the study. The control group consisted of 30 subjects (12 men and 18 women), healthy volunteers or patients with other neurological diseases, without PNS damage. PNS parameters obtained by the nerve conduction study (NCS) were analysed, including latency, sensory and motor response amplitude, conduction velocities and minimal F-wave latency in the median, ulnar, peroneal and sural nerves. Based on the values obtained, the prevalence of peripheral nervous system damage in MS patients/PwMS and the nature of this damage were determined.

Results: Abnormalities in nerve conduction studies were demonstrated in 66.6% of patients in the study group compared to 43.3% of the control group ($p < 0.005$). The peripheral nerve damage in patients with MS/PwMS was axonal-demyelinating in nature and tended to involve proximal segments, mainly in the ulnar and peroneal nerves. The peripheral nervous system damage was statistically significantly more frequent in patients with MS/PwMS than in the control group.

Conclusions: Electrophysiological testing, in specific cases, can be useful in the algorithm of care for a patient with MS.

Introduction

For many decades, multiple sclerosis (MS) was considered a disease of the central nervous system (CNS) only, but in recent years, the co-occurrence of damage to the peripheral nervous system (PNS) has been increasingly emphasized. So far, the frequency of damage has not been determined, nor has the mechanism leading to it been defined. Based on current research using electrophysiological methods, it is estimated that PNS damage occurs in 5–74% of patients with multiple sclerosis (PwMS). This range of results shows that there is still a lot of uncertainty on this topic [1–6].

The nature of the damage, despite the most frequently described demyelinating changes, is not entirely clear because the latest scientific reports also describe axonal damage [3]. The research noted the temporal relationship between the diagnosis of polyneuropathy and multiple sclerosis in patients with a double diagnosis, as well as the co-occurrence of the chronic inflammatory demyelinating polyradiculoneuropathy (CIDP) and MS [4, 7–10].

Apart from electrophysiological methods, PNS abnormalities in MS have been investigated by nerve biopsy, magnetic resonance imaging, sudomotor reflex testing, histamine response test and vibration perception threshold (VPT), electrochemical skin conductance (ESC) and DN4 questionnaire [6, 10, 11].

The analysed scientific studies have not been shown/have not shown a coherent picture that would allow us to clearly determine the mechanism of damage. However, it is known that the disease process tends to affect proximal nerve segments, which may explain the uncertainties in the results of electrophysiological tests [12]. Most changes are demyelinating in nature in the form of prolonged terminal latencies, prolonged minimum F wave delays and segmental slowing of conduction velocity [5, 10]. Studies of larger groups indicate demyelinating damage [10], but studies indicating axonal changes have also been published [1].

Moreover, the analysis of scientific research shows that several points have also been identified that may suggest a common pathological mechanism leading to myelin damage in the CNS and PNS. These are

similarities in the structure of CNS and PNS myelin, especially in the presence of myelin basic protein (MBP), myelin proteolipid protein (PLP), 2,3'-cyclic nucleotide 3-phosphodiesterase (CNP) and myelin-associated glycoprotein (MAG) proteins [13]; the occurrence of antibodies against neurofascin in patients with demyelination in the CNS and PNS (combined central and peripheral demyelination, CCPD) [9]; the presence of specific antibodies against MBP in the serum of patients with MS [14]; increased citrullination of MBP in the CNS and PNS in PwMS [15]; loss of MAG in the CNS and PNS in PwMS [15]; loss of nodal and perinodal structures stabilizing myelin in the CNS and PNS in MS patients/PwMS [16] and increased oxidative stress in the CNS and peripheral blood in patients with MS/PwMS [17].

Aim of the research

The main aim of the study was to estimate the frequency of damage to the PNS in PwMS compared to the control population and to assess selected electrophysiological parameters of the PNS in these patients.

Material and methods

The study included 93 patients of both sexes (38 men and 55 women) aged between 18 and 65 years, diagnosed with multiple sclerosis according to the modified 2017 McDonald criteria [18]. The research was conducted from 24 January 2019 to 27 August 2021 in four centres in the Świętokrzyskie Voivodeship.

The control group consisted of 30 patients (12 men and 18 women) aged between 18 and 59 years, without clinical signs of PNS or CNS damage, who were hospitalized for other reasons (e.g. headache, epilepsy, dementia). The control group also included volunteers without diseases predisposing to damage to the PNS (Table 1).

The criteria for exclusion from the study were: lack of informed and voluntary consent to participate in the study, co-occurrence of diseases predisposing to

damage of the PNS, such as diabetes, chronic renal failure, monoclonal gammopathies, connective tissue diseases, alcoholism, malnutrition (BMI < 18.5 kg/m²), cancer diseases.

The research procedure consisted of:

- Analysis of available medical documentation (medical history regarding the onset and course of MS, duration of the disease, number of relapses, dominant clinical symptoms, duration of immunomodulatory therapy and currently used medications);
- Assessment of motor impairment based on the EDSS scale;
- Nerve conduction study (NCS) performed with the Neurosoft Neuro-EMG-Micro device covering the median, ulnar, peroneal and sural nerves (using the antidromic sensory method, motor method, with F wave assessment).

For the purposes of this study, nerve damage was defined as exceeding the norms applicable in the laboratory of at least one indicator of neurographic examination (final latency, response amplitude, segmental conduction velocities and minimum F-wave latency).

Statistical analysis

Statistical analysis was performed using MedCalc® Statistical Software version 20.014 (MedCalc Software Ltd, Ostend, Belgium) and Microsoft Excel. Statistical description techniques and the Shapiro-Wilk test of normality were applied for descriptions of the groups and the variables. The main research hypothesis was tested by comparing the average results of the two groups. The parametric Student's *t*-test was applied for quantitative, normally distributed variables. To verify the secondary hypotheses, the same method was applied for the comparison of quantitative variables (e.g., age) between the groups, and a contingency table analysis with χ^2 analysis was performed to compare the groups in terms of nominal and ordinal variables. *P*-values < 0.05 were considered statistically significant.

Results

Of the 93 PwMS included in the study, the vast majority (93.5%) consisted of relapsing-remitting form of multiple sclerosis (RRMS). The median duration of the disease was 5 years. They were characterized by different disease activity and were taking various available disease-modifying drugs (clinical characteristics of the study group are presented in Table 2).

The electrophysiological study showed that 66.6% of the patients in the study group (61 persons) had damage to at least one nerve manifested by a deviation from the norm of at least neurographic examination indicator (Figure 1). In the control group, similar damage was found in 43.3% (13 patients) (*p* < 0.05).

The electroneurographic parameters of the PNS were compared between the study group and the control group, separated into sensory and motor fibres.

Table 1. Comparison of the PwMS group and the control group

Variables	PwMS (<i>n</i> = 93)	Control group (<i>n</i> = 30)	<i>P</i> -value
Sex, <i>n</i> (%):			
Men	38 (40.9)	12 (40)	0.9338
Women	55 (59.1)	18 (60)	
Age [years]:			
Range	18–66	18–59	0.5400
Mean (SD)	41 (12.4)	39 (10.6)	
Median (Q ₁ -Q ₃)	41 (34-46)	38 (32-44)	

PwMS – patients with multiple sclerosis.

Table 2. General characteristics of the study group

Variables	Value
Disease course, <i>n</i> (%):	
RRMS	87 (93.5)
SPMS	3 (3.2)
PPMS	3 (3.2)
Disease duration [years]:	
Mean (SD)	6.99 (7.41)
Median	5.00
Range	0–43
EDSS:	
Mean (SD)	2.15 (2.1)
Median	2.0
Range	1–4.5
Number of relapses:	
Mean (SD)	2.36 (0.24)
Median	1
Range	1–10
Disease activity [number of relapses/disease duration]:	
Mean (SD)	0.556 (0.49)
Median	0.380
Range	0.02–3.00
Percentage of treatment time [total treatment time/disease years]:	
Mean (SD)	0.69 (0.35)
Median	0.710
Range	0–1

Table 3 shows a comparison of the average conduction parameters of sensory fibres of the examined nerves.

In the study group, a statistically significant decrease in the amplitude of the sensory response was observed in the median and ulnar nerves, compared to the control group, amounting to 14.3 μ V in the median nerve and 11.2 μ V in the ulnar nerve. This phenomenon was not observed in the sural nerve. No statistical significance was observed in terms of parameters that may indicate demyelination, i.e. an increase in distal latency and a decrease in conduction velocity in the above nerves.

The conduction parameters in motor fibres in the median nerve in MS patients did not differ from the control group. However, in the ulnar nerve, these differences were significant and were manifested by

Variables	Value
Current DMT, <i>n</i> (%):	
Interferon β	33 (35.5)
Dimethyl fumarate	28 (30.1)
Teriflunomide	8 (8.6)
Glatiramer acetate	4 (4.3)
Fingolimod	3 (3.2)
Ocrelizumab	2 (2.2)
Siponimod	1 (1.1)
Without DMT	14 (15.1)
Changing DMT during disease, <i>n</i> (%):	
Only one drug	77 (82.8)
More than one drug	16 (17.2)
Clinical symptoms, <i>n</i> (%):	
Sensory	56 (60.2)
Motor	55 (59.1)
Cerebellar	21 (22.6)
Mental	18 (19.4)
Visual	12 (12.9)
Autonomic	10 (10.8)
Brainstem	4 (4.3)
Other	22 (23.9)

DMT – disease modifying therapy, PPMS – primary progressive multiple sclerosis, RRMS – relapsing remitting multiple sclerosis, SPMS – secondary progressive multiple sclerosis, SD – standard deviation.

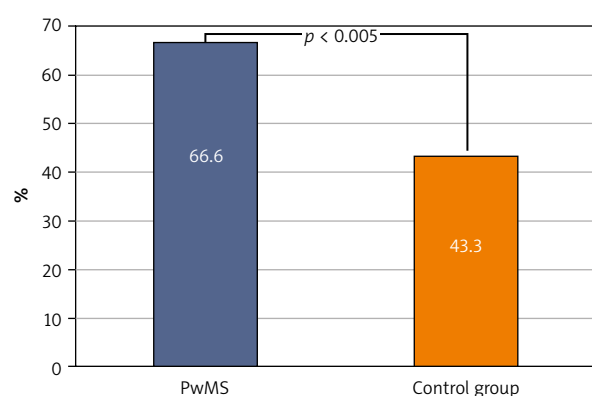


Figure 1. Frequency of damage to at least 1 peripheral nerve in the patients with multiple sclerosis (PwMS) and control groups

a statistically significant decrease in the response amplitude and an increase in the minimum F-wave latency (Table 4).

Table 3. Comparison of abnormalities in peripheral sensory fibres between the persons with multiple sclerosis (PwMS) and control groups

Nerve	Parameter	PwMS			Control group			P-value
		Mean (SD)	Range	Median (Q1–Q2)	Mean (SD)	Range	Median (Q1–Q2)	
Median	Latency [ms]	2.4 (0.4)	1.8–3.8	2.3 (2.2–2.6)	2.4 (0.3)	2.1–3.3	2.3 (2.2–2.5)	0.4453
	Amplitude [mV]	40.3 (19.4)	6.5–86.3	39.9 (28.5–52.6)	54.8 (27.1)	12.6–124.0	47.5 (35.1–74.5)	0.0016
	Velocity [m/s]	59.4 (7.6)	36.8–77.8	60.9 (54.9–63.6)	60.0 (7.2)	42.0–68.3	62.2 (57.1–65.1)	0.4998
Ulnar	Latency [ms]	2.4 (0.3)	1.7–3.2	2.3 (2.2–2.5)	2.3 (0.2)	1.9–2.7	2.2 (2.1–2.4)	0.0580
	Amplitude [mV]	29.1 (15.8)	5.1–71.6	26.9 (15.4–40.1)	40.3 (20.3)	7.2–93.2	35.9 (25.3–52.2)	0.0052
	Velocity [m/s]	59.5 (6.2)	43.4–82.7	59.6 (56.1–63.0)	62.0 (5.5)	52.8–73.7	62.2 (57.1–66.7)	0.0561
Sural	Latency [ms]	2.9 (0.4)	0.0–3.6	2.9 (2.7–3.1)	2.8 (0.3)	2.4–3.3	2.8 (2.6–3.1)	0.3206
	Amplitude [mV]	14.9 (9.1)	0.0–41.9	12.9 (7.6–20.0)	15.7 (7.0)	5.4–30.2	13.6 (11.9–20.4)	0.2798
	Velocity [m/s]	48.5 (7.2)	0.0–66.1	49.0 (45.5–52.8)	50.3 (5.1)	42.4–58.8	49.0 (45.5–52.8)	0.2373

SD – standard deviation; statistically significant differences are shown in bold.

Table 4. Comparison of test results in peripheral motor fibres between the patients with multiple sclerosis (PwMS) and control groups

Nerve	Parameter	PwMS			Control group			P-value
		Mean (SD)	Range	Median (Q1–Q2)	Mean (SD)	Range	Median (Q1–Q2)	
Median	Latency [ms]	2.9 (0.5)	2.1–4.9	2.9 (2.6–3.1)	2.9 (0.4)	2.2–3.8	2.8 (2.7–3.1)	0.7613
	Amplitude [mV]	10.0 (2.9)	3.0–19.1	9.7 (8.5–11.9)	10.6 (2.4)	6.1–16.4	10.3 (9.8–12.0)	0.3120
	V1 [m/s]	59.7 (3.5)	53.2–68.4	59.6 (57.1–62.3)	60.5 (5.0)	51.1–74.0	59.6 (57.1–62.3)	0.3349
	V2 [m/s]	60.0 (7.4)	40.0–82.4	59.3 (55.2–65.0)	62.6 (8.2)	50.0–87.5	61.4 (57.9–66.7)	0.1175
	F wave latency [ms]	26.2 (2.2)	20.3–32.2	25.8 (24.6–27.6)	25.5 (2.1)	20.6–30.3	25.1 (24.2–26.4)	0.9363
Ulnar	Latency [ms]	2.2 (0.3)	1.7–3.4	2.2 (2.1–2.4)	2.2 (0.3)	1.8–3.1	2.1 (2.1–2.2)	0.1003
	Amplitude [mV]	9.1 (2.4)	4.0–15.1	9.2 (7.2–10.6)	10.3 (1.9)	6.4–13.9	10.5 (8.8–11.5)	0.0195
	V1 [m/s]	60.8 (5.1)	43.1–75.6	60.0 (58.1–63.4)	62.1 (4.8)	51.1–72.0	61.6 (59.0–65.5)	0.1465
	V2 [m/s]	60.4 (7.8)	38.1–78.6	61.5 (55.2–66.7)	61.1 (7.6)	45.9–75.9	59.4 (56.2–67.6)	0.6815
	V3 [m/s]	66.2 (9.4)	41.3–90.9	65.5 (59.4–71.5)	66.0 (8.4)	52.2–88.9	65.5 (59.4–71.6)	0.9256
Peroneal	F wave latency [ms]	26.7 (2.3)	19.9–32.1	26.5 (25.1–28.1)	25.6 (2.2)	21.6–30.7	25.3 (24.2–26.5)	0.0283
	Latency [ms]	4.5 (0.8)	3.3–9.2	46.8 (43.7–50.4)	4.1 (0.6)	2.8–5.7	4.0 (3.8–4.3)	0.0016
	Amplitude [mV]	5.9 (2.3)	0.4–11.1	6.3 (4.3–7.4)	6.7 (2.3)	4.1–12.2	6.0 (5.2–7.6)	0.2945
	V1 [m/s]	48.1 (4.6)	29.3–56.2	49.2 (45.8–50.6)	50.8 (4.1)	41.8–58.0	51.1 (47.1–53.1)	0.0052
	V2 [m/s]	51.9 (7.9)	30.7–70.4	51.7 (46.9–57.1)	53.3 (6.4)	41.7–65.5	54.4 (47.1–57.9)	0.3915
Peroneal	F wave latency [ms]	47.4 (4.7)	32.9–58.2	47.1 (44.1–50.8)	45.1 (14.2)	37.7–52.7	44.8 (42.3–47.1)	0.0191

V – velocity; statistically significant differences are shown in bold.

The largest differences were found in the motor fibres of the peroneal nerve. A statistically significant increase in the latency of the final response from the extensor digitorum brevis muscle, the minimum F-wave latency and a decrease in conduction velocity in the lower leg (V1) was found. These changes were demyelinating damage (Figures 2 and 3).

Discussion

Our study showed that 66.6% of PwMS (63 patients) had damage to at least one nerve. In the control group, this damage occurred in 43.3% (13 patients). This is a lower frequency than in Pogorzelski *et al.*'s study, which found PNS damage in 74.2% of PwMS [2],

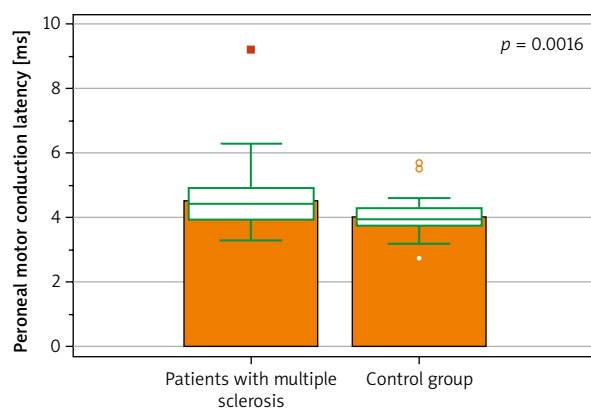


Figure 2. Motor conduction latency of the peroneal nerve

but slightly higher than in other such studies [1–5, 7]. In the Sarova-Pinhas *et al.*'s study [1], PNS damage was observed in 45.5% of PwMS, while Yin *et al.* estimates this percentage at 53.5% [3]. Clearly lower rates of PNS involvement in patients with MS are reported by Gartzon *et al.* (29.6%) [5] and Misawa *et al.* (10%) [7]. These differences result from different research methodologies and indicate the lack of uniform and universally accepted electrophysiological criteria for nerve damage.

Our study focused on comparing the peripheral nerve conduction parameters of the PwMS group and the control group. Sensory fibres showed axonal damage in the median and ulnar nerves (decrease in response amplitude). This phenomenon was not observed in the sural nerve. No significant demyelinating changes were observed, but as subsequent analyses showed, minor changes occurred in the group of men. This confirms the results of Pogorzelski *et al.*'s study [2] and studies by other authors, which also demonstrated axonal damage [1]. However, they contrast with the 2020 Turkish study, which did not show such damage [12].

No significant differences were found in the motor fibres of the median nerve. The ulnar nerve showed mixed changes (statistically significant decrease in response amplitude, prolongation of minimal F-wave latency). As in most previous studies, the most significant differences were observed in the peroneal nerve. There was a prolongation of the latency of the final motor response and the minimal F-wave latency, as well as a slowing of the conduction velocity on the lower extremity. These parameters indicate demyelinating damage. No axonal damage was demonstrated. Due to the lack of anatomical isthmus in the section where the release occurred and unchanged conduction at the level of the head of the fibula, these changes are not secondary to compression. The mechanical causes of PNS damage can also be excluded due to the fact that the vast majority of respondents/patients did not require or use orthopaedic aids (the average EDSS was 2.15 points).

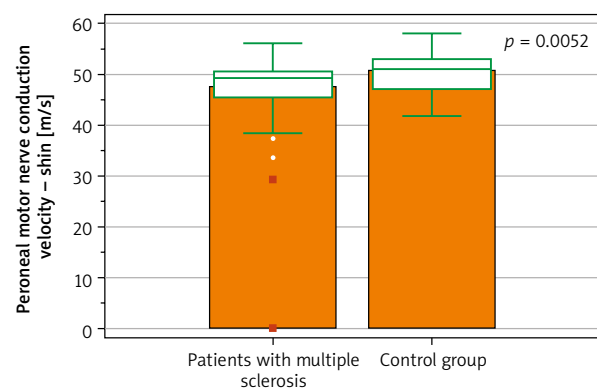


Figure 3. Decrease of conduction velocity in the motor fibres of the peroneal nerve

Similar results were presented by Misawa *et al.*, where the most commonly affected nerves in patients with MS/PwMS were the ulnar, peroneal and sural nerve [7]. However, in Pogorzelski *et al.*'s study, changes in multiple sclerosis had a predilection for the ulnar and peroneal nerve [2].

Analysing the results of our study, it can be concluded that the damage to the peripheral fibres was of a mixed axonal-demyelinating type and occurred mainly in the ulnar and peroneal nerve. Similar conclusions were represented in previous Polish studies [2, 6].

A significant prolongation of the minimum latency of the F wave in the ulnar and peroneal nerves is worth noting, which may indirectly indicate a greater involvement of the proximal sections of these nerves. This observation confirms the result of the study by Jende *et al.* using magnetic resonance imaging (MRI) [11].

Reports of the co-occurrence of CIDP and MS are very interesting [4, 7–10]. However, in our study, no case meeting the CIDP criteria was diagnosed. One patient was referred for further diagnostics at the home centre due to severe sensory syndrome and weak signs of demyelinating damage to the examined nerves, which did not meet the criteria for the diagnosis of CIDP.

Our study had several important limitations. The study group was not very large because data collection took place during the peak of the COVID-19 epidemic/pandemic. The homogeneity of the study group would probably be increased by excluding patients with PPMS and SPMS and focusing on the assessment of patients with RRMS only. However, patients with progressive forms constituted a small percentage and were retained in the study. There was a noticeable difference in the mean age between the study and control groups, but it was not statistically significant. However, the observed differences in amplitude and latencies cannot be explained by age-related changes, because in sensory fibres the final latency decreases on average by only 0.02 ms/cm and the amplitude by 2.3 μ V for every 5 years of age [19, 20].

It should be noted that in the majority of PwMS, the abnormalities revealed by neurographic examination were not symptomatic. The current diagnostic criteria focus only on indicators of the central damage, with particular emphasis on the use of imaging studies. We need to be aware that symptoms such as paraesthesia and disturbances of superficial sensation may also be caused by damage to peripheral sensory nerves, and pathological fatigue and heat intolerance – by damage to autonomic fibres. It has also been suggested that symptoms of autonomic nervous system damage may be a predictor of disability associated with disease progression [15]. Additionally, the more frequent occurrence of inflammatory polyneuropathy in PwMS may indicate the need to change the diagnostic and therapeutic approach. In special cases, the electrophysiological testing may be useful in the algorithm of care for a patient with MS.

Conclusions

The common belief that MS is a disease of the CNS only is not entirely true. Conduction abnormalities in the PNS in MS are more common than commonly believed and affect more than 66% of PwMS (compared to 43.3% of the control group). The PNS damage is axonal-demyelinating in nature and tends to affect the proximal segments, mainly in the ulnar and peroneal nerve.

Funding

No external funding.

Ethical approval

The consent to conduct the study was obtained from the Bioethics Committee in Kielce at the Świętokrzyska Medical Chamber (Resolution of the Bioethics Committee No. 10/2019 - VII).

Conflict of interest

The authors declare no conflict of interest.

References

1. Sarova-Pinhas I, Achiron A, Gilad R, Lampl Y. Peripheral neuropathy in multiple sclerosis: a clinical and electrophysiological study. *Acta Neurol Scand.* 1995 Apr; 91(4): 234-238.
2. Pogorzelski R, Baniukiewicz E, Drozdowski W. Subkliniczne uszkodzenie obwodowego układu nerwowego u chorych na stwardnienie rozsiane [Subclinical lesions of peripheral nervous system in multiple sclerosis patients]. *Neurol Neurochir Pol.* 2004 Jul-Aug; 38(4): 257-264.
3. Yin H, Nair KP, Rao DG, Hariharan S, Spencer A, Baster K. Upper limb entrapment neuropathies in multiple sclerosis. *Mult Scler J Exp Transl Clin.* 2020 Jun; 6(2): 2055217320930774.
4. Warabi Y, Yamazaki M, Shimizu T, Nagao M. Abnormal nerve conduction study findings indicating the existence of peripheral neuropathy in multiple sclerosis and neuro-myelitis optica. *Biomed Res Int.* 2013; 2013: 847670.
5. Gartzon K, Katzarava Z, Diener HC, Putzki N. Peripheral nervous system involvement in multiple sclerosis. *Eur J Neurol.* 2011 May; 18(5): 789-91.
6. Koszewicz M, Martynów R. Electrophysiological analysis of changes in peripheral nervous system in multiple sclerosis. *Neurol Neurochir Pol.* 1999 Jan-Feb; 33(1):53-62.
7. Misawa S, Kuwabara S, Mori M, Hayakawa S, Sawai S, Hattori T. Peripheral nerve demyelination in multiple sclerosis. *Clin Neurophysiol.* 2008 Aug; 119(8): 1829-1833.
8. Falcone M, Scalise A, Minisci C, Romito D, Cancelli I, Gigli GL. Spreading of autoimmunity from central to peripheral myelin: two cases of clinical association between multiple sclerosis and chronic inflammatory demyelinating polyneuropathy. *Neurol Sci.* 2006 Apr; 27(1): 58-62.
9. Kawamura N, Yamasaki R, Yonekawa T, Matsushita T, Kusunoki S, Nagayama S, Fukuda Y, Ogata H, Matsu-se D, Murai H, Kira J. Anti-neurofascin antibody in patients with combined central and peripheral demyelination. *Neurology.* 2013 Aug; 81(8): 714-722.
10. Suanprasert N, Taylor BV, Klein CJ, Roforth MM, Karam C, Keegan BM, Dyck PJB. Polyneuropathies and chronic inflammatory demyelinating polyradiculoneuropathy in multiple sclerosis. *Mult Scler Relat Disord.* 2019 May; 30: 284-290.
11. Jende JME, Hauck GH, Diem R, Weiler M, Heiland S, Wildemann B, Korporel-Kuhnke M, Wick W, Hayes JM, Pfaff J, Pham M, Bendszus M, Kollmer J. Peripheral nerve involvement in multiple sclerosis: demonstration by magnetic resonance neurography. *Ann Neurol.* 2017 Nov; 82(5): 676-685.
12. Görgülü Ü, Ergün U, Ertuğrul L. Peripheral nerve conduction in relapsing remitting multiple sclerosis (RRMS) patients. *J Clin Neurosci.* 2020 Apr; 74: 93-97.
13. Oudejans E, Luchicchi A, Strijbis EMM, Geurts JGG, van Dam AM. Is MS affecting the CNS only? Lessons from clinic to myelin pathophysiology. *Neurol Neuroimmunol Neuroinflamm.* 2020 Nov; 8(1): e914.
14. Belogurov AA Jr, Kurkova IN, Friboulet A, Thomas D, Misikov VK, Zakharova MY, Suchkov SV, Kotov SV, Alehin AI, Avalle B, Souslova EA, Morse 3rd HC, Gabibov AG, Ponomarenko NA. Recognition and degradation of myelin basic protein peptides by serum autoantibodies: novel biomarker for multiple sclerosis. *J Immunol.* 2008 Jan; 180(2): 1258-67.
15. Pan B, Fromholt SE, Hess EJ, Crawford TO, Griffin JW, Sheikh KA, Schnaar RL. Myelin-associated glycoprotein and complementary axonal ligands, gangliosides, mediate axon stability in the CNS and PNS: neuropathology and behavioral deficits in single- and double-null mice. *Exp Neurol.* 2005 Sep; 195(1): 208-17.
16. Koch M, Mostert J, Arutjunyan A, Stepanov M, Teelken A, Heersema D, De Keyser J. Peripheral blood leukocyte NO production and oxidative stress in multiple sclerosis. *Mult Scler.* 2008 Mar; 14(2): 159-65.
17. Ohl K, Tenbrock K, Kipp M. Oxidative stress in multiple sclerosis: central and peripheral mode of action. *Exp Neurol.* 2016 Mar; 277: 58-67.
18. Thompson AJ, Banwell BL, Barkhof F, Carroll WM, Coetzee T, Comi G, Correale J, Fazekas F, Filippi M, Freedman MS, Fujihara K, Galetta SL, Hartung HP, Kappos L, Lublin FD, Marrie RA, Miller AE, Miller DH, Montal-

- ban X, Mowry EM, Soelberg Sorensen P, Tintoré M, Tra-
boulsee AL, Trojano M, Uitdehaag BMJ, Vukusic S, Wau-
bant E, Weinshenker BG, Reingold SC, Cohen JA. Diagno-
sis of multiple sclerosis: 2017 revisions of the McDonald
criteria. *Lancet Neurol.* 2018; 17(2): 162-173.
19. Tong HC, Werner RA, Franzblau A. Effect of aging on sen-
sory nerve conduction study parameters. *Muscle Nerve.*
2004 May; 29(5): 716-20.
20. Siutka D, Bonek K, Fudala M, Brola W. Diagnosis of com-
pression syndromes in neurological practice.

Address for correspondence**Waldemar Brola**

Department of Neurology
Collegium Medicum
Jan Kochanowski University
Kielce, Poland
E-mail: wbrola@wp.pl

Received: 28.02.2024

Accepted: 28.04.2025

Online publication: 30.06.2025