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Improving Diagnostic Accuracy for Peripheral Neuropathy: Use of Tibial Nerve Somatosensory Evoked Potentials

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Abstract

Background: We investigated whether combining sural nerve sensory conduction study (s-SCS) and tibial nerve SEPs (t-SEPs) improves diagnostic accuracy for peripheral sensory neuropathy. **Methods**: We retrospectively reviewed 74 consecutive cases (114 lower limbs) of patients suspected of having neuropathy or radiculoneuropathy who underwent s-SCS and t-SEPs between July 2021 and December 2024. Abnormal s-SCS was defined as an amplitude <3.8 μV or a conduction velocity <39.3 m/s. Abnormal t-SEPs were defined as the failure to evoke N20, N20 latency >24.37 ms, the failure to evoke P37, or P37 latency >44.35 ms. **Results**: No cases showed s-SCS abnormalities with normal t-SEPs. Group 1 (G1) had normal s-SCS and normal t-SEPs, which were observed in 31 limbs (27.2%). Group 2 (G2) had normal s-SCSs and abnormal t-SEPs, which were found in 45 limbs (39.5%). Subgroups of G2 included normal N20 with abnormal P37, abnormal N20 with normal P37 and N20/P37 abnormalities. Group 3 (G3) had abnormal s-SCSs with abnormal t-SEPs, which was seen in 38 limbs (33.3%). **Conclusions**: Electrophysiological testing reveals normal distal and proximal sensory nerves in G1, suggesting preserved sensory nerve function. The distal sensory nerves are normal in G2. However, abnormal N20/P37 and abnormal N20 with nP37 indicate proximal sensory nerve involvement. Normal N20 with abnormal P37 indicates posterior column dysfunction. In G3, both the distal and proximal sensory nerve segments are abnormal. Therefore, adding t-SEPs to s-SCSs allows us to evaluate the full length of the peripheral nerves, which is useful for diagnosis and assessing treatment efficacy.

Keywords: peripheral neuropathy; sural nerve; sensory conduction study; tibial nerve; somatosensory evoked potentials

1. Introduction

Traditionally, the diagnosis of peripheral nerve disorders has depended on medical history and clinical examination [1–3]. Electrodiagnostic studies provide a quantitative assessment of disease severity and elaborate on the pathophysiology. They may also confirm the location of the nerve injury [4–6]. Peripheral neuropathy is best categorized by the location of the nerve injury. One of the most common localizations is a diffuse, length-dependent process called distal symmetric polyneuropathy (DSP). Another common site of nerve injury is mononeuropathy, as in the case of carpal tunnel

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syndrome. Rare locations of peripheral neuropathy include diffuse, non-length-dependent neuropathies and multiple mononeuropathies, such as chronic inflammatory demyelinating polyradiculoneuropathy (CIDP), vasculitic neuropathy, and diabetic neuropathy [2,3]. A main purpose of electrodiagnostic studies in polyneuropathy is differentiating between axonal degeneration and demyelination, as clinical information alone cannot distinguish between these two processes [7]. Demyelination is generally indicated by decreased nerve conduction velocity (NCV), conduction block, or increased temporal dispersion. Axonal loss is indicated by reduced amplitude or area of the sensory nerve action potential (SNAP) or compound muscle action potential (CMAP). However, differentiating between demyelinating and axonal pathophysiologies using nerve conduction studies can sometimes be difficult [7].

CIDP is the most common form of autoimmune polyneuropathy. CIDP is characterized by the selective involvement of peripheral nerves, plexuses, or nerve roots [5,6]. In our neurology department, we routinely perform tibial nerve somatosensory evoked potentials (t-SEPs) testing on patients with polyneuropathy (DSP, CIDP, etc.) and/or radiculoneuropathy to evaluate proximal sensory nerve function, the spinal entry zone, and the posterior column. We coincidentally noticed dissociation between the results of the sensory conduction study (SCS) of the sural nerve (s-SCS) and the t-SEP results: some patients showed normal s-SCS with abnormal t-SEPs. We assumed that this was probably because s-SCS cannot evaluate proximal sensory nerve damage, whereas t-SEPs are useful for assessing the function of the proximal sensory nerve and the spinal entry zone [8,9]. Although expert recommendations for the clinical use of SEPs did not specifically address diffuse peripheral neuropathies [10], the value of SEPs in polyneuropathy has been demonstrated by several studies published in the literature [11–19], especially in CIDP [12–18]. However, dissociated results between s-SCS and t-SEPs have not received much attention in the context of peripheral neuropathies. Therefore, we investigated whether combining s-SCS and t-SEPs improves diagnostic accuracy for peripheral sensory neuropathy using a retrospective cohort design.

2. Materials and Methods

2.1. Study Design and Setting

We performed a retrospective observational study at Fukuoka Central Hospital, a 140-bed hospital with a neurological center located in southern Japan. The hospital performs approximately 250 motor and sensory nerve conduction studies and 250 evoked potential studies per year. We performed both s-SCS and t-SEPs to evaluate various neurological disorders, such as CIDP and multiple sclerosis. Our institutional ethics committee approved this study, waiving the requirement for individual patient consent due to its retrospective nature.

2.2. Patient Population

We conducted a retrospective review of 74 consecutive cases (24 females) of suspected peripheral neuropathy or neuro-myelopathy. Their ages ranged from 27 to 91 years old. Table 1 shows a spectrum of peripheral nerve diseases with various etiologies. The most common causes are immune-mediated conditions (CIDP and Guillain-Barré syndrome (GBS)), as well as collagen vascular disorders associated with vasculitic neuropathy and radiculoneuropathy. These patients underwent s-SCS and t-SEPs at our institution between July 2021 and December 2024.

Table 1. Etiologies of the patients.

Immune-mediated neuropathy	**n = 16**
Vasculitic neuropathy	**n = 15**
Radiculoneuropathy	**n = 12**
Vitamin deficiency neuropathy	**n = 4**
Viral infectious neuropathy	**n = 4**
Diabetic neuropathy	**n = 4**

 doi:10.20944/preprints202603.2358.v1

Toxic neuropathy	**n = 3**
Inherited neuropathy	**n = 1**
Unclassified	**n = 15**

2.3. Sural Nerve Conduction Study

A board-certified clinical neurophysiologist (SI) from the Japanese Society of Clinical Neurophysiology carried out s-SCS with the assistance of a laboratory technician (MO) and two other doctors (AS and YN). s-SCS was performed using a Neuropack X1 electromyograph system (Nihon Koden, Japan) with standard techniques. The skin temperature over the lateral malleolus was kept above 31 °C. The sural nerves were stimulated antidromically using a surface stimulator. The surface recording electrode was placed posterior to the lateral malleolus, and the reference electrode was placed 3 cm distally. The stimulation site was the posterior aspect of the calf, with the cathode placed 14 cm proximal to the recording electrode. A ground electrode was placed between the recording and stimulating electrodes. The stimulus intensity was gradually increased until a supramaximal sensory response was obtained. Standard filter settings were used to record SNAPs, and responses were averaged at least ten times, or more if necessary, until a stable waveform was obtained. Latencies were calculated from stimulus onset to the initial negative deflection for biphasic SNAPs, or to the first positive peak for triphasic SNAPs, to determine CV. Amplitudes were measured from the lowest positive peak to the negative peak. According to a previous publication, SNAP amplitudes of less than 3.8 μV and conduction velocities of less than 39.3 m/s were considered abnormal [20].

2.4. Somatosensory Evoked Potential Recording

A board-certified clinical neurophysiologist (ST) from the Japanese Society of Clinical Neurophysiology performed t-SEPs with the help of MO and three other doctors (AS, AY and KH). SEPs were obtained by stimulating the posterior tibial nerve at the ankle at a frequency of 2 Hz [13,21,22]. The recording electrodes were placed over the 12th thoracic vertebra and Cz, which was 2 cm posterior to Cz according to the international 10-20 system. An electrode placed over the contralateral iliac crest and Fz was used as the reference. A Neuropack X1 amplifier (Nihon Kohden, Japan) with a bandpass of 5–2000 Hz was used, and 350 responses were averaged. At least two trials were superimposed to establish reproducibility. The peak latencies of the responses were measured: N20 (Th12) and P37 (sensory cortex) for tibial nerve SEPs). Central sensory conduction time (CSCT) was calculated as P37–N20. The normal mean values were 20.02 ms for N20, 36.91 ms for P37, and 16.88 ms for N20–P37 (CSCT), respectively [13,21,22]. Latencies exceeding the mean + 3 SD from the established normal values for SEPs were considered abnormal.

2.5. Criteria for Abnormal SCSs and SEPs

Abnormal s-SCS was defined as an amplitude of less than 3.8 μV or a conduction velocity of less than 39.3 m/s [20]. An abnormal t-SEP was defined as a failure to evoke an N20 response, an N20 latency greater than 24.37 ms, a failure to evoke a P37 response, a P37 latency greater than 44.35 ms, or a CSCT greater than 21.83 ms [12,20].

2.6. Clinical Data Collection

Clinical information was extracted from electronic medical records. Board-certified neurologists from the Japanese Society of Neurology (AY, AS, YY, KH, YI, YN, KY and JK) made diagnoses according to the Society's established diagnostic criteria [1–4].

3. Results

Although we tested t-SEPs bilaterally in all 74 subjects, s-SCSs were not always examined bilaterally. Thus, the total number of s-SCS examined was 114 limbs. Figure 1 summarizes the results of this study. Since 80 limbs showed abnormal N20 responses, CSCT was not included as an index of

 doi:10.20944/preprints202603.2358.v1

t-SEP abnormality. As expected, no cases showed s-SCS abnormalities with normal t-SEPs (Figure 1). From Figure 1, three groups were found based on the results of s-SCS and t-SEPs. Group 1 had normal s-SCSs and normal t-SEPs, which was observed in 31 limbs (27.2%) (Figure 2A). Group 2 had normal s-SCS with abnormal t-SEPs, which was observed in 45 limbs (39.5%). Within Group 2, there were three subgroups: normal N20 with abnormal P37 (Group 2a, 2.6%; Figure 2B); abnormal N20 with normal P37 (Group 2b, 13.2%; Figure 3); and abnormal N20 with abnormal P37 (Group 2c, 23.7%; Figure 3). Group 3 had abnormal s-SCS with abnormal t-SEPs. This combination was observed in 38 limbs (33.3%; see Figure 4): five limbs showed normal P37 (Group 3a) while 33 limbs had abnormal P37 (Group 3b). Abnormal N20 with normal P37 was found in 20 limbs (17.5%) in combination with Groups 2b and 3a.

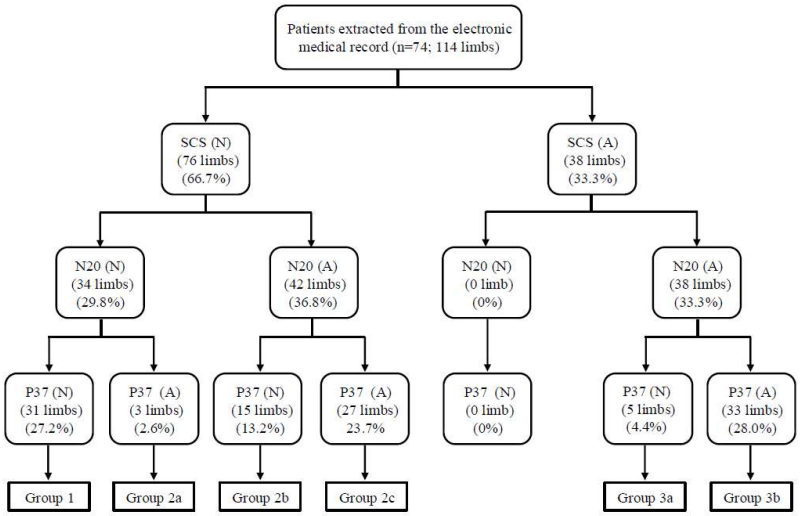

Figure 1. Flowchart of patients. Patients were categorized into three groups based on the results of s-SCS and t-SEPs (N20/P37). Abbreviations: s-SCS, sural nerve sensory conduction study; t-SEPs, tibial nerve SEPs; N, Normal; A, Abnormal.

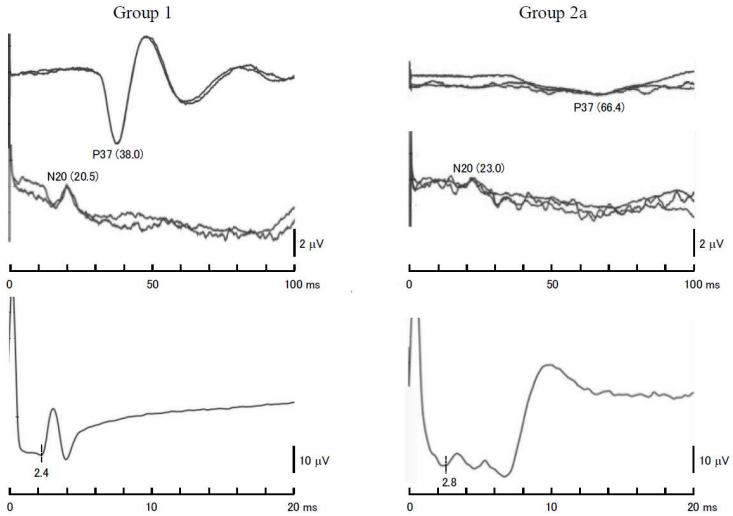

Figure 2. Illustrated cases of Groups 1 and 2a. A 50-year-old female patient with small fiber sensory neuropathy showed normal s-SCS and t-SEPs (Group 1). A 39-year-old male patient with CIDP had normal s-SCS and N20 but his P37 was prolonged and temporally dispersed (Group 2a).

 doi:10.20944/preprints202603.2358.v1

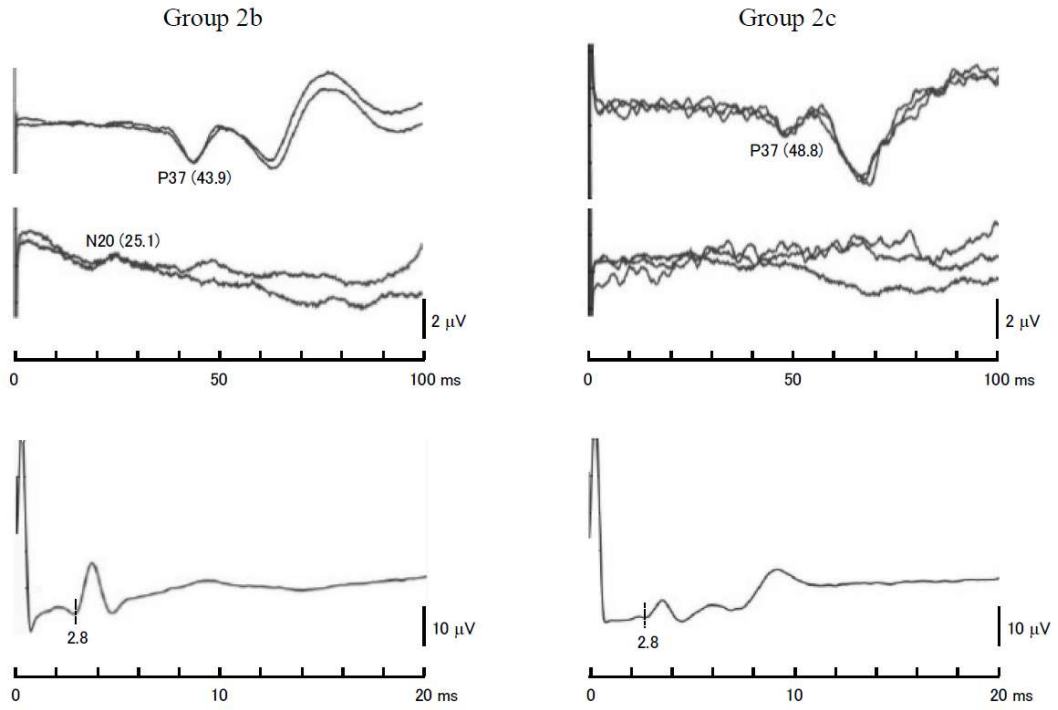

Figure 3. Illustrated cases of Groups 2b and 2c. A 66-year-old male patient with diabetic polyneuropathy had normal s-SCS and prolonged N20 with normal P37 (Group 2b). A 77-year-old male patient with vitamin deficiency polyneuropathy had normal s-SCS, as well as prolonged N20 and P37 (Group 2c).

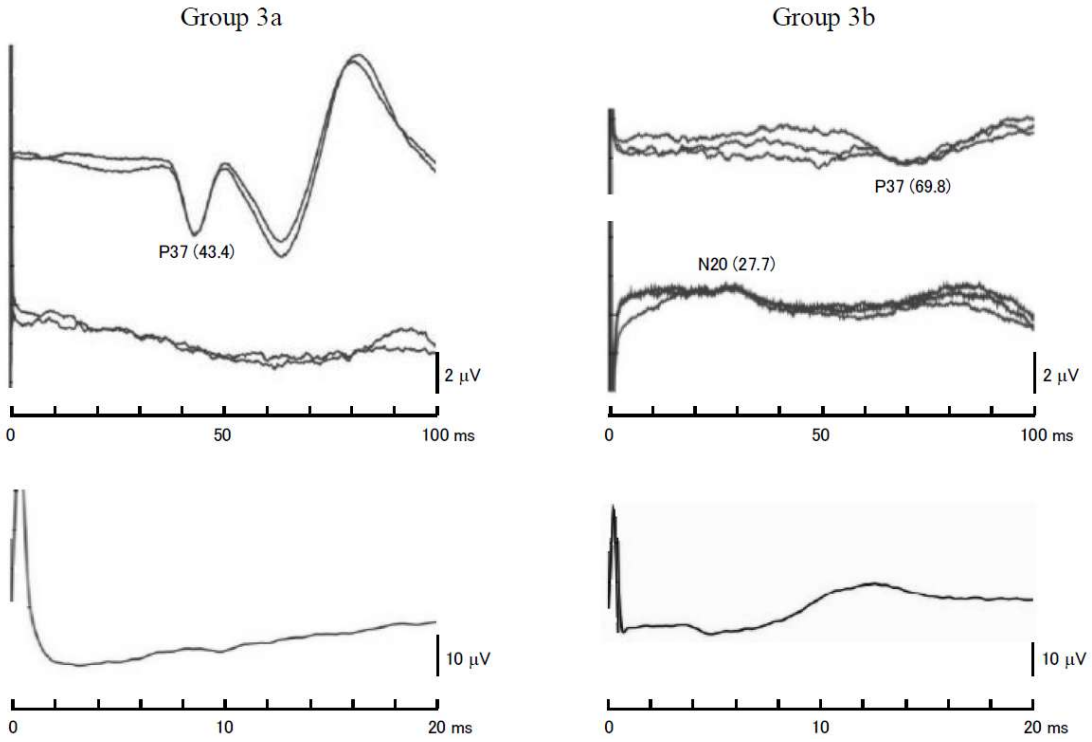

Figure 4. Illustrated cases of Groups 3a and 3b. A 75-year-old female patient with vasculitic neuropathy showed absent SNAP and prolonged N20 with normal P37 (Group 3a). A 65-year-old male patient with alcoholic neuropathy had absent SNAP and prolonged N20 and P37 responses (Group 3b).

 doi:10.20944/preprints202603.2358.v1

4. Discussion

The diagnostic utility of t-SEPs in demyelinating diseases, such as multiple sclerosis, is well-established. In particular, prolonged CSCT indicates abnormal t-SEPs [21,23]. This study examined the usefulness of t-SEPs for diagnosing and classifying peripheral neuropathies and neuromyelopathies. However, CSCT was not included as an index of abnormality because 80 limbs showed abnormal N20 responses. The utility of t-SEPs was best demonstrated in patients classified as Group 2 (normal s-SCS with abnormal t-SEPs; 39.5%), for whom the presence of proximal sensory nerve damage could not be confirmed by conventional nerve conduction studies. Group 2 appears to be divided into three subtypes: normal N20 with abnormal P37 (Figure 2); abnormal N20 with normal P37 (Figure 3); and abnormal N20 and P37 (Figure 3). Group 2a indicates spinal entry zone or dorsal column dysfunction, while Groups 2b and 2c suggest proximal sensory nerve damage. However, it is unclear why t-SEPs can be recorded over the scalp when peripheral responses are abnormal in Groups 2b and 3a (17.5%). Presumably, there is some reorganization and amplification of impulse traffic within the central nervous system [8,9,24]. Peripheral SNAPs may not be recorded when peripheral impulse traffic is dispersed; however, there may be central synchronization at different synaptic levels to amplify the volleys. In accordance with the "cortical amplification" theory, we found that t-SEPs could be obtained even when the N20 is abnormal. Overall, our findings suggest that t-SEP studies may be an important ancillary neurophysiological tool for classifying sensory neuropathies and diagnosing non-length-dependent polyneuropathies, such as CIDP, alongside conventional NCS.

We found that of the 76 limbs with normal s-SCS and normal t-SEPs were observed in 31 limbs (27.2%, Group 1). Upon reviewing the medical records, we discovered that the motor conduction velocity of the tibial nerve was abnormal in five limbs, indicating the presence of motor-dominant neuropathy. All four cases of GBS were included in this group. The remaining patients with normal s-SCS and t-SEPs may not have large fiber neuropathy. Conversely, all patients with abnormal s-SCS had abnormal t-SEPs (Group 3). This finding suggests the presence of severe, length-dependent neuropathy caused by severe axonal dysfunction of the peripheral nerve. Electrophysiological differentiation between demyelinating and axonal pathologies can be difficult using peripheral nerve conduction studies alone. Thus, the combined use of s-SCS and t-SEPs enables differentiation between length-dependent and non-length-dependent neuropathies.

The value of SEPs in CIDP is well established [12–18]. In accordance with previous studies' results, nine out of ten CIDP patients and two patients with combined central and peripheral demyelination (CCPD) were classified as Groups 2 and 3. Interestingly, one CIDP patient (Group 1) showed abnormal N20 with normal P37 in left t-SEPs, though left-SCS was not performed. The 2010 European Federation of Neurological Societies (EFNS)/Peripheral Nerve Society (PNS) criteria [26] and the 2021 European Academy of Neurology (EAN)/PNS guidelines for diagnosing CIDP do not recommend using SEPs for electrodiagnosis [27]. CIDP is confirmed if there is at least one demyelinating feature in two motor nerves and is only "possible" if demyelinating features are identified in only one nerve. The EAN/PNS diagnostic criteria also require sensory nerve conduction study (NCS) abnormalities in two or more nerves, which are defined as prolonged distal latency, reduced SNAP amplitude, or slowed conduction velocity. Doneddu et al. [28] performed a comparison of the diagnostic accuracy of the 2010 EFNS/PNS and 2021 EAN/PNS criteria for CIDP. They found that the EAN/PNS criteria are more specific for probable or definite CIDP, but less sensitive than the EFNS/PNS criteria. Thus, they concluded that more extensive nerve conduction studies improve the diagnostic sensitivity of the EAN/PNS criteria while maintaining a high specificity. Based on the results of previous studies [12–18] and our study, t-SEPs allow for a more thorough evaluation of the peripheral nervous system than conventional techniques.

The wide variety of etiologies of peripheral neuropathy and radiculoneuropathy is a significant limitation of the present study. Although we could not overcome this limitation, our study clearly demonstrated the effectiveness of using s-SCS and t-SEPs together on the electrodiagnosis of

 doi:10.20944/preprints202603.2358.v1

peripheral neuropathy. In the future, we plan to focus on differentiating between length-dependent and non-length-dependent neuropathies using s-SCS and t-SEPs.

5. Conclusions

Adding t-SEPs to s-SCS allows for an electrophysiological evaluation of both the distal and proximal segments of peripheral nerves and is useful for diagnosing and assessing the efficacy of treatment. Therefore, t-SEPs appear to be a second-line test that provides valuable and complementary information on the central and proximal nervous systems.

Author Contributions: Miki Oka: writing – original draft, data curation, resources, visualization. Shozo Tobimatsu: conceptualization, formal analysis, supervision, writing – review and editing. Akira Yokote, Ayako Sakoda, Saeko Inamizu: data curation, resources, visualization. Yuri Nakamura, Keiko Hara, Yuki Yanagihara, Yasutaka Iwanaga, Ken-ichiro Yamashita: resources. Jun-ichi Kira: supervision, writing – review and editing.

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Informed Consent Statement: Informed consent was waived due to retrospective nature of the study.

Data Availability Statement: The data that support the findings of this study are available on request from the corresponding author. The data are not publicly available due to privacy or ethical restrictions.

Conflicts of Interest: All authors of this manuscript declare that they have no conflicts of interest.

Abbreviations

The following abbreviations are used in this manuscript:

CCPD	Central and peripheral demyelination
CIDP	Chronic inflammatory demyelinating polyradiculoneuropathy
CMAP	Compound motor action potential
CSCT	Central sensory conduction time
DSP	Distal symmetric polyneuropathy
EFNS	European Federation of Neurological Societies
EAN	European Academy of Neurology
G	group
GBS	Guillain-Barré syndrome
NCS	Nerve conduction study
NCV	Nerve conduction velocity
PNS	Peripheral Nerve Society
SCS	Sensory conduction study
SEPs	Somatosensory evoked potentials
SNAP	Sensory nerve action potential
s-SCS	Sural verve SCS
t-SEPs	Tibial nerve SEPs

References

1. Watson, J.C.; James, P.B.; Dyck, B. Peripheral neuropathy: A practical approach to diagnosis and symptom management. *Mayo Clin. Proc.* **2015**, 90:940-951.
2. Callaghan, B.C.; Price, R.S.; Chen, K.S.; Feldman, E.L. Distal symmetric polyneuropathy: A review. *JAMA*. **2015**, 314:2172-2181.
3. Callaghan, B.C.; Price, R.S.; Chen, K.S.; Feldman, E.L. The importance of rare subtypes in diagnosis and treatment of peripheral neuropathy: A Review. *JAMA Neurol*. **2015**, 72:1510-1518.
4. Callaghan, B.C.; Price, R.S.; Feldman, E.L. Diagnostic and therapeutic advances: distal symmetric polyneuropathy. *JAMA* **2015**, 314:2172-2181,

 doi:10.20944/preprints202603.2358.v1

5. Dziadkowiak, E.; Waliszewska-Prosół, M.; Nowakowska-Kotas, M.; Budrewicz, S.; Koszewicz, Z.; Koszewicz, M. Pathophysiology of the different clinical phenotypes of chronic inflammatory demyelinating polyradiculoneuropathy (CIDP). *Int. J. Mol. Sci.* **2022**, 23, 179.
6. Hannaford, A.; Paling, E.; Silsby, M.; Vincenten, S.; van Alfen, N.; Simon, N.G. Electrodiagnostic studies and new diagnostic modalities for evaluation of peripheral nerve disorders. *Muscle Nerve* **2024**, 69:653-669.
7. Tankisi, H.; Pugdahl, K.; Johnsen, B.; Fuglsang-Frederiksen, A. Correlations of nerve conduction measures in axonal and demyelinating polyneuropathies. *Clin. Neurophysiol*. **2007**, 118: 2383-2392.
8. Eisen, A.; Elleker, G. Sensory nerve stimulation and evoked cerebral potentials. Neurology **1980**, 30: 1097-1105.
9. Parry, G.; Aminoff, M.J. Somatosensory evoked potentials in chronic acquired demyelinating peripheral neuropathy. *Neurology* **1987**, 37:313-316.
10. Cruccu, G.; Aminoff, M.J; Curio, G.; Guerit, J.M.; Kakigi, R.; Mauguiere, F.; Rossini, P.M.; Treede, R.-D.; Garcia-Larrea, L. Recommendations for the clinical use of somatosensory-evoked potentials. *Clin. Neurophysiol.* **2008**, 119:1705-1719.
11. Morizot-Koutlidis, R.; André-Obadia, N.; Antoine, J.-C.; Attarian, S.; Ayache, S.S; Azabou, E.; Benaderette, S.; Camdessanché, J.-P.; Cassereau, J.; Convers, P.; et al. Somatosensory evoked potentials in the assessment of peripheral neuropathies: Commented results of a survey among French-speaking practitioners and recommendations for practice. *Neurophysiol. Clin*. **2015**, 131-142.
12. Koutlidis, R.M.; Ayrignac, X.; Pradat, P.-F.; Le Forestier, N.; Léger, J.-M.; Salachas, F.; Maisonobe, T.; Fournier, E.; Viala, K. Segmental somatosensory-evoked potentials as a diagnostic tool in chronic inflammatory demyelinating polyneuropathies, and other sensory neuropathies. *Neurophysiol. Clin*. **2014**, 44:267-280,
13. Pineda, A.A.M.; Ogata, K .; Osoegawa, M.; Murai, H., Shigeto H.; Yoshiura, T.; Tobimatsu, S.; J.-I. Kira, J.-I. A distinct subgroup of chronic inflammatory demyelinating polyneuropathy with CNS demyelination and a favorable response to immunotherapy. *J. Neurol. Sci*. **2007**, 255:1-6.
14. Yannikas, C.; Vucic, S. Utility of somatosensory evoked potentials in chronic acquired demyelinating neuropathy. *Muscle Nerve* **2008**, 38:1447-1454.
15. Tsukamoto, H.; Sonoo, M.; Shimizu, T. Segmental evaluation of the peripheral nerve using tibial nerve SEP for the diagnosis of CIDP. *Clin. Neurophysiol*. **2010**, 121: 77-84.
16. Ayrignac, X.; Viala, K.; Koutlidis, R.M.; Taïeb, G.; Stojkovic, T.; Musset, L.; Léger, J.M.; Fournier, E.; Maisonobe, T.; Bouche, P. Sensory chronic inflammatory demyelinating polyneuropathy: an under-recognized entity? *Muscle Nerve* **2013**, 48: 727-32.
17. Salhi, H.; Corcia, P.; Remer, S; Praline J. Somatosensory evoked potentials in chronic inflammatory demyelinating polyradiculoneuropathy. *J. Clin. Neurophysiol*. **2014**, 31: 241-245.
18. Devic, P.; Mauguiere F. Diagnostic utility of somatosensory evoked potentials in chronic polyradiculopathy without electrodiagnostic signs of peripheral demyelination. *Muscle Nerve* **2016,** 53: 78–83.
19. Ziegler, D.; Mühlen, H.; Dannehl, K.; Gries, F.A. Tibial nerve somatosensory evoked potentials at various stages of peripheral neuropathy in insulin dependent diabetic patients. *J. Neurol. Neurosurg. Psychiatry* **1993,** 56: 58-64.
20. Kimura, J., Kohara, N. For Those Learning Nerve Conduction Studies and Electromyography. 2nd Ed. Igakushoin, Tokyo, 2010 (in Japanese).
21. Suga, R.; Tobimatsu, S.; Kira, J.; Kato, M. Motor and somatosensory evoked potential findings in HLTV-1 associated myelopathy. *J. Neurol. Sc*i. **1999,** 167:102-106.
22. Watanabe, A.; Matsushita, T.; Doi, H.; Matsuoka, T.; Shigeto, H.; Isobe, N.; Kawano, Y.; Tobimatsu, S.; Kira J.-I. Multimodality-evoked potential study of anti-aquaporin-4 antibody-positive and –negative multiple sclerosis patients. *J. Neurol. Sci*. **2009,** 28: 34-40.
23. Kanbayashi, T.; Ogawa, G.; Ito, T.; Hokkoku, K.; Oishi, C.; Hatanaka, Y.; Sonoo, M. Utility of the tibial nerve somatosensory evoked potentials in differentiating between neuromyelitis optica spectrum disorders and multiple sclerosis. *Mult. Scler. Relat. Disord*. **2023** Feb:70:104503.
24. Eisen, A.; Purves, S.; Hoirch, M. Central nervous system amplification: Its potential in the diagnosis of early multiple sclerosis. *Neurology* **1982,** 32:359-364.

 doi:10.20944/preprints202603.2358.v1

25. Kira, J.-I. Anti-Neurofascin 155 antibody-positive chronic inflammatory demyelinating polyneuropathy/combined central and peripheral demyelination: Strategies for diagnosis and treatment based on the disease mechanism. *Front. Neurol*. **2021** Jun 10:12:665136.
26. Van den Bergh, P.Y.,; Hadden, R.D.; Bouche, P.; Cornblath, D.R.; Hahn, A.; Illa, I.; Koski, C.L.; Léger, J.M.; Nobile-Orazio, E.; Pollard, J.; et al. European Federation of Neurological Societies; Peripheral Nerve Society, European Federation of Neurological Societies/Peripheral Nerve Society guideline on management of chronic inflammatory demyelinating polyradiculoneuropathy: report of a joint task force of the European Federation of Neurological Societies and the Peripheral Nerve Society - first revision. *Eur. J. Neurol*. **2010,** 17:356–363.
27. Van den Bergh, P.Y.K.; van Doorn, P.A.; Hadden, R.D.M.; Avau, B.; Vankrunkelsven, P.; Allen, J.A.; Attarian, S.; Blomkwist-Markens, P.H.; Cornblath, D.R.; Eftimov, F.; et al. European Academy of Neurology/Peripheral Nerve Society guideline on diagnosis and treatment of chronic inflammatory demyelinating polyradiculoneuropathy: Report of a joint Task Force–Second revision. *Eur. J. Neurol*. **2021,** 28:3556-3583. Correction in *Eur. J. Neurol*. **2022,** 29:1288. https://doi.org/10.1111/ene.15225.
28. Domeddu, P.E.; De Lorenzo, A.; Manganelli, F.; Cocito, D.; Fazio, R.; Briani, C.; Mazzeo, A.; Filosto, M.; Cosentino, G.; Benedetti, L.; et al. Comparison of the diagnostic accuracy of the 2021 EAN/PNS and 2010 EFNS/PNS diagnostic criteria for chronic inflammatory demyelinating polyradiculoneuropathy. *J. Neurol. Neurosurg. Psychiaty* **2022**, 93:1239-1246.