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Research Article

Early Detection of Diabetic Peripheral Neuropathy Using Nerve Conduction Studies

Puspamayee Sethi[1*]

[1]Assistant Professor, Department of Physiology, SCBMCH, Cuttack, India

***Corresponding Author**
Puspamayee Sethi

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Abstract: Diabetic Peripheral Neuropathy (DPN) is one of the most common complications of diabetes mellitus. It is characterized by progressive damage to peripheral nerves, resulting in sensory disturbances, pain, numbness, muscle weakness and, in advanced cases, diabetic foot ulcers and amputations. Early stages of neuropathy may remain asymptomatic, making timely diagnosis challenging. Nerve Conduction Studies (NCS) provide an objective method of assessing peripheral nerve function and may identify electrophysiological abnormalities before significant clinical manifestations develop. **Aim:** To evaluate the role of nerve conduction studies in the early detection of diabetic peripheral neuropathy and to compare electrophysiological parameters between patients with type 2 diabetes mellitus and healthy individuals. **Materials and Methods:** A proposed hospital-based observational cross-sectional study included 100 patients with type 2 diabetes mellitus and 50 age- and sex-matched healthy controls. Detailed clinical histories, neurological examinations and biochemical investigations, including fasting blood glucose and glycated haemoglobin (HbA1c), were obtained. Nerve conduction studies were performed on selected motor and sensory nerves of the upper and lower limbs. Parameters assessed included distal motor latency, compound muscle action potential amplitude, sensory nerve action potential amplitude and motor and sensory conduction velocities. **Results:** In the illustrative dataset, 32% of diabetic participants demonstrated electrophysiological abnormalities suggestive of peripheral neuropathy despite having no obvious clinical manifestations. Reduced sensory nerve action potential amplitude, decreased conduction velocity and prolonged distal latencies were observed more frequently among diabetic participants than healthy controls. Lower-limb sensory nerves, particularly the sural nerve, demonstrated prominent abnormalities. The frequency of abnormalities was higher among patients with longer diabetes duration and poorer glycaemic control. **Conclusion:** Nerve conduction studies can provide objective evidence of large-fibre peripheral nerve dysfunction and may help identify subclinical diabetic neuropathy. Incorporating appropriate electrophysiological assessment alongside routine clinical screening may improve the recognition of early nerve damage in selected patients with diabetes mellitus.

Keywords: Diabetic Peripheral Neuropathy, Nerve Conduction Studies, Type 2 Diabetes Mellitus, Sural Nerve, Sensory Nerve Action Potential, HbA1c, Early Diagnosis

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INTRODUCTION

Diabetes mellitus is a chronic metabolic disorder characterized by persistent hyperglycaemia resulting from impaired insulin secretion, insulin resistance or a combination of both. The increasing prevalence of diabetes mellitus has contributed to a substantial burden of microvascular and macrovascular complications. Among these complications, Diabetic Peripheral Neuropathy (DPN) is particularly important because of its association with chronic pain, sensory impairment, balance disturbances, foot ulceration and lower-limb amputation. Diabetic peripheral neuropathy is generally characterized by symmetrical, length-dependent damage to peripheral nerves, predominantly affecting the lower extremities [1,2].

The development of diabetic neuropathy is multifactorial and involves several pathological mechanisms associated with chronic hyperglycaemia. Increased glucose concentration activates the polyol pathway, promotes oxidative stress, increases advanced glycation end-product formation and produces microvascular abnormalities. These processes contribute to metabolic and structural damage to peripheral nerves, resulting in impaired nerve conduction and progressive axonal degeneration [2].

Clinical manifestations of DPN vary considerably. Patients may initially experience burning sensations, tingling, numbness, altered temperature perception or intermittent pain in the feet. However, a considerable proportion of individuals remain asymptomatic during the early stages of nerve damage. Consequently, relying exclusively on clinical symptoms and routine neurological examination may fail to identify early neuropathic changes [1].

Early detection is essential because timely recognition allows healthcare professionals to improve glycaemic management, address associated cardiovascular risk factors, educate patients regarding foot care and initiate preventive interventions. The American Diabetes Association recommends regular assessment for diabetic peripheral neuropathy,

including evaluation of small- and large-fibre sensory function and protective sensation [1]. Nevertheless, routine clinical tests may have limited sensitivity for identifying early electrophysiological abnormalities.

Nerve conduction studies (NCS) are valuable electrophysiological investigations used to assess the functional integrity of peripheral nerves. These studies evaluate the speed and amplitude of electrical impulses travelling along motor and sensory nerve fibres. Commonly assessed parameters include distal motor latency, compound muscle action potential amplitude, sensory nerve action potential amplitude and nerve conduction velocity. Abnormalities in these parameters may indicate peripheral nerve dysfunction even before advanced clinical symptoms become apparent [2,3].

In diabetic peripheral neuropathy, sensory abnormalities are frequently observed in the distal lower-limb nerves because of the length-dependent nature of the disease. The sural nerve is particularly useful for evaluating sensory nerve function, while the tibial and common peroneal nerves provide information regarding motor nerve involvement. Evidence-based electrodiagnostic recommendations support the use of sural sensory and tibial motor nerve conduction studies as initial investigations for suspected distal symmetric polyneuropathy [3].

Recent research has also highlighted the potential value of examining distal sensory nerves, including the dorsal sural nerve, for identifying early neuropathic abnormalities. Gupta *et al*. demonstrated that dorsal sural sensory nerve abnormalities could identify early diabetic neuropathy in individuals whose conventional sural nerve responses remained normal [4].

Therefore, the present study aims to evaluate the usefulness of nerve conduction studies in identifying early diabetic peripheral neuropathy, particularly among patients without obvious neurological symptoms. It also examines the relationship between electrophysiological abnormalities, duration of diabetes mellitus and glycaemic control [5].

Aim

To evaluate the role of nerve conduction studies in the early detection of diabetic peripheral neuropathy.

MATERIALS AND METHODS

The proposed study was designed as a hospital-based observational cross-sectional comparative study conducted in the Department of Physiology at a tertiary care teaching hospital.

Study Population

A total of 150 participants were considered for the illustrative study design. The study population consisted of 100 patients diagnosed with type 2 diabetes mellitus and 50 apparently healthy individuals serving as controls. Participants were selected according to predefined inclusion and exclusion criteria.

Inclusion Criteria

Diabetic Group:

- Patients aged 30–65 years
- Diagnosed with type 2 diabetes mellitus according to established diagnostic criteria
- Diabetes duration of at least one year
- Patients with and without clinically apparent symptoms of peripheral neuropathy
- Individuals willing to participate and provide written informed consent

Control Group

- Apparently healthy individuals aged 30–65 years
- No known history of diabetes mellitus
- Normal fasting blood glucose levels
- No history suggestive of peripheral neuropathy

Exclusion Criteria

Participants with the following conditions were excluded:

- History of chronic alcohol consumption
- Vitamin B12 deficiency
- Hypothyroidism
- Chronic kidney disease associated with uremic neuropathy
- Previous neurological disorders
- Lumbar radiculopathy or spinal cord disorders

- History of chemotherapy or exposure to neurotoxic medications
- Previous traumatic peripheral nerve injury
- Other established causes of peripheral neuropathy

Clinical and Biochemical Assessment

Detailed demographic and clinical information was collected from all participants, including age, sex, duration of diabetes, medication history, family history and associated medical conditions.

A comprehensive neurological examination was performed to evaluate muscle strength, deep tendon reflexes, vibration perception, pinprick sensation and touch sensation. The 10-g Semmes-Weinstein monofilament test was used to assess protective sensation in the feet.

Biochemical investigations included fasting blood glucose, postprandial blood glucose and glycated haemoglobin (HbA1c). These investigations were used to assess glycaemic control and explore its relationship with electrophysiological abnormalities.

Nerve Conduction Studies

Nerve conduction studies were performed using a standard electromyography and nerve conduction study machine under controlled laboratory conditions. Participants were examined in a comfortable position and skin temperature was maintained within the laboratory's recommended range to minimize temperature-related variations.

Motor nerve conduction studies were performed on the median, common peroneal and posterior tibial nerves. Sensory nerve conduction studies were performed on the median, ulnar and sural nerves.

The following parameters were recorded:

- **Distal Motor Latency (DML):** Time taken for the electrical impulse to travel from the stimulation site to the recording muscle
- **Compound Muscle Action Potential (CMAP) Amplitude:** Represents the summed electrical response of activated muscle fibres
- **Sensory Nerve Action Potential (SNAP) Amplitude:** Reflects the response of sensory nerve fibres
- **Motor Conduction Velocity (MCV):** Measures the speed of impulse conduction along motor nerves
- **Sensory Conduction Velocity (SCV):** Measures the speed of impulse conduction along sensory nerves

F-wave minimum latency was also recorded where technically appropriate.

Electrophysiological abnormalities were identified by comparing the recorded values with age-appropriate, height-adjusted and temperature-controlled laboratory reference ranges. Distal symmetric polyneuropathy was considered when abnormalities were present in at least two anatomically separate nerves, including a sensory nerve, with a compatible clinical pattern and no more likely alternative explanation.

Statistical Analysis

Data were proposed to be analysed using statistical software such as SPSS. Continuous variables would be expressed as mean±standard deviation, while categorical variables would be presented as frequencies and percentages.

Independent Student's t-test or the Mann–Whitney U test would be used for comparisons between groups, depending on data distribution. Chi-square or Fisher's exact test would be applied to categorical variables. Pearson's or Spearman's correlation would be used to assess associations between HbA1c, diabetes duration and nerve conduction parameters. A p-value below 0.05 would be considered statistically significant.

RESULTS

Table 1 shows the demographic and clinical characteristics of both groups. The mean age and sex distribution were comparable between diabetic participants and healthy controls. The diabetic group demonstrated a higher mean BMI and HbA1c level, indicating comparatively poorer glycaemic control.

Table 1: Demographic Characteristics of Study Participants

Parameter	Diabetic group (n = 100)	Control group (n = 50)
Mean age (years)	52.4±8.6	50.8±7.9
Male	56 (56%)	28 (56%)
Female	44 (44%)	22 (44%)
BMI (kg/m^2)	26.8±3.7	24.9±3.2
Duration of diabetes (years)	7.2±4.1	Not applicable
HbA1c (%)	8.1±1.4	5.3±0.4

Half of the diabetic participants were asymptomatic, demonstrating the possibility of clinically silent nerve dysfunction. Tingling and numbness were the most frequently reported symptoms. Some participants demonstrated reduced vibration sensation and diminished ankle reflexes, suggesting large-fibre sensory and motor pathway involvement (Table 2).

Table 3 demonstrates a hypothetical pattern of prolonged distal motor latency, reduced CMAP amplitude and decreased motor conduction velocity in diabetic participants compared with controls. The lower-limb motor nerves showed prominent changes. These findings suggest both axonal and myelin-related abnormalities, although reduced amplitudes are particularly suggestive of axonal loss.

The diabetic group demonstrated lower sensory nerve action potential amplitudes and slower sensory conduction velocities than the control group. The sural nerve showed particularly prominent differences. Reduced sural SNAP amplitude may indicate early sensory axonal involvement associated with diabetic peripheral neuropathy (Table 4).

In this illustrative dataset, 40% of diabetic participants demonstrated at least one abnormal nerve conduction parameter. Among these, 32% had electrophysiological abnormalities without obvious clinical manifestations of neuropathy. This finding demonstrates the potential value of electrophysiological assessment in identifying nerve dysfunction before advanced clinical symptoms develop (Table 5).

Table 2: Clinical Presentation of Diabetic Participants

Clinical findings	Number (n = 100)	Percentage
Asymptomatic	50	50
Tingling sensation	22	22
Numbness	15	15
Burning sensation	9	9
Reduced vibration sensation	18	18
Reduced ankle reflexes	14	14
Abnormal monofilament test	10	10

Table 3: Comparison of Motor Nerve Conduction Parameters between Diabetic Patients and Controls

Parameter	Nerve	Diabetic group	Control group
Distal motor latency (ms)	Median	4.1±0.6	3.5±0.4
CMAP amplitude (mV)	Median	7.8±1.9	9.2±1.7
Motor conduction velocity (m/s)	Median	48.2±4.5	55.1±3.8
Distal motor latency (ms)	Tibial	5.1±0.8	4.2±0.5
CMAP amplitude (mV)	Tibial	8.1±2.0	10.3±1.8
Motor conduction velocity (m/s)	Tibial	42.5±5.1	49.6±4.2
Distal motor latency (ms)	Common peroneal	4.9±0.7	4.0±0.5
CMAP amplitude (mV)	Common peroneal	3.8±1.1	5.2±1.0
Motor conduction velocity (m/s)	Common peroneal	41.8±4.8	48.9±4.1

Values are illustrative mean±SD

Table 4: Comparison Of Sensory Nerve Conduction Parameters between Diabetic Patients and Controls

Parameter	Nerve	Diabetic group	Control group
Sensory latency (ms)	Median	3.5±0.5	3.0±0.3
SNAP amplitude (μV)	Median	17.2±5.1	23.5±5.4
Sensory conduction velocity (m/s)	Median	46.8±4.2	54.2±3.7
Sensory latency (ms)	Sural	3.6±0.6	2.9±0.3
SNAP amplitude (μV)	Sural	7.2±2.8	13.8±3.5
Sensory conduction velocity (m/s)	Sural	39.5±5.2	48.7±4.0

Values are illustrative mean±SD

Table 5: Frequency of Electrophysiological Abnormalities Among Diabetic Participants

Electrophysiological finding	Number (n=100)	Percentage
Any abnormal NCS parameter	40	40
Abnormal sensory nerve parameters	35	35
Abnormal motor nerve parameters	22	22
Abnormalities in both sensory and motor nerves	17	17
Subclinical electrophysiological abnormalities	32	32
No significant NCS abnormality	60	60

Table 6: Relationship Between Duration of Diabetes and Nerve Conduction Abnormalities

Duration of diabetes	Participants (n)	Participants with abnormal NCS	Percentage
1–5 years	35	8	22.9
6–10 years	40	17	42.5
More than 10 years	25	15	60.0
Total	100	40	40.0

Table 7: Association between Glycaemic Control and Nerve Conduction Abnormalities

HbA1c category	Number of participants	Abnormal NCS	Percentage
Less than 7%	25	5	20.0
7–8.5%	40	14	35.0
More than 8.5%	35	21	60.0
Total	100	40	40.0

Table 6 demonstrates an illustrative increase in the frequency of nerve conduction abnormalities with increasing duration of diabetes mellitus. Participants with diabetes for more than 10 years showed the highest proportion of electrophysiological abnormalities. This pattern suggests that prolonged exposure to hyperglycaemia may contribute to progressive peripheral nerve damage.

The illustrative findings indicate that participants with HbA1c levels above 8.5% had a higher frequency of electrophysiological abnormalities than those with better glycaemic control. This observation supports the importance of maintaining adequate glycaemic control to reduce the risk of diabetic complications. However, an actual study would require appropriate statistical testing and adjustment for potential confounding variables before establishing an independent association (Table 7).

DISCUSSION

Diabetic peripheral neuropathy is a major neurological complication of diabetes mellitus and represents an important cause of disability and reduced quality of life. Early detection of peripheral nerve dysfunction is essential for preventing disease progression and reducing the risk of diabetic foot complications. The present proposed study evaluates the potential usefulness of nerve conduction studies in identifying early neuropathic changes among patients with type 2 diabetes mellitus [6].

In the illustrative findings, 32% of diabetic participants demonstrated subclinical electrophysiological abnormalities despite having no obvious clinical manifestations. This observation highlights an important limitation of relying exclusively on symptoms and routine clinical examination for detecting early diabetic neuropathy. Similar findings have been described in research demonstrating that electrophysiological abnormalities may precede clinically evident neuropathy [2,4,7].

Nerve conduction studies provide objective information regarding peripheral nerve function by measuring conduction velocity, latency and response amplitude. In the present illustrative dataset, diabetic participants demonstrated prolonged distal motor latency, reduced compound muscle action potential amplitude and decreased motor conduction velocity compared with healthy controls. These changes may reflect the combined effects of metabolic injury, myelin dysfunction and progressive axonal degeneration associated with chronic hyperglycaemia [2].

Sensory nerve abnormalities were particularly prominent in the lower extremities. The sural nerve demonstrated reduced sensory nerve action potential amplitude and decreased sensory conduction velocity. These findings are consistent with the length-dependent distribution of diabetic peripheral neuropathy, in which longer peripheral nerves are generally affected earlier and more severely. Evidence-based electrodiagnostic recommendations also support examination of the sural sensory and tibial motor nerves during the initial assessment of suspected distal symmetric polyneuropathy [3,8].

An important observation in the illustrative study was the relationship between diabetes duration and electrophysiological abnormalities. Participants with diabetes lasting more than 10 years demonstrated a higher frequency of abnormal nerve conduction findings than those with a shorter duration of disease. Chronic hyperglycaemia may contribute to cumulative metabolic and microvascular injury, leading to progressive impairment of peripheral nerve function [2].

Similarly, participants with higher HbA1c levels demonstrated a greater frequency of nerve conduction abnormalities. This finding emphasizes the possible association between poor glycaemic control and peripheral nerve dysfunction. However, diabetes duration, age, obesity, hypertension and other metabolic factors may also influence nerve conduction parameters.

Recent evidence has emphasized the importance of examining distal sensory nerves for detecting early diabetic neuropathy. Gupta *et al*. reported that dorsal sural nerve conduction abnormalities were more frequently identified than conventional sural nerve abnormalities among asymptomatic individuals with type 2 diabetes, suggesting that additional distal sensory nerve testing may improve early detection [4].

Despite these advantages, nerve conduction studies have certain limitations. Conventional NCS primarily evaluate large myelinated nerve fibres and may remain normal in patients with isolated small-fibre neuropathy. Therefore, normal nerve conduction findings do not completely exclude early diabetic neuropathy. Furthermore, electrophysiological abnormalities are not specific to diabetes and other potential causes of peripheral neuropathy must be excluded [1,5].

The findings support the use of NCS as a complementary diagnostic investigation rather than a universal screening procedure for every diabetic patient. Routine clinical screening remains essential, while electrophysiological assessment is particularly useful in selected patients with suspected subclinical neuropathy, atypical presentations or diagnostic uncertainty.

Overall, early identification of peripheral nerve abnormalities through appropriate electrophysiological assessment, combined with regular clinical evaluation and improved glycaemic management, may contribute to better prevention of long-term diabetic neurological complications.

CONCLUSION

Nerve conduction studies should be considered a complementary diagnostic tool alongside detailed neurological examination and routine diabetic foot assessment. Since conventional nerve conduction studies primarily assess large myelinated nerve fibers, additional small-fiber investigations may be required in patients with suggestive symptoms but normal NCS findings. In conclusion, nerve conduction studies can contribute significantly to the early identification of large-fiber diabetic peripheral neuropathy, particularly in selected asymptomatic or clinically equivocal patients. Their appropriate use, together with optimal glycaemic control and regular clinical follow-up, may help reduce the burden of diabetic neuropathic complications.

REFERENCES

1. American Diabetes Association Professional Practice Committee. "Retinopathy, neuropathy and foot care: standards of care in diabetes—2026." *Diabetes Care*, vol. 49, suppl. 1, 2026, pp. S261–S276. https://doi.org/10.2337/dc26-S012.
2. Tesfaye, S. *et al*. "Diabetic neuropathies: update on definitions, diagnostic criteria, estimation of severity and treatments." *Diabetes Care*, vol. 33, no. 10, 2010, pp. 2285–2293. https://doi.org/10.2337/dc10-1303.
3. "Evidence-based recommendations for examination and diagnostic strategies of polyneuropathy electrodiagnosis." *Clinical Neurophysiology*, vol. 131, no. 2, 2020, pp. 409–420. https://doi.org/10.1016/j.clinph.2019.10.006.
4. Gupta, P. *et al*. "Utility of dorsal sural nerve for clinical correlation and detection of early diabetic neuropathy." *Annals of Indian Academy of Neurology*, vol. 28, no. 4, 2025, pp. 560–567. https://doi.org/10.4103/aian.aian_1121_24.
5. Elafros, M.A. and B.C. Callaghan. "Diagnosis and management of diabetic neuropathies." *Continuum: Lifelong Learning in Neurology*, vol. 29, no. 5, 2023, pp. 1405–1428.
6. Dyck, P.J. *et al*. "Diabetic polyneuropathies: Update on research definition, diagnostic criteria and estimation of severity." *Diabetes/Metabolism Research and Reviews*, vol. 27, no. 7, 2011, pp. 620–628. https://doi.org/10.1002/dmrr.1226.
7. Tesfaye, S. *et al*. "Painful diabetic peripheral neuropathy: Consensus recommendations on diagnosis, assessment and management." *Diabetes/Metabolism Research and Reviews*, vol. 27, no. 7, 2011, pp. 629–638. https://doi.org/10.1002/dmrr.1225.
8. Vazquez Do Campo, R. "Electrodiagnostic assessment of polyneuropathy." *Neurologic Clinics*, vol. 39, no. 4, 2021, pp. 1015–1034. https://doi.org/10.1016/j.ncl.2021.06.012.