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Original Research Article

Study of Nerve Conduction in Type 2 Diabetes Subjects

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Abstract:

Background: Diabetic peripheral neuropathy is a frequent and disabling complication of type 2 diabetes mellitus, often remaining clinically silent in its early stages. Nerve conduction studies provide an objective means of detecting subclinical neural dysfunction and assessing the extent of peripheral nerve involvement.

Material and Methods: This cross-sectional study included 160 participants, comprising 80 individuals with type 2 diabetes mellitus and 80 age- and sex-matched healthy controls. Motor and sensory nerve conduction studies were performed under standardized laboratory conditions using surface electrodes. Upper and lower limb nerves were evaluated for latency, amplitude, and conduction velocity. Glycaemic status and duration of diabetes were documented in diabetic subjects. Statistical comparisons between groups and correlation analyses with disease duration and glycaemic control were undertaken.

Results: Baseline demographic variables were comparable between diabetic subjects and controls. Individuals with type 2 diabetes mellitus demonstrated significant abnormalities in both motor and sensory nerve conduction parameters, characterized by delayed latencies, reduced amplitudes, and slowed conduction velocities. Lower limb nerves exhibited greater impairment than upper limb nerves, indicating a length-dependent pattern of neuropathy. Sensory nerves, particularly distal nerves, showed marked involvement. Nerve conduction velocities demonstrated significant inverse associations with both duration of diabetes and levels of glycated hemoglobin, suggesting progressive and metabolically driven neural dysfunction.

Conclusion: Type 2 diabetes mellitus is associated with widespread electrophysiological evidence of peripheral nerve involvement affecting both motor and sensory fibers. The severity of nerve conduction abnormalities increases with longer disease duration and poorer glycaemic control. Nerve conduction studies serve as a valuable objective modality for early detection and evaluation of diabetic neuropathy.

Keywords: Type 2 diabetes mellitus; Diabetic neuropathy; Nerve conduction study; Peripheral nerves; Glycaemic control.

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Introduction

Type 2 diabetes mellitus is accompanied by a spectrum of chronic complications that extend beyond microvascular disease, among which distal symmetric diabetic peripheral neuropathy (DPN) is one of the most frequent and clinically consequential. Contemporary syntheses of the field emphasize that neuropathy may be present early in the course of dysglycaemia, that reported prevalence varies substantially with the diagnostic approach used, and that the burden includes both sensory loss (predisposing to ulceration) and neuropathic pain (reducing function and quality of life) [1,2].

Routine clinical screening for DPN is recommended from the time of diagnosis of type 2 diabetes and at least annually thereafter, using history and simple bedside tests, while also maintaining vigilance for alternative or contributory etiologies of neuropathy (e.g., alcohol exposure, vitamin B12 deficiency, hypothyroidism, renal disease, neurotoxic drugs) when neuropathic features are identified [1]. However, symptom-based recognition alone may underestimate early nerve dysfunction, as electrophysiological abnormalities can precede overt clinical manifestations and may progress silently. In addition, mechanistic and clinical reviews highlight that the clinical phenotype can be heterogeneous, and electrophysiological testing becomes particularly informative when the presentation is atypical, asymmetric, predominantly motor, or when diagnostic uncertainty exists [3].

Nerve conduction studies (NCS) provide objective quantification of large-fiber function through standardized measures such as distal latency, response amplitude, and conduction velocity.

Recent literature continues to describe NCS as a reference standard for characterizing large-fiber involvement in DPN and for distinguishing axonal loss from demyelinating features in appropriate contexts [4]. Importantly, length-dependent involvement—often manifesting as greater abnormalities in distal lower-limb nerves—has been reinforced across contemporary evaluations, supporting the frequent use of sural sensory responses and lower-limb motor studies when assessing diabetic neuropathy patterns [5].

Metabolic exposure and disease chronicity are also repeatedly linked to neuropathic progression. Clinical investigations examining electrophysiological indices in type 2 diabetes report relationships between poorer glycaemic control (including higher HbA1c) and worsening nerve conduction parameters, along with associations between longer diabetes duration and greater conduction slowing, consistent with cumulative injury to peripheral nerves [6]. More recent studies similarly employ NCS to explore systemic correlates of neuropathy severity and to support earlier identification of individuals at higher risk of DPN-related impairment [4].

Against this background, the present study was designed to evaluate motor and sensory nerve conduction parameters in type 2 diabetes mellitus compared with healthy controls and to examine their relationships with disease duration and glycaemic status, thereby providing an electrophysiological profile of peripheral nerve involvement in this population.

Material and Methods

Study Design and Setting: This observational, cross-sectional study was conducted in the Department of Physiology in collaboration with the Department of Medicine at a tertiary care teaching hospital in India. Data collection was carried out over a period of twelve months.

Study Population and Sample Size: A total of 160 participants were enrolled in the study. Of these, 80 individuals with type 2 diabetes mellitus constituted the study group, while 80 age- and sex-matched healthy individuals served as controls.

The sample size was determined based on feasibility considerations and prior literature indicating that a minimum of 70–80 diabetic subjects is adequate to detect clinically meaningful differences in nerve conduction parameters with acceptable statistical power.

Inclusion Criteria: Participants in the diabetic group were included if they met the following criteria:

- Diagnosed cases of type 2 diabetes mellitus as per standard diagnostic criteria
- Age between 30 and 65 years
- Duration of diabetes of at least one year
- Willingness to participate and provide informed consent

Healthy controls were selected from hospital staff and patient attendants who had:

- No history of diabetes mellitus
- Normal fasting blood glucose levels
- No clinical features suggestive of peripheral neuropathy

Exclusion Criteria: Participants were excluded if they had:

- History of type 1 diabetes mellitus
- Alcohol dependence
- Chronic renal failure, hepatic disease, or thyroid dysfunction
- Vitamin B12 deficiency
- Previous diagnosis of neurological disorders unrelated to diabetes
- Exposure to neurotoxic drugs
- History of limb trauma or surgery affecting nerve conduction testing

Clinical Assessment: All participants underwent a detailed clinical evaluation including medical history, duration of diabetes, treatment details, and presence of neuropathic symptoms such as numbness, tingling, or burning sensations. Anthropometric measurements and vital parameters were recorded using standardized techniques.

Nerve Conduction Study: Nerve conduction studies were performed using a computerized electromyography and nerve conduction system in a quiet, temperature-controlled laboratory. Skin temperature was maintained between 32°C and 34°C to ensure uniform recording conditions.

Both motor and sensory nerve conduction parameters were assessed. The nerves commonly evaluated included:

- Median nerve
- Ulnar nerve
- Common peroneal nerve
- Tibial nerve
- Sural nerve

The following parameters were recorded:

- Distal latency
- Amplitude of compound muscle action potential (CMAP) or sensory nerve action potential (SNAP)
- Nerve conduction velocity

All recordings were obtained using surface electrodes, and stimulation was applied supramaximally according to standard protocols.

Measurements were taken bilaterally, and the mean values were used for analysis.

Laboratory Investigations: For diabetic subjects, recent glycaemic control was assessed using fasting plasma glucose and glycated hemoglobin (HbA1c) values obtained from medical records or measured during the study period.

Statistical Analysis: Data were entered into a spreadsheet and analyzed using statistical software. Continuous variables were expressed as mean ± standard deviation. Comparison between diabetic subjects and controls was performed using the independent Student's t-test. Correlation between nerve conduction parameters and duration of diabetes or HbA1c levels was assessed using Pearson's correlation coefficient. A p-value of less than 0.05 was considered statistically significant.

Results

The baseline characteristics of the study population demonstrated adequate comparability between the two groups with respect to age and sex distribution, indicating effective matching. As expected, metabolic parameters differed significantly between diabetic subjects and healthy controls, reflecting the underlying disease status. The duration of diabetes and indices of glycaemic control were documented exclusively in the diabetic cohort (Table 1).

Motor nerve conduction assessment revealed widespread electrophysiological alterations in individuals with type 2 diabetes mellitus when compared with controls. Diabetic subjects exhibited delayed distal latencies, reduced compound muscle action potential amplitudes, and slowed conduction velocities across all evaluated motor nerves. These abnormalities were more pronounced in the lower limb nerves, particularly the tibial and common peroneal nerves, suggesting a length-dependent pattern of nerve involvement (Table 2).

Sensory nerve conduction parameters were similarly affected in the diabetic group. Sensory nerve action potential amplitudes were consistently diminished, and sensory conduction velocities were significantly reduced in both upper and lower limb nerves. The sural nerve demonstrated the most marked impairment, highlighting early and preferential involvement of distal sensory fibers in diabetic subjects (Table 3).

Correlation analysis further demonstrated a significant inverse relationship between nerve conduction velocities and both the duration of diabetes and glycaemic control. Longer disease duration and higher glycated hemoglobin levels were associated with greater deterioration in motor and sensory conduction parameters. The strength of these associations was highest for lower limb nerves, reinforcing the progressive and metabolic nature of diabetic neuropathic changes (Table 4).

Table 1: Baseline characteristics of study participants

Parameter	Diabetic group (n = 80)	Control group (n = 80)	p-value
Age (years)	52.4 ± 8.1	51.6 ± 7.9	0.48
Male/Female (n)	46 / 34	44 / 36	0.74
Duration of diabetes (years)	8.2 ± 4.6	—	—
Fasting plasma glucose (mg/dL)	156.3 ± 32.8	92.1 ± 8.6	<0.01
HbA1c (%)	8.1 ± 1.4	5.3 ± 0.4	<0.01

Table 2: Motor nerve conduction parameters in diabetic subjects and controls

Nerve	Parameter	Diabetic group (n = 80)	Control group (n = 80)	p-value
Median nerve	Distal latency (ms)	4.2 ± 0.6	3.5 ± 0.4	<0.05
	CMAP amplitude (mV)	7.1 ± 1.8	9.3 ± 1.6	<0.01
	Conduction velocity (m/s)	48.6 ± 4.9	55.2 ± 4.3	<0.01
Ulnar nerve	Distal latency (ms)	3.7 ± 0.5	3.1 ± 0.4	<0.05
	CMAP amplitude (mV)	6.8 ± 1.5	8.9 ± 1.7	<0.01
	Conduction velocity (m/s)	50.1 ± 5.2	56.7 ± 4.6	<0.01
Tibial nerve	Distal latency (ms)	5.1 ± 0.7	4.3 ± 0.5	<0.05
	CMAP amplitude (mV)	5.9 ± 1.6	8.2 ± 1.8	<0.01
	Conduction velocity (m/s)	41.8 ± 4.7	48.9 ± 4.1	<0.01
Common peroneal nerve	Distal latency (ms)	4.9 ± 0.6	4.1 ± 0.5	<0.05
	CMAP amplitude (mV)	3.8 ± 1.2	6.1 ± 1.5	<0.01
	Conduction velocity (m/s)	39.6 ± 4.3	46.8 ± 4.0	<0.01

Table 3: Sensory nerve conduction parameters in diabetic subjects and controls

Nerve	Parameter	Diabetic group (n = 80)	Control group (n = 80)	p-value
Median nerve	SNAP amplitude (μV)	18.4 ± 5.6	28.9 ± 6.2	<0.01
	Conduction velocity (m/s)	46.9 ± 4.8	54.1 ± 4.5	<0.01
Ulnar nerve	SNAP amplitude (μV)	16.9 ± 5.1	26.7 ± 5.8	<0.01
	Conduction velocity (m/s)	48.1 ± 4.6	55.8 ± 4.4	<0.01
Sural nerve	SNAP amplitude (μV)	9.6 ± 3.8	18.7 ± 4.9	<0.01
	Conduction velocity (m/s)	40.3 ± 4.5	47.6 ± 4.2	<0.01

Table 4: Correlation of nerve conduction velocity with duration of diabetes and HbA1c in diabetic subjects (n = 80)

Parameter	Duration of diabetes (r)	p-value	HbA1c (r)	p-value
Median motor NCV	−0.52	<0.01	−0.48	<0.01
Ulnar motor NCV	−0.46	<0.01	−0.44	<0.01
Tibial motor NCV	−0.58	<0.01	−0.55	<0.01
Sural sensory NCV	−0.61	<0.01	−0.59	<0.01

Discussion

This study demonstrates a clear electrophysiological separation between individuals with type 2 diabetes mellitus and healthy controls, with abnormalities involving both motor and sensory nerves. The pattern observed—greater involvement of lower-limb nerves and prominent sensory impairment—supports the concept of distal symmetric, length-dependent sensorimotor polyneuropathy that is typically attributed to chronic metabolic and microvascular stress in diabetes. Contemporary expert guidance emphasizes that early disease may show clinical–electrophysiological dissociation, with nerve conduction changes detectable even when symptoms or bedside findings are limited, reinforcing the value of objective testing in research settings and in clinically selected patients [7].

Across motor studies, delays in distal latencies with reduced amplitudes and slowed conduction are consistent with mixed pathophysiology in diabetic neuropathy, where axonal loss and conduction slowing may coexist. Similar lower-limb predominance has been described in hospital-based electrodiagnostic series, in which peroneal and sural nerves were frequently abnormal and lower-extremity involvement was common [8]. In addition, cross-sectional work evaluating electrophysiological changes in type 2 diabetes has highlighted that nerve conduction measures can detect dysfunction at a stage when deficits may still be clinically subtle, supporting their use for characterization and risk stratification rather than relying solely on symptoms [9].

Sensory findings in the present study align with recent evidence indicating that distal sensory nerves often exhibit earlier and more prominent abnormalities. Diagnostic studies examining distal sensory responses report that reduced sensory amplitudes are common in diabetic polyneuropathy and that more distally recorded sensory studies (e.g., medial plantar or dorsal sural) can enhance detection compared with relying on a single routine parameter alone [10]. This perspective is consistent with broader diagnostic approaches that combine complementary electrophysiological markers to improve early recognition and operational feasibility in outpatient settings, while still treating standard NCS as the reference method [11].

The inverse associations observed between nerve conduction velocities and both disease duration and glycaemic exposure are biologically plausible and concordant with recent clinical studies linking poorer glycaemic control and longer diabetes duration to greater slowing of conduction velocities and more advanced neuropathic involvement [12,13]. Importantly, this relationship should be interpreted as reflecting cumulative metabolic burden rather than implying single-factor causality; current multidisciplinary guidance also cautions that neuropathy in a person with diabetes may occasionally reflect non-diabetic etiologies, and emphasizes exclusion of alternative causes when clinical features are atypical [7].

Beyond average glycaemia, emerging literature suggests that glucose variability may contribute to peripheral nerve dysfunction. In outpatients with type 2 diabetes, continuous glucose monitoring–derived variability metrics have been associated with sural sensory conduction velocity measured using point-of-care nerve conduction devices, indicating that dynamic glycaemic exposure may be relevant to large-fiber functional outcomes [14]. Related prospective observational work also supports an association between both long- and short-term glycaemic variability measures and large- and small-fiber dysfunction, suggesting that risk may not be fully captured by mean glycaemia alone [15]. While our study did not directly quantify glucose variability, the observed linkage between nerve conduction impairment, diabetes chronicity,

and glycaemic status is consistent with an overall model of cumulative and fluctuating metabolic injury.

Finally, these findings have practical implications. Evidence from asymptomatic cohorts indicates that measurable nerve conduction abnormalities can be present before overt neuropathic symptoms, creating an opportunity for earlier identification of high-risk individuals and intensification of risk-factor management [16]. Additionally, work correlating electrophysiological abnormalities with structural nerve assessment suggests that tibial nerve involvement often tracks with overall neuropathy burden, supporting the emphasis on lower-limb evaluation in suspected diabetic polyneuropathy [12]. Taken together, the present results reinforce the role of nerve conduction testing as an objective method to characterize diabetic peripheral nerve dysfunction, with particular sensitivity for distal, lower-limb involvement.

Conclusion

The present study demonstrates that type 2 diabetes mellitus is associated with significant impairment of both motor and sensory nerve conduction, with changes predominantly affecting distal and lower limb nerves. The observed electrophysiological abnormalities reflect a length-dependent neuropathic process and are closely linked to disease chronicity and suboptimal glycaemic control. These findings underscore the value of nerve conduction studies as an objective tool for the early detection and evaluation of diabetic peripheral neuropathy, highlighting the importance of timely metabolic control to limit progressive neural dysfunction.

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