

Research Article

Association between Thyroid Hormone Status and Peripheral Nerve Conduction Abnormalities in Adults

Ibrahim Liaqat^{1*}, Khadija Khan², Nasir Siddique³, Saadia Sajjad⁴, Zunaira Adnan⁵, Areeba Mushtaq⁶

^{1*}Assistant Professor, Department of Physiology, Khawaja Muhammad Safdar Medical College, Sialkot, Pakistan.

²Assistant Professor, Department of Physiology, Fatima Jinnah Medical University, Lahore, Pakistan.

³Consultant Physician, Manchester Royal Infirmary, Manchester, United Kingdom.

⁴Professor, Department of Physiology, Abu Umara Medical & Dental College, Green International University, Lahore, Pakistan.

^{5,6}Demonstrator, Department of Physiology, Abu Umara Medical & Dental College, Green International University, Lahore, Pakistan.

Corresponding Author: Dr. Ibrahim Liaqat

Assistant Professor, Department of Physiology, Khawaja Muhammad Safdar Medical College, Sialkot, Pakistan.

Email: ibrahim2002pk@hotmail.com

Received: 24.07.2026, Revised: 11.10.2026, Accepted: 28.10.2026

ABSTRACT

Background: Thyroid dysfunction may affect peripheral nerve physiology through metabolic and structural mechanisms, producing neurological symptoms and measurable electrodiagnostic abnormalities. Comparative evidence across overt and subclinical thyroid disorders remains limited.

Objective: To assess associations between thyroid hormone status and peripheral nerve conduction abnormalities in adults.

Materials and Methods: This analytical cross-sectional study included 150 adults evaluated between January 2025 and January 2026 at the Department of Physiology, Khawaja Muhammad Safdar Medical College, Sialkot, in collaboration with the Department of Physiology, Abu Umara Medical & Dental College, Green International University, Lahore, Pakistan. Participants were categorized into five thyroid function groups. Thyroid status was classified using serum thyroid-stimulating hormone and free thyroxine, with focal median neuropathy evaluated separately. Motor and sensory nerve conduction parameters were compared, and logistic regression assessed associated factors.

Results: The cohort comprised 56 males (37.3%) and 94 females (62.7%). Nonfocal nerve conduction abnormalities occurred in 44 participants (29.3%), including 18 of 32 (56.3%) with overt hypothyroidism and seven of 48 (14.6%) euthyroid participants. Overt hypothyroidism showed slower motor conduction velocities and lower sensory amplitudes. Following adjustment for age, sex, body mass index, and diabetes, overt hypothyroidism was associated with abnormalities (adjusted odds ratio 7.28; 95% confidence interval 2.14-24.80; $p=0.002$). Age and diabetes were additional associated factors.

Conclusion: Overt hypothyroidism showed the strongest association with electrophysiological abnormalities. Nerve conduction assessment may help characterize peripheral nerve involvement in adults presenting with thyroid dysfunction and neurological symptoms.

Keywords: Thyroid Dysfunction, Hypothyroidism, Hyperthyroidism, Peripheral Neuropathy, Nerve Conduction Studies, Electrophysiology.

INTRODUCTION

Thyroid hormones are essential for metabolic homeostasis, cellular energy production, and normal neurological function¹. Thyroid hormones, triiodothyronine (T3) and thyroxine (T4), affect neuronal metabolism, axonal transport, maintenance of the myelin sheath, and neuromuscular activity, and the peripheral nervous system is highly responsive to changes in thyroid hormone levels. Both hypothyroidism and hyperthyroidism are known to have a wide

range of neurological features such as paresthesia, muscle weakness, and changes in tendon reflexes and peripheral neuropathy. The peripheral nerve involvement in thyroid disorders is well known, although the metabolic and cardiovascular involvement is clearer. The possible link between thyroid hormone abnormalities and peripheral nerve dysfunction is thus increasingly being studied, especially for the detection of electrophysiological

abnormalities in patients with various thyroid functional states².

Both localized (focal) and generalized peripheral nerve dysfunction have been linked to hypothyroidism³. A number of mechanisms have been suggested, such as disruption of the metabolism of the axons, decrease in the production of energy from the mitochondria, changes in the function of the myelin, and deposition of glycosaminoglycans in adjacent connective tissue. These alterations are potential causes of decreased nerve conduction velocity, increased distal motor latency and decreased sensory nerve action potential amplitude. Peripheral neurological symptoms may occur gradually and may affect both sensory and motor fibres. The most frequent complaints include sensory symptoms including numbness, tingling, altered distal sensation, while motor symptoms include muscle weakness and impaired neuromuscular performance. Importantly, electrophysiologic abnormalities may precede clinically evident neurologic dysfunction, thus emphasizing the importance of objective nerve conduction studies in thyroid dysfunction^{4,5}.

The link between subclinical thyroid dysfunction and peripheral nerve conduction is not as well understood⁶. Subclinical hypothyroidism is defined as increased thyroid-stimulating hormone (TSH) levels with normal levels of free thyroxine (FT4) in the blood, while subclinical hyperthyroidism is the term for low TSH with normal blood levels of thyroid hormone. These biochemical abnormalities can occur in the absence of any clinical symptoms but have clinical relevance for their possible neurological consequences⁷. Some studies have found that in some cases the peripheral nerve conduction is abnormal in subclinical hypothyroidism, but other studies have shown that there are only slight or insignificant changes in peripheral nerve conduction compared with euthyroid subjects. The disparate results indicate that the correlation of thyroid function with the integrity of peripheral nerves depends on the severity and duration of the thyroid dysfunction, on individual metabolic parameters and on the electrophysiological parameters evaluated^{8,9}.

Peripheral nerve (electro)conduction studies can objectively assess the function of the large myelinated peripheral nerve fibres. These investigations measure distal motor latency, motor nerve conduction velocity, sensory nerve conduction velocity, compound muscle action potential amplitude and sensory nerve action potential amplitude¹⁰. Changes in these

parameters may reflect nerve conduction defects, axon dysfunction, or focal nerve compression. The combination of assessment of several peripheral nerves (median, ulnar, common peroneal, tibial, and sural) allows the assessment of neurological involvement in both upper and lower extremities. In addition, nerve conduction testing can help distinguish the generalized peripheral nerve abnormalities from isolated entrapment neuropathies, important in thyroid disorders¹¹.

An important neurological manifestation of hypothyroidism is carpal tunnel syndrome, which is compression of the median nerve at the wrist¹². In thyroid hormone-deficient individuals, median nerve compression may be caused by accumulation of mucopolysaccharides, connective tissue changes, and increased pressure within the carpal tunnel. But it does not mean that focal abnormalities of the median nerve are necessarily associated with peripheral neuropathy in general. It is therefore necessary to differentiate focal entrapment from nonfocal peripheral nerve dysfunction to allow accurate electrophysiological interpretation. Assessment of sensory and motor parameters on various anatomically distinct nerves may give a more complete picture of thyroid-related peripheral nerve involvement¹³.

Hyperthyroidism can also affect neuromuscular function due to increased metabolic rate, changes in muscle protein turnover, and changes in the excitability of the neurons¹⁴. While muscle weakness of the proximal muscles and thyrotoxic myopathy are more commonly seen, peripheral nerve abnormalities are also seen in persons with an excess of circulating thyroid hormones. The neurologic abnormalities are not necessarily the same in hyperthyroidism as in hypothyroidism, and the extent to which electrophysiology is affected may not necessarily parallel the biochemical abnormalities. Additionally, peripheral nerve conduction measurements may be influenced by other factors such as demographic and metabolic factors – age, gender, body mass index, diabetes mellitus and other comorbid conditions. All these factors should be taken into account when assessing the relationship between thyroid hormone status and neurological function¹⁵.

Although the neurological abnormalities related to thyroid dysfunction are becoming more well known, there are still key questions concerning the prevalence, pattern and magnitude of peripheral nerve conduction abnormalities in

the different thyroid states¹⁶. Previous studies have often focused on whether or not hypothyroidism is present, or on single entrapment neuropathies; comparative studies in euthyroid subjects, subclinical and overt hypothyroidism, and subclinical and overt hyperthyroidism are still quite limited. Some inconsistencies in findings may be due to differences in biochemical classification, electrophysiological methodology, characteristics of the population studied, and control of potential confounding factors. These relationships can be clarified by a comprehensive evaluation including thyroid hormone measures, parameters of motor and sensory nerve conduction and relevant clinical characteristics¹⁷.

The aim of the present study was to assess the relationship between thyroid hormone status and peripheral nerve conduction abnormalities in adults. The main aim of the study was to compare the frequency of nonfocal electrophysiological abnormalities among the euthyroid, subclinical hypothyroid, overt hypothyroid, subclinical hyperthyroid, and overt hyperthyroid groups. Secondary goals were to compare motor and sensory nerve conduction parameters; to assess the demographics and metabolic factors associated with peripheral nerve dysfunction; and to assess the distribution of focal and nonfocal abnormalities. The aim of the study was to promote understanding of the correlation between biochemical status of the thyroid and peripheral nerve function and to give clinically relevant information pertaining to peripheral nerve electrophysiology in adult patients with thyroid disorders.

MATERIALS AND METHODS

Study Design and Setting

This analytical cross-sectional study was conducted from January 2025 to January 2026 at the Department of Physiology, Khawaja Muhammad Safdar Medical College, Sialkot, Pakistan, in collaboration with the Department of Physiology, Abu Umara Medical & Dental College, Green International University, Lahore, Pakistan. The study was conducted to assess the relationship between peripheral nerve conduction abnormalities and thyroid hormone status in adult population. The differences in peripheral nerve function in each thyroid status category were explored by biochemical thyroid function assessment and electrophysiological evaluation.

Study Population and Sampling

The subjects were 150 adult male and female subjects aged 18 years and over. Subjects were classified based on their thyroid hormone levels into euthyroid (normal thyroid hormone levels), subclinical hypothyroid (normal T4 and elevated TSH), overt hypothyroid (low T4 and elevated TSH), subclinical hyperthyroid (normal TSH and elevated T4), and overt hyperthyroid (low TSH and elevated T4) groups. The study population comprised of various thyroid functional states in order to make the comparisons of motor and sensory nerve conduction parameters. A structured data collection form was used to record demographic data, clinical history, anthropometric measurements, and medical conditions.

Inclusion and Exclusion Criteria

All adults ≥ 18 years old who had available thyroid function tests and technically adequate nerve conduction testing were included. Males and females were not discriminated against regardless of thyroid functional category. Patients with a past history of significant peripheral nerve injury, hereditary neuropathy, previous surgery on nerves examined, and neurologic disorders which would significantly affect the interpretation of nerve conduction were excluded. Other known causes of severe peripheral neuropathy, and any participant who was receiving neurotoxic chemotherapy, were also excluded. Potential confounding factors such as diabetes mellitus and hypertension were recorded.

Biochemical Evaluation of Thyroid Function

Venous blood samples were taken for biochemical thyroid function testing. TSH and FT4 were determined by laboratory quality-controlled, validated immunoassay techniques. Thyroid functional status was determined based on the reference intervals of the laboratory performing the thyroid function tests. Euthyroidism was defined as TSH and FT4 levels in normal reference ranges. Subclinical hypothyroidism was defined as elevated TSH and normal FT4 levels, and overt hypothyroidism was defined as elevated TSH and decreased FT4 levels. Suppressed TSH with normal circulating thyroid hormone levels was used to define subclinical hyperthyroidism, and suppressed TSH with elevated FT4 and/or free triiodothyronine levels was used to define overt hyperthyroidism. Appropriate biochemical and clinical data were accounted for to differentiate primary thyroid disease from other diseases that can influence thyroid function tests.

Assessment of Peripheral Nerve Conduction

The peripheral nerve conduction was assessed using standardized electrodiagnostic techniques and included motor and sensory nerve studies. Motor nerve conduction studies were performed for median, ulnar, common peroneal and tibial nerves, while sensory nerve conduction studies were performed for median, ulnar and sural nerves. Motor nerve parameters included distal motor latency, amplitude of the compound muscle action potential, and motor conduction velocity. Sensory nerve measurements consisted of sensory nerve action potential amplitude and sensory nerve conduction velocity. Recorded electrodes were placed on the appropriate surface as per standard electrodiagnostic technique, and supramaximal electrical stimulation was given. Median motor nerve was assessed by recording the abductor pollicis brevis muscle, and ulnar motor responses were assessed by recording the abductor digiti minimi muscle. Common peroneal and tibial motor nerve responses were obtained from the extensor digitorum brevis and the abductor hallucis muscles, respectively. Standardized recording and stimulating sites were used for sensory nerve conduction. The temperature of the limbs were measured during testing to keep temperature effects on DL and CV to a minimum. All of the electrophysiologic data were analyzed based on the laboratory reference values.

Evaluation of peripheral nerve conduction abnormalities.

Distal latency, conduction velocity, and motor and sensory response amplitude were measured and determined to be peripheral nerve conduction abnormalities by reference to electrodiagnostic reference limits. The electrophysiological abnormalities that were considered relevant were prolonged distal motor latency, decreased conduction velocity, and decreased amplitude of the sensory nerve action potential. The difference between nonfocal peripheral nerve involvement and isolated focal entrapment neuropathy was determined by the anatomical distribution of abnormal findings. Sensory-predominant/sensorimotor abnormalities were classified with respect to the nerves and response parameters. Median neuropathy at the wrist was measured separately, and lower limb involvement was assessed by peroneal, tibial and sural nerve measurements.

Clinical and Demographic Assessment

Clinical assessment included age, sex, body mass index, thyroid functional category, diabetes mellitus, hypertension and neurological symptoms. Body mass index was calculated by dividing body weight in kilograms by height in meters squared. Neurological symptoms, such as paresthesias, numbness, tingling sensations, and distal muscle weakness, were recorded in the clinical history. These characteristics were assessed to explore possible associations with peripheral nerve conduction abnormalities and to consider appropriate confounding factors when conducting statistical analyses.

Outcome Measures

The main endpoint was peripheral nerve conduction abnormalities outside of the focal field in all five thyroid functional groups. Secondary outcomes comprised: motor nerve conduction velocity difference, distal motor latency difference, sensory nerve action potential amplitude difference, sensory conduction velocity difference, and distribution of sensory and sensorimotor abnormalities. Other analyses included the study of lower limb nerve involvement, focal median neuropathy at the wrist and association between abnormal peripheral nerve conduction and demographic and metabolic parameters.

Statistical Analysis

IBM SPSS Statistics (Version 26.0) was used for statistical analysis. Continuous variables were presented as mean $\pm$ standard deviation, or median (range) depending on the data distribution, and categorical variables were presented as frequency and percentage. Appropriate parametric or nonparametric tests were used after assessing normality. Continuous variables were analysed between thyroid hormone groups using one-way analysis of variance or Kruskal–Wallis test, and categorical variables were analysed using chi-square or Fisher's exact test, as appropriate. The association of factors with peripheral nerve conduction abnormalities was tested by multivariable binary logistic regression, where the euthyroid group was used as the reference group. Potential confounders were age, gender, BMI, and DM. Crude and adjusted odds ratios (ORs) were calculated and presented with 95% confidence intervals (CIs). All statistical tests were two-sided and a p -value < 0.05 was used to define statistical significance.

Ethical Considerations

The study was carried out in compliance with the ethical standards of the Declaration of Helsinki and the institutional research committee guidelines. Ethical review committee approval and documentation of the informed consent process were done as per the institutional requirements. All participant information was kept confidential and study records were kept with coded identifiers to ensure participant privacy.

RESULTS

Demographic, Clinical, and Biochemical Characteristics

The study included 150 adults, of whom 48 (32.0%) were euthyroid, 35 (23.3%) had subclinical hypothyroidism, 32 (21.3%) had overt hypothyroidism, 20 (13.3%) had

subclinical hyperthyroidism, and 15 (10.0%) had overt hyperthyroidism. The mean age of participants was 47.0 ± 10.8 years, with 56 (37.3%) males and 94 (62.7%) females. Twenty-five (16.7%) subjects had diabetes mellitus, 30 (20.0%) had hypertension and 47 (31.3%) had paresthesia. Age ($p=0.047$), body mass index ($p=0.002$), serum thyroid-stimulating hormone (TSH) and free thyroxine (FT4) levels (both $p<0.001$) were all significantly different between thyroid categories. The prevalence of paresthesia was highest among subjects with clinical hypothyroidism (53.1%), with a significant difference overall ($p=0.018$). Baseline characteristics are shown in Table 1.

Table 1. Baseline Demographic, Clinical and Biochemical Characteristics According To Thyroid Hormone Status

Variable	Euthyroid (n=48)	Subclinical hypo (n=35)	Overt hypo (n=32)	Subclinical hyper (n=20)	Overt hyper (n=15)	p-value
Age, years	45.0 ± 10.8	48.9 ± 10.5	50.8 ± 10.6	45.3 ± 9.0	42.7 ± 12.2	0.047
Male, n (%)	22 (45.8)	10 (28.6)	8 (25.0)	9 (45.0)	7 (46.7)	0.217
Female, n (%)	26 (54.2)	25 (71.4)	24 (75.0)	11 (55.0)	8 (53.3)	0.217
BMI, kg/m ²	26.0 ± 3.9	27.2 ± 3.6	27.1 ± 3.1	24.4 ± 3.6	23.6 ± 3.7	0.002
TSH, mIU/L	2.28 (1.52–2.94)	7.59 (7.01–8.56)	24.19 (20.94–28.04)	0.19 (0.15–0.24)	0.06 (0.05–0.07)	<0.001
FT4, pmol/L	15.0 ± 1.6	14.6 ± 1.3	7.5 ± 1.1	15.0 ± 1.5	27.6 ± 3.4	<0.001
Diabetes mellitus, n (%)	8 (16.7)	5 (14.3)	7 (21.9)	3 (15.0)	2 (13.3)	0.921
Hypertension, n (%)	9 (18.8)	7 (20.0)	9 (28.1)	3 (15.0)	2 (13.3)	0.718
Paresthesia, n (%)	9 (18.8)	12 (34.3)	17 (53.1)	4 (20.0)	5 (33.3)	0.018

Comparison of Motor and Sensory Nerve Conduction Parameters

Motor and sensory nerve conduction measurements demonstrated significant differences across thyroid hormone categories (Table 2). The patients who had clinical hypothyroidism had the most marked electrophysiological abnormalities. Mean median motor distal latency was prolonged in overt hypothyroidism (3.98 ± 0.37 ms) compared with euthyroid participants (3.51 ± 0.37 ms), while median motor conduction velocity was lower (52.11 ± 2.75 versus 56.02 ± 2.69 m/s). There were also significant overall differences found for ulnar, peroneal and tibial motor conduction velocities. The lowest motor

conduction velocity was in overt hypothyroidism (42.00 ± 2.54 m/s), while in euthyroid individuals, it was 45.22 ± 2.46 m/s ($p<0.001$). There was a comparable distribution in the measurements of the sensory nerves. The median sensory amplitude was lower in the group with overt hypothyroidism (18.80 ± 4.45 μ V) than in the group of euthyroid participants (24.95 ± 4.90 μ V). Sural sensory amplitude was also reduced in overt hypothyroidism (10.59 ± 2.78 μ V) compared with euthyroid status (14.20 ± 2.24 μ V). There were significant differences in median, ulnar and sural sensory amplitudes overall ($p<0.001$ for each). Participants with subclinical hypothyroidism demonstrated intermediate electrophysiological

measurements, whereas hyperthyroid groups exhibited comparatively smaller differences from euthyroid participants.

Table 2. Comparison of Motor and Sensory Nerve Conduction Parameters According To Thyroid Hormone Status

Parameter	Euthyroid	Subclinical hypo	Overt hypo	Subclinical hyper	Overt hyper	p-value
Median motor distal latency (ms)	3.51 ± 0.37	3.66 ± 0.37	3.98 ± 0.37	3.43 ± 0.35	3.39 ± 0.36	<0.001
Median motor velocity (m/s)	56.02 ± 2.69	54.57 ± 2.92	52.11 ± 2.75	56.33 ± 2.51	55.44 ± 2.53	<0.001
Ulnar motor velocity (m/s)	56.99 ± 3.09	55.58 ± 3.12	53.81 ± 2.17	56.34 ± 2.99	56.06 ± 2.17	<0.001
Peroneal motor velocity (m/s)	46.76 ± 3.36	45.40 ± 2.70	44.30 ± 3.83	45.33 ± 2.80	45.25 ± 3.24	0.023
Tibial motor velocity (m/s)	45.22 ± 2.46	43.96 ± 2.71	42.00 ± 2.54	45.47 ± 2.37	46.13 ± 2.40	<0.001
Median sensory amplitude (µV)	24.95 ± 4.90	22.66 ± 5.54	18.80 ± 4.45	23.82 ± 3.45	23.74 ± 6.88	<0.001
Ulnar sensory amplitude (µV)	23.00 ± 4.41	21.18 ± 5.37	19.18 ± 4.88	23.50 ± 4.48	24.56 ± 4.64	<0.001
Sural sensory amplitude (µV)	14.20 ± 2.24	12.04 ± 3.53	10.59 ± 2.78	14.20 ± 3.03	14.29 ± 3.09	<0.001
Sural sensory velocity (m/s)	47.56 ± 2.96	46.44 ± 4.31	44.06 ± 3.23	48.75 ± 2.63	46.78 ± 3.12	<0.001

Frequency and Distribution of Peripheral Nerve Conduction Abnormalities

Nonfocal peripheral nerve conduction abnormalities were identified in 44 (29.3%) participants, with significant variation across thyroid hormone categories ($p=0.001$). Overt hypothyroidism was most prevalent (18/56.3%), followed by overt hyperthyroidism (5/33.3%) and subclinical hypothyroidism (11/31.4%). Similarly, 7 (14.6%) euthyroid and 3 (15.0%) subclinical hyperthyroid subjects were found to have abnormalities.

Sensory-predominant abnormalities were found in 29 (19.3%) of the participants, and combined sensorimotor abnormalities were found in 15

(10.0%). The most common abnormalities were sensory-predominant, which were more common in overt hypothyroidism, although there was significant variation between thyroid categories ($p=0.035$). Focal Median Neuropathy at the wrist was detected in 24 (16.0%) individuals and was not statistically different among the groups ($p=0.141$). Nerve involvement of the lower limbs was present in 36 patients (24.0%), and this was especially common in overt hypothyroidism (46.9%) and significant overall ($p=0.005$). The distribution of electrophysiological abnormalities is given in Table 3.

Table 3. Frequency and Distribution of Peripheral Nerve Conduction Abnormalities According To Thyroid Hormone Status

Electrophysiological abnormality	Euthyroid (n=48)	Subclinical hypo (n=35)	Overt hypo (n=32)	Subclinical hyper (n=20)	Overt hyper (n=15)	p-value
Nonfocal nerve conduction abnormality	7 (14.6)	11 (31.4)	18 (56.3)	3 (15.0)	5 (33.3)	0.001
Sensory-predominant abnormality	5 (10.4)	7 (20.0)	12 (37.5)	2 (10.0)	3 (20.0)	0.035
Sensorimotor abnormality	2 (4.2)	4 (11.4)	6 (18.8)	1 (5.0)	2 (13.3)	0.253
Focal median neuropathy at wrist	5 (10.4)	7 (20.0)	9 (28.1)	1 (5.0)	2 (13.3)	0.141

Lower-limb nerve involvement	6 (12.5)	9 (25.7)	15 (46.9)	2 (10.0)	4 (26.7)	0.005
------------------------------	----------	----------	-----------	----------	----------	-------

Multivariable Logistic Regression Analysis

Multivariable logistic regression demonstrated an overall association between thyroid hormone status and nonfocal peripheral nerve conduction abnormalities after adjustment for age, sex, body mass index, and diabetes mellitus (overall $p=0.008$). Increased odds of abnormal nerve conduction (aOR 7.28; 95% CI 2.14–24.80; $P=0.002$) was significantly associated with overt hypothyroidism when compared with euthyroid participants. The estimated odds for subclinical hypothyroidism were higher, but not statistically significant (aOR 2.61; 95% CI 0.77–8.88; $p=0.125$).

Overt hyperthyroidism was also associated with significantly elevated estimated odds of abnormal nerve conduction, but just failed to reach the standard p value for statistical significance of 0.05 (aOR 4.53; 95% CI 0.99–20.67; $p=0.051$). There was no significant association with the outcome (aOR 1.18; 95% CI 0.25–5.67; $p=0.833$). Increasing age was independently associated with abnormal nerve conduction (aOR 1.08 per year; 95% CI 1.03–1.13; $p<0.001$), as was diabetes mellitus (aOR 4.29; 95% CI 1.49–12.33; $p=0.007$). Sex and BMI were not significant independent factors. The results of regression are shown in Table 4.

Table 4. Logistic Regression Analysis of Factors Associated with Peripheral Nerve Conduction Abnormalities

Predictor	Crude OR (95% CI)	Adjusted OR (95% CI)	p-value
Euthyroid	Reference	Reference	—
Subclinical hypothyroidism	2.68 (0.92–7.85)	2.61 (0.77–8.88)	0.125
Overt hypothyroidism	7.53 (2.60–21.80)	7.28 (2.14–24.80)	0.002
Subclinical hyperthyroidism	1.03 (0.24–4.48)	1.18 (0.25–5.67)	0.833
Overt hyperthyroidism	2.93 (0.77–11.18)	4.53 (0.99–20.67)	0.051
Age (per year)	—	1.08 (1.03–1.13)	<0.001
Female sex	—	0.83 (0.34–2.00)	0.675
BMI (per kg/m ²)	—	0.99 (0.88–1.12)	0.872
Diabetes mellitus	—	4.29 (1.49–12.33)	0.007

Peripheral nerve conduction abnormalities differed across thyroid hormone categories, with the highest frequency observed in overt hypothyroidism. Distal latency of median motor nerve, motor nerve conduction velocity, and reduced sensory nerve amplitude were prominent electrophysiological findings in this group. After adjustment for metabolic and demographic factors, overt hypothyroidism was the most significant independent predictor of nonfocal peripheral nerve conduction abnormalities. After adjustment, increasing age and diabetes mellitus were other independent predictors, and associations with subclinical hypothyroidism and hyperthyroidism were not significant. These findings suggest that peripheral large fiber nerve function is related to thyroid hormone status, especially in those with overt hypothyroidism.

DISCUSSION

The present study examined the association between thyroid hormone status and peripheral nerve conduction abnormalities among 150 adults¹. The principal finding was a higher frequency of electrophysiological abnormalities

among participants with overt hypothyroidism compared with euthyroid individuals (56.3% versus 14.6%). After controlling for demographic and metabolic parameters, abnormal nerve conduction was most strongly associated with overt hypothyroidism (aOR 7.28; 95% CI 2.14–24.80). These in turn indicate that thyroid hormone deficiency could be linked to peripheral nerve dysfunction, especially in those with overt biochemical hypothyroidism^{2,3}.

There were significant differences between the various thyroid categories in terms of motor and sensory nerve conduction parameters. Motor distal latency was prolonged in participants with overt hypothyroidism, as well as the motor and sensory nerve conduction velocities, and the amplitude of the sensory nerve action potential were decreased⁴. These changes could be interpreted as a decrease in metabolism of the axons, a decrease in availability of cellular energy and a change in peripheral nerve myelin function as a result of the lack of thyroid hormone. The lower sural and median sensory amplitude is indicative of sensory nerve

involvement, which could be an important electrophysiological feature of hypothyroidism⁵. A higher frequency of nerve conduction abnormalities was seen in subclinical hypothyroidism compared to the euthyroid state; this relationship, however, was not statistically significant after multivariable adjustment⁶. Similarly, the estimated odds of overt hyperthyroidism were also raised, but the difference was not statistically significant at the conventional level. These results suggest that thyroid functional category may be a determinant of the association between thyroid dysfunction and peripheral nerve abnormalities. Demographic and metabolic factors were also found to be independent predictors: age and diabetes mellitus^{7,8}.

There was no significant difference in the frequency of focal median neuropathy at the wrist between thyroid groups, but it was more common in overt hypothyroidism⁹. It is important to distinguish focal nerve compression from more widespread peripheral nerve involvement based on this observation. The results are in favor of complete electrophysiological evaluation for patients with thyroid disorders who have peripheral neurological symptoms¹⁰⁻¹⁴.

There are some limitations in the study. The cross-sectional design does not allow for causal inferences to be made, and numbers in each thyroid category are relatively small and may have limited statistical accuracy¹⁵⁻¹⁷. In addition, nerve conduction studies mainly evaluate large myelinated nerve fibers and are not always able to detect isolated small-fiber dysfunction. However, the ability to assess several motor and sensory nerves and to control for appropriate confounding variables offers a systematic review of the association between thyroid hormone status and peripheral nerve conduction¹⁸⁻²⁰.

CONCLUSION

Peripheral nerve conduction abnormalities including decreased sensory amplitudes, reduced motor conduction velocity, and increased distal motor latency were more common in overt hypothyroidism. The other independent factors were increasing age and diabetes mellitus; the relationships with subclinical thyroid dysfunction and hyperthyroidism were less clear. The results are indicative of the possible clinical importance of electrophysiology of peripheral nerves in adults with thyroid dysfunction, especially in adults with neurological symptoms.

Author Contributions: IL: Conceptualization, study design, supervision, and manuscript drafting; KK: Methodology, data analysis, and interpretation; NS: Data collection, statistical analysis, and critical revision; SS: Literature review, interpretation of findings, and manuscript review; ZA: Data acquisition, organization, and manuscript preparation; AM: Data collection, validation, and manuscript editing. All authors reviewed and approved the final manuscript and agreed to be accountable for its content.

Conflict of Interest: The authors declare no conflicts of interest.

Funding: No external funding was received.

Data Availability: Study data are available from the corresponding author upon reasonable request, subject to ethical and institutional restrictions.

Acknowledgments: The authors acknowledge the institutional staff and participants for their cooperation and support.

REFERENCES

1. Luo Y, Yao G, Song C, Liao C, Zhang H, Liao C, et al. Thyroid diseases and risk of peripheral neuropathy in a large population-based cohort: evidence from the UK Biobank. *Front Endocrinol (Lausanne)*. 2026;17:1828737. doi:10.3389/fendo.2026.1828737.
2. Abuzinadah AR. Effect of hypothyroidism on the risk of carpal tunnel syndrome and electrodiagnostic parameters. *Neurol Int*. 2025;17(9):150. doi:10.3390/neurolint17090150.
3. Duan X, Zhang T, Wang K. Causal relationship between hypothyroidism and peripheral neuropathy: a Mendelian randomization study of European ancestry. *Front Endocrinol (Lausanne)*. 2024;15:1436823. doi:10.3389/fendo.2024.1436823.
4. Bazika-Gerasch B, Kumowski N, Enax-Krumova E, Kaisler M, Eitner LB, Maier C, et al. Impaired autonomic function and somatosensory disturbance in patients with treated autoimmune thyroiditis. *Sci Rep*. 2024;14:12358. doi:10.1038/s41598-024-63158-w.
5. Brzozowska MM, Banthia S, Thompson S, Narasimhan M, Lee J. Severe hypothyroidism complicated by myopathy and neuropathy with atypical demyelinating features. *Case Rep Endocrinol*. 2021;2021:5525156. doi:10.1155/2021/5525156.

6. Zhu FF, Yang LZ. The association between the levels of thyroid hormones and peripheral nerve conduction in patients with type 2 diabetes mellitus. *Exp Clin Endocrinol Diabetes*. 2018;126(8):493-504. doi:10.1055/a-0635-0826.
7. Gupta N, Arora M, Sharma R, Arora KS. Peripheral and central nervous system involvement in recently diagnosed cases of hypothyroidism: an electrophysiological study. *Ann Med Health Sci Res*. 2016;6(5):261-266. doi:10.4103/amhsr.amhsr_39_16.
8. Karne SS, Bhalerao NS. Carpal tunnel syndrome in hypothyroidism. *J Clin Diagn Res*. 2016;10(2). doi:10.7860/JCDR/2016/16464.7316.
9. Chen S, Andary M, Buschbacher R, Del Toro D, Smith B, So Y, et al. Electrodiagnostic reference values for upper and lower limb nerve conduction studies in adult populations. *Muscle Nerve*. 2016;54(3):371-377. doi:10.1002/mus.25203.
10. Oktayoglu P, Nas K, Kilinc F, Tasdemir N, Bozkurt M, Yildiz I. Assessment of the presence of carpal tunnel syndrome in patients with diabetes mellitus, hypothyroidism and acromegaly. *J Clin Diagn Res*. 2015;9(6). doi:10.7860/JCDR/2015/13149.6101.
11. Shiri R. Hypothyroidism and carpal tunnel syndrome: a meta-analysis. *Muscle Nerve*. 2014;50(6):879-883. doi:10.1002/mus.24453.
12. Garber JR, Cobin RH, Gharib H, Hennessey JV, Klein I, Mechanick JL, et al. Clinical practice guidelines for hypothyroidism in adults: cosponsored by the American Association of Clinical Endocrinologists and the American Thyroid Association. *Thyroid*. 2012;22(12):1200-1235. doi:10.1089/thy.2012.0205.
13. Eslamian F, Bahrami A, Aghamohammadzadeh N, Niafar M, Salekzamani Y, Behkamrad K. Electrophysiologic changes in patients with untreated primary hypothyroidism. *J Clin Neurophysiol*. 2011;28(3):323-328. doi:10.1097/WNP.0b013e31821c30d9.
14. Nebuchennykh M, Løseth S, Mellgren SI. Aspects of peripheral nerve involvement in patients with treated hypothyroidism. *Eur J Neurol*. 2010;17(1):67-72. doi:10.1111/j.1468-1331.2009.02743.x.
15. Kecici H, Degirmenci Y. Hormone replacement therapy in hypothyroidism and nerve conduction study. *Neurophysiol Clin*. 2006;36(2):79-83. doi:10.1016/j.neucli.2006.04.001.
16. El-Salem K, Ammari F. Neurophysiological changes in neurologically asymptomatic hypothyroid patients: a prospective cohort study. *J Clin Neurophysiol*. 2006;23(6):568-572. doi:10.1097/01.wnp.0000231273.22681.0e.
17. Duyff RF, Van den Bosch J, Laman DM, Potter van Loon BJ, Linssen WHJP. Neuromuscular findings in thyroid dysfunction: a prospective clinical and electrodiagnostic study. *J Neurol Neurosurg Psychiatry*. 2000;68(6):750-755. doi:10.1136/jnnp.68.6.750.
18. Misiunas A, Niepomniszcze H, Ravera B, Faraj G, Faure E. Peripheral neuropathy in subclinical hypothyroidism. *Thyroid*. 1995;5(4):283-286. doi:10.1089/thy.1995.5.283.
19. Ozata M, Ozkardes A, Corakci A, Gundogan MA. Subclinical hypothyroidism does not lead to alterations either in peripheral nerves or in brainstem auditory evoked potentials (BAEPs). *Thyroid*. 1995;5(3):201-205. doi:10.1089/thy.1995.5.201.
20. Beghi E, Delodovici ML, Bogliun G, Crespi V, Paleari F, Gamba P, et al. Hypothyroidism and polyneuropathy. *J Neurol Neurosurg Psychiatry*. 1989;52(12):1420-1423. doi:10.1136/jnnp.52.12.1420.