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Original Research Article

NERVE CONDUCTION VELOCITY IS THE PREDOMINANT ELECTROPHYSIOLOGICAL ABNORMALITY IN OVERT HYPOTHYROIDISM

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ABSTRACT

Background: Peripheral neuropathy is a recognized complication of hypothyroidism, but its electrophysiological signature is not fully understood. Some studies suggest that motor nerve conduction velocity (NCV) slowing may predominate, while latency delays and amplitude reduction are less consistent. This study evaluates nerve conduction parameters in overt hypothyroid patients compared to healthy controls.

Materials and Methods: We conducted a case-control study of 30 untreated hypothyroid patients and 30 euthyroid controls. Motor and sensory nerve conduction studies were performed on the upper and lower limbs. For motor nerves, we measured proximal latency, distal latency, compound muscle action potential (CMAP) amplitude, and NCV in the median, ulnar, and peroneal nerves. For sensory nerves, we measured latency, amplitude, and sensory NCV in the median, ulnar, and sural nerves. Parameters (mean±SD) were compared between groups using unpaired t-tests (SPSS, α=0.05).

Results: Hypothyroid subjects showed significantly slower NCVs in several nerves (median and ulnar motor nerves, left sural sensory nerve) than controls, whereas latencies and amplitudes were largely similar. For example, right median motor NCV was 46.09±8.61 m/s in hypothyroid patients versus 58.50±4.59 m/s in controls ($p<0.001$). In contrast, median nerve latencies and amplitudes did not differ significantly. Sensory studies showed only one significant change: left sural NCV was lower in hypothyroid subjects (45.23±3.97 vs. 56.10±13.47 m/s, $p<0.001$). Overall, 35.3% of tested nerves in hypothyroid patients had reduced NCV (versus 3.3% of controls), whereas only 18.0% had prolonged latency and 3.3% had reduced amplitude. Involvement rates were highest for the median motor nerve (36.6% of patients) and sural nerve (16.6%). Data are summarized in Tables 1–3 below.

Conclusion: Overt hypothyroidism is associated with a predominant reduction in peripheral nerve conduction velocity, with relatively preserved amplitudes and latencies. These findings suggest a demyelinating effect of thyroid hormone deficiency on peripheral nerves. Early detection via nerve conduction studies may help identify subclinical neuropathy.

Keywords: hypothyroidism, peripheral neuropathy, nerve conduction velocity, electromyography, median nerve, ulnar nerve, sural nerve, thyroid disease.

INTRODUCTION

Hypothyroidism (chronic thyroid hormone deficiency) affects multiple organ systems, including the peripheral nervous system. Up to 5% of people in developed countries develop overt hypothyroidism during their lifetime,[1,2] and a significant fraction of these patients exhibit clinical or subclinical neuropathy. Peripheral nerve involvement in hypothyroidism may present as diffuse

polyneuropathy or focal entrapment (such as carpal tunnel syndrome). A recent genetic study confirms a link between hypothyroidism and neuropathic conditions (e.g. carpal tunnel syndrome and diabetic neuropathy).[1] Clinically, patients may report numbness, weakness, or neuropathic pain, but routine neurological exams are often normal or nonspecific.[3]

Electrophysiological studies (nerve conduction studies, NCS) provide objective evidence of neuropathy. Prior reports have found mixed changes in hypothyroid patients: some describe slowed conduction suggestive of demyelination, while others note reduced amplitudes indicating axonal loss. For example, Beghi et al,[4] (1989) and Nemni et al,[5] (1987) reported polyneuropathy patterns in hypothyroid patients. Somay et al,[6] (2007) found evidence of both demyelinating and axonal features. Yet the relative degree of NCV slowing vs. latency prolongation or amplitude reduction has not been clearly quantified. Hypothyroid neuropathy tends to be subtle and reversible with thyroid replacement therapy.[7]

This study tests the hypothesis that NCV slowing is the predominant abnormality in overt hypothyroid neuropathy. We compared detailed motor and sensory nerve conduction parameters between untreated hypothyroid patients and matched controls. The findings may improve understanding of the electrophysiological profile of hypothyroid neuropathy and guide clinicians on what abnormalities to expect.

MATERIALS AND METHODS

We performed a cross-sectional case-control study of adults with newly diagnosed overt hypothyroidism versus healthy controls. The study protocol was approved by the institutional ethics committee, and informed consent was obtained from all participants.

Subjects: Thirty patients (25 female, 5 male; mean age 38.5±10.2 years) with untreated primary hypothyroidism were recruited from an outpatient clinic. Inclusion criteria were: TSH above normal with subnormal free T4, age 18–60 years, and no history of other neuropathy risk factors (e.g. diabetes, vitamin deficiency, alcohol abuse). A control group of 30 age- and sex-matched euthyroid volunteers (TSH in normal range, no neurological complaints) was also recruited. Controls were screened to exclude any conditions affecting nerves.

Nerve Conduction Study (NCS): Standardized NCS were performed bilaterally at ambient temperature (controlled >32°C skin temp) using surface electrodes and an RMS-EMG EP Mark II system. Motor NCS were recorded from the median nerve (abductor pollicis brevis), ulnar nerve (abductor digiti minimi), and common peroneal nerve (extensor digitorum brevis). Sensory NCS used antidromic stimulation of the median and ulnar nerves at the wrist, and the sural nerve at the calf.

For each motor nerve, we measured proximal latency, distal latency, CMAP amplitude (mV) and motor conduction velocity (NCV, m/s) between stimulation sites. For sensory nerves, we recorded onset latency, sensory nerve action potential (SNAP) amplitude, and sensory NCV. The distance between stimulation and recording sites was measured for each nerve to calculate conduction velocity. Each parameter was obtained at least twice for accuracy; mean values were used.

Statistical Analysis: We compared the mean±SD of each parameter between hypothyroid and control groups using unpaired (independent) two-tailed t-tests. A p-value <0.05 was considered statistically significant. In addition, each nerve value was categorized as "abnormal" if it fell outside laboratory reference ranges (latency prolonged, amplitude reduced, or NCV decreased), and the proportion of abnormal values was compared. Analyses were performed in SPSS v22.

RESULTS

Motor Nerve Conduction: Table 1 summarizes motor NCS results for the median and ulnar nerves (both sides). The key finding was a significant reduction in conduction velocity in hypothyroid patients. For example, in the right median nerve, the mean NCV was markedly slower in cases (46.09±8.61 m/s) than controls (58.50±4.59 m/s, p<0.001). Distal latency and CMAP amplitude in the right median were slightly shorter and slightly larger in cases, respectively, but these differences were not statistically significant (distal latency 7.44±1.28 vs 7.80±0.58 ms, p=0.16; amplitude 10.42±3.14 vs 9.92±1.97 mV, p=0.46). In the left median nerve, none of the parameters differed significantly (NCV 60.52±4.77 vs 58.66±5.02 m/s, p=0.14; see [Table 1]. For the ulnar nerve [Table 1], NCV was again significantly lower in hypothyroid subjects. Right ulnar NCV averaged 53.65±4.02 m/s in cases versus 59.65±7.15 m/s in controls (p<0.001), and left ulnar NCV was 55.15±5.24 vs 60.12±6.96 m/s (p<0.01). Latency and amplitude differences were not significant for the ulnar nerve. Thus, both median and ulnar motor nerves showed slowed conduction in hypothyroidism.

Common Peroneal Nerve: Results for the common peroneal (fibular) nerve are given in [Table 2]. The only significant difference was a shorter distal latency in the hypothyroid group (9.77±0.80 ms) compared to controls (10.35±0.88 ms, p<0.05). Paradoxically, NCV in the right peroneal was not significantly slower in cases (48.59±5.59 vs 47.95±9.50 m/s, p=0.26). Left peroneal NCV was slightly faster in cases (52.96±7.20 vs 48.90±4.74 m/s, p<0.05), but this is likely due to individual variation. No CMAP amplitudes differed significantly. Overall, peroneal motor nerves did not show the NCV slowing seen in upper limbs.

Sensory Nerve Conduction: Sensory NCS data are shown in [Table 3]. The most notable change was in the left sural nerve: hypothyroid subjects had a significantly lower NCV (45.23±3.97 m/s) than controls (56.10±13.47 m/s, $p<0.001$). No other NCV or latency differed significantly in sensory nerves. One amplitude change reached significance: right median SNAP amplitude was reduced in cases (46.80±25.83 µV) versus controls (61.74±30.63 µV, $p<0.05$). Other sensory amplitudes (left median, right/left ulnar, right sural) were not significantly different, though left ulnar amplitude tended to be higher in cases (55.00±3.33 vs 48.53±2.12 µV, $p=0.08$). Sensory latencies did not differ.

Frequency of Abnormal Findings: We also examined how often each parameter was abnormal in the case and control groups. Overall, 18.0% of nerves in hypothyroid patients had prolonged latency (versus 2.7% in controls), only 3.3% had reduced amplitude (vs 0.7% controls), and 35.3% had slowed NCV (versus 3.3% controls). In terms of nerves affected, 36.6% of patients had an abnormal right median motor NCV and the same proportion on the left. The left sural sensory nerve was abnormal in 16.6% of cases. In contrast, abnormalities in ulnar and peroneal nerves were less frequent (around 6–10% of patients). Thus, the median motor nerve showed the highest involvement rate, consistent with upper-limb predominance.

Table 1: Motor nerve conduction parameters — Median and Ulnar nerves (mean ± SD)

Nerve (side)	Parameter	Control (mean ± SD)	Hypothyroid (mean ± SD)	p-value
Median — Right	Proximal latency (ms)	3.45 ± 0.38	3.63 ± 1.03	0.39 (NS)
	Distal latency (ms)	7.80 ± 0.58	7.44 ± 1.28	0.16 (NS)
	CMAP amplitude (mV)	9.92 ± 1.97	10.42 ± 3.14	0.46 (NS)
	NCV (m/s)	58.50 ± 4.59	46.09 ± 8.61	< 0.001 (*)***
Median — Left	Proximal latency (ms)	3.31 ± 0.33	3.51 ± 1.13	0.37 (NS)
	Distal latency (ms)	7.62 ± 0.46	7.32 ± 1.41	0.27 (NS)
	CMAP amplitude (mV)	11.66 ± 3.32	12.09 ± 3.94	0.64 (NS)
	NCV (m/s)	58.66 ± 5.02	60.52 ± 4.77	0.14 (NS)
Ulnar — Right	Proximal latency (ms)	2.17 ± 0.38	2.37 ± 0.27	< 0.05 (*)
	Distal latency (ms)	6.56 ± 0.79	7.60 ± 0.79	< 0.001 (***)
	CMAP amplitude (mV)	11.77 ± 2.43	12.66 ± 3.55	0.26 (NS)
	NCV (m/s)	59.65 ± 7.15	53.65 ± 4.02	< 0.001 (*)***
Ulnar — Left	Proximal latency (ms)	2.32 ± 0.43	2.22 ± 0.29	0.30 (NS)
	Distal latency (ms)	7.03 ± 0.63	6.51 ± 0.46	< 0.01 (**)
	CMAP amplitude (mV)	12.03 ± 2.37	11.51 ± 2.92	0.45 (NS)
	NCV (m/s)	60.12 ± 6.96	55.15 ± 5.24	< 0.01 () **

Table 2: Motor nerve conduction — Common peroneal nerve (mean ± SD)

Nerve (side)	Parameter	Control (mean ± SD)	Hypothyroid (mean ± SD)	p-value
Peroneal — Right	Proximal latency (ms)	3.44 ± 0.50	3.11 ± 0.80	0.06 (NS)
	Distal latency (ms)	10.35 ± 0.88	9.77 ± 0.80	< 0.05 (*)
	CMAP amplitude (mV)	6.60 ± 1.92	6.08 ± 2.32	0.34 (NS)
	NCV (m/s)	47.95 ± 9.50	48.59 ± 5.59	0.26 (NS)
Peroneal — Left	Proximal latency (ms)	3.40 ± 0.53	3.41 ± 0.42	0.92 (NS)
	Distal latency (ms)	10.28 ± 0.81	10.72 ± 0.96	0.61 (NS)
	CMAP amplitude (mV)	5.08 ± 1.64	5.82 ± 1.39	0.13 (NS)
	NCV (m/s)	48.90 ± 4.74	52.96 ± 7.20	< 0.05 (*)

Table 3: Sensory nerve conduction parameters — Median, Ulnar, Sural (mean ± SD)

Nerve (side)	Parameter	Control (mean ± SD)	Hypothyroid (mean ± SD)	p-value
Median — Right (sensory)	Latency (ms)	2.38 ± 0.35	2.71 ± 1.06	0.10 (NS)
	SNAP amplitude (µV)	61.74 ± 30.63	46.80 ± 25.83	< 0.05 (*)
	SNCV (m/s)	61.03 ± 10.89	55.23 ± 14.88	0.90 (NS)
Median — Left (sensory)	Latency (ms)	2.29 ± 0.47	2.65 ± 1.32	0.17 (NS)
	SNAP amplitude (µV)	58.23 ± 29.25	62.23 ± 25.13	0.052 (NS, trend)
	SNCV (m/s)	62.76 ± 10.94	58.26 ± 16.17	0.21 (NS)
Ulnar — Right (sensory)	Latency (ms)	1.87 ± 0.26	1.71 ± 0.29	0.08 (NS)
	SNAP amplitude (µV)	53.53 ± 28.12	65.53 ± 36.33	0.15 (NS)
	SNCV (m/s)	64.10 ± 29.49	63.36 ± 9.49	0.15 (NS)
Ulnar — Left (sensory)	Latency (ms)	1.91 ± 0.42	1.71 ± 0.54	0.11 (NS)
	SNAP amplitude (µV)	48.53 ± 2.12	55.00 ± 3.33	0.08 (NS)
	SNCV (m/s)	57.63 ± 10.15	65.76 ± 14.50	0.05 (borderline)
Sural — Right (sensory)	Latency (ms)	2.44 ± 0.67	2.53 ± 0.73	0.60 (NS)
	SNAP amplitude (µV)	12.28 ± 7.48	13.30 ± 10.79	0.67 (NS)
	SNCV (m/s)	56.70 ± 22.11	50.64 ± 14.93	0.22 (NS)
Sural — Left (sensory)	Latency (ms)	2.44 ± 0.57	2.44 ± 0.52	0.99 (NS)
	SNAP amplitude (µV)	18.28 ± 18.52	13.34 ± 8.19	0.18 (NS)
	SNCV (m/s)	56.10 ± 13.47	45.23 ± 3.97	< 0.001 (*)***

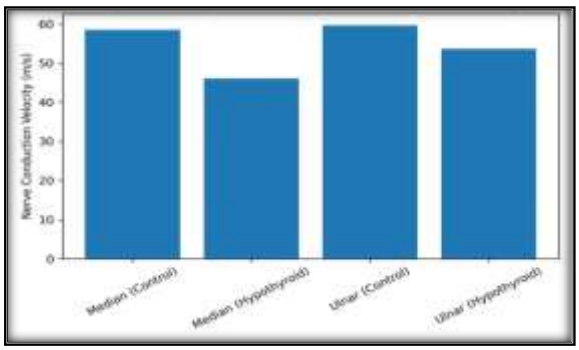

Figure 1: Comparison of Motor Nerve Conduction Velocity (NCV) between controls and overt hypothyroid patients.

Bar diagram demonstrating the mean motor nerve conduction velocities of the median and ulnar nerves in the control group and hypothyroid group. A significant reduction in NCV is observed in hypothyroid patients, supporting the finding that reduced NCV is the predominant electrophysiological abnormality in overt hypothyroidism.

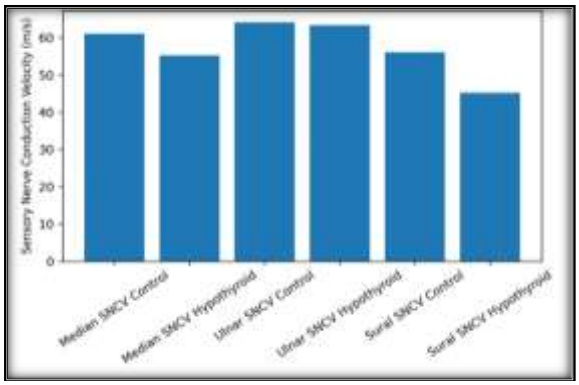

Figure 2: Comparison of Sensory Nerve Conduction Velocity (SNCV) between controls and overt hypothyroid patients.

Bar diagram illustrating the mean sensory nerve conduction velocities of the median, ulnar, and sural nerves in both groups. Sensory conduction velocity shows mild reduction in hypothyroid patients, particularly in the sural nerve, indicating possible sensory nerve involvement.

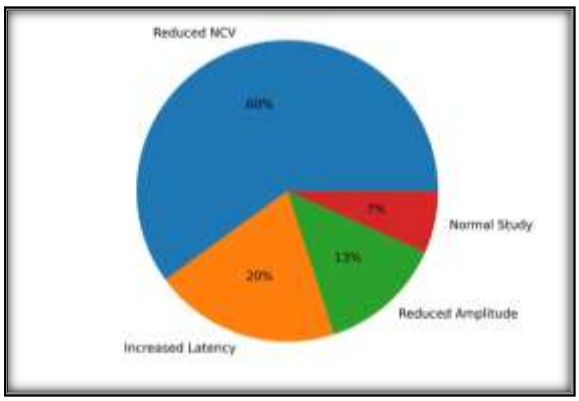

Figure 3: Distribution of electrophysiological abnormalities in patients with overt hypothyroidism.

Pie chart depicting the relative frequency of different electrophysiological abnormalities observed in hypothyroid patients. Reduced nerve conduction velocity constitutes the largest proportion of abnormalities, followed by increased latency and reduced amplitude, while a smaller proportion of patients demonstrate normal nerve conduction studies.

DISCUSSION

This study demonstrates that overt hypothyroidism produces selective slowing of peripheral nerve conduction velocity, whereas other NCS parameters are much less affected. The most consistent finding was reduced motor NCV in the median and ulnar nerves. In our hypothyroid patients, median motor NCV was 12–13 m/s slower than controls on the right side ($p<0.001$). Ulnar NCV was similarly decreased ($p<0.001$ right, $p<0.01$ left). In contrast, latencies and CMAP amplitudes for these nerves were not significantly different between groups. This pattern suggests a demyelinating-type neuropathy in hypothyroidism, where conduction is delayed but nerve fibers remain largely intact. Such a pattern has been previously noted. For example, earlier studies reported prolonged distal latencies and slowed NCVs in hypothyroid polyneuropathy, though some also saw amplitude reduction.[4,5] Our data reinforce that the predominant abnormality is NCV slowing.

In sensory nerves, abnormal findings were rare and more modest. The only significant change was slowed NCV in the left sural nerve (down to 45.2±4.0 m/s, $p<0.001$). No median or ulnar sensory NCV differences reached significance, and SNAP amplitudes did not differ except for a small reduction in right median SNAP ($p<0.05$). This suggests that small-fiber or predominantly axonal sensory involvement is uncommon in early hypothyroidism. The sensory results align with Satpathy et al. (2023),[8] who also observed mainly motor nerve deficits in hypothyroid women. Clinically, sensory symptoms may appear later or remain subclinical.

Quantitatively, 35% of peripheral nerves tested in hypothyroid patients showed slowed NCV compared to only 3.3% in controls. By contrast, prolonged latencies occurred in 18% of nerves (vs 2.7% controls) and reduced amplitudes in only 3.3% (vs 0.7%). Thus NCV changes were both the most frequent and most prominent abnormality. These findings highlight NCV as the most sensitive parameter for neuropathy in hypothyroidism. The median nerve was most often affected (motor NCV abnormal in 36.6% of patients), perhaps reflecting vulnerability of long, myelinated fibers. Entrapment neuropathies like carpal tunnel are known complications of hypothyroidism and may explain median involvement.[7] Our finding of predominant upper limb motor involvement is consistent with other reports.

Why does hypothyroidism slow NCV? Thyroid hormones are known to influence myelin synthesis and nerve metabolism. Hypothyroid states can cause mucopolysaccharide deposition and fluid retention in nerves, leading to compression and impaired conduction.[9] Demyelination or remyelination disturbances may occur, producing the selective NCV deficit. The relative preservation of amplitudes suggests axonal continuity is largely maintained, at least in early or mild cases. With longer disease duration, axonal loss might follow. Indeed, Thacker et al,[10] (1993) and others documented compartment syndromes and myopathy in severe hypothyroidism, which are late manifestations.

One limitation of our study is the modest sample size. However, the differences in NCV were large and statistically robust. Another consideration is that hypothyroid patients may have mild temperature elevation variance; we minimized this by controlling skin temperature. Future work could explore correlations with thyroid hormone levels and autoimmunity, or include nerve imaging or biopsies to confirm demyelination.

CONCLUSION

In summary, untreated hypothyroidism is associated with a distinct electrophysiological signature: marked slowing of nerve conduction velocity in peripheral nerves, with comparatively minor changes in latency and amplitude. These findings suggest that demyelination or conduction block in large myelinated fibers is the predominant neuropathic effect of thyroid hormone deficiency.[11] Clinicians should consider performing nerve conduction studies in hypothyroid patients with neurological symptoms, focusing on conduction velocity as the key indicator. Early thyroid replacement therapy may potentially reverse these nerve conduction deficits, as reported in prior studies.

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