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Original Research Article

# Hypothyroidism Effect on Motor and Sensory Nerve Functions: A Cross-sectional Nerve Conduction Study on Tibial and Sural Nerves

**Sanghamitra Mukherjee[1], Ashmita Sengupta[2], Deepak Chandra[3], Amiya Kumar Sarkar[4], Indira Maisnam[5], Achyut Ghosal[6], Sukanta Sen[7]**

[1]Demonstrator, Department of Physiology, College of Medicine and JNM Hospital, Kalyani, Nadia 741235, West Bengal, India

[2]Associate Professor, Department of Physiology, College of Medicine and Sagore Dutta Hospital, Kamarhati 700058, North 24 Parganas, West Bengal, India

[3]Associate Professor, Department of Anaesthesiology, ICARE Institute of Medical Sciences & Research, Haldia 721645, Purba Medinipur, West Bengal, India

[4]Professor and HOD, Department of Physiology, College of Medicine and Sagore Dutta Hospital, Kamarhati 700058, North 24 Parganas, West Bengal, India

[5]Assistant Professor, Department of Endocrinology, Institute of Postgraduate Medical Education & Research, 244, A. J. C. Bose Road, Kolkata 700020, West Bengal, India

[6]Professor and HOD, Department of Physiology, Santiniketan Medical College, Gobindapur, P.O- Muluk, Bolpur 731204, West Bengal, India

[7]Professor and HOD, Department of Pharmacology, ICARE Institute of Medical Sciences & Research, Haldia 721645, Purba Medinipur, West Bengal, India

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**Corresponding author: Dr. Deepak Chandra**

**Conflict of interest: Nil**

**Abstract**

**Background:** Altered levels of thyroid hormones can impact various body systems, including the nervous system. Hypothyroidism may disrupt nerve conduction due to pathophysiological changes associated with hormone deficiency. To study nerve conduction abnormalities and to correlate electrodiagnostic findings with clinical features in patients with primary hypothyroidism.

**Materials & Methods:** History regarding the duration of the disease, clinical and neurological complaints, use of medicines and level of control of hypothyroidism was recorded in history record sheet. After history taking, general clinical examination was done. Then neurological examination was conducted with special attention. Neuro-MEP-Micro (version 2009) Machine and it's all accessories (Manufactured by Neurosoft Medical Diagnostics Limited, Ivanova, Russia). Electrophysiological parameters like nerve conduction study parameters (latency, amplitude, conduction velocity) of bilateral tibial, and sural motor nerves and tibial, and sural sensory nerves were recorded. Biochemical parameters like T3, T4, TSH values were recorded from patients' test reports and from Endocrinology OPD prescriptions.

**Results:** Study results show mean (±SD) of serum T3 was significantly less in cases (5.27±4.83) than that of the control (15.93±26.63) whereas mean (±SD) of TSH was significantly increased in cases (20.09±28.16) than that of the control (2.62±0.93). Similarly, there was significant increase in mean (±SD) of serum FT4 levels in cases (1.66±1.740) as compared to control subjects (1.22±0.37). In lower limbs the mean (±SD) of distal motor latency (DML) of both tibial motor nerves were significantly increased in cases (Right: 6.71±0.77, Left: 6.19±1.16) than that of the control (Right: 4.24±0.69, Left: 4.16±0.74). No significant differences were found in mean (±SD) of compound muscle action potential (CMAP) of both tibial motor nerves than that of the control. Table 6 shows DSL of right sural nerve decreases but DSL of left sural nerve increases with increase in duration of disease. SNAP of right sural shows positive correlation with duration of disease but the findings are not significant (r=0.072, p= 0.479). SNAP of left sural nerve decreases with increase in duration of disease but it is not significant (r= -0.131, p= 0.193). Positive correlation is there in between SNCV of sural nerve with duration of disease in both sides but these relations are not strong and significant (r= 0.016, p=0.875 and r=0.193, p=0.054).

**Conclusion:** In our study we found that there were significant differences of both sensory and motor NCS parameters between cases and control. We found that CMAP, SNAP and MNCV, SNCV were significantly decreased and distal motor and sensory latencies were significantly increased in cases than controls which indicate mixed type of polyneuropathy, both axonal and demyelinating in hypothyroid patients. Therefore, we can conclude that peripheral neuropathy in hypothyroidism due to axonal loss and/or demyelination can be evaluated effectively by nerve conduction studies. So, the hypothyroid patients should be routinely screened by nerve conduction study.

**Keywords:** Hypothyroidism, Nerve Conduction Study, Distal Motor Latency (DML), Distal Sensory Latency (DSL), Sensory Nerve Action Potential (SNAP), Compound Muscle Action Potential (CMAP), Motor Nerve Conduction Velocity (MNCV), Sensory Nerve Conduction Velocity (SNCV), Tibial Nerve, Sural Nerve.
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## Introduction

Hypothyroidism is a clinical condition associated with low levels of thyroid hormones with variably raised TSH. Hypothyroidism is a multi-organ endocrine disorder resulting in neurological dysfunction. The brain, peripheral nerves and muscular systems also are affected in the disease process [1]. In India, approximately one in ten individuals suffers from hypothyroidism, with females experiencing it more frequently than males [2]. Despite the fact that a decrease in thyroid secretion affects nearly all systems of the body, patients with hypothyroidism frequently present with symptoms and signs of neuromuscular dysfunction, sensorimotor axonal neuropathy and other neurological disorders [3].

Polyneuropathy in hypothyroidism has been described in many studies. Mechanism of development of neuropathy in hypothyroidism is not well-understood. Metabolic alterations induced by hypothyroidism are responsible for peripheral neuropathy. Adenosine triphosphate (ATP) deficiency and reduced ATPase activity with decreased N+/K+ pump activity cause alteration in axonal transport leading to peripheral neuropathy. These changes initially damage function in the nerve, and later induce structural alterations. There are studies which reported primary axonal degeneration while some reported demyelination as the predominant feature of neuropathy in hypothyroidism [4-7].

The prevalence of neuromuscular dysfunctions in thyroid disorders was found to be 20%–80% [8]. Usually, hypothyroidism has both central and peripheral nerve involvement [9]. Patients develop the usual manifestations of peripheral neuropathy as loss of reflexes, proximal muscle weakness, numbness, paresthesia, decreased sensations, and slowed muscle contraction and relaxation with prolonged [10]. The severity of peripheral neuropathy correlates with the degree and duration of hormonal deficiency in hypothyroidism patients [8]. A previous study by AG Unnikrishnan et al [11]. showed that, the prevalence of undetected hypothyroidism was almost one-third of their study population and hypothyroid patients were diagnosed for the first time during the study-related screening. This indicates that even as it continues to impair an individual's daily quality of life, work performance, and economic productivity, a considerable proportion of patients do not seek medical attention until the problems are severe enough. Illiteracy and of low socioeconomic status could be the reason of their ignorance regarding the consequences and the complications of delayed or irregular treatment. So, a large portion of hypothyroid cases may go undetected and untreated [11]. Nerve conduction studies have an important role in evaluation of peripheral neuropathies by confirming the clinical suspicion of neuropathy. It can identify the predominant pathophysiology such as axonal or demyelinating, sensory or motor and also the temporal course of the disease i.e. acute, subacute or chronic. Electrodiagnostic studies can provide an objective and quantitative measure of nerve function and also help in predicting the prognosis of neuropathy. Latent subclinical neuropathy in hypothyroidism can also be investigated using electrophysiological study [12].

As neuromuscular dysfunction is associated with hypothyroidism; the nerve conduction parameters are expected to be altered in these patients. Early electrophysiological diagnosis of neuropathy can help the clinicians to determine the extent of disease and the course of the treatment. A very few similar studies have been done in eastern India. On this background we conducted our study.

## Materials and Methods

The Observational Cross-Sectional, Case-control study was conducted at the Department of Physiology, R.G. Kar Medical College in collaboration with department of Endocrinology, R.G. Kar Medical College & Hospital. Clinical cases of primary hypothyroidism (between 20-60 years of age, both Male and Female), diagnosed and referred from the Endocrinology OPD, R.G Kar Medical College and Hospital, Kolkata were included in the study. Approval from the Institutional Ethics Committee was obtained before commencement of the study.

**Criteria for diagnosing primary hypothyroidism:** A normal TSH level excludes primary hypothyroidism. If the TSH is elevated, an unbound T4 level is needed to confirm the presence of clinical hypothyroidism, but T4 is inferior to TSH when used as a screening test, because it will not detect subclinical hypothyroidism. Circulating unbound T3 levels are normal in about 25% of

patients, reflecting adaptive deiodinase responses to hypothyroidism. T3 measurements are, therefore, not indicated. Once clinical or subclinical hypothyroidism is confirmed, the etiology is usually easily established by demonstrating the presence of TPO antibodies [12].

**Inclusion criteria & exclusion criteria of cases:** Clinical cases of primary hypothyroidism aged between 20-60 years, irrespective of gender and duration of disease and referred from the Endocrinology Department of R.G Kar Medical College, Kolkata were considered for the study. Sixty apparently healthy controls were taken. Study subjects were selected as per the following inclusion and exclusion criteria.

**Exclusion Criteria**

- Diabetes Mellitus
- Secondary hypothyroidism
- Alcoholism
- Neuromuscular Disorder
- Leprosy
- Drug Induced /Toxic Neuropathy
- Family H/o Neuropathy
- Malignancy
- HIV
- Liver diseases
- Kidney Disease
- Myopathy
- Pregnancy
- Patients with permanent pacemaker implants
- Patients unwilling to participate in the study

**Inclusion Criteria**

- Primary hypothyroidism
- Aged between 20-60 years
- Males and females
- Diagnosed in the department of Endocrinology, RGKMC&H
- Patients willing to participate in the study

**Sample Size:** One hundred (100)

Sample size is calculated using the formula:

$n = (Z\alpha/2)2\ pq / d^2$

Prevalence of NCS confirmed Neuropathy ranges between 20 – 80%.[6, 9] So, in this study p has been taken to be 50%.

Margin of error (d) has been taken to be 10%

Therefore $n = [(1.96)2*0.5*(1-0.5)]/ (0.1)^2 = 96.04 \sim 96$ (rounded off to 100) where

$Z\alpha/2 = 1.96$, $p = 0.5$, $q = (1-p) = 0.5$, and $d = 0.1$

History regarding the duration of the disease, clinical and neurological complaints, use of medicines and level of control of hypothyroidism was recorded in history record sheet. After history taking, general clinical examination was done. Then neurological examination was conducted with special attention.Neuro-MEP-Micro (version 2009) Machine and it's all accessories (Manufactured by Neurosoft Medical Diagnostics Limited, Ivanova, Russia)

**Study variables, laboratory investigations, and parameters:** Electrophysiological parameters: Nerve conduction study parameters (latency, amplitude, conduction velocity) of bilateral tibial and sural motor and sensory nerves.

Biochemical parameters: T3, T4, TSH values were recorded from patients' test reports and from Endocrinology OPD prescriptions.

The subjects were placed in a supine position at a temperature of 22-24$^{o}$C in the Neurophysiology Laboratory of the Department of Physiology and the procedures were explained. Nerve conduction study was performed by three electrodes: recording electrodes, a reference electrode, and ground electrode. The electrodes (disposable adhesive surface electrode) were placed after cleaning the skin with Neuro-Prep gel to reduce the skin impedance. Nerve conduction study was performed by using Neuro-MEP-Micro (version 2009) Machine. Nerve conduction study parameters of both tibial and sural nerve were recorded in the study. The latency, amplitude, and velocity of motor and sensory conduction were recorded. Distal Motor Latency (DML), Distal sensory Latency (DSL), Sensory Nerve Action Potential (SNAP), Compound muscle action potential (CMAP), Motor nerve conduction velocity (MNCV) and Sensory nerve conduction velocity (SNCV) were evaluated by Belly Tendon Montage and antidromic stimulation respectively.

**Motor Nerve Conduction Study:** Motor NCS was performed by electrical stimulation of a peripheral nerve and recording from a muscle supplied by this nerve, characterized by its latency, amplitude, and conduction velocity. Latency in milliseconds (Ms) is the time from the onset of stimulus to the point of take-off from baseline. It is an index of speed of impulse travel. Size of the response called amplitude (in mV), measured from the baseline to the top of the motor response. Conduction velocity (in M/s) reflects the fastest motor axons. CV(M/s) =Distance(mm) / Proximal Latency–Distal Latency(ms)

**Results**

**Table 1: Anthropometric Characteristics of Study Subjects**

| Variables | Group | N | Mean | Std. Deviation | Std. Error Mean | p- value |
|---|---|---|---|---|---|---|
| Age (years) | Control | 60 | 37.15 | 10.13 | 1.31 | |
| | Case | 100 | 34.77 | 10.61 | 1.06 | 0.160 |
| Height (cm) | Control | 60 | 153.85 | 6.02 | 0.78 | |
| | Case | 100 | 153.73 | 6.39 | 0.64 | 0.905 |
| Weight (kg) | Control | 60 | 60.10 | 12.05 | 1.56 | |
| | Case | 100 | 61.33 | 13.19 | 1.32 | 0.547 |
| BMI | Control | 60 | 25.43 | 4.71 | 0.61 | |
| | Case | 100 | 25.94 | 4.94 | 0.49 | 0.520 |
| WHR | Control | 60 | 0.64 | 0.38 | 0.05 | |
| | Case | 100 | 0.62 | 0.39 | 0.04 | 0.775 |

Table 1 shows that there was no statistically significant difference between control and cases of hypothyroidism in respect to age and other anthropometric parameters like height, weight, body mass index (BMI) and waist circumference to hip circumference ratio (WHR). The study shows the gender distribution of study subjects. Among the study subjects 8% were male.

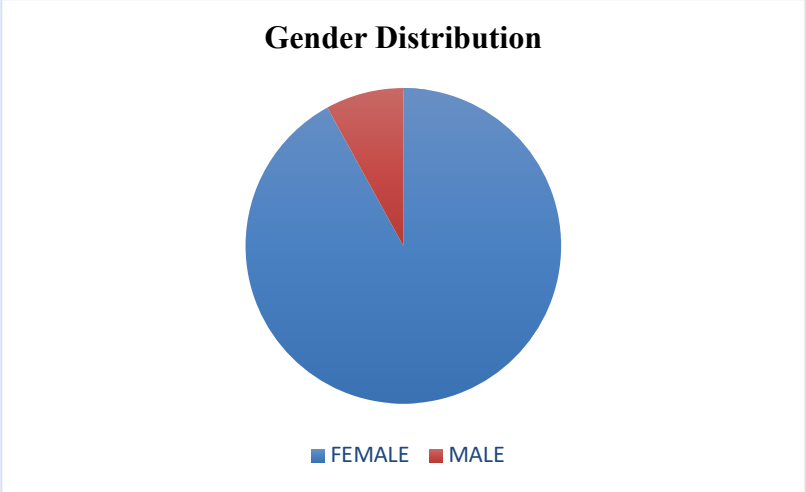



**Figure 1: Showing gender distribution among study subjects**

The pie chart shows the gender distribution of study subjects. Among the study subjects 8% were male [Fig. 1].

**Table 2: Comparison of biochemical parameters among the study subjects**

| | GR | N | Mean | Std. Deviation | Std. Error Mean | P- Value |
|---|---|---|---|---|---|---|
| T3 (ng/L) | Control | 60 | 15.93 | 26.63 | 3.44 | |
| | Case | 100 | 5.27 | 4.83 | 0.48 | 0.001 |
| T4 (µg/dL) | Control | 60 | 8.62 | 1.94 | 0.25 | |
| | Case | 100 | 6.61 | 4.29 | 0.43 | 0.001 |
| FT4 (ng/dL) | Control | 60 | 1.22 | 0.37 | 0.05 | |
| | Case | 100 | 1.66 | 1.74 | 0.17 | 0.05 |
| TSH (mIU/L) | Control | 60 | 2.62 | 0.93 | 0.12 | |
| | Case | 100 | 20.09 | 28.16 | 2.82 | 0.001 |

$P < 0.05$ was considered as level of significance.

Table 2 shows mean (±SD) of serum T3 was significantly less in cases (5.27±4.83) than that of the control (15.93±26.63) whereas mean (±SD) of TSH was significantly increased in cases (20.09±28.16) than that of the control (2.62±0.93). Similarly, there was significant increase in mean (±SD) of serum FT4 levels in cases (1.66±1.740) as compared to control subjects (1.22±0.37) as shown in Table 2.

**Table 3: Comparison of nerve conduction parameters of both tibial nerves in hypothyroid patients and controls**

| Tibial Motor | | Group | N | Mean | Std. Deviation | Std. Error Mean | P- Value |
|---|---|---|---|---|---|---|---|
| DML (ms) | Right | Control | 60 | 4.24 | 0.69 | 0.09 | 0.0001 |
| | | Case | 100 | 6.71 | 0.77 | 0.08 | |
| | Left | Control | 60 | 4.16 | 0.74 | 0.1 | 0.0001 |
| | | Case | 100 | 6.19 | 1.16 | 0.12 | |
| CMAP (µV) | Right | Control | 60 | 7.64 | 2.89 | 0.37 | 0.894 |
| | | Case | 100 | 7.57 | 2.95 | 0.3 | |
| | Left | Control | 60 | 7.8 | 3.21 | 0.41 | 0.992 |
| | | Case | 100 | 7.81 | 3.43 | 0.34 | |
| MNCV (m/s) | Right | Control | 60 | 47.58 | 4.74 | 0.61 | 0.0001 |
| | | Case | 100 | 44.28 | 5.59 | 0.56 | |
| | Left | Control | 60 | 48.18 | 3.03 | 0.39 | 0.0001 |
| | | Case | 100 | 43.86 | 3.7 | 0.37 | |

$p < 0.05$ was considered as level of significance.

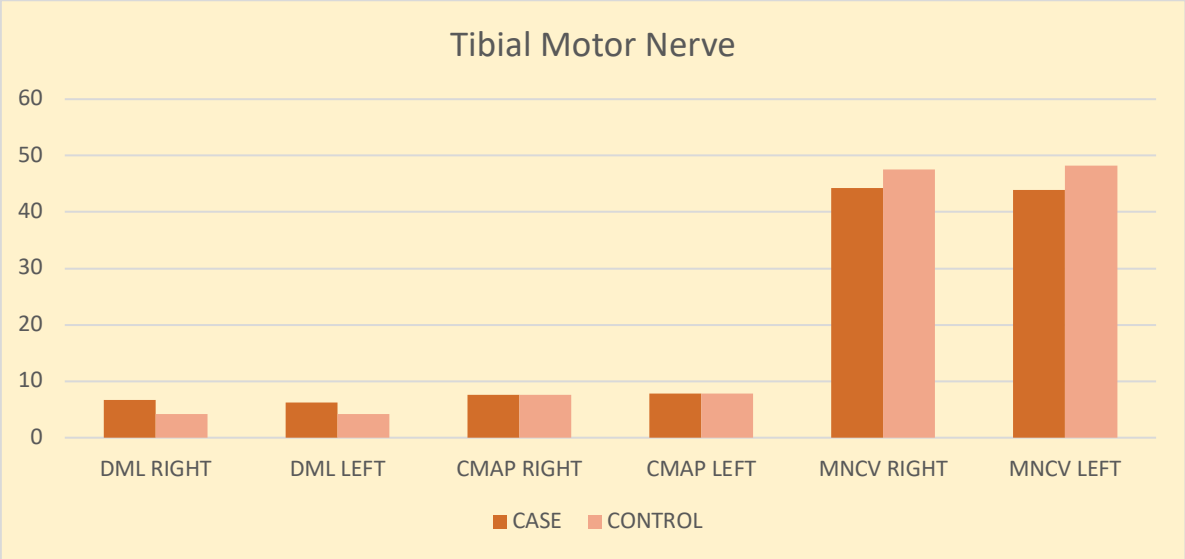



**Figure 2: Showing changes in tibial motor nerve conduction parameters among study subjects**

Table 3 shows the comparison of the latency, amplitude and velocity parameters between cases and controls.

In lower limbs the mean (±SD) of distal motor latency (DML) of both tibial motor nerves were significantly increased in cases (Right: 6.71±0.77, Left: 6.19±1.16) than that of the control (Right: 4.24±0.69, Left: 4.16±0.74). No significant differences were found in mean (±SD) of compound muscle action potential (CMAP) of both tibial motor nerves than that of the control. Though MNCV of both sided tibial nerves in cases (Right: 44.28±5.59, Left: 43.86±3.7) showed significant decrease in their means as compared to controls (Right: 47.58±4.74, Left: 48.18±3.03).

Bar diagram Fig. 2 shows significant increase in DML and decrease in MNCV of bilateral tibial nerves in comparison to control. It also shows that there is no significant difference between CMAP of bilateral tibial nerves with that of the control.

**Table 4: Comparison of nerve conduction parameters of both sural nerves in hypothyroid patients and controls**

| Sural Sensory | | Group | N | Mean | Std. Deviation | Std. Error Mean | P- Value |
|---|---|---|---|---|---|---|---|
| DSL | Right | Control | 60 | 3.69 | 0.38 | 0.05 | 0.0001 |
| (ms) | | Case | 100 | 5.27 | 1.33 | 0.13 | |
| | Left | Control | 60 | 3.63 | 0.34 | 0.04 | 0.0001 |
| | | Case | 100 | 5.19 | 1.74 | 0.17 | |
| SNAP | Right | Control | 60 | 24.39 | 3.56 | 0.46 | 0.0001 |
| (µV) | | Case | 100 | 15.06 | 3.4 | 0.34 | |
| | Left | Control | 60 | 23.57 | 3.9 | 0.5 | 0.0001 |
| | | Case | 100 | 15.33 | 2.89 | 0.29 | |
| SNCV | Right | Control | 60 | 48.69 | 1.87 | 0.24 | 0.0001 |
| (m/s) | | Case | 100 | 20.15 | 7.42 | 0.74 | |
| | Left | Control | 60 | 49.01 | 4.53 | 0.59 | 0.0001 |
| | | Case | 100 | 18.45 | 7.65 | 0.77 | |

$p<0.05$ was considered as level of significance.

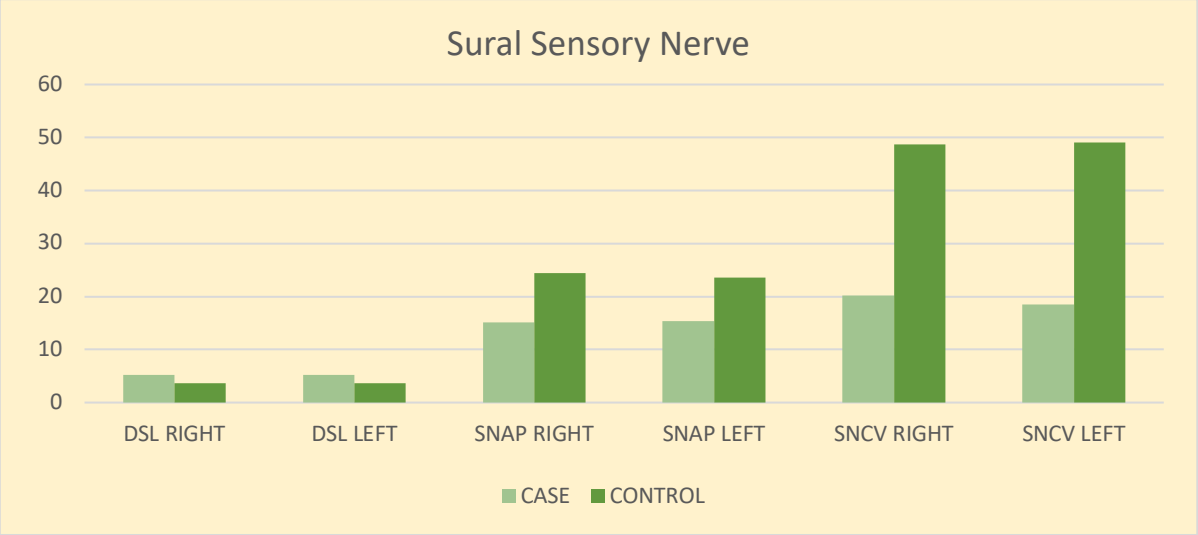


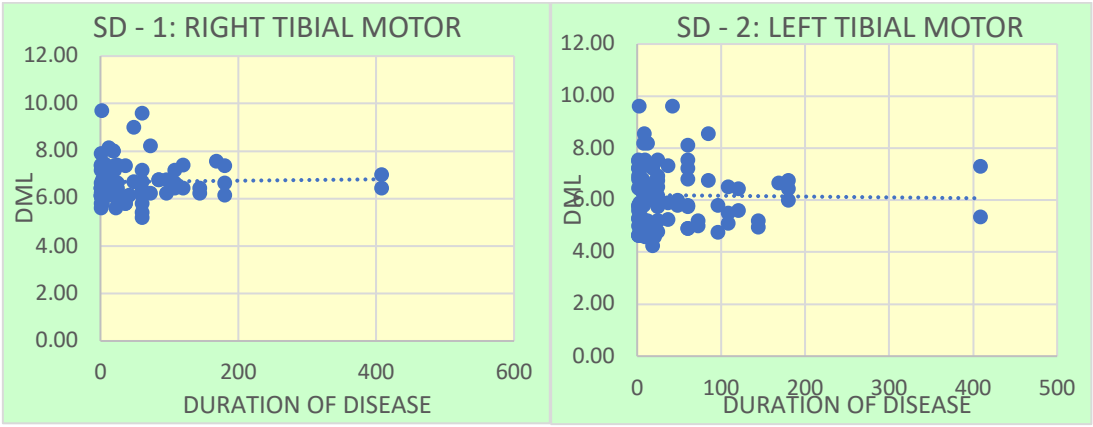

**Fig. 3: Showing changes in sural nerve conduction parameters among study subjects**

Table 4 shows distal sensory latency (DSL) of bilateral sural nerve was significantly increased in cases (Right: 5.27±1.33, Left: 5.19±1.74) than that of the controls (Right: 3.69±0.38, Left: 3.63±0.34). The of SNAP amplitude were reduced significantly in cases (Right: 15.06±3.4, Left: 15.33±2.89) as compared to controls (Right: 24.39±3.56, Left: 23.57±3.9). SNCV were reduced significantly in cases (Right: 20.15±7.42, Left: 18.45±7.65) as compared to controls (Right: 48.69±1.87, Left: 49.01±4.53). The bar diagram Fig. 3 shows significant increase in DSL and significant decrease in SNAP and SNCV of bilateral sural nerves with that of the control.

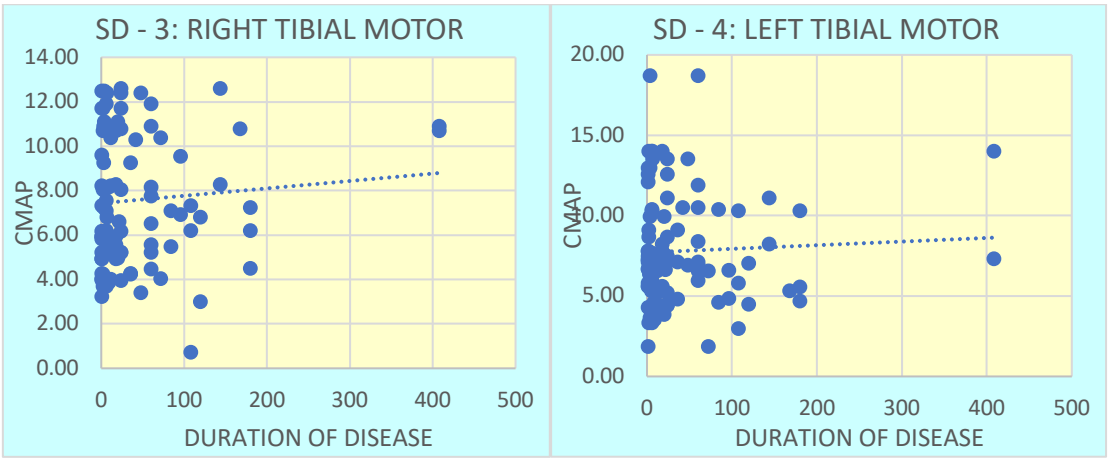
**Table 5: Showing relation between tibial motor NCS parameters with duration of disease**

| Tibial Motor Parameter | | Pearson Co-Relations Coefficient (R) | P-Value |
|---|---|---|---|
| DML (ms) | Right | 0.031 | 0.763 |
| | Left | -0.109 | 0.28 |
| CMAP (µV) | Right | 0.016 | 0.873 |
| | Left | 0.068 | 0.503 |
| MNCV(m/s) | Right | -0.166 | 0.098 |
| | Left | 0.039 | 0.698 |

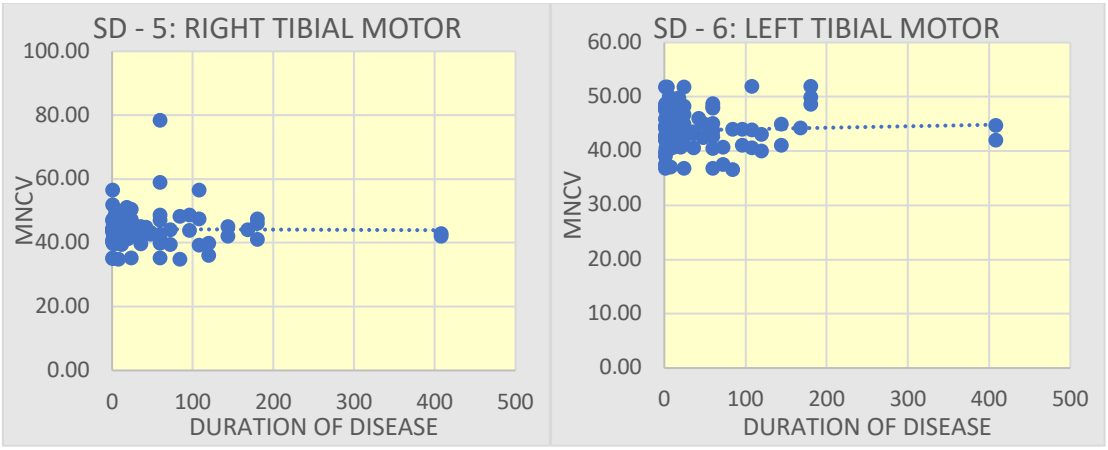
Table 5 shows DML of right tibial nerve increases with duration of disease, but it is not a significant finding (r= 0.031, p=0.763).no significant relation is there with DML of left tibial nerve with duration of disease (r=-0.109, p=0.28). CMAP of bilateral tibial nerves show positive correlation with duration of disease. These findings are not significant (r=0.016, p=0.873 and r =0.068, p= 0.503 of right and left side respectively). Scattered diagrams 01- 06 shows the same findings.




**Scattered Diagram – 1 and 2: Showing relation between distal motor latency of bilateral tibial nerve with duration of disease.**




**Scattered Diagram – 3 and 4: Showing relation between compound muscle action potential of bilateral tibial nerve with duration of disease.**




**Scattered Diagram – 5 and 6: Showing relation between motor nerve conduction velocity of bilateral tibial nerve with duration of disease.**

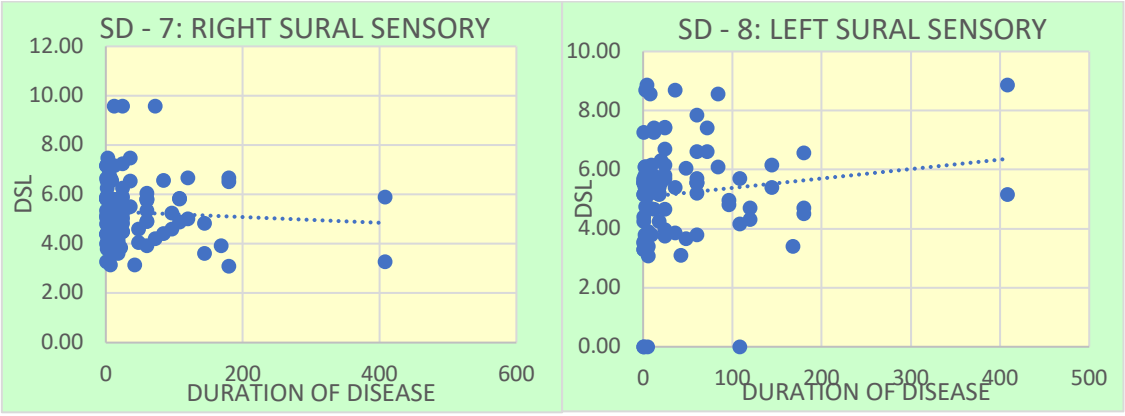
**Table 6: Showing relation between sural NCS parameters with duration of disease**

| Sural Sensory Parameter | | Pearson Co-Relations Coefficient (R) | P-Value |
|---|---|---|---|
| DSL (ms) | Right | -0.148 | 0.141 |
| | Left | 0.018 | 0.859 |
| SNAP (µV) | Right | 0.072 | 0.479 |
| | Left | -0.131 | 0.193 |
| SNCV (m/s) | Right | 0.016 | 0.875 |
| | Left | 0.193 | 0.054 |

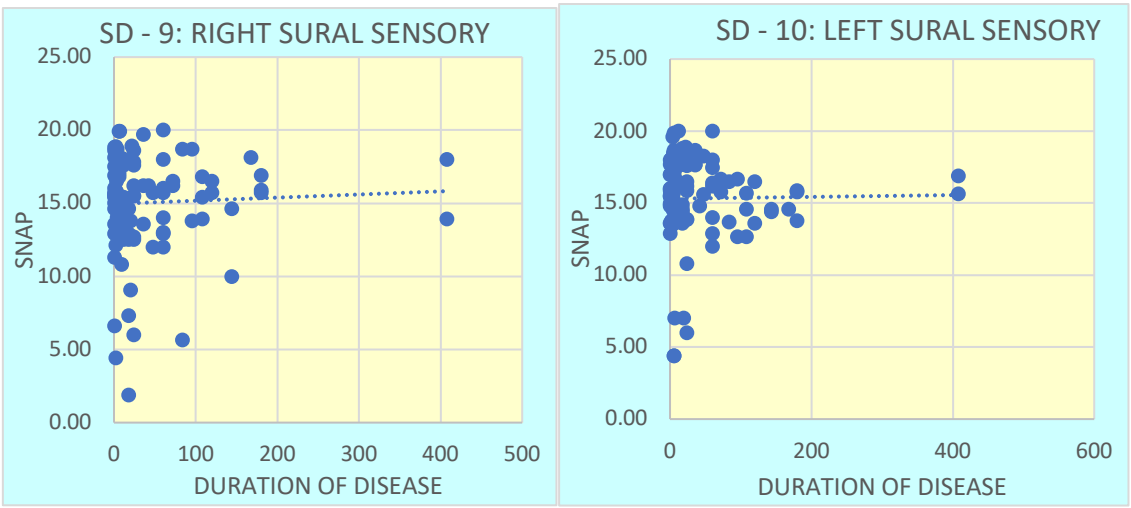
Table 6 shows DSL of right sural nerve decreases but DSL of left sural nerve increases with increase in duration of disease.

SNAP of right sural shows positive correlation with duration of disease but the findings are not significant (r=0.072, p= 0.479). SNAP of left sural nerve decreases with increase in duration of disease but it is not significant (r= -0.131, p= 0.193). Positive correlation is there in between SNCV of sural nerve with duration of disease in both sides but these relations are not strong and significant (r= 0.016, p=0.875 and r=0.193, p=0.054).

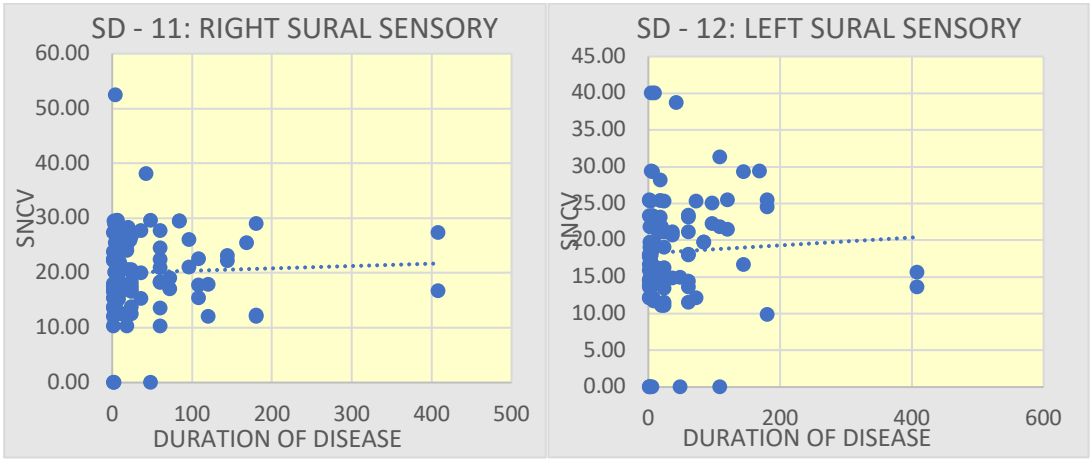
Similar findings are observed in scattered diagrams 07-12.




**Scattered Diagram – 7 and 8: Showing relation between distal sensory latency of bilateral sural nerve with duration of disease.**




**Scattered Diagram – 9 and 10: Showing relation between sensory nerve action potential of bilateral sural nerve with duration of disease.**




**Scattered Diagram – 11 and 12: Showing relation between sensory nerve conduction velocity of bilateral sural nerve with duration of disease.**

**Table 7: Neurological features of hypothyroidism among study subjects**

| Symptoms | Present (%) |
|---|---|
| Diminished Sweating | 35 (N = 35) |
| Dry Skin | 54 (N = 54) |
| Cold Intolerance | 54 (N = 54) |
| Weight Increase | 59 (N = 59) |
| Constipation | 59 (N = 59) |
| Hoarseness | 38 (N = 38) |
| Deafness | 41 (N = 41) |
| Slow Movements | 65 (N = 65) |
| Coarse Skin | 64 (N = 65) |
| Cold Skin | 34 (N = 34) |
| Periorbital Puffiness | 31 (N = 31) |
| Pulse Rate (Bradycardia) | 32 (N = 32) |
| Delayed Ankle Jerk | 57 (N = 57) |
| Tingling Numbness of Limbs | 69 (N=69) |
| Paresthesia | 7 (N=7) |
| Pain And Cramps | 48 (N=48) |
| Weakness | 15 (N=15) |

Table 7 shows that 69% of the patients reported with tingling numbness in limbs,7% of the cases showed paresthesia.48% of them complained of pain in limbs along with cramps in the lower limbs.15% 0f the cases came with weakness and fatigue. It also shows that thirty-five (35%) patients had diminished sweating, fifty-four (54%) patients reported with dry skin, fifty-four (54%) had cold intolerance. Fifty-nine patients (59%) complained of weight gain. 59% patients had complaint of constipation. Thirty-eight patients had hoarseness; 41 patients complained of deafness; 65% patients complained of slow movements; 64% complained of coarse skin; 34% patients complained of cold skin and 31% complained of periorbital puffiness. Thirty-two (32%) of the patients on examination showed bradycardia. Delayed ankle jerk was present in 57% patient.

**Discussion**

In the present study, we evaluated the clinical, biochemical and electro-physiological findings in diagnosed hypothyroid patients. In the study period, 100 cases fulfilled the inclusion and exclusion criteria and participated in the study. Sixty, subjects were taken as controls. We performed the nerve conduction study on the subjects in the neurophysiology laboratory of R.G. Kar Medical College and Hospital under suitable temperature and other conditions.

Our study showed there was no statistically significant difference between control and cases of hypothyroidism in respect to age and other anthropometric parameters (Table 1). Mean ±SD of the age and height was 37.15±10.13(yrs.) and 153.85±6.02(cm) in comparison to 34.77 ± 10.61(yrs.) and 153.73±6.39(cm) in controls, showing non-significant association ($p<0.05$). Our study also showed that mean (±SD) of serum T3 was significantly less in cases (5.27±4.83) than that of the control (15.93±26.63) whereas mean (±SD) of TSH was significantly increased in cases (20.09±28.16) than that of the control (2.62±0.93). Similarly, there was significant increase in mean (±SD) of Serum FT4 levels in cases (1.66±1.740) as compared to control subjects (1.22±0.37). A study by Ruchika Garg et al [13] showed the similar findings.

**Neurological Features:** In our study we found that 69% of the patients reported with tingling numbness in limbs, 7% of the cases showed paresthesia.48% of them complained of pain in limbs along with cramps in the lower limbs. About 15% of the cases came with weakness and fatigue. The prevalence of neurological complications has been reported to be around 79% in hypothyroidism [14].

Our study showed (table 7) that thirty-five (35%) patients had diminished sweating, fifty-four (54%) patients reported with dry skin, fifty-four (54%) had cold intolerance. Fifty-nine patients (59%) complained of weight gain and 59% patients had complaint of constipation. Thirty-eight patients had hoarseness, 41 patients complained of deafness, 65%patients complained of slow movements, 64% complained of coarse skin, 34% patients complained of cold skin, 31% complained of periorbital puffiness. Thirty-two (32%) of the patients on examination showed bradycardia. Delayed ankle jerk was present in 57% patient.

Our findings did not corroborate with the study by Zulewski et al [15]. They showed most frequent in the hypothyroid patients were prolonged ART (77%) and complaints about dry skin (76%). The results of their study demonstrated that the modern laboratory tests for thyroid function have

completely changed the clinical picture of hypothyroidism.

Other studies have shown that, at neurological examination hypo or areflexia was mostly noted (81.2%) followed in frequency by: muscle pain (37.5%), Tinnel sign after percussion of median nerve at the wrist (31.2%), distal hypoesthesia on median nerve distribution (12.5%) and muscle weakness (12.5%). The Achillean reflex was abolished in 31% of the patients and hypoactive in 31%. Biceps, triceps and patellar reflexes were found diminished in 56%, 31% and 25% of the cases respectively [16]. According to our data, the prevalence of the typical hypothyroid symptoms in overt hypothyroidism is remarkably different from that described in the classical, but also in recent, literature [17]. In other studies, the classical symptoms and signs (e.g. coarse skin, cold intolerance, decreased sweating, or puffiness) were still described as having a high frequency of 90–97%, but as in other studies, they observed much less often in their patients (50–64%). These findings also did not corroborate with ours.

**Tibial Nerve:** In our study no significant differences were found in mean (±SD) of compound muscle action potential (CMAP) of both tibial motor nerves than that of the control (table 3). Though MNCV of both sided tibial nerves showed significant decrease in their means as compared to control (table 3). It also showed that there is no significant difference between CMAP of bilateral tibial nerves with that of the control.

Though the study by Ruchika Garg et al [13] found shortening of the amplitudes (CMAPs) in tibial motor nerves when compared to controls and found to be statistically significant. Their study agrees with us in case of significant prolongation of DML and significant increase in MNCV of both the tibial nerves in both sides. However, findings of tibial nerve conduction being significantly affected, didn't agree with the results of other studies [18, 19].

**Sural Nerve:** As per a study by Beghi et al [20] sural nerves were the most commonly affected nerves and 69% of the patients showed abnormal sural nerve conduction velocity. Eslamian et al [21] described significant decrease in amplitude and velocity parameters while significant increase in latency in sural NCS. Ajeena et al [18] also reported high prevalence of sensory neuropathy (44%), where sural mononeuropathy was noted in 43% of the cases. As per a study by Ruchika Et al [13], out of total patients,12.5% had isolated sural neuropathy.

In our study, we have also found that SNAP amplitude and conduction velocity (SNCV) of bilateral sural nerves were reduced significantly as compared to controls.

Our result agreed with the results of other studies [18, 22]. We have found that distal sensory latencies (DSL) of bilateral sural nerve were significantly increased in cases than that of the controls. These results corroborate with the studies done by Ajeena Ihsan M et al [18], Kececi H et al [22] and many other studies.

**Sensorimotor Polyneuropathy:** Most of the studies, reported neuropathies in hypothyroidism were mild and mainly sensory in type [23]. Involvement of sensory nerves may be due to axonal degeneration. It is seen that sensory nerves are affected earlier than motor. Reason though is obscure. A study by Adikesavan Balaraman et al [24] showed that Most of the neuropathy remain latent in the early phase. They have also found that the sensory nerve conduction is decreased in hypothyroidism. There were no changes in motor nerve conduction velocity in their study. Other studies done by Ruud F Duyff et al. [25] and O Malley et al [26] also showed the same findings. A study by Marcia W. Cruz et al [16] showed sensory type polyneuropathy in 68.7% of patients with primary hypothyroidism. However, Eslamian F et al reported that mild sensory type neuropathy was uncommon [21]. As per a study by Schutt et al [27] motor nerve velocity was decreased in hypothyroidism.

Many studies by Ruurd F Duyff et al Dyck and Lambert [28]; Shirabe et al [29]; Pollard et al [30], reported individual cases and showed that slowing of both motor and sensory nerve conduction velocities. Electro physiological evaluation of hypothyroid patients by Somay G. et al [31], Ruchika Garg et al [13] and Yeasmin S.et al [23], have revealed occurrence of sensory-motor polyneuropathy in the cases. We have also found sensorimotor polyneuropathy in the present study so these reports coincide with our findings.

**Association of NCS Parameters with Duration of Disease:** The metabolic changes in hypothyroidism, causing electrophysiological changes may occur early in the course of the disease [18]. Long term accumulation of mucinous tissue is also suggested by Kececi H et al [22] as a possible cause of irreversibility even after hormone replacement therapy. The cause of irreversibility to replacement therapy in hypothyroid patients may be related to differences in illness durations, severity and treatment regimens as per many other studies [19, 21, 22, 32].

As per study by Karne et al [33] occurrence of neuropathy was more common in patients with duration of disease more than 5 years (44.44%) but these findings were not statistically significant (P =

0.08). As per them this relation is possibly due to long-term accumulation of mucinous tissue. This is also supported by Nemni et al [34]. Our study was in agreement with these studies; we also did not find any significant correlation with duration of disease. This might be possibly due the fact that the population of patients are ignorant about their signs and symptoms and disease condition. Often, they do not understand the exact nature of their illness. Some patients receive treatment but discontinue in the course of disease, so they cannot give proper history of the onset of their disease.

**Peripheral Neuropathy:** Peripheral neuropathy can be divided into those that primarily affect axons and those that primarily affect the myelin sheath. Axon loss in hypothyroidism may occur due to metabolic changes in peripheral nerves. Amplitude of compound muscle action potential (CMAP) correlates with the number of motor nerve axons, and similarly, the amplitude of the sensory nerve action potential (SNAP) reflects the number of sensory nerve axons. Lesions causing axon loss generally result in reduced CMAP and SNAP amplitudes90..Whereas loss of myelin is associated with slowing of conduction velocity marked prolongation of distal latencies or both [7, 32]. Primarily axonal type of polyneuropathy in hypothyroid patients was noted by Nemni R Bottachi et al [34]. Evidence of primary axonal degeneration with secondary demyelination has also been reported in some studies [30]. However, in many studies mixed axonal degeneration and demyelinating polyneuropathy was reported [35]. Our findings are in agreement with these findings as we have also observed mixed (axonal and demyelinating) type of neuropathy in the cases.

The probable cause of axonal degeneration could be due to thyroid hormone deficiency which normally increase ATPase activity and, consequently, the activity of the ATP-dependent Na+/K+ pump. The increase in ATPase activity would be associated with an increase of ATP transport through the mitochondrial membranes. In hypothyroidism, the ATP deficiency and the reduced activity of the ATPase enzyme induce a decrease in Na+/K+ pump activity, with consequent alterations of pump-dependent axonal transport. This leads to axonal degeneration and peripheral neuropathy in hypothyroidism [34].

Demylination can occur due to compressive, immune mediated cause or abnormal metabolism in schwan cells or oxidative damage to myelin membrane. The mucinous infiltrations found in the peripheral nerves could interfere mechanically with metabolic exchange of nutrients and catabolic products to and from the neuron resulting in entrapment neuropathy in hypothyroidism [18]. The deposition of mucopolysaccharide or the myxomatous tissue around the peripheral nerves may also lead to its compression and thereby results in swelling and degeneration of those nerves leading to peripheral neuropathy in hypothyroidism [29]. So, in our study we have found mixed (axonal and demyelinating) polyneuropathy. Nemni et al [34] proposed that degeneration of peripheral nerve in hypothyroidism is primarily axonal causing axonal polyneuropathy. This is not in agreement with our findings as we observed mixed type of lesion in the cases.

In our study we found both motor and sensory neuropathy in hypothyroid patients. We found that, this neuropathy is axonal and demyelinating polyneuropathy. Thyroid affects the peripheral nervous system via its role in gene expression, myelin production, its effects on the neurotransmitter system and axonal transportation [36, 37]. Primary axonal degeneration has also been shown electro physiologically.

Initially only functional loss is seen in the nerve, but as the disease progresses, structural modification may happen later [38]. This might be the reason why did we get mixed (demyelinating and axonal) type of polyneuropathy in our patients. Deposition of glycosaminoglycans in nerves and soft tissues surrounding them with resultant axonal degeneration and secondary segmental demyelination forms the pathogenic basis of alterations in peripheral nerve function in thyroid hormone deficiency [39].

## Conclusion

In our study we found that there were significant differences of both sensory and motor NCS parameters between cases and control. We found that CMAP, SNAP and MNCV, SNCV were significantly decreased and distal motor and sensory latencies were significantly increased in cases than controls which indicate mixed type of polyneuropathy, both axonal and demyelinating in hypothyroid patients. Therefore, we can conclude that peripheral neuropathy in hypothyroidism due to axonal loss and/or demyelination can be evaluated effectively by nerve conduction studies. So, the hypothyroid patients should be routinely screened by nerve conduction study.

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