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**Research Article**

# Nerve Conduction Studies, SEP and Blink Reflex Studies in Recently Diagnosed, Untreated Thyroid Disease Patients

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## Abstract

The purpose of this study was to investigate the electrophysiological changes in the peripheral and central nervous system in recently diagnosed subclinical thyroid disease patients before any treatment. This study included 22 patients with hypothyroidism and 13 patients with hyperthyroidism who had no other disease which could affect peripheral or central nervous system. We performed nerve conduction studies, median nevre somatosensorial evoked potentials and blink reflex studies in 35 patients and 30 age and gender matched controls. Distal latency, compound muscle action potential amplitude, nerve conduction velocity, minumum F-latency were recorded in the motor nerve conduction studies, distal latency, sensorial nerve action potential amplitüde, nerve conduction velocity were recorded in the sensorial nerve conduction studies and cortical N20-P25 potential latencies were recorded during the median somatosensorial evoked potential study. Bilaterally R1, R2 and contralaterally R2 potentials were evaluated during the blink reflex studies. In the subclinical hypothyroid patients median motor and sensorial nerves were the most commonly affected nerves with carpal tunnel syndrom being positive in 54,5 % of these patients. Sensorimotor polyneuropathy was find in 8-9% of subclinical hypothyroid patients while this ratio was 15,38% in subclinical hyperthyroid patients. Sural neuropathy was found in 23,07% of subclinical hyperthyroid patients. A decrease in median, ulnar and sural sensorial nerve action potential amplitüdes and tibial motor nerve conduction velocities were the other findings in this group. During the median nerve SEP studies the most common pathological finding in all patients was an increase in P25 cortical latency. This was found in 45,45%

**Keywords:** Polyneuropathy, hyperthyroidism, hypothyroidism

# Tedavi Başlanmamış Tiroid Hastalarında Sinir İletileri, Median Sup Ve Göz Kırpma Refleksi

## Özet

**Amaç:** Tiroid bezi hastalıklarının nöromuskuler disfonksiyona bağlı birçok semptom ve bulguları olabilir. Tiroid hastalıklarına bağlı nörolojik komplikasyonlar, hormonal değişiklikler ya da immün mekanizmalarla meydana gelir (1,19). Hem periferik hem de santral sinir sistemi etkilenebilir. Hastalık bazen bu nörolojik komplikasyonlarla ortaya çıkabileceği ve uygun tedavi ile kolayca düzelebileceği için erken tanı ve değerlendirme çok önemlidir. Bu çalışmanın amacı; yeni tanı almış, henüz tedaviye başlanmamış olan tiroid hastalarındaki periferik ve santral sinir sistemi etkileniminin araştırılmasıdır.

**Çalışma Planı:** Çalışmaya periferik ve santral sinir sistemini etkileyecek başka bir hastalığı olmayan 22 hipotiroidili, 13 hipertiroidili olmak üzere toplam 35 hasta alındı. Elektrofizyolojik inceleme olarak; tek taraflı (sağ) üst ve alt ekstremitede sinir iletimleri, median uyarımlı somatosensoriyel uyandırılmış potansiyeller (SUP), iki taraflı göz kırpma refleksi çalışıldı. Yaş ve cinsiyet açısından eşleştirilmiş 30 sağlıklı birey kontrol grubu olarak alındı, sonuçlar karşılaştırıldı.

**Sonuçlar:** Sinir iletimi incelemesinde; 13 (%59.09) hipotiroidi, 7 (%53,84) hipertiroidili hastada elektrofizyolojik patoloji tespit edildi. Hipotiroidi hasta grubunda; en fazla etkilenen sinir median motor ve duysal sinirlerdi. Karpal tünel sendromu (KTS) 11 (%50) hastada görüldü. Ilımlı düzeyde (%8-9) sensorimotor polinöropati vardı. Hipertiroidide ise nispeten polinöropati daha fazla (%15,4) idi ve sural nöropati ön plandaydı (%23,1). SUP incelemesinde; P25 latans uzaması en belirgin patolojik bulguydu ve hipotiroidide %45,45, hipertiroidide ise %38,46 oranında görülmekteydi. Göz kırpma refleksinde ise ipsi-kontralateral R2 süresindeki uzama en fazla rastlanan anormallikti. Bu da hipotiroidide %36,36 oranında ve hipertiroidide ise %23,07 oranında görülmekteydi.

**Anahtar Kelimeler:** Polinöropati, hipertroidizm, hipotroidizm

## INTRODUCTION

Thyroid hormones are involved in many functions of the central and peripheral nervous system and as a result both hyperthyroidism and hypothyroidism may cause various neurological signs and symptoms [1,11]. The prevelance of neuromusculer disorders related to thyroid dysfunction has been reported to be between 20-80 % [1,7].

Two different types of peripheral nerve abnormalities are associated with hypothyroidism. Although the most common disorders are the entrapment neuropathies, especially carpal tunnel syndrome (CTS), sensorimotor polyneuropathies can also be seen in these patients [15,17]. The severity of the neuromuscular signs and symptoms are known to be related to the duration and degree of hormonal deficiency and clinical, electrophysiological and morphological improvement following hormone replacement therapy is typical [2,14,15,17,23]. Hyperthyroidism is less commonly associated with neuromuscular disorders and polyneuropathy is a relatively rare complication of hyperthyroidism [7,12,15,23].

After a detailed neurological examination the most important investigation to diagnose the neuromuscular complications in electroneuromyography. The evoked potentials (EP) can also be used to evaluate the nervous system function. The metabolic and hormonal changes in hypothyroidism causes central nervous system disturbances in up to 78% of the patients [11,16]. Therefore EP's may be important for demonstrating this effect in asymptomatic.

Most of the electrophysiological studies regarding patients with thyroid diseases are retrospective and involves patients under treatment. The aim of this study was to evaluate the neuromuscular symptoms and signs, as well as the electrophysiological changes in nerve conduction, median nerve somatosensorial evoked potentials (SEP) and blink reflex in patients with recently diagnosed thyroid disease.

## METHODS

Patients consecutively diagnosed of thyroid dysfunction (abnormal serum concentrations of thyroid stimulating hormone (TSH), free thyroxine (FT4) or free triiodothyronine (FT3) at Haydarpaşa Numune Education and Research Hospital Endocrinology or Internal Medicine outpatient clinics were included in this study. Patients who had other illnesses or were taking medications which could affect the peripheral and/or central nervous system were excluded. Electrophysiologial studies were done on 22 patients with hypothyrodism (62,8 %) and 13 patients with hyperthyroidism (37,1 %) before any hormonal treatment.

Clinical evaluation included a detailed history with a set of screening questions refering to principal symptoms of thyroid disease, including palpitations, abnormal sweating, alopecia, cramp, weakness, myalgia, sensorial symptoms, tremor, hot or cold intolerance and cognitive changes and a standart neurological examination. The free T3, T4 and TSH leveles were recorded.

### Electrophysiological Evaluation:

A Medelec Sapphire II FO4/00 EMG machine was used for all studies. The nerve conduction studies were done unilaterally for the median , ulnar, posterior tibial and peroneal motor nerves and median, ulnar, radial and sural sensory nerves with conventional methods. Distal latencies, motor and sensory action potential amplitudes and conduction velocities for all studied nerves and F minimum latencies for the motor nerves were evaluated. For the SEP study, the stimulus was given from the median nerve at the right wrist and the intensity was increased until thumb movement was seen. The electrode placements were done according to the 10/20 system.

The active recording electrode was 2 cm behind and 7 cm lateral to Cz, the reference electrode was placed on Fz and the ground electrode was placed on the same extremity proximally. The cortical responses, N20 and P22(25) were evaluated during this study.

For the blink reflex studies the active electrode was placed over the orbicularis oculi muscle laterally, the reference electrod was placed 2 cm medially on the lateral aspect of the nose and the ground electrode was placed on the forehead. The stimulatıon was bilateral from the supraorbital nerve. The duration and latencies of the ipsilateral R1 and R2 as well as contralateral R2 responses were evaluated.

30 aged and sex matched controls were used for comparison, the ± 2SD results from the control group was accepted as normal values for the electrophysiological studies.

**Statistics:** Statistical package for social scienses (SPSS) for Windows 10.0 was used for statistical analysis. The Student's t, and Mann Whitney U tests were used for quantitative comparisons. Qhi square and Fisher's exact Qhi square testes were used for the comparison of categorical variables. The results were evaluated within 95% confidence interval and $p< 0.05$ was considered significant.

## RESULTS

35 patients (22 hypothyroid, 13 hyperthyroid) were included in this study. Most patients were females (n=32, 91,4 %), with only 3 male patients (8,5 %). The mean age was 45.0 ± 12,4 in hypothyroid and 45,0 ± 13,4 in the hyperthyroid patients. The control group consisted of 24 females (80%) and 6 males (20%) with a mean age of 38.8 ± 11.4.

Hypothyroid patients complained more about myalgia, fatigue, intolerance to cold and cognitive changes while the hyperthyroid patients complaind more about palpitations, hot intolerance, abnormal sweating, alopecia, cramps and tremor (Table 1).

**Table 1:** Symptoms and signs in hyperthyoid and hypothyroid patients

| | **Hyperthyroid Patients** | | **Hypothyroid patients** | | ***P*** |
|---|---|---|---|---|---|
| | **n** | **%** | **n** | **%** | |
| **Palpitation** | 7 | 53,8 | 8 | 36,4 | *0,313* |
| **Abnormal sweating** | 7 | 53,8 | 9 | 40,9 | *0,458* |
| **Alopecia** | 9 | 69,2 | 3 | 13,6 | ***0,001***** |
| **Myalgia** | 6 | 46,2 | 12 | 54,5 | *0,631* |
| **Weakness** | 4 | 30,8 | 12 | 54,5 | *0,172* |
| **Fatigue** | 8 | 61,5 | 19 | 86,4 | *0,091* |
| **Cramps** | 5 | 38,5 | 8 | 36,4 | *0,901* |
| **Paresthesia** | 7 | 53,8 | 9 | 40,9 | *0,458* |
| **Tremor** | 4 | 30,8 | 1 | 4,5 | ***0,050**** |
| **Cognitive changes** | 6 | 46,2 | 11 | 50,0 | *0,826* |
| **Hot intolerance** | 10 | 76,9 | 8 | 36,4 | *0,020*** |
| **Cold intolerance** | - | - | 4 | 18,2 | ***0,274*** |

**Electrodiagnostic Studies:**

Nerve Conduction Studies:

The results of the nerve conduction studies in 22 hypothyroid and 13 hyperthyroid patients compared to the control group are shown in table 2 and table 3.

**Table 2:** The electrophysiological parameters in the hypothyrioid patients and controls

| Parameter | Hypothyroid patients (mean±SD) | Controls (mean±SD) | P value |
|---|---|---|---|
| **Latencies (ms)** | | | |
| median motor | 3,43 ± 0,5 | 3,03 ± 03 | 0,003** |
| median sensory | 2,80 ± 0,3 | 2,49 ± 0,2 | 0,002** |
| ulnar motor distal latency | 2,66 ± 0,9 | 2,37 ± 0,2 | 0,163 |
| ulnar sensory | 2,19 ± 0,1 | 2,22 ± 0,2 | 0,624 |
| Radial sensory | 1,94 ± 0,2 | 1,83 ± 0,2 | 0,151 |
| posterior tibial motor | 3,79 ± 0,64 | 3,99 ± 0,7 | 0,319 |
| peroneal motor | 4,04 ± 0,5 | 3,82 ± 0,4 | 0,154 |
| sural sensory | 2,62 ± 0,3 | 2,29 ± 0,3 | 0,001** |
| F min latencies (ms) | | | |
| median motor | 25,47 ± 1,9 | 25,63 ± 5,8 | 0,900 |
| ulnar motor | 24,94 ± 1,5 | 24,59 ± 1,7 | 0,454 |
| posterior tibial motor | 47,79± 4,0 | 45,5 ± 3,6 | 0,041** |
| peroneal motor | 45,6 ± 3,6 | 43,5 ± 3,3 | 0,033** |
| **Conduction velocities (m/s)** | | | |
| median motor | 57,0 ± 4,8 | 57,87 ±4,8 | 0,566 |
| median sensory | 48,5 ± 5,2 | 51,7 ± 6,7 | 0,020** |
| ulnar motor forearm segment | 62,5 ± 4,6 | 64,1 ± 7,6 | 0,359 |
| ulnar motor elbow segment | 57,32 ± 6,8 | 57,17 ± 6,7 | 0,938 |
| ulnar sensory | 51,91 ± 2,3 | 1,86 ± 0,1 | 0,693 |
| Radial sensory | 56,1 ± 5,5 | 57,9 ± 7,6 | 0,335 |
| posterior tibial motor | 49,1 ± 3,3 | 58,5 ± 10,7 | 0,001** |
| peroneal motor | 50,4 ± 3,9 | 51,3 ± 4,3 | 0,447 |
| sural sensory | 46,9 ± 5,0 | 48,0± 4,73 | 0,421 |
| **Motor action potential amplitudes (mV)** | | | |
| median | 6,10 ± 1,9 | 6,55± 2,2 | 0,445 |
| ulnar | 5,34 ± 1,0 | 5,38 ± 1,5 | 0,918 |
| peroneal | 2,65 ±0,8 | 3,23 ± 2,3 | 0,074 |
| posterior tibial | 4,18 ± 1,32 | 4,74 ± 1,7 | 0,213 |
| **Sensory action potential amplitudes (µV)** | | | |
| median | 16,53 ± 6,4 | 26,67 ± 12,2 | 0,001** |
| ulnar | 16,72 ± 6,6 | 24,82 ± 11,3 | 0,002** |
| radial | 31,70 ± 27,7 | 25,54 ± 9,2 | 0,261 |
| sural | 21,32 ± 5,0 | 23,63 ± 10,6 | 0,421 |

**Table 3:** The electrophysiological parameters in the hyperthyroid patients and controls

| Parameter | Hyperthyroid patients (mean±SD) | Controls (mean±SD) | p value |
|---|---|---|---|
| **Latencies (ms)** | | | |
| median motor | 3,23 ± 0,4 | 3,03 ± 03 | 0,135 |
| median sensory | 2,65 ± 0,3 | 2,49 ± 0,2 | 0,105 |
| ulnar motor distal latency | 2,46 ± 04 | 2,37 ± 0,2 | 0,438 |
| ulnar sensory | 2,24 ± 0,2 | 2,22 ± 0,2 | 0,802 |
| Radial sensory | 1,91 ± 0,2 | 1,83 ± 0,2 | 0,387 |
| posterior tibial motor | 3,93 ± 0,7 | 3,99 ± 0,7 | 0,789 |
| peroneal motor | 4,19 ± 0,7 | 3,82 ± 0,4 | 0,054 |
| sural sensory | 2,61 ± 0,5 | 2,29 ± 0,3 | 0,013** |
| F min latencies (ms) | | | |
| median motor | 24,37 ± 1,4 | 25,63 ± 5,8 | 0,451 |
| ulnar motor | 24,42 ± 1,8 | 24,59 ± 1,7 | 0,783 |
| posterior tibial motor | 46,45± 3,7 | 45,5 ± 3,6 | 0,460 |
| peroneal motor | 44,35 ± 3,6 | 43,5 ± 3,3 | 0,468 |
| **Conduction velocities(m/s)** | | | |
| median motor | 57,4 ± 5,4 | 57,87 ±4,8 | 0,788 |
| median sensory | 50,7 ± 5,0 | 51,7 ± 6,7 | 0,509 |
| ulnar motor forearm segment | 61,30 ± 6,1 | 64,1 ± 7,6 | 0,246 |
| ulnar motor elbow segment | 59,89 ± 9,6 | 57,17 ± 6,7 | 0,292 |
| ulnar sensory | 51,78 ± 3,03 | 1,86 ± 0,1 | 0,660 |
| Radial sensory | 57,17 ± 5,0 | 57,9 ± 7,6 | 0,725 |
| posterior tibial motor | 47,7 ± 4,5 | 58,5 ± 10,7 | 0,001** |
| peroneal motor | 48,88 ± 3,7 | 51,3 ± 4,3 | 0,089 |
| sural sensory | 46,48 ± 4,4 | 48,0± 4,73 | 0,312 |
| **Motor action potential amplitudes (mV)** | | | |
| median | 6,91 ± 2,9 | 6,55± 2,21 | 0,663 |
| ulnar | 5,57± 1,9 | 5,38 ± 1,5 | 0,733 |
| peroneal | 2,63 ±0,9 | 3,23 ± 2,3 | 0,137 |
| posterior tibial | 4,92 ± 2,3 | 4,74 ± 1,7 | 0,795 |
| **Sensory action potential amplitudes (µV)** | | | |
| median | 18,90 ± 7,9 | 26,67 ± 12,2 | 0,042** |
| ulnar | 15,43 ± 9,29 | 24,82 ± 11,3 | 0,013** |
| radial | 26,47 ± 10,8 | 25,54 ± 9,2 | 0,776 |
| sural | 16,80 ± 5,1 | 23,63 ± 10,6 | 0,007** |

When we evaluated the results according to the normal values obtained from the controls, 13 hypothyroid (59%) and 7 hyperthyroid (53,8%) patients had abnormal nerve conduction studies. Carpal tunnel syndrome (CTS) was positive in 12 patients (34 %); 11 hypothyroid and 1 hyperthyroid patient (Table 4). CTS was significantly higher in the hypothyroidism group (p:0,027). Two patients had sensorial neuropthy while only one side sural nevre was affected in 3 patients (Table 4).

**Table 4:** Electrophysiological abnormalities in hypothyroid and hyperthyroid patients

| Finding | Hypothyroid | Hyperthyroid |
|---|---|---|
| CTS | **9 (40.90)** | **1 (7,69)** |
| CTS +sural neuropathy | **1 (4.54)** | **---** |
| CTS +sensorimotor polyneuropathy | **1 (4,54)** | **---** |
| Sensorimotor polyneuropathy | **1 (4,54)** | **2 (15,38)** |
| Sensorial polyneuropathy | **1 (4,54)** | **1 (7,69)** |
| Sural mononeuropathy | **---** | **3 (23,07)** |

Median nerve SEP studies:

N20 and P25 latencies were prolonged in the hypothyroid patients compared to the control group ($p < 0,05$) . In the hyperthyroid patient group both latencies were also prolonged but this was only statistically significant fort he P25 latency. (Table 5-6).

When compared to the normal values 11 (50%) patients with hypothyroidism and 5 (38,4 %) patients with hyperthyroidism had abnormal SEP studies. The most common finding was a prolonged P25 latency which was seen in 10 (45%) hypothyroid and 5(38%) hyperthyroid patients. Patients with abnormal nerve conduction studies had significangly prolonged N20 latencies (p:0,011).

**Blink Reflex Studies:**

İpsilateral and contralateral R2 duration was longer in hypothyroid patients ($p<0.05$). None of the variables were significantly different from the control group in the hyperthyroid patients.

Overall 18 patients (51,4%) had blink reflex abnormalities (11 hypothyroid, 7 hyperthyroid patient). Eight hypothyroid and 3 hyperthyroid patients had longer ipsilateral and contralateral R2 durations.

**Table 5:** Comparison of median nerve SEP latencies in hypothyroid patients

| | Hypothyroid patients | Control | p value |
|---|---|---|---|
| N 20 latency | **18,89 ± 0,99** | **18,08 ± 1,31** | **0,022**** |
| P 25 latency | **23,79 ± 2,01** | **20,87 -±1,39** | **0,001**** |

**Table 6:** Comparison of median nerve SEP latencies in hyperthyroid patients

| | **Hyperthyroid patients** | **Control** | **p value** |
|---|---|---|---|
| **N 20 latency** | 18,48 ± 0,94 | 18,08 ± 1,31 | 0,343 |
| **P 25 latency** | 22,78 ± 1,86 | 20,87 -±1,39 | 0,001** |

## DISSCUSSION

Neurological dysfunction associated with disorders of the thyroid gland may be a result of hormonal imbalance or may be related to the immune mechanisms associated with thyroid dieseses [6,7,23,22]. The thyroid hormone affects the central and peripheral nervous systems via it's role in gene expression, myelin production, it's effects on the neurotransmitter system and axonal transportation [1,13,15].

Overall neurological complications have been reported to be around 79% in hypothyroidism [7,15,24]. Proximal muscle weakness, mental changes, constipation, intolerance to cold are some of the usual signs of hypothyroidism. Common symptoms of hyperthyroidism are alopecia, palpitations, proximal muscle weakness, fatigue, abnormal sweating [10,12,23]. In our study as expected hypothyroid patients complained more about myalgia, fatigue, intolerance to cold and cognitive changes, while the hyperthyroid patients complained more about palpitations, hot intolerance, abnormal sweating, alopecia, cramps and tremor.

A high ratio of polyneuropathy (20-70%) associated with hypothyroidism has been reported and the mechanisms has been studied extensively [3]. The metabolic alteration caused by hormonal imbalance affects the Schwann cell, inducing a segmental demyelination. Primary axonal degeneration has also been shown electrophysiologically and pathologically [15]. Only the function of the nerve is affected initially, but later structural alterations may ocur [5,18]. Since the distal and sensory nerves are affected earlier, the most commonly involved nerves are the sural nerve and median nerve sensory fibers. CTS is caused by the deposition of mucinous material in the tissue surrounding the median nerve combined with mild hypothyroidism induced demyelinization [11,15,18]. The incidence of CTS also varies has been reported in 5-92% of hypothyroid patients [11,22,24].

Our findings are compatible with the literature. In this study 59% of the hypothyroid patients had at least one type of electrophysiological abnormality. The most common finding was found in median motor and sensory nerves (54%). Eleven patients (50%) had isolated CTS. One patient had sensorimotor and 1 patient had sensorial neuropathy. Sural nerve was affected in 2 (18%) cases.

The prevelance of neuropathy in hyperthyroidism is lower, around 19-24%. The mechanism is unknown [11,22]. It has been suggested that in severe thyrotoxicosis peripheral nerves are affected as well as dorsal root ganglion and anterior horn cells. In a study involving 141 recently diagnosed untreated thyroid disease patients, 20 % of the hyperthyroid patients were reported to have an axonal sensorimotor polyneuropathy (6,7,19). The sural nerve is the most commonly affected nerve in these patients. The incidence of CTS has been reported as 5 % [6,7,9]. How does an excess of thyroidal hormones induce peripheral nerve damage is still unknown [6,8,19].

In our study, we found electrophysiological disturbances in 7 out of 13 hyperthyroid patients. Similar with the literature sural nerve was the most commonly involved nerve in this group of patients (23 %). Only 1 patient had electrophysiological evidence of CTS (8,3%).

SEP gives sensitive, objective information about abnormalities of the sensory system [16,19]. Abnormal SEPs can result from dysfunction at the level of the peripheral nerve, plexus, spinal root, spinal cord, brain stem, thalamocortical projections, or primary somatosensory cortex [16,19,20,21]. There are only a few studies with different conclusions regarding SEP in thyroid diseases. Some studies found prolonged latencies in both hypothyroid and hyperthyroid patients while others did not find any difference between patients and controls. Overall central nervous system

dysfunction has been reported in up to 78% of the patients with hypothyroidism (16,20). In this study 50% of the hypothyroid and 38% of the hyperthyroid patients had median nerve SEP abnormalities. We further analyzed the patients who had nerve conduction abnormalities and they had more prolonged SEP latencies as we expected ($p<0,05$). Therefore we believe the prolonged latencies in the present study might be a result of the peripheral nervous system involvement. Absolute SEP latencies vary with limb length and a limitation of our study is that we were not able to measure limb length.

Blink reflex has been reported to be normal in hypothyroidism [4]. We found 50 % of the patients had long ipsilateral and contralateral R2 durations. It has been suggested that the duration of R2 responses might be shorter in hyperthyroidism due to the inhibitor effect of the excess thyroid hormone on polysynaptic reflex arc of the blink reflex [4]. In this study there was no significant difference between hyperthyroid patients or controls in any of the blink reflex parameters. We believe that the different conclusions might be related to our small sample size.

Our data confirm the involvement of peripheral nervous system in thyroid diseases. It is known that the severity of neuromuscular symptoms and signs correlates well with the degree and duration of hormonal imbalance [24]. Our study shows that the hormonal and and metabolic changes which are responsible for the electrophysiological changes may occur early in the disease course and can cause symptoms before the diagnosis of the thyroid disease.

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