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ORIGINAL ARTICLE

Brainstem auditory evoked potential in clinical hypothyroidism

Kirti Sharma, Naresh Kumar, Joshil Kumar Behera[1], Sushma Sood[2], Sibadatta Das, Harnam Singh Madan[3]

Departments of Physiology, and [3]Orthopedics, Shaheed Hasan Khan Mewati Government Medical College, Nalhar, Mewat, [2]Department of Physiology, Post Graduate Institute of Medical Sciences, Rohtak, Haryana, [1]Department of Physiology, Government Medical College, Srinagar, Pauri Garhwal, Uttarakhand, India

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ABSTRACT

Objectives: The association of hypothyroidism with impairment of hearing is known to occur. It may be of any kind i. e., conductive, sensorineural or mixed. The aim of this study is to assess auditory pathway by brainstem auditory evoked potential (BAEP) in newly diagnosed patients of clinical hypothyroidism and healthy sex- and age-matched controls. **Materials and Methods:** The study included 25 healthy age- and sex-matched controls (Group I) and 25 patients of newly diagnosed clinical hypothyroidism (Group II). The recording was taken by using RMS EMG EP MK2 equipment. **Statistical Analysis Used:** Unpaired Student's t test. **Results:** There was a significant increase in wave IV (5.16 ± 0.85 ms) and wave V (6.17 ± 0.89 ms) latencies of right ear BAEP of Group II in comparison to wave IV (4.66 ± 0.39 ms) and wave V (5.49 ± 0.26 ms) of Group I. Wave V of left ear BAEP of Group II was also prolonged (6 ± 0.61 ms) in comparison to Group I (5.47 ± 0.35 ms). There was a significant difference in inter-peak latencies IPL I -V (4.44 ± 0.66 ms) and IPL III -V (2.2 ± 0.5 ms) of right ear BAEP of Group II in comparison to IPL I -V (3.94 ± 0.31 ms) and IPL III -V (1.84 ± 0.34 ms) of Group I. A significant prolongation was also found of IPL I -V (4.36 ± 0.59 ms) and IPL III -V (2.2 ± 0.42 ms) of left ear BAEP of Group II in comparison to IPL I -V (3.89 ± 0.3 ms) and IPL III -V (1.85 ± 0.3 ms) of Group I. **Conclusion:** Prolongation of wave IV and V along with inter-peak latencies in BAEP of both ears suggests that central auditory pathway is affected significantly in clinical hypothyroid patients.

Key words: BAEP, clinical hypothyroidism, hearing impairment

INTRODUCTION

Thomas Wharton coined the term thyroid for the first time in 1656. Adult thyroid is the largest endocrine gland in human body consisting of two lobes. Thyroid gland produces tetraiodothyronin (T4), a prohormone and triiodothyronin (T3), active hormone.[1] Hypothyroidism results from functional or structural derangement that interferes with the adequate production of thyroid hormone. This again is divided into primary and secondary depending on whether the defect is intrinsic or due to hypothalamus or pituitary defect.[2] The association of hypothyroidism with impairment of hearing cannot be ruled out. It was first documented by Kemp in 1907 in a severely hypothyroid female.[3] The extent of hearing loss in acquired hypothyroidism was audiometrically documented by Hilger.[4] Brainstem auditory evoked potentials (BAEP) are potentials recorded from ear and vertex to access the conduction through auditory pathway up to midbrain by giving a brief auditory stimulation.[5] We are assessing auditory pathways of hypothyroid patients by BAEP, because some workers have reported prolongation of both peripheral

Address for correspondence: Dr. Kirti Sharma, Department of Physiology, Shaheed Hasan Khan Mewati Government Medical College, Nalhar, Nuh, Mewat - 122 107, Haryana, India. E-mail: kirti75kirti@yahoo.co.in

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and central conduction time in hypothyroidism while some studies showed that there were no statistically significant alteration in BAEP in hypothyroidism.[6-8]

MATERIALS AND METHODS

The present study was conducted in Department of Physiology and Endocrinology of a tertiary health center of North India. The study included 25 healthy age- and sex-matched controls and 25 patients of newly diagnosed clinical hypothyroidism. Newly diagnosed stands for those patients who have been labeled as hypothyroid after doing their thyroid profile for the first time and put on medication only after doing BAEP for evaluation of their auditory functions. The subjects were divided into two groups:

Group I - 25 healthy control subjects of either sex, in age group below 50 years.

Group II - 25 newly diagnosed patients of clinical hypothyroidism of either sex, in age group below 50 years.

Inclusion criteria

Hypothyroidism is diagnosed by increased thyroid stimulating hormone (TSH) and decreased thyroid hormone level. For assessment of these hormones, chemiluminescence immunoassay method was adopted.

- Clinical hypothyroidism TSH level >5.1 mIU/L and T_4 < 57.9 nmol/L.[9]

Exclusion criteria

- Any neurological or psychiatric illness; altered sensorium; patients with any other major medical disorder that can affect evoked potentials i. e., diabetes mellitus, anemia, hypertension, chronic obstructive pulmonary disease, acute or chronic liver and kidney disease
- History of drug abuse including alcoholism
- Subjects with more than 25 dB loss were not included as they come under category of mild hearing impairment according to WHO classification of hearing impairment.

Investigations

The following investigations were done in all subjects and results were written in a specific proforma.

- Thyroid profile
- Brainstem Auditory Evoked Potential (using RMS EMG EP MK2 equipment, Chandigarh, India).

Procedure for BAEP

Subjects were explained about the nature of the procedure. The patients were put at ease and were made to lie down to relax. Procedure was carried out in a silent, sound proof and air conditioned room. Patients were encouraged to sleep during testing as this helped in relaxation and would not alter recordings. After thoroughly cleaning the electrode-recording sites on scalp with cleansing material (alcohol or spirit), electrolyte paste was applied on recording surface of disc electrodes and then Ag/AgCl electrodes were affixed at predetermined positions on scalp according to 10/20 international system of electrode placement. The auditory stimulus as described in stimulus parameter for BAEP was provided to subject. The signals were picked by electrodes and were filtered, amplified, averaged, displayed on the screen of RMS EMG EP MK2 and recorded. Artifacts were automatically rejected. Two recording were taken and they were superimposed to assess reproducibility of BAEP waveforms. Only the reproducible readings were taken and averaged together to obtain the final measurement. For ideal reproducibility, the latency values measured on two separate repetitions should agree with each other within 0.1 ms and the amplitude values should agree with each other within 10%.

Stimulus parameters

Auditory stimulus of 60 dB at a rate of 0.1 ms square pulse through shielded headphones was given. The other non testing ear was masked by 40 dB sound. In this way a total of 1,000 stimulations were applied at a rate of 10/sec. Signals were filtered with band pass 100 Hz and 3 K Hz. Stimulus with alternating polarity were used and the skin electrode impedance was kept below 5 K ohms.

Channels were named according to designation given to reference electrodes attached to mastoids. We named them as A1 and A2 so the names of channels are as given below:

Channel 1: Cz - A1

Channel 2: Cz - A2

Ground : Fz

We recorded the BAEPs by monaural stimulation so two montages Cz – A1 and Cz – A2 were used.

Recordings of BAEPs

The normal BAEP recording consists of five or more vertex positive and vertex negative waves arising within 10 ms of auditory stimulus. Absolute peak latencies of waves I, II, III, IV and V together with inter-peak latencies of I-III, I-V and III-V; and amplitudes of wave I and V was recorded. Amplitudes were very variable and difficult to use clinically. Therefore they were not considered in our study. For BAEPs,

polarity was such that positivity was upward and negativity was downward.

RESULTS

The difference in right ear absolute peak latency of wave IV and V between two groups was statistically significant ($P < 0.05$), whereas there was no significant difference in latencies of other waves. Also there was a significant increase in inter-peak latencies I-V and III-V [Table 1].

The difference in left ear absolute peak latency of wave V between two groups was statistically significant ($P < 0.05$), whereas there was no significant difference in latencies of other waves. There was a significant increase in inter-peak latencies I-V and III-V [Table 2].

DISCUSSION

In the present study, we compared 25 healthy age- and sex-matched controls (Group I) and 25 patients of newly diagnosed clinical hypothyroidism (Group II). We found that there was no significant difference in the age, height, weight and body mass index between the two groups. Also, there was no significant difference in random plasma glucose, hemoglobin level and lipid profile between the two groups, hence both the groups were comparable.

Table 1: Comparison of right ear BAEP absolute latencies and inter-peak latencies of control (group I) and newly diagnosed clinical hypothyroid patients (group II)

Latencies and Inter-peak latencies (ms)	Mean±SD (*n*=25)	
	Group I	Group II
Wave I	1.55±0.16	1.75±0.31
Wave II	2.64±0.23	2.87±0.44
Wave III	3.65±0.28	3.98±0.8
Wave IV	4.66±0.39	5.16±0.85*
Wave V	5.49±0.26	6.17±0.89*
IPL I – III	2.1±0.3	2.24±0.61
IPL I – V	3.94±0.31	4.44±0.66*
IPL III – V	1.84±0.34	2.2±0.5*

*P value<0.05. BAEP: Brainstem auditory evoked potential

Table 2: Comparison of left ear BAEP absolute latencies and inter-peak latencies of control (group I) and newly diagnosed clinical hypothyroid patients (group II)

Latencies and Inter-peak latencies (ms)	Mean±SD (*n*=25)	
	Group I	Group II
Wave I	1.59±0.19	1.67±0.25
Wave II	2.74±0.24	2.76±0.3
Wave III	3.64±0.21	3.86±0.47
Wave IV	4.7±0.4	4.91±0.51
Wave V	5.47±0.35	6±0.61*
IPL I – III	2.05±0.22	2.17±0.42
IPL I – V	3.89±0.3	4.36±0.59*
IPL III – V	1.85±0.3	2.2±0.42*

*P value<0.05. BAEP: Brainstem auditory evoked potential

In BAEP, wave I represents peripheral nervous system involvement as any change in it depicts the effect of hypothyroidism on auditory nerve. Other absolute waves represents central nervous system involvement as any change in them represents effect of hypothyroidism on brainstem as generators of waves II, III, IV and V are cochlear nucleus, superior olivary nucleus, lateral lemniscus and inferior colliculus, respectively. IPL I-III measures neuronal conduction of acoustic nerve across subarachnoid space into core of lower pons. IPL I-V measures central neuronal conduction from proximal acoustic nerve through pons to midbrain. IPL 111-V measures or indirectly reflects neuronal conduction from lower pons to midbrain.[10-12] BAEP might be a useful diagnostic tool in exploring neurological dysfunctions due to hypothyroidism.

In our study, there was statistically significant increase in right ear BAEP absolute latencies of waves IV (5.16 ± 0.85 ms) and V (6.17 ± 0.89 ms) of Group II in comparison to waves IV (4.66 ± 0.39 ms) and V (5.49 ± 0.26 ms) of Group I. Chandrasekhar *et al*., reported prolongation of absolute latency of wave III (5.2 ± 0.63 ms) and wave V (6.7 ± 1.01 ms) in hypothyroid patients compared to wave III (3.6 ± 0.28 ms) and wave V (5.6 ± 0.3 ms) of control group.[13] Anjana *et al*., observed that the latency of wave III was prolonged from (3.57 ± 0.1 ms) in control to (3.6 ± 0.23 ms) in hypothyroid patients.[6] Anand *et al*., also observed prolongation of latency of wave V in alignment with our results,[7] whereas Vanasse *et al*., did not find any change in the latency of different waves.[8]

In the present study, wave V of left ear BAEP was significantly prolonged ($P < 0.01$) in Group II (6 ± 0.61 ms) in comparison to Group I (5.47 ± 0.35 ms). Similar observations were reported in various other studies,[7,13] whereas Vanasse *et al*., observed no significant change in brainstem auditory evoked potential absolute latencies between clinical and control groups.[8]

The difference of IPL I-III between the two groups in right ear was non-significant. But IPL I-V (4.44 ± 0.66 ms) and IPL III-V (2.2 ± 0.5 ms) were significantly increased ($P < 0.01$) in Group II as compared to IPL I-V (3.94 ± 0.31 ms) and IPL III-V (1.84 ± 0.34 ms) in Group I. In left ear, the difference of IPL I-III between the two groups was non-significant. But IPL I-V (4.36 ± 0.59 ms) and IPL III-V (2.2 ± 0.42 ms) were significantly increased ($P < 0.01$) in Group II as compared to IPL I-V (3.89 ± 0.3 ms) and IPL III-V (1.85 ± 0.3 ms)

in Group I. Chandrasekhar *et al*., in their study found prolongation of IPL I-V (6.7 ± 1.95 ms) in clinical group compared to IPL I-V (4.1 ± 0.18 ms) in control group in right ear as well as in left ear.[13] Anand *et al*., reported similar results as in our study.[7] Thornton *et al*., found prolongation of IPL I-V in their study.[14] On the other hand, Anjana *et al*., found a significant decrease in IPL I-III from (2.08 ± 0.16 ms) to (2 ± 0.17 ms) in hypothyroid patients after treatment.[6]

Exact pathophysiological changes leading to hearing loss in hypothyroidism is not fully known. It is believed by many researchers that hypothyroidism leads to decrease in cell energy production, compromising the microcirculation and consequently the metabolism and oxygenation of the involved organ. In the case of hearing loss, this affects the inner ear structures.[15,16] Thyroid hormone is also known to control protein synthesis and myelin production in the central auditory pathway. In addition, T4 also acts as a neurotransmitter in central nervous system. Thus, it can be speculated that in hypothyroidism hearing impairment can originate in the cochlea, central auditory pathway and/or in the retrocochlear region.[17]

So the present study confirmed the involvement of central auditory pathway, evidenced by increase in latency of waves IV and V in clinical hypothyroidism patients. A statistically significant increase in latency of wave V bilaterally in our study indicates that inferior colliculus was affected.

BAEP, a simple and noninvasive method, can act as a clinically useful diagnostic tool in detecting peripheral (auditory nerves) and central neuropathy (brainstem). So we recommend that these electrophysiological studies should be included in routine investigations to find out any CNS dysfunction as early as possible in newly diagnosed patients of clinical hypothyroidism.

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Conflicts of interest
There are no conflicts of interest.

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