# Brainstem Auditory Evoked Potentials Study in Patients with Diabetes Mellitus

Chi-Ren Huang[1], Chen-Hsien Lu[1], Hsueh-Wen Chang[2], Nan-Wen Tsai[1], Wen-Neng Chang[1]

**Abstract-**

***Objectives:*** To analyze the correlation between brainstem-auditory evoked potentials (BAEP) and nerve conduction (NC) studies in patients with diabetes mellitus (DM).

***Methods:*** We retrospectively reviewed the results from the subjects who received our neurological screening test including BAEP and NC studies. A DM group and a control group were applied. The DM group was subdivided 4 subgroups including neuropathy, non-neuropathy, infarct and non-infarct.

***Results:*** A total of 43 DM patients and 43 control subjects were included. The inter-peak latencies (IPL) I-III and IPL I-V of the BAEP showed a statistical significance between the DM and control groups. In the IPL I-III study, the DM neuropathy subgroup showed a statistical significance in either the DM non-neuropathy or control subgroup. The IPL I-III showed moderate correlation (correlation coefficient-0.334) with tibial motor NC velocity.

***Conclusion:*** Patients with DM have a prolongation in IPL I-III, especially in the neuropathy subgroup. This prolongation in IPL I-III would best be explained by acoustic neuropathy. The tibial motor, median sensory, and sural NC velocities correlated with the acoustic neuropathy in patients with DM.

**Key Words:** Brainstem-auditory evoked potentials, Nerve conduction study, Diabetes mellitus, Tibial nerve, Neuropathy

*Acta Neurol Taiwan 2010;19:33-40*

## INTRODUCTION

The inter-peak latencies (IPL) of brainstem auditory evoked potentials (BAEP) study between DM and non-DM subjects have been reported to have statistical significance in the IPL I-III[(1-11)], IPL III-V[(2-5,12)], and IPL I-V[(2-15)]. In addition to the above findings, two reports included brain magnetic resonance imaging (MRI) studies of some of their patients[(2,14)]. Dual pathogenesis including silent infarct and metabolic disturbance of the brain has been proposed as the explanation of the above findings, especially diabetic angiopathy[(14)]. Neuropathy is commonly found in patients with diabetes mellitus (DM) including cranial neuropathy. A longer latency of responses in patients with DM than control subjects in facial nerve conduction studies and blink reflex studies

From the [1]Department of Neurology, Chang Gung Memorial Hospital-Kaohsiung Medical Center, Chang Gung University College of Medicine, Kaohsiung, Taiwan; [2]Department of Biological Science, National Sun Yat-Sen University, Kaohsiung, Taiwan.
Received June 10, 2009. Revised July 8, 2009.
Accepted August 14, 2009.

Reprint requests and correspondence to: Wen-Neng Chang, MD. Department of Neurology, Chang Gung Memorial Hospital-Kaohsiung, Kaohsiung County, 833, Taiwan.
E-mail: cwenneng@ms19.hinet.net

have been reported[16-18]. Sensorineural hearing loss could be clinically unapparent in some patients with DM[1-3,11]. A correlation between the BAEP findings and nerve conduction (NC) studies has been suggested, including velocies of median sensory and peroneal motor nerve studies[5,10]. Acoustic neuropathy in DM would be another pathogenesis in the prolongation of IPL I-III.

Some of BAEP studies in DM have been reported in Taiwan before[5,19,20] including one animal study[19]. Of them, one study reported BAEP in patients with DM[5]. A correlation between the BAEP findings and velocity of the median sensory NC study was reported[5]. The above study lacked complete comparison with all routine late response and NC studies. Although the BAEP findings in subjects with DM and non-DM have been widely reported before, most of the reports do not have comprehensive brain MRI studies to exclude intracranial lesions. Moreover, studies concerning the correlation between BAEP and NC have rarely been reported. The purposes of this study were (1) to evaluate the statistical difference of BAEP between DM and non-DM groups, (2) to evaluate the statistical difference of BAEP between the DM with infarct, DM without infarct, and control subgroups, (3) to evaluate the statistical difference of BAEP between the DM with neuropathy, DM without neuropathy, and control subgroups, (4) to evaluate which parameters in NC had the best correlation with BAEP in the patients with DM group.

## METHODS

We retrospectively reviewed the clinical and electrophysiological results from the subjects who had ever received our special protocol for neurological screening testing. Such screening which is not covered by the Taiwan National Health Insurance Bureau included blood examination, BAEP, NC studies, brain and spine MRI, clinical diagnosis, and so on. All subjects who had significant systemic diseases other than DM (such as autoimmune disease, thyroid diseases, and cancer), evidence of brainstem or cerebellar infarct in MRI, or any lesion occupying the intracranial space other than infarct in the MRI study, myelopathy, radiculopathy, plexopathy, neuromuscular junction disorders, or myopathy were also excluded from this study.

The DM and control groups of subjects were included in this study. The diagnostic method of DM was based on the criteria from the American Diabetic Association[21]. Subjects in the DM group were sub-classified into DM neuropathy and DM non-neuropathy subgroups or DM infarct and DM non-infarct subgroups. Patients who had evidence of neuropathy in NC studies were included in the neuropathy subgroup. Patients who had evidence of infarct in brain MRI studies were considered as the infarct subgroup. Control group subjects were considered by excluding those who had DM, neuropathy, or any evidence of infarct from MRI studies.

The BAEP studies were performed according to the recommended standards of the American Clinical Neurophysiology Society[22] by using the machine of Nicolet Bravo. The method is summarized as follows. Monaural click stimulation at rates of 10 Hz with masking sounds in the contralateral ear was applied to test subjects. Two thousand clicks were averaged by a filter setting of 100 and 3000 Hz. Two or more responses were obtained to show replicability. Bipolar activity was recorded from a mastoid electrode ipsilateral to stimulation and a reference electrode at the vertex (position Cz of the 10-20 system). The ground was placed on the scalp in a midline frontal location (position Fz of the 10-20 system). Electrode impedances were 5 kOhm. The latencies of reproducible waves I, III, V and the interpeak latencies I-III, I-V and III-V were determined.

The late responses and NC studies were conducted using our laboratory standard methods including temperature control using a Nicolet Viking Select system for the analysis of NC studies. Surface recording and stimulation were performed in all these studies. The motor NC studies included median, ulnar, peroneal and tibial nerves studies. In general, the belly-tendon montage was used. Supramaximal stimulation was applied in this study. The median motor NC study was recorded at the abductor pollicis brevis muscle and stimulated at distal (6 cm, wrist) or proximal (elbow) sites. The ulnar motor NC study was recorded at the abductor digiti minimi muscle and stimulated at distal (6 cm, wrist) or proximal

(below elbow) sites. The peroneal motor NC study was recorded at the extensor digitorium brevis muscle and stimulated at distal (6 cm, ankle) or proximal (knee) sites. The tibial motor NC study was recorded at the abductor hallucis muscle and stimulated at distal (6 cm, ankle) or proximal (popliteal fossa) sites. For each patient, the nerve conduction velocity (NCV) data were included in this study.

The sensory NC studies were carried out by using an antidromic study. The sensory NC studies included median, ulnar and sural nerves studies. The median sensory NC study was recorded at digit 2 (index finger) and stimulated at the wrist with a distance of 14 cm. The ulnar sensory NC study was recorded at digit 5 and stimulated at the wrist with a distance of 11 cm. The sural sensory NC study was recorded at the lateral malleolus and stimulated at the lateral calf with a distance of 14 cm. The onset latency was the time from the stimulus to the initial negative deflection from baseline from biphasic sensory nerve action potentials (SNAP) or to the initial positive peak for triphasic SNAP (22). For each patient, the data of sensory NCV were included in this study. The late response study included a H reflex study and median, ulnar, peroneal and tibial F wave studies. The stimulation site and recording site were the same as for the motor nerve conduction study except for when the cathode was placed distally. The stimulation was given by supramaximal stimulation. Ten artifact free responses were recorded. The data of minimal latency in an F-wave study were included in this study. The H reflex study was recorded at the soleus muscle and stimulated at the popliteal fossa of the tibial nerve. The stimulation intensity was increased gradually to yield the maximum H response. The latency of H reflex was included in this study.

Three separate statistical analyses were performed. First, the demographic data between study and control groups were compared. Categorical variables were compared using Chi-square test or Fisher exact test. Continuous variables within 2 groups were compared using independent t test for parametric data and Mann-Whitney U test for non-parametric data, respectively. The reference limits from the control group were derived from the mean $\pm$2.5 standard deviation (SD) if they followed a normal distribution. The data above the reference limits were considered to be "outside reference data". Second, continuous variables among three groups were compared using one-way analysis of variance (ANOVA) followed Scheffe's multiple comparison procedures. Third, since some NC studies of individual nerves did not follow a normal distribution. The correlation between BAEP study and NC study was analyzed by using Spearman's correlation.

## RESULTS

A total of 43 DM patients and 43 healthy subjects were included in this study. Twenty-one men and twenty-two women were included in each group. Their basic data are listed in Table 1. Basic data including age, height, and weight did not show a statistical significance between the DM patients and control subjects, the absolute latencies the I control and DM groups were 1.63$\pm$0.02 and 1.67$\pm$0.02, in wave I 3.72$\pm$0.02 and 3.84$\pm$ 0.02 in wave III, and 5.65$\pm$0.02 and 5.65$\pm$0.02 in wave Ⅴ, respectively. The BAEP results between the DM patients and control subjects are listed in Table 2. "outside reference data", were found in 26% (11 in 43 subjects) in the IPL I-III, 0% (0 in 43) III-V and 14% (6 in 43) I-V studies in the DM group. "Outside reference data" in the IPL I-III study were found to be 37% (10 in 27 subjects) in the DM neuropathy group in the 6% (1 in 16) and DM non-neuropathy subgroups. "Outside reference data" in IPL I-V study were noted in 5 persons in the DM neuropathy subgroup and in 1 in the DM non-neuropathy subgroup. "Outside reference data" in the IPL I-III study were found to be 33% (3 in 9) and 24% (8 in 34) in the DM infarct and DM non-infarct subgroups, respectively. "Outside reference data" in the IPL I-V study were observed in 1 person in DM infarct subgroup and in 5 in the DM non-infarct subgroup. In the comparison with the data from the BAEP study, the IPL I-III and IPL I-V studies showed in statistical significance between the DM and control group.

The BAEP results between patients in the DM neuropathy, DM non-neuropathy, and control subgroups or

***Table 1.*** *Basic data of the 43 diabetic mellitus (DM) patients and 43 control subjects*

| Basic Data | DM (N =43) | | Control (N =43) | | *p*-value* |
|---|---|---|---|---|---|
| | Median | Range | Mean ± SD | Range | |
| Age (years) | 60.5 | 40 - 79 | 59.0 | 40 - 76 | 0.840 |
| Height (cm) | 157.8 | 141.5 - 177.5 | 158.5 | 143.5 - 172.5 | 0.552 |
| Body weight (kg) | 67.1 | 44 - 105.4 | 63.0 | 42.0 - 84.3 | 0.267 |

* P values were calculated by the Mann-Whitney U test

***Table 2.*** *Comparison of Control and diabetes mellitus groups in the brainstem auditory evoked potentials study*

| | Brainstem auditory evoked potentials study | | |
|---|---|---|---|
| | IPL I-III (ms) | IPL III-V (ms) | IPL I-V (ms) |
| Control (n = 86) | | | |
| Mean ± SD | 2.08±0.11 | 1.93±0.15 | 4.01±0.18 |
| Range | 1.84 - 2.28 | 1.64 - 2.32 | 3.64 - 4.40 |
| Reference limit | 2.35 | 2.30 | 4.46 |
| Diabetes mellitus (n = 86) | | | |
| Mean ± SD | 2.17±0.17 | 1.96±0.15 | 4.13±0.23 |
| Range | 1.80 - 2.68 | 1.60 - 2.24 | 3.68 - 4.64 |
| P value | <0.001 | 0.151 | <0.001 |

P values were calculated by an independent-samples T test

Reference limit in IPL I-III, III-V, and I-V was calculated by mean ± 2.5 SD

IPL: inter-peak latency, SD: standard deviation

***Table 3.*** *Comparison of diabetes mellitus (DM) with neuropathy, DM without neuropathy, and control groups in the brainstem auditory evoked potentials study*

| IPL | Result | Groups | | | ANOVA |
|---|---|---|---|---|---|
| | | Control (N = 86) | DM with PN (N = 54) | DM without PN (N = 32) | *P* value |
| IPL I-III | Mean±SD | 2.08±0.11 | 2.20±0.18 | 2.12±0.13 | <0.001 |
| | Range | 1.84 - 2.28 | 1.80 - 2.68 | 1.80 - 2.36 | |
| IPL III-V | Mean±SD | 1.93±0.15 | 1.97±0.15 | 1.95±0.16 | 0.269 |
| | Range | 1.64 - 2.32 | 1.60 - 2.24 | 1.64 - 2.24 | |
| IPL I-V | Mean±SD | 4.01±0.18 | 4.17±0.23 | 4.07±0.21 | <0.001 |
| | Range | 3.64 -2.32 | 3.68 - 4.64 | 3.72 - 4.60 | |

IPL: inter-peak latency; SD: standard deviation; DM: diabetes mellitus; PN: peripheral neuropathies; ANOVA: The Analysis of Variance

Post Hoc analysis of ILP I-III

DM with neuropathy group and DM without neuropathy group (*P*= 0.043)

DM with neuropathy and control group (*P* <0.001)

Post Hoc analysis for IPL I-V

DM with neuropathy group and control group (*P* <0.001)

***Table 4.*** *Comparison of diabetes mellitus (DM) with infarct, DM without infarct, and control groups in the brainstem auditory evoked potentials study*

| IPL | Result | Groups | | | ANOVA |
|---|---|---|---|---|---|
| | | Control (N = 86) | DM with infarct (N = 18) | DM without infarct (N = 68) | *P* value |
| IIPL I-III | Mean±SD | 2.08± 0.11 | 2.17±0.19 | 2.17±0.16 | 0.001 |
| | Range | 1.84 - 2.28 | 1.80 - 2.48 | 1.80 - 2.68 | |
| IPL III-V | Mean ± SD | 1.93±0.15 | 1.95±0.16 | 1.97±0.15 | 0.316 |
| | Range | 1.64 - 2.32 | 1.64 - 2.24 | 1.60 - 2.24 | |
| IPL I-V | Mean ± SD | 4.01±0.18 | 4.12±0.24 | 4.13±0.24 | 0.001 |
| | Range | 3.64 - 2.32 | 3.68 - 4.60 | 3.72 - 4.64 | |

IPL: inter-peak latency; SD: standard deviation; DM: diabetes mellitus; ANOVA: The Analysis of Variance

Post Hoc analysis of IPL I-III

DM without infarct group and control group (*P*= 0.001)

Post Hoc analysis for IPL I-V

DM without infarct group and control group (*P* =0.001)

***Table 5.*** *Correlation study between the BAEP study and nerve conduction study from 29 subjects with 58 pieces of data in DM patients*

| | IPL I-III | | IPL I-V | |
|---|---|---|---|---|
| | Correlation | *p*-value | Correlation | *p*-value |
| Motor nerve conduction velocity | | | | |
| Median | -0.252* | 0.028 | | 0.085 |
| Ulnar | | 0.407 | | 0.091 |
| Peroneal | | 0.315 | | 0.129 |
| Tibial | -0.318** | 0.008 | -0.350** | 0.004 |
| Sensory nerve conduction velocity | | | | |
| Median | | 0.128 | -0.352** | 0.003 |
| Ulnar | | 0.209 | | 0.123 |
| Sural | -0.251* | 0.029 | -0.302* | 0.011 |
| Late response | | | | |
| Median F-wave | | 0.152 | | 0.115 |
| Ulnar F-wave | | 0.056 | | 0.133 |
| Peroneal F-wave | | 0.382 | | 0.283 |
| Tibial F-wave | | 0.121 | | 0.298 |
| H reflex | | 0.056 | | 0.098 |

Spearman's correlation test

IPL: inter-peak latency

*Correlation is significant at the 0.05 level

** Correlation is significant at the 0.01 level

DM infarct, non-infarct, and control subgroups are listed in Table 3 and 4. DM neuropathy subgroup showed a statistical significance either with the DM non-neuropathy or control subgroups in ILP I-III study. DM non-neuropathy subgroup did not show a statistical significance with the control group in ILP I-III study. The IPL I-III, III-V, and I-V studies did not show a statistical significance between the two subgroups of DM infarct and non-infarct patients.

The correlation study between the BAEP study and NC study were analyzed from 58 pieces of data from 29 subjects in DM group by excluding the NC study done by the machine other than Nicolet. The results are listed in Table 5. The tibial motor nerve velocity was most significantly correlated with the IPL I-III study (Spearman rho = -0.318). The IPL I-III study also showed correlation with median MNCV (Spearman rho = -0.252), and sural sensory NCV (Spearman rho = -0.251). The tibial motor (Spearman's rho = -0.350), median sensory (Spearman's rho = -0.352) and sural (Spearman's rho = -0.302) NCV were significantly correlated with IPL I-V studies.

## DISCUSSION

Patients with DM may have subclinical sensorineural hearing loss[12]. Sensorineural hearing loss is more commonly found in patients with DM than non-DM controls [22]. The atrophy of spiral ganglion in the cochlear, demyelination and beading of the myelin sheaths of the VIII cranial nerve, and lack of degenerative change in central auditory pathways are the main pathological findings in patients with DM[25]. In our study, the IPL I-V and I-III but not IPL III-V showed a statistical difference between the DM and control group. We agree with the opinion that an increase of IPL I-V results from an increase of IPL I-III[8]. The findings of an increase of IPL I-III and I-V indicated a retro-cochlear dysfunction in the DM group. The working hypothesis in most BAEP studies has assigned waves I, II, III, and V to the segment of nerve closest to the cochlear, cochlear nucleus, superior olivary complex, and inferior colliculus, respectively[26]. We did not agree with the proposal of brain stem dysfunction in the explanation of the prolongation in IPL I-III. The IPL III-V study was conducted in the auditory pathway of the brain stem. No "outside reference data" were found in the analysis of IPL III-V in our patients with DM. The negative results of statistical difference in IPL III-V between the DM and control groups went against the reasoning of brain stem dysfunction. The nonspecific findings in the central nervous system of a previous pathological study also did not agree with the opinion of brain stem dysfunction in IPL I-III[25].

Dual pathogenesis including silent infarct and metabolic disturbance of the brain has been proposed as the explanation of the above findings, especially diabetic angiopathy[14]. Two previous studies were found to have BAEP and MRI studies. The first report concerned about 13 subjects who were randomly selected for brain MRI studies from 40 persons with DM, including two in nine "outside reference data" (exceed mean±2SD) in negative brain MRI studies and three in four of lacunar infarct in MRI studies[2]. Brain MRI studies were selected for seven persons who had abnormal (exceed mean± 3SD) BAEP studies of IPL I-III from 53 subjects with DM, including five persons in an infarct group and two in a non-infarct subgroup[14]. In our study, all of our 43 subjects received brain MRI studies. "Outside reference data" were found in three of nine (33%) subjects of the infarct subgroup and eight of 34 (24%) in the non-infract subgroup. Our study did not show statistical significance of IPL I-III between the infarct and non-infarct subgroups in patients with DM. The number of cases in our DM infarct subgroup was limited to achieve a statistical significance.

Neuropathy is commonly found in patients with DM, including cranial neuropathy. A longer latency of blink reflex and facial nerve conduction studies in patients with DM than the control subjects has been reported[17-19]. "Outside reference data" in our patients with DM were more commonly found in the DM neuropathy subgroup (37%) than the DM non-neuropathy subgroup (6%). In our study, the result of IPL I-III showed a statistical difference in the DM neuropathy and non-neuropathy subgroups, but not in the study of the DM infarct and non-infarct subgroups. Previously, acoustic neuropathy has been considered in the analysis of BAEP studies in patients with peripheral neuropathies other than DM[27,28].

Furthermore, acoustic neuropathy has been considered as cranial nerve neuropathy of BEAP in patients with DM[4]. A correlation between the BAEP findings and NC studies has been suggested, including velocity of median sensory and peroneal motor nerve studies[5,10]. The above studies lacked complete comparisons with all routine late response and nerve conduction studies. In our study, the results of IPL I-III of BAEP studies showed the best correlation with tibial motor NC velocity following median motor and sural NCV studies. We cannot make a definite conclusion for the above findings. In our consideration, the abnormality in nerve conduction velocity was more significantly found in lower limb (such as the tibial and sural nerve studies) than upper limb nerve studies in patients with length-dependent diabetic polyneuropathy. We propose that this may be the explanation for correlation between tibial motor and sural sensory nerve conduction velocity and IPL I-III.

In conclusion, patients with DM had the findings of a delay in IPL I-III and IPL I-V in BAEP studies, especially in the neuropathy subgroup. The increase in IPL I-V could result from an increase in IPL I-III. DM related acoustic neuropathy was the best explanation in the findings of the prolongation in IPL I-III. The tibial motor NC velocity had the best correlation with neuropathy in patients with DM. Further large scale studies should be considered due to the limitation in case numbers of the present study.

## REFERENCES


1. Uzun N, Uluduz D, Mikla S, et al. Evaluation of asymptomatic central neuropathy in type I diabetes mellitus. Electromyogr Clin Neurophysiol 2006;46:131-7.
2. Nakamura Y, Takahashi M, Kitaguti M, et al. Abnormal brainstem evoked potentials in diabetes mellitus: evoked potentials testings and magnetic resonance imaging. Electromyogr Clin Neurophysiol 1991;31:243-9.
3. Durmus C, Yetiser S, Durmus O. Auditory brainstem evoked responses in the insulin-dependent and non-insulin-dependent diabetic subjects with normal hearing. Int J Audiol 2004;43:29-33.
4. Al-azzawi LM, Mirza KB. The usefulness of the brainstem auditory evoked potential in the early diagnosis of cranial neuropathy associated with diabetes mellitus. Electromyogr Clin Neurophysiol 2004;44:387-94.
5. Pan CH, Chen TJ, Chen SS. Brainstem auditory evoked potentials in diabetes mellitus. Zhonghua Yi Xue Za Zhi (Taipei) 1992;49:244-52.
6. Donald MW, Bird CE, Lawson JS, et al. Delayed auditory brainstem responses in diabetes mellitus. J Neurol Neurosurg Psychiatry 1981;44:641-4.
7. Tóth F, Várkonyi TT, Rovó L, et al. Investigation of auditory brainstem function in diabetic patients. Int Tinnitus J 2003;9:84-6.
8. Dolu H, Ulas UH, Bolu E, et al. Evaluation of central neuropathy in type II diabetes mellitus by multimodal evoked potentials. Acta Neurol Belg 2003;103:206-11.
9. Bayazit Y, Yilmaz M, Kepekçi Y, et al. Use of the auditory brainstem response testing in the clinical evaluation of the patients with diabetes mellitus. J Neurol Sci 2000;181:29-32.
10. Martini A, Comacchio F, Fedele D, et al . Auditory brainstem evoked response in the clinical evaluation and follow-up of insulin-dependent diabetic subjects. Acta Otolaryngol (Stockh) 1987;103:620-7.
11. Fedele D, Martini A, Cardone C, et al. Impaired auditory brainstem-evoked response in sinsulin-dependent diabetic subjects. Diabetes 1984;33:1085-9.
12. Díaz de León-Morales LV, Jáuregui-Renaud K, Garay-Sevilla ME, et al: Auditory impairment in patients with type 2 diabetes mellitus. Arch Med Res 2005;36:507-10.
13. Kondo J, Tachibana H, Inuzumi K, et al. Involvement of central nervous system in patients with diabetes mellitus detected by evoked potentials. Rinsho Byori 1990;38:457-62.
14. Kurita A, Mochio S, Isogai Y. Changes in auditory P300 event-related potentials and brainstem evoked potentials in diabetes mellitus. Acta Neurol Scand 1995;92:319-23.
15. Comi G. Evoked potentials in diabetes mellitus. Clin Neurosci 1997;4:374-9
16. Irkeç C, Nazliel B, Yetkin I, et al. Facial nerve conduction in diabetic neuropathy. Acta Neurol Belg 2001;101:177-9.
17. Al-Azzawi LM, Mizra K, Kummoona R. The usefulness of the blink reflex in the diagnosis of cranial nerve neuropathy associated with diabetes mellitus. Electromyo Clin Neurophysiol 2004;44:323-7.
18. Kazem SS, Behzad D. Role of blink reflex study in diagno-

sis of subclinical cranial neuropathy in diabetic mellitus type II. Am J Phys Med Rehabil 2006;85:449-52.
19. Wu HP, Cheng TJ, Tan CT, et al. Diabetes impairs recovery from noise-induced temporary hearing loss. Laryngoscope 2009;119:1190-4.
20. Wang CT, Huang TW, Kuo SW, et al. Correlation between audiovestibular function tests and hearing outcomes in severe to profound sudden sensorineural hearing loss. Ear Hear 2009;30:110-4.
21. American Diabetes Association. Diagnosis and classification of diabetes mellitus. Diabetes Care 2007; 30(S1):S42-7
22. American Clinical Neurophysiology Society. Guidelines on short-latency auditory evoked potentials. From http://www.acns.org/pdfs/ACFDE93.pdf
23. Preston DC, Shapiro BE: Basic nerve conduction study. In: Electromyography and Neuromuscular Disorders: Clinical-Electrophysiologic Correlations, 2nd ed. Philadelphia: Elsevier, 2005:25-45.
24. Kakarlapudi V, Sawyer R, Staecker H. The effect of diabetes on sensorineural hearing loss. Otology & Neurotology 2003;24:382-6.
25. Makishima K, Tanaka K. Pathological changes of the inner ear and central auditory pathway in diabetics. Ann Otol Rhinol Laryngol 1971;80:218-28.
26. Chiappa KH, Hill RA. Brain stem auditory evoked potentials: interpretation. In: Chiappa KH. Evoked potentials in clinical medicine, 3rd ed. Philadelphia: Lippincott-Raven, 1997:199-249.
27. Pareyson D, Scaioli V, Berta E, et al. Acoustic neuropathy in peripheral neuropathy: a BAEP study. Electromyo Clin Neurophysiol 1995;35:359-64.
28. Kowalski JW, Rasheva M, Zakrzewska B. Visual and brainstem auditory evoked potentials in hereditary motor-sensory neuropathy. Electromyo Clin Neurophysiol 1991;31:167-72.