International Journal of Otorhinolaryngology and Head and Neck Surgery
Joshi KD et al. Int J Otorhinolaryngol Head Neck Surg. 2017 Apr;3(2):347-353
http://www.ijorl.com
pISSN 2454-5929 | eISSN 2454-5937

Original Research Article

DOI: http://dx.doi.org/10.18203/issn.2454-5929.ijohns20171192

A comparative study on effects of diabetes on auditory functions as measured by BERA and DPOAE

K. D. Joshi[1]*, J. R. Galagali[2], Manoj Kumar Kanzhuly[3], I. D. Singh[2]

[1]Department of ENT, Military Hospital, Roorkee, Uttarakhand, India
[2]Department of ENT, Command Hospital, Pune, India
[3]Department of ENT, Armed Forces Medical College, Pune, India

Received: 19 November 2016
Accepted: 24 December 2016

***Correspondence:**
Dr. K D Joshi,
E-mail: kdeepjoshi@rediff.com

ABSTRACT

Background: Diabetes is prevalent endocrine disorder associated with many complications. However, the link between auditory dysfunctions and diabetes is still vague. The current study aims to correlate auditory dysfunction caused by DM, measured by distortion product otoacoustic emission (DPOAE) & brainstem evoked otoacoustic emissions (BERA).
Methods: This is a descriptive (comparative) study in which auditory functions of 100 diabetics and 100 matched non-diabetics were assessed by distortion product otoacoustic emission (DPOAE) & brainstem evoked otoacoustic emissions (BERA). The data for diabetic and non- diabetic group was compared and analysed. Effects of age of individual on auditory functions were also analysed separately using suitable statistical tests. The data collected was analysed with suitable statistical tests were performed with a significance level of α=0.1 using SPSS 2.0 software.
Results: The demographical variable was comparable in both the groups. The results showed decline in free field hearing, which are further adversely affected by duration of diabetes and patient's age. The pure tone thresholds were not significantly higher in diabetics; however the thresholds were significantly higher in diabetics in older age groups. The hearing loss appear at early age in diabetics but gradually become indistinguishable from age related hearing loss.
Conclusions: Although the auditory dysfunction can be linked to diabetes, but are usually not detectable at earliest stages with routine clinical and audiological tests. The DPOAE and BERA have role to play in monitoring of the auditory dysfunction.

Keywords: Diabetes mellitus, Hearing loss, BERA, DPOAE, OAE, neuropathy, Auditory dysfunction, Hyperglycemia

INTRODUCTION

Diabetes (DM) is most commonly encountered endocrine disorder and is proven to be linked with risk of cardiovascular disorders, retinopathy, nephropathy and neuropathies. The available literature on diabetes and hearing loss is exhaustive, yet the relation between diabetes and hearing loss is elusive. The incidence of sensorineural hearing loss (SNHL) in DM is estimated to be 78.2% as compared to 38% in non-diabetics.[1] However the hearing loss resulting from DM is non-characteristic symptom and usually remains underemphasized. Even the world health organization has not mentioned it as a common consequence of diabetes.[2]

The current study aims to correlate auditory dysfunction caused by DM, measured by distortion product otoacoustic emission (DPOAE) & brainstem evoked otoacoustic emissions (BERA). The microangiopathy due to DM can result in loss of blood supply to stria vascularis, atrophy of stria vascularis and damage to outer hair cells; resulting in deterioration of otoacoustic

emissions (OAE). DM can also lead to neuropathy, resulting in changes in BERA recordings.

METHODS

The present study is a comparative study with study population of 100 diagnosed patients of diabetes mellitus, without any subjective hearing related complaints and receiving outpatient and inpatient care at tertiary care hospital during the year January 2015 to January 2016. The control population comprised of 100 age and sex matched non-diabetic subjects. Exclusion criteria were history of exposure o loud sound, intake of ototoxic drugs, chronic smoking, severe head injury, neoplastic diseases, family history of deafness, anatomical defects in ear or conductive hearing loss of any etiology. Diabetic patients were metabolically controlled as determined by Hb1AC, with cut off of normal value at less than 7%. Complications like diabetic retinopathy, neuropathy or nephropathy were also excluded. Prior approval of ethical committee and informed consent from all patients were taken.

A through clinical examination was done for all patients to rule out external and middle ear pathology. DPOAE were measured using GSI audioscreener and analysed using software audiotrac. The stimulus was two equal pure tone primaries at two different frequencies f1 and f2; f1:f2 being 1.2. The geometric mean of f1& f2 corresponded to standard audiometric frequencies 2, 3, 4, 5 & 6 kHz. The intensities of f1 & f2 were (L1) 65 dB SPL & (L2) 55dB SPL respectively. The overall amplitude of DPOAE & its relation to noise floor (signal to noise ratio- SNR) was measured to establish presence of DPOAE. DPOAE SNR has been considered to be better in detecting hearing loss than DPOAE amplitude.[3,4]

BERA recording was performed using LABAT epic audiology evoked potential system with 3000 broadband click stimuli of 100 µsec each at rate of 21 per second at 70 dB & averaged. The absolute latency of wave I to V and interpeak latency (IPL) I-III, III-V and I-V were measured.

The sample size was calculated by using statistical formula for sample size for estimation of difference between proportions, with proportion of subjects having sensorineural hearing loss among diabetics as 74% and proportion of subjects having sensorineural hearing loss among Non-Diabetics as 7% with an alpha-error level of 10% and a power of 90%.[5]

Statistical analyses were performed on the per-protocol population. Statistical testing was performed at a significance level of α=0.1. Adjusted means and corresponding two-sided 90% confidence intervals (CI) were calculated. The data are presented as mean ± standard deviation values and suitable statistical methods were applied. Statistical analyses were performed using IBM SPSS v2.0 software.

RESULTS

The study included 100 subjects for diabetic group with mean age 50.47 years with range 19-79 yrs. The mean age of non-diabetic individuals was 47.86 years. Demographically, both groups were statistically comparable as shown in Table 1. In diabetic group mean fasting blood sugar was 155.65 ± 57.09 with range 77 to 361 mg/dl. The mean post-prandial blood sugar in diabetes was 222.86 mg/dl ± 79.71 with range 121- 501 mg/dl. The duration of diabetes was ranging from newly diagnosed (02 days) to 30 years.

Table 1: Demographic details.

	Diabetic group	Non-diabetic group
Number	100 (Male- 62, Female- 38)	100 (Male- 64, Female- 36)
Mean age (years)	50.47 (SD 13.57)	47.88 (SD 13.78)
Mean fasting blood sugar	155.65 ± 57.09 (Range 77- 361)	93.34 ± 11.12 (Range 68-133)
Mean post prandial blood sugar	222.86 ± 79.77 (Range 121-501)	113.06 ±12.82 (Range 86 -142)
Duration of diabetes	02 days – 30 yrs	-

The comparison of BERA records of two groups revealed that the absolute latencies of BERA were significantly delayed in for wave III, V and significant delay was noted in IPL I-III and I-V as shown in Table 2.

On comparing effects of duration of diabetes on BERA as shown in Table 3, it was noted that only absolute latency of wave V was significantly affected with increase in duration of diabetes in subgroups less than 5 years, 5 to 10 years and more than 10 years.

When BERA recordings were compared in respect of age of the diabetics as shown in Table 4, it was found that there was no significant difference in BERA recording of diabetics and non-diabetics for age group less than 30 years. In age group between 31 to 50 years, it was found that absolute latency for wave III, V, IPL I-III and IPL III-V were significantly affected for diabetics, while in age group more than 50 years, wave III, V and IPL I-III were significantly affected.

The DPOAE showed that mean SNR values and amplitude were decreased for all frequencies in diabetics, when compared to non-diabetics as shown in Table 5.

When DPOAE SNR were compared as shown in Table 6, the values were lower in all frequencies in diabetics, however the values were significantly reduced in higher frequencies (4, 5 & 6 kHz) in age group less than 30 years, and not significantly reduced in age group less than

Table 2: BERA: Absolute latencies and interpeak latencies.

		Min	Max	Mean	SD	P-value
Wave I	DM	1.20	1.90	1.59	0.18	0.276
	Non DM	1.21	1.92	1.61	0.17	
Wave III	DM	3.48	4.23	3.87	0.16	< 0.001*
	Non DM	3.21	4.18	3.72	0.22	
Wave V	DM	5.30	6.50	5.85	0.31	< 0.001*
	Non DM	5.40	6.10	5.73	0.18	
Wave I - III	DM	1.74	2.85	2.28	0.23	< 0.001*
	Non DM	1.52	2.84	2.11	0.27	
Wave III - V	DM	1.32	2.85	1.98	0.31	0.442
	Non DM	1.33	2.67	2.00	0.29	
Wave I-V	DM	3.56	5.28	4.26	0.34	0.020*
	Non DM	3.52	4.83	4.19	0.28	
*** significant**						

Table 3: BERA on basis of duration of diabetes: absolute latencies & interpeak latencies.

Frequency	Duration of diabetes mellitus	BERA				P-value
		Min	Max	Mean	SD	
Wave I	≤ 5 years	1.20	1.90	1.58	0.18	0.617
	5 - 10 years	1.21	1.88	1.57	0.17	
	> 10 years	1.33	1.88	1.67	0.14	
Wave III	≤ 5 years	3.48	4.23	3.86	0.15	0.255
	5 - 10 years	3.54	4.12	3.89	0.16	
	> 10 years	3.50	4.20	3.91	0.21	
Wave V	≤ 5 years	5.30	6.50	5.84	0.32	0.029*
	5 - 10 years	5.36	6.44	5.85	0.28	
	> 10 years	5.33	6.49	5.94	0.34	
Wave I – III	≤ 5 years	1.74	2.74	2.28	0.23	
	5 - 10 years	1.80	2.75	2.32	0.23	0.285
	> 10 years	1.82	2.85	2.24	0.25	
Wave III – V	≤ 5 years	1.32	2.69	1.97	0.31	
	5 - 10 years	1.33	2.66	1.96	0.32	0.194
	> 10 years	1.34	2.85	2.03	0.34	
Wave I - V	≤ 5 years	3.56	5.28	4.26	0.36	
	5 - 10 years	3.82	4.84	4.28	0.27	0.195
	> 10 years	3.64	5.06	4.27	0.37	
*** significant**						

50 years. In age group between 31 to 50 years, mean SNR were significantly reduced in all frequencies and similar findings were noted for mean DPOAE amplitude as shown in Table 7. In age group less than 30 years, amplitudes were reduced only for 5 kHz and for age group more than 50 years, it were reduced only for 2 kHz.

When DPOAE SNR were compared as shown in Table 6, the values were lower in all frequencies in diabetics, however the values were significantly reduced in higher frequencies (4, 5 & 6 kHz) in age group less than 30 years, and not significantly reduced in age group less than

50 years. In age group between 31 to 50 years, mean SNR were significantly reduced in all frequencies and similar findings were noted for mean DPOAE amplitude as shown in Table 7. In age group less than 30 years, amplitudes were reduced only for 5 kHz and for age group more than 50 years, it were reduced only for 2 kHz.

The duration of diabetes did not seem to have any effect on mean DPOAE amplitude and SNR as shown in Table 8, as they were not significantly elevated with duration of diabetes.

Table 4: BERA : Age groupwise absolute and interpeak BERA latencies.

Group		Age group (< 30)			Age group (31 - 50)			Age group (> 50)		
		BERA			BERA			BERA		
		Number of ears examined	Mean	P-value	Number of ears examined	Mean	P-value	Number of ears examined	Mean	P-value
Wave I	DM	10	1.58	0.484	98	1.57	0.192	92	1.61	0.983
	Non DM	18	1.63		104	1.60		78	1.61	
Wave III	DM	10	3.74	0.081	98	3.87	0.000	92	3.89	< 0.001
	Non DM	18	3.60		104	3.70		78	3.79	
Wave V	DM	10	5.88	0.130	98	5.80	0.048	92	5.91	< 0.001
	Non DM	18	5.72		104	5.73		78	5.72	
Wave I - III	DM	10	2.15	0.126	98	2.30	< 0.001	92	2.28	0.006
	Non DM	18	1.97		104	2.09		78	2.17	
Wave III - V	DM	10	2.15	0.779	98	1.93	0.013	92	2.01	0.095
	Non DM	18	2.11		104	2.03		78	1.94	
Wave I - V	DM	10	4.30	0.302	98	4.22	0.228	92	4.30	0.107
	Non DM	18	4.14		104	4.17		78	4.22	
*** significant**										

Table 5: DPOAE: SNR values & Amplitude (Ldp).

		SNR			Amplitude (LDP)		P-value
		Mean	SD		Mean	SD	
2000	DM	8.32	8.19	0.001*	-0.35	12.18	0.002*
	Non DM	11.05	10.15		2.60	12.66	
3000	DM	11.61	9.26	< 0.001*	-3.96	8.83	< 0.001*
	Non DM	14.87	10.54		0.95	9.13	
4000	DM	8.65	10.97	0.001*	-7.18	9.08	< 0.001*
	Non DM	12.10	11.48		-3.86	8.93	
5000	DM	6.80	8.38	< 0.001*	-6.93	9.41	0.006*
	Non DM	10.60	10.04		-4.50	8.81	
6000	DM	5.56	7.50	< 0.001*	-8.70	9.04	0.002*
	Non DM	8.75	8.79		-5.88	8.39	
*** significant**							

Table 6: DPOAE: Age group wise SNR values.

Frequency in Hz		Age group (< 30)			Age group (31 - 50)			Age group (> 50)		
		SNR			SNR			SNR		
	Group	Number of ears examined	Mean	P-value	Number of ears examined	Mean	P-value	Number of ears examined	Mean	P-value
2000	DM	10	12.35	0.869	98	8.34	0.000	92	7.84	0.489
	Non DM	18	12.74		104	12.75		78	8.43	
3000	DM	10	13.50	0.191	98	12.06	0.000	92	10.92	0.197
	Non DM	18	17.97		104	16.89		78	11.48	
4000	DM	10	12.10	0.035	98	10.73	0.024	92	6.07	0.216
	Non DM	18	19.82		104	13.83		78	8.00	
5000	DM	10	7.37	0.001	98	7.35	0.012	92	6.16	0.051
	Non DM	18	18.09		104	10.77		78	8.64	
6000	DM	10	11.16	0.049	98	5.88	0.001	92	4.61	0.217
	Non DM	18	13.04		104	9.76		78	6.42	
*** significant**										

Table 7: DPOAE: Age group wise DPOAE amplitude (Ldp) values.

Frequency	Group	Age group (< 30) AMPLITUDE (LDP) Number of ears examined	Mean	P-value	Age group (31 - 50) AMPLITUDE (LDP) Number of ears examined	Mean	P-value	Age group (> 50) AMPLITUDE (LDP) Number of ears examined	Mean	P-value
2000	DM	10	6.17	0.408	98	-0.03	0.012	92	-1.40	0.489
	Non DM	18	5.59		104	4.52		78	-0.65	
3000	DM	10	-0.29	0.064	98	-3.39	0.000	92	-4.95	0.197
	Non DM	18	4.51		104	3.01		78	-2.62	
4000	DM	10	-1.34	0.494	98	-6.29	0.008	92	-8.74	0216
	Non DM	18	4.81		104	-3.00		78	-7.00	
5000	DM	10	-4.54	0.012	98	-6.32	0.046	92	-7.84	0.051
	Non DM	18	3.16		104	-3.91		78	-7.06	
6000	DM	10	-0.40	0.382	98	-9.48	0.003	92	-8.78	0.217
	Non DM	18	-2.37		104	-6.01		78	-6.52	
*** significant**										

Table 8: DPOAE on basis of duration of diabetes: SNR values & DPOAE amplitude (Ldp).

Frequency in Hz	Duration of Diabetes Mellitus	SNR Mean	SD	P-value	DPOAE amplitude (Ldp) Mean	SD	P-value
2000	≤ 5 years	8.54	8.24	0.767	-0.06	13.76	0.460
	5 - 10 years	8.54	7.95		-0.65	8.54	
	> 10 years	6.76	8.34		-1.34	8.21	
3000	≤ 5 years	12.06	9.35	0.681	-3.22	8.81	0.313
	5 - 10 years	11.37	9.07		-4.86	8.35	
	> 10 years	9.72	9.25		-6.23	9.46	
4000	≤ 5 years	9.50	10.63	0.109	-6.59	8.95	0.093
	5 - 10 years	8.83	11.50		-6.83	9.20	
	> 10 years	4.07	11.11		-10.70	9.06	
50000	≤ 5 years	7.48	8.46	0.054	-6.61	9.48	0.416
	5 - 10 years	6.90	8.71		-6.49	10.02	
	> 10 years	3.20	6.59		-9.25	7.94	
6000	≤ 5 years	6.01	7.71	0.103	-8.07	9.35	0.474
	5 - 10 years	5.94	7.26		-9.45	8.47	
	> 10 years	2.65	6.28		2.65	8.20	
*** significant**							

DISCUSSION

BERA is graphical representation of electrical activity generated in auditory pathway. In present study, measurement of absolute and interpeak latencies of BERA was considered for determining site of affection.

The absolute latencies of wave III and V were found to be significantly increased in diabetics, which signify brainstem involvement. The IPL I-III and III-V were significantly higher in diabetic group as compared to non-diabetics, which is indicative of upper and lower nerve function and central conduction delay. Age wise subgroup analysis showed that increased latency of wave III and increased IPL I-III which suggest role of diabetes in causing auditory dysfunction. Only wave V was prolonged significantly with increase in duration of diabetes.

Previous literature also supports the finding of the present study. Dabrowski et al confirmed involvement of auditory organ early in diabetes, with no significant association with duration of diabetes.[6] Gupta et al found significant difference in latencies of wave III, V, IPL I-III, III-V and I-V.[7] Latency of wave I was not higher in diabetes and control population. However, they also reported the larger duration of diabetes is a risk factor for central neuropathy. In contrast to present study,

Chaudhary et al concluded wave I, III, V, IPL I-III, III-V and I-V were not significantly elevated in diabetes.[8] Only II-V was significantly prolonged in group with diabetes for more than 5 yrs. Takkar et al also could not find statistically different BERA parameters in diabetics and non-diabetics.[9]

The OAE are measurable sound produced due to cochlear functions and are by-products of this cochlear amplifier mechanism. OAEs are recordable and absence of these can provide indication of cochlear pathology, even earlier to other clinical tests. TEOAE evaluate cochlea as a whole and is restricted to 1 kHz to 5 kHz. The DPOAE evaluates specific part of cochlea and varies from 0.5 to 8 kHz. It is observed that the TEOAE recordings are not very frequency selective as compared to DPOAE.[10, 11]

In the present study, mean DPOAE SNR values for diabetics were lower in all frequencies [2000, 3000, 4000, 5000 and 6000 Hz] for diabetics. An early onset high frequency cochlear dysfunction in diabetics was indicated by lower SNR values with significant decrease for 4, 5 and 6 kHz. In age group 31- 50 years, all frequencies showed significant decrease in SNR in diabetic patients. However in age group more than 50 years, mean SNR values were comparable for two groups, reflecting a possible combined effect of aging and diabetes.[12] Similar results were seen on comparing DPOAE amplitude. However, the present study failed to detect change in DPOAE SNR and DPOAE amplitude (Ldp) value, when compared on basis of duration of diabetes. Similar results were reported by Lisowska et al in their study, where DPOAE amplitudes (Ldp) were significantly reduced in diabetic group, but no correlation was ascertained between DPOAE amplitude (Ldp) and duration of diabetes.[13] Abo-Elfetoh et al also reported decrease in DPOAE amplitude (Ldp) in diabetes especially at higher frequencies suggesting peripheral auditory system dysfunction.[14] Walter di Nardo studied 47 diabetic patients and compared with controls. A significant difference in mean amplitude between patient and control was found. DPOAE was reduced below 2SD of control mean in 32% of diabetic patients, however no significant association between EOAEs and duration and control of diabetes was found.[11] The results of study conducted by Eren et al were contradictory to present study, in which no statistically significant difference is there between DPOAE amplitude (Ldp) between 40 diabetics and 24 control group in frequencies 1, 2, 2.5, 3,4, 5, 6, 7, and 8 kHz, however small study group can be a limitation of their study.[15] Bayindir et al investigated role of glycaemic control on outer hair cell functions.[16] They found that there is no statistical difference in DPOAE function in controlled and non-controlled diabetes.

The present study substantiate that BERA & DPOAE are valuable in assessing subclinical auditory dysfunction as a consequence of diabetes and can be utilised in early screening of central as well as outer hair cells damage, before conventional methods of audiological assessment. Diabetes mellitus can not only affect peripheral and autonomic nervous system but can also equally involve central nervous system. The auditory functions are also affected by diabetes and various variables like age, sex, age of onset, duration, severity and control of diabetes are subject of research. In the present study an effort was made to study the effects of diabetes in relation to age and duration of diabetes. The results of the study suggest that diabetes have a definite and adverse effect on all constituent of auditory system, which usually go unnoticed. The cochlear and retrocochlear auditory system needs regular evaluation in patients of diabetes. BERA can prove to be an advantageous method to detect both eighth nerve and CNS impairment at the earliest. Apart from BERA, DPOAE is a reliable, non-invasive test for early identification of damage to cochlear functions. In view of diverse result from the studies on the subject, standardisation of results might be required before implementation into routine clinical practice, which will require studies with large sample size and probably multicentric studies.

This study concludes that the diabetics are at definite risk of developing auditory dysfunction. It is recommended that all newly diagnosed diabetic case should undergo a complete audiological evaluation on diagnosis and a regular half yearly or yearly follow up is warranted for early detection of damage to auditory functions.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: The study was approved by the Institutional Ethics Committee

REFERENCES

1. Bhaskar KN, Chalihadan S, Vaswani R, Rehaman CPA. Clinical and audiometric assessment of hearing loss in diabetes mellitus. Int J Scientific Study. 2014;2(4):2-17.
2. World Health Organisation. Diabetes, Fact sheet. WHO media centre. 2015. Available at http: // www.who.Int/mediacentre/factsheets/fs312/en/. Accessed on 2 January2017.
3. Shera CA, Abdala C. Section I: Otoacoustic emissions—mechanisms and applications .In: Kelly L. Tremblay, editor. Translational perspectives in auditory neuroscience hearing across the life span - assessment and disorders. Plural publishing; 2012.
4. Gorga MP, Neely ST, Bergman B, Beauchaine KL, Kaminski JR, Peters J, et al. Otoacoustic emissions from normal-hearing and hearing-impaired subjects: distortion product responses. J Acoust Soc Am. 1993;93:2050-60.
5. Rajendran S, Anandhalakshmi, Mythili B, Viswa. Evaluation of the Incidence of Sensorineural hearing loss in Patients with Type 2 Diabetes Mellitus. Int J Biol Med Res. 2011;2(4):982-87.

6. Dabrowski M, Mielnik-Niedzielska G, Nowakowski A. Involvement of the auditory organ in type 1 diabetes mellitus. Endokrynol Pol. 2011;62(2):138-44.
7. Gupta R, Aslam M, Hasan SA, Siddiqi SS. Type- 2 diabetes mellitus and auditory brainstem response- a hospital based study. Indian J Endocrinol Metab. 2010;14(1):9-11.
8. Shatdai C, Karki P, Baja BK, Patel S. Evaluation of brainstem auditory evoked potential in diabetes. Journal of universal college of medical sciences. 2013;1(2):8-12.
9. Takkar J, Manchanda KC, Bansal B, Gahlot S. Grover GS. Evaluation of auditory neuropathy in type-2 diabetes mellitus using brainstem auditory evoked potentials. International Journal of Research in Health Sciences. 2013;1(2):74-9.
10. Fernandes LC, Casais-Silva L, Ladeia AM. Dysfunction of the peripheral and central auditory pathway in patients with type 1 diabetes mellitus. J Diabetes Mellitus. 2012;2(1):76-81.
11. Di Nardo W, Ghirlanda G, Paludetti G, Cercone S, Saponara C, Del Ninno M, et al. Distortion-product otoacoustic emissions and selective sensorineural loss in IDDM. Diabetes care. 1998;21(8):1317-21.
12. Sharma B. Distortion product otoacoustic emission fine‐structure: An insight into the ear asymmetries. Indian Journal of Otology. 2014;20(2):56-9.
13. Lisowska G, Namyslowski G, Morawski K, Strojek K. Early identification of hearing impairment in patients with type 1 diabetes mellitus. Otol & Neurotol. 2001;22(3):316-20.
14. Abo-Elfetoh NM, Mohamed ES, Tag LM, El-Baz MA, Ez Eldeen ME. Auditory dysfunction in patients with type 2 diabetes mellitus with poor versus good glycemic control. Egypt J Otolaryngol. 2015;31:162-9.
15. Eren E, Harman E, Arslanoglu S, Onal K. Effects of type 2 diabetes on otoacoustic emissions and the medial olivocochlear reflex. Otolaryngol- Head and Neck Surg. 2014;150(6):1033-9.
16. Bayindir T, Erdem T, Uze E, Toplu Y, San R, Ozturan O. Evaluation of the auditory effects in controlled and uncontrolled type 2 diabetes mellitus using otoacoustic emissions. Journal of Turgut Ozal medical center. 2010;17(4):337-41.

Cite this article as: Joshi KD, Galagali JR, Kanzhuly MK, Singh ID. A comparative study on effects of diabetes on auditory functions as measured by BERA and DPOAE. Int J Otorhinolaryngol Head Neck Surg 2017;3:347-53.