Efferent Auditory System Functioning and Speech Perception in Noise in Individuals with Type II Diabetes Mellitus

Prashanth Prabhu and SP Shanthala

All India Institute of Speech and Hearing, Mysore-570006, Karnataka, India

Corresponding author: Prashanth Prabhu, All India Institute of Speech and Hearing, Mysore-570006, Karnataka, India, Tel: +91 8904353390; E-mail: prashanth.audio@gmail.com

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Abstract

Objective: There could be efferent system damage in individuals with type II diabetes mellitus. The study attempts to determine the efferent auditory system functioning through contralateral suppression of otoacoustic emissions (OAE) and speech perception in noise (SPIN) in individuals with type II diabetes mellitus (DM).

Method: The study was carried out on twenty five participants (50 ears) with type II DM patients who were diagnosed by an endocrinologist and twenty five healthy age matched controls (50 ears) were included in the study. Contralateral suppression of OAE and SPIN were administered on both the groups and they were compared and correlated.

Results: The results of the study show that individuals with type II DM had statistically significant lower contralateral suppression values and had poorer speech perception in noise. In addition, there were significant correlations between SPIN scores and the amount of suppression in individuals with type II DM.

Conclusion: The individuals with DM showed difficulty understanding speech in presence of noise and that can be attributed to efferent system damage. Thus, contralateral suppression can be a useful marker in early diagnosis of cochleopathy and auditory efferent disorder. The present study can provide important evidence for early diagnosis and early treatment of diabetic complications.

Keywords: Contralateral suppression; Efferent system; Cochleopathy; Diabetes mellitus; Otoacoustic emissions

Introduction

Type II diabetes mellitus (DM) is an age-related metabolic disorder affecting up to 7% of the population worldwide [1,2]. Diabetes alters the normal blood glucose levels and insulin and affects the intra- and extracellular biochemical signaling pathways of various physiological systems of the body. Diabetes has been associated with hearing impairment in several population-based studies [3,4]. Sasso et al. [5] reported significantly lower otoacoustic emissions amplitudes for diabetics relative to controls. They also noticed in pure tone audiograms that hearing loss increased relative to the duration of DM. These results are similar to other clinical studies on differences in pure-tone audiogram thresholds in individuals with DM [6]. Sasso et al. [5] also found longer auditory brainstem response (ABR) latencies in the diabetics, but there was no significant correlation of the ABR latency changes with decreased emissions amplitudes. Other studies which have used less sensitive hearing measures and/or fewer participants have revealed no differences in hearing loss comparing type II diabetics and controls [7,8].

The histopathological studies of the cochlea in individuals with diabetes show damage to the nerve and vessels of the inner ear [9-11]. The medial olivocochlear (MOC) efferent nerve fibers originate from the medial part of the superior olivary complex on both sides, and project through the vestibular nerve, and terminate on the outer hair cells (OHC) of the cochlea [12-16]. The medial olivocochlear bundle (MOCB) is believed to have an inhibitory function on the outer hair cells, due to its direct connection with the outer hair cells whereby it changes the active micromechanics of the cochlea [17]. The stimulation of MOCB alters the micromechanics of OHC and results in a reduction of otoacoustic emission amplitudes in the opposite ear, an effect referred to as suppression of the otoacoustic emissions (OAE). The OAE suppression can be recorded by providing suppressor noise into the contralateral ear, same ear, or both ears. During contralateral suppression of OAE, a continuous noise is presented to the opposite ear and the OAE is recorded [18]. The lack of contralateral suppression (CS) is a pathologic finding indicating dysfunction of the efferent auditory system [14].

The role of the efferent auditory system in speech recognition in noise, specifically, the medial olivocochlear system (MOCS), has been extensively studied [14,15,19]. It has been suggested that the MOCS enhances the detection of sounds in background noise [20,21]. MOCB helps in enhancement of the signal-to-noise ratio by reduction in the response of the auditory nerve fibers to continuous noise in favor of transient sounds such as speech signals [19]. In addition, studies report that there is correlation between MOCB functioning and speech intelligibility in noise [18,20,22]. Giraud et al. [23] reported correlation between MOCS functioning and speech recognition in noise in a group of patients with vestibular neuromotor and a group of normal-hearing subjects. They found that contralateral noise improved speech recognition in noise in normal-hearing subjects but not in patients with vestibular neuromotor disorders. This suggests that the MOCS can play an
antimasking role in speech perception in noise. There are other studies in the literature which support the possible role of the MOCB in reducing the masking effect [15,19].

The review of literature on otoacoustic emissions in patients with DM shows that they had significantly lower amplitude OAE relative to controls [5,24,25]. Cai [26] studied contralateral suppression of TEOAE in patients with diabetes mellitus and reported significant lower suppression values in them compared to controls. Ugur et al. [27] obtained similar results and suggested a possible medial ololovocochear bundle abnormality. Wang and Zhong [28] studied contralateral suppression of DPOAE and reported absence of suppression and concluded MOCS abnormality in patients with diabetes. The majority of studies are done on Type I diabetes and there are very limited studies on type II diabetes. In addition, there are no studies reported in the literature which attempt to correlate speech perception in noise and contralateral suppression of OAE in individuals with type II diabetes mellitus. Thus, the present study aims to assess efferent auditory system functioning and speech perception in noise in older adults with and without type II diabetes mellitus. The goal is to measure contralateral suppression of TEOAE and DPOAE in both groups. The study also seeks to determine if there is any difference in speech perception in noise scores across the groups. The study also asks if there is a correlation between speech perception in noise and the amount of suppression.

Material and Methods

Twenty five participants (50 ears) with type II DM patients who were diagnosed by an endocrinologist and twenty five healthy age matched controls (50 ears) were included in the study. The mean age of participants with DM was 49.8 ± 5.1 years (range 40-60 years) and the control group was 47.9 ± 4.8 years (range 40-59 years). The groups were age matched but not gender matched across the groups. Criteria of the World Health Organization were used to diagnose type II diabetes [29]. A total of 38 participants without diabetes and 40 participants with diabetes were screened for their hearing sensitivity and 25 participants in each group with normal hearing were selected for the study. None of the members had a history of using ototoxic drug, noise exposure, ear surgery, chronic middle ear disease, Meniere’s disease, cranial trauma or metabolic diseases, only for DM. All the participants had normal hearing sensitivity within 250 Hz to 4000 Hz.

Procedure

To estimate the pure-tone air conduction thresholds and speech identification scores, a calibrated dual channel Grason Stadler (GSI-61) diagnostic audiometer with TDH-39 headphones housed in MX-41/AR ear cushions was used. The bone conduction thresholds were estimated with a Radio Ear B-71 bone vibrator. Pure tone testing was done with a Modified Hughson and Westlake procedure [30]. Speech identification testing was done with monitored live voice presentation of phonemically balanced words in Kannada [31] at 40 dB SL (re: SRT). Immitance evaluation (tympanometry and acoustic reflex testing at 500, 1000, 2000 and 4000 Hz) was carried out with a calibrated middle ear analyzer (GSI-Tympstar V 2.0) and a 226 Hz probe tone. Speech perception in Noise test (SPIN) was administered at 0 dB SNR with ipsilateral speech noise using the word list developed by Yathiraj and Vijayalakshmi [31].

All OAE measurements were performed for both ears separately, and recorded using the ILO 292 II OAE analyzer, version 6 in a sound treated room. TEOAEs were measured with a calibrated OAE analyzer ILO (V6) for non-linear click trains presented at 80 dB peak equivalents SPL. An emission was considered to be present if the waveform reproducibility was more than 50%, and the overall signal to noise ratio was more than 3 dB at least at two frequency bands. The mean TEOAE amplitude (dB SPL) from 800 Hz width frequency bands centered at 1000, 1500, 2000, 3000 and 4000 Hz with and without contralateral white noise stimulus of 55 dB SPL was recorded and analyzed. Distortion product signal amplitude across the range of frequencies corresponding to the following frequencies values for f2: 1000, 1500, 2000, 3000, 4000, 5000, 6000 Hz, and 8000 Hz was recorded with and without contralateral white noise at 55 dB SPL.

Statistical analysis

The results obtained in the study were analyzed statistically with appropriate tests using IBM Statistical Package for Social Sciences (SPSS) Statistics for Windows, Version 20.0. Armonk, New York: IBM Corp.

Ethical considerations

In the present study, all the testing procedures done were using non-invasive techniques adhering to conditions established by ethical approval committee of the Institute and also complied with the Declaration of Helsinki. All the test procedures were explained to participants before testing and informed consent was given by them before participating in the study.

Results

The results of the study show that there was significant reduction (p<0.01) in DPOAE and TEOAE amplitude in individuals with DM compared to the control group. The results of the study also showed a
significant reduction (p<0.05) in SPIN scores at 0 dB SNR in individuals with DM compared to control group. The mean and SD of SPIN scores across the two groups are shown in figure 1.

The contralateral suppression (CS) was significantly reduced (p < 0.001) at mid frequencies in DM group for DPOAE. The mean and SD of DPOAE suppression values across the two groups are shown in figure 2.

![Figure 2: Mean and SD of amount of suppression for DPOAE across normal and DM group.](image)

TEOAE was absent in majority of patients (34 out of 50 ears) with DM. TEOAE when present in individuals with DM had significantly lower suppression values (p < 0.001) at all frequencies compared to the control group. The mean and SD of suppression of TEOAE across DM and the control group is shown in figure 3.

![Figure 3: Mean and SD of amount of suppression for TEOAE across normal and DM group.](image)

The Pearsons product moment correlation at r=0.76, p<0.01 between the mean CS of DPOAE and SPIN scores was obtained.

Correlation analysis was not done for TEOAE because of limited amount of TEOAE data (16 ears) in patients with DM.

## Discussion

The results obtained in the present study supports efferent system dysfunction in individuals with type II diabetes mellitus. The reduction in suppression of OAE is reported in type I diabetics by Namyolowski et al. [32] and Ugur et al. [27]. Thus, the study suggests that efferent system damage and poor speech perception in noise is noticed even in individuals with type II diabetes mellitus. This could be an early manifestation of diabetic neuropathy affecting the efferent auditory system. It can also be attributed to changes in central neural transmission in these individuals due to metabolic changes caused by type II diabetes mellitus [32] (Namyolowski et al. 2001). Makishama and Tanaka [11] described atrophy of spiral ganglion neurons and demyelination of the 8th cranial nerve in four DM subjects. Histopathological studies of the inner ear in the patients with DM showed a thickening in the walls of capillaries in the stria vascularis and degeneration in the organ of corti and outer hair cells [33]. Additionally, abnormal auditory brainstem response results can also suggest impairment in the central neural conduction process of the auditory system in DM [11,34].

The results of the study showed that DM group had poorer SPIN scores and had lower suppression values for TEOAE and DPOAE. In addition, there was also a substantial correlation between SPIN scores and suppression amplitude for DPOAE. This supports the theory that improvement in speech understanding in presence of noise is greater for those who had larger suppression values [20,22]. Thus, the study also confirms that the efferent auditory system is important for understanding speech in the presence of noise [20,21]. Hence, the study shows that there is efferent system damage even in individuals with type II diabetes mellitus. In addition, there is difficulty understanding speech in the presence of noise because of the efferent damage. The study thus provides valuable clinical data which suggests a possible cochleopathy in individuals with DM. These results can also serve as early marker for efferent system damage and difficulty understanding speech in presence of noise in individuals with DM. This would lead to earlier diagnosis of audiological difficulties in individuals with DM and early rehabilitation. However, it is also essential to rule out processing disorders due to aging which could affect the efferent system along with DM.

## Conclusions

Individuals with type II DM had lower contralateral suppression values and had poorer speech perception in noise. In addition, there was substantial correlation between SPIN scores and the amount of suppression in individuals with type II DM. Thus, the present study shows efferent system damage in individuals with type II diabetes mellitus. The individuals with DM also have difficulty understanding speech in the presence of noise that can be attributed to efferent system damage. Thus, contralateral suppression may be a useful marker in early diagnosis of cochleopathy and auditory efferent disorder. The present study may contribute to early diagnosis and early treatment of diabetic complications.

## Conflict of interest statement

The author reports no conflicts of interest. The author alone is responsible for the content and writing of the paper.
References

1. Groop LC (1999) Insulin resistance: the fundamental trigger of type 2 diabetes. Diabetes Obes Metab 1: S1-7.
2. Cavaghan MK, Ehrmann DA, Polonsky KS (2000) Interactions between insulin resistance and insulin secretion in the development of glucose intolerance. J Clin Invest 106: 329-333.
3. Bairbridge KE, Hoffman HJ, Cowie CC (2008) Diabetes and hearing impairment in the United States: audiometric evidence from the National Health and Nutrition Examination Survey, 1999 to 2004. Ann Intern Med 149: 1-10.
4. Dalton DS, Cruickshanks KJ, Klein R, Klein BE, Wiley TL (1998) Association of NIDDM and hearing loss. Diabetes Care 21: 1540-1544.
5. Sasso FC, Salvatore T, Tranchino G, Cozzolino D, Caruso AA, et al. (1999) Cochlear dysfunction in type 2 diabetes: a complication independent of neuropathy and acute hyperglycemia. Metabolism 48: 1346-1350.
6. Tay HL, Ray N, Ohri R, Frootko NJ (1995) Diabetes mellitus and hearing loss. Clin Otolaryngol Allied Sci 20: 130-134.
7. Malpas S, Blake P, Bishop R, Johnson R (1989) Does autonomic neuropathy in diabetes cause hearing deficits? NZ Med J 102: 434-435.
8. Ologe FE, Okoro EO, Oyejoda BA (2005) Hearing function in Nigerian children with a family history of type 2 diabetes. Int J Pediatr Otorhinolaryngol 69: 387-391.
9. Kovar M (1973) The inner ear in diabetes mellitus. ORL J Otorhinolaryngol Relat Spec 35: 42-51.
10. Smith TL, Raynor E, Praza J, Buenting JE, Pillsbury HC (1995) Insulin-dependent diabetic microangiopathy in the inner ear. Laryngoscope 105: 236-240.
11. Makishima K, Tanaka K (1971) Pathological changes of the inner ear and central auditory pathway in diabetics. Ann Otol Rhinol Laryngol 80: 218-228.
12. Brown MC, Nuttall AL (1984) Efferent control of cochlear inner hair cell responses in the guinea-pig. J Physiol 354: 625-646.
13. Al-Mana D, Ceranic B, Djahanbakhch O, Luxon LM (2008) Hormones and the auditory system: a review of physiology and pathophysiology. Neuroscience 153: 881-900.
14. Guinan JJ Jr (2006) Olivocochlear efferents: anatomy, physiology, function, and the measurement of efferent effects in humans. Ear Hear 27: 589-607.
15. Liberman MC, Guinan JJ Jr (1998) Feedback control of the auditory periphery: anti-masking effects of middle ear muscles vs. olivocochlear efferents. J Commun Disord 31: 471-482.
16. Maia CA, Campos CA (2005) Diabetes mellitus as etiological factor of hearing loss. Braz J Otorhinolaryngol 71: 208-214.
17. Maison S, Micheyl C, Collet L (2001) Influence of focused auditory attention on cochlear activity in humans. Psychophysiology 38: 35-40.
18. Collet L, Kemp DT, Veuillet E, Duclaux R, Moulin A, et al. (1990) Effect of contralateral auditory stimuli on active cochlear micro-mechanical properties in human subjects. Hear Res 43: 251-261.
19. Kawase T, Delgutte B, Liberman MC (1993) Antimasking effects of the olivocochlear reflex. II. Enhancement of auditory-nerve response to masked tones. J Neurophysiol 70: 2533-2549.
20. Micheyl C, Collet L (1996) Involvement of the olivocochlear bundle in the detection of tones in noise. J Acoust Soc Am 99: 1604-1610.
21. Winslow RL, Sachs MB (1987) Effect of electrical stimulation of the crossed olivocochlear bundle on auditory nerve response to tones in noise. J Neurophysiol 57: 1002-1021.
22. Micheyl C, Perrot X, Collet L (1997) Relationship between auditory intensity discrimination in noise and olivocochlear efferent system activity in humans. Behav Neurosci 111: 801-807.
23. Giraud AL, Garnier S, Micheyl C, Lina G, Chays A, et al. (1997) Auditory efferents involved in speech-in-noise intelligibility. Neuroreport 8: 1779-1783.
24. Pessin AB, Martins RH, Pimenta Wde P, Simões AC, Marsiglia A, et al. (2008) Auditory evaluation in patients with type 1 diabetes. Ann Otol Rhinol Laryngol 117: 366-370.
25. Díaz de León-Morales LV, Jüregui-Renaud K, Garay-Sevilla ME, Hernández-Prado J, Malacara-Hernández JM (2005) Auditory impairment in patients with type 2 diabetes mellitus. Arch Med Res 36: 507-510.
26. Cai Y (2011) [Contralateral suppressions of transient evoked otoacoustic emissions in diabetes mellitus patients]. Lin Chung Er Bi Yan Hou Tou Jing Wai Ke Za Zhi 25: 292-294.
27. Ugur AK, Kemaloglu YK, Ugur MB, Gunduz B, Saridogan C, et al. (2009) Otoacoustic emissions and effects of contralateral white noise stimulation on transient evoked otoacoustic emissions in diabetic children. Int J Pediatr Otorhinolaryngol 73: 555-559.
28. Wang H, Zhong N (1997) [A study on the contralateral suppressive effects of distortion product otoacoustic emissions]. Lin Chung Er Bi Yan Hou Ke Za Zhi 11: 489-492.
29. Alberti KG, Zimmet PZ (1998) Definition, diagnosis and classification of diabetes mellitus and its complications. Part I: diagnosis and classification of diabetes mellitus provisional report of a WHO consultation. Diabet Med 15: 539-553.
30. Carhart R, Jerger JF (1959) Preferred method for clinical determination of pure-tone thresholds. J Speech Hear Disord 24: 330-45.
31. Yathiraj A, Vijayalakshmi CS (2005) Phonomically balanced wordlist in Kannada. University of Mysore.