Review Article

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The otoacoustic emissions-applicability in the diabetic individual: a brief review

Manish Munjal¹, Hardeep Kaur¹, Shubham Munjal²*, Vineeta Arora³, Kshitij Nanda¹, Pritish Gupta², Tegbir S. Binepal², Ayame D. Patel¹

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*Correspondence: Dr. Shubham Munjal,

E-mail: manishmunjaldr@yahoo.com

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ABSTRACT

Untoward effects in the organ of corti and the stria vascularis are documented on histopathology in the diabetic individual with consequent hearing impairment. Hearing loss is even hypothesized to be due to the synergistic effect of hyperglycemias plus oxidative stress causing cochlear microangiopathy as well as auditory neuropathy. Control of the glycemic index in the diabetic improves the quality of life (QOL).

Keywords: Diabetes mellitus, Otoacoustic emissions, HB I Ac outer hair cells, Hearing impairment

INTRODUCTION

In the inner ear originate, low intensity sounds from the hair cells labelled the otoacoustic emissions (OAE). Cochlear damage disturbs these ermissions. In retro cochlear pathology otoacoustic emissions will be normal but auditory brain stem responses are abnormal.^{1,2} A microphone inserted in the ear canal collects the sounds produced by the cochlear hair cells, on auditory stimulation; captioned the otoacoustic emissions. The brainstem auditory evoked responses (BERA) evaluate. the electrophysiological activity of the auditory pathway. This non-invasive test assesses early impairment of acoustic nerve and integrity of the auditory pathway.³ Fukushima et al study documented that basilar membrane and the stria vascularis vasculature had thickened walls with loss of cochlear hair cells in insulin taking group with respect to the controls.⁴

Spontaneous otoacoustic emissions

In less than one half to 60% of individuals with normal thresholds of hearing, otoacoustic emissions are noted

without an extraneous stimulus. Their frequency ranges between 500 to 4500 Hz and are picked up and measured with a microphone inserted in the ear canal. These emissions are the spontaneous otoacoustic emissions.⁵

Transient otoacoustic emissions

Utilizing click and tone burst stimuli the cochlear function acoustic data is acquired. The elicited responses are the transient otoacoustic emissions (TOAE). Transient otoacoustic emission test assesses cochlear non linearity and outer hair cell function.⁶ Abrupt onset slow duration click stimuli till the frequency range of 4 kHz are used to stimulate multiple nerve fibres. Burst stimuli can be even administered at a narrow frequency range especially at lower frequencies to get a more specific response tone.8 Multiple stimuli are required and averaged. Corresponding to the frequency of the stimulus response emissions are recorded at 2 to 23-milli second latencies. Higher frequencies propagate a shorter distance along the basilar membrane to the base and need a shorter latency whereas lower frequencies travel further towards the cochlear apex and require a longer latency.9

¹Department of ENT and Head and Neck Surgery, Dayanand Medical College, Ludhiana, Punjab, India

²Department of ENT, Dayanand Medical College, Ludhiana, Punjab, India

³Department of Gynaecology, GTB Hospital, Ludhiana, Punjab, India

Distortion product otoacoustic emissions

To detect a high frequency hearing loss, distortion product otoacoustic emissions (DPOAE) is the most sensitive screening test modality which can be customized to assess frequencies that match the patient's audiogram. Two simultaneous pure tone stimuli (f1-f2) are given in this procedure. The frequency is calculated using the formula: 2f1-f2, when measuring DPOAE. To separate normal hearing patients from those with hearing loss the frequency ratio (f2/f1) of 1.2 provides the best accuracy. In normal hearing people and healthy cochlea OAE will be present. Mild hearing impairment can be determined by the OAE test. Outer hair cells damage from ototoxic medications or noise trauma can appear on OAE before presenting on an audiogram. In dysfunction of cochlea, OAE will be absent. In

Test environment

Pure tone audiometry DPOAE and BERA recordings are undertaken in a standard sound treated two room setup with ambient noise levels within permissible limits according to standard for maximum permissible ambient noise levels.

Measurement of DPOAES

DPOAEs are recorded using the OAE equipment. An appropriate probe that fits snugly and includes a source that delivers sounds of particular frequency and a microphone is inserted in the external auditory canal.

The prerequisite for obtaining the otoacoustic emissions are: a patent external auditory canal, tight seal of the ear canal probe, an optimum placement of the test probe, a non-restless patient, and a relatively silent recording environment.

DPOAEs are measured by delivering two simultaneous pure tone stimuli conventionally, of frequencies f1 and f2 (in hertz) and at intensity levels designated L1 and L2 (In decibels).

Conventionally utilized are f1 > f2 with f2 corresponding to 1, 2, 3, 4, 6 and 8 kHz. Audiometric frequencies. The BERA is suggestive of retro cochlear pathology and the DPOAEs of cochlear damage. Similar to audiometric assessment, the evoked otoacoustic emissions are measured independently in each ear.

Measurement of HbA1c

HbA1c is formed by non-enzymatic union of glucose to the N-terminal valine of HbA molecule of the beta-chain. This is a covalent linkage that modifies the molecule's structure as well as charge. These properties are explored by the under mentioned techniques: ion exchange chromatography, immunoassay, electrophoresis, high performance liquid chromatography, and measured as a percent of total haemoglobins.

Data is compiled and detailing all the relevant variables and appropriate statistical tests applied for analysis.

DISCUSSION

A significant decrease in distortion product otoacoustic emission amplitudes at 4, 6, 8, and 10 kHz in the high blood glucose and diabetes mellitus groups w.r.t. the control groups were documented by Akcay et al.¹³

Otoacoustic emissions

In the Ottaviani et al study, seventeen patients (28.3%) had no OAEs in at least one ear and 10% in both ears. Participants were 40 adolescents with DM1 and 40 healthy subjects. DPOAE amplitude at medium frequencies (2.8-4 kHz) was significantly lower in patients with diabetes. 14 The amplitude of otoacoustic emissions was significantly lower in the diabetic patients in Dąbrowski et al study. 15 Botelho et al detected cochlear damage by DPOAE responses with 32% belonging to the diabetic group, versus 3.7% in the control group. 16 Eren et al 40 subject study noted that 40 diabetic patients had decreased OAE amplitude.¹⁷ Balakrishnan et al 90 patient analysis noted that in 60 diabetic patients (30 type 1, 30 type 2 diabetic) DPOAE amplitude reduction in both experimental groups (type 1, and type 2 DM) is comparatively equal. ¹⁸ Hilali et al, 21 subject study documented that the mean TEOAE amplitude was to be significantly reduced (p<0.001) in the diabetic patients (mean 9.0±1.7 dB SPL).19

In the DMCH 30 subject study, 29 (97%) patient had no otoacoustic emissions.²⁰

In the Munjal et al, 30 subjects' analysis, only 3.3% of patients had otoacoustic emissions. With increase in HbA1C (p=0.001) DPOAE the amplitude reduced. Maximum subjects 12 (41.3%) had a reduced amplitude of DPOAE at 6 kHz frequency followed by 9 (31.2%) at 4 kHz and 5 (17.2%) at 2 kHz with a minimal number 3 (10.3%) at 8 kHz Majority 21 (72.4%) were males with reduced amplitude of DPOAE with females 9 (27.5%). Maximum subjects 21 (72.4%) were males with reduced amplitude of DPOAE at a frequency of 6 kHz.²⁰

Otoacoustic emissions are representative of the function of the outer hair cells of the cochlea and the latter, responsible for qualitative sound perception. Therefore, outer hair cell trauma leads to a subclinical hearing dysfunction, as suggested by the above study.

CONCLUSION

Otoacoustic emission is a non-invasive screening technique to evaluate the function of cochlea in chronic health states with significant morbidity and mortality. This can detect early and manage the hearing impairment in the

diabetic patient. Both type 1 and type 2 diabetes have been associated with hearing loss secondary to changes in the organ of corti and stria vascularis.

Hearing deficit is due to hyperglycemia and oxidative stress to cochlear microangiopathy and auditory neuropathy.

With control of glycemic index, the quality of life improves in diabetic patients. On increase in HbA1C, amplitude of recorded waves for OAE is significantly lower which is attributed to disorder in the hair cells of the cochlea.

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