

Gender difference in TEOAEs and contralateral suppression of TEOAEs in normal hearing adults

Farzaneh Zamiri Abdollahi* ; Yones Lotfi, MD.

University of Social Welfare and Rehabilitation sciences, Tehran, Iran

Introduction: Otoacoustic emissions (OAEs) are sounds that originate in cochlea and are measured in external auditory canal and provide a simple, efficient and non-invasive objective indicator of healthy cochlear function. Olivo cochlear bundle (OCB) or auditory efferent system is a neural feedback pathway which originated from brain stem and terminated in the inner ear and can be evaluated non-invasively by applying a contralateral acoustic stimulus and simultaneously measuring reduction of OAEs amplitude. In this study gender differences in TEOAE amplitude and suppression of TEOAE were investigated.

Method and Materials: This study was performed at Akhavan rehabilitation centre belonging to the University of Social welfare and rehabilitation sciences, Tehran, Iran in 2011. 60 young adults (30 female and 30 male) between 21 and 27 years old (mean= 24 years old, SD=1.661) with normal hearing criteria were selected. Right ear of all cases were tested to neutralize side effect if there is any.

Results: According to Independent T-test, TEOAE amplitude was significantly greater in females with mean value of 24.98 dB (p-value <0.001) and TEOAE suppression was significantly greater in males with mean value of 2.07 dB (p-value <0.001).

Conclusion: This study shows that there is a significant gender difference in adult's TEOAE (cochlear mechanisms) and TEOAE suppression (auditory efferent system). The exact reason for these results is not clear. According to this study different norms for males and females might be necessary.

Key words: TEOAEs, Contralateral suppression of TEOAEs, Efferent system, Androgen

Submitted: 17 May 2011

Accepted: 012 Sep 2011

Introduction

Otoacoustic emissions (OAEs) are sound waves that originate from cochlea and emit back into the external ear canal. These sounds can be recorded in ear canal using a sensitive microphone (1). They are produced by active motions of the sensory hair cells of cochlea in response to auditory stimuli (2). OAEs are generated in the outer hair cells (OHCs) which have motility function. Active contractions of the actin and myosin in these cells produce a mechanism of frequency specific cochlear amplifier (3).

There is consensus that OAEs are simple, efficient and non-invasive objective indicators of healthy cochlear function especially OHCs and OAE screening are widely used as a part of universal newborn hearing screening programs. OAEs, as part of the audio logical diagnostic test, can help for differential diagnosis among some hearing pathologies, can be used to monitor the effects of ear disease treatments and are useful in the selection of

hearing aids and surgical options. As a research tool, OAEs are non invasive tools for intra cochlear processes and have brought a new understanding about the nature of sensory hearing impairments (2). OAEs can be classified into two main responses: First, spontaneous otoacoustic emissions (SOAEs) - if response is being recorded in the external acoustic meatus without any auditory stimulation; Second, Evoked Otoacoustic Emissions (EOAEs) - when ear energy is being recorded in response to a kind of sound stimulus. Evoked otoacoustic emissions are also divided (based on stimulus type) into three emissions: Transient (TEOAE) - evoked by a brief sound stimulus, usually a click that has a wide range of frequencies; Distortion product (DPOAE) - evoked by two pure and simultaneous tones (f_1 and f_2) to produce a response based on intermodulation distortion in cochlea (for example $2f_1 - f_2$); Stimulus-frequency (SFEAOE) - evoked by a continuous and low intensity tone (4).

* All correspondence to: Farzaneh Zamiri Abdollahi ; Email:<Audiology_zamiri@yahoo.com>

Auditory system consists of afferent and efferent systems that operate in together (5). Olivocochlear bundle (OCB) or auditory efferent system is a neural feedback loop which is originated from brain stem nuclei and terminated in the inner ear hair cells. This system has two subsystems: Medial olivocochlear bundle (MOCB) and Lateral olivocochlear bundle (LOCB). MOCBs originate in medial portion of superior olivary complex (SOC) and LOCBs originate from lateral part of SOC. Both of these subsystems have crossed (mainly MOCB) and uncrossed (mostly LOCB) projections (6).

Stimulation of auditory efferent has been shown to have a suppressive effect on cochlear responses like OAEs and suppression of OAE has been used frequently in clinical and research settings because it assesses efferent pathways quickly and non-invasively (5). Activation of MOCS can be performed by delivering a contralateral acoustic stimulation and simultaneously measuring OAEs amplitude in test ear. Contralateral acoustic stimulation leads to attenuation of the OAE (7).

Efferent auditory pathway modulates OHCs of cochlea, reduces action potentials of auditory nerve fibers, and involves in locating sources of sound and improving sound detection in noisy context (5). Auditory efferent system involves in anti-masking, protection from damage due to loud noise, auditory and visual attention and auditory development (6). Stimulation of MOCS provides protection against moderate levels of noise, encoding noise signals as well as selecting hearing attention (7).

In this study gender difference in TEOAE amplitude and suppression of TEOAE was investigated.

Materials and Methods

This study was performed at Akhavan rehabilitation centre belonging to the university of Social welfare and rehabilitation sciences, Tehran, Iran in 2011. 60 young adults (30 female and 30 male) between 21 and 27 years old (mean age of both groups 24 years old with 1.66 standard deviation and 0.30 standard error of mean) from students and staff of Akhavan rehabilitation centre were selected. They had not any

previous ear disease or ear surgery and they were volunteers. The inclusion criteria were as follow: Normal otoscopy (by using Riester otoscope), hearing threshold ≤ 15 dBHL between 250 and 8000 HZ (by using Clinical Audiometer AC 33 and headphone TDH-39p of Telephonics), tympanogram type A_n and existence of acoustic reflex threshold between 500 and 4000 HZ (by using Zodiac 901 of Madsen).

Right ear of all cases were selected for TEOAE and TEOAE suppression tests to neutralize side effect if there is any. Cases were instructed to lie down without movement on examination table. OAE and OAE suppression was tested (by using ILO292 of Otodynamics with ILO v6 software in an acoustic room). Probe was calibrated before examinations on daily bases with probe test cavity of Otodynamics. Nonlinear click with $80\mu s$ electrical pulse at a rate of 50/s, mean intensity of 84 dBpeak and 20 ms time window after stimulation was used. Rejection level was 6 percent. TEOAE stimuli were presented through probe 1 of ILO292 of Otodynamics. The contralateral acoustic stimulation (CAS) was a 70 dB SPL white noise delivered by probe 2 of device. Contralateral noise was linear and intermittent (every 3 seconds was turned on/off automatically).

TEOAE test in right ear was done while intermittent white noise was simultaneously presented in contralateral ear. Device shows TEOAE amplitude without and with contralateral noise in two separate windows on screen at once. The difference between TEOAE amplitude with and without contralateral stimulation is suppression magnitude and it is due to efferent system activation.

SPSS software ver. 13 was used for analyzing the data. Independent T-test was selected for analyzing data. The significance level for the statistic tests was set at 5% ($p < 0.05$).

Results

Table 1 and 2 respectively show summary of TEOAE amplitude and TEOAE suppression in males and females.

Table 1: TEOAE amplitude in males and females

TEOAE amplitude (dB)	Males	Females
Mean	20.96	24.98
Standard error of mean	0.34	0.42
Median	20.99	25.03
Standard Deviation (SD)	1.87	2.30
95% confidence interval for mean	Lower 20.26	24.11
	Upper 21.66	25.84
Total number	30	30

Table 2: TEOAE suppression in males and females

TEOAE suppression (dB)	Males	Females
Mean	2.07	1.54
Standard error of mean	0.05	0.03
Median	2.04	1.54
Standard Deviation (SD)	0.27	0.16
95% confidence interval for mean	Lower 1.97	1.48
	Upper 2.18	1.60
Total number	30	30

One sample Kolmogorov-Smirnov test was used to determine if distribution of variables is normal. According to this test all variables were within normal distribution (p -value > 0.05): TEOAE amplitude p -value in males was 0.96 and in females was 0.98, TEOAE suppression p -value in males was 0.52 and in females was 0.97. So parametric Independent t-test was used to compare TEOAE amplitude and TEOAE suppression between men and women. Leven's test for equality of variances was not significant with p -value of 0.22 (p -value > 0.05) so variances of two groups were equal.

The Independent T-test results show that there is a significant difference between males and females in TEOAE amplitude and TEOAE suppression. TEOAE amplitude was significantly greater in females with mean value of 24.98 dB (p -value < 0.001) and TEOAE suppression was significantly greater in males with mean value of 2.07 dB (p -value < 0.001).

Discussion

Several studies have shown gender influence on OAEs and contralateral suppression of OAEs. Cassidy and Ditty (2001) showed that in female newborns TEOAE is more powerful than male newborns. They suggested that OHCs in females respond more sensitive than in males (8).

Durante and Carvallo (2006) found that gender has significant effect on TEOAE and contralateral suppression of TEOAE in neonates. They have shown that TEOAE was larger in female infants and suppression of TEOAE was larger in male infants. They explained this finding with differences in prevalence of SOAEs (SOAEs are more prevalent in female) and cochlear length (cochlea is longer in males) (9). Miller JD. (2007) examined cochlea length in males and females and found that gender difference in cochlea length was 3.36% (corresponds to 1.11 mm difference in length with 0.49 SD) (10). The shorter cochlea in females could lead to the higher amplitude of females' TEOAE response (9).

McFadden D. (1993) proposed that the amount of efferent inhibition is relatively less in females than in males. So OAE amplitude is greater in females and contralateral suppression of OAE is greater in males (11).

McFadden D. et al (2006) continued study of gender effects on OAE and indicated that in human beings and Rhesus monkeys, Click-Evoked Otoacoustic emissions (CEOAEs) are more powerful in females than males, and this gender difference is the result of greater exposure to androgens prenatally in males (12). Other works showed that this gender difference in OAE amplitude fluctuated seasonally and is related to the annual fluctuations of testosterone levels in male Rhesus. The CEOAEs of male Rhesus monkeys were weaker in the breeding season (when male androgen levels are high) than in the birthing season (when male androgen levels fall) (13).

Al-Mana D. et al (2008) stated that it is possible that hormones contribute to pathophysiology of some auditory dysfunctions, including hyper acusis, tinnitus, Menière's disease and pre-menstrual auditory dysfunction and play role in modulating the auditory functions (14).

McFadden D. et al (2009_{a,b}) showed that in humans, OAEs have significant differences between males and females. From early studies on OAEs in humans, ear (right ear versus left ear) and gender differences were apparent. These effects have been shown in newborns and adults. In general, human females have stronger and more prevalent SOAEs and more powerful CEOAEs than males (15, 16).

McFadden D. et al (2009) insisted that one obvious explanation for the gender difference in newborns is the differential prenatal exposure to androgens in two sexes (15, 16). All male mammals early in the course of prenatal development develop embryonic testes that begin producing the androgens that are responsible for masculinizing the prenatal body and brain (16).

OHCs are the most important part in the production of OAEs. Thus, OHCs might have some differences

between males and females. The electro motility of OHCs is dependent to the prestin molecules in the walls of the OHCs, so any differences in prestin could be a major contributor to the OAE differences. Perhaps, for some reasons, women have, on average, more prestin molecules per OHC than men, or perhaps the prestin molecules in women OHCs are better aligned along the cell's contraction axis. In either case, female OHCs would be capable of greater electro motility than male OHCs (16).

Maruska K. and Fernald R. (2010) stated that gonadal and stress-related steroid hormones have influences on auditory function across vertebrates but the cellular and molecular mechanisms of steroid-mediated auditory plasticity at the level of the inner ear remain unknown. The peripheral and central auditory system of vertebrates is sensitive to sex- and stress-related steroid hormones, which can have strong effects on how an animal perceives acoustic information and behaves during social interactions. The steroid receptors have been found in the inner ear which suggests there might be a direct pathway for hormones to act on the peripheral auditory system. The expression levels of steroid receptors differ between the genders. In mammals, females often have "better" hearing (e.g., better high frequency hearing; shorter auditory brainstem response wave latencies) and presbycusis begins in

older age than males. Further, postmenopausal women who are on estrogen-based hormone replacement therapy (HRT) have better hearing than those who are not, while progesterin-based HRT can diminish hearing ability. These sex and female ovarian cycle variations in hearing are attributed to the protective effects of estrogen and may be partially related to estrogen receptor (ER) expression in the cochlea (17).

Conclusion

This study among others shows that there is a significant gender difference in TEOAE (which is by product of cochlear mechanisms) and TEOAE suppression (which is due to effects of auditory efferent system on cochlea). The exact reason for these results is not clear but there are some hormonal and structural explanations. According to this study and other results, it might be necessary to have different norms for males and females, especially in newborn OAE testing to avoid any wrong interpretation.

Acknowledgement:

The authors wish to acknowledge the assistance of the students and staff of Akhavan rehabilitation centre of the University of Social Welfare and Rehabilitation Sciences, Tehran, Iran, in collecting data.

References

1. McFadden D, Pasanen EG, Raper J, Lange HS, Wallen K. Sex differences in otoacoustic emissions measured in rhesus monkeys (*Macaca mulatta*). *Hormones and Behavior*. 2006 Aug;50(2):274–284.
2. Kemp DT. Otoacoustic emissions, their origin in cochlear function, and use. *Br Med Bull*. 2002 Oct 1;63(1):223–241.
3. A L-D. [Otoacoustic emissions]. *HNO*. 1992 Nov; 40(11): 415–421.
4. Vasconcelos RM, Serra LSM, Aragão VM de F. Transient evoked otoacoustic emissions and distortion product in school children. *Revista Brasileira de Otorrinolaringologia*. 2008 Aug;74(4):503–507.
5. Fronza AB, Barreto DCM, Tochetto TM, Cruz IBM da, Silveira AF da. Association between auditory pathway efferent functions and genotoxicity in young adults. *Brazilian Journal of Otorhinolaryngology*. 2011 Feb; 77(1): 107–114.
6. Zeng F-G, Martino KM, Linthicum FH, Soli SD. Auditory perception in vestibular neurectomy subjects. *Hearing research*. 2000;142(1):102–112.
7. Komazec Z, Filipović D, Milo\vsević D. Contralateral acoustic suppression of transient evoked otoacoustic emissions: Activation of the medial olivocochlear system. *Medicinski preglod*. 2003;56(3-4):124–130.
8. Cassidy JW, Ditty KM. Gender differences among newborns on a transient otoacoustic emissions test for hearing. *Journal of Music Therapy*. 2001;38(1):28–35.
9. Durante AS, Carvallo RMM. Changes in transient evoked otoacoustic emissions contralateral suppression in infants. *Pró-Fono Revista de Atualização Científica*. 2006 Jan; 18(1):49–56.
10. Miller JD. Sex differences in the length of the organ of Corti in humans. *The Journal of the Acoustical Society of America*. 2007;121(4):EL151–EL155.
11. McFadden D. A speculation about the parallel ear asymmetries and sex differences in hearing sensitivity and otoacoustic emissions. *Hearing Research*. 1993 Aug;68(2):143–151.
12. McFadden D, Pasanen EG, Weldele ML, Glickman SE, Place NJ. Masculinized otoacoustic emissions in female spotted hyenas (*Crocuta crocuta*). *Hormones and behavior*. 2006;50(2):285–292.
13. McFadden D, Pasanen EG, Raper J, Lange HS, Wallen K. Sex differences in otoacoustic emissions measured in rhesus monkeys (*Macaca mulatta*). *Hormones and Behavior*. 2006 Aug;50(2):274–284.
14. Al-Mana D, Ceranic B, Djahanbakhch O, Luxon LM. Hormones and the auditory system: A review of physiology and pathophysiology. *Neuroscience*. 2008 Jun 2; 153(4): 881–900.
15. McFadden D, Martin GK, Stagner BB, Maloney MM. Sex differences in distortion-product and transient-evoked otoacoustic emissions compared. *J Acoust Soc Am*. 2009 Jan;125(1):239–246.
16. McFadden D. Masculinization of the Mammalian Cochlea. *Hear Res*. 2009 Jun;252(1-2):37–48.
17. Maruska KP, Fernald RD. Steroid receptor expression in the fish inner ear varies with sex, social status, and reproductive state. *BMC Neuroscience*. 2010 Apr 30;11(1):58.