# OLIVOCOCHLEAR REFLEX IN MIGRAINEURS WITH PHONOPHOBIA

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## Abstract

**Background:** Phonophobia describes sound intolerance, one of the characteristic symptoms associated with migraine attacks (Vingen et al., 1998).

**Methods:** This study included 25 normal hearing migraineurs with ictal phonophobia, tested with transient evoked otoacoustic emission (TEOAE) with and without contralateral acoustic stimulation (CAS) with white noise and speech in noise intelligibility (SIN) in the inter-ictal phase. They were compared to 25 well-matched controls.

**Objective:** to assess the function of the cochlear outer hair cells (OHC) and their efferent regulation by the medial olivocochlear reflex.

**Results:** Migraineurs showed statistically significant lower TEOAE than controls in the higher frequency bands as well as in overall response, overall reproducibility and mean AB value reflecting OHC dysfunction. But the majority of cases showed pass TEOAE bilaterally. After CAS, migraineurs showed statistically non-significant weaker TEOAE suppression than controls. Around 60% of ears had suppressed TEOAE overall response. Loudness discomfort level (LDL) was significantly lower than controls but only 3 cases showed hyperacusis, LDL and migraine duration were not correlated. TEOAE suppression was significantly correlated with word discrimination% in different signal to noise ratios (SNR) at certain frequency bands; but was not correlated with SNR of speech reception threshold in noise. TEOAE and SIN tests were not correlated with LDL or migraine duration.

**Conclusions:** Outer hair cells and olivocochlear reflex dysfunction can occur in migraineurs with phonophobia, but still the majority remains unaffected, suggesting other mechanisms of phonophobia than efferent system dysfunction, while the affected minority may have associated sub-clinical hyperacusis.

## Background

Migraine is a neurovascular disorder, characterized by repeated attacks of headache, autonomic dysfunction and gastrointestinal symptoms [1]. Phonophobia describes sound intolerance and occurs in 70–80% of migraineurs during an acute attack [2]. It may also be related to, caused by, or confused with hyperacusis (oversensitivity to sounds), which is an abnormally strong reaction to sound, occurring within the auditory pathways, and efferent auditory dysfunction was proposed as one of its possible mechanisms [3]. Contralateral acoustic stimulation (CAS) has a suppressive effect on acoustic emissions (OAEs) amplitude mediated by medial olivo-cochlear (MOC) reflex, which provides a feedback gain-control at moderate sound levels, to enhance dynamic range and has an anti-masking role in speech perception in noise [4,5].

### Aim of work

Our aim of work was to assess the integrity of efferent control of the cochlea in migraineurs to find its relation to phonophobia.

## Materials and Methods

This study included 25 normal hearing migraineurs having phonophobia in their aura, diagnosed according to the Headache Classification Committee [6] diagnostic criteria, whose mean age was 29.80±6.28 (ranging from 19–41 years), 4 males and 21 females, with a mean migraine duration of 7.09±6.84 years. They were selected from the neurology outpatient clinic at Kasr-Al-Aini hospital, Cairo University, Egypt and were assessed in the headache free (inter-ictal) period, not on any regular anti-migrainous treatment for at least 3 months prior to testing, and free of any vertigo or dizziness. Effects of aging, ototoxic drugs, middle or inner ear diseases, skull, neck trauma, noise exposure or ear surgery on the cochlear biomechanics and hair cell population were eliminated. They were compared to 25 healthy normal hearing volunteers well-matched to them in age (26.92±6.56 ranging from 19–40 years) and gender (7 males and 18 females).

All were submitted to: **1**) Full history taking. **2**) E.N.T. examination. **3**) Basic audiologic evaluation. **4**) Loudness discomfort level (LDL), using pure tones at frequencies of 0.5, 1, 2 & 4 kHz. Dynamic range (DR) was calculated. **5**) Monaural speech intelligibility [word discrimination score

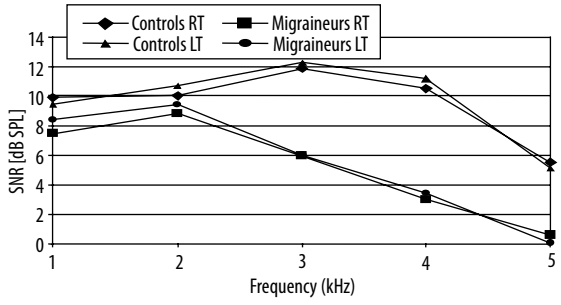


**Figure 1.** TEOAEs signal to noise spectra without CAS of the right and left ears of the 2 studied groups.

(WRS)] in the presence of noise [speech in noise (SIN) test], using Arabic phonetically-balanced words [7], presented at the most comfortable loudness level [40 dB sensation level (SL)]. The level of speech-spectrum noise presented from the same side TDH 39 earphone by the second channel of the audiometer – Orbiter 922 in a sound treated room- was varied at 3 signal-to-noise ratios (SNRs): 0, –5, and –10 dB. Speech reception threshold (SRT) in noise test [difference in dB between speech and noise levels (SRT-SNR) at which the subject understands 50% of the presented words] was also done. **6**) Transient evoked otoacoustic emission (TEOAE), using ILO-96, cochlear emission analyzer (Otodynamics, version 5, London, UK). Stimulus level was 80±3 dB peak sound pressure level. Response included mean AB value, overall response, reproducibility, echo SNR level and reproducibility% at frequency bands: 1, 2, 3, 4, 5 kHz. An overall reproducibility of ≥70% was considered a "pass" result and <70% but >50% was considered a "partial pass" result (8). TEOAE was recorded without and with contralateral acoustic stimulation (CAS) using white noise presented at a level of 40 dBSL according to De Ceeulaer et al. [9].

### Statistical analyses

Statistical analyses were done using the statistical package SPSS version 12 for Windows (SPSS Inc., Chicago, IL, USA).

## Results

There was no statistically significant difference between migraineurs and their controls with regards to the mean SIN tests in both ears: Migraineurs showed mean values of –5.60±1.26, 78.92±10.50, 61.08±13.04 & 34.96±16.59 in the SRT-SNR, WRS-SNR 0, –5 & –10 respectively compared to –5.64±2.40, 80.96±5.17, 63.64±6.82 & 30.28±7.38 of their controls. LDL and DR were statistically significantly lower than the controls. The mean ULL was 99.60±8.89; 103.80±9.05; 105.60±8.08; 106.00±7.22 106.40±3.96 at 0.5, 1, 2 & 4 kHz respectively in migraineurs compared to 113.80±3.32, 113.20±2.84; 114.20±2.36 for the controls. But only 3/25 cases showed hyperacusis. LDL and migraine duration were not correlated (p>0.05). Migraineurs showed a statistically significant (p=0.000) lower TEOAE amplitude than the controls in the higher frequency bands (Figure 1) as well as in overall response, reproducibility and mean AB value. But the majority of cases (18/25) showed pass TEOAE bilaterally. After CAS, migraineurs showed statistically non-significant (p>0.05) weaker TEOAE suppression than their controls (Table 1). The majority of the pass (11/18 right & 10/18 left ears) and (5/7 right & left ears) of the partial pass TEOAEs were suppressed by CAS. But this was not statistically significant (p>0.05). TEOAE amplitude suppression at 2 kHz was significantly correlated with WDS-SNR-5 in the right ear (Pearson's correlation coefficient r=0.309, p=0.029); and -10 in the left ear (r=0.331, p=0.019). TEOAE amplitude suppression at 4 kHz was significantly correlated with WDS-SNR-5 in the left ear (r=0.314, p=0.026). TEOAE reproducibility suppression at 2 kHz was significantly correlated with WDS-SNR-5 in the right ear (r=0.286, p=0.044); and at 4 kHz was significantly correlated with WDS-SNR-5 in the left ear (r=0.396, p=0.004). But there was no correlation between TEOAE amplitude or reproducibility suppression and SRT-SNR. TEOAE and SIN tests were not correlated with LDL or migraine duration (p>0.05).

## Discussion

Our LDL results suggest that phonophobia can sub-clinically affect the LDL, or phonophobia may precede hyperacusis. Migraine is one of the central nervous system disorders causing hyperacusis [10]. Migraineurs had significantly lower LDL in dB between attacks (90.4±0.8) than the controls (105.9±1.1) that is further augmented during acute attacks (76.0±0.9), at 1, 4 & 8 kHz [2,11,12]. Migraineurs had significantly lower LDL of 1 kHz, during headache [13]. Woodhouse and Drummond, [14] did not find any increased between-attack LDL to an 8 kHz tone. Their findings do not support the view that phonophobia in migraine is a manifestation of loudness recruitment, although cochlear disturbances might mediate hearing loss in some cases. Our study showed that migraineurs face the same difficulty as their controls in SIN, their WDS decreased when ipsilateral noise increased, which agree with Persson et al. [5]. Our results showed OHC dysfunction in migraineurs group as a whole but 72% of cases showed pass TEOAE bilaterally suggesting that in the majority, OHC remain unaffected, in agreement with Bolay et al. [15], but they found that TEOAEs were not suppressed by CAS in migraineurs with and without aura. Although around 60% of our cases showed suppression, efferent dysfunction may not be the only mechanism of phonophobia in migraineurs. In agreement with Murdin et al. [16], we did not find any correlation between TEOAE suppression and phonophobia or migraine duration. Vestibular migraine was significantly associated with abnormal suppression [16]. None of our migraineurs had vestibular symptoms, but failure of suppression in some cases may be due to sub-clinical vestibular affection. We did not find any correlation between TEOAE suppression and LDL which agrees with Baguley et al. [17] who showed no change in LDL after section of OC bundle fibers in vestibular neurectomy for disabling vertigo. Our correlation between SIN and TEOAE tests at 2 & 4 kHz, reflected the importance of the efferent system in extracting Arabic speech from background noise. In comparison, Lautenschlager et al. [18] found an association between self-reported difficulties in discriminating SIN and DPOAEs suppression, especially at middle frequencies. But Wagner et al. [19] concluded that SIN does not correlate with efferent activity in humans with normal auditory threshold.

**Table 1.** Mean and standard deviation of TEOAEs amplitude suppression in (dBSPL) and reproducibility suppression in (%) at different frequency bands, overall response, overall reproducibility and mean AB value for both groups.

| Frequency in kHz | Controls (n=25) | | | | Migraineurs (n=25) | | | | t-value | p-value |
|---|---|---|---|---|---|---|---|---|---|---|
| | Mean | SD | Min | Max | Mean | SD | Min | Max | | |
| **Right** SNR 1 | 4.00 | 4.04 | –4 | 13 | 2.16 | 4.53 | –3 | 12 | 1.515 | 0.136 |
| SNR 2 | 2.48 | 2.29 | –1 | 7 | 1.48 | 3.73 | –4 | 13 | 1.141 | 0.259 |
| SNR 3 | 1.64 | 2.74 | –2 | 8 | 1.24 | 3.06 | –5 | 6 | 0.487 | 0.628 |
| SNR 4 | 2.08 | 3.52 | –3 | 11 | –0.12 | 5.16 | –15 | 8 | 1.761 | 0.085 |
| SNR 5 | 0.16 | 2.10 | –4 | 4 | 0.92 | 2.58 | –4 | 6 | –1.143 | 0.259 |
| Reproducibility 1 | 12.04 | 13.11 | –16 | 39 | 7.68 | 15.12 | –10 | 48 | 1.089 | 0.281 |
| Reproducibility 2 | 13.20 | 15.55 | –2 | 60 | 5.20 | 14.84 | –17 | 54 | 1.861 | 0.069 |
| Reproducibility 3 | 2.76 | 5.78 | –7 | 24 | 5.12 | 14.73 | –24 | 38 | –0.746 | 0.460 |
| Reproducibility 4 | 7.12 | 14.00 | –8 | 48 | 4.92 | 30.15 | –61 | 78 | 0.331 | 0.742 |
| Reproducibility 5 | 6.32 | 19.29 | –8 | 69 | 5.28 | 13.45 | –12 | 41 | 0.221 | 0.826 |
| Overall response | 0.70 | 1.23 | –1.6 | 3.7 | 1.18 | 2.37 | –4.5 | 6.8 | –0.907 | 0.369 |
| Overall reproducibility | 6.36 | 5.35 | –3 | 22 | 6.00 | 8.69 | –8 | 25 | 0.176 | 0.861 |
| Mean AB value | 0.52 | 1.31 | –1.7 | 3.7 | 0.98 | 2.14 | –4.3 | 6.1 | –0.919 | 0.363 |
| **Left** SNR 1 | 3.40 | 4.04 | –4 | 13 | 1.40 | 4.27 | –5 | 12 | 1.700 | 0.096 |
| SNR 2 | 2.36 | 2.64 | –1 | 9 | 1.84 | 2.53 | –3 | 5 | 0.711 | 0.481 |
| SNR 3 | 1.60 | 3.29 | –5 | 8 | 0.84 | 2.59 | –5 | 6 | 0.907 | 0.369 |
| SNR 4 | 1.92 | 3.37 | –3 | 11 | –0.04 | 4.08 | –15 | 7 | 1.854 | 0.070 |
| SNR 5 | 0.56 | 2.68 | –4 | 9 | 0.56 | 2.65 | –5 | 6 | 0.000 | 1.000 |
| Reproducibility 1 | 10.24 | 13.72 | –16 | 39 | 2.84 | 14.20 | –25 | 44 | 1.874 | 0.067 |
| Reproducibility 2 | 13.96 | 23.92 | –2 | 98 | 3.80 | 9.24 | –17 | 26 | 1.981 | 0.053 |
| Reproducibility 3 | 4.92 | 11.32 | –7 | 47 | 3.92 | 10.34 | –14 | 38 | 0.326 | 0.746 |
| Reproducibility 4 | 4.84 | 12.37 | –8 | 48 | 3.84 | 23.17 | –61 | 82 | 0.190 | 0.850 |
| Reproducibility 5 | 12.56 | 28.13 | –8 | 91 | 6.92 | 15.10 | –2 | 57 | 0.883 | 0.382 |
| Overall response | 0.74 | 1.37 | –1.6 | 3.7 | 1.11 | 1.91 | –1 | 6.8 | –0.791 | 0.433 |
| Overall reproducibility | 5.16 | 4.89 | –3 | 20 | 4.12 | 7.69 | –12 | 23 | 0.571 | 0.571 |
| Mean AB value | 0.60 | 1.37 | –1.7 | 3.7 | 0.96 | 1.71 | –1 | 6.1 | –0.840 | 0.405 |

## Conclusion

Outer hair cells and olivocochlear reflex dysfunction can occur in migraineurs with phonophobia, but still the majority remains unaffected, suggesting other mechanisms of phonophobia than efferent system dysfunction.

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