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ORIGINAL ARTICLE

Acoustic Reflexes in Individuals Having Hyperacusis of the Auditory Origin

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Abstract Functional role of the acoustic reflex in preventing over stimulation of the inner auditory system by decreasing sound intensity along with the previous reports of acoustic reflex abnormalities in individuals having hyperacusis point towards the involvement of acoustic reflex deficit in the origin of hyperacusis especially when any medical condition leading to hyperacusis is not associated. However this issue remains contradictory owing to limited comprehensive investigation. This study was undertaken to ascertain the relationship between hyperacusis and the acoustic reflex. Threshold, amplitude and latency of the acoustic reflex were measured in two different groups of individuals having hyperacusis; Group 1: 14 individuals having hyperacusis with hearing loss (HwHL) and Group 2: 17 individuals having hyperacusis without hearing loss (HwoHL). Control group (Group 3) consisted of 15 normal hearing individuals who never experienced hyperacusis. Result showed a significant group effect on all the measured characteristics of the acoustic reflex. ARTs were found to be significantly higher in HwHL and HwoHL when compared to NHwoH. ARTs were statistically similar for HwoHL and NHwoH. HwoHL's ARAs and ARLs were significantly smaller and prolonged, respectively, when compared to HwHL and NHwoH. HwHL and NHwoH had statistically similar ARAs and ARLs. This study confirms acoustic reflex abnormalities in some individuals having hyperacusis with or without hearing loss. It further highlight the importance of involving acoustic reflex testing in the assessment of hyperacusis especially when hyperacusis is not associated with hearing loss or any other medical condition that may lead to hyperacusis.

Keywords Hyperacusis · Acoustic reflexes · Threshold · Latency · Amplitude

Introduction

Hyperacusis is one of the most arguable topics in hearing sciences. The phenomena, its definition, site of origin, assessment and management are yet not conclusive [1]. Defined differently by authors, hyperacusis refers to an auditory dysfunction in which the sufferers are over sensitive to sound [1–4]. Individuals having hyperacusis report themselves uncomfortable, hyper-responsive or intolerant to sound intensity levels that are otherwise rated "normally loud" by others who do not have the problem [1, 5–7].

Various hypotheses have been put forward to understand the underlying mechanism of hyperausis. These hypotheses are based on anatomical and physiological conditions of the auditory system, associated medical disorders/diseases and psychological status of sufferer [1, 8, 9]. Hyperacusis reported by individuals with cochlear hearing loss is

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commonly explained based on loudness model. This model explains hyperacusis as the over-excitation of auditory neurons, excitation of neurons more in number when compared to normal hearing individuals, due to the loss of cochlear tuning in individuals with cochlear hearing loss [1, 10, 11]. Another potential hypothesis is that the enhanced central gain as compensation for peripheral hearing loss can cause hyperacusis in individuals with cochlear hearing loss [12, 13].

Hyperacusis is also attributed to the absence/abnormality of the acoustic reflex, for example in patients with Bell's palsy and William Syndrome due to the dysfunction of facial nerve and auditory nerve, respectively, which are important junctures in the pathway of the acoustic reflex [1, 9, 14–18]. Functionally, the acoustic reflex is known to attenuate loud sounds reaching the cochlea. When sound is sufficient enough to activate the acoustic reflex pathway, it contracts stapedius muscle which then pulls the stapes. This process increases impedance of the middle ear and thereby restricts louder sound reaching the inner auditory system [19, 20]. Any anatomical or functional dysfunction in this process may result in hyperacusis [1, 9, 14–18, 20].

Some authors have pointed out an involvement of the efferent auditory system in hyperacusis [1, 8, 21, 22]. Efferent auditory system on activation reduces electomotility function of outer hair cells in the cochlea which in turn attenuates incoming sound reaching the inner auditory system [22]. Therefore, in case there is deficit in the efferent auditory system, the inner auditory system may receive higher sound stimulation resulting in hyperacusis.

Others have linked hyperacusis to cochlear neuropathy; over-excitation of the auditory nervous system; anatomical damage to and/or degeneration of the auditory structures, plasticity of the afferent auditory system and inheritance of syndromes affecting the auditory pathways [1, 8, 9, 23, 24]. There also exist some non-auditory theories of hyperacusis. Some individuals having psychological disorders may develop hyperacusis. Further, hyper-excitability of the central nervous system may also result in hyperacusis [1, 8, 9].

Clinically, it is important to understand the physiological basis of any disorder for accurate assessment diagnosis and effective management. Out of the various hypotheses mentioned earlier, abnormalities of the acoustic reflex appear to be a very relevant auditory cause of hyperacusis when there is no associated medical or psychological condition. Surprisingly, there is limited literature on this necessary clinical issue. Therefore, in this study we focused on understanding the relationship between hyperacusis and the acoustic reflex.

The acoustic reflex is a feedback loop of the auditory system [25]. It occurs when stapes bone, in the middle ear, gets pulled due to the contraction of the stapedius muscle in response to sounds of sufficient intensity. The acoustic reflex pathway includes peripheral hearing system (tympanic membrane to cochlea), auditory nerve, nuclei of the auditory brainstem (cochlear nucleus and superior olivary complex, facial nerve and stapedius muscle [26]. Functionally, it is known that the acoustic reflex prevent over stimulation of the inner auditory system by decreasing the level of sound intensity reaching the cochlea. Therefore, if both exists together, abnormalities of the acoustic reflex (arising from deficit in one or more anatomical unit of the acoustic reflex pathway) can be directly related to hyperacusis. However, literature till date is rare and contradictory on this relation. Few studies have shown absent acoustic reflex, lower acoustic reflex thresholds (ARTs; intensity level at which acoustic reflex activation takes place) and reversed acoustic reflexes in individuals having hyperacusis while others have reported normal acoustic reflexes [27–30]. One of the reasons for this ambiguity is the fact that research investigating acoustic reflexes in individuals having hyperacusis was limited to the check of its presence/absence and to measure ART. In addition to ART, Acoustic Reflex Amplitude (ARA) and Acoustic reflex latency (ARL) are other characteristics of the acoustic reflex that can provide much better and detailed information about the integrity of its anatomical pathway and functioning. ARA is the magnitude of the acoustic reflex [26, 31–33]. ARL, defined differently by authors, most consistently refers to the time taken by the acoustic reflex to grow (or stapedius muscle to contract) to its 10% its maximum amplitude (or maximum contraction) after the onset of reflex activator stimulus [26, 31, 34, 35]. In order to obtain comprehensive understanding of the acoustic reflex in individuals having hyperacusis, we investigated ART, ARA and ARL in this clinical population.

Methods

A total of 196 individual came to our institute with symptom like hyperacusis over a period of 3 years. The entire 196 individual were tested with Modified Khalfa Hyperacusis Questionnaire. Score of > 28 was the labeling criterion for hyperacusis as suggested by Khalfa (2002) [36]. One hundred thirteen turned out to be hyperacusic. In next step, we took detailed medical history to assure that hyperacusis in these participants is not related to condition such as Bell's palsy, head trauma, temporo-mandibular joint syndrome, chronic fatigue syndrome, Lyme disease, posttraumatic stress disorder, depression, autism and endocrine disorders. This was done to control non-auditory variables that may lead to symptoms like hyperacusis. Following this, 64 individuals were excluded and experimental group was formed which consisted of 49 individuals

having hyperacusis of the origin of auditory dysfunction. It is known that hyperacusis may or may not be associated with hearing loss and also that acoustic reflex measurement changes in presence of hearing loss [1, 8, 9, 26, 31, 34, 35]. Therefore, to study the relation between hyperacusis and the acoustic reflex more precisely, the experimental group was further divided into two based on the status of hearing; Group 1 of 23 individuals having hyperacusis with hearing loss and Group 2 included 26 individuals having hyperacusis but no hearing loss. Group 1 and group 2 were abbreviated as HwHL (hyperacusis with hearing loss) and HwoHL (hyperacusis without hearing loss), respectively. Control group (Group 3) consisted of 15 normal hearing individuals who never experienced hyperacusis and had no medical condition stated above that may cause hyperacusis. Group 3 was abbreviated as NHwoH (Normal hearing without hyperacusis).

Pure tone audiometery was done in all the participants to estimate hearing thresholds and diagnose hearing loss. Maico MA 42 diagnostic audiometer, Telephonics TDH39 headphones and Radioear B71 bone vibrator were used for conducting pure tone audiometry. Air conduction hearing thresholds were measured at 250, 500, 1000, 2000 4000 and 8000 Hzs. Bone conduction hearing thresholds were measured at 250, 500, 1000, 2000 and 4000 Hzs. Out of 23 participants in HwHL, 21 were found to have sensorineural hearing loss while 2 had mixed hearing loss. Acoustic reflex measurements were done following pure tone audiometry. Participants were told to inform clinicians if they feel discomfort from sound and were also asked to withdraw anytime from testing if they wish to. One withdrew from HoHL and 3 from HwoHL.

Acoutic reflex measurements were done using GSI TympStar Middle Ear Analyzer. Broad band noise was used as the stimulus for the measurement of ARTs, ARAs and ARLs. Broadband noise was used as it is known to trigger acoustic reflex at intensity level 20 dB lower than that of pure tone stimulus and therefore would be more comfortable for the participant of the study. All the parameters measured in one ear for each participant. It is the ear reported to have hyperacusis in unilateral cases and right ear for bilateral cases and NHwoH. Acoustic reflex were found to be absent in 8 HwHL and 6 HwoHL. At this point of study, 14 out of 21 HoHLs, 17 out of 26 HwoHLs and 15 NHwoHs proceeded for further acoustic reflex measurements.

ARTs were measured using 5 dB step size. A change of 0.02 ml or greater in static compliance was used as the criteria for establishing ART (dB HL). As clinically suggested, stimulus was presented at 10 dB above ART (of respective participant) for the estimation of ARA and ARL [26, 31–35]. Measurement were done while presenting stimulus at intensity relative to ART help in avoiding variable related to individual differences in ART. Validity of all the acoustic reflex measurements was confirmed by repeating the measurement at least two times.

Results

Mean, standard deviation, maximum and minimum of ARTs, ARAs and ARLs in HwHL, HwoHL and NHwoH are shown and given in Online Table 1, Figs. 1, 2 and 3. Results of Multivariate Analysis of Variance showed a significant effect of group on all the measured characteristics of acoustic reflex; ART [$F(2, 43) = 12.470$; $p < 0.001$], ARA [$F(2, 43) = 7.245$; $p = 0.002$] and ARL [$F(2, 43) = 9.713$; $p < 0.001$].

Figures 1, 2 and 3 show ART, ARA and ARL data, respectively, in HwHL, HwoHL and NHwoH. Mean ART, ARA and ARL of each group is represented by unfilled triangle in respective figure. Open circles show ARTs, ARAs and ARLs of all the participants. There was over lapping of circles (data points) in figures due to same ART, ARA and ARL in two or more participants. This was overcome by changing ART, ARA and ARL values (slightly, only in the figures) of few participants by 0.5 or 1 dB, 0.005mmho and 5 ms, respectively, for better data display. Dashed lines represent 1 standard deviation with reference to NHwoH (control group).

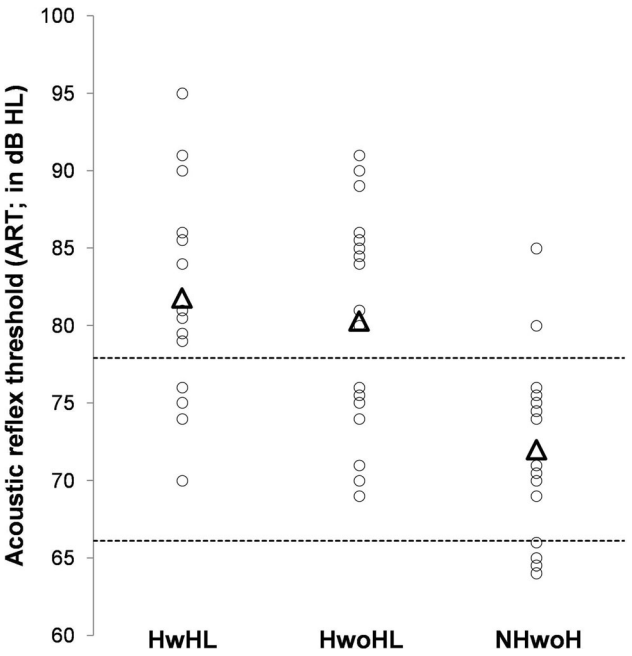

Fig. 1 Show mean ART (unfilled triangles) in HwHL, HwoHL and NHwoH. Individual ART of all the participants is represented by open circles. Dashed lines represent 1-standard deviation with reference to NHwoH (control group)

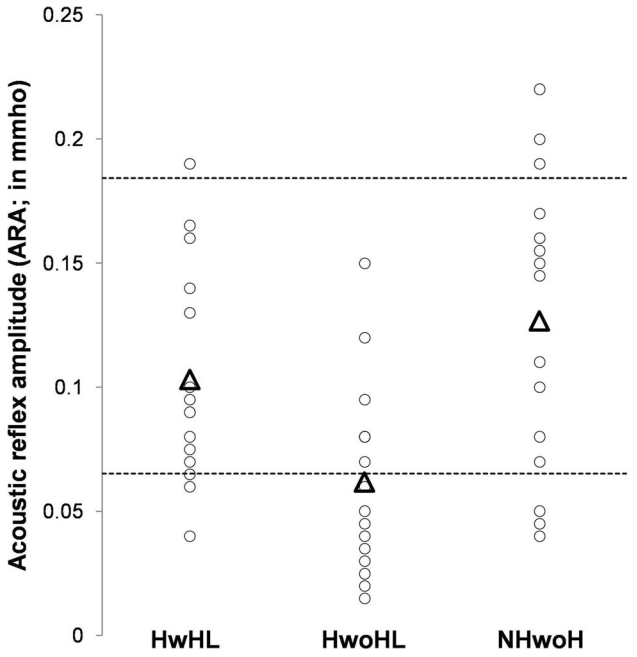

Fig. 2 Show mean ARA (unfilled triangles) in HwHL, HwoHL and NHwoH. Individual ARA of all the participants is represented by open circles. Dashed lines represent 1-standard deviation with reference to NHwoH (control group)

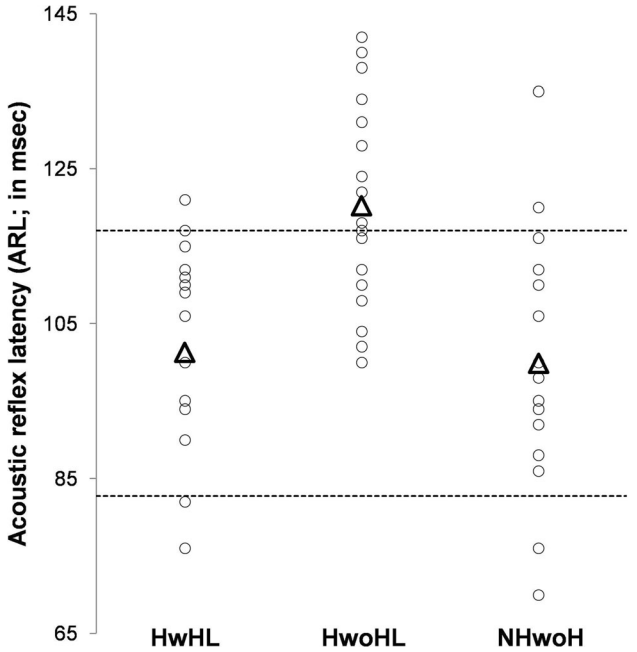

Fig. 3 Show mean ARL (unfilled triangles) in HwHL, HwoHL and NHwoH. Individual ARL of all the participants is represented by open circles. Dashed lines represent 1-standard deviation with reference to NHwoH (control group)

Further statistical analysis involved pair-wise comparisons for ART, ARA and ARL separately. ARTs were found to be significantly higher in HwHL ($p < 0.001$) and HwoHL ($p < 0.001$) when compared to NHwoH (Fig. 1). ARTs were statistically similar for HwHL and HwoHL ($p = 0.493$). HwoHL's ARAs were significantly smaller than HwHL ($p = 0.020$) and NHwoH ($p = 0.001$) [Fig. 2]. HwHL and NHwoH had statistically similar ARAs ($p = 0.232$). ARLs were statistically prolonged in HwoHL contrary to HwHL ($p = 0.001$) and NHwoH ($p < 0.001$) [Fig. 3]. However, ARLs were similar in HwHL and NHwoH ($p = 0.795$).

Discussions

Previous researches suggest a complex threesome relationship between hearing loss, acoustic reflex and hyperacusis. For example, (1) Individuals having mechanical disorder of the middle ear and inner ear were found to have hyperactive acoustic reflex associated with hyperacusis but no hearing loss [27],(2) Williams Syndrome patients have dysfunction of auditory nerve dysfunction, they showed hearing loss, absent acoustic reflex and hyperacusis symptoms [18],(3) Patients with confirmed brainstem lesions and complaints of hyperacusis are associated with hyperactive acoustic reflexes but did not had hearing loss [28],(4) Patients with Bells Palsy, disruption in facial nerve, had normal hearing threshold, absent acoustic reflex and complaints of hyperacusis [14–17]. This relationship was revisited in details through this study by measuring different characteristics of the acoustic reflex including ART, ARA and ARL in two different groups of individuals having hyperacusis, HwoHL and HwHL (grouped based on the status of hearing loss) & in NHwoH.

Acoustic Reflexes in HwHL

Out of the total 23 HwHLs originally recruited for the study; 1 withdrew, acoustic reflexes were absent in 8 HwHLs & measurements (of ART, ARA and ARL) were done in the rest 14. Results showed higher ARTs in HwHL when compared to NHwoH. In contrast, ARA and ARL were similar to NHwoH. ARA and ARL are relative measures of the acoustic reflex; they were measured 10 dB above the ART. Therefore, sound energy reaching stapedius muscle for the activation of acoustic reflex would be same in the two groups even when their ARTs were different, resulting in same ARA and ARL. The fact that only ARTs were abnormal (elevated) in HwHL, not ARAs and ARLs, could just be attributed to their hearing loss not hyperacusis. This also stands for HwHLs who had absent acoustic reflexes. Considering these results in HwHLs, loudness model that account distorted cochlear filter for hyperacusis in individuals with cochear hearing loss looks more appropriate reason of hyperacusis in this group.

Loudness model also gets support from the fact that 4 out of 14 HwHLs had normal ARTs (Fig. 1), within 1-standard deviation of the ARTs of NwoHL. However, an expected relation between hyperacusis and the acoustic reflex cannot be completely denied in HwHL because of the presence of acoustic reflex abnormalities (absent acoustic reflexes and elevated ARTs) found in this group and the functional role of the acoustic reflex in decreasing sound input to the inner auditory system.

Acoustic Reflexes in HwoHL

There were 26 HwoHLs selected for the study, out of which 3 withdrew and 6 did not have acoustic reflexes leaving 17 in whom ART, ARA and ARL were measured. Unlike HwHL, HwoHL had all the three measured characteristics of the acoustic reflex; ART, ARA and ARL, different from NHwoH. ARTs, ARAs and ARLs were elevated, smaller and delayed, respectively, in HwoHL when compared to NHwoH. Abnormal ARTs, ARAs and ARLs in HwoHL indicate dysfunction in one or more anatomical units of the acoustic reflex pathway. As there was no associated hearing loss in this group deficit in the acoustic reflex pathway could be beyond the peripheral hearing system; probably at the level of auditory nerve, nuclei of the auditory brainstem (cochlear nucleus and superior livery complex, facial nerve and/or stapedius muscle. Abnormalities of ARTS, ARAs and ARLs in HwoHL including 6 HwoHLs who had absent acoustic reflexes point towards a strong relation between hyperacusis and the acoustic reflex (abnormality) in HwoHLs. There were 5 participants in HwoHL who had no abnormality in any of the measured characteristic of the acoustic reflex (Figs. 1, 2 and 3). Their ARTs, ARAs and ARLs were within 1-standard deviation of NHwoH. Since they had normal hearing, it is possible that hyperacusis in these 5 HwoHLs have origin involving the higher auditory system.

Several other inferences can be drawn from the results showing abnormal ARTs, ARLs and ARLs in HwoHL. There are a number of studies that have associated hyperacusis in patients having acute facial paralysis (all had normal hearing) with acoustic reflex abnormalities. It is likely that patients showing hyperacusis without hearing loss may be at high risk of facial paralysis. Stadepius muscle is the final anatomical unit in the acoustic reflex pathway, its weakness may lead to hyperacusis and as stapedius muscle don't have any role in hearing, hearing thresholds may remain normal. Mechanical disorder of the middle ear as a reason for hyperacusis was also suggested by Gordon (1986) previously [27].

Conclusion

This study further confirms acoustic reflex abnormalities in some individuals having hyperacusis. A weaker acoustic reflex pathway in such individuals may allow loud sound to reach the inner auditory system and cause discomfort. Clinically this study showed the efficacy to involve acoustic reflex testing in the assessment of hyperacusis especially when hyperacusis is not associated with hearing loss or any other medical condition that may lead to hyperacusis. This study provides evidence for the theoretical role of the acoustic reflex in loudness management and highlights the importance of studying hyperacusis with the perspective of the acoustic reflex.

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Availability of Data and Material The datasets generated during and/or analysed during the current study are available from the corresponding author on reasonable request.

Compliance with Ethical Standards

Conflict of interest The authors declare that they have no conflict of interest.

Ethical Approval All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

Informed Consent Informed consent was obtained from all individual participants included in the study.

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