Original Article

Insights from the Third International Conference on Hyperacusis: Causes, Evaluation, Diagnosis, and Treatment

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Abstract

Background: Hyperacusis is intolerance of certain everyday sounds that causes significant distress and impairment in social, occupational, recreational, and other day-to-day activities. **Objective:** The aim of this report is to summarize the key findings and conclusions from the Third International Conference on Hyperacusis. **Topics covered:** The main topics discussed comprise (1) diagnosis of hyperacusis and audiological evaluations, (2) neurobiological aspect of hyperacusis, (3) misophonia, (4) hyperacusis in autism spectrum disorder, (5) noise sensitivity, (6) hyperacusis-related distress and comorbid psychiatric illness, and (7) audiologist-delivered cognitive behavioral therapy for hyperacusis. **Conclusions:** Implications for research and clinical practice are summarised.

Keywords: Audiology, auditory system, hyperacusis, misophonia, noise sensitivity

Introduction

Hyperacusis is intolerance of certain everyday sounds that causes significant distress and impairment in social, occupational, recreational, and other day-to-day activities.[1] The sounds may be perceived as uncomfortably loud, unpleasant, frightening, or painful.[2] The Third International Conference on Hyperacusis (ICH3) gathered researchers and clinicians from 16 countries at Guildford, United Kingdom, on July 6 and 7, 2017. This report summarizes the key presentations and discussions at ICH3 and highlights their implications for research and clinical practice. The main topics discussed include (1) diagnosis of hyperacusis and audiological evaluations, (2) neurobiological aspect of hyperacusis, (3) misophonia, (4) hyperacusis in autism spectrum disorder (ASD), (5) noise sensitivity, (6) hyperacusis-related distress and comorbid psychiatric illness, and (7) audiologist-delivered cognitive behavioral therapy (CBT) for hyperacusis.

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Diagnosis of Hyperacusis and Audiological Evaluations

In the United Kingdom, audiologists who are specialized in tinnitus rehabilitation play a major role in providing support and therapeutic services to patients who experience hyperacusis.[2-4] Assessment of hyperacusis usually involves pure-tone audiometry, the measurement of uncomfortable loudness levels (ULLs), and self-report questionnaires, typically the hyperacusis questionnaire (HQ).[5] The pure-tone average (PTA) threshold across the frequencies 0.5, 1, 2, and 4 kHz provides a measure of the weakest sounds that can be detected for tones with different frequencies. In contrast, ULLs provide a measure of the sound

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level above which tones with different frequencies become uncomfortably loud. The average ULL across the audiometric frequencies is about 100 dB hearing level (HL) for normal-hearing people without hyperacusis.[6]

The average ULLs reported for patients with hyperacusis (when diagnosed *via* measures other than ULLs, such as questionnaires) vary widely across studies, from 66-dB HL (standard deviation, SD = 15),[7] to 77-dB HL,[8] and 83-dB HL (SD = 17).[9] This makes the diagnosis of hyperacusis based on ULLs difficult. The criteria for diagnosing hyperacusis handicap based on HQ scores are not generally agreed either. Khalfa *et al.*[10] suggested a cutoff score of 28 as indicating hyperacusis handicap. Meeus *et al.*[11] suggested reducing the cutoff score to 26, whereas Fackrell *et al.*[12] suggested that the cutoff score of 28 needs to be revaluated but did not propose a definitive value.

At ICH3, Brian C.J. Moore (University of Cambridge, UK) and Hashir Aazh (Royal Surrey County Hospital, UK) presented their recent research findings regarding the diagnosis of hyperacusis. They assessed the criteria for diagnosing hyperacusis based on measurements of ULLs and scores for the HQ for 573 consecutive patients who attended the Tinnitus and Hyperacusis Therapy Specialist Clinic.[13] Their results showed that a diagnosis of hyperacusis based on HQ scores can be made consistent with a diagnosis based on ULLs if the following cutoff scores are adopted for a positive diagnosis: the average ULL at 0.25, 0.5, 1, 2, 4, and 8 kHz for the ear with the lower average ULL, ULL_{min}, should be $\leq$77-dB HL, and the HQ score should be $\geq$22. With these cutoff values, 95% of patients with HQ scores meeting the criterion will also meet the criterion based on ULLs and *vice versa*. However, the cutoff values for ULLs and HQ scores proposed by Aazh and Moore[13] lead only to a binary decision; hyperacusis is either present or absent. Further work needs to be conducted in developing psychometric instruments to determine the severity of hyperacusis, its subtypes, and its impact on a patient's life.

At ICH3, Aazh and Moore also discussed possible problems that can arise during measurement of audiometric thresholds and ULLs when patients have unusually low ULLs. In extreme cases of hyperacusis, ULLs can be as low as 10 dB HL.[14] Such low ULLs raise the possibility that some patients will experience discomfort during routine audiometry and measurement of ULLs. The proportion of patients for whom this might happen was assessed in a recent study by Aazh and Moore.[15] The study was based on 362 consecutive patients who attended a National Health Service (NHS) audiology clinic for tinnitus and/or hyperacusis rehabilitation. Pure-tone audiometry was conducted using the procedure recommended by the British Society of Audiology (BSA)[16] for frequencies of 0.25, 0.5, 1, 2, 3, 4, 6, and 8 kHz. A similar procedure is used in many countries. According to this procedure, once the threshold has been determined at a given frequency, the initial level when assessing the threshold for the next frequency should be "at a clearly audible level (e.g. 30 dB above the adjacent threshold)" (p. 11), but never more than 80 dB HL. An experience of discomfort during pure-tone audiometry was deemed to be present when a test tone with a given frequency presented at 30 dB above the threshold for an adjacent frequency exceeded the ULL at the test frequency for at least one of the measured frequencies. Remarkably, the results showed that discomfort would have occurred for 21% of the patients. The incidence of discomfort would have been reduced to 10, 2.7, and 0.8% if the starting level had been 20 dB above, 10 dB above, or at the same level as the threshold for the adjacent frequency, respectively.

In the study of Aazh and Moore,[15] ULLs were also measured using the BSA recommended procedure.[16] According to this, the audiologist should "Start testing at 60 dB HL or at the subject's hearing threshold level for that ear at that frequency, whichever is highest, unless otherwise indicated (Section 2.2)" (p. 7). An experience of discomfort during measurement of ULLs was deemed to be present if the starting level of 60 dB HL exceeded a patient's ULL for at least at one of the measured frequencies. Discomfort would have occurred for 24% of the patients using this criterion. The incidence of discomfort would have been reduced to 3.6% if the starting level had been reduced to 30 dB HL and to 0.5% if the starting level had been reduced to 15 dB HL.

Given the high prevalence of anxiety and stress in patients seeking help for tinnitus and hyperacusis,[17] it is very important to ensure that any evaluation procedures do not lead to unnecessary discomfort. If discomfort is experienced, this might trigger further anxiety and stress, leading to worsening of the symptoms and to possible loss of trust in the audiologist. This in turn might reduce the effectiveness of any therapy performed by the audiologist after the initial evaluation. To avoid discomfort during PTA, Aazh and Moore[15] suggested using an initial level of 0 dB HL at the starting frequency of 1 kHz and setting the level for subsequent frequencies to be equal to the level at threshold for the previously tested frequency. To avoid discomfort during measurement of ULLs, they recommended that the starting level for a given test frequency should be equal to the measured audiometric threshold at that test frequency and that levels above 80 dB HL should not be used.

Neurobiological Aspect of Hyperacusis

The mechanisms underlying hyperacusis are unknown. One possibility is that neurons that normally respond at higher sound levels begin to respond to sounds with lower levels, leading to the perception of increased loudness. Another possibility is that hyperacusis (as well as tinnitus) may result from increased neural synchrony and reorganization of the tonotopic map in the auditory cortex. Although there is limited evidence for increased brain activity in the auditory cortex of people with hyperacusis, there is a growing body of

literature suggesting such changes in the brains of animals with salicylate-induced or noise-induced hearing loss.[18-23] Behavioral experiments with these animals showed enhanced acoustic startle responses, which are assumed to be related to hyperacusis. However, the interpretation of the results of such animal experiments is difficult, and it is not clear whether the observed neurophysiological changes are related to hearing loss, hyperacusis, or tinnitus.[18]

Marlies Knipper (Department of Molecular Physiology of Hearing, Hearing Research Institute Tübingen, Germany) presented data from neurobiological laboratory studies that aimed to distinguish the effects of hyperacusis from those of tinnitus and hearing loss. Her laboratory previously developed a behavioral animal model of tinnitus.[24,25] With this model, certain biomarkers could be used to distinguish equally hearing-impaired animals with and without tinnitus. These biomarkers included molecular changes in hair cells and their synapses, changes in the number of auditory fiber numbers, changes in activity-dependent plasticity genes, and several physiological changes, including tests of outer hair cell function, summed auditory nerve activity, suprathreshold early and late sound-evoked response amplitudes, and field potentials. For reviews, see Knipper *et al.*[26,27] and Ruttiger *et al.*[28] Building on this work, Knipper's laboratory has now developed an animal model for hyperacusis. It is known that exposure to a very intense noise often, but not always, leads to tinnitus and/or hyperacusis in humans. In Knipper's laboratory, animals were exposed to the type of noise that produces tinnitus and/or hyperacusis in humans. It seems reasonable to assume that some animals exposed to the noise will develop tinnitus and/or hyperacusis, and some will not. Despite no distinguishable hearing threshold difference (based on the measurement of auditory brainstem responses), it was found that the animals could be subdivided on the basis of behavioral measures into groups with (i) no tinnitus and no hyperacusis, (ii) tinnitus but no hyperacusis, (iii) hyperacusis but no tinnitus, and (iv) tinnitus and hyperacusis. The results also confirmed what has been reported previously for men and rodents: hyperacusis is not primarily linked to an elevation of hearing thresholds or impairment of outer hair cell function. Rather, whether or not hyperacusis and tinnitus occur is related to differences in central responsiveness to peripheral auditory fiber damage. The findings also indicate that differences in central responsiveness linked with tinnitus and hyperacusis are associated with differences in a memory reinforcement system that is involved in strengthening auditory circuits. Moreover, the findings support a crucial role of the history of stress levels in driving central adaptive responses to peripheral neuronal impairments that lead to tinnitus or hyperacusis.[25] The differences in central response pattern observed between animals with various combinations of hyperacusis and tinnitus are currently being compared with features in defined patient groups with matched degrees of hearing loss.

Martin Schecklmann (Department of Psychiatry and Psychotherapy, University of Regensburg, Germany) described earlier work of his group demonstrating that hyperacusis as indicated by screening questions from the Tinnitus Research Initiative (TRI) database[29,30] is associated with specific demographic, tinnitus, and clinical characteristics.[31] For example, patients with chronic tinnitus and hyperacusis (in contrast to patients with only tinnitus) were more seriously handicapped, showed a higher influence of stress on their tinnitus, and rated the pitch and loudness of their tinnitus as higher and their hearing function as worse. However, measures of tinnitus pitch, tinnitus loudness, and hearing thresholds did not reveal group differences. In another work, Schecklmann *et al.*[32] validated the screening questions of the TRI database[29,30] by using the German hyperacusis questionnaire "GÜF" for a sample of patients with chronic tinnitus, some of whom also had hyperacusis.[33] The original proposed factor structure of the GÜF could not be replicated. Factors of the GÜF for this sample of patients were found to be quality of life, hearing difficulties, and fear–pain hyperacusis. These factors match well with the characteristics of patients with hyperacusis as determined using the TRI database analysis.[31] Relative to patients with tinnitus alone, patients with hyperacusis were more seriously handicapped and had a reduced quality of life. The latter also reported that their tinnitus was more strongly modulated by stress induced by emotional factors. These findings highlight the need to consider hyperacusis subtypes both in clinical settings and for scientific work.

Schecklmann then presented preliminary data on resting state electroencephalography (EEG) for a sample of 42 patients with chronic tinnitus, some of whom also had hyperacusis. The aim of this work was to assess whether those without and with hyperacusis had different resting state brain oscillatory activity. This was done by determining the correlation between scores for single items of the GÜF and the amount of EEG activity in different frequency bands. Theta activity in bilateral temporal and frontal areas was correlated with emotional aspects of the GÜF, central beta-3 activity was correlated with quality of life, and gamma activity over all sensors was correlated with hyperacusis in general. These findings corroborate the existence of hyperacusis subtypes on a phenotypic and neuronal level. EEG might be helpful in disentangling different forms of hyperacusis for patients with chronic tinnitus.

Misophonia

Misophonia is defined as an abnormally strong emotional and behavioral reaction to particular sounds or groups of sounds that have a specific pattern and meaning to the patient,[34] although a consensus paper bringing together experts from a wide range of disciplines suggested using the term "annoyance hyperacusis" for the experience of negative emotional reaction to sound.[2] Andrea E. Cavanna (Department of Neuropsychiatry, Birmingham, UK) discussed the possible mechanisms underlying misophonia. Although misophonia is not listed in any major psychiatric classification system, a few psychiatric clinics have reported

treating patients with misophonia.[35-38] Systematic studies of misophonia are very limited.[39] In the field of psychiatry, there is a growing debate as to whether misophonia is a distinct psychiatric disorder or a variety of hyperacusis that is often comorbid with neuropsychiatric disorders.[37,39-42] Specifically, neurodevelopmental disorders including Tourette syndrome[43] and ASD (see the following section) have been shown to be associated with misophonia in a proportion of cases, suggesting shared neurodevelopmental trajectories.

Most research studies in the field of audiology have not distinguished misophonia from hyperacusis.[9,12,31,32,44-46] Jastreboff and Jastreboff[47] reported that people with severe hyperacusis always experience misophonia. Recent studies conducted within the NHS in the United Kingdom suggest that severe hyperacusis is typically characterized by strong across-frequency variations in sensitivity to sound, which is an indication of adverse reaction only to specific sounds, a feature that is associated with misophonia.[13,14]

Based on the above mentioned studies, it is likely that misophonia forms part of the spectrum of hyperacusis and needs to be considered when assessing patients with sound intolerance complaints.

Hyperacusis in Autism Spectrum Disorders (ASDs)

Many researchers have studied the hearing status and auditory features of children with autism or ASDs. Ali A. Danesh (Florida Atlantic University, USA) described how abnormalities of the auditory system can result in hearing loss, tinnitus, and hyperacusis.[48] Additional auditory abnormalities may include inability to localize the source of a sound and difficulty understanding speech, particularly in noisy environments.[48,49] Of these problems, hyperacusis is probably the most common. Unusual behaviors such as social avoidance and exaggerated covering of the ears are two common traits in the ASD population, and these are a result of an emotional response to sounds *via* stimulation of the limbic system and the autonomic nervous system.[50-52] This behavior suggests a defensive response to an obnoxious stimulus. When this behavior is exaggerated, it manifests itself as hyperacusis. Previous studies have shown that hyperacusis occurs in 18% of individuals with autism.[53] Danesh *et al*.[54] reported that 38 out of 55 patients (69%) with Asperger's syndrome had hyperacusis and 19 (35%) had tinnitus.

Hyperacusis could be a sign of auditory pathway problems or a sign of abnormalities in the limbic system.[55,56] It has been suggested that the limbic system generates a negative emotional reaction to sounds and relays it to the auditory cortex. The auditory cortex in turn triggers the perception of the negative reaction, causing hyperacusis.[57,58] However, clinicians should be aware that hyperacusis in the ASD population may originate from other and less expected factors. An imaging study on 14 patients with ASD investigated the relationship between hypersensitivity to sound and superior semicircular canal dehiscence disorder.[59] This is a disorder where the bone between the superior semicircular canal and the cranial vault is very thin or absent, and its incidence in the general population is about 2%.[60] Remarkably, Thabet and Zaghloul[59] reported that 29% of individuals with ASD combined with hypersensitivity to sound had superior semicircular canal dehiscence as revealed by computerised tomography (CT) imaging.

Some cases of hyperacusis may be linked to reduced effectiveness of the efferent pathways in the auditory system.[2] One aspect of the operation of the efferent system can be assessed by recording otoacoustic emissions (OAEs) with and without contralateral noise stimulation; this assesses the integrity of the medial olivocochlear bundle (MOCB) pathway.[49] It is well known that reduction of OAE responses by contralateral stimulation reflects the normal inhibitory function of the MOCB on the outer hair cells.[61] Danesh and Kaf[62] measured distortion-product OAE (DPOAE) amplitudes for children with autism and compared them with those for an age-matched control group. In the absence of noise, the DPOAE amplitudes were reduced for the children with autism, suggesting cochlear dysfunction. When DPOAEs were recorded in the presence of contralateral noise, the suppression effect was weaker for the children with autism than for the control group, suggesting dysfunction of the MOCB. The findings of Danesh and Kaf[62] may explain why many children with autism suffer from hypersensitivity to sounds and difficulty hearing in background noise, as the efferent system appears to play a role in the latter effect.[63]

A similar study by Kaf and Danesh[64] measured DPOAEs and contralateral suppression in children with Asperger's syndrome (N = 18 males) and an age-matched control group. Interestingly, there was no significant difference in DPOAE amplitudes and contralateral suppression between the two groups. Comparison of the studies of Danesh and Kaf[62] and Kaf and Danesh[64] suggests that the diminished DPOAE suppression effect may provide neurophysiological evidence for reduced MOCB function mainly in children with low-functioning autism, but not in children with high-functioning autism.

Noise Sensitivity

Noise sensitivity is a personality trait characteristic involving underlying attitudes toward noise in general.[65,66] It refers to the physiological and psychological internal states of the individual, which affect the degree of reactivity to noise.[67] Noise sensitivity aggregates in families; the presence/absence of noise sensitivity is higher for first-degree relatives than for the general population, with heritability estimated as 36%.[68] Noise sensitivity is a predictor of noise annoyance.[69] Annoyance is a multifaceted psychological concept, covering immediate

behavioral effects of noise, such as disturbance of concentration and interference with activities, and evaluative aspects like "nuisance," "disturbance," "unpleasantness," and "getting on one's nerves."[70]

Psychometric tools have been designed to assess noise sensitivity and annoyance.[71-74] Examples include Weinstein's noise sensitivity (WNS) scale[74] and the noise annoyance sensitivity scale.[71] Although these questionnaires have been widely used in public health, occupational health, and environmental research, they have not been used in clinical settings for patients with hyperacusis. Most of the items on these questionnaires were developed to assess the individual's attitude toward noise, with a focus on the impact of environmental noise as opposed to assessment of the severity of sensitivity to sound and its impact on the individuals' quality of life; the latter are needed in the clinical assessment of patients with hyperacusis. Nevertheless, it seems that individuals with high noise sensitivity exhibit similar symptoms to patients with hyperacusis.[75,76]

In some medical reports, mainly in the literature related to traumatic brain injury and postconcussion syndrome, the terms noise sensitivity and hyperacusis have been used interchangeably to describe intolerance to sound.[76,77] Viziano *et al.*[78] reported a strong correlation between scores on the WNS and scores on the HQ for a group of patients affected by multiple chemical sensitivity ($r = 0.9$, $P < 0.01$).

Marja Heinonen-Guzejev (Department of Public Health, University of Helsinki, Finland) discussed neurophysiological studies of noise sensitivity. Similar to hyperacusis, a comprehensive model of the mechanisms underlying noise sensitivity is lacking. Both psychological and biological factors may be involved. Noise-sensitive individuals seem to exhibit less sensory gating than noise-resistant individuals.[79] Marja Heinonen-Guzejev discussed a recent study conducted at the University of Helsinki that assessed neuronal sound processing in relation to noise sensitivity using combined EEG and magnetoencephalography (MEG).[80] Patients were tested using a fast multifeature mismatch negativity (MMN) paradigm that included six types of sound feature deviations from a reference sound (a piano tone). The MMN can be used to assess sound discrimination accuracy.[81] Patients with high noise sensitivity had smaller P1 amplitudes than less noise-sensitive patients, suggesting that the former may have difficulties with sound feature encoding. Furthermore, their MMN, a response that reflects deviance detection, was diminished. This was especially apparent for a deviant with increased noisiness. Noise sensitivity was specifically related to the processing of noise-like properties, but not other features. The results of this study indicate that at least two stages of preattentive cortical sound processing are affected by noise sensitivity. The functional changes in the auditory system observed for noise sensitive individuals could result from the susceptibility of their central auditory system to detrimental noise effects. Studying the neuronal mechanisms of sound processing may help in understanding the origin of noise sensitivity.[80]

Johan Paulin and Linus Andersson (both from the Department of Psychology, Umeå University, Sweden) presented preliminary results from an ongoing noise-exposure study, inspired by studies of chemical intolerance (often referred to as odor hypersensitivity). Individuals with chemical intolerance differ from healthy controls in how they rate the perceptual properties of and react to odorous exposure, especially after extended exposure.[82,83] Paulin and Andersson assessed whether individuals with noise sensitivity react to extended white noise exposure in a way that is comparable to how people with chemical intolerance react to smells. Participants with and without self-reported noise sensitivity were screened for hearing deficits, fitted with electrodes to register their pulse, and seated inside a sound-attenuating chamber. Following 11 min of silence, white noise was gradually increased in level for 9 min and then held constant at 60 dB sound pressure level (SPL) during the remaining 25 min of the session. Gradually changing stimuli were used to remove the availability of perceptual anchors. Without the possibility of anchoring ratings to an unchanging stimulus, possible perceptual changes due to sensitization and habituation processes were arguably enhanced.

Paulin and Andersson discussed two ways of analyzing the data. One method was to assign participants to high, intermediate, and low noise-sensitivity groups based on their self-reported problems in daily life, as assessed with the WNS.[74] The high-sensitivity group rated the exposure as more intense, unpleasant, and symptom-eliciting than did the low-sensitivity group, with the intermediate-sensitivity group giving intermediate ratings. The high noise-sensitivity group had lower heart rate variability throughout the session, which is an autonomic nervous system measure of distress.[84] The other method of analysis involved assigning participants to three groups according to the rated unpleasantness of the white noise. The outcomes were similar to those found in the first analysis, but the effect sizes were generally larger. Those who regarded the white noise as least unpleasant also rated the noise as decreasing in magnitude over time, which can be interpreted as a form of perceptual adaptation or habituation. This effect did not occur for the other two groups. Finally, there was a tendency for those who rated the noise as unpleasant to rate the smell inside the chamber as more intense than did the other two groups.

In summary, the preliminary analyses revealed considerable variability between individuals, not only in terms of affective responses and symptoms but also in terms of the perceived intensity of the noise exposure. However, assigning participants to different groups in terms of noise sensitivity is not a trivial matter, and different ways of assigning participants can lead to different outcomes. Given the overlap between noise sensitivity and chemical intolerance,[85] the similarities in other measures of

distress,[86] and comparable responses to extended exposure, Paulin and Andersson suggested that it may be fruitful to look for intolerances other than to sound in hyperacusis patients.

Hyperacusis-Related Distress and Comorbid Psychiatric Illness

Hashir Aazh and Brian Moore discussed the process of distinguishing hyperacusis-related distress from the distress caused by other psychological disorders for people who have hyperacusis, using self-reported psychological questionnaires and in-depth interviews. The first task is to screen for any comorbid psychological disorders, as these seem to be highly prevalent among patients with hyperacusis.[17] The following questionnaires were assessed for their acceptability and personal relevance to patients with tinnitus and/or hyperacusis (n = 150): Generalized Anxiety Disorder (GAD-7), Short Health Anxiety Inventory (SHAI), MINI-Social Phobia Inventory (Mini-SPIN), Obsessive Compulsive Inventory-Revised (OCI-R), Panic Disorder Severity Scale-Self Report (PDSS-SR), Patient Health Questionnaire (PHQ-9), and Penn State Worry Questionnaire-Abbreviated version (PSWQ-A).[17] All questionnaires except the PDSS-SR were rated as relevant and recommended for use. There were significant relationships between hyperacusis handicap and responses on the PHQ-9, SHAI, Mini-SPIN, PDSS-SR, and PSWQ.[17] The relative risk ratio of abnormal scores on the PHQ-9, SHAI, Mini-SPIN, PDSS-SR, and PSWQ increased by factors of 2.7 [95% confidence interval (CI): 1.04–7.13], 4.05 (95% CI: 1.59–10.3), 3.4 (95% CI: 1.4–8.09), 4.4 (95% CI: 1.5–12.8), and 2.5 (95% CI: 1.2–7.3), respectively, for patients with HQ scores above 26 relative to scores for those with scores below 26.[17]

The use of these questionnaires can help audiologists to screen for underlying psychological disorders in patients with hyperacusis and make appropriate onward referral to mental health services. In addition, abnormal scores on these questionnaires highlight the possible effect of the underlying psychological condition on a patient's experience of sound intolerance, which can help the audiologist when they conduct in-depth interviews to explore hyperacusis-related distress.

Abnormal scores on the psychological questionnaires do not necessarily mean that the patient would not benefit from audiologist-delivered CBT for their hyperacusis. Hashir Aazh proposed that in-depth interviews should be used to explore the patient's experience, behavior, emotions, and perceptions.[87] During such interviews, patients are encouraged to talk about a typical day (e.g., tell me a bit more about how your hyperacusis affects your activities and/or mood on a typical day?). Throughout, the principle of guided discovery[88] is employed, in which the patient makes discoveries guided by careful questioning from the clinician. CBT for hyperacusis is only needed if the patient experiences current hyperacusis-related distress, in the other words, if their day-to-day activities or mood are affected due to their sound intolerance. Hashir Aazh reported that 68.5% (124/181) of patients with abnormal scores on the HQ and/or tinnitus handicap inventory (THI) (in the case of comorbid tinnitus) presented with tinnitus- and/or hyperacusis-related distress. For 31.5% patients (57/181), there was no current tinnitus and/or hyperacusis-related distress. For 17 out of 57 patients, it was agreed that the emotional disturbances they were experiencing did not seem to be related to their tinnitus and/or hyperacusis and were more likely to be related to an underlying psychological disorder. Hence, they were referred for further psychological evaluations and treatment (when needed). Hashir Aazh discussed a case study of a patient who was referred for hyperacusis management for whom the in-depth interview revealed that the main reason for the distress she was experiencing was her symptoms of psychosis and visual hallucinations, which she felt were more likely to happen in noisy situations. The conclusion was that although she was experiencing intolerance to sound, the root cause of the problem was not hyperacusis. Hence, she was referred to the Early Intervention in Psychosis Service.

Brian Moore and Hashir Aazh discussed the mechanisms by which depressive symptoms can develop for patients with hyperacusis. Data were gathered from the records of 620 consecutive patients who sought help concerning their tinnitus or hyperacusis from an audiology clinic in the United Kingdom. One-third of the patients had borderline abnormal or abnormal scores on the depression subscale of the Hospital Anxiety and Depression Scale (HADS-D). Mediation analyses, which attempt to infer the likely path of influence of one variable upon another, showed that the small influence of ULLs on HADS-D scores was fully mediated by hyperacusis handicap and anxiety.[89] Therefore, the mechanism that produces depression in patients with hyperacusis does not seem to be explained by reduced ULLs. Future research should focus on factors that might lead to depression for patients with hyperacusis.

Audiologist-Delivered Cognitive Behavioral Therapy (CBT) for Hyperacusis

In collaborative work between researchers in audiology and clinical psychology, Aazh and Allott[90] published an audiologist-delivered CBT protocol for hyperacusis rehabilitation. Aazh and Moore[1] assessed patients' views about the effectiveness of audiologist-delivered CBT for the management of tinnitus and hyperacusis. Of 75 patients who received audiologist-delivered CBT as a part of their tinnitus and/or hyperacusis rehabilitation, 56% ranked its effectiveness as 5/5 (where 1 represents no effect and 5 represents very effective) and a further 29% as 4/5. A comparison of patients' feedback about the effectiveness of audiologist-led CBT between patients with tinnitus only and those with hyperacusis (with or without tinnitus) showed no significant difference (P = 0.05).

Hashir Aazh and Anna Julia (Royal Surrey County Hospital, UK) discussed the procedures involved in audiologist-

delivered CBT and its clinical effectiveness. The treatment involves six CBT sessions across 6 weeks. Each session involves individual face-to-face interaction with an audiologist and lasts for about 60 min. Although audiologist-delivered CBT is resource intensive, only 17% of patients receive the full treatment, as only patients with clear tinnitus and/or hyperacusis-related distress are offered the treatment, and some decline the treatment or drop out. The audiologist-delivered CBT using this protocol showed promising results with regard to changes in HQ score.[91] It is, therefore, recommended that researchers consider using this protocol when designing future randomized controlled trials assessing the effect of audiologist-delivered CBT for hyperacusis management. Details of audiologist-delivered CBT are described elsewhere.[92]

Some Take-Home Messages

(1) The diagnosis of hyperacusis based on ULLs can be made consistent with a diagnosis based on HQ scores by appropriate choice of cutoff values for the two measures. Recommended cutoff values are ULL_{min} $\leq$77 dB HL and HQ score $\geq$22.

(2) The procedures recommended by the BSA for conducting pure-tone audiometry and measuring ULLs need modification to avoid discomfort for patients seeking help for tinnitus and/or hyperacusis. For audiometry, the recommendation is to use an initial level of 0 dB HL at the starting frequency of 1 kHz and to set the level for subsequent frequencies to be equal to the level at threshold for the previously tested frequency. For ULLs, the starting level for a given test frequency should be equal to the measured audiometric threshold at that test frequency and levels above 80 dB HL should not be used.

(3) Animal models of tinnitus, hyperacusis, and the combination of the two have been developed. Animals falling into different subgroups can be distinguished using both physiological and behavioral measures. The results suggest that hyperacusis is not primarily linked to an elevation of hearing thresholds or impairment of outer hair cell function. Rather, whether or not hyperacusis and tinnitus occur is related to differences in central responsiveness to peripheral auditory fiber damage.

(4) Subgroups of humans with different combinations of tinnitus and hyperacusis can be distinguished using the amount and cortical distribution of different types of EEG activity. Relative to patients with tinnitus alone, patients with hyperacusis were more seriously handicapped and had a reduced quality of life. The latter also reported that their tinnitus was more strongly modulated by stress induced by emotional factors.

(5) Based on the literature, it is not clear whether misophonia is a symptom of a distinct psychiatric disorder or represents a comorbidity of hyperacusis with an underlying psychiatric illness. Further research on this topic is needed.

(6) Hyperacusis occurs commonly in individuals with ASD and seems to be associated with subtle cochlear dysfunction and reduced effectiveness of the efferent system.

(7) Individuals with high noise sensitivity seem to exhibit similar symptoms to patients with hyperacusis. High noise sensitivity can be detected using EEG and MEG and is associated with reduced sensitivity to noise-like deviant sounds.

(8) The GAD-7, SHAI, Mini-SPIN, OCI-R, PSWQ-A, and PHQ-9 psychological questionnaires are recommended for evaluation of psychological problems for patients seeking help for tinnitus and/or hyperacusis. Abnormal results on these questionnaires may indicate the need for referral for possible treatment of psychological problems.

(9) In-depth interviews can be used in addition to self-report questionnaires to assess whether a patient is experiencing hyperacusis-related distress, that is, to assess whether their day-to-day activities or mood are affected by their sound intolerance. About two-thirds of patients with abnormal scores on the HQ and/or THI may have current distress linked to their hyperacusis and/or hyperacusis. These patients may benefit from CBT.

(10) Audiologist-delivered CBT gave promising results with regard to changes in hyperacusis handicap scores. Hence, it is recommended that researchers consider using this procedures described by Aazh and Moore[91,92] when designing future controlled trials.

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Conflicts of interest

There are no conflicts of interest.

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