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Insights from the first international conference on hyperacusis: Causes, evaluation, diagnosis and treatment

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Abstract

The First International Conference on Hyperacusis gathered over 100 scientists and health care professionals in London, UK. Key conclusions from the conference included: (1) Hyperacusis is characterized by reduced tolerance of sound that has perceptual, psychological and social dimensions; (2) there is a growing awareness that children as well as adults experience symptoms of hyperacusis or misophonia; (3) the exact mechanisms that give rise to hyperacusis are not clear, but the available evidence suggests that functional changes within the central nervous system are important and in particular, hyperacusis may be related to increased gain in the central auditory pathways and to increased anxiety or emotional response to sound; (4) various counseling and sound therapy approaches seem beneficial in the management of hyperacusis, but the evidence base for these remains poor.

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Full Text

Introduction

Hyperacusis is an umbrella term for several types of intolerance to sound. Although such symptoms are common, the topic has attracted little academic interest to date. The First International Conference on Hyperacusis was organized by the tinnitus and hyperacusis therapy team of the Royal Surrey County Hospital and held at Birkbeck College,

University of London, UK, on 1 st -2 nd March 2013. Over 100 scientists and health care professionals from 27 countries took part in this event. This report summarizes the highlights from the conference on topics related to assessment, hyperacusis in pediatric populations, mechanisms of hyperacusis and treatment strategies including sound therapy.

Assessment of Hyperacusis

Tyler et al. (University of Iowa, USA) suggested the following classification of hyperacusis:

"Loudness hyperacusis" - some moderately loud sounds are very loud;"Annoyance hyperacusis" - some sounds are annoying (not always loud);"Fear hyperacusis" - patients are afraid of some sounds (not always loud).

Questionnaires to assess the severity and disability associated with each of the above categories [1] were used in a study of over 200 people with hyperacusis. This showed that louder sounds are generally more annoying and softer sounds are less likely to evoke fear hyperacusis. The most common etiological factor is noise exposure. In 10% the symptom is unilateral. About 90% report concurrent tinnitus (link first identified by Tyler and Conrad-Arnes [2]). Music, screaming and sirens are the most common sounds associated with fear hyperacusis. Loudness and annoyance hyperacusis are more closely linked to one another than they are to fear hyperacusis. Some patients with severe hyperacusis are also bothered by strong smells, tastes and bright lights and report headaches and balance problems. It was suggested that use of this classification facilitates the treatment of hyperacusis patients using a protocol called tinnitus activities treatment. [3]

Jastreboff and Jastreboff (Emory University, USA) proposed a different taxonomy for decreased sound tolerance (DST); defining DST as the presence of negative reactions experienced by a subject as a result of exposure to sounds that would not evoke such aversive reactions in the average listener. They suggested that DST can be subdivided into hyperacusis, misophonia, or a combination of these conditions. [4],[5],[6] In this classification, hyperacusis is defined as a negative reaction to a sound dependent only on its physical characteristics, namely its spectrum and intensity. The sound's meaning and the context in which it occurs are irrelevant. Misophonia is characterized as negative reactions to a sound with a specific pattern and meaning to an individual patient. In misophonia the physical characteristics of the sound are secondary and the reactions to the sound are thought to depend on non-auditory factors such as the patient's previous evaluation of that sound and the context in which the sound is presented. Misophonia can include a variety of negative emotions such as dislike, annoyance, hate, discomfort, fear. In this classification a further type of DST, phonophobia, is defined as a subset of misophonia in which fear is the dominant factor. A hypothesis for the mechanisms of hyperacusis and misophonia was proposed together with a treatment paradigm based on tinnitus retraining therapy (TRT) (see below).

Both the Tyler and Jastreboff classifications have pros and cons: Tyler's uses simple terminology and is perhaps more adaptable; Jastreboff's is the current dominant classification and has been in use for a longer period of time. Both are linked to management strategies. Both classifications, however, have attracted criticism and people with reduced sound tolerance have devised other terms such as "selective sound sensitivity syndrome". In this complex field, standardization of terminology would be beneficial.

Hyperacusis in Children

Veronica Kennedy (Bolton NHS Foundation Trust, UK) described the different profiles of children presenting to a pediatric audiology service with reported impaired tolerance to sounds. Prevalence studies in children are difficult to undertake and figures vary widely from 6% to 42% increasing to 90% in those with Williams syndrome. [8],[9],[10] She suggested that reduced tolerance to sounds seems particularly common in children who have had a temporary hearing loss due to wax or otitis media with effusion that has then been corrected. Anecdotally it is commonly reported to occur temporarily after insertion of ventilation tubes. It is also noted in children with learning and/or social and communication difficulties including those on the autistic spectrum or with Williams Syndrome. Common troublesome sounds include sounds of vacuum cleaners, washing machines or emergency vehicles. The child's

reaction ranges from covering ears to crying and screaming. For children with mild distress, management may involve an explanation of the symptom and advice on coping strategies. For those children who are markedly distressed, the management may involve auditory desensitization, behavioral desensitization or both. A multi-agency or multi-disciplinary approach is recommended.

Mechanisms of Hyperacusis

Salvi et al. (University at Buffalo, USA) discussed the possible role of increased gain within the central auditory pathways in the development of hyperacusis. Sensorineural hearing loss reduces the neural output from the inner hair cells through the auditory nerve and into the central auditory system. The central auditory pathway, unlike a hardwired device, shows a remarkable capacity to respond to a severely diminished input by increasing its gain and upregulating its response to sound, a phenomenon often referred to as auditory plasticity. Increases in central gain are frequently, but not always, seen in the local field potentials and single unit firing rates recorded from the auditory cortex, medial geniculate body and inferior colliculus following cochlear injury. Despite the reduced output from the cochlea, neural responses in the auditory cortex and inferior colliculus can be enhanced by more than 50%. Using an animal model of drug induced hearing loss using salicylate, there is a substantial increase in the auditory evoked potential in the auditory cortex. This is accompanied by a substantial increase in the amplitude of the acoustic startle response, a behavioral reflection of enhanced sensitivity to supra-threshold sounds. Although the biological mechanisms responsible for the enhanced sensitivity to high level sounds are not fully understood, there is growing evidence that it may arise from the loss of gamma-aminobutyric acid-mediated inhibition at multiple levels along the auditory pathway. While increasing the gain of the central auditory system may partially compensate for peripheral hearing loss, excessive gain may lead to hypersensitivity to supra-threshold sounds. [11],[12],[13]

This neural plasticity theory remains contentious. First, although behavioral experiments in animals illustrate enhanced acoustic startle responses which is assumed to be related to hyperacusis, interpretation of the results of such experiments is difficult and it is not clear whether the observed neurophysiological changes relate to hearing loss, hyperacusis or tinnitus. [14] Second, many patients suffering from hyperacusis present with normal or near normal audiometric thresholds. Therefore, the existence of abnormally increased auditory gain in humans remains to be evaluated. Thirdly, human studies indicate that auditory deprivation can only decrease loudness discomfort levels (LDLs) by 7 dB relative to their normal levels. [15],[16] This is not consistent with the 40-50 dB shift in LDLs which is common among patients with hyperacusis.

Sound Therapy

Implications of experimental studies for clinical practice

Wideband noise generators (WNG) are commonly used in the management of hyperacusis. Human studies indicate that just as sound tolerance can be decreased by sound deprivation, sound exposure can increase LDLs by 7 dB. [15],[16] Studies conducted in animals indicate that long-term sound exposures can alter the response properties of the auditory cortex to acoustic stimulation. [17],[18] General consensus among the conference delegates was that use of WNGs is safe as long as the sound is adjusted to a comfortable level for the patient. However, there is a lack of supporting evidence and this is an area where research is required.

Management Strategies

Jastreboff and Jastreboff (Emory University School of Medicine, USA and JHDF, Inc.) discussed their approach to DST based on the neurophysiological model of tinnitus [19] and their clinical experience treating patients using TRT. [4],[5],[6],[7] The principles and implementation of counseling, sound therapy protocols and optimal use of ear-level instrumentation were presented for the treatment of hyperacusis and misophonia. Challenging cases, including tensor tympani syndrome and autism were used to illustrate the critical differences between treatments of DST dominated by

hyperacusis or misophonic components. It was postulated that the proper diagnosis and treatment of hyperacusis and/or misophonia frequently result in complete resolution of DST in affected patients. Anecdotally, TRT has been reported [20] to give good results for the treatment of DST. In a prospective clinical cohort study, 68 patients with DST treated with TRT had statistically significant improvement of their LDLs. [21]

Hashir Aazh (Royal Surrey County Hospital, UK) introduced psychological models of hyperacusis with a focus on theoretical underpinnings and clinical implementation using cognitive behavioral therapy (CBT). Although a causal relationship between anxiety and hyperacusis is unproven, there is a growing body of evidence suggesting a possible link. CBT is a psychological intervention which is aimed at helping the patient to modify their unhelpful thoughts and safety-seeking behaviors so as to alleviate anxiety. [22] In the absence of a cure for hyperacusis, treatment of accompanying anxiety could be beneficial.

There are no randomized controlled trials comparing various treatments for hyperacusis and therefore further research studies are required to elucidate optimal treatment options for patients. Furthermore, in the case of CBT, a stripped down version of the technique has been being taught to some audiologists in the UK. It is not yet known whether CBT delivered in this fashion is as efficacious as that delivered by clinical psychologists and this is another area where further work is required.

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