Hyperacusis

Jastreboff, M. M., and Jastreboff, P. J. (2001). Hyperacusis. Audiology Online. (accessed July 11, 2025).

Margaret M. Jastreboff,Pawel J. Jastreboff

Hyperacusis has recently attracted professional attention. Previously, this topic was not well researched or documented. In many instances, due to a lack of understanding regarding the diagnosis, the pathophysiology and treatment options, patients' complaints were ignored.

Terms such as 'oversensitive hearing,' 'hyperacusis,' 'phonophobia,' 'recruitment,' 'dysacusis,' and 'auditory hyperesthesia,' were used interchangeably (and incorrectly) to describe decreased sound tolerance, and discomfort or pain in the ears, associated with sound exposure. Hyperacusis has been reported as an isolated finding or as a component of multi-symptom diagnosis.

Epidemiologic studies and prevalence rates are not well described, mechanisms are speculative and anatomic sites and etiology are, unfortunately, not well documented. Most typically, advice offered to those who insisted upon help was to 'use ear plugs,' or 'learn to live with it.'

This report is not a comprehensive review of the literature regarding hyperacusis. Rather, this article will offer a brief description of the problem and will attempt to bring to your attention some questions and topics you might find interesting, or choose to explore, relating to hyperacusis.

'My ears are painful when I am exposed to sound.' 'My ears are very sensitive to sounds.' 'I am afraid about my hearing.' 'When I am in a noisy surrounding I experience a troublesome sensation in my ears.' These, and similar statements, are not unusual in audiology or otolaryngology clinics.

Decreased sound tolerance can have an extremely strong impact on patients lives. It can *prevent* people from exposing themselves to louder environments and it can prevent people from working and interacting socially. Importantly, it can prevent patients from enjoying a variety of life activities. In extreme cases, decreased sound tolerance can totally control a patient's life. In these cases, patients do not leave their homes. Their lives, and the lives of their families, are totally controlled by the issue of avoidance of sound.

Clinical observations reveal that in many cases, decreased sound tolerance is a complex phenomenon and frequently consists of more that one problem. In some cases, it is not necessarily loud sounds, but even quiet sound, which can cause discomfort. It has been recognized that decreased sound tolerance might reflect a physical discomfort, or can be related to a dislike or a
fear of sound.

Our approach to decreased sound tolerance is based on neurophysiology. We attempt to recognize neuronal systems which may be involved in decreased sound tolerance including the peripheral and central auditory systems, the limbic and the autonomic nervous systems. Consequently, we propose the following specific definitions.

Hyperacusis can be defined as an abnormally strong reaction to sound occurring within the auditory pathways. At the behavioral level, it is manifested by a patient experiencing physical discomfort as a result of exposure to sound (quiet, medium or loud). The same sound would not evoke a similar reaction in an average listener. The strength of the reaction is controlled by the

physical characteristics of the sound, e.g., its spectrum and intensity.

Misophonia and *phonophobia* can be defined as abnormally strong reactions of the autonomic and limbic systems resulting from enhanced connections between the auditory and limbic systems. Importantly, misophonia and phonophobia do not involve a significant activation of the auditory system. At the behavioral level, patients have negative attitude to sound (misophonia), or are afraid of sound (phonophobia). In cases of misophonia and phonophobia, the strength of the patient's reaction is only partially determined by the physical characteristics of the upsetting sound and is dependent as well on the patient's previous evaluation and recollection of the sound (e.g., sound as a potential threat, and/or the belief that the sound can be harmful), the patient's psychological profile and the context in which the sound is presented.

Importantly, note that neither hyperacusis, misophonia or phonophobia have any relation to hearing thresholds. Patients with hyperacusis, misophonia or phonophobia may have normal hearing, and they may also have impaired hearing.

There is limited data available regarding the prevalence of decreased sound tolerance. Nonetheless, our research indicates that hyperacusis and tinnitus frequently co-exist in the same ear. Approximately 40% of tinnitus patients exhibit some degree of decreased sound tolerance, with 27% requiring specific treatment for hyperacusis. Conversely a study of 100 patients with hypersensitivity to sound showed that 86% of them suffered form tinnitus.

Therefore, based on clinical observation that approximately 27% of tinnitus patients required treatment for hyperacusis, considering that 86% of hyperacusis patients reported tinnitus [1], and accepting that about 4-5% of general population have clinically significant tinnitus, it is possible to extrapolate that significant hyperacusis probably exists in at least 1 - 1.5% of the general population.

In the majority of cases the etiology of hyperacusis is unknown. Hyperacusis has been linked to sound exposure (particularly short, impulse noise), head injury, stress, and medications. The lack of strong epidemiological data, and the lack of an animal model for hyperacusis prevents us from proving the validity of any theory of potential mechanism responsible for hyperacusis.

At the peripheral level it is possible to speculate that the abnormal enhancement of vibratory signals within the cochlea by the outer hair cells (OHC) might result in overstimulation of inner hair cells (IHC) and subsequently result in hyperacusis. Indeed, in some cases it is possible to observe high amplitude Distortion Product Otoacoustic Emissions (DPOAE) and DPOAE evoked by low level acoustic stimulation. Interestingly, the presence of asymmetric hyperacusis might support a peripheral based etiology, as central mechanisms would likely impact both sides.

Laboratory research has shown that damage to the cochlea, or a decrease of the auditory input, results in decrease of the threshold of response in significant proportion of neurons in the ventral cochlear nucleus and inferior colliculus. Studies with auditory evoked potentials indicated abnormal increase of the gain in the auditory pathways after such manipulations. Some medical conditions can be linked to the central processing of signals, and modification of the level of neuromodulators as a factor inducing or enhancing hyperacusis. Moreover, serotonin was indicated as involved in hyperacusis, and a recent case report indicated that serotonin reuptake inhibitors might be helpful for hyperacusis.

Mechanisms of misophonia could involve enhancement of the functional links between the auditory and limbic systems, both at the cognitive and subconscious levels. Alternatively, tonic high level of activation of the limbic and autonomic nervous systems may result in strong behavioral reactions to moderate sounds.

Decreased sound tolerance can exist as an independent medical diagnosis, or may be associated with more complex problems. Medical conditions previously linked to decreased sound tolerance include: tinnitus, Bell's palsy, Lyme Disease, Williams Syndrome, Ramsay Hunt Syndrome, stapedectomy, perilymphatic fistula, head injury, migraine, depression, withdrawal from benzodiazepines, increased Cerebral Spinal Fluid (CSF) pressure and Addison's disease [3,4].

Most frequently, significantly decreased sound tolerance results from a combination of hyperacusis and misophonia/phonophobia. It is important to assess the presence and the extent of all these phenomena in each patient, as these phenomena need to be treated using different methods.

While there is no clearly accepted 'consensus' method for the evaluation of decreased sound tolerance, there appears to be general agreement that loudness discomfort levels (LDLs) provide a reasonable estimation of the problem.

There are several variations of clinical protocols for establishing LDLs with various stimuli, e.g., continuous or pulsed, beeps of sound, pure tones or narrow band noise, etc. [2,5]. The approach we advocate incorporates modifications of standard procedures aimed at decreasing the effects of misophonic and phonophobic components to a minimum. To minimize the impact of these components, a situation is created (during testing) in which the patient has full control over the maximal sound level to which they will be exposed.

A detailed pre-test interview is needed with each patient to determine the relative contribution of hyperacusis, misophonia and phonophobia to decreased sound tolerance, reflected in decreased behavioral LDLs. As normative data are not uniform, and as there is substantial individual variability (even when using identical methodologies to obtain and measure LDLs), it is advisable to pay particular attention to the potential presence of hyperacusis when average LDLs values are lower than 95 - 100 dB HL [6,7].

TREATMENT OPTIONS:

Regarding hyperacusis treatment, there are two, diametrically opposed treatment options.

First, the most common approach is to advise patients to avoid sound and use hearing protection. This is based on reasoning that because patients became sensitive to sound this may indicate that they are more susceptible to sound exposure and consequently need extra protection. Patients easily embrace this philosophy and start to protect their ears, even to the extent of using ear plugs in quiet environments. Unfortunately, this approach makes the auditory system even more sensitive and further exacerbates hyperacusis.

The second approach involves the desensitization of patients by exposure to a variety of sounds. The desensitization approach has been promoted for some time with a variety of protocols and types of sounds utilized; such as the recommendation of using sound with certain frequencies removed, short exposures to moderately loud sound, or prolonged exposures to low level sounds. According to principles of the neurophysiological model of tinnitus, the latter approach is recommended and it is used as a part of Tinnitus Retraining Therapy [7].

Misophonic/phonophobic components cannot be removed by desensitization and a separate approach needs to be implemented [7]. Our approach involves systematic exposure to sounds, associated with a pleasant situation, with gradually increasing sound levels.

Decreased sound tolerance, including hyperacusis, misophonia, and phonophobia, is a challenging topic to study and a challenging symptom to treat.

Many questions are unanswered; etiology is not clear, neural mechanisms are speculative and treatments are not yet proven. Above all, the general recognition of decreased sound tolerance, as a problem requiring attention and proper treatment, should be considered a priority in the community of hearing professionals.

In our next report, we will detail individual patient's case histories, their treatment plans, and their individual outcomes.

REFERENCES:

1. Anari M, Axelsson A, Elies W, Magnusson L. Hypersensitivity to sound -- questionnaire data, audiometry and classification. Scand Audiol 1999; 28(4):219-230.

2. Cox RM, Alexander GC, Taylor IM, Gray GA. The Countour test of loudness perception. Ear & Hearing 1997; 18(5):388-400.

3. Klein AJ, Armstrong BL, Greer MK, Brown FR. Hyperacusis and otitis media in individuals with Williams syndrome. J Speech Hear Disord 1990; 55:339-344.

4. Nields JA, Fallon BA, Jastreboff PJ. Carbamazepine in the treatment of Lyme disease-induced hyperacusi. J Neuropsychiatry Clin Neurosci 1999; 11(1):97-99.

5. Ricketts TA, Bentler RA. The effect of test signal type and bandwidth on the categorical scaling of loudness. J Acoust Soc Am 1996; 99:2281-2287.

6. Stephens SD, Anderson C. Experimental studies on the uncomfortable loudness level. J Speech and Hearing Res 1971; 14:262-270.

7. Jastreboff, P.J., Jastreboff, M.M. Tinnitus Retraining Therapy (TRT) as a method for treatment of tinnitus and hyperacusis patients. J. Am. Acad. Audiol., 11(3):156-161, 2000.