Treatments for Decreased Sound Tolerance (Hyperacusis and Misophonia)

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ABSTRACT

Decreased sound tolerance (DST) is an underappreciated condition that affects the lives of a significant portion of the general population. There is lack of agreement regarding definitions, specific components, prevalence, methods of evaluation, and methods of treatment. Limited data are available on the results of treatments. Research is scant and constrained by the lack of an animal model. This article proposes a definition of DST and its division into hyperacusis and misophonia. The potential mechanisms of these phenomena are outlined, and the results of treatment performed at Emory University are presented. Out of 201 patients with DST, 165 (82%) showed significant improvement. Of 56 patients with hyperacusis (with or without misophonia), 45 (80%) showed significant improvement. This proportion was higher for the group with hyperacusis and concurrent misophonia (33 of 39, or 85%) and lower for patients with hyperacusis only (13 of 17, or 76%). Effectiveness of treatment for misophonia with or without hyperacusis was identical (152 of 184, 83% and 139 of 167, 83%, respectively, for misophonia accompanied by hyperacusis and for misophonia only). Even with current limited knowledge of DST, it is possible to propose specific mechanisms of hyperacusis and misophonia and, based on these mechanisms, to offer treatments in accordance with the neurophysiological model of tinnitus. These treatments are part of Tinnitus Retraining Therapy (TRT), which is aimed at concurrently treating tinnitus and DST and alleviating the effects of hearing loss. High effectiveness of the proposed treatments support the postulated mechanisms.

KEYWORDS: Decreased sound tolerance, hyperacusis, misophonia, definitions, treatment

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Learning Outcomes: As a result of this activity, the participant will be able to (1) classify subsets of decreased sound tolerance; (2) list potential mechanisms for hyperacusis and misophonia.

Most people have preferences regarding specific sounds or the level of sound, but these preferences do not affect their everyday lives and interaction with others. Nevertheless, a significant number of people have negative reactions such as irritation, tension, anxiety, or fear of ordinary sounds that, in turn, negatively affect their lives. Individuals with decreased sound tolerance (DST) are unable to tolerate everyday sounds that do not bother other people. Interestingly, the sounds that evoke negative responses in these individuals (see Table 1) are not necessarily loud (e.g., power tools or low-flying planes) or moderate (e.g., electric shaver or average office sounds). Indeed, some individuals may be affected by soft sounds, such as breathing or chewing. For many years, DST was underestimated and therefore not fully investigated. Perhaps this was because affected individuals sought help from clinicians with a variety of specialties including otology, neurology, psychiatry, psychology, audiology, and occupational therapy. As a result, DST had no research home or treatment.

DST can be present with different levels of severity and may not necessarily require intervention and treatment. This text focuses on clinically significant DST (i.e., DST that bothers the individual to the extent that he or she wants to alleviate it).

Individuals affected by DST can be divided into two distinct groups: (1) Patients who respond consistently to sounds above a certain intensity; their reactions can be correlated with the physical parameters of the sound. These patients are diagnosed with *hyperacusis*. (2) Patients who react to specific patterns of sound and/or react to sound that occurs in specific situations or settings whereas tolerating other sounds that are frequently much louder. These patients are diagnosed with *misophonia*. Although the same sounds may be reported to induce identical reactions in both groups of patients, the treatment of hyperacusic and

misophonic patients is quite distinct and therefore they require careful diagnosis.

DEFINITIONS

Over the years, various terms have been used to describe oversensitivity to ordinary sounds that induce negative reactions. These terms include *hyperacusis*, *recruitment*, *hyperacousia*, *auditory hyperesthesia*, *dysacousis*, *auditory dysesthesia*, *odynacusis*, *auditory allodynia*, *phonophobia*, *increased noise sensitivity*, *collapsed tolerance level*, and *decreased sound tolerance*, with the term *hyperacusis* used most frequently.[1–3] These terms have not been precisely defined. Based on the literature and our accumulated knowledge from clinical practice, we proposed to define DST as present when a subject exhibits negative reactions following exposure to sound that would not evoke the same response in an average listener.[2,4,5] We also postulated that DST consists of two components: hyperacusis and misophonia, which frequently coexist.

Hyperacusis, the first component of DST, is present when negative reactions to a sound depend only on its *physical* characteristics (i.e., its spectrum and intensity). The sound's meaning and the context in which it occurs are irrelevant. In turn, this implies that the mechanisms responsible for the emergence of hyperacusis are within the subconscious part of the auditory pathways.[2,4,5] For example, a patient will react identically to the sound of a knife hitting china in *any* situation or setting. This individual also will react negatively to all other high-intensity sounds.

The second component of DST, misophonia, is present when an abnormally strong reaction occurs to a sound with a specific pattern and/or meaning to an individual.[4,5] The reaction may depend on the environment where the offensive sound is presented. The physical characteristics of the sound are secondary. Indeed, the strength of the misophonic patient's reaction is only partially determined by the

Table 1 Sounds that Evoke Negative Reactions as Reported by Patients[*]

Loud rings (phone, doorbell)

School bell

Announcements in a metro, train station, airport, or supermarket

Announcement on airplane or train

Train on a track, particularly under bridge

Low-flying airplanes

Street sounds

Sound of car brakes or bicycle brakes

Sounds of driving on street or highway

Dogs barking

Warning sounds (e.g., sirens, car horns, beepers)

Slamming doors

Sudden sounds (e.g., object dropped on a hard surface)

Sounds of surgical instruments in an operating room

Sounds of a dental procedure

Drilling

Power tools

Mechanical/motor sounds

Leaf blower, lawnmowers

Swimming pool pump

Shoveling cement

Movie theaters, concerts

Social gatherings, parties

Restaurants, pubs, bars, nightclubs

School breaks, cafeterias

Sporting events

Church services

TV, radio with volume set by family member with normal hearing

Other people singing

A musical instrument

Vacuum cleaner

Boiling water

Sound of a refrigerator

Utensils hitting china

Cutlery and plates

Loading dishes in dishwasher

Garbage disposal

Popping popcorn

Supermarket freezer

Grocery stores

Shopping malls

Crinkly bags

Crumpling or wrinkling paper

Hum of electricity

Hum of a computer

Office sounds (typing on a keyboard, printers, copy machine, fax)

Other people's headphones

One's own voice

Specific type of laughter

Children's voices

Screaming, crying babies

High-pitched voices

Sniffing

Other person breathing in bed, snoring

Other people breathing

Lip smacking

Sounds of eating

Swallowing

Chewing

Crunching sound

Clipping and filing fingernails

Toothbrush

Electric shaver

Hair dryer

Flushing toilet

Keys rattling

Moving hand on a surface

Chalk on a blackboard

Sound of drawing with a felt-tipped pen

Cat walking on a hardwood floor

[*]From various texts.[3,5,21] Note that sounds such as bird song, running water, wind, and rain are rarely reported as negative.[21]

sound's physical characteristics. Frequently, a person with misophonia will respond strongly to a soft sound of a specific pattern (e.g., a voice, the sounds of eating), but not react to other, much louder sounds (e.g., loud music). Furthermore, the individual may react to a given sound in one setting (such as in his or her home) but not react to the *same* sound in another setting (such as in the home of a friend). The patient's negative reaction to the sound depends on nonauditory factors such as his or her previous evaluation of the sound, on the belief that the sound is a potential threat, or that exposure to it will be harmful. The sound may be associated with a previous negative experience.[2–5] The patient's psychological profile and the context in which the sound occurs are important as well.[2,4,5]

A specific category of misophonia called *phonophobia* occurs when the patient's fear of

sound is the dominant emotion.[2,4,5] Phonophobia is recognized in clinical psychology, where it is treated the same way as other types of sensory phobias, and in neurology, where it is one of the symptoms associated with migraines and treated as part of a chronic neurological disorder characterized by recurrent headaches.

The concept of misophonia was created in 2001, after 10 years of clinical work with patients with tinnitus and what we believed at the time was hyperacusis, when we recognized the presence of the two subgroups of patients with DST. Patients who reacted negatively to specific patterns of sound did not improve as well as hyperacusic patients to classical sound desensitization treatment. In introducing a new categorization of patients with DST, including a new concept and a specific term to describe patients who reacted in a negative manner to specific patterns of sound, we combined the word *miso* (meaning *hate* in Greek) to represent patients' strong negative attitudes with the term *phonia* (meaning *voice, sound*) to form the term *misophonia*.[4,6] Obviously, it is incorrect to translate misophonia literally as hatred or dislike of sound, just as it would be incorrect to translate photography to mean light drawing. Unfortunately, such an incorrect interpretation has been made in the literature. There are patients who exhibit a general dislike of sound, typically combined with phonophobia, but they are rare.

Although the term *misophonia* was introduced in the literature in 2002[4] and described in the following years,[2,3,7,8] in January 2013, Schröder et al redefined misophonia based on their work in a psychiatric center and proposed to classify the condition as a new psychiatric disorder.[9] In our opinion, Schröder studied a population of *psychiatric* patients who happened to have misophonia as well. In our clinical work, we have seen several hundred misophonic patients (compared with 42 cases reported by Schröder), all evaluated by physicians, and in only a few cases did patients exhibit psychiatric problems. Moreover, our misophonic patients showed significant improvement when treated with a combination of counseling and a specific version of sound therapy (described later) without any need for psychiatric intervention.

PREVALENCE OF DST

Information on the prevalence of DST is vague. There is a lack of agreement on terminology and definitions, and even on how such an evaluation should be performed. No validated questionnaire exists to detect the presence of DST. Some reports indicate high prevalence; for example, data obtained from 10,349 randomly selected subjects showed that 15.3% reported DST.[10] Other authors reported lower prevalence.[11–13]

There is general agreement that with regard to hyperacusis, pure tone loudness discomfort levels (LDLs) must have decreased values. That being said, there is no consensus on how LDLs should be evaluated and, indeed, a variety of methods have been proposed.[14–18] Furthermore, LDLs are not typically part of a routine audiological evaluation. Importantly, low LDLs by themselves do not prove the presence of hyperacusis as low values may be due to misophonia. A careful inspection of publications reveals that, in some cases, misophonia rather than hyperacusis was present, such as in reports on Williams syndrome.[19,20]

On the basis of the tinnitus literature, for which more detailed data are available, it is possible to estimate the prevalence of DST in the general population. Results from the Emory Tinnitus and Hyperacusis Center indicate ~60% of patients with tinnitus examined have significant DST.[4] Results from other clinicians and researchers show that ~25 to 30% of patients with tinnitus have hyperacusis that requires treatment.[3,4,21–24] In addition, it has been reported that 86% of patients with hyperacusis suffer from tinnitus.[25] By accepting an average prevalence of clinically significant tinnitus reported in the literature as 4%,[26] it is possible to calculate that clinically significant hyperacusis is present in ~1.75% of the general population. As our data indicate, hyperacusis is present in about half of patients with DST, and the prevalence of DST in the general population can be estimated to be 3.5%. Because our data indicate 92% of patients with DST have misophonia, the prevalence of misophonia in the general population can be estimated at 3.2%.

PRESUMED MECHANISMS

The mechanisms associated with the emergence of DST, and how negative reactions to sound

develop, are hypothetical and none of the proposed mechanisms have been proven. The lack of an established animal model of hyperacusis makes it difficult to assess the validity of the proposed mechanisms. Both peripheral and central mechanisms have been postulated.[3,27,28] The impact of stress on the peripheral and central auditory system involving dynorphins has been proposed,[29] as has the involvement of serotonin.[30,31] Dynorphins are a class of opioid peptides that have been shown to be involved in modulating pain response, addiction to cocaine, stress and depression, appetite and circadian rhythms, and temperature regulation. Minimal experimental and clinical support exists to support either proposition.

Another proposed mechanism involves dysfunction of the efferent olivocochlear systems,[32,33] which have an inhibitory action on the outer hair cell (OHC) and inner hair cell (IHC) systems. The available data argue against this hypothesis in the following ways. Recordings of otoacoustic emissions in hyperacusis patients have not revealed enhanced emissions, thereby arguing against overamplification provided by the cochlear amplifier. Importantly, the possibility of involvement of the efferent olivocochlear bundles can be excluded due to the observation that severance of the vestibular nerve in humans, within which all efferent olivocochlear fibers are contained, has no impact on DST[34] nor on auditory performance.[35]

In analyzing the potential mechanisms of hyperacusis and misophonia, it is essential to consider the following. First, the negative reactions induced by hyperacusis and misophonia are identical and it is impossible to distinguish between the two conditions on that basis. We can imply, however, that common brain systems are responsible for the negative reactions evoked by hyperacusis and misophonia and that these reactions (see Table 2) argue for the dominant role played by the limbic and autonomic nervous systems. Other systems in the brain are involved, but are secondary.

Second, the differentiating factor between the two conditions is that for hyperacusis the physical characterization of the offending sound is sufficient to assess the other sounds that would create a problem, whereas for misophonia the specific pattern of sound, its

Table 2 Negative Reactions Frequently Reported by Patients with DST

Irritation

Annoyance

Anger

Tension

Frustration

Urge to escape (run)

Urge to cry

Feeling of physical pain

Feeling of being restrained in doing things

Feeling uncomfortable (discomfort)

Inability to concentrate

Inability to enjoy things, particularly involving louder or specific sounds

Increased awareness of sounds (forced to monitor sounds)

Fear of sounds

Emotional distress

Uneasiness

Worry

Stress

DST, decreased sound tolerance.

meaning, and the situation in which it occurs play a predominant role. Therefore, the reactions depend on the misophonic's past history and on the association of the sound with something negative. Taken together, these facts suggest that although the negative reactions to sound are identical, the mechanisms yielding these reactions are different for hyperacusis and misophonia.

In cases of clinically significant DST, a sound evokes the overactivation of the limbic and autonomic nervous systems. Taking into account the factors listed previously, it is possible to consider two different scenarios leading to such a state. In one scenario, when the sound reaches the cochlea it is overamplified exclusively within the auditory system, (1) during the transduction process performed by the IHCs and OHCs or (2) at a higher level of the subconscious part of the auditory pathways.[2,3,36] In the second scenario, the auditory system works normally, but functional connections between the auditory system and the limbic and autonomic nervous systems are enhanced.

At the peripheral level of the auditory pathways, it is possible to envision enhanced activity of the OHCs that would result in the overstimulation of IHCs or the enhanced release of neurotransmitters from the IHCs.[2,3,36] Available data, however, argue against a dominant peripheral mechanism for DST. For example, clinical observations show the nearly universal dominance of bilateral, symmetrical hyperacusis,[24] which suggests the central mechanism as dominant. In other words, as in the case of cochlear dysfunction, there should be cases of clear unilateral hyperacusis. Furthermore, if overamplification of the OHCs was present, then a high level of distortion product otoacoustic emissions should be observed, which is not the case.

Research and available clinical data mentioned later support the involvement of the central part of the auditory pathways in DST. As is the case for all other sensory systems, the auditory system works according to the dynamic balance principle, where the gain on the system is modified depending on the input level. Thus, when the sound level is low, the sensitivity and gain of the auditory system increases. Animal research has shown that damage to the cochlea or a decrease in auditory input yields a corresponding decrease of the response threshold in a significant proportion of neurons in the ventral cochlear nucleus and inferior colliculus.[37] These findings have been corroborated by a study of evoked potentials, which indicated an abnormal increase of gain in the auditory pathways after such manipulations.[38] The hypothesis of increased gain within the central part of the auditory pathways has been discussed and promoted in recent approaches to the mechanisms of tinnitus and hyperacusis.[39]

In the previously mentioned scenario, the auditory system alone would be involved with no corresponding evaluation of the sound's meaning and without regard to memory, previous exposure (which would involve other brain systems), or emotional status. The patient's reactions would be determined solely by the physical characterization of the sound (i.e., its spectrum and intensity). When this behavior is observed in patients with DST, we diagnose them as having hyperacusis.

In misophonia, patients react to specific patterns of sound (soft or loud sounds), but do not have problems with other, even louder sounds. This observation demonstrates that the mechanism for misophonia is not within the auditory pathways, but in functional connections of the auditory pathways with other systems in the brain. The neurophysiological model of tinnitus can be used to explain the mechanisms of misophonia as well as to delineate the differences between the mechanisms of hyperacusis and misophonia.

THE NEUROPHYSIOLOGICAL MODEL OF TINNITUS AS A BASIS FOR PROPOSED MECHANISMS OF HYPERACUSIS AND MISOPHONIA AND THEIR TREATMENT

The fundamental principle of the neurophysiological model of tinnitus is that in cases of clinically significant tinnitus or DST, several brain systems are dominant other than the auditory system. Furthermore, the activation of these other systems is responsible for the negative emotional and autonomic reactions evoked by these conditions. Analysis of these reactions points to the essential involvement of the limbic and autonomic nervous systems.

In cases of hyperacusis, it is proposed that the problem results from abnormally high amplification occurring within the auditory pathways, which, in turn, results in the stimulation of the limbic and autonomic nervous systems (Fig. 1A). Consequently, the neuronal activity evoked by exposure to (for example) a 70-dB hearing level (HL) sound in a patient with hyperacusis is similar to activity evoked in an individual without hyperacusis, following exposure to a 120-dB HL sound. In the same way that such a level of activity would evoke an aversive negative reaction in a normal subject, negative reactions would be evoked in a hyperacusis patient who is stimulated by a 70-dB HL sound. Note, for hyperacusis, the functional properties of the connections between the auditory system and the limbic and autonomic nervous systems are normal and the strong reaction of these systems reflects a high level of input to the limbic and autonomic nervous systems coming from the auditory system.
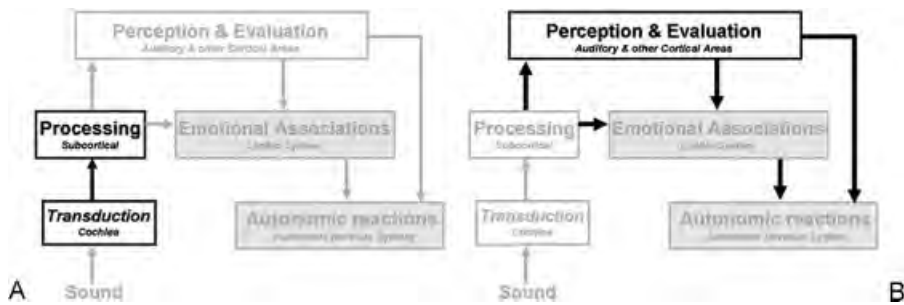
Figure 1 Block diagram of potential mechanisms responsible for decreased sound tolerance (DST). (A) Block diagram of potential mechanisms responsible for hyperacusis. Hyperacusis results from abnormal amplification of sound-evoked activity occurring within the auditory pathways (marked by black lines). (B) Block diagram of potential mechanisms responsible for misophonia. Misophonia results from enhanced functional connections between the auditory and the limbic and autonomic nervous systems for a specific pattern of sound-evoked activity (marked by black lines). Note overamplification of the limbic and autonomic system occurring in both conditions and marked by cross pattern. (Modified from Jastreboff and Jastreboff[3]).

The situation is dramatically different in cases of misophonia (Fig. 1B). In these cases, the auditory system works normally. However, the functional connections between the auditory and the limbic and autonomic nervous systems are enhanced for specific *patterns* of sound.[2–5] These connections involve the conscious, cognitive part and the subconscious path, with the subconscious path governed by the principle of conditioned reflexes. Our results indicate that the subconscious connections play a dominant role in overactivating the limbic and autonomic nervous systems.[40] As a result, even if a subject fully understands a given sound is not dangerous or threatening, strong negative reactions are still evoked.

The proposed mechanisms of misophonia were recently supported by the results of physiological investigations by Edelstein et al.[41] The authors found experimental evidence that misophonia produces distinct autonomic effects, and suggested that the mechanisms of misophonia concern aberrant functional connections between the auditory and limbic systems. Furthermore, the overactivation of the autonomic nervous system may, in turn, activate the tensor tympani muscle,[5,7] resulting in tensor tympani syndrome (e.g., fullness, pulsation, pain in the ear).[42] The presence of tensor tympani syndrome can be documented by measuring immittance over a 30- to 60-second period. Tensor tympani syndrome is frequently observed in patients with severe misophonia and

treatment of misophonia results in its elimination.[4,5]

The concept of a complex conditioned stimulus[43] plays an important role in misophonia. Behavioral neuroscience has documented that reaction to a stimulus occurs as a whole, and not to its individual components. A classic example has been described in the case of a tinnitus patient.[36] The patient perceived tinnitus as a hissing sound that was highly intrusive during the day and markedly affected his concentration and life. Paradoxically, although the patient had difficulty sleeping at night prior to developing tinnitus, his sleep improved after he had tinnitus. A detailed interview revealed he had a happy, carefree childhood and that the sound of his tinnitus was similar to the loud hissing sound produced by a water cistern in his attic bedroom that he heard as he drifted off to sleep. At bedtime, the similarity of the patient's tinnitus to the water cistern, in combination with the darkness and other factors he associated with going to sleep, created a complex stimulus that the patient perceived as positive and relaxing. On the other hand, the patient's tinnitus was highly intrusive during the day. Thus, the patient's tinnitus evoked opposite reactions depending on the copresence of other stimuli, which created the complex stimulus.

Similarly, for misophonia the environment may play a significant role and the patient may react to the same sound differently depending on the context and setting in which the sound is

heard. The purposeful creation of a complex conditioned stimulus, with the offensive sound as its component, is used in one of the protocols to treat misophonia, as discussed later.

For hyperacusis and misophonia, the extent of a patient's reaction to sound will depend on his or her emotional status and the coexistence of factors that induce a negative emotional state (e.g., pain, a vestibular problem, etc.). These factors directly affect the limbic and autonomic nervous systems, the centers responsible for inducing a negative reaction, and increase their sensitivity and reactivity (Fig. 1A and 1B).

EVALUATION OF HYPERACUSIS AND MISOPHONIA

There is lack of agreement as well as significant confusion regarding how to evaluate DST (see the previous discussion of LDL measurement), assess its severity, and discriminate between the presence and relative contribution of hyperacusis and misophonia. Only two questionnaires have been published that assess the extent of hyperacusis: the Hyperacusis Questionnaire[44] and the Multiple-Activity Scale for Hyperacusis (MASH).[45]

There are some concerns regarding the specificity and selectivity of these questionnaires. The Hyperacusis Questionnaire was created by testing the general population of subjects who did *not* complain of hyperacusis. The questionnaire primarily focuses on the psychological and social aspects of hearing rather than on hyperacusis per se. Indeed, only 4 of 14 questions are related to hyperacusis whereas the remaining questions are related to other aspects of hearing, including hearing loss (for example, "5. Do you have difficulty listening to conversations in noisy places?").[44] The MASH questionnaire consists of a list of 14 activities, and respondents are asked to indicate their level of annoyance related to a given activity on a scale from 0 to 10.[45] Although both questionnaires have been reported to be effective in evaluating hyperacusis, interestingly, there was no correlation of their scores with audiological measurements of discomfort levels such as the LDL test and a speech discomfort level assessment.[45] Therefore, these question-

naires need to be further evaluated. Notably, neither questionnaire differentiates between hyperacusis and misophonia. There is no published, validated questionnaire for misophonia.

The approach we are using to evaluate DST and its components, as well as to the administration and evaluation of LDLs, is based on published forms for structured initial and follow-up interviews.[46] Detailed instructions on how to apply these interviews have been published.[47] The expanded interview is crucial to assess DST. During this interview, stress is placed on identifying sounds that evoke negative reactions in the patient as well as sounds that are well tolerated. It is important to check for the presence of any discrepancies between the patient's reactions and the intensity of the sound. The existence of these discrepancies indicates the presence of misophonia. There is a need to create a structured interview and a questionnaire oriented to detect the presence and assess the severity of misophonia.

LDLs are useful for evaluating the potential presence of hyperacusis. Normal LDL values (~100-dB HL[18]) exclude the presence of hyperacusis. As low LDL values could be due to misophonia, the presence of low values alone does not provide sufficient proof that the patient has hyperacusis. Notably, hyperacusis and misophonia frequently occur together. Indeed, misophonia is inevitable in cases of severe hyperacusis, as the negative reaction evoked by hyperacusis will provide negative reinforcement and create a conditioned reflex linking specific sounds with something negative. Misophonia, however, does not induce hyperacusis.

LDL values reflect the contribution of hyperacusis and misophonia. The goal is to obtain LDLs that reflect hyperacusis and minimize the contribution of misophonia. Typically, the misophonic component is enhanced by patients' concern that they will be exposed to a loud sound that would make their tinnitus and/or DST worse; unfortunately, this may reflect the past experience of some patients. To decrease the impact of misophonia on LDLs, we promote a modification of the standard procedure for administering LDLs.[48] Patients are given the full power to stop the LDL test at any time. In addition, patients are

presented with short beeps of sound separated by a second or more of silence, so they have time to think and decide if they will allow for a further increase of the sound level.[3,49] LDL measurements are administered twice and the second set of results is used to assess the potential presence of hyperacusis. Note that misophonic patients tend to have higher LDL values during the second test. For example, the first set of values may be 85-, 90-, 85-, and 80-dB HL for 1, 3, 4, and 6 kHz, respectively, and the second set may be 90-, 95-, 90-, and 90-dB HL, respectively.

In cases of pure hyperacusis, LDLs are in the 60- to 85-dB HL range.[4] In cases of pure misophonia, LDL values as low as 30-dB HL to as high as 120-dB HL can be observed. Therefore, LDLs alone are insufficient to diagnose hyperacusis or misophonia and a specific, detailed interview is crucial to diagnose and assess the relative contribution of hyperacusis and misophonia to DST.

TREATMENT OF HYPERACUSIS AND MISOPHONIA

There are very limited data in the literature on the results of hyperacusis treatment. According to anecdotal reports, treatment has taken two opposite directions. The most common approach is to advise patients to avoid sound and use ear protection. This approach is based on the reasoning that because patients became sensitive to sound, they are supposedly more susceptible to sound exposure and consequently require extra protection. Patients readily embrace this advice and begin to protect their ears, even to the extent of using earplugs in quiet environments. Unfortunately, this well-intended approach makes the auditory system even more sensitive to sound and further exacerbates hyperacusis.[13,50–53]

The opposite approach to treating hyperacusis involves desensitization wherein patients are exposed to a variety of sounds. The desensitization approach has been promoted for some time with several protocols and types of sounds used.[13] For example, sounds with certain frequencies removed, short exposure to moderately loud sound, or prolonged exposure to relatively low-level sounds have been used.[13,54] Pink noise therapy as proposed by Vernon and Press has gained some recognition.[13] For this protocol, patients are advised to stop overprotecting their ears and to listen to pink noise through headphones set at the highest comfortable level for 2 hours a day. A group of patients were provided with pink noise cassette tapes, and questionnaires were mailed to 30 participants. Of 20 patients who responded, 13 reported using these tapes in a systematic manner and 7 (54%) reported improvement in their hyperacusis.[13] A version of pink noise therapy also has been proposed recently for tinnitus and hyperacusis by Johnson.[55] The protocol combines the use of pink noise with a 16-week cognitive exercise program, divided into sections and devoted to different topics, such as recognition and relief. To date, no results have been published in the peer-reviewed literature regarding the effectiveness of this approach.

Medications have been recommended for DST aimed at decreasing anxiety, depression, and other negative reactions. Cognitive behavioral therapy (CBT) has been proposed as well.[56] There are no reports in the literature showing the positive effects of medication on DST (except for migraine) and there is limited information on the efficacy of CBT to treat DST.[57]

In rare cases of hyperacusis, exposure to normal, everyday sound can evoke vestibular disturbances (e.g., sound-induced loss of balance, a feeling of motion sickness, nausea, vertigo, or dizziness). This is called the *Tullio phenomenon* (sometimes *vestibular hyperacusis*), and it is one of the common symptoms of superior canal dehiscence syndrome and may be successfully treated by surgery.

As discussed previously, hyperacusis results from enhanced gain within the auditory system, particularly the central part of the auditory pathways. The sensory systems work according to the principle of automatic gain control; depending on the strength of the signal, the sensitivity and gain changes continuously. Consequently, the lack of sensory input results in an increase of gain within the auditory system, yielding a corresponding enhancement of neuronal activity representing a sound, whereas exposure to louder sound yields a decrease in gain. Therefore, according to the principles of

the neurophysiological model of tinnitus, as well as the proposed mechanisms of hyperacusis, the systematic enhancement of the patient's auditory background should result in decrease of the gain within the auditory pathways. Consequently, this approach is recommended and used as part of Tinnitus Retraining Therapy (TRT).

IMPLEMENTATION

When TRT is used to treat hyperacusis, specific counseling is provided with the focus on the proposed mechanisms of hyperacusis, auditory toughening (i.e., preexposure to a nondamaging level of noise provides protection against loud noise[58–60]), and sound therapy.[2,3] Sound enrichment can be achieved by various means, with tabletop sound machines and ear-level sound devices (i.e., sound generators or combination devices that feature amplification and a sound generator) being most common. The sound used in sound therapy for hyperacusis cannot evoke annoyance or other problems for any reason, as the negative activation of the limbic and autonomic nervous systems would then occur, which, in turn, enhances conditioned reflexes and, as a consequence, enhances negative reactions to sound.

The sound emitted by the wearable sound generators should be set at or above a level below which stochastic resonance (i.e., enhancement of the signal by adding low-level broadband noise) could be evoked (i.e., 6-dB sensational level (SL) or higher).[61] At the same time, the sound should not be too high as to make it difficult to understand speech (above 20-dB SL). Stochastic resonance might enhance a loudness match of coexisting tinnitus by ~10%,[61] and anecdotal reports indicate the possibility of inducing tinnitus in patients treated with sound generators set close to threshold of a patient hearing.

Our results show that, on average, hyperacusis patients use sound at ~9-dB SL (range from 0 to 20 dB SL) as evaluated by real ear measurement.[62] There is no need to initially set the sound to a lower level, nor are patients asked to change the sound level of the devices in a systematic manner during treatment.[62] In practical terms, it is recommended that patients use

sound that is not annoying to them, even when listening to the sound for many hours. At the same time, patients should be able to easily notice the sound when focusing their attention on it. Patients may temporarily increase the sound level when going to louder places.

Independent studies support the use of TRT for the treatment of hyperacusis and have shown that the desensitization approach used in TRT has a statistically and clinically significant impact on hyperacusis.[53,63] It is possible to observe significant improvement in hyperacusis and improvements in LDLs within a few months. In some cases, it is possible to achieve a cure for and elimination of hyperacusis.

As discussed previously, the mechanisms of misophonia and hyperacusis are very different. In the case of misophonia, because the auditory system works within the norm, the misophonic component of DST cannot be removed by classical desensitization therapy. A different approach must be used for misophonia that includes specific counseling and therapy involving several specific protocols of sound use.[3–5,64] As misophonia results from enhanced functional connections between the auditory and the limbic and autonomic nervous systems, which are governed by principles of conditioned reflexes, treatment for misophonia is aimed at weakening and then removing these connections using protocols appropriate for the extinction of conditioned reflexes. Specifically, the active extinction of conditioned reflexes is performed by linking currently offensive sounds with something positive. Note the similarity between the mechanisms of misophonia and tinnitus. The difference is in the initial signal (internally generated neuronal activity in the case of tinnitus and neuronal activity evoked by an external sound for misophonia), but the remaining proposed mechanisms are the same. Consequently, treatment for misophonia usually takes a similar amount of time as the treatment for tinnitus.

When hyperacusis and misophonia coexist, offensive sound evokes the overactivation of neuronal activity within the auditory pathways; in turn, this signal is further enhanced while conveyed to the limbic and autonomic nervous systems via connections that are tuned to the specific pattern of the sound. Clinical

observations indicate that even when coexisting hyperacusis is treated successfully, as shown by changes in LDLs to normal values, the behavioral reactions of patients remain the same in that patients do not perceive improvement. Presumably, misophonia becomes even stronger, which keeps the strength of the negative reactions the same.

For misophonic patients, the sound therapy component includes the same recommendations as for hyperacusis. However, specific additional protocols for sound use are recommended. There are four classes of sound protocols for misophonia and all of them contain a component whereby positive associations are formed with sound. The protocols differ in terms of the extent of control the patient has over the environment, the sounds used, and the length of exposure. Additionally, for protocol category 4, complex conditioned stimuli are created by combining offensive sounds with sounds that have highly positive connotations, and then gradually modifying the relative levels of those two types of sound. Notably, the protocol must be tailored to the individual. Frequently more than one protocol is used concurrently.

Protocol category 1 provides the patient with full control over the selection of sound used, its level, and its duration.[4] This protocol can be used even in cases of coexisting significant hyperacusis from the beginning of DST treatment.

When implementing this protocol, patients are asked to select a sound they like very much and listen attentively to it once or twice a day for 15 to 30 minutes each time. Patients typically select music, but any favorite sound can be used such as an audiobook on CD or a favorite TV show (particularly in the case of children). In the following example, music will be used. The specific music selected for a given day can vary depending on what the patient feels like listening to at a given moment. It is important that the patient listen attentively to the music and is not involved in any other activity that requires his or her attention (e.g., the patient should not read a book at the same time or drive a car). During the first week, every session begins with the patient selecting music and then adjusting the sound level to achieve maximum pleasure. During the second week, the patient selects music he or she wants to hear

and adjusts the sound level to a volume that is optimal for listening pleasure. Before beginning the listening session, the patient is then asked to increase the volume by a just noticeable difference. In practice, the increase in volume will be ~2 dB or a little more. During the third week, the patient is asked to increase the initial optimal level by 2 steps, which would yield a volume increase of ~4 to 5 dB.

This protocol has two goals: (1) to create a positive association with a favorite type of sound and (2) to gradually increase the level of sound considered to be optimal. Due to the principle of stimulus generalization, the positive associations that are formed will spread to include other, similar types of sound. This protocol is completely safe, as the patient fully controls the type, level, and duration of sound he or she is listening to. Protocol category 1 is not aimed at exposing the patient to a variety of sounds or to offensive sounds, but at creating a positive association with sound in general. In cases of coexisting hyperacusis, this is the only protocol used.

Protocol category 2 also provides the patient with full control over the type of sound used (e.g., music, movie, TV program), but only partial, indirect control over the sound level. In this case, control is given to someone close to the patient who is instructed to set the sound volume to a level he or she thinks the patient will accept. After a listening session, the patient should provide feedback as to whether the sound level was too high, too low, or just fine, so that the individual can adjust the level for the next session.

The following example shows a movie being used to implement this protocol. Patients are asked to select one of their favorite movies and watch it in its entirety on DVD (or download it from the Internet) one to two times a week. Patients should select a movie they feel like watching at a given time. The sound level is set by somebody living with the patient, but modified from session to session on the basis of feedback from the patient. Patients who use sound generators or combination devices are advised to increase the sound level of the generators before starting the movie.[3]

This protocol also creates a positive association with sound, but decreases the patient's direct control over the sound level. As the

average movie contains a variety of sounds, patients will attain a more positive association with several sounds because they are presented while the patient is enjoying the movie.

Protocol category 3 enables the patient to select the type of sound, but the sound level is outside his or her control. This protocol is aimed at exposing the patient to a variety of sounds encountered in everyday life and therefore going to a movie theater is recommended. For some patients, it is recommended that they go to shopping malls and gradually visit louder stores. This protocol is used only when significant hyperacusis is absent or has been eliminated by treatment. (Patients can always choose to leave a noisy environment when annoyed by sound or for any other reason.)

When implementing this protocol, patients are asked to go to a movie theater once or twice a week and select only their most favorite movies. Patients who use sound generators or combination devices are advised to increase the sound level of the generators before entering the movie theater. If the situation causes strong discomfort, the patient should leave the theater and return 10 to 15 minutes later.

This protocol works similarly to protocol category 2 but removes patients' control over the sound level to which they are exposed. It creates a positive association with a broad variety of sounds even when some sounds are presented at a typically high level.

Protocol category 4 is more complex and addresses specifically offensive sounds. It implements the concept of complex conditioned stimuli, which are created by combining offensive sounds with sounds that have highly positive connotations, and then gradually modifying the relative levels of the two types of sound. Once the new complex stimulus is established, the ratio of positive to negative sound is gradually decreased. The environment where this protocol is used must be taken into account, as many patients react differently depending on where they are exposed to a bothersome sound (i.e., home, school, a restaurant, a friend's home). Patients are advised to start with less offensive sounds and gradually work with more challenging sounds and situations. For example, if the sounds in a school cafeteria are less bothersome to a patient than the sounds of eating at home, the protocol should first be implemented at school. The multisensory aspect of stimuli also is taken into account as some patients react even to *seeing* someone produce offensive sounds (e.g., eating). Consequently, patients are advised to observe offensive situations while listening to highly positive sounds.

An example of implementation: Let's assume that a patient strongly reacts to the sounds of eating when at home. In this case, the patient is asked to select a highly favorable sound, such as music he or she likes a lot. In the first stage of this protocol, before the meal, the patient is asked to start playing the music at a level that still allows the offensive sounds to be heard. An open-field sound presentation is preferred, as headphones or earbuds will isolate patients from other people, but if the type of music or its level is unacceptable to others, then headphones or earbuds are recommended, provided the patient can still hear the offensive sounds. Typically, it is recommended that the positive sound should not mask the offensive sounds; however, full masking is permitted for a limited time for those patients who exhibit an extremely strong reaction to the offensive sounds.

After the patient follows this protocol for 1 week, and provided the newly created complex stimulus has a positive or neutral connotation for the patient and is well tolerated, the level of positive sound is gradually decreased over a period of weeks to months.

Protocol category 4 is applied to all offensive sounds; if it is difficult to create a situation with certain offensive sounds, the sounds can be recorded and played back to create the complex stimulus described previously. Furthermore, to facilitate treatment, stimuli from other sensory modalities in addition to sound (i.e., vision, taste, smell), which are associated with positive emotions and have some connection with the offensive sound, are introduced. For example, if the crunching sound of a cookie as it is being eaten is aversive to a patient but the patient likes to eat cookies, the smell of freshly baked cookies is presented at the same time the crunching sound is made.

Some "desensitization protocols" resemble a simplified version of protocol category 4. However, protocol category 4 appears to be

more powerful and consistent in offering help to patients even in challenging cases, such as for autistic children, perhaps because it is based on specific postulated neurophysiological mechanisms and on well-established methods from behavioral psychology.[43]

RESULTS OF TREATMENT OF HYPERACUSIS AND MISOPHONIA AT EMORY TINNITUS AND HYPERACUSIS CENTER

Detailed information was available for 201 consecutive patients diagnosed with DST. Of these 201 patients, 184 (92%) exhibited misophonia; 17 patients (8%) had hyperacusis alone, and 56 patients (28%) had hyperacusis and misophonia concurrently. The proportion of patients with significant hyperacusis (with or without misophonia) who required specific treatment is similar to the 25 to 30% reported in the literature[3,21–24] and to the 26% we have reported previously.[4]

All patients were treated with an appropriate version of TRT. The initial and follow-up structured interviews were used to monitor the treatment, with several questions aimed at assessing DST. Patients were asked to judge the severity of their DST, the annoyance induced by it, the effect of DST on their lives, and to rate DST as a problem on a scale from 0 to 10 (where 0 corresponded to an absence of DST and 10 indicated DST was as big of a problem as they could imagine). Patients were asked to consider an average over the last month in their responses.

Improvement in hyperacusis was judged on the basis of changes in LDLs combined with responses obtained during the structured interviews, whereas improvement in misophonia was solely based on the interviews. This decision was based on the observation that in cases of misophonia, any LDL values could be seen and that the values were not correlated with patients' judgment of the problems due to DST.

Of 201 patients with DST, 165 patients (82%) showed significant improvement. For 56 patients with hyperacusis (with or without misophonia), 45 patients (80%) showed significant improvement. This proportion was higher for the group with hyperacusis and concurrent misophonia (33 of 39, or 85%) and lower for patients with hyperacusis alone (13 of 17, or 76%). The effectiveness of treatment for misophonia with or without hyperacusis was identical (152 of 184 patients with misophonia accompanied by hyperacusis, or 83%, and 139 of 167 patients with misophonia alone, or 83%).

In some cases, it is possible to achieve a cure for misophonia, as is also the case for hyperacusis.[5,65] Furthermore, our clinical observations demonstrated that treatment of misophonia is crucial to achieving a successful outcome for tinnitus treatment.[66]

SUMMARY AND CONCLUSIONS

DST remains a complex and elusive phenomenon that has only recently attracted larger attention. There is limited information on epidemiology, potential mechanisms, and results of a variety of treatments that are used. The situation is particularly complex and difficult with misophonia (reaction to specific patterns of sound). Consequently, many patients with DST are left without help. The proposed mechanisms of hyperacusis and misophonia, which are based on the neurophysiological model of tinnitus, indicated a potential approach to their treatment. These treatments have been used for over a decade as part of TRT with a high level of success for both hyperacusis and misophonia. This observation supports the proposed mechanisms of DST. There is still need for improvement in the diagnosis and treatment of DST, as well as for research that could lead to a better understanding of the problem.

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