Chapter 57
Tinnitus and Hyperacusis/Phonophobia

Carlos Herráiz and Isabel Diges

Keypoints

1. Hyperacusis is a decreased sound tolerance.
2. Prevalence of the disease is described in 9–15% of the population, but increases among tinnitus patients.
3. Pathophysiological mechanisms involve some disruptions in the amplification and regulation processes of the external hair cells, disorders of the efferent system (medial and lateral olivocochlear pathways), or effects to the central sound processing at the subcortical level.
4. The role of some neurotransmitters (serotonin, GABA), which are also involved in other hyperacusis-related diseases (migraine, depression), can be relevant in this disorder.
5. Other theories confirm the effect of the endorphins that activates the excitatory function of the glutamate, the main auditory neurotransmitter, increasing its toxicity.
6. The activation of the limbic and autonomic nervous systems produces the emotional reaction of the hyperacusis (anxiety, fear, and depression).
7. Proposed treatments are based on acoustic stimulation: progressive introduction of white sound (tinnitus retraining therapy TRT) and customized sounds based on the damaged hearing frequencies.
8. Noise generators and hearing aids can be fitted in severe cases.
9. The role of some drugs involved in the metabolism of serotonin and GABA opens new approaches for the management of hyperacusis.

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Abbreviations

LDL	Loudness discomfort level
DST	Decreased sound tolerance
OHC	Outer hair cells
IHC	Inner hair cells
MOCB	Medial olivocochlear bundle
LOCB	Lateral olivocochlear bundle
5HT	Serotonin
ABR	Auditory brain responses
THS	Test of Hypersensitivity to Sound
BBNG	Broad band noise generators

Introduction

Hyperacusis is defined as a decreased tolerance to environmental sounds or the abnormal avoidance response to sounds that they are not annoying to the general population (see Chap. 3). It is a disorder of the normal amplification process of the auditory pathways. A decrease in the loudness discomfort levels (LDL) to environmental noise is observed in individuals with hyperacusis scores below 90 dBHL for some authors [1] or below 100 dBHL according to others [2]. Auditory hypersensitivity affects all sounds, although some specific noises can be more annoying according to their frequency spectrum or intensity.

The hyperacusis has to be distinguished from other symptoms that could co-exist simultaneously or develop as isolated forms. Misophonia (from the

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Greek "miso: hate") is a "dislike of certain specific sounds," and is different from phonophobia – a fear of certain sounds [2] (see Chap. 4). The anatomical and physiological basis is generally unknown, and these clinical entities have been regarded as belonging to the field of psychology. Phonophobia and misophonia are related to the type or the source of the sound and not specifically to its loudness. Hyperacusis is an abnormally low tolerance of sounds and may have to do with faulty gain control in the auditory pathways causing an abnormal activation of emotional reactions from the limbic and autonomous systems. Conversely, phonophobia is an abnormal reaction from the limbic and autonomous systems with normal auditory neural activity.

Recruitment of loudness is a pure cochlear physical phenomenon that depends on the outer hair cells. It is caused by the stimulation of the neighboring neural fibers to the damaged cochlear areas after exposure to intense sounds. There is a breakdown in the relation between the stimulus loudness and the intensity of the patient's acoustic sensation. The result is a distortion of, and an annoyance to, the sound.

There are a few epidemiological studies related to hyperacusis and decreased sound tolerance (DST). Fabijanska performed a wide study, sending a specific questionnaire to the general population by postal mail. Of 10,349 returned questionnaires, the study showed that 15.2% of the population referred hypersensitivity to sound [3].

The study published by Andersson in 2002 was conducted in Sweden through the Internet. Nine percent of the 595 responders reported a DST. These data were confirmed through postal mail to 589 individuals, where 8% of the sample showed the same results [4].

Some studies have described the prevalence of DST among tinnitus patients. Between 40% [2] and 59% [5] in a tinnitus clinic sample reported symptoms of hyperacusis. The prevalence of tinnitus in DST patients rises up to 86% [6].

Mechanisms of Hyperacusis

The mechanisms of hyperacusis generation and persistence could involve a peripheral origin, principally the cochlea, or could be a disorder of the central auditory pathways.

The amplification of the acoustic pressure wave from the active movements of the outer hair cells (OHC) facilitates the stimulation of the inner hair cells (IHC). This mechanism can be damaged due to an increased amplification of sound from the OHC [2]. Hyperexcitability of these cells would overstimulate the action of the IHC. The OHC's active movements would excessively amplify a sound of moderate intensity and, therefore, it will be annoying. Distortion product measurements in these patients would show increased values [2, 7].

Contralateral otoacoustic emission suppression through white noise stimulation is a useful tool to test the efferent system function. We found some abnormalities in the medial olivocochlear bundle (MOCB) pathways as the cause of DST [8]. Other authors, such as Baguley, have not found any change in LDL scores after section of olivocochlear fibers (efferent fibers) when performing a vestibular neurectomy for disabling vertigo (the MOCB travels with the vestibular nerve at the point where it is sectioned) [9].

The lateral olivocochlear bundle (LOCB) originates in the lateral superior olivary complex and innervates through unmyelinated axons, the primary afferent dendrites of the cochlear nerve near their synapses with inner hair cells. LOCB terminals are more complex, with evidence for cholinergic, GABAergic, dopaminergic, and peptidergic transmission [10]. Activation of the LOCB can evoke either slow enhancement (cholinergic) or suppression (dopaminergic) of auditory nerve response. LOCB feedback maintains the binaural balance in neural excitability required for accurate localization of sounds in space [11]. Its function has been associated with the control to glutamate excitotoxicity in afferent nerve terminals in acute acoustic injury [12], and it has a protective effect over neural damage from intense sound exposition, mainly based on the dopaminergic regulation.

DST could be caused by LOCB impairment. Clinical diagnosis of LOCB function is based on auditory brainstem responses (ABR) [13], which would increase by ipsilateral stimulation and decrease in response to contralateral stimulation. Otoacoustic emissions will not be affected because the LOCB does not affect the function of the outer hair cells. It can be hypothesized that the dopaminergic LOCB synapses would be a suitable target for treatments. It has been shown that after acute acoustic injury, perfusion with dopaminergic agonists reduces cochlear damage [12].

Table 57.1 Cause of hyperacusis from ear disorders range

Cochlear diseases	Ménière's disease/endolymphatic hydrops
	Perylimphatic fistula
	Sudden deafness
	Acoustic trauma/noise induced hearing loss
	Otoesclerosis
After surgical procedures	Post stapedectomy
	After transtympanic tube placement
	After wax removal
Stapedial reflex disorders	Sdr. Ramsay hunt
	Bell's facial palsy
Muscular disorders	Mystenia gravis

Table 57.2 Causes of hyperacusis related to central nervous system disorders

Migraine
Depression
Sd. Postraumatic stress
Craneoencephalic trauma
Lyme's disease (*Borrelia burgdorferi*)
Williams Sdr.
BZD dependence Sdr.
Chronic postviral fatigue Sdr.
Serotonine disfunction
Tay-Sachs Sdr. (*gangliosidosis 2*)
Multiple sclerosis
Benign intracranial hypertension Sdr.

BZD benzodiazepines

Other possible mechanisms of peripheral disorders that could cause DST would be the recruitment phenomenon; although recruitment of loudness is regarded to be different from hyperacusis, it is included in DST. Cochlear hearing loss (which occurs in Ménière's disease), sudden sensorineural hearing loss, or immunological inner ear disease shows a reduction in LDL and the presence of acoustic distortion. Other possible causes of hyperacusis would be damage of the acoustic middle-ear reflex mediated by the facial nerve. Bell's palsy, other facial palsies, neuro-muscle disorders such as myasthenia gravis, or stapes surgery may present DST in many patients. This kind of DST, however, usually abates spontaneously over time. Table 57.1 shows the most relevant etiologies.

Serotonin (5HT) has been involved in some diseases such as migraine, depression, or posttraumatic stress syndrome – disorders associated with DST that may modulate auditory signals [14]. (5HT) has an important role for central auditory processing (CAP) and can be decreased in older people. A study performed in elderly patients showed that treatment with a selective serotonin release inhibitor (citalopram) improved the results of auditory processing and speech discrimination tests [15].

A second mechanism described in hyperacusis is based on the role of the endogen endorphins [16]. Anxiety and stress increase the liberation of endorphins in the IHC–auditory nerve synapses. These substances potentiate the excitatory effect of the glutamate and therefore may increase excitation in the auditory periphery. Table 57.2 gives a list of central disorders associated with hyperacusis.

The inhibitory neurotransmitter GABA acts at several levels on the acoustic pathways. Even the function of the cochlea depends on GABA transmission at IHC synapses. A decrease in the action of GABA will increase neural activity and could be a correlate for hyperacusis. $GABA_A$ receptor agonists, such as benzodiazepines, could be used for some forms of hyperacusis. The author has used pregabalin for DST management with good results in some patients. Pregabalin affects many receptors and produces a dose-dependent increase in glutamic acid decarboxylase activity, increasing neuronal GABA levels.

Diagnosis of Hyperacusis

There is no objective measurement of hyperacusis because it is a subjective symptom (Table 57.3). A complete audiological examination, however, can be useful in the diagnosis of hyperacusis. Tonal and speech audiograms, tympanometry, and the study of the acoustic middle-ear reflex should be performed in all patients. ABR can rule out vestibular schwannoma and other retrocochlear diseases (multiple sclerosis) and is also useful for the diagnosis of auditory nerve neuropathy. An increase in the amplitude of the ipsilateral ABR responses and a decrease in the contralateral ABR in normal hearing subjects would rule out a LOCB disorder [13].

The study of OHC function and the MOCB efferent system can be performed through otoacoustic emissions (OEA). Study of the MOCB efferent system can be useful for diagnosis of some causes of DST. The discomfort threshold, which is the sound intensity that is annoying and not tolerable, can be determined. Its

Table 57.3 Classification of hyperacusis according to the loudness discomfort level and dynamic range

Degree	Dynamic range	Loudness discomfort level
No hyperac.	≥60 dB	≥95 dB in all the frequencies
Mild	50–55 dB in any frequency	80–90 dB in 2 or more frequencies
Moderate	40–45 dB in any frequency	65–75 dB in 2 or more frequencies
Severe	≤35 dB in any frequency	≤60 dB in 2 or more frequencies

Table 57.4 List of affected or avoided activities due to DST

Concerts	Social life	Sport spectacles
Going to the restaurants	Going to church	House keeping
Going to the cinema	Working	Taking care of the children
Shopping	Driving	Others

normal values are more than 90 dBHL, which are lower than the pain thresholds. It has to be tested several times because patients may have an initial fear of sounds, which would initially give lower thresholds than the real tolerance level.

Many individuals with DST will avoid different activities, affecting quality of life. The use of visual analogue scales for evaluation of hyperacusis handicap is useful (described in Table 57.4). Another system that has been proposed, named MASH, classifies the hyperacusis in four grades according to a broad list of activities: mild (≤3), moderate (from 3.1 to 5), severe (from 5.1 to 7), and very severe (≥7) [17].

In recent years, some specific questionnaires for DST have been developed and are useful tools in clinical diagnosis. The "self-rating Questionnaire on Hypersensitivity to Sound" published by Nelting and Rienhoff [18], evaluated DST according to three factors: cognitive reactions to hyperacusis, behavioral changes, and emotional responses to external sound. It was based on 15 questions; the scores went from 0 to 45. Every question had four possible answers: never (0 points), sometimes (1 point), often (2 points), and always (3 points). The score obtained can be divided into four grades, as we can see in Table 57.5. This questionnaire was originally written in German and it has been translated into Spanish [19]. Another published questionnaire was written by Khalfa [20]. It is based on 14 items and evaluates three dimensions: attention, social interaction, and emotion.

Table 57.5 Grades of hyperacusis considering the GUF

Degree	Score
Mild	≤10
Moderate	From 11 to 17
Severe	From 18 to 25
Very severe	26–45

Studies of Hyperacusis in a Tinnitus Clinic

In a study on 250 consecutive patients [5], we described the clinical characteristics of hyperacusis and tinnitus in DST patients. Direct questions and specific questionnaires were used to evaluate the interference of DST and tinnitus on quality of life. Auditory and psychoacoustic measurements were done on all participants. The answer to a question "do you feel more uncomfortable with environmental sounds than a majority of people?" was affirmative for 54% of the participants. Fifty-two participants had to stop one or more activities from a list of eleven (shopping, driving, taking care of children, going to church, etc.) because of DST. Sixty-three percent of the tinnitus clinic population showed LDL ≤ 90 dBHL, which was our definition of hyperacusis. Sixty-one percent were women, whose average age was 51 years (±14). Anxiety or stress was reported by 65% of the group, and 15% described the presence of different phobias: height, closed spaces, or insects. Sleeping problems were also very common (51%), and in two-thirds of the cases, tinnitus was the main problem for lack of sleep. A hearing impairment over 25 dBHL in any frequency was present in 83% of the participants.

The tinnitus of the DST group was predominantly in the left ear (52%), 27% in the right ear, and bilateral or cephalic in 21%. The average time the participants had experienced their symptoms was 6.6 years. The symptoms were present all day in 81% and had fluctuant intensity in 42% of the participants. The tinnitus increased by anxiety in 63% of the participants by loud external sound (27%) and postural changes (10%). The Tinnitus Handicap Inventory (THI) was used for evaluation of the severity and degree of annoyance. An average of 47 points was obtained. The visual analogue scale on tinnitus loudness scored 6.5 ± 2 (range 1–10).

Psychoacoustic measurement of tinnitus pitch showed that 46% had high- frequency tinnitus (>2 kHz) 34% from 0.5 to 2 kHz, and 14% of the participants matched their tinnitus to low frequencies. Average loudness was 9.8 dB ± 8.5 and minimum masking level was 19.3 dB ± 18.5. Two percent reported a temporally complete elimination of the tinnitus with residual inhibition, whereas 56% obtained a partial reduction. Forty-two percent had no changes after sound exposure.

The Spanish version of the Sound Hypersensitivity Questionnaire (THS) was evaluated in another study with 40 participants with DST who were referred to our Tinnitus and Hyperacusis Clinic.

Seventy percent of the participants were female; average age was 48 ± 11 years. Hearing loss was present in 77% of the participants. THS average was 20.1 ± 10.0 points (range 1–45). The questions "I cannot listen or pay attention when intense or annoying sounds from my surroundings are present," "I have to leave when there are intense surrounding sounds," and "I am worried of hearing loss because of exposure to intense sounds" were answered with "yes" by most of the participants. There was a significant correlation ($p < 0.05$) between higher scores of the THS and a higher score in the visual analogue scale and the number of affected activities. The group of DST patients with hearing loss had higher scores in THS, but there was no correlation between the degree of hearing loss (pure-tone average) and the THS scores. Ninety percent of the participants presented tinnitus. The presence of tinnitus and its handicap, according to a visual analogue scale and the THI evaluation, were also correlated with higher THS scores. There was no significant relation between THS values and sex, age, possible etiology, duration of the disease, and loudness discomfort levels [19].

Therapeutical Approaches

There is one basic pillar for hyperacusis treatment: acoustic stimulation. Reaching this objective requires two steps. The first one is counseling. A professional can be able to change the patient's negative feelings about causes of hyperacusis, possibilities for its control, treatment options, and prognosis. Counseling should be focused on positive and evidence-based medicinal information, reducing the patient's emotional reaction and behaviors.

The second step is acoustic stimulation. Controlled and progressive exposure to sound has been demonstrated to be a useful tool in hyperacusis management, as we will see later in this chapter. Patients should avoid regular use of hearing plugs, except for the activities they are not able to perform without ear protection. The continuous use of the earplugs will increase the loudness discomfort levels and will decrease sound tolerance. The combination of counseling and white noise stimulation was developed by Jastreboff on the basis of a neurophysiologic model of tinnitus and named *Tinnitus Retraining Therapy (TRT)* (see Chap. 73). TRT has demonstrated its efficacy for hyperacusis management [2] and is now in routine use in many clinics. According to TRT, sound therapy can be delivered using three systems.

- *Environmental sound enrichment.* Different devices are useful for sound enrichment. A progressive increase in the volume of different kinds of sounds is used to increase sound tolerance in a slow but constant way. This method is effective for mild or moderate hyperacusis.
- *Broad band noise generators (BBNG).* According to Jastreboff's criteria, broad band noise generators should be used when LDL were 80–85 dB or less. Jastreboff reported that 30 percent of the tinnitus patients required hyperacusis management before treatment of their tinnitus [21]. The BBNG is designed to produce two sounds with different spectrums: one covers low and middle frequencies and the other one covers some high frequencies. The digital noise generators can be customized to each patient's preferences. The patient starts the therapy at the maximum volume tolerated without feeling annoyance. In some patients, the volume and time of exposure to the generator has to be increased on a weekly or monthly basis and extended to up to 8 h a day.
- *Hearing Aids.* Patients with hearing loss and moderate or severe hyperacusis will require DST management before being fitted with a hearing aid. This symptom could lead the patient to reject the device. There is also a possibility that the hyperacusis and tinnitus could increase. In a recent study, 41 percent of DST patients in our clinic experienced increased loudness of their tinnitus after exposure to loud sounds [5] (see also Chap. 74). The fitting process has to be slow, progressive, and made in accordance to the patient's tolerance. We recommend that patients first use their hearing aid in quiet places. The use of the device and environmental sound exposure should be increased after this initial period of adjustment. The hearing aid compression systems and the maximum output of the device should be adjusted to avoid annoyance. The use of auditory training and broad band noise generators before the hearing aid fitting helps improve the LDL, dynamic range, and the speech comprehension. This method has been used by other authors, such as Knáster [22, 23], who obtained a reduction of the LDL (recruitment coefficient) in 59% of participants who had unilateral DST and 94% in bilateral DST.

The results of TRT in the management of DST are convincing. Gold reduced the LDL for 2, 3, and 4 kHz in more than 12 dBHL [24] at the end of treatment. Hazell reported that 45 percent of the patients he treated returned to regular LDL after 6 months, and 61% of the patients

had regular LDL in 2 years. The number of activities the patient had to give up because of his DST was reduced from 3.5 to 1.1 after 15 months of TRT [25].

Noreña and Chery-Croze [26] have hypothesized that hyperacusis is caused by enhancement of neural activity in the auditory pathways caused by deprivation of input to the auditory nervous system at the hearing impaired frequencies (see also Chap. 11). The introduction of external sound limited to the impaired frequencies (inverse to the one showed at the audiogram) could progressively reduce the amplification in the auditory nervous system and thereby decrease hyperacusis. The intensity of the sound stimulus should be customized according to the hearing loss as it appears in the audiogram. The differences between TRT's recommended noise stimulation and that suggested in Noreña's study is that in Norena's study, it is limited to the impaired frequencies; there is not a progression of the stimulus intensity. The intensity of the sound used should be kept the same during all training. Our method is based on stimulation with sound (or CDs) of different frequency ranges (2–8 kHz, 4–12 kHz, etc.), but there is no customized intensity for each frequency. The sound intensity is increased gradually according to patient's improvement.

Although TRT is the most used method to treat hyperacusis worldwide, treatment with drugs can be used alone or in combination with sound treatment. Those patients we suspect of having a cochlear hyperexcitability may improve with administration of salicylates because of their ototoxicity [27]. Typical recruitment from non-compensated cochlear diseases (Ménière's disease, sudden deafness, fluctuant sensorineural hearing loss, etc.) can be managed through steroid therapy (systemically or transtympanic delivery). The use of diuretics, betahistine, and sulpiride are common on these clinical entities and can give some relief during acute crisis.

One of the mechanisms described for central hyperacusis has been associated with a decrease in serotonin. Drugs that are selective serotonin reuptake inhibitors (paroxetine, fluoxetine, sertraline) can therefore be helpful to some patients with DST [14, 28]. Drug or cognitive management of anxiety and depression can successfully treat the emotional component of hyperacusis. DST mechanisms based on GABA disorders can be alleviated using $GABA_A$ agonists such as benzodiazepines. Other drugs, such as pregabalin and gabapentin, facilitate the GABA transport over the blood–brain barrier, among other effects. The authors' personal experiences have shown pregabalin to be a useful drug for acute and severe DST in patients with normal hearing.

Conclusions

Hyperacusis is a decreased tolerance to sounds and is estimated to affect 9% of the general population. Pathophysiological mechanisms can be cochlear diseases or disorders in the central auditory pathways, with an abnormal activation of the limbic system that increases the psychological and emotional reaction to the symptom. The combination of professional counseling and acoustic stimulation using controlled sounds (TRT) has been proven to provide relief of decreased sound tolerance in many patients with DST.

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