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Decreased Sound Tolerance and Tinnitus Retraining Therapy (TRT)

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The main objective of Tinnitus Retraining Therapy (TRT) is habituation of activation of the autonomic nervous system, evoked by signals present in the auditory pathways. Sound therapy aims at decreasing the strength of these signals. The same systems in the brain are involved in tinnitus and decreased sound tolerance, and the same basic neurophysiological mechanisms are utilised for decreasing the tinnitus-related neuronal activity and, in case of hyperacusis, abnormally enhanced activity induced by external sounds. The similarity of TRT treatment between tinnitus and misophonia is even closer, as in both situations the goal is to achieve extinction of functional connections between the auditory and the limbic and autonomic nervous systems. The increased gain within the auditory pathways that are presumably responsible for hyperacusis could enhance the tinnitus signal, thus it is possible to expect coexistence of tinnitus and hyperacusis, and the predisposition of hyperacusis patients to develop tinnitus. As such, for some patients tinnitus and hyperacusis may be considered the double manifestation of the same internal phenomenon.

Tinnitus perception is not governed by the same principles as perception of external sounds. Its suppression is not frequency dependent and does not show presence of the critical band, with contralateral suppression frequently being at least as effective as ipsilateral (Feldmann, 1971, 1988). It is impossible to observe beats of tinnitus with external tones, and to re-synthesise tinnitus by any combination of external sounds (Penner, 1993). These observations yielded a postulate that tinnitus is a phantom auditory perception (i.e., it is a real perception of a sound for which there is no corresponding physical correlate) (Jastreboff, 1990).

Clinically-significant tinnitus (i.e., creating problems to an extent that subjects are seeking professional help) involve a number of systems in the brain. Since patients perceive tinnitus as a sound, therefore the tinnitus-related neuronal activity is present within the auditory pathways. The symptoms which patients experience (anxiety, annoyance, strong emotional reactions), strongly suggest that the limbic and autonomic nervous systems are involved as well, and that activation of these systems is responsible for tinnitus becoming clinically-significant. Recent studies, using variety of techniques, strongly supported this postulate (Lockwood et al., 1996; Lockwood et al., 1998; Langner & Wallhdusser-Franke, 1999; Andersson et al., 2000; Mirz et al., 2000; Lockwood et al., 2001).

These concepts were developed in the 80s, published in 1990 (Jastreboff, 1990), and are presently known as the neurophysiological model of tinnitus. The model postulates the involvement of both auditory and non-auditory systems in cases of clinically-significant tinnitus. In these cases, the focus of attention is predominantly on the neurophysiology of the limbic and autonomic nervous systems, with the auditory system playing only secondary role in reactions to tinnitus. Part of the model is devoted to neurophysiological mechanisms,

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which could potentially enhance the tinnitus-related neuronal activity or even contribute to its generation, while the main part focuses on functional connections between auditory and other systems in the brain. There is a consensus that auditory systems display an automatic gain control mechanism, and its sensitivity depends on the average environmental sound level. Experimental results have shown that the sensitivity of the single neurons and their assembles in the auditory pathways indeed increases when sound level is low, when conductive hearing loss is induced, and finally when the cochlea is partially or totally damaged (Gerken, 1979; Sasaki et al., 1980; Gerken et al., 1984; Gerken et al., 1985; Gerken et al., 1986; Salvi et al., 1992; Salvi et al., 1996).

On the other hand, the observation that 94% of subjects developed tinnitus when staying in the anechoic chamber for few minutes (Heller & Bergman, 1953), collaborated the possibility that enhanced sensitivity of the peripheral and central part of the auditory system could contribute to tinnitus (Jastreboff, 1990). It is possible to envision, that in some cases tinnitus and decreased sound tolerance are two manifestations of the same internal phenomenon of increased gain within the auditory pathways (Jastreboff, 1990; Jastreboff & Hazell, 1993b). This hypothesis yielded in turn a prediction that substantial proportions of tinnitus patients should exhibit decreased sound tolerance as well. This postulate was in contrast to literature stating that only 0.1% of tinnitus patients showed symptoms of hyperacusis (Vernon, 1987). This was rather surprising, as analysis of physiological mechanisms of the neurophysiological model of tinnitus suggested that the same networks are involved in tinnitus and some components of decreased sound tolerance.

The only approach to test if these theoretical predictions were correct was by evaluating tinnitus patients for their sound tolerance. While there is no consensus regarding a protocol for the evaluation of decreased sound tolerance, there is a general agreement that pure tone loudness discomfort level (LDL) provides a reasonable estimation of the problem. From 1990, both at University of Maryland at Baltimore and at Emory University in Atlanta, we were routinely evaluating LDL in all new patients, and during some follow-up visits. High prevalence of decreased sound tolerance was evident from the results of the first group of patients (Jastreboff et al., 1994) and indicated that it might affect about 40% of tinnitus patients, with approximately 30% requiring specific treatment for hyperacusis (Jastreboff et al., 1999; Jastreboff & Jastreboff 2000; Jastreboff & Jastreboff, 2002). This observation has been confirmed by other centers (Coles & Sood, 1988; Hazell & Sheldrake 1992; Lux-Wellenhof, 1999; Fabijanska et al., 1999; Gold et al., 1999; Pilgramm et al., 1999; Gold et al., 2002).

Decreased sound tolerance can have an extremely strong impact on patients' lives. It can prevent people from working, social interaction, or participating in variety of pleasant life activities. In extreme cases, decreased sound tolerance can totally control patients' lives. Some patients do not even leave their homes. Not only their lives, but also their families' are totally controlled by the issue of avoidance of sound.

The most recent results from a group of 149 consecutive patients seen at Emory Tinnitus and Hyperacusis Center in Atlanta confirmed high level of prevalence of decreased sound tolerance in tinnitus patients. For each patient, during the initial visit the pure tone LDL were evaluated for both ears, for frequencies 0.5, 1, 2, 3, 4, 6, 8, 12 kHz. The average value of LDL for all frequencies and both ears, was calculated for each patient. The mean LDL value for all patients was 88.66 dB HL with standard deviation equal to 17.85 dB.

The cumulative distribution of the data (Figure 1) provides more insight into the patients' status: 62.4% of all patients have an average LDL value below 100 dB HL, 58.4% below 95 dB HL, 38.3% below 90 dB HL, and 22.8% below 80 dB HL. At the same time 65.8% of these patients stated

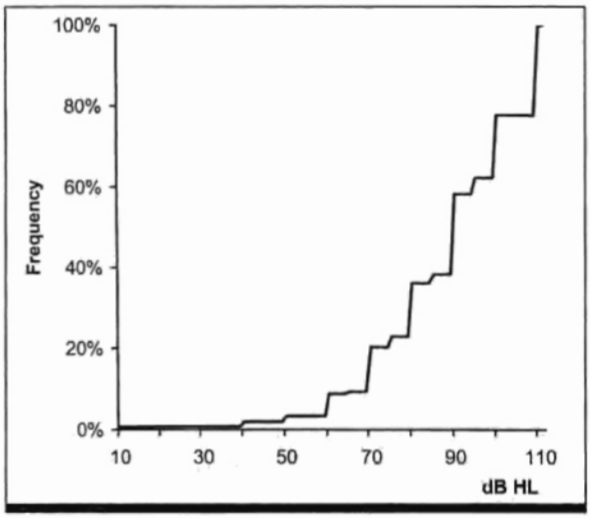

FIGURE 1

Cumulative distribution of loudness discomfort levels (LDL). Vertical axis — proportion of population showing the average value of LDL equal or smaller than value on horizontal axis. Results are presented from 149 patients. Note that about 40% of all cases have average LDL value smaller than 90 dB HL.

during the initial interview that they experience decreased sound tolerance.

The average of LDL for patients reporting problems with sound tolerance was 81.7 dB HL, while for patients not perceiving a problem was 102.0 dB HL. These data are consistent with our past results where the average level below 100 dB HL was suggested as a indicator of potential problems (Jastreboff & Hazell, 1993a), while an average value below 90 dB HL indicates the need for a specific treatment because of decreased sound tolerance.

While average values of LDL provide some guideline for diagnosis, individual data show large variability, and patients' complaints do not simplistically correlate with values of LDL. Some patients with normal values of LDL exhibit strong reactions to everyday sounds (acceptable by others in their surrounding), which significantly affect their life. Other patients, with LDL in the range of 90 dB HL, do not report any noticeable impact on life by these sounds. Another group may have LDL in the range of 20–40 dB HL but, while reporting the problem of the decreased sound tolerance, nevertheless they are able to tolerate some louder than indicated by LDL sounds (e.g., normal level of speech). Many patients exhibit a different level of tolerance to various sounds, which does not correspond to physical characterisation of these sounds (i.e., their spectrum and overall intensity).

The simplest assumption that patients have phonophobia was difficult to accept. The majority of these patients do not show elements of a classical phobia, and furthermore they are not afraid of sound but rather express negative feelings and dislike of sound. The analysis of potential neurophysiological mechanisms underlying reactions of people to sound, resulted in the conclusion that the same systems in the brain are involved in tinnitus and in decreased sound tolerance (see Figure 2).

Considering potential neurophysiological mechanisms involved in processing of signals within the auditory pathways, and interaction of the auditory with other systems in the brain, we proposed that decreased sound tolerance actually consists of two components: (a) hyperacusis which reflects abnormally strong reactivity of the auditory pathways to sound,

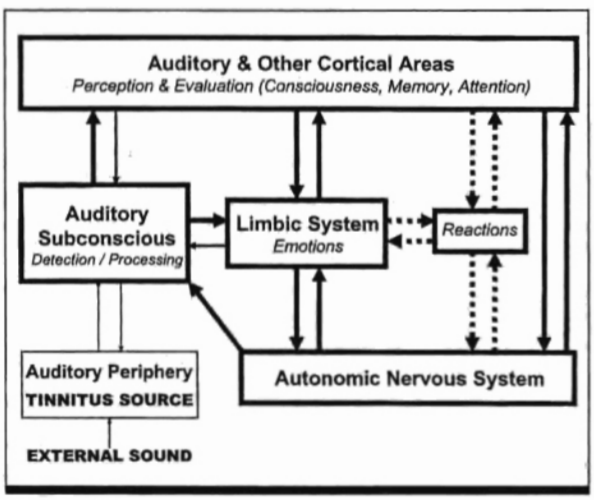

FIGURE 2

Diagram of the neurophysiological model of tinnitus. On this and all subsequent Figures the thickness of arrows and boxes indicates the strength of activations. Note, that both, tinnitus or external sound can serve as initial signal evoking activation of the limbic and autonomic nervous systems, and consequently negative reactions.

and only secondary yielding activation of the limbic and autonomic nervous systems (see Figure 3); and (b) misophonia (dislike / hate of sound) which reflects abnormally strong reactions of the autonomic and limbic systems resulting from enhanced connections between the auditory, limbic and autonomic systems, or enhanced reactivity of the limbic and autonomic system to sound (see Figure 4) (Jastreboff & Jastreboff, 2001; Jastreboff & Jastreboff, 2002). Misophonia includes a broad spectrum of emotions including, but not limited to fear. When fear is a dominant factor, patients are experiencing phonophobia, which is a specific subtype of misophonia.

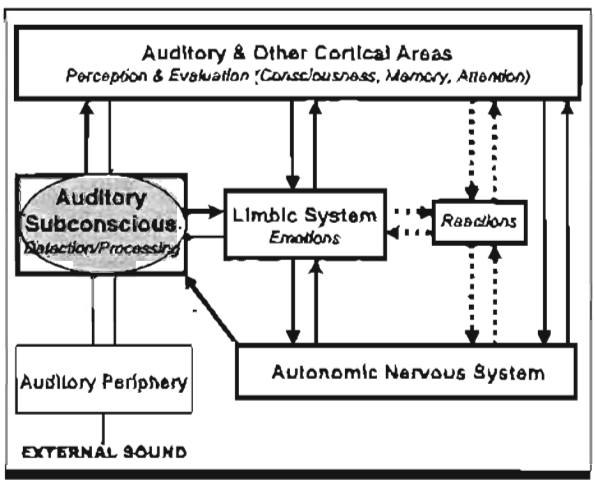

FIGURE 3

Neural systems and mechanisms involved in hyperacusis. Gray oval indicates the primary brain area responsible for hyperacusis.

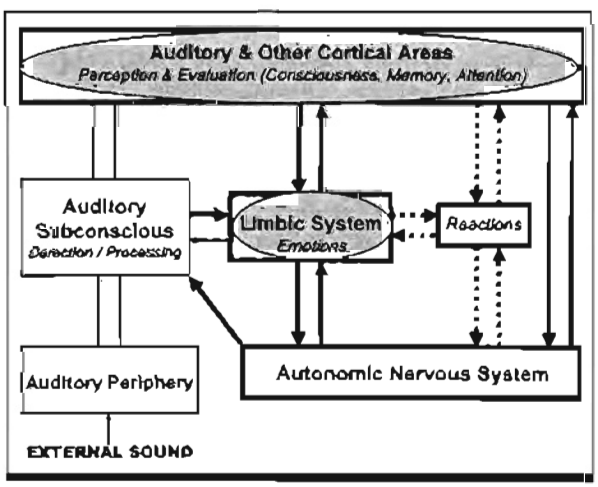

FIGURE 4

Neural systems and mechanisms involved in misophonia. Gray ovals indicate the primary brain areas responsible for misophonia.

In the case of pure hyperacusis, a patient experiences discomfort as a result of exposure to a 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). The reaction to a specific sound will be the same disregarding the context in which it occurs. 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. It is also dependent on the patient's previous evaluation and recollection of the sound, the patient's psychological profile and the context in which the sound is presented.

Abnormally high activation of the autonomic nervous system due to either high amplification / sensitivity of the auditory system subsequently passed to autonomic nervous system (hyperacusis), or by over amplification occurring at connections between the auditory pathways and autonomic nervous system (misophonia) results in behavioral reactions to a sound. Note, that connections involved in misophonia are controlled by conditioned reflexes principles, the same as in the case of tinnitus.

In addition to the LDL evaluation a detailed interview is necessary to determine the presence and extent of hyperacusis and misophonia. LDLs provide an assessment of total decreased sound tolerance (e.g., the sum of hyperacusis and misophonia). Decreased values of LDL are necessary, but not sufficient condition to prove the presence of hyperacusis.

Note, that misophonic reactions are strongly dependent on verbal instructions given to patients, their general feeling at particular day, and the level of trust to audiologist performing the test (the fear of being exposed to highly uncomfortable level of sound). As the extent of misophonia affects the LDL, these factors strongly increase variability of the LDL. Moreover,

for proper treatment of patients with tinnitus and decreased sound tolerance it is important to identify and characterise the hyperacusis component, as it has to be treated first.

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) (Ricketts & Bentler, 1996; Cox et al., 1997). The approach we advocate incorporates modifications of standard procedures aimed at decreasing the effects of misophonic and phonophobic components to a minimum. To minimise the impact of these components, and to assess as closely as possible the presence and the extent of hyperacusis, testing is conducted in such a manner that the patients are aware that they have real full control over the maximal sound level to which they will be exposed. Moreover, testing is performed in a manner allowing patients to become familiar with the procedure and consequently decrease the anxiety and concerns patients may have regarding LDL evaluation. Therefore, we introduced several modifications to standard LDL procedure aimed at decreasing the impact of the misophonic component on LDL values.

Every patient is getting a very specific set of instructions regarding testing procedure, and sound level when response is expected. The measurements of LDL are performed using short pure tones, with the initial level below threshold of hearing and increased in 5 dB (or 1 dB — typically) increments. Testing is performed at the previously mentioned frequencies, and all frequencies are tested twice (i.e., after performing testing for all frequencies the testing is repeated). Only the second set is recorded on an audiogram. It is common for misophonic patients to have the first set of measurements 10–15 dB lower than the second set. While this protocol does not assure total removal of the misophonic component, it decreases its impact on final LDL. As a result the measurements are more stable, and there is high test–retest repeatability.

In the presented group of patients, 65.8% reported some problem with sound tolerance (average LDL = 81.7 dB HL), and 66.4% were diagnosed with decreased sound tolerance (LDL = 82.2 dB HL). However, only 32.9% of all patients were diagnosed with hyperacusis requiring specific treatment (average LDL = 73.5 dB HL). While 57.0% were diagnosed with misophonia (LDL = 81.9 dB HL), only 28.9% had pure misophonia without hyperacusis (LDL = 92.8 dB HL). Finally, 2.7% had only decreased sound tolerance and no tinnitus (LDL = 85.0 dB HL) (see Table 1).

Patients with significant hyperacusis typically develop misophonia as well. Since LDLs represent an estimation of a sum of hyperacusis and misophonia, as expected the average LDLs for patients with hyperacusis, some of whom have misophonia as well, was the lowest. Note, that all LDLs values tend to be around 80 dB HL, for patients exhibiting some problems with decreased sound tolerance, suggesting 80–85 dB HL as an indicator of the need for sound tolerance treatment.

It is important to recognise, that neither hyperacusis, misophonia nor phonophobia have any relation to hearing thresholds, and

TABLE I
Decreased Sound Tolerance and LDL in Discussed Patients' Population

Subgroups	% of population	LDL (dB HL)
Subjective DST	65.8	81.7
Diagnosed DST	66.4	82.2
Diagnosed with misophonia (with or without hyperacusis)	57.0	81.9
Diagnosed with misophonia (no hyperacusis)	28.9	92.8
Diagnosed with hyperacusis (with or without misophonia)	32.9	73.5
Diagnosed with hearing loss and hyperacusis	17.4	75.6
Diagnosed with DST (no tinnitus)	2.7	85.0

Note: Data from 149 consecutive patients. DST — decreased sound tolerance. All subgroups, except last one, represent data from patients with or without tinnitus.

can appear in patients with normal hearing, and various types of hearing loss. These phenomena are not related to recruitment. Recruitment results from the elevated threshold of hearing, and is governed by purely peripheral mechanisms resulting from loss of outer hair cells (OHC) in the cochlea. On the other hand the mechanisms of hyperacusis can be peripheral, central, or mixed, and misophonia always involves only central mechanisms. Potential peripheral mechanisms of hyperacusis might involve dysfunction of the mechanisms controlling decrease of the OHC produced amplification with the increase of a sound level or abnormally high amplification provide by OHC. Indeed, in some patients there is an indication of inappropriate functioning of OHC system, as evaluated by distortion product otoacoustic emission. Central mechanisms of hyperacusis might involve the increase of the sensitivity of neurons in the central auditory pathways after decreased auditory input (dorsal cochlear nuclei, inferior colliculi) (Gerken, 1979; Sasaki, Kauer, & Babitz, 1980; Gerken, Saunders, & Paul, 1984; Gerken, Simhadri-Sumithra, & Bhat, 1986; Salvi, Wang, & Powers, 1996).

Potential mechanisms of misophonia might involve an abnormally strong functional connection between the limbic system and the auditory system, or abnormally high tonic activation of the limbic system. Most frequently, significantly decreased sound tolerance results from a combination of hyperacusis and misophonia.

Our data confirm a lack of relationship between decreased sound tolerance and hearing. There is rather common opinion that hyperacusis is presented only, or predominantly in people with normal hearing. Contrary to this belief, our results show that 55.5% of patients requiring treatment for hyperacusis had hearing loss as well.

At the same time, in the population of patients with hearing loss (91 subjects) 62.6% reported decreased sound tolerance, 57.0% were diagnosed with misophonia, and 29.7% with hyperacusis. 72.5% had high frequency hearing loss, 5.5% conductive and 22.0% other type.

At this moment, it appears to be a consensus that decreased sound tolerance affects a significant proportion of tinnitus patients and of the general population. In this study 65.8% of tinnitus patients exhibited some degree of decreased sound tolerance, with 32.9% requiring specific treatment for hyperacusis. Therefore, assuming that approximately 30% of tinnitus patients require treatment for hyperacusis, considering that 86% of hyperacusis patients reported tinnitus (Anari et al., 1999), and accepting that about 4% of general population have clinically significant tinnitus, it is possible to extrapolate that significant hyperacusis probably exists in at least 1.4% of the general population, and decreased sound tolerance affects a proportion twice as large.

Data from an epidemiological study performed in Poland on 10,349 subjects strongly suggest that this assessment might be highly conservative, as 15.2% of subjects reported decreased sound tolerance (Fabijanska, Rogowski, Bartnik, & Skarzynski, 1999). Notably, tinnitus data from the same population are in agreement with findings from other studies (i.e., 20.1% of subjects experienced tinnitus lasting more than 5 minutes, 5.4% reported clinically-significant tinnitus for which they seek professional help and 40% of tinnitus patients reported hyperacusis as well).

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, medications, and a number of medical conditions such as tinnitus, Bell's palsy, Lyme Disease, Williams Syndrome, Ramsay Hunt Syndrome, failed stapedectomy, perilymphatic fistula, head injury, migraine, depression, withdrawal from benzodiazepines, increased Cerebral Spinal Fluid (CSF) pressure and Addison's disease (Katznell & Segal, 2001; Jastreboff & Jastreboff, 2002). Audiological data indicate that hyperacusis can have both peripheral and

central components (Jastreboff, Jastreboff, & Sheldrake, 1999).

TRT offers an opportunity to treat not only tinnitus but also decreased sound tolerance. Initially, the treatment of decreased sound tolerance was focused on treating hyperacusis. The approach involves the desensitisation of patients through exposure to a variety of sounds (provided by sound generators, table top sound machines, nature sounds, radio, TV, etc.). Sound therapy for treatment of hyperacusis has several differences, as compared with sound therapy used for tinnitus. Avoidance of silence and continued exposure to background sound is even more important than for tinnitus patients. The sound level from sound generators needs to be closer monitored (which results in standard for recommendation using sound generators), and may require modification during the treatment.

The desensitisation approach has been promoted for some time with a variety of protocols and types of sounds utilised; such as the recommendation of using sound with certain frequencies removed, short exposures to moderately loud sound, or prolonged exposures to low level sounds (Vernon & Press, 1998). According to principles of the neurophysiological model of tinnitus, desensitisation is recommended and it is used as a part of TRT (Jastreboff & Jastreboff, 2000). Both theory and our results of treatment of hyperacusis were presented already (Jastreboff et al., 1996a; Jastreboff et al., 1996b; Jastreboff et al., 1998; Jastreboff 1998).

However, the misophonic component cannot be removed by desensitisation and a separate approach needs to be implemented. Recognising the similarity of neural networks involved in tinnitus and misophonia and the involvement of conditioned reflexes we promote a method based on active extinction of conditioned reflexes (Konorski, 1948; Konorski, 1967), which involves systematic exposure of patients to sounds associated with a pleasant situation, with gradually increasing sound levels. A handout with protocol for misophonia, as given to patients, is shown in the Appendix.

Both, desensitisation and active extinction process produce effects relatively fast and, typically, patients with decreased sound tolerance show fast recovery, with a number of them reaching the maximal levels of sound allowed for testing LDLs without experiencing discomfort. Effectively, it is possible to achieve not only improvement, but a cure for decreased sound tolerance in a significant proportion of patients. If only hyperacusis is treated without recognising and treating misophonia results are unpredictable and recovery might take a long time, or even treatment might be unsuccessful.

As 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 both hyperacusis and misophonia in each patient (Jastreboff, Jastreboff, & Sheldrake, 1999; Jastreboff & Jastreboff, 2000), as they need to be treated using different methods (Jastreboff & Jastreboff, 2001; Jastreboff & Jastreboff, 2002). After a detailed interview and audiological testing, a medical evaluation is advisable to rule out medical conditions, which might be related to tinnitus or decreased sound tolerance.

In conclusion, several issues are worth pointing out. The same systems in the brain are involved in tinnitus and decreased sound tolerance, and the neurophysiological model of tinnitus provides theoretical guidance for treatment of both conditions. Basically, the main difference between tinnitus and misophonia is that in the case of tinnitus there is no external sound, and the tinnitus signal is stable. However, for both phenomena, the same neuronal pathways are activated, with the final product being activation of the sympathetic part of the autonomic nervous system, which in turn is responsible for behaviourally observed problems.

Analysis of the neural mechanisms responsible for hyperacusis and misophonia suggests specific clinical approaches aimed at removing these problems. In the case of pure hyperacusis, where the problem is constrained to the auditory pathways and

conditional reflexes are not involved, effective treatment can be based on desensitisation implemented by exposing the subject to relatively low levels of neutral sounds. This approach would not work for misophonia where conditioned reflexes linking the auditory with the limbic and autonomic nervous systems play a crucial role. In the case of misophonia the reflexes are the same, with substitution of an internally generated signal (tinnitus) by a variety of external sounds (misophonia).

For both tinnitus and hyperacusis, part of the problem arises from the presence of unusual activity within the auditory pathways: for tinnitus — an abnormal, typically weak neuronal activity; for pure hyperacusis — abnormally enhanced neuronal activity induced by external sounds. For both tinnitus and hyperacusis, the problem arises from overactivation of the autonomic nervous system by these signals. In the case of misophonia, the activity within the auditory system is normal, but it causes abnormal activation of the limbic and autonomic nervous systems.

TRT is aimed at habituation of activation of autonomic nervous system evoked by the signal present in the auditory pathways, and a part of protocol (sound therapy) aims at decreasing the strength of this signal. In the case of tinnitus, the strength of the tinnitus-related neuronal activity is directly decreased by increasing background neuronal activity. This is accomplished by exposing the patient to additional external sounds. Sounds used to provide this additional auditory background should not induce annoyance or any other negative reactions. These sounds decrease the overall gain of the auditory system as well. In the case of hyperacusis, a partial decrease of the signal strength is achieved immediately, due to the same principle of difference with background. For example, measurements of LDLs, while the patient is provided with sound from sound generators, result in higher values of LDLs. It is common for hyperacusis patients to note immediate protective properties of sound from sound generators. We recommend increasing the sound level of sound generators when patients expect to be in a louder environment. As amplification of the sound-induced neuronal activity in the case of significant hyperacusis appears to be relatively large, the main effect of desensitisation is a gradual decrease of this gain, typically achieved in a span of several weeks or months.

Nevertheless, for both tinnitus and hyperacusis the aim of TRT is to decrease the strength of neuronal activity within auditory pathways and to eliminate activation of the autonomic nervous system. The similarity of TRT treatment between tinnitus and misophonia is even closer. In both cases, the signal coming from the auditory system to the limbic and autonomic nervous systems is weak (in case of tinnitus), or normal (for misophonia). However, abnormal amplification of these signals, occurring at the connections between the auditory, the limbic and autonomic nervous systems, is responsible for the problem. TRT is aimed at elimination of these functional connections, that is, an extinction of conditioned reflexes linking these systems (habituation of reactions). The main difference between tinnitus and misophonia is that we can manipulate external sounds (but not tinnitus). Therefore, it is possible to implement a more powerful method of active extinction of these conditioned reflexes by associating sounds with positive reward. This is impossible in the case of tinnitus where the more cumbersome method of possible extinction is utilised. Nevertheless, the basics of the methods are identical for tinnitus and misophonia.

An interesting aspect of decreased sound tolerance is that affected subjects typically have a tendency to notice, and to be aware of somatosounds (e.g., swallowing, chewing, jaw movements), as well as external sounds, with patients concentrating on all sounds. This is turn, causes patients to avoid sound, enhances tendency to remain in silence, and overprotect their ears. All these factors, result in further enhancement of misophonia, hyperacusis, and tinnitus. TRT results in habituation of perception of these sounds

(similarly to habituation of tinnitus perception), removes factors worsening these problems, and thus provides an extra benefit to these patients.

Finally, as increased gain within the auditory pathways will enhance an already existing low level of the tinnitus-related neuronal activity and consequently may cause crossing the threshold of its detection, it is possible to expect that subjects with hyperacusis may have the tendency to develop tinnitus as well. On the other hand decrease of this gain should result in a decrease of the tinnitus signal. As such, for some patients tinnitus and hyperacusis may be considered the double manifestation of the same internal phenomenon — "two sides of the same coin".

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APPENDIX

TREATMENT OF MISOPHONIA

Misophonia (dislike of sound) is treated by training involving engagement in activities which you enjoy, and which have sound as an inevitable component.

The main concept is to create association of sound with a pleasant situation. This can be achieved by engaging in activities which you like, and which have sound as an indispensable part, such as listening to music, shopping in a mall, going to parties, restaurants and so forth. You should always have a full control over the situation and to be able to discontinue sound exposure at any time, if you would like to (for example, listening to music at home but not going to a concert).

The most common method is to listen to music at home. In this case, you should select your most favourite type of music and listen to it attentively once / twice every single day for 20–40 minutes. The music should have reasonably stable level without going from very quiet to very loud.

The treatment consists of 3-week cycles, repeated as many times as it is needed.

- For the first week, the sound volume should be set every day for the most comfortable level.
- For the second week, the level should be increased by one just noticeable step louder than the most comfortable level for a given day.
- For the third week, sound volume should be increased by another step, that is, sound is initially set at most comfortable level and then increased by two just noticeable steps louder.

Then this 3-week cycle is repeated.

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