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Tinnitus Retraining Therapy (TRT) as a Method for Treatment of Tinnitus and Hyperacusis Patients

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Abstract

The aim of this paper is to provide information about the neurophysiologic model of tinnitus and Tinnitus Retraining Therapy (TRT). With this overview of the model and therapy, professionals may discern with this basic foundation of knowledge whether they wish to pursue learning and subsequently implement TRT in their practice. This paper provides an overview only and is insufficient for the implementation of TRT.

Key Words: Habituation, hyperacusis, neurophysiologic model, tinnitus, Tinnitus Retraining Therapy, treatment

Abbreviations: DPOAE = distortion product otoacoustic emission, IHC = inner hair cells, LDL = loudness discomfort level, OHC = outer hair cells, THT = Tinnitus Habituation Therapy, TRT = Tinnitus Retraining Therapy

Tinnitus, commonly referred to as "ringing in the ears," is a common problem affecting many people around the world. According to studies performed in various countries, tinnitus affects 10 to 20 percent of the general population (McFadden, 1982; Coles, 1987; Drukier, 1989); in the United States, this translates into 25 to 52 million Americans. This affliction is even more prevalent in the elderly over the age of 65 years, with approximately 30 percent reporting tinnitus (Sataloff et al, 1987; Salomon, 1989). For about 5 percent of the general population (about 13 million Americans), prolonged tinnitus is moderately or significantly annoying, causing them to seek help (McFadden, 1982). Consequently, this population is labeled as having clinically significant tinnitus. Finally, 1 out of 100 adults reports tinnitus as a debilitating problem (Coles, 1996) (about 2.6 million Americans). Typically, tinnitus is associated with hearing loss, otosclerosis, ear infections, acoustic neuroma, Meniere's syndrome, and aging. Also, more than 200 prescription and nonprescription drugs list tinnitus as a potential side effect.

Much less is known about the prevalence of increased sensitivity to sound, hyperacusis. Currently, in the literature there are limited data published by Vernon (Vernon, 1987; Vernon and Press, 1998), Coles (Coles and Sood, 1988), Hazell and Sheldrake (Hazell and Sheldrake, 1992), and Jastreboff (Jastreboff et al, 1996b, 1998). Data presented by Vernon are incongruent with the remaining reports, stating that only 0.3 percent of tinnitus patients have hyperacusis, whereas other data indicate that about 40 percent of tinnitus patients have some degree of hyperacusis. Furthermore, our data indicate that about 25 percent of tinnitus patients are bothered more by their hyperacusis than their tinnitus, and thus require specific treatment for hyperacusis.

We are not aware of epidemiologic data related to the prevalence of hyperacusis in the general population. This is an unfortunate situation, since these data could help in estimating the need for health services and for planning cost-effective, yet quality, services. Assuming, however, that in our practice we are working

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with patients with clinically significant tinnitus (5% of the general population), and 25 percent of those have significant hyperacusis, then about 1.25 percent of the general population (3.25 million Americans) has significant hyperacusis. This is a rather conservative estimate, as there are cases of hyperacusis without tinnitus.

In spite of a long recorded history of tinnitus, reaching as far back as the ancient Babylonian and Egyptian civilizations (Feldmann, 1988), and its high prevalence today, there is no cure for tinnitus. It appears that all approaches used in the past failed to provide systematic relief to tinnitus patients. Practically all of the treatments previously used were effective only on a subpopulation of patients, had to be continued through the patient's life, and were frequently accompanied by significant side effects. Furthermore, the very existence of a long list of treatments that may potentially provide help, and the fact that the single most common approach is telling patients "to learn to live with it," argues strongly against their effectiveness. In this paper, we propose that Tinnitus Retraining Therapy (TRT), when implemented properly, (1) is highly effective, (2) does not have side effects, (3) needs to be implemented over a finite amount of time, and (4) can be used on all patients.

Definitions

As there are various definitions of tinnitus, hyperacusis, and phonophobia, we are presenting the definitions, as proposed by us, to ensure a clearer understanding.

Tinnitus is commonly defined as a noise in the ears or head, frequently described as ringing, buzzing, humming, hissing, the sound of escaping steam, etc. In 1982, the Committee on Hearing, Bioacoustics and Biomechanics proposed a definition of tinnitus as "the conscious experience of a sound that originates in the head of its owner" (McFadden, 1982). The definition of tinnitus we are promoting is "the perception of a sound which results exclusively from the activity within the nervous system without any corresponding mechanical, vibratory activity within the cochlea" (Jastreboff, 1995), that is, tinnitus as an auditory phantom perception (Jastreboff, 1990, 1995).

Somatosounds are sounds generated by structures in and adjacent to the ear, including spontaneous otoacoustic emission. The term "objective tinnitus" has been used to describe somatosounds. This classification is inaccurate because it is dependent on the equipment used and the skill of the observer, not the pathophysiology of the sound.

Hyperacusis is defined as abnormally strong reactions occurring within the auditory pathways resulting from exposure to moderate sound; as a consequence, patients express reduced tolerance to suprathreshold sounds. This phenomenon may be, but typically is not, related to recruitment (Moore, 1995; Jastreboff, 1998; Jastreboff et al, 1998).

Phonophobia is defined as abnormally strong reactions of the autonomic and limbic systems (without abnormally high activation of the auditory system by sound), resulting from enhanced connections between the auditory and limbic systems. This can be described at the behavioral level as "patients being afraid of sound."

Increased sound sensitivity is abnormally high sensitivity to a sound resulting from the sum effects of hyperacusis and phonophobia.

The seemingly simple definition of tinnitus has profound implications on proposing the mechanisms of tinnitus and consequently on its treatment. This definition stresses the involvement of the nervous system as a key component responsible for the emergence of tinnitus and problems arising from its presence, thus moving its mechanisms away from the cochlea to the central nervous system. The definition further indicates the existence of a link between the mechanisms of tinnitus and that of the phantom limb and phantom pain phenomena, which indeed appear to exist. Certain common aspects of tinnitus and phantom pain are used for the classification of tinnitus and hyperacusis patients and their treatment.

OUTLINE OF THE NEUROPHYSIOLOGIC MODEL

Proposed in the 1980s (Jastreboff, 1990), development of the neurophysiologic model of tinnitus was initiated by several observations. First, epidemiologic studies revealed that tinnitus induces distress in only about 25 percent of the tinnitus population (McFadden, 1982), and there is no correlation of the distress with psychoacoustic characterization of tinnitus, that is, average loudness of tinnitus, its pitch, and maskability are similar in people who are only experiencing tinnitus to those who suffer because of it (Jastreboff, 1995). Second, the psychoacoustic characterization of tinnitus in the patient population is not related to the severity of

tinnitus (i.e., two people with a similar characterization of tinnitus often differ dramatically in the level of distress created by their tinnitus). The same observation applies to the treatment outcome, which is not correlated to the loudness, pitch, or maskability of tinnitus (Jastreboff et al, 1994).

The above facts argued very strongly for the auditory system as secondary only and other systems in the brain being dominant in clinically relevant tinnitus. Moreover, Heller and Bergman (1953) showed that the perception of tinnitus cannot be pathologic, since essentially everyone (94% of people without tinnitus experience tinnitus when isolated for several minutes in a anechoic chamber) experiences it when put in a sufficiently quiet environment.

Consequently, the neurophysiologic model of tinnitus postulates that both abnormalities in the cochlear function and the processing of a tinnitus-related signal within the nervous system must be included in the analysis of tinnitus phenomenon. Specifically, in the emergence of clinically relevant tinnitus, it is possible to distinguish the following stages: (1) the generation of tinnitus-related neuronal activity initiated in the periphery of the auditory system (the cochlea, auditory nerve), (2) the detection of this signal occurring in subcortical auditory centers, (3) the perception and evaluation of the signal at cortical areas (auditory and others), and (4) the sustained activation of the limbic (emotional) and autonomic nervous systems. If negative associations are not attached to the person's tinnitus, then only the first three stages occur; therefore, the person only experiences tinnitus without being annoyed by its presence. The fourth stage is crucial for creating distress and, consequently, clinically relevant tinnitus. Tinnitus-induced activation of the limbic and autonomic nervous systems is responsible for the distress caused by tinnitus.

The involvement of these two systems was indicated by the problems reported by tinnitus patients. Patients with clinically significant tinnitus exhibit a strong emotional reaction to its presence, a high level of anxiety, and a number of psychosomatic problems (Jastreboff, 1990; Kuk et al, 1990; Stouffer and Tyler, 1990; Stouffer et al, 1991; Newman et al, 1995). In general, these reactions depend on the activation of the limbic and autonomic nervous systems. The limbic system consists of an array of brain structures including the hippocampal formation, amygdala, septum, and hypothalamus. This system has a direct influence on neuroendocrine and autonomic function and controls emotional expression, seizure activity, memory storage and recall, and the motivational and mood states (Swanson, 1987). The limbic system plays a role in all aspects of life, which involve motivation, mood, and emotions. Furthermore, it activates the autonomic nervous system.

The autonomic nervous system, one of the two main divisions of the nervous system, controls the action of glands, as well as the functions of the respiratory, circulatory, digestive, and urogenital systems. The system also has some control over the production of hormones. The autonomic nervous system consists of two distinct, mutually antagonistic components, the sympathetic and parasympathetic. The sympathetic division stimulates the heart, dilates the bronchi, contracts the arteries, inhibits the digestive system, and prepares the organism for physical action. The parasympathetic division has the opposite effect and prepares the organism for feeding, digestion, and rest (Brooks, 1987). Because of the exacerbated activation of the autonomic nervous system, many body functions are affected. Typically, patients exhibit syndromes that indicate the sympathetic division of the autonomic nervous system as dominant, which stimulates the heart, inhibits the digestive system, and, in general, prepares the body for physical action. The overactivation of the sympathetic nervous system leads to problems with sleep, a very common situation among tinnitus patients (Coles, 1996).

Abnormally high activation of the limbic and autonomic nervous systems results in stress, anxiety, and loss of well-being. The patients are getting extremely annoyed by their tinnitus. Feedback loops connecting the auditory, limbic, and autonomic nervous systems (Fig. 1A) are getting stronger, and patients continue to get worse.

The feedback loops develop in the following manner. The continuous, uncontrollable presence of tinnitus, or belief that something bad is going to happen, causes the tinnitus-related neuronal activity to become linked to a negative reaction in the brain (i.e., annoyance, anxiety, general stress). Due to this negative reaction, more attention is automatically directed toward tinnitus, which, in turn, enhances the detection of the tinnitus signal by subcortical auditory centers. This enhanced detection of the tinnitus signal increases activation of the limbic and autonomic nervous systems, which, in turn, increases the attention devoted toward tinnitus, etc. Consequently, the sustained activation of limbic and autonomic nervous systems occurs.

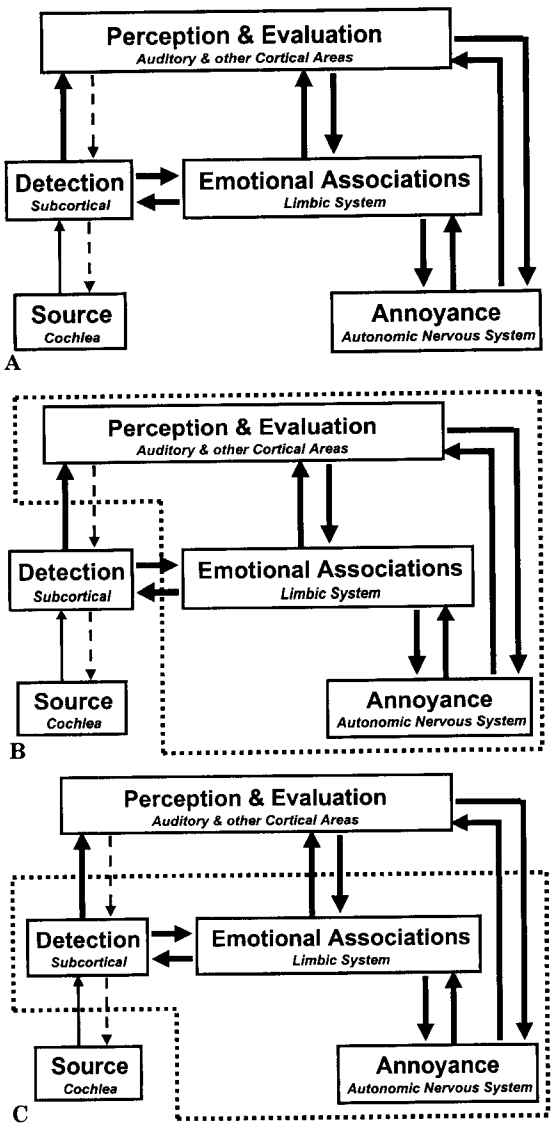

Figure 1 Block diagram outlining systems involved in clinically relevant tinnitus and changes occurring as a result of tinnitus habituation. Thickness of the arrows indicates the significance of a given connection. *A*, main diagram, *B*, upper loop, *C*, lower loop.

Certain aspects of this activation are important. First, the activation level depends on the strength of the negative associations and not on the perceptual description of tinnitus. The increase of annoyance and anxiety is a self-propelling process, with the maximal level depending upon the association of tinnitus with something negative, as well as on the psychological profile of the patient, but is not related to psychoacoustic characterization of the perceived sound of tinnitus. Second, a high level of activation of the sympathetic part of the autonomic nervous system induces the fight or flight reaction and suppresses the ability of the patient to enjoy life. Frequently, in cases of severe tinnitus, patients no longer enjoy activities previously pleasant to them, which, in turn, may yield depression.

From a clinical point of view, it is interesting to recognize that tinnitus can acquire negative associations through (1) a prolonged, continuous presence of a neutral stimulus (e.g., a neighbor's son playing the same song over and over again); (2) fear of a new, unknown danger; and (3) "negative counseling" (e.g., "nothing can be done; you will have to learn to live with it; let's do an MRI to exclude a brain tumor"). Unfortunately, negative counseling is very common and triggers the development of a vicious cycle, and patients devote increasingly more time to monitoring their tinnitus and experience problems with attention, work, sleep, etc.

Once established, the reactions of the limbic and autonomic nervous systems are induced, along the principle of conditioned reflex. In the diagram presented in Figure 1, two loops are depicted: (1) upper, cortical—verbal (Fig. 1B), which may involve beliefs and can be directly affected by counseling, and (2) lower, subcortical—nonverbal (Fig. 1C), which can be controlled only indirectly. Cognitive therapies acting on high cortical levels affect the upper loop. Since there is no consistently successful outcome of cognitive therapies in a majority of patients, this argues that the lower, subconscious loop plays the dominant role.

While we postulate that the auditory system, including the cochlea, plays a secondary rather than primary role in the emergence of clinically significant tinnitus, it does, in fact, provide the initial signal that starts the cascade of events resulting in the development of clinically significant tinnitus. The specific mechanisms triggering the source of tinnitus can vary from patient to patient.

Neurophysiologic experiments provide insight to potential mechanisms, yielding the perception of tinnitus. The emergence of tinnitus in subjects placed in a very quiet environment may be explained by the following observation: there is a high level of spontaneous activity within the auditory pathways that is random and is not perceived under normal conditions. This activity could be labeled as a code for silence and, under normal conditions, is filtered out by subcortical centers and does not result in the perception of sound. However, when the level of cochlear stimulation is decreased, it has been shown that the sensitivity of the auditory pathways increases with about 25 percent of

the neurons exhibiting abnormally low threshold to any stimulation (Boettcher and Salvi, 1993; Salvi et al, 1996). Under a condition of increased neuronal sensitivity, the auditory system starts to detect the inevitable fluctuation of randomness of the spontaneous activity, which is perceived as tinnitus.

The concept of tinnitus-related neuronal activity, resulting from the compensation performed by the auditory system to even mild dysfunction within the cochlea or the auditory nerve, has been further elaborated in the postulate of discordant damage/dysfunction theory (Jastreboff, 1990, 1995), which is an extension of the theory proposed by Tonndorf (Tonndorf, 1987). Typically, damage of the cochlea affects more outer (OHC) than inner (IHC) hair cells, resulting in unbalanced activity, which reaches the dorsal cochlear nucleus through Type I and Type II auditory nerve fibers. If the imbalance is large enough, it evokes a compensation within the auditory pathways to such an extent that tinnitus-related neuronal activity is generated as a side effect of this compensation. Potential mechanisms are speculative but may involve lateral inhibition (Liberman and Kiang, 1978; Liberman and Mulroy, 1982; Gerken, 1992, 1993) or disinhibition (Chen and Jastreboff, 1995). Indeed, abnormal, bursting, epileptic-like spontaneous neuronal activity has been recorded from the inferior colliculus in animals with salicylate-induced tinnitus (Chen and Jastreboff, 1995) and recently in animals with sound overexposure (Jastreboff et al, 1999).

Hyperacusis and Phonophobia

We propose that the increased sound sensitivity (decreased sound tolerance) consists of two components: hyperacusis and phonophobia. Hyperacusis may result from both peripheral and central mechanisms. In the cochlea, two types of dysfunction in the active amplification, provided by the OHC, could result in hyperacusis. Normally, OHCs amplify weak sounds (<10–20 dB SPL) by 66 to 76 dB, and the amplification gradually decreases to 0.2 dB/dB in the range of 40 to 80 dB (Ruggero et al, 1997). The first type of peripheral overamplification occurs if OHCs continue to amplify louder sounds, and then IHCs become overstimulated for moderately loud sounds levels. The second type of peripheral overamplification occurs when OHC mechanical amplification increases to larger values than present at a normal state. Indeed, measurements of the distortion product otoacoustic emission (DPOAE) suggest that, in some patients, dysfunction in the OHC system may be responsible for their hyperacusis.

The central type of mechanism involves oversensitivity of neurons in the auditory pathways. About 25 percent of cells in the cochlear nuclei complex and the inferior colliculi exhibit abnormally high sensitivity and abnormally strong evoked potentials if the auditory inputs is decreased (Boettcher and Salvi, 1993; Gerken, 1993). An abnormally high central gain might result in the detection of fluctuations in the spontaneous activity or a weak abnormal pattern of activity that would otherwise not be detected as tinnitus. Thus, for some patients, tinnitus and hyperacusis can be two manifestations of the same neuronal mechanism.

In the case of pure hyperacusis, the abnormal gain is constrained to the auditory system and moderate sounds induce a high level of activity within the auditory pathways, and only secondary in the limbic and autonomic nervous systems, involved in preparing the subject to withdraw from the unpleasant sound level. Since in pure hyperacusis (i.e., without simultaneous phonophobia), the actual amplitude of the cochlear basilar membrane vibrations and the processed physical intensity of the sound are the dominant factors, the physical parameters of the sound are the determining factors for the extent of discomfort. The context in which the sound occurs (e.g., home, doctor's office, movie theater) is irrelevant, and the reaction to a given sound is the same under all of these various conditions. The physical parameters of the transduction of the cochlea and the relation of dB SPL and HL can result in a flat curve of loudness discomfort levels (LDLs), with tendency of decreased values for very low and very high frequencies (Fig. 2A).

After prolonged exposure to sound perceived by an individual as too loud, an activation of these systems could occur for increasingly lower sound levels, as phonophobia, the fear of sound, may develop. Moreover, pure phonophobia (i.e., without the presence of hyperacusis) can be found in patients believing in the harmful effects of sound and their attempts to overprotect their ears (ear plugs, etc.). In pure phonophobia, the auditory system works normally. However, a relatively low level of the activation within the auditory system results in an overactivation of the limbic and autonomic nervous systems due to the enhanced connectivity between these systems. The reaction to a given sound depends upon the context in which the sound occurs.

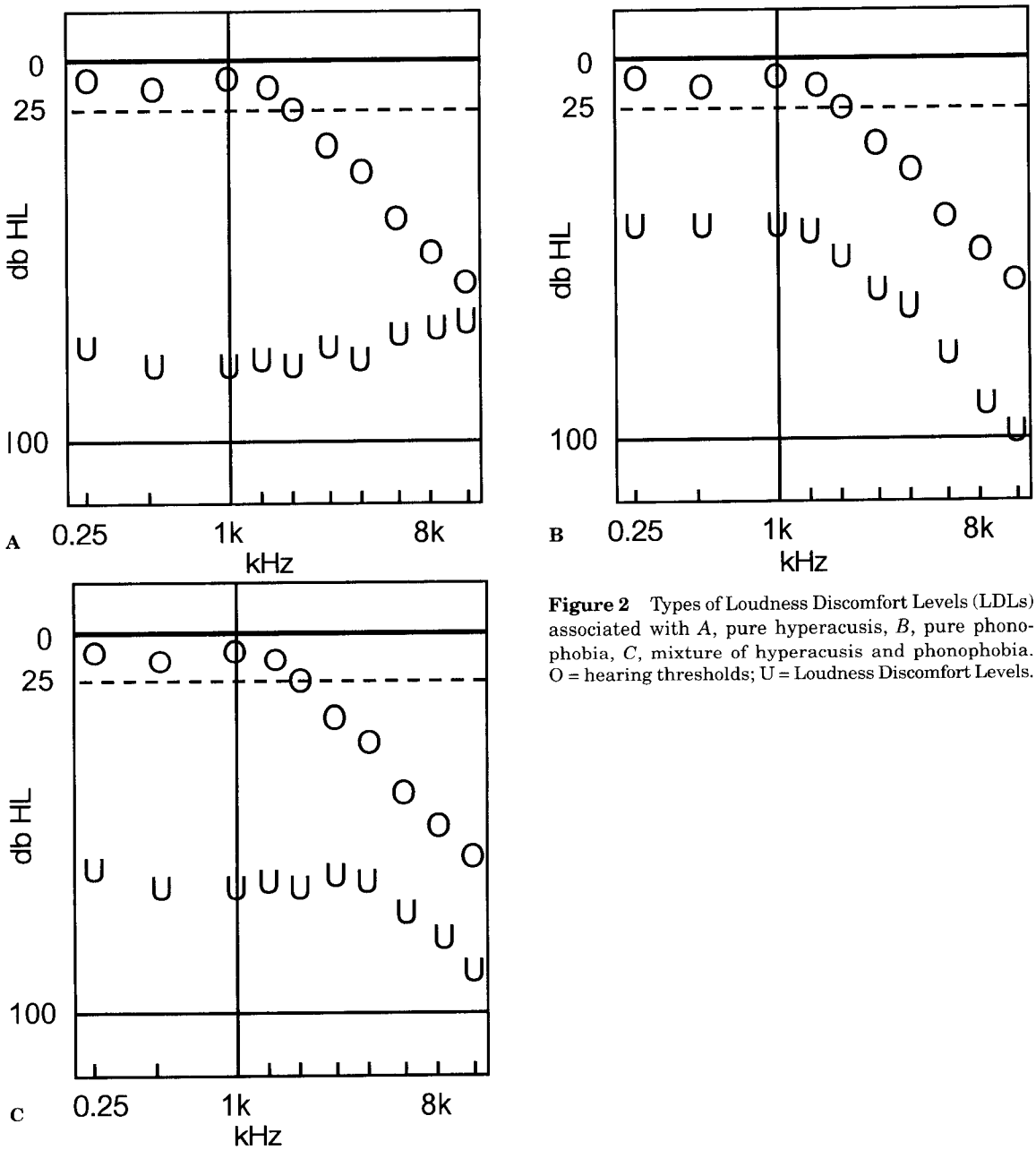

Figure 2 Types of Loudness Discomfort Levels (LDLs) associated with *A*, pure hyperacusis, *B*, pure phonophobia, *C*, mixture of hyperacusis and phonophobia. O = hearing thresholds; U = Loudness Discomfort Levels.

Patients may over-react to certain sounds, while not reacting to more intense sounds. Since the reactions depend upon the perceived loudness of the sound (related to dB SL) and not its physical intensity, the LDLs will keep a relatively constant distance from the audiogram following in consequence the shape of audiogram (Fig. 2B). In patients with exclusive or strong phonophobia, there is a tendency of extremely low values of LDLs. Most frequently, hyperacusis and phonophobia coexist, with typical LDLs, as shown in Figure 2C.

Some patients exhibit prolonged (days of weeks) worsening of their tinnitus and/or hyperacusis as a result of an exposure to sound. There is a need to separate this phenomenon from the temporarily enhanced phonophobia, which is treated differently.

In summary, it is postulated that tinnitus-related neuronal activity results predominantly from the compensatory action of the auditory pathways to a peripheral dysfunction, perhaps a difference in the damage of OHCs versus IHCs. In about 75 percent of tinnitus cases, this activity is contained within the auditory system and is frequently blocked before it reaches the level of awareness. Consequently, these people experience tinnitus but do not suffer because of it.

However, in clinically relevant cases, as a result of initial negative associations, tinnitus-

related neuronal activity inappropriately activates the limbic and autonomic nervous systems, resulting in the development of annoyance, anxiety, sleep disturbances, and a number of somatic problems. Connections between the auditory and other systems are based upon the principle of conditioned reflexes and as such cannot be easily and directly changed. Furthermore, the limbic and autonomic nervous systems may be completely normal, and the problem arises from their activation by inappropriate stimulus (i.e., tinnitus-related neuronal activity).

Hyperacusis and phonophobia activate the limbic and autonomic nervous systems as well, but with different mechanisms than tinnitus. Once established, reactions of these systems are controlled by the conditioned reflex principle but triggered by external sounds rather than tinnitus-related neuronal activity.

How to Treat Tinnitus and Hyperacusis

The neurophysiologic model offers an approach to treat both tinnitus and hyperacusis. For tinnitus, the approach is based on observations that, although there is no reliable method for attenuating the tinnitus source (cure), the brain exhibits a high level of plasticity. It is possible to habituate to any sensory signal, as long as the signal is not associated with any negative implications. Thus, the conclusion is to induce habituation of tinnitus (by interfering with tinnitus-related neuronal activity above the tinnitus source).

Habituation is a normal, common, and necessary function of the brain, as pointed out by Konorski in 1967 (Konorski, 1967) following the original postulate from Pavlov in 1928. Its necessity results from the fact that although the brain can detect very weak sound patterns if significant (our name called out in a noisy room, cry of our baby, our language), it cannot handle more than one conscious task at any given time (inability to read a book and write a letter; to understand someone talking while reading; to listen to two people talking at the same time). The question is how do we manage the huge amount of sensory stimulations that bombard us all of the time? For example, how are we able to drive a car?

The solution to this problem performed by the brain is to (1) select and block all unimportant stimuli from reaching our awareness at the subconscious level and block reactions that these stimuli would otherwise evoke (habituate); (2) automize as many tasks as possible into the subconscious, nonverbal reflexes (driving a car, eye movements when reading, walking, running, etc.); (3) prioritize all remaining tasks; and (4) perform one task at a time, starting from the most important.

Note that the selection and blockage of unimportant signals have to occur at a subconscious level on the basis of past experiences. The selection process cannot be done on a conscious level, as it would consume our attentional abilities and nullify the purpose of selection.

On the basis of the neurophysiologic model, it has been proposed to treat tinnitus by inducing its habituation. To achieve habituation, a specific approach based on the general principles of the brain function (e.g., the physiologic mechanisms of perception, the role of subcortical auditory pathways, the functional properties of the limbic and autonomic nervous systems and their interaction with the auditory system, mechanisms of conditioned reflexes, and the physiologic mechanisms of the brain plasticity) was suggested (Jastreboff, 1990, 1995; Jastreboff et al, 1996a). Use of habituation to help tinnitus patients was first proposed by Hallam et al (1984), but their model was psychological rather than physiologic. Consequently, their approach to induce and sustain habituation was different, focused on reassurance, relaxation, and attention distraction (Jakes et al, 1986), and did not appear to produce significant, sustained improvement in tinnitus patients.

Habituation of tinnitus has two main goals. The primary clinical goal is to habituate reactions of the limbic and autonomic nervous systems (Fig. 3A). Tinnitus-related neuronal activity is blocked from reaching both systems. Consequently, patients who achieve full habituation of their reactions do not experience annoyance, anxiety, or any other negative reactions of their brain and the body. Note that the patients still perceive their tinnitus and, in case of exclusive habituation of reactions, the proportion of the time when they are aware of tinnitus is still the same. Nevertheless, since these patients no longer are bothered by their tinnitus, even when perceived, tinnitus ceases to be an issue.

The secondary goal is to achieve a habituation of the perception (Fig. 3B). In this case, tinnitus-related neuronal activity is blocked before it reaches the level of awareness, and patients are unaware of the presence of tinnitus. Note that even when a very high level of habituation of the reaction and perception is achieved, it is still not a cure for tinnitus, as patients can

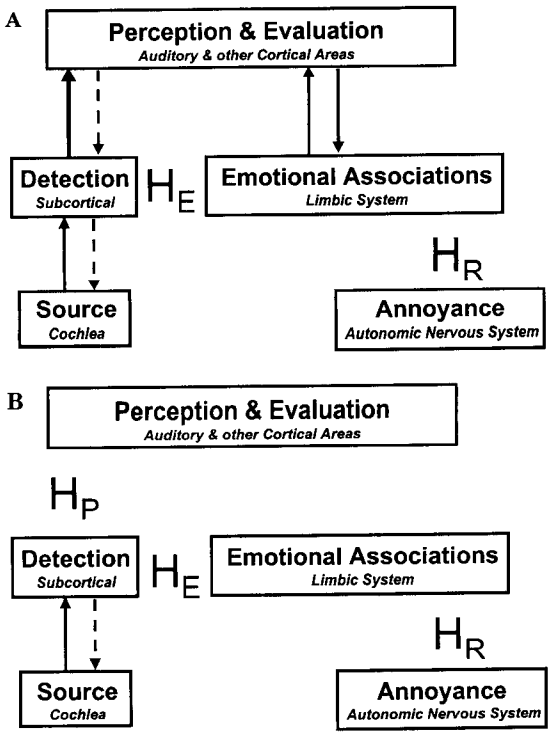

Figure 3 Classes of habituation. *A*, habituation of reactions, *B*, habituation of perception and reactions. Abbreviations: H_R = habituation of reactions of the autonomic nervous system, H_E = habituation of emotional reactions involving the limbic system, H_P = habituation of perception.

hear their tinnitus anytime when their attention is focused on it; also, the tinnitus pitch and loudness are the same as at the beginning of the treatment. As predicted by the model, tinnitus maskability may decrease a few decibels (Jastreboff et al, 1994), but the change is too small to have a practical significance for patients.

Although some patients achieve a high level of tinnitus habituation, to be of clinical significance, habituation does not have to be complete, and patients can experience significant improvement even with partial habituation of reactions and perception. The final goal of the treatment is that tinnitus ceases to have an impact on the patient's life.

In conclusion, Tinnitus Habituation Therapy (THT), aimed at inducing tinnitus habituation, has been proposed as a treatment for tinnitus on the basis of the neurophysiologic model of tinnitus (Jastreboff, 1990). Since the habituation-related modifications of the nervous system occur above the source of tinnitus, the etiology of tinnitus is irrelevant, and any type of tinnitus, as well as somatosounds, can be treated by inducing habituation.

TINNITUS RETRAINING THERAPY

Outline of the Treatment

Habituation can be achieved or facilitated by a number of approaches, including counseling, combined, for example, with medications, biofeedback, hypnosis, etc. However, the easiest implementation of THT involves a parallel use of retraining counseling with sound therapy. This therapy has become popular under the name of Tinnitus Retraining Therapy (TRT). These two components perform different functions and both have to be used for a method to be called TRT.

Retraining counseling acts to decrease the level of stimulation from the cortical areas of the brain to the limbic and autonomic nervous systems and to decrease the general level of activity within these two systems. During counseling, a number of points are presented to the patient: (1) the perception of tinnitus results from a compensation occurring within the auditory system, (2) tinnitus is a problem because of the activation of emotional (limbic) and autonomic nervous systems, and (3) by using the plasticity of the nervous system, it is possible to retrain the brain to achieve habituation of tinnitus-induced reactions and tinnitus perception. Once the patient accepts these ideas as realistic and "making sense" to them, this puts tinnitus into the category of neutral stimuli, to which they may gradually habituate. The crucial point to recognize, for both the therapist and the patient, is that tinnitus-induced reactions are governed by the conditioned reflex principle. Consequently, the conscious realization of the benign nature of tinnitus is not sufficient to remove these reactions, and a significant amount of time is needed for their gradual extinction.

Sound therapy provides significant help in the process of habituation by decreasing the strength of tinnitus-related neuronal activity within the auditory system and from the auditory system to the limbic and autonomic nervous systems. Therefore, the strength of tinnitus-related activation within all loops presented in Figure 1 decreases, making habituation of tinnitus easier.

Specifically, sound therapy acts by providing the auditory system with constant, low-level, neutral auditory signals to (1) decrease the contrast between tinnitus-related neuronal activity and background neuronal activity, (2) interfere with the detection of the tinnitus signal, and (3) decrease enhanced gain within the auditory pathways.

All of our senses are acting upon the principle of gradient or the difference between a signal and its background. The absolute physical intensity of stimulus is secondary. The same sound appears louder and evokes more detectable change in the neuronal activity when there are no other competing sounds. When the same sound is presented with some additional auditory background, it will appear to be softer, and the neuronal activity evoked by it will become more difficult to detect. Presently, we cannot directly decrease the tinnitus-related neuronal activity. However, by enhancing background neuronal activity, through exposing patients to low-level sounds, the relative strength of the tinnitus signal decreases, thereby making habituation easier.

Enhancement of the background sound can be provided by enrichment of environmental sounds, which can be further amplified by hearing aids for patients with a significant hearing loss or by the use of special sound generators. Notably, the sound is of importance and not any particular means or device providing it.

From the perspective of achieving habituation, the masking of tinnitus is counterproductive, since it prevents detection of tinnitus (signal to be habituated), thus preventing, by definition, habituation. Even partial masking is not recommended, as it will change the tinnitus signal, and habituation would occur to this modified signal. Once the external sound is removed and the tinnitus signal is restored to its initial characteristics, it will not be effectively habituated. The relationship of the effectiveness of habituation from the sound intensity is presented in Figure 4.

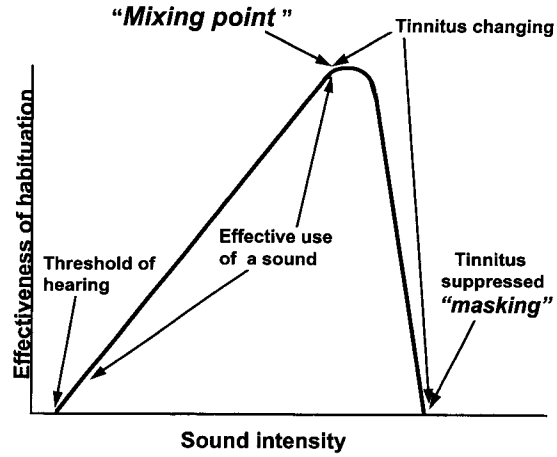

Figure 4 Theoretical dependence of the effectiveness of habituation on the intensity of the sound used for the sound therapy.

Stages of the Protocol

The treatment consists of an initial appointment and a number of follow-up visits. The initial visit, which is described in some detail below, consists of (1) initial contact with the patient, (2) audiologic evaluation, (3) medical evaluation, (4) diagnosis with decision regarding the treatment category, (5) retraining counseling, and (6) fitting/counseling. Although the initial appointment is of great importance, the follow-up visits, even if not very frequent, are crucial in order to achieve a high level of TRT effectiveness.

Initial Contact with the Patient

The first contact with a patient establishes the basis for future interactions and allows significant information, essential for a specific diagnosis to be gathered and for the discernment of an individualized treatment for the patient. The initial contact with a patient is made through a questionnaire sent in the mail to those individuals who expressed an interest to be treated in the center. This questionnaire is further expanded by an interview, performed before an audiologic or medical evaluation, which is guided by the Tinnitus/Hyperacusis History form. The main goals of the interview are to (1) identify complaints and any resulting problems, (2) determine the impact of tinnitus on the patient's life, (3) assess emotional status and the degree of distress, and (4) evaluate the influence of sound exposure on the problems.

Audiologic Evaluation

The audiologic evaluation is helpful in assessing and separating issues of hearing, tinnitus, hyperacusis, and phonophobia in each case. The results are used as the basis for individualized counseling, choosing appropriate treatment, and as a reference for evaluation of the treatment outcome during follow-up visits. After otoscopic examination of the ear canal and tympanic membrane, a series of tests are performed. Routine audiologic testing, consisting of an evaluation of pure-tone thresholds (up to 12 kHz) and word recognition scores, provides an assessment of a patient's hearing and a basis for subsequent tinnitus measurements.

Specific tinnitus/hyperacusis measurements consist of the evaluation of pitch matching (for the most troublesome tinnitus), loudness match-

ing, the minimal masking level, and LDLs. Pitch and loudness matching provide information useful for counseling but not for diagnosis. The crucial measurement is that of LDLs, evaluated using pure tones up to 12 kHz, as well as the frequency that corresponds to the tinnitus pitch. Measurements are performed twice, and the second set is recorded.

DPOAEs allow assessment of the function and integrity of the OHC. This information is predominantly used for counseling patients, but it can also be useful in the characterization of OHC-related hyperacusis (Jastreboff et al, 1998).

Acoustic immittance, which provides some insight into the integrity of the ascending pathways of the auditory nerve, is not routinely tested. This test is not performed if probe tone levels or acoustic reflex levels exceed LDLs (probe tone signal of 226 Hz is approximately 60 dB HL).

Other tests, such as auditory brainstem response or electronystagmography, are performed only if there is an indication of medical problems extending beyond tinnitus. Both tests are interesting for research purposes, but at this stage they do not provide any clinically useful information for patient treatment.

In summary, a basic audiogram with LDLs is the crucial test for diagnosis and assessment of the treatment outcome. The remaining measurements are useful for individualized counseling and population studies.

Medical Evaluation of Tinnitus/Hyperacusis Patients

The medical evaluation of patients with tinnitus/hyperacusis is directed at identifying medical conditions that may cause, contribute to, or have an impact on the treatment of tinnitus. The main goal is to exclude any known medical condition that has tinnitus as one of its symptoms. Typical examples would include an acoustic neuroma, Meniere's disease, or Lyme disease. In fact, if such a condition is diagnosed, the treatment is focused on alleviating this (primary) cause of tinnitus. There is a danger of delaying appropriate treatment of the medical problem if TRT is successfully used to treat tinnitus before proper medical diagnosis. Moreover, during counseling, the perception of tinnitus is presented to patients as benign, resulting from a compensatory mechanism within the auditory system. Therefore, it is necessary to be sure that this statement is true before presenting it to patients.

During the medical evaluation, the main information regarding tinnitus and hyperacusis is rechecked, followed by a detailed otolaryngologic and general medical evaluation. Somatosounds might also be detected during this evaluation. However, since TRT is effective in their treatment, typically, somatosounds are not treated medically or surgically.

A frequently asked question from professionals who work in an audiologic clinic without physicians on site is whether they can treat tinnitus patients. The answer is yes, but they should require a statement from an otolaryngologist that the patient's tinnitus can be treated without the risk of removing tinnitus as a symptom of another medically treatable disease.

Patient Categories

The patients are placed into one of five general categories (Table 1) (Jastreboff, 1998). Although all patients receive counseling and sound therapy, including the advice to "avoid silence," there are substantial differences for both components in each category.

Categories of patients and their treatments are based on four factors. The first is the extent of impact tinnitus or hyperacusis has on the patient's life and the duration of tinnitus. This reflects the strength of the connection formed between the auditory system and the limbic and autonomic nervous systems. The second is the patient's subjective perception of hearing loss, with stress placed on the subjectivity of this perception. The third is the presence or absence of hyperacusis; threshold of significant hyperacusis is defined as average LDLs below 100 dB HL. It is necessary to assess the relative contribution of hyperacusis and phonophobia since LDLs reflect the sum of both phenomena. The fourth characteristic is a prolonged worsening of hyperacusis and/or tinnitus following exposure to moderate or loud sounds. This effect is of particular significance, as it is a characteristic feature of patients with hyperacusis difficult to treat, including hyperacusis resulting from Lyme disease. Forty-eight percent of patients with Lyme disease have hyperacusis, which exhibits prolonged worsening of the symptoms as a result of exposure to moderate or even very low sound levels (Fallon et al, 1992). Some patients without Lyme disease may also exhibit a similar effect.

Although a number of patients report worsening of their tinnitus or hyperacusis as a result of exposure to sound, in most cases, this wors-

Table 1 Categories of Tinnitus and Hyperacusis Patients

Category	Hyperacusis	Prolonged Sound-Induced Exacerbation	Subjective Hearing Loss	Impact on Life	Treatment
0	—	—	—	Low	Counseling only
1	—	—	—	High	Sound generators set at mixing point
2	—	—	Present	High	Hearing aid with stress on enrichment of the auditory background
3	Present	—	Not relevant	High	Sound generators set above threshold of hearing
4	Present	Present	Not relevant	High	Sound generators set at the threshold; very slow increase of sound level

Hyperacusis: significant sensitivity to environmental sounds typically associated with LDLs below 100 dB HL; prolonged sound-induced exacerbation of tinnitus/hyperacusis when the effects persist to the following day; subjective hearing loss: perceived subjectively by a patient as having a significant impact on patient's life; impact on life: the extent of impact of tinnitus and/or hyperacusis on patient's life; common treatment for each category involves counseling and the use of enriched auditory background.

ening lasts only a few minutes or hours. However, in some cases, it can last for days or weeks. If the patient experiences worsening of their tinnitus and/or hyperacusis the morning after the sound exposure, then the patient is classified as having a prolonged impact to noise exposure. The resulting categories from this classification are presented in Table 1.

Category 0 consists of patients who do not have hyperacusis or any significant hearing loss, and whose tinnitus has little impact on their life. For these patients, the directive counseling session, including the advice to avoid silence and to enrich their sound environment, is usually sufficient and there is no need for any instrumentation. Patients with a recent onset of tinnitus, not exceeding more than 2 months, and who have not received any "negative counseling," leaving them with many concerns and a little hope for the potential improvement (Jastreboff and Hazell, 1993), belong to this category as well.

Category 1 consists of patients who have significant tinnitus but no hyperacusis and no subjective hearing loss. For these patients, the most effective approach is the use of sound generators set at the level close to the "mixing"/"blending" point. This is the sound level corresponding to the beginning of partial suppression ("partial masking"). Patients describe it as when the external sound and tinnitus can be heard separately but start to interfere or intertwine with each other. Most tinnitus patients belong to this category and exhibit a high level of success in controlling their tinnitus.

Category 2 consists of patients with the characteristics of Category 1 but additional significant subjective hearing loss. For these patients to achieve improvement in both tinnitus and hearing, we recommend hearing aids. We instruct the patient to wear them all of the time while enriching their sound environment. It is stressed to the patient that sound is important for the treatment and not the hearing aids. The main purpose of the hearing aids is to amplify sound, whereas providing better communication is secondary.

Category 3 consists of patients with significant hyperacusis, which is not enhanced, for a prolonged period of time, as a result of sound exposure. Tinnitus may or may not be present. Sound generators are necessary to help desensitize the auditory system and consequently to decrease/remove hyperacusis. The desensitization protocol begins with the sound level set close to, but clearly above, the threshold of hearing. This level is increased during the treatment to the level appropriate to their tinnitus (if present). These patients tend to recover faster than patients with tinnitus only.

Category 4 consists of patients who have tinnitus and/or hyperacusis and exhibit prolonged worsening of their symptoms as a result of sound exposure. This is the most difficult category of patients to treat, and the success rate is lower than in the other four categories. In this case, we set the level of sound generators at the threshold of hearing. In cases where there is general hypersensitivity of perception of any type, not just sound, the patients are advised to wear the devices for a week without turning them on. This is done in order to desensitize the patient's perception of the touch to devices in their ears. As the treatment progresses, the

sound level is increased very slowly. These patients need continuous monitoring and typically exhibit profound phonophobia.

Specific Issues of Treatment of Hyperacusis and Phonophobia

In most cases, hyperacusis can be treated directly by a process of gradual desensitization of the auditory system. If hyperacusis is present (e.g., Categories 3 and 4), then it must be treated first, before the tinnitus. After the patient shows improvement in his/her hyperacusis, the tinnitus is addressed more directly. Frequently, however, as the patient gets the hyperacusis under control, the tinnitus becomes less of an issue. For the hyperacusis patient, it is even more important than for patients with tinnitus only to have an enriched sound environment in addition to the use of instruments. It is also important to discontinue the overuse of ear protection as it causes an increase in the sensitivity of the auditory system due to decreased auditory input.

Typically, patients combine and confuse hyperacusis and phonophobia. LDLs, although essential, are not sufficient for the diagnosis of hyperacusis; therefore, a detailed interview is necessary. Once hyperacusis is at least partially under control, patients are treated for their phonophobia by training involving engagement in activities that they enjoy and have sound as an inevitable component.

Instruments

In theory, all patients who do not have hyperacusis can be treated without instrumentation. In practice, however, it is advisable to use some form of instrumentation for most patients (except Category O) due to the following reasons. First, about 40 percent of patients have hyperacusis (Jastreboff et al, 1996b). For these patients, there is a need for a well-controlled, stable sound source, such as sound generators. Second, the increased ease of implementing sound therapy by patients who use sound generators results in better compliance with the protocol. Third, for patients with significant subjective hearing loss (Category 2), the use of hearing aids provides an additional benefit in improved hearing. A wide variety of hearing aids may be used. The general principle is the use of a high-quality, programmable hearing aid, fit with an open mold, to ensure significant improvement of hearing under various environmental conditions, protection against overstimulation (by use of higher than typically selected compression ratio), and prevention of the attenuation of low-frequency environmental sound. In-the-canal hearing aids are not recommended except in cases with a significant low-frequency hearing loss, since they will attenuate low-frequency environmental sound, which, in turn, typically results in the increase of tinnitus.

In cases of unilateral deafness and tinnitus, CROS, BICROS, or transcranial stimulation, combined with training to improve space localization of the sound, is recommended. The goal is to reactivate parts of the auditory pathways that received decreased input as a result of deafness. This approach is based on recent developments in neuroscience.

It is common knowledge that the nervous system exhibits an enormous amount of plasticity and that information from various sensory systems is integrated into a coherent entity. The visual and vestibular systems are classic examples of such a collaboration. It also has been recognized that, in the absence of sensory input, phantom perception occurs (phantom limb, phantom pain, tinnitus), with accompanied reorganization of receptive fields. A few years ago, a new dramatic development was reported for controlling phantom pain and phantom limb by using multisensory interaction (Ramachandran and Rogers-Ramachandran, 1996). Phantom pain and phantom limb frequently cannot be controlled by any pharmacologic or surgical approach. However, by introducing visual input, it turned out to be possible to control phantom pain in patients with one of their hands amputated. These patients were instructed to put the healthy hand into the box with a glass top and the mirror inside, so they saw only the healthy hand and its mirror reflection, which mimicked the missing hand, and to move the hand. After several sessions, the phantom pain, which could not be controlled by other means, disappeared. Presumed mechanisms of action involved reorganization of receptive fields of somatosensory representation of the hands by visual input and partially restoring the balance disturbed by the lack of sensory input from the missing hand. Recent data with fMRI strongly supported this postulate (Borsook et al, 1998).

This information had direct effect on the treatment of tinnitus in patients with profound unilateral hearing loss or unilateral deafness. Although the high level of plasticity of the nervous system was recognized long ago, the extent of plasticity and reorganization of receptive fields within the auditory system was not sufficiently appreciated. Recent data changed this situation

dramatically, with results showing reorganization of the tonotopic cortical maps due to the presence of tinnitus (Muhlnickel et al, 1998). Based on the results with phantom pain, the ideas was to use the combined actions of the auditory and visual systems by fitting patients with CROS, BICROS, or transcranial systems. This provided them with auditory information from a whole auditory space, which, in combination with the information from the visual system, would restore spatial localization of the sound and modify receptive fields in the auditory pathways. The clinical results confirmed that these patients had partially restored their space localization of auditory stimuli (tested with closed eyes). Furthermore, as hoped, this method also was helpful for their tinnitus. A systematic study on a large number of cases is needed, but results obtained so far are very encouraging.

There are a variety of sound generators. The ideal sound generator should (1) minimally interfere with the perception of external sound, (2) allow for a smooth increase in the sound level from the threshold of hearing, (3) provide a reasonably wide frequency range of generated sound, (4) provide a stable amplitude and spectrum of sound, and (5) be cosmetically pleasing.

Although none of the 18 models of sound generators evaluated by us were ideal, a number of them may be used for treatment. At the moment, we recommend the three most interesting devices: (1) Silent Star (Viennatone/ReSound, USA), (2) Tranquil (General Hearing Instruments, class D model with enhanced high frequencies), and (3) the sound generator from Audifon (Spain).

Two sound generators are always recommended, including cases of unilateral tinnitus. This is to ensure symmetric stimulation of the auditory pathways and avoidance of abnormal modification of connections and receptive fields within the auditory system, thereby pushing perception of tinnitus to one side.

Follow-up Visits

To achieve the habituation of tinnitus, TRT is aimed at the reversal or retraining of the feedback loops formed between the auditory, limbic, and autonomic nervous systems. Modifications of these loops occur in a dynamic balance scenario. Under a dynamic balance condition, the annoyance that the person experiences acts (continuously) to increase the strength of connections between the auditory system and other systems in the brain. When actively promoted, habituation acts to decrease the strength of those connections. The outcome depends upon the relative strength of the patients' annoyance and habituation. Consequently, the continuous push toward habituation is needed to counteract continuous negative reinforcement and enhancement of reflex loops connecting the tinnitus signal with reactions of the limbic and autonomic nervous systems provided by tinnitus-evoked annoyance. Therefore, the counseling component of the follow-up visits is essential in order to provide a patient with continuous promotion and facilitation of habituation. The sound therapy alone without directive counseling is not sufficient. However, once weakening of the insidious connections reaches a sufficient level, further habituation occurs spontaneously.

Results

The process of habituation is slow and gradual, with fluctuations of tinnitus severity, including transient perception of tinnitus worsening observed in some patients about 3 to 4 weeks after the initiation of the treatment. This paradoxical phenomenon is actually a positive sign, as it probably indicates the beginning of habituation. Patients experience temporary relief due to partial habituation, but when tinnitus returns to the previous state it appears to be worse, due to the contrast with the previous period of habituation. Initial improvement is typically seen within the first few months, followed by further gradual improvement. Clear results can be seen in about a year, but to prevent a relapse it is recommended that treatment last at least 18 months. Some patients follow the protocol even longer and experience further improvement.

Due to the lack of an objective method for measuring tinnitus, selecting the proper criteria for assessing the effectiveness of the treatment outcome is of fundamental importance. Multidimensional evaluation is necessary with an emphasis on changes and increased involvement in life activities that were previously prevented or interfered with by tinnitus or hyperacusis (e.g., the patient has resumed square dancing). Comparison of the initial questionnaire with the follow-up questionnaire allows observation of changes that may have occurred in life activities.

Moreover, patients are asked to assess their tinnitus awareness, annoyance, and effect on life on a scale of 0 to 10 before, during, and after

treatment is performed. To classify a patient as showing "significant improvement," the following criteria are used: (1) at least one activity previously prevented/interfered with is no longer affected or all activities show improvement; (2) tinnitus awareness is decreased by at least 20 percent, the impact of tinnitus on life is decreased by at least 20 percent, and tinnitus annoyance is decreased by at least 20 percent; (3) evaluation was performed after at least 6 months of treatment and is repeated at least once, with the last assessment performed not later than 3 years after initiation of the treatment; and (4) an improvement in more than one category. If there is improvement in only one category, then the patient is classified as showing no improvement.

Note that the results described below are not an outcome of the planned studies designed to validate TRT protocol but rather a brief summary of the retrospective analysis of the treatment outcome of a nonpreselected sample of the University of Maryland Tinnitus and Hyperacusis Center patients. The progress of the treatment was monitored by the use of questionnaires during initial and follow-up visits or telephone interviews. Of 263 patients with whom we were able to keep contact, 90.1 percent received instruments (82.5% sound generators and 7.6% hearing aids) and stated that they were following TRT. Of these patients, 9.9 percent received one session of counseling, including information about sound therapy and no instruments. These patients typically did not follow TRT.

Combining results obtained from all 263 patients, including those who decided not to follow TRT, revealed that 75 percent of patients reached the level of significant improvement as defined above. The results are even more optimistic (above 80%) for patients using noise generators or hearing aids as a part of TRT. On average, the indices for awareness, annoyance, and life quality decreased to about half of their pretreatment values. Additionally, the analysis of the relationship of the treatment outcome with the presence of hyperacusis performed on 163 cases revealed that patients with hyperacusis (combined Categories 3 and 4) showed a higher rate of improvement than patients with tinnitus only (Categories 1 and 2).

CONCLUSIONS

As with any method, TRT has both positive and negative aspects. On the positive side, TRT appears to be highly effective for both tinnitus and hyperacusis, can be used to treat all types of patients, does not require frequent visits, and does not interfere with hearing, and there are no negative side effects. The protocol requires limited time for treatment, and many successful patients who completed the treatment and stopped using the devices have no need to use them several years later; tinnitus is not a problem in their life. Moreover, approximately 20 percent of these patients achieved a block of the perception of their tinnitus to the extent that they could not hear tinnitus even when focusing their attention on it (Sheldrake et al, 1996).

The main negative aspect of TRT is that the protocol has to focus on the individual needs and profile of a patient, consequently requiring significant time involvement of the personnel providing the treatment, who have to be specifically trained. Moreover, the development of specific plastic changes within the nervous system (leading to habituation of tinnitus) requires about 18 to 24 months, as estimated from our retrospective observation of the patients.

Although we are not claiming that TRT is the only method to treat tinnitus patients, nor that it is finalized, we believe, that when implemented properly, TRT is effective in helping tinnitus and hyperacusis patients. Since it is based upon a scientific model, it can be further tested and refined. It is not a cure, since it does not remove tinnitus, but by inducing habituation of tinnitus-induced reactions and tinnitus perception, it allows patients to achieve control of their tinnitus, live a normal life, and participate in everyday activities. TRT does not promise a 100 percent success rate, but we feel it is justified to promote it as an effective treatment for tinnitus and hyperacusis patients. Nevertheless, this should not stop efforts to search for better methods, particularly research on mechanism-based tinnitus alleviation.

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