TINNITUS – STATE OF THE ART AND RETRAINING THERAPY

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SUMMARY – Tinnitus is an abnormal noise in the ear. About six percent of the general population suffer from what they consider to be 'severe' tinnitus. Tinnitus can come and go, or be continuous. It can sound like a low roar, or a high-pitched ring. Tinnitus may be bilateral or unilateral. The causes of tinnitus are various, e.g., inner ear injury, 8th nerve lesion, injury of the brainstem, and rarely of the brain. There also are many extracranial causes of tinnitus. Upon making the diagnosis of tinnitus, medical therapy may occasionally help lessen the noise even though the cause has not been identified. Current therapy for tinnitus, so-called tinnitus retraining therapy, first includes learning about what does actually cause the tinnitus. This process is called habituation of reaction. Tinnitus then becomes quieter for long period of time and may eventually disappear, or becomes part of the background 'sound of silence' (habituation of perception). In some cases, changes in the inner ear function may be important in triggering the occurrence of tinnitus (e.g., Meniere's disease or acute acoustic trauma), however, the retraining approach works independently of the triggering factor. Despite the importance of hearing loss, a recent study in tinnitus patients showed that there was no significant difference in hearing between the tinnitus group and control group of healthy subjects.

Key words: *Tinnitus, etiology; Tinnitus, diagnosis; Tinnitus, therapy*

Introduction

The conscious awareness of sound takes place near the surface of the brain when a pattern of electrical activity traveling up the nerve of hearing from the ear reaches a point just below the auditory cortex. The auditory nerve has about 30,000 different fibers, and patterns of electrical activity in these fibers are matched with other patterns kept in the auditory or hearing memory. The cochlea or inner ear, which transforms sound waves into these electrical patterns, is a noisy place, where continuous mechanical and electrical activity in 17,000 hair cells can now be monitored with sensitive, computer enhanced, listening devices[1]. Most of what we hear is a sequence of sounds, like speech or music. In infancy, new sound experiences are stored in a relatively empty auditory cortex. Later on, there is a continuous process of matching familiar memory patterns with those coming from the ear. Each time a pattern from the ears is matched with a pattern in the auditory memory we have the experience of hearing a sound[2]. Putting together these matched patterns starts a process of evaluation. Another part of the brain close to the hearing center is involved in the meaning of what we hear, and in interpreting the language. If it is a foreign language, we can hear the sound but may not understand the meaning.

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The Meaning of Sound

Sound is of enormous importance in monitoring our environment. In animals hearing has to be very sensitive

and specific. The ability of animals to develop extremely acute hearing to detect very weak sounds of a distant predator has contributed to the survival of the species to the present. These warning signals produce acute anxiety prompting appropriate action to avoid attack, so-called survival reflex.

We respond in the same way to the sound of motorcar horn, stepping automatically back on the pavement or sidewalk. Some sounds can be identified as warning signals, while others can evoke a feeling of security or pleasure. We have this experience every day with sounds that alarm us, or sounds that soothe us such as music, or the sounds of nature.

When a sound has a special meaning, like a baby waking at night, or floor board creaking, or the sound of our first name, we respond to it in an automatic manner. This happens after a short learning period but the responses can remain as strong as ever throughout one's life. During sleep, the conscious part of the brain is 'shut down', so we do not hear, see or feel anything. This shows that weak sound patterns, if of great meaning, can be detected by the subconscious filters in the hearing pathways, between the ear and the brain (auditory cortex). The conditioned response also triggers the activity outside the auditory system where there are large numbers of connections with the limbic system, which is concerned with emotion and learning. Also activated is the autonomic nervous system, which turns on the body's 'ignition key' to prepare for any possibility, particularly 'fight or flight', and gets the body ready for action. High levels of autonomic function tense muscles, raise heart and breathing rates, cause sweating, and are the complete opposite to the state of relaxation. They rightly preclude sleep, or concentration on other, less important tasks. Most of our day to day activity consists of a series of conditioned responses, executed to order. So, each and every sound that we hear and learn the meaning of, has an 'emotional label' attached to it, which may change from time to time according to how we feel it ourselves and the context in which we hear it. For example, the sound of a neighbor's television set may be acceptable, or unpleasant and intrusive, depending on whether it belongs to a well loved friend or relative, or to somebody else whom for various reasons we dislike.

The Meaning of Tinnitus Sounds

Just as the animal alerted to danger by the sound of a predator focuses solely on that sound in order to survive, so those who consider that tinnitus is a threat or warning signal are unable to do anything but listen to it. It is part of the mechanism that humans have developed for self-preservation, although clearly in this situation it is not working to our advantage. Many people complain of the loss of silence, something they previously greatly treasured and enjoyed, before tinnitus has become persistent.

So, what happens, even in mild cases of persistent irritating tinnitus, is that a conditioned response (reaction) is set up to the tinnitus sound (the sound evoked by background electrical activity in auditory nerve cells). As this conditioned response occurs in the subconscious part of the brain, what you may be thinking about tinnitus at any time (or even if you are not thinking about it), is irrelevant to the reaction produced[3].

Moreover, it is the reaction to tinnitus which is important in creating distress. That is, the degree to which unpleasant feelings about it (from the limbic system) and increased tension (from autonomic system stimulation) are experienced, every time the tinnitus signal is detected.

When tinnitus first emerges it is a new signal, there are no memory patterns, and no means of categorizing it. Any new experience tends to produce a feeling of discomfort due to the loss of the *'status quo'* and a change in what was previously a stable environment (a change in 'homeostasis'). Until proper evaluation has been undertaken of what tinnitus means, it will be regarded with understandable suspicion. Many 'sufferers' only experience mild annoyance from tinnitus as a result of this mechanism, but it may be sufficient to promote the need to seek help. For many sufferers, tinnitus is quite threatening. Some people fear that tinnitus means they have some kind of serious illness. Others are convinced that the experience of 'disco tinnitus' means permanent damage to the ear rather than temporary protective 'shut down'. There are patients who worry about the possibility that it heralds a brain tumor, blood clot, or some serious mental illness ("... it will drive me mad!"). These anxieties are almost always unfounded. Many people fear that tinnitus will get louder, continue for ever, and cannot be cured. Even the concept that tinnitus is invading one's 'right to silence' constitutes a threat, very similar to the territorial invasions that animals experience. It is often feared that tinnitus will continue to spoil peace and quiet, interfere with concentration at work, quiet recreational activity and ability to sleep at night. Unfortunately, these fears may be enhanced by professional advice or reports from others of their own, phobic reaction to tinnitus. Many physicians and other professionals still advise patients that there is nothing that can be done about tinnitus and that it will go on forever. Oth-

er people fear that tinnitus may mean that their hearing is becoming impaired. Tinnitus may be the consequence of a mild hearing impairment rather than the other way around, but is still only twice as common in hearing impairment to normal hearing. In any event, the threatening qualities of tinnitus are enhanced[4-8].

Finally, many tinnitus sufferers are angry about the treatment, or lack of treatment, or inappropriate advice that they have received. They may feel guilty for having submitted to treatment which they think is the cause of their tinnitus. Fear, anger and guilt are very powerful emotions which are intended to enhance survival-style, conditioned, reflex activity, and consequently greatly increase attention on the tinnitus. In our experience, tinnitus improves when the patient overcomes these feelings and stops dwelling on thoughts of injustice.

In some patients extreme fear of tinnitus results in a phobic state developing, very similar to that of the fear of spiders, frogs, small spaces, flying, etc. Many tinnitus sufferers also have these phobias, suggesting common mechanisms at work. In any phobic state, a slow process of 'desensitization' has to be used, confronting the feared object, and learning first to tolerate it, and then to accept it as a normal phenomenon that does not in any way threaten. Many aspects of tinnitus retraining are common to these techniques.

In many people the response to tinnitus is milder, though still negative in its meaning. Annoyance or unease exists, and although strong emotions may not be evoked, the limbic and autonomic systems are still being stimulated to produce aversive and intrusive emotions which reduce the quality of life. These properties of tinnitus, which make people seek help, are created outside the hearing mechanism, and therefore cannot be helped by a purely audiologic or ear-related approach.

The Causes of Tinnitus

Tinnitus mostly arises from damage to the inner ear, specifically the cochlea. Tinnitus can also result from 8th nerve lesion, injury to the brainstem, and rarely, to the brain itself[3]. There are some specific causes. Ear wax can rarely cause tinnitus. Other causes include middle ear infection or fluid, Meniere's disease, microvascular compression syndrome, and tumors of the 8th nerve. Pulsatile tinnitus can be caused by aneurysms, increased intracranial pressure (hydrocephalus), and hardening of the arteries. Vitamin B12 deficiency is common in tinnitus patients[9,10].

The etiology is at first an assumption based upon available information, the most common causes being noise exposure, presbyacusis, acoustic tumors, and vascular anomalies (Table 1).

The pathophysiology of hydrops had not been recognized until 1938, when Hallpike and Cairns identified dilatation of the saccule and scala media within the cochlea. The cause of Meniere's disease is unknown, however, many disease processes can lead to endolymphatic hydrops, yielding similar clinical findings: systemic infectious diseases (syphilis, mumps, measles), autoimmune mediated diseases, chronic otitis media, labyrinthine concussion, systemic illnesses such as leukemia or von Hipple-Lindau disease, congenital inner ear dysplasia, bone disease, labyrinthitis and temporal bone trauma. Since endolymphatic hydrops can be produced experimentally by blocking or obliterating the endolymphatic sac, abnormal absorption of endolymph is considered to be the etiologic factor. Unilateral tinnitus should always be evaluated thoroughly because of the possible presence of an acoustic tumor. Vestibular schwannoma is the most common CPA tumor, which usually presents with asymmetric hearing loss and unilateral, high-pitched tinnitus. There have been numerous reports of successful resolution of tinnitus concomitant with treatment of temporomandibular joint syndrome, which includes pain, crepitus, joint locking, tinnitus, aural fullness, vertigo, hyper- or hypoacusis, blurred vision, hoarseness, and orofacial dysesthesia. Myoclonic activity of the tensor and levator veli palatini muscles, salpingopharyngeus, and superior constrictor muscle can give rise to involuntary movement of the soft palate at a rate of 40 to 240 beats *per* minute. Although rare, it can lead to 'clicking' tinnitus, presumably due to forceful closure of the eustachian tube, associated with contractions. Although the precise etiology is unknown, experimental lesions of the inferior olive have produced myoclonic activity in monkeys. Palatal myoclonus has also been identified after viral encephalitis, malaria, syphilis, demyelinating diseases and vascular infarction. According to preliminary reports, the use of clostridium botulinum toxin injected into the soft palate produced complete resolution of tinnitus. Middle ear myoclonus has also been reported to cause pulsatile tinnitus due to the activity of tensor tympanic and stapedius muscles. Characteristically, tinnitus is described as rhythmic clicking or buzzing, which is usually unilateral. The sound produced could be caused by the propagation of the muscle contraction noise or vibration of the tympanic membrane during clonic activity. There are numerous vascular causes of pulsatile tinnitus, the most common of which are arteriovenous malformations (AVM) and fistulas (AVF). Carotid abnormalities (atherosclerotic dis-

Table 1. Classification of tinnitus causes

Objective tinnitus causes	Subjective tinnitus causes
Glomus tumor	Presbyacusis
Meningioma	Noise exposure
Adenoma	Noise trauma
Hemangioma	Whiplash injury
Acquired arterial alteration	Cochlear concussion
Arterial stenosis	Pharmacologic factors
Arterial tortuosity	Nontoxic drugs
Arterial dissection	Ototoxic drugs
Arterial aneurysm	Meniere's disease
Persistent stapedial artery	Acoustic neuroma
Aberrant internal carotid	Acute otitis media
Dural or cervical arteriovenous malformation	Serous otitis media
High jugular bulb	Temporal bone neoplasm
Jugular diverticulum	Chronic otitis media
Jugular bulb dehiscence	Neurologic abnormalities
Enlarged jugular vein	Dental factors
Dural arteriovenous fistula	Psychologic factors
Extracranial arteriovenous fistula	Metabolic function
Stenotic transverse sinus	
Benign intracranial hypertension	
Great vessel bruits	
High cardiac output	
Palatal myoclonus	
Temporomandibular joint dysfunction	
Tensor tympanic muscle spasm	

ease, fibromuscular dysplasia, and aneurysms) can also cause the sensation of pulsating tinnitus. Recently, benign intracranial hypertension has been increasingly reported as a major cause of pulsatile tinnitus in women, which can be identified by papilledema on funduscopic examination. Dural aretriovenous malformations account for 10 to 15 percent of intracranial AVMs and most commonly present with pulsatile tinnitus. The most frequently involved vessels are the transverse, cavernous and sigmoid sinuses, with communication to the occipital and great auricular arteries[11,12]. Dural AVFs comprise about 15 percent of all intracranial vascular malformations, and most commonly involve the transverse and sigmoid sinuses. Carotid artery pathology can also cause pulsatile tinnitus. The more common clinical conditions are atherosclerosis, abberance, aneurysm, and fibromuscular dysplasia. Tinnitus is probably related to turbulence within the vessel secondary to caliber change near the skull base. Turbulent blood flow through a normal internal jugular vein can also cause pulsatile tinnitus. This condition is often referred to as venous hum. Paragangliomas (glomus tumors, chemodectomas) are the most common vascular neoplasms of the skull base, frequently presenting with pulsatile tinnitus, with or without hearing loss. The pulsatile tinnitus in these patients may be caused by the formation of microvascular shunts within the tumor mass. In addition to glomus tumors, other skull base tumors are capable of causing pulsatile tinnitus. These include acoustic neuroma, meningioma, and hemangioma. In some series, benign intracranial hypertension was the most common cause of pulsatile tinnitus, often associated with headaches, visual disturbances, dizziness, and aural fullness[13-19]. Loud noise is the leading cause of damage to the inner ear. Advancing age may also be accompanied by inner ear damage and tinnitus. Many medications also can cause tinnitus (Table 2). Generally, this is thought to arise from their effect on the cochlea (ear)[20,21].

Thyroid dysfunction can be associated with tinnitus. Hyperthyroidism, by increasing cardiac output, can cause a pulsatile or rushing noise. Hypothyroidism has also caused this complaint. It is severe in about 4 percent of this population. Hyperlipidemia is being increasingly reported as a factor in tinnitus, particularly in association with

fluctuating sensorineural hearing loss and associated dizziness. Vitamin A or B deficiency has been described as causing tinnitus. Nine percent of patients who had disabling tinnitus reported it to be the result of major head trauma, often in conjunction with other factors such as hearing loss. The trauma generally included skull fracture or closed head injury. Seven percent of patients had tinnitus initiated by whiplash injury or cervical trauma, suggesting that abnormal proprioceptive input from nerve fibers in the neck and shoulders, or possibly brain stem injuries, may have been involved. Tinnitus after whiplash injury usually occurs 7 to 10 days of the accident, and the occurrence of tinnitus immediately after head trauma without clearly defined ear abnormalities or vestibular disease is uncommon[22].

The Diagnosis of Tinnitus

Clinically, tinnitus measurements are reasonable to perform only if they are useful. What is useful, depends on the individual needs specific to particular departments. The reasons for measuring tinnitus are as follows:

1) to provide reassurance to the patients that their tinnitus is real;
2) to be able to reproduce a similar sound to demonstrate to the family and significant others some of the characteristics of the tinnitus experienced by the patient;
3) to provide information that might assist in determining the site in the auditory system where the tinnitus originates;
4) to distinguish different subcategories of tinnitus;
5) to determine whether the tinnitus has changed;
6) to determine whether the treatment has had an effect;
7) to provide treatment guidelines;
8) to determine which patients are likely to benefit from some types of treatment; and
9) to assist in legal issues.

Table 2. Drugs that commonly cause or enhance tinnitus

DRUGS
Aspirin and other salicylates
Furosemide and other 'loop' diuretics
Aminoglycoside antibiotics
Quinine and related drugs
Nonsteroidal anti-inflammatory drugs
Heterocycline antidepressants
Chemotherapy

The evaluation of a patient with tinnitus begins, as does the evaluation of any symptom or complaint, with a thorough history. Attempts should be made to ascertain the age at onset, mode of progression, family history, and association with other audiovestibular symptoms. Additional history should include information regarding aural discharge, head trauma, noise exposure, and exposure to ototoxic drugs. A complete otoneurologic and head and neck examination should be performed. Many diagnostic modalities are available to study patients suffering from tinnitus (Table 3). Angiography will confirm the presence of vascular lesions. Tympanometry helps identify subtle changes in tympanic membrane stiffness due to respiratory influence through a patent eustachian tube or to myoclonus of the stapedial muscle, tensor tympanic muscle, or muscles of the palate. Electromyography will confirm myoclonus of the palatal muscles. Audiometric assessment plays a substantial role in the evaluation of patients suffering from nonvibratory tinnitus because of the poor correlation of the subjective complaint with actual acous-

Table 3. Evaluation of tinnitus

TESTING
Audiology
audiometry
speech discrimination score
speech reception threshold
brain stem response audiometry
impedance audiometry
electron nystagmography
Radiology
computed tomography scanning
magnetic resonance imaging
tomography of temporal bones
angiography
jugular venography
Hematology
MHA-TP (FTA-ABS)
thyroid functions
blood tests (ANA, B12, FTA, ESR, SMA-24, HBA-IC, fasting glucose, TSH, antimicrosomal antibodies)
blood count (CBC)
Allergy
allergic evaluation
Miscellaneous
electromyography

tic properties. This assessment should begin with pure-tone audiometry. Attempts at matching the frequency of tinnitus to the identified hearing loss may produce a reasonable approximation by a narrow-band noise in the same frequency range. Speech discrimination should be tested in and out of noise. Tone decay test should be performed as well as alternate binaural loudness balance (ABLB), tympanometry (stapedial reflex, reflex latency and reflex decay, tympanic membrane compliance), short increment sensitivity index (SISI), and brain stem response audiometry (BSR). Further investigation into the known causes of tinnitus should be dictated by the index of suspicion aroused by the history, physical examination, and audiometric profile. Electron nystagmography will give an insight into the status of the vestibular system, x-ray of the petrous apex may be helpful in diagnosing cerebellopontine angle lesions, and computed tomography or magnetic resonance imaging will help demonstrate most intracranial lesions[23]. Laboratory data should include serologic tests for syphilis, complete blood count, serum thyroxine level, and glucose tolerance test. Based on these tests, tinnitus can be classified into categories of cochlear, retrocochlear, central, and tinnitus of unknown cause[24-27].

The Treatment of Tinnitus

If a specific cause of tinnitus is found, the noise can be eliminated. Examples of specific causes include medication, tumors, infections, Meniere's disease, TMJ and otosclerosis. Identification of a specific cause may require quite extensive workup including x-ray, balance testing and blood tests. However, even after extensive workup most causes of tinnitus remain undiagnosed[28,29]. If a specific cause of tinnitus is not found, it is unlikely that tinnitus will be able to eliminate. At best, one might get partial relief from some of the strategies to be described in the next few paragraphs. However, even though no treatment may be available, tinnitus should be examined since it may be a warning sign of a serious disorder such as tumor of the 8th nerve, or some other disorder which may imply hearing impairment[30]. Medicines may occasionally help lessen the noise even though no cause has been identified[31]. Medications used in the treatment of tinnitus are:

- benzdiazepines
- tricyclics
- Tegretol
- verapamil
- ginkgo
- betahistine
- B12

Tinnitus Retraining Therapy (TRT)

Successful tinnitus management is the result of retraining and relearning. Once the tinnitus loses its sinister meaning, however loud it has been or unpleasant it may seem, it does begin to diminish, and in many cases may not be heard for long periods of time. In most cases firmly held beliefs are hard to change.

Retraining the subconscious auditory system to accept tinnitus as something that occurs naturally, does not spell a lifetime of torture and despair, and is not a threat or a warning signal, can take months and sometimes even years. Such retraining should be guided by professionals with experience in this field forming part of a multidisciplinary team. For people who also have coexisting or pre-existing anxiety or depression it can take longer to change their feelings about their tinnitus.

When we talk about retraining, this is not simply an abstract learning exercise. In the subconscious part of the brain concerned with learning, beyond the inner ear but before the act of conscious perception of sound takes place, the subconscious filter networks of nerve cells (neuronal networks) are programmed to pick up signals on a 'need to hear' basis.

Think again of the way we invariably detect the sound of our own name, or a distant car horn, or a new baby stirring in sleep, whereas we may be unaware of the sound of rain pounding on the roof or surf beating on a sea shore. Retraining therapy involves reprogramming or resetting these networks, which are selectively picking up the 'sound life' in the auditory system. Although these are 'nature sounds', they become a problem because they have been identified as a threat, either to life or to life quality[32-34].

Tinnitus retraining first involves learning about what is actually causing the tinnitus. This begins with proper examination by an ear specialist, followed by full explanation of what is going on in the ear and the brain to produce the tinnitus sound. However, specialists who themselves believe that tinnitus is an 'ear' phenomenon cannot help. We are in a difficult situation where the classical training of tinnitus being due to inner ear damage is still very dominant, rather than an understanding based on the neurophysiological model[35,36]. We need to learn that the sounds of tinnitus, which we may interpret as distressing, affecting life quality, and seemingly unending, are in reality the sounds of nature, coming from weak electrical signals in the auditory pathways that have always been there. With appropriate directive counseling (or 'retraining') from professionals, we can change even strongly held views that

tinnitus is a threatening and unpleasant experience which cannot be altered.

The presence of any continuous stimulus usually results in a process called habituation, whereby the individual responds less and less to the stimulus as long as it does not have any special negative meaning. The final stage in this process is when the signal is no longer detected, and cortical neurons are unresponsive. With tinnitus this means that it is no longer heard, even if it is listened for[37-39].

Retraining therapy can achieve this. As the process takes a long time (often two years or so), during the initial stages tinnitus becomes gradually less unpleasant (but may still be perceived as a loud sound). This process is called habituation of reaction. Tinnitus then becomes quieter for longer periods of time, and eventually disappears or becomes a natural part of the background 'sound of silence' (habituation of perception). This cannot happen, however, while tinnitus is still classified as a threat, or negative experience that demands further monitoring[30,40-42].

One way in which the ear itself does contribute to tinnitus is if there is a hearing loss. This may be quite slight, or just in the high frequencies. Any tendency to 'straining to hear' can increase amplification of sound signals in the subconscious part of the brain, and increase the ease by which tinnitus signals can be picked up. This is why it is important to correct any significant hearing loss with appropriate hearing aids, as part of overall tinnitus management. However, inept and inappropriate hearing aid fitting by those unpracticed in tinnitus management can make it worse, a frequent finding in ENT departments.

In some cases, changes in the inner ear function may be important in triggering tinnitus emergence (e.g., Meniere's disease or acute acoustic trauma), however, the retraining approach works independently of the triggering factor.

It is important to distinguish between the role of the ear in the emergence of tinnitus (e.g., disco tinnitus) and the role of central processing in the brain, outside the auditory system, in determining the persistence of tinnitus and our emotional response to it. Despite the importance of hearing loss, a recent study in tinnitus patients has shown that there was no significant difference in hearing between the tinnitus group and normal population statistics[43].

Wide Band Noise Generators

Wide band noise generators (WNGs; previously called 'maskers') have a different role to play. Tinnitus masking was at one time thought to be useful in that it simply made tinnitus inaudible. In fact, this proved to be counterproductive, as tinnitus, the object of an exercise in habituation, must be audible for habituation to occur. Habituation to any signal cannot occur in the absence of its perception. Imagine trying to habituate your response to spiders, which you hate, simply by avoiding them.

Much better longterm results can be obtained if wide band noise is used at low intensities while the tinnitus can be heard at the same time. Wide band noise contains all frequencies, and therefore very gently stimulates all nerve cells in these subconscious networks, allowing them to be more easily programmed or reset, so that tinnitus signals are no longer detected. It also reduces the contrast between tinnitus and otherwise total silence. WNGs on their own may give temporary reduction in the distress from tinnitus, but will not achieve longterm habituation without other essential elements of TRT, i.e. teaching – demystification – re-evaluation – densitization.

The emergence of tinnitus is often dependent on silence. Tinnitus is mostly first heard at night in a well sound-proof bedroom, or a quiet living room[44]. Tinnitus persistence depends on the meaning attached to it, but also on the contrast it creates with the auditory environment. Contrast contributes greatly to the intensity of any perception. Thus, a small candle in the corner of a large darkened room seems dazzlingly bright until flood lights are switched on, making it virtually invisible. Tinnitus patients should avoid extreme silence, and retraining programs will often use white noise therapy as a means to reduce this contrast. In all cases, sound enrichment should be practiced. Make sure there is always a pleasant, nonintrusive background sound (like a large slow fan or an open window). Choosing what is right for you may take some time. Avoid masking tinnitus but have some sound present during day and night[45,46].

Many tinnitus patients have hyperacusis (sensitivity to external noise) and for this reason often seek very quiet environments. In all cases, sound enrichment should be practiced, using unobtrusive sound sources (e.g., a domestic fan, an open window) to break the silence.

Although using wide band noise on its own can give some temporary relief from tinnitus discomfort, there is very little chance of permanent relief unless it is combined with the retraining approach. Information and teaching and demystification about tinnitus and tinnitus mechanisms are the most vital parts of therapy. Otherwise, where strong beliefs about the threatening nature of tinnitus are maintained, the survival-style conditioned response mecha-

nisms in the subconscious brain ensure that it is continuously monitored.

At present, TRT is available in relatively few centers, but the techniques are being spread, and gradually learned and used in an increasing number of otology and audiology departments around the world[47,48].

Conclusion

In summary, what to do about tinnitus?

It is recommended that persons with tinnitus limit salt (no added salt) and refrain from drinking caffeinated beverages, other stimulants (like tea), and chocolate. Salt restriction is intended for those who might have a subclinical form of Meniere's disease. Caffeine and similar substances increase tinnitus in a nonspecific fashion. Otherwise, the diet should be balanced and have normal amounts of fruit and veggies. Hearing aids and other devices known as maskers may also help alleviate tinnitus[49]. If the patients have tinnitus associated with hearing loss, a hearing aid is the first thing to try. Maskers are based on the idea that tinnitus is usually worst when things are very quiet. Listening to the interstation static on the FM radio, tapes of ocean surf, and the like may be helpful. Tinnitus maskers are fitted by audiologists. However, controlled studies of maskers have failed to demonstrate efficacy. Sometimes, anxiety or depression which accompanies tinnitus may be as big a problem as the tinnitus itself. In this instance, consultation with a psychologist or psychiatrist expert in this field may be helful. Hypnosis may be effective and increase tolerance to tinnitus[50-52]. Only rarely is surgical treatment indicated, and even more rarely is tinnitus thus relieved. One should certainly consider surgery if the tinnitus is due to a tumor. Surgery may be an option to consider if the diagnosis is otosclerosis, fistula or Meniere's disease[53]. Tinnitus is a somewhat terrifying chronic symptom that can be disabling to the patient. It is important to rule out significant medical etiologies, as outlined above. For many patients, reassurance that there are no underlying tumors or impending medical emergencies can reduce their anxiety and symptoms. A thorough medical evaluation must accompany the management of tinnitus, as underlying medical etiologies of tinnitus may go undiagnosed for years with increased morbidity.

References

1. LOCKWOOD AH, SALVI RJ, COAD BA, TOWSLEY ML, WACK DS, MURPHY BW. The functional neuroanatomy of tinnitus. Evidence for limbic system links and neural plasticity. Neurology 1998;50:114-20.
2. STOUFFER A, TYLER RS. Characterization of tinnitus by tinnitus patients. J Speech Hear Disord 1990;55:439-53.
3. LANSKA DJ, LANSKA MJ, MENDEZ MF. Brainstem auditory hallucinosis. Neurology 1987;37:1685.
4. GOLDSTEIN B, SHULMAN A. Tinnitus-hyperacusis and the loudness discomfort level test: a preliminary report. Int Tinnitus J 1996;2:83-9.
5. CHAN SWY, READ PC. Tinnitus and temporomandibular pain-dysfunction disorder. Clin Otolaryngol 1994;19:370-80.
6. PENNER MJ, SARAN A. Simultaneous measurement of tinnitus pitch and loudness. Ear Hear 1994;15:416-21.
7. PENNER MJ. Tinnitus as a source of internal noise. J Speech Hear Res 1986;29:400-6.
8. CHANDLER JR. Diagnosis and cure of venous hum tinnitus. Laryngoscope 1983;93:892-5.
9. ATTIAS J, FURMAN V, SHEMESH Z, BRESLOFF I. Impaired brain processing in noise-induced tinnitus patients as measured by auditory and visual event-related potentials. Ear Hear 1996;17:327-33.
10. HARROP-GRIFFITH J, KATON W, DOBIE R, SAKAI C, RUSSO J. Chronic tinnitus: association with psychiatric diagnosis. J Psychosom Res 1987;31:613-21.
11. YAMASOBA T, KIKUCHI S, HIGO R. Deafness associated with vertebrobasilar insufficiency. J Neurol Sci 2001;187:69-75.
12. MATSUSHIMA JI, SAKAI N, UEMI N, IFUKUBE T. Effects of greater occipital nerve block on tinnitus and dizziness. Int Tinnitus J 1999;5:40-6.
13. SAVASTANO M. A protocol of study for tinnitus in childhood. Int J Pediatr Otorhinolaryngol 2002;64:23-7.
14. MRENA R, SAVOLAINEN S, KUOKKANEN JT, YLIKOSKI J. Characteristics of tinnitus induced by acute acoustic trauma: a long-term follow-up. Audiol Neurootol 2002;7:122-30.
15. KHODAEI I, ROWLEY H, FARRELL M, COLLINS CM, VIANNI L, RAWLUK D. An unusual cause for tinnitus. Ir Med J 2001;94:312-3.
16. JACOBSON GP, McCASLIN DL. A search for evidence of a direct relationship between tinnitus and suicide. J Am Acad Audiol 2001;12:493-6.
17. YONEOKA Y, FUJII Y, NAKADA T. Central tinnitus: a case report. Ear Nose Throat J 2001;80:864-6.
18. BROOKLER KH. Unilateral tinnitus occurring with a peripheral vestibular disorder in the contralateral ear. Ear Nose Throat J 2001;80:860-1.
19. PULEC JL. The enigma of tinnitus. Ear Nose Throat J 2001;80:851.
20. TYLER RS, BABIN RW. Tinnitus. In: CUMMINGS CW, ed. Otolaryngology – head and neck surgery. New York: Mosby, 1993:3031-53.

21. SISMANIS A, SMOKER WRK. Pulsatile tinnitus: recent advances in diagnosis. Laryngoscope 1994;104:681-7.
22. FAINGOLD CL. Role of GABA abnormalities in the inferior colliculus pathophysiology – audiogenic seizures. Hear Res 2002;168:223-37.
23. MIRZ F, GJEDDE A, SODKILDE-JORGENSEN H, PEDERSEN CB. Functional brain imaging of tinnitus-like perception induced by aversive auditory stimuli. Neuroreport 2000;11:633-7.
24. WILSON PH, HENRY JL. Psychological approaches in the management of tinnitus. Aust J Otolaryngol 1993;1:296-302.
25. KAHARIT K, ZACHAU G, EKLOF M, SANDSJO L, MOLLER C. Assessment of hearing and hearing disorders in rock/jazz musicians. Int J Audiol 2003;42:279-88.
26. BAGULEY DM, ANDERSSON G. Factor analysis of the Tinnitus Handicap Inventory. Am J Audiol 2003;12:31-4.
27. SISMANIS A. Pulsatile tinnitus. Otolaryngol Clin North Am 2003;36:389-402.
28. SHEMESH Z, ATTIAS J, ORNAN M, SHAPIRA N, SHAHAR A. Vitamin B12 deficiency in patients with chronic tinnitus and noise-induced hearing loss. Am J Otolaryngol 1993;14:94-9.
29. HENRY JL, WILSON PH. The psychological management of tinnitus: comparison of a combined cognitive educational program, education alone and a waiting-list control. Int Tinnitus J 1996;2:9-20.
30. HOUSE HW. Treatment of severe tinnitus with biofeedback training. Laryngoscope 1978;88:406-12.
31. MORGENSTERN C, BIERMANN E. The efficacy of ginkgo special extract EGb 761 in patients with tinnitus. Int J Clin Pharmacol Ther 2002;40:188-97.
32. HAZELL JWP. Models of tinnitus: generation, perception – clinical implications. In: MOELLER VJ, ed. Tinnitus mechanisms. Boston, DC: Allyn & Bacon, Boston Chapter, 1995:7:57-72.
33. HAZELL JWP. Tinnitus as the manifestation of a survival-style 5 reflex – an anthropological approach. Proceedings of the Vth International Tinnitus Seminar, Portland, OR, USA, 1995:579-82.
34. HAZELL JWP. Support for a neurophysiological model of tinnitus: research data and clinical experience. Proceedings of the Vth International Tinnitus Seminar, Portland, OR, USA, 1995:51-7.
35. JASTREBOFF PJ. Phantom auditory perception (tinnitus): mechanisms of generation and perception. Neurosci Res 1990;8:221-54.
36. JASTREBOFF PJ, HAZELL JWP. A neurophysiological approach to tinnitus: clinical implications. Br J Audiol 1993;27:1-11.
37. TERRY AMP, JONES DM, DAVIS BR, SLATER R. Parametric studies of tinnitus masking and residual inhibition. Br J Audiol 1983;17:245-56.
38. VERNON J, GRIEST S, PRESS L. Attributes of tinnitus and the acceptance of masking. Am J Otolaryngol 1990;11:44-50.
39. ZWISLOCKI JJ. Central masking and neural activity in the cochlear nucleus. Audiology 1971;10:48-59.
40. JOHNSON R, GRIEST S, PRESS L, STORTER K, LENTZ B. A tinnitus masking program: efficacy and safety. Hear J 1989;42:18-25.
41. SEIDMAN MD, BABU S. Alternative medications and other treatments for tinnitus: facts from fiction. Otolaryngol Clin North Am 2003;36:359-81.
42. STEENERSON RL, CRONIN GW. Tinnitus reduction using transcutaneous electrical stimulation. Otolaryngol Clin North Am 2003;36:337-44.
43. SHELDRAKE JB, JASTREBOFF PJ, HAZELL JWP. Perspectives for the total elimination of tinnitus perception. Proceedings of the Vth International Tinnitus Seminar, Portland, OR, USA, 1995:531-7.
44. HELLER MF, BERGMAN M. Tinnitus in normally hearing persons. Ann Otol 1953;62:73-83.
45. RUBINSTEIN JT, TYLER RS, JOHNSON A, BROWN CJ. Electrical suppression of tinnitus with high-rate pulse trains. Otol Neurootol 2003;24:478-85.
46. MIANI C, PASSON P, BRACALE AM, BAROTTI A, PANZOLLI N. Treatment of hyperacusis in Williams syndrome with bilateral conductive hearing loss. Eur Arch Otorhinolaryngol 2001;258:341-4.
47. TYLER RS. Tinnitus in the profoundly hearing-impaired and the effects of cochlear implants. Ann Otol Rhinol Laryngol 1995;104:25-30.
48. SALLUSTIO V, PORTALINA P, SOLEO L, CASSANO F, PESOLA G, LASORSA G, QUARANTA N, SALONNA I. Auditory dysfunction in occupational noise exposed workers. Scand Audiol Suppl 1998;27:95-110.
49. BAGULEY DM, McFERRAN DJ. Current perspectives on tinnitus. Arch Dis Child 2002;86:141-3.
50. ATTIAS J, SHEMESH Z, SHOHAM C, SHAHAR A, SOHMER H. Efficacy of self-hypnosis for tinnitus relief. Scand Audiol 1990;19:245-54.
51. ATTIAS J, SHEMESH Z, SOHMER H, GOLD S, SHOAM C, FARAGI D. A comparison between self-hypnosis and attentiveness for alleviation of chronic tinnitus. Audiology 1993;32:205-12.
52. ATTIAS J, PRATT H, RESHEF I, BRESLOFF I, HOROWITZ G, POLYAKOV A, SHEMESH Z. Detailed analysis of auditory brainstem responses in patients with noise-induced tinnitus. Audiology 1996;35:259-70.
53. FAHY C, NIKOLOPOULOS TP, O'DONOGHUE GM. Acoustic neuroma surgery and tinnitus. Eur Arch Otorhinolaryngol 2002;259:299-301.

Sažetak

ŠUM U UHU – SADAŠNJE STANJE I TERAPIJA PRIVIKAVANJEM

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Šum u uhu je pojava nenormalne buke u uhu. Otprilike 6% populacije pati od tzv. jakog šuma u uhu. Šum se može pojaviti i nestati, ali može biti i trajan. Može zvučati poput duboke tutnjave ili zvonjave visokih tonova. Može nastati u oba uha ili samo u jednom. Uzroci šuma mogu biti različiti, npr. oštećenje unutarnjeg uha, ozljeda osmog moždanog živca ili moždanog debla, ili pak rjeđe ozljeda mozga. Ekstrakranijski uzroci šuma također su brojni. Nakon postavljanja dijagnoze osjet buke u uhu može se ublažiti upotrebom lijekova, iako uzrok šuma još nije utvrđen. U suvremenom načinu liječenja šuma, tzv. liječenju metodom privikavanja (*tinnitus retraining therapy*), najprije treba utvrditi što je zapravo prouzročilo nastanak šuma. Taj se proces zove 'privikavanje na nastalu situaciju'. Šum se tako može ublažiti na dulje vrijeme, a na kraju može i sasvim nestati ili se stopiti sa zvučnom pozadinom (habituacija percepcije). Promjene u unutarnjem uhu u nekim slučajevima mogu potaknuti naglu pojavu šuma u uhu (npr. Meniereova bolest ili akutna akustička trauma), ali valja naglasiti da liječenje metodom privikavanja daje dobre rezultate bez obzira na to što je u osnovi nastanka šuma. Unatoč važnosti gubitka sluha najnovija ispitivanja u bolesnika sa šumom pokazuju da nema značajnih razlika u sluhu između bolesnika sa šumom i skupine zdravih ispitanika.

Ključne riječi: *Tinitus, etiologija; Tinitus, dijagnostika; Tinitus, terapija*