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Review

A neurophysiological approach to tinnitus: clinical implications

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Abstract

This paper presents a neurophysiological approach to tinnitus and discusses its clinical implications. A hypothesis of discordant damage of inner and outer hair cells systems in tinnitus generation is outlined. A recent animal model has facilitated the investigation of the mechanisms of tinnitus and has been further refined to allow for the measurement of tinnitus pitch and loudness.

The analysis of the processes involved in tinnitus detection postulates the involvement of an abnormal increase of gain within the auditory system. Moreover, it provides a basis for treating patients with hyperacusis, which we are considering to be a pre-tinnitus state. Analysis of the process of tinnitus perception allows fur the possibility of facilitating the process of tinnitus habituation for the purpose of its alleviation.

The combining of theoretical analysis with clinical findings has resulted in the creation of a multidisciplinary Tinnitus Centre. The foundation of the Centre focuses on two goals: the clinical goal is to remove tinnitus perception from the patient's consciousness, while directing research toward finding a mechanism-based method for the suppression of tinnitus generators and processes responsible for enhancement of tinnitus-related neuronal activity.

Neurophysiological approach

Introduction

Tinnitus presents an enormous challenge for clinicians and researchers. It is an extremely prevalent disorder, seriously affecting about 10 million people in the USA, with the total number of tinnitus sufferers estimated to be in the range of 40 million in USA (McFadden, 1982) with a similar ratio in the UK (Coles, 1987). For about one million of the afflicted, tinnitus presents a debilitating problem, excluding them from normal life and, in some cases, causing such suffering that they consider committing suicide. About 85% of the patients seeking help from otologists have tinnitus (McFadden, 1982). Importantly, if nothing is done to help tinnitus patients, these numbers will only increase since tinnitus affects about one third of the population over 65 years of age (Sataloff *et al.*, 1987) and in industrial countries, life span increases continuously.

Tinnitus management is practised variably, and often separately, by physicians, audiologists, hearing therapists, psychologists and other health care professionals. Long-term results are generally poor and many patients continue to suffer from chronic tinnitus which significantly interferes with many aspects of their life. The common approach from most professionals is to send the patient home with the advice that 'nothing can be done and you will have to learn to live with it'.

Neurophysiological analysis of the tinnitus phenomenon and experimental work using our animal model of tinnitus (Jastreboff *et al.*, 1988; Jastreboff *et al.*, 1991; Brennan and Jastreboff, 1991; Jastreboff, 1992; Jastreboff and Brennan, 1992a; Jastreboff and Brennan, 1992b), however, has provided a new approach to tinnitus and its treatment. A behavioural paradigm allows us to measure the presence of tinnitus, estimate its pitch and loudness (Brennan and Jastreboff, 1991; Jastreboff and Brennan, 1992a), and then use electrophysiological, biochemical and immunocytochemical techniques to reveal abnormalities responsible for tinnitus perception and through this to seek new methods of its alleviation. The process of identifying effective treatment strategies is facilitated by combining basic science research and clinical approaches. For example, hypotheses about tinnitus mechanisms can be validated by testing them on our clinic patients, and simultaneously models of tinnitus based on our clinical experience can be subjected to rigorous analysis in animal models.

In this paper, we are describing tinnitus from the point of view of general neurophysiology, indicating the clinical implications of theoretical divagations, and their correspondence to present clinical findings. The concept of our Tinnitus Centres, their structures, and the process of diagnosis and treatment of patients is explained using neurophysiological knowledge as a frame of reference.

Development of the Tinnitus Centre concept

In Baltimore, the combination of theoretical analysis and basic research findings with clinical data from

London, accumulated since 1976, has resulted in the creation of the multidisciplinary Tinnitus Centre. The foundation of the Centre is based on two goals: (i) a clinical goal to implement the most effective methods to help tinnitus patients; and (ii) a basic research goal to find a mechanism-based method of suppression of tinnitus generators.

We started by trying to answer the question, 'Why is tinnitus so difficult to deal with'? There are a number of reasons. First of all, in the vast majority of cases tinnitus is a phantom auditory perception, perceived exclusively by the patient. In only about 4% of all eases may there be an objective correlate to spontaneous otoacoustic emissions (Penner, 1990), and the frequency of other somatosounds is similar (McFadden, 1982).

Phantom perception cannot be measured objectively, as yet. All work, until recent years, has been based on verbal interviews with patients or by performing a psychoacoustical estimation of tinnitus parameters. Evaluations of treatment based on these data are generally unhelpful, and contaminated by placebo effects, often in the range of 40%. Therefore, all data so obtained should be assessed with great care before it becomes a basis fur accepted clinical practice.

The optimal formula would be an objective method for tinnitus evaluation in humans. Some attempts have been made (Jastreboff *et al.,* 1982a; Jastreboff *et al.,* 1990; Schreiner *et al.,* 1990**),** but none has been validated. Furthermore, human research is restricted for ethical reasons and significant progress in the work on the mechanisms of tinnitus and new methods for its alleviation has been restrained because of the lack of a valid animal model of tinnitus. Only recently such a model has been proposed and validated (Jastreboff *et al.,* 1987; Jastreboff *et al.,* 1988; Jastreboff *et al.,* 1986), opening new dimensions for the work on tinnitus.

The second difficulty is that tinnitus is a symptom and not a disease. There is an enormous variety of dysfunctions within the peripheral or central parts of the auditory system which might result in tinnitus. Moreover, many of these abnormalities are not easily identified by any effect on hearing. Therefore, each case has to be evaluated individually; we cannot expect to find one treatment which will be effective in the majority of cases.

The third problem results from a lack of a proven mechanism(s) of tinnitus. There are many different hypotheses (Kiang *et al.,* 1970; Salvi and Ahroon, 1983; Hazell, 1987; Moller, 1984; Moller 1987; Eggermont, 1990; Tonndorf, 1981; Tonndorf, 1987; Jastreboff, 1990) but none has been proven yet, making it impossible to work a mechanism-based approach to treatment.

The fourth problem is created by the extremely strong imprinting of the tinnitus sound pattern in the patients' central nervous system. The persistence of even psychoacoustically weak tinnitus perception and its frequent resistance to masking are corollaries of this postulate. One important clinical prediction is that even if a tinnitus generator is suppressed to a large extent, the perception of tinnitus is affected to a much smaller degree.

Finally, clinical data clearly shows that in patients with significant tinnitus, the perception of tinnitus has powerful connections with the emotional system (Hallam *et al.,* 1984), which are difficult to modify. Patients exhibit strong emotional reactions to the sound, which when evaluated psychoacoustically appear insignificant, but nevertheless are capable of being perceived as intrusive sounds, causing considerable distress (Meikle *et al.,* 1984).

Neurophysiological basis for tinnitus

A theoretical analysis of tinnitus from the point of view of neuroscience has already been published (Jastreboff, 1990) together with some of the clinical implications. In this paper, only the main points are presented, stressing their clinical importance.

The first result of this analysis yields the hypothesis that even tinnitus of the cochlear type must involve abnormalities of both cochlear function and the processing of tinnitus-related signals within the nervous system (Jastreboff, 1990). In each ease we may have to account for several peripheral as well as central components, which contribute, each to a different extent, to the final product - tinnitus perception and evaluation by the patient. As such, tinnitus is a disorder of perception and should not be thought of as a perception of signals originating from a single, localised generator. We therefore reject the simplified classification of tinnitus based on its generator or supposed aetiology. Our hypothesis reflects the well-established concept of parallel processing of information within the nervous system by interconnected neuronal networks (Goldman-Rakic, 1988; Jastreboff, 1990). Although this hypothesis sounds esoteric, it has profound clinical implications.

The next conclusion is that the differentiation of the process by which tinnitus emerges, can be divided into three stages; (i) generation; (ii) detection, and (iii) perception and evaluation. Generation occurs usually at the periphery (although it may be central) and in the majority of cases can be associated with disorders occurring in the cochlea or the cochlear nerve. The process of detection occurs at the level of the subcortical centres and is based on pattern recognition. Finally, perception and evaluation of tinnitus-related activity occurs in the auditory cortex, with considerable and significant participation of the limbic system, the prefrontal cortex and several other cortical areas (Jastreboff, 1990).

The generation of an abnormal signal, which

will in a cascade of events yield the tinnitus perception, can depend on a number of different mechanisms and their interaction, a detailed description of which has already been published (Jastreboff, 1990). Briefly, some main categories can be listed (Kiang *et al.*, 1970; Salvi and Ahroon, 1983; Hazell, 1987; Moller, 1984; Moller, 1987; Eggermont, 1990; Tonndorf, 1981; Tonndorf, 1987; Jastreboff, 1990): (1) discordant damage of outer (OHC) and inner (IHC) hair cell systems; (2) crosstalk between the VIII nerve fibres; (3) ionic imbalance in the cochlea; (4) dysfunction of cochlear neurotransmitter systems; (5) heterogeneous activation of the efferent system; (6) heterogenous activation of Type I and II cochlear afferents.

The hypothesis of discordant damage to OHC and IHC system might be of particular interest since it provides a useful link in combining a number of seemingly different proposals about tinnitus generation; for example, the edge-effects (Kiang *et al.*, 1970; Salvi and Ahroon, 1983; Penner, 1980), efferent system activation, salicylate-induced tinnitus (Stypulkowski, 1990; Jastreboff, 1992; Jastreboff', 1990), and proposals in which analogies are drawn between tinnitus and chronic pain (Tonndorf 1987). The discordant damage hypothesis postulates that tinnitus is generated on the portion of the basilar membrane where there is preservation of IHC but damaged or temporarily dysfunctional OHC, a common pathological finding (Bohne and Clark, 1982; Bohne *et al.*, 1987; Liberman, 1987; Liberman and Dodds, 1987; Liberman and Mulroy, 1982; Liberman and Kiang, 1978).

One of the predictions of this theory is that the prevalence of perceived pitch of tinnitus should be localized close to the slope on the audiogram in cases of patients with hearing loss. Such a situation is frequently observed in clinical practice (Hazell, 1987; Hazell *et al*, 1985). Furthermore, this hypothesis explains the occurrence of tinnitus in patients without hearing loss, as diffuse damage of up to 30% of OHC can occur without any associated detectable hearing loss (Bohne arid Clark, 1982). However, a patient with localized damage of OHC, but intact IHC, will not exhibit a hearing loss in standard audiological evaluation, but due to the discordant damage of OHC and IHC, the patient may have tinnitus with a pitch close to the characteristic frequency of the affected part of the cochlea. Furthermore, it might be possible to identify these areas of limited damage by means of otoacoustic emission (OAE) distortion products which are thought to relate to OHC activity (Brown e*t al.*, 1989; Harris *et al.*, 1989; Wier *et al.*, 1988). Preliminary data from our patients indicates that tinnitus pitch may well be associated with abnormal OAE distortion products even before any abnormality is noted on pure tone audiometry.

The majority of approaches in the past have been aided by the concept of attenuating a tinnitus generator by the sole process of manipulating it with pharmacological, surgical or prosthetic means (Shulman, 1992; Dobie *et al*, 1992; Podoshin *et al.*, 1991; Hulshof and Vermeij, 1987). These attempts were seldom effective and none have provided prolonged and consistent benefit in a significant proportion of cases.

Generation and detection

Our experimental work, utilizing an animal model of tinnitus, points out the importance of calcium homeostasis in tinnitus generation, as well as the possibility of using certain calcium channel blockers in tinnitus alleviation (Jastreboff and Brennan, 1988; Bobbin *et al.*, 1990; Jastreboff *et al.*, 1992b; Jastrebuff *et al*, 1991). This work, once properly adapted for human application, might yield one specific method of dealing with cochlear generators of tinnitus.

The next stage in the emergence of tinnitus is the detection of tinnitus-related signals from the background of neuronal spontaneous activity. Neuroscience principles point out that this process is based on a pattern recognition principle and occurs at subcortical levels (Carpenter and Grossberg, 1987; Grossberg, 1987). The ability of the central auditory system to extract signals on a basis of preset criteria allows for the detection of, and orientation towards, important sounds (even though they be of low intensity) while at the same time ignoring other environmental sounds (which may be quite loud). The understanding of this mechanism helps in the explanation of several otherwise puzzling features of tinnitus: its usually rapid emergence, persistence in spite of low levels of perceived loudness, and the phenomenon of residual inhibition (Jastreboff, 1990).

From the clinical viewpoint, other features of the detection process are of significance, particularly neural-network based decoding of information (Carpenter and Grossberg, 1987; Grossberg, 1987) and the plasticity of the nervous system. The concept of decoding information by neuronal networks dictates that once a certain pattern has been classified, even if the initial stimulus is significantly reduced, the detection of this signal is affected to much smaller extent. An everyday experience of this feature is our ability to detect and interpret the soft sounds of our native language, even when immersed in environmental noise, while clear, louder sounds of a foreign language are difficult to classify and are frequently simply ignored.

In the case of tinnitus, once the abnormal pattern of neural activity is detected and classified, it can be very persistent. Basic neuroscience predicts that it should be possible to reverse this pattern recognition process by exposing the patient to low levels of white noise for long periods of time. White noise should interfere with the pattern recognition process making it more difficult to separate the tinnitus signal from background neuronal activity, and if there are no other processes enhancing recognition of this signal, then the

pattern could become no longer detected.

Although theoretically possible, this process is difficult to accomplish. Surprisingly, however, it does seem to occur in a gradually increasing proportion of tinnitus patients after several years of treatment (Sheldrake and Hazell, 1992). These patients, after years of using so-called 'tinnitus maskers' become unable to perceive their tinnitus any more. There is still the question of whether these patients are significantly different from a few untreated patients who experience spontaneous full recovery and more data are needed to clarify this point. Whereas this observation is interesting for a better understanding of the mechanisms of tinnitus, it has limited impact on clinical practice due to the time required for the symptomatic improvement.

Tinnitus and Hyperacusis

Plasticity in the auditory system has been clearly demonstrated (Sasaki *et al.,* 1980**;** Gerken *et al.,* 1986; Gerken *ci al.,* 1985; Gerken *et al.,* 1984; Gerken, 1979; Salvi *et al.,* 1992; Salvi *et al.,* 1990). Importantly, even a temporal decrease in auditory input results in an increase of sensitivity of neurons within the subcortical centres (Gerken *et al.,* 1986; Gerken *et al.,* 1985; Gerken *et al.,* 1984; Gerken, 1979; Salvi *et al.,* 1992; Salvi *et al.,* 1990). These observations provide an explanation to the well-recognized phenomenon that if a person with normal hearing is deprived of auditory input in an anechoic chamber, the person will experience increased hearing sensitivity, start to hear extremely weak sounds, and frequently experience tinnitus. The implication of these findings and the postulate of the involvement of subcortical processing in tinnitus is that in certain cases tinnitus might be due to such an increased gain within the subcortical centres of the auditory system. As such it can be treated by a method aimed at decreasing this gain; that is, hearing aids or white noise generators incorporated into a protocol which employs a very gradual increase of such auditory input over prolonged periods of time. Data from the field of neuroscience stresses that reversal of these plastic changes requires prolonged periods of time, measured in weeks or months. Clinical data fully support this approach (Sheldrake and Hazell, 1992).

Another ramification of this hypothesis is that hyperacusis can be regarded as a pretinnitus state. Hyperacusis is a manifestation of increased central gain (Coles and Sood, 1988; Hazell and Sheldrake, 1992; Jastreboff, 1990). The frequent occurrence of hyperacusis with tinnitus, often before tinnitus onset, further implies its relation with tinnitus. This opens the possibility of tinnitus prevention in cases of hyperacusis without tinnitus. We have postulated that hyperacusis both with and without significant tinnitus can be effectively treated by the gradual introduction of white noise and should be treated similarly to tinnitus resulting from abnormal gain increase (Jastreboff, 1990). The effectiveness of this approach has now been documented (Hazell and Sheldrake, 1992), and we are utilizing it in our tinnitus clinics in Baltimore and London. Importantly, this hypothesis offers the possibility of early detection of a susceptibility to tinnitus and early prevention of it.

Association and evaluation

The final stage of tinnitus emergence is its perception and evaluation, with the involvement of several cortical areas and the limbic system. The pattern representing tinnitus is related to a library of patterns in auditory memory and also, by reference to the limbic system, associated with an emotional state. Unfortunately for those suffering (rather than just experiencing) tinnitus, the emotional associations are usually extremely negative. When tinnitus emerges, a person is concerned that it might indicate the presence of a brain tumour, stroke, the first stage of total hearing loss, or 'going mad', and that tinnitus will last forever and nothing can be done about it. Usually the tinnitus sufferer seeks advice from a family physician and later an otolaryngologist to learn that there is no clear explanation for tinnitus and that indeed, nothing can be done and they will have to live with tinnitus forever. Commonly, this prompts a search for further specialist opinions, only to have this initial diagnosis confirmed on each occasion. Indeed severe tinnitus distress can often be linked to the large number of consultations (often in excess of 20) patients have obtained on their travels seeking the cure for tinnitus.

This process can be classified as 'negative counselling' and results in the enhancement and solidification of the initial aversive association of the perception of the tinnitus sound with emotional state. Once established, this process becomes a vicious circle (aptly termed the 'headless chicken stage'), resulting in a firm consolidation of detection of the tinnitus-related pattern and establishment of a strong negative reaction towards tinnitus. As a result, the normal auditory habituation to repeated sounds cannot occur, and the more a patient is concerned about tinnitus and focuses on its presence, the more prominent tinnitus becomes. Finally, patients may react strongly to even relatively weak and intermittent tinnitus, living in constant fear and dread of it.

This stage of the evaluation of the tinnitus signal is of fundamental clinical significance, because these cortical associations can be modified much more easily than connections within subcortical centres, involved in the pattern recognition process. Behavioural neurophysiology has shown that it is possible to train a subject to be afraid of any particular external sound and, using more time and effort, to retrain the subject to habituate to this sound and ignore it totally. The same principle can be applied to tinnitus patients.

Thus, the present clinical goal in treating

tinnitus is to remove its perception from the patient's consciousness and to initiate and facilitate the process of tinnitus habituation. At the same time, animal research is centred on finding a mechanism-based method for suppressing tinnitus generators.

Clinical implementation

Introduction

The theoretical analysis already outlined in this paper has profound clinical implications which determine the process of diagnosis and treatment of patients. Focusing treatment on the behavioural retraining of, the associations induced by tinnitus **perception** and on inducing and further facilitating the process of habituation of tinnitus **perception** is consequential to all stages of interaction with the patient.

In determining the optimal treatment, the exact cause of local cochlear damage is largely irrelevant (e.g. noise, viral infection, ototoxic drugs). However, it is critical to determine if abnormally high gain within the auditory system occurs. Similarly, while it is interesting to have an audiometric estimation of tinnitus loudness, more informative is data on tinnitus maskability, including changes in tinnitus perception while gradually increasing the level of white noise presented to the patient. The patient's association of the perceived sound of tinnitus with past experiences is important, as well as patient evaluation of psychological and social problems resulting from hearing loss. These issues dictate our diagnostic strategy, and determine what are the most important questions to ask and tests to perform.

Therefore, the diagnosis is focused on the evaluation of a psysiological basis for altered perception, and identifying the psychological aspects of perceived tinnitus. The standard general medical and otolaryngological evaluations are included, but on their own these are usually uninformative. While important in detecting other medical problems and somatosounds, which are relevant to the tinnitus complaint in only a small proportion of cases (less than 5% in our clinics), these are dealt with separately. Additionally, data is collected to estimate the integrity of OHC and IHC systems and is used in the evaluation of a potential cochlear generator.

According to this neurophysiological approach, the present standard treatment is aimed at the retraining of subcortical and cortical centres involved in processing tinnitus signals, without attempting to suppress the tinnitus generator, which at the present stage of our knowledge does not seem to be systematically possible. These processes involve changing the strength of functional connections between neurons involved in the processing of tinnitus-related signal, with the goal of decreasing tinnitus detectability and the transmission of tinnitus-related signals to emotional centres in the brain. This process involves the occurrence of permanent metabolic and morphological changes at the synapses connecting these neurons and therefore requires a substantial amount of time. The time required for transformation of already established synaptic connections is responsible for the relatively slow process of clinical improvement measured in months, with the ultimate benefit being reached after about a year to 18 months. On the other hand, the positive consequence of aiming our approach on plastic synaptic transformations is that, contrary to other methods, once this stage is achieved there is no need fur further systematic intervention. The modification in the processing of the tinnitus signal is achieved by utilizing methods of cognitive therapy with highly specific and directive counselling, and at the same time decreasing detectability of tinnitus by introducing a low level, neutral acoustic signal, typically white noise. Hyperacusis is dealt with by a process of desensitization, again using wearable white noise generators, with the signal level adjusted gradually according to our protocol.

This approach seems to us to be optimal, while we do not presently have an effective method of significantly attenuating tinnitus generators or the possibility of pharmacologically altering abnormal gain increase or patterns of abnormal activity within the auditory pathways. Once progress is made in this respect and it is possible to attenuate rapidly the perception of tinnitus, the behavioural retraining technique should become complementary to pharmacology. Nevertheless, we anticipate that this approach will still be required, short of total therapeutic elimination of tinnitus perception. Otherwise the perception of even weak residual tinnitus signals may undergo enhancement, returning the patient to the initial stage of distress, which, as our clinical experience and research data point out (Hazell 1985), is not simplistically related to tinnitus loudness.

Diagnostic procedures

In accordance with our approach every patient is evaluated in an individual manner. There are certain differences in emphasis between Baltimore and London clinics, with higher stress on accuracy of measurement, hyperacusis, and (previously) with evaluation of OAE distortion products only in Baltimore.

In Baltimore, the basic audiological evaluations similar to the one performed in the majority of audiological centres, except that by incorporating some methods used in psychoacoustical laboratories the accuracy of measurements is increased. This particularly applies to the hearing threshold evaluation, pitch and loudness of tinnitus, maskability of tinnitus, and loudness discomfort levels. To assess damage of the OHC, we are performing measurements of the OAE distortion product, with a high frequency resolution, when typical 10 points per octave measurements indicate the need for higher precision. The measurement

of the distortion product allows us to estimate the functional integrity of the OHC system (Avan and Bonfils, 1992; Lonsbury-Martin *et al.*, 1987; Martin *et al.*, 1987; Siegel *et a/.*, 1982; Lonsbury-Martin *et al.*, 1991; Harris *et al.*, 1989), and by comparing these data with a detailed audiogram it is possible to estimate the presence of discordant damage in the hair cell systems. Furthermore, these data are related to the estimated pitch of tinnitus. In accordance with the hypothesis of discordant damage of the OHC and IHC systems (Jastreboff, 1990) we are comparing the pure tone audiogram with distortion product measurements searching for frequency ranges where distortion products are disproportionally smaller than that which might be predicted from the hearing loss. Such data suggest the presence of localized areas on the basilar membrane where there is noticeable OHC loss and where IHC are reasonably intact. Since the theory predicts that the dominant tinnitus pitch is localized near the bottom of the slope of audiogram, this prediction is checked as well.

Presently there are no data proving or disproving the hypothesis of involvement of the efferent system in tinnitus generation and modulation, nor is there a proven tool for the measurement of efferent activity, precluding the evaluation of the state of the efferent system as a routine clinical tool for tinnitus patients. However, it is plausible that the efferent system might be involved in modulation of primary cochlear tinnitus generators, since physiological data clearly show the changes of the mechanical properties of the organ of Corti and the modulation of the active processes which occur in the cochlea as a result of increased efferent input (Pujol, 1991; Plinkert *et al.*, 1989; Liberman, 1989; Liberman and Brown, 1986; Siegel and Kim, 1982; Warren and Liberman, 1989). In this respect, the accumulation of data on inhibition of the otoacoustic emission by enhancement of the activity of the efferent system induced by contralateral low level auditory stimulation is of interest (Mott *et al.*, 1989; Norton *et al.*, 1989; Veuillet *et al.*, 1991). This method might provide a useful tool for investigating the involvement of the efferent system in tinnitus. The experimental human work on evaluation of the correctness of the efferent system hypothesis is presently in progress in London, while in Baltimore the modulation of hearing threshold by contralateral low level noise is used to estimate involvement of the efferent system in some patients.

Finally, speech recognition tests combined with interview help us in the relative ranking of the importance of impaired communication skills *v*. the impact of tinnitus. Information obtained from all audiological measurements helps to assure that audiological problems other than tinnitus are not overlooked, assists in the decision of whether specifically selected hearing aids might be appropriate in tinnitus treatment, and contributes to a determination of whether the tinnitus generator is localized in the cochlea or at a higher level.

Specific tinnitus tests include evaluation of hyperacusis and psychoacoustical properties of tinnitus, particularly the process of its maskability. Theoretical analysis points out that even mild hyperacusis indicates the presence of increased gain within the auditory pathways, which then yields abnormal enhancement of peripheral signals, including tinnitus. In some cases, this factor alone can be responsible for tinnitus, similarly to tinnitus perceived by the majority of people who are temporally deprived of sound in an anechoic chamber (Heller and Bergmann, 1953). Therefore, hyperacusis is evaluated carefully by both direct frequency specific measurements of loudness discomfort, and by a detailed assessment to detect any intolerance of specific environmental sounds. Alleviation of such hyperacusis is the first step in dealing with any coexistent and distressing tinnitus. Measurements of hyperacusis performed during subsequent visits provide an important indication of the progress of treatment (Hazell and Sheldrake, 1992).

A detailed medical examination follows. This examination evaluates the general medical status of the patient and other potential medical problems but focuses particularly on psychological aspects of tinnitus perception. The questions are aimed at finding out the inter-relations between tinnitus perception and emotional reactions to it, a precise determination of the patient's view of tinnitus, its likely development and its possible impact on life or life quality. This information is essential for proper adjustment of the treatment protocol.

Treatment strategies

From the presented material it is clear that our approach is based on the work of the multidisciplinary team, where every member of the team contributes specialized knowledge to the patient evaluation. Consequently, in the next step there is a short meeting of otolaryngologist, audiologist and neurophysiologist, who, together, review the data and establish an appropriate treatment programme tailored to the individual case. Our approach does not involve drug treatment or surgery, unless this is separately and medically indicated. Even where otosurgery is a clear option, proper work must first be done to prepare the patient for further tinnitus treatment which will be necessary if tinnitus attenuation is not complete.

The primary element of therapy is to provide the patient with the understanding of what causes their tinnitus. Therefore, directive counselling plays a vital role in each case. The physiology of the auditory system is explained to the patient, with a detailed explanation of the mechanism of tinnitus which is most probably acting in his/her ease. In nearly all cases of tinnitus the patient

feels threatened by the tinnitus. It is important to identify these specific anxieties about tinnitus, to retrain thinking and to present tinnitus as a benign and harmless phenomenon, which can be reduced and sometimes eradicated by appropriate treatment. At the very least we find it is possible to significantly reduce distress evoked by tinnitus.

When patients first visit us they are often very depressed, anxious, alarmed and fixed in their belief that nothing can be done about their tinnitus. Even if others have been helped, they are unique in their position of being unhelpable. They are terrified that their problem will be labelled 'psychological'. Strongly held beliefs take time to change, and adequate discussion time (never less than one hour, and often more than two) must be allowed to start the process of changing these beliefs. In addition, appropriate sequential reinforcement is needed so that these new ideas can first be understood, and then used to replace the original in-appropriate feelings about tinnitus, which were the cause of distress. Typically four to six visits may be needed over an 18-month period. Only professionals perceived by the patient to understand the ear and the auditory system are effective in this early stage of directive counselling.

The process of a cognitive therapy, as outlined above, is sufficient alone in about 15% of eases (including both authors). Providing the patient with knowledge on the physiological basis of tinnitus creates an environment for introducing the second, acoustic element of the treatment, aimed at decreasing tinnitus delectability and facilitating the process of tinnitus habituation, initiated by the cognitive therapy.

In the majority of cases, this element of the treatment is based on the use of low level, stable, white noise to interfere with tinnitus perception and reverse the increase of the gain in the auditory system. This is achieved by using a wearable white noise generator (previously called a 'masker') but with the clear understanding that the goal is not to mask the tinnitus in the psychoacoustical sense. According to behavioural retraining theory, masking is actually counterproductive since for retraining to occur the stimulus being retrained has to be perceived during training! Therefore, perception of tinnitus has to be present during the process of retraining, although for changing of the synaptic connection it is helpful when tinnitus signal within the auditory pathways is partially attenuated. White noise causes an increase of the mean neuronal activity within the auditory system, making detection of the tinnitus-related signal more difficult and thus facilitating the process of habituation. At the same time, the perception of white noise signal is relatively easy to habituate by the patient, and when kept at low level it interferes to the least extent with the perception of external sounds. Furthermore, as predicted by theory (Jastreboff, 1990) and confirmed by experimental data (Hazell *et al.*, 1985), the observation that tinnitus can or cannot be totally masked has no relevance for predicting the outcome of the treatment. Importantly, patients in whom it is impossible to mask tinnitus, and who therefore would not be accepted for masker therapy, are perfectly proper candidates and are being helped systematically. Since masking of tinnitus is not crucial, another obvious implication of the neurophysiological theory is the irrelevance of shaping the masking noise to optimalize masking of tinnitus. The best signal is the one that provides stable, random excitation of neurons involved in processing of acoustic information in as wide frequency range as possible. Such a signal is optimal for interfering with the extraction of tinnitus related activity from the background and for easy habituation.

On the other hand, the theory predicts that as long as an additional external signal is emotionally neutral, so it is not inducing anxiety or increasing the general arousal level, and includes the frequency range of tinnitus-related activity, it should be effective too, although it might require a longer time for retraining. Therefore, this additional external signal can be provided by specifically tailored and used hearing aids, which amplify environmental sounds. It has been shown that in some cases where there is hearing impairment (often in the high frequencies and undetected by the patient), hearing aids, appropriately fitted and counselled can be equally effective (Sheldrake and Hazell, 1992). Importantly, the primary use of the device is not to improve the hearing, but to counteract the process of tinnitus detection. Specific protocols for the use of white noise generators and hearing aids are of fundamental importance for the successful outcome of the treatment.

In each case the rationale of the proposed treatment is discussed with the patient, providing detailed justification of the particular approach. If maskers' or 'hearing aids' are appropriate, the patient is placed under the management of an audiologist trained in tinnitus treatment who fits the patient with the proper device and explains in detail the protocol for its use in tinnitus alleviation.

Negative emotional associations with tinnitus are more likely to be made in those with anxiety (Hazell, 1987, 1990; Dobie *et al.*, 1992; Halford and Anderson, 1991; Collet *et al.*, 1990) or with a history of depressive illness. Additionally, increased levels of general arousal counteract the process of habituation. Thus, parallel management of related emotional disturbance is often needed from a clinical psychologist, an important member of the tinnitus team. The psychologist can be essential in continuing cognitive therapy sessions, as well as teaching techniques for anxiety control, such as relaxation, thought-blocking techniques, and insomnia strategies.

The process outlined above takes about one

year to 18 months to reach the level when the patient is not aware of tinnitus majority of the time, or at all, except when he/she focuses attention on tinnitus. At this stage white noise generators are not needed any more and many patients discontinue their use. Some patients find the use of generators assuring and continue their use '… unless I forget to put them on'. From time to time, particularly at periods of increased stress, tinnitus resurfaces, but then, as predicted by neurophysiology, short repetition of the treatment, which last a few weeks, is sufficient to restore the previous level of comfort.

Summary

The combination of theoretical neurophysiological analysis with previous clinical findings accumulated in London resulted in the creation of a multidisciplinary Tinnitus Centre in Baltimore, which pursues a double goal: the present clinical goal is to remove the perception of tinnitus from the patients' consciousness by initiating and facilitating the process of tinnitus habituation, and the research goal of finding a mechanism-based method for the suppression of tinnitus generators and processes that enhance the initial tinnitus related signal. The results obtained in the clinical branches of the Centres are encouraging and offer help to tinnitus sufferers now, while experimental results suggest new approaches, which in the future can be incorporated to our protocol, further improving our effectiveness in dealing with tinnitus.

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During the process of review of this paper, Mark Lutman pointed out the possibility that hair cell regeneration following damage could set up unstable conditions that could lead to tinnitus. His interesting suggestion expands the discordant damage hypothesis and although at the moment there are no data indicating the presence of hair cell regeneration in humans, and therefore no indication of tinnitus based on this mechanism, nevertheless, such an unstable condition could occur following the recovery of hair cells experiencing metabolic dysfunction during temporary or permanent threshold shift.

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