
Editorial

3  GD Searchfield, R Goodey

Articles

4  A surgeon’s understanding of hearing and tinnitus
   R Goodey

9  Personality and perception of tinnitus
   D Welch, PJD Dawes

18 Noise-induced hearing loss and tinnitus
    PR Thorne

25 Acoustic shock disorder (ASD)
    M Westcott

32 Animal models of tinnitus
    CL Darlington, PF Smith, Y Zheng

39 Neural synchrony and neural plasticity in tinnitus
    LE Roberts, DJ Bosnyak

51 Similarities between chronic pain and tinnitus: what we’ve learned from chronic pain and how it applies to tinnitus
    JE Magnusson

59 Medical evaluation and management of tinnitus
    R Goodey

66 Promising medications for tinnitus
    PF Smith, Y Zheng, CL Darlington

73 Tinnitus management with repetitive transcranial magnetic stimulation
    CM Stinear

77 Cortical electrical suppression of tinnitus and modulation of its related neural activity
    JS Zhang, ZL Guan, XG Zhang, H Beydoun, J Zhang, M Seidman, K Elisevich, S Bowyer, Q Jiang, J Moran

89 Auditory attention and tinnitus
    K Wise, D Singh, GD Searchfield
101  Tinnitus assessment  
GD Searchfield, C Jerram

112  Sound therapies and instrumentation for tinnitus management  
GD Searchfield, H Cameron, S Irving, K Kobayashi

126  Tinnitus perception and the effects of a self-programmable hearing aid on hearing fluctuation due to Ménière’s disease  
C McNeill, A Taylor

136  Combination open ear instrument for tinnitus sound treatment  
L Carrabba, G Coad, M Costantini, L Del Bo, O Dyrlund, S Forti, GD Searchfield

141  Perceptual training of tinnitus  
K Jepsen, M Sanders, GD Searchfield, K Kobayashi

154  Hyperacusis: a clinical perspective on understanding and management  
M Westcott

161  Multidisciplinary team approaches to tinnitus  
G Shakes
Editorial

GD Searchfield, R Goodey

Tinnitus is a perception of sound in the absence of sound. It is a very common symptom. Conservatively we estimate that 5% of adult New Zealanders experience annoying tinnitus. Like pain, tinnitus is a sensation which affects different individuals in different ways. It is difficult to quantify and often difficult to treat. It follows a complex cascade of neural responses usually initiated by damage in the auditory system. In the last 20 years there have been considerable advances in the understanding of its pathogenesis and in the treatment of its effects though not in the treatment of the perception itself.

In September 2009 the Oticon Foundation Hearing Education Centre at The University of Auckland, in association with Otago University, hosted “Tinnitus Discovery: Asia and Pacific Tinnitus Symposium”. This symposium brought together many of the most prominent tinnitus clinicians and researchers in our region. This special issue is a collection of papers arising from that meeting.

We greatly appreciate the support of the NZMJ in bringing this collection of papers to print.

We would like to thank the generous sponsorship of the following organizations for contributing to a very successful symposium:

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- Auckland UniServices
- TRI Auditory Stimulation Working Group
- National Foundation for the Deaf
- Oticon NZ
- GN ReSound Denmark/USA
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A surgeon’s understanding of hearing and tinnitus

R Goodey

Abstract

The diffuse, extra-lemniscal, non-classical auditory pathways are presented as the primitive and basic auditory pathways. They have close interactions with limbic and autonomic systems and are polysensory and shared with somatosensory inputs. The classical auditory pathways are presented as a later development enabling sophisticated analysis of sound in the primary auditory cortex and facilitating its use for communication and the development of language, but influenced by and competing with the non-classical pathways. This view impacts on our understanding of tinnitus generation, perception and reaction to it.

Tinnitus generation can now be accepted as a central phenomenon. However, the triggering and perpetuating effects of altered sensory inputs, emotional factors, and central injury mean that our routes for treating it are still the same.

Review of an extensive tinnitus database reveals changing patterns. For a clinician concerned primarily with treating patients who have tinnitus, there is reason for concern at the quantity of knowledge being accumulated and how difficult it is to extract from it information which improves the treatments we prescribe and the prospects for discovering complete cures.

Previously, when I thought of hearing I thought of the classical auditory pathways, conscious awareness of sound and acquisition of language. Now, I find it better to think first of the non-classical auditory pathways, which are more primitive and basic, and have much in common with other primitive sensory inputs such as olfaction.

Primitive responses to sound stimulation pass in the diffuse, extra-lemniscal, non-classical auditory pathways, and have close interactions with the limbic and autonomic systems as they pass to the secondary auditory cortex to produce reactions and responses which are largely at an unconscious level but which also influence the primary auditory cortex. These pathways are polysensory and are shared with somatic sensory inputs from V, VII, IX, X, and C2 (Figure 1).

I think of the classical auditory pathway as a later development, capitalising on increasingly sophisticated transduction in the inner ear and passing more ventrally on its way to the primary auditory cortex. It enables increasingly sophisticated analysis of sound, its use for communication, the development of spoken language, and subsequently of written language and the evolution of our sophisticated civilisation. However, the classical pathways and our conscious perception of sound are constantly being influenced by the non-classical pathways and the systems with which they interact (Figure 2).
Figure 1: Non-Classical Auditory Path. Diffuse, extra-lemniscal and polysensory (somatosensory). C2: second cervical vertebra; CN: Cochlear nucleus; IHC: Inner hair cell; ICC: Inferior colliculus; IX: Glossopharyngeal nerve; MGB(d): Dorsal medial geniculate body; MSN: Medullary somatosensory nuclei; V: Trigeminal nerve; VII: Facial nerve; X: Vagus nerve.
I have always been aware of the importance of central factors in determining whether the neural activity we perceive as tinnitus reaches a level of conscious perception, intrusiveness and annoyance. Dr Pamela Melding and I wrote about it as early as 1979. Previously I thought in terms of both peripheral and central generators of tinnitus. I am now comfortable to think of all tinnitus as being generated centrally. However, effects of altered sensory inputs (auditory, somatosensory and probably others) from the periphery are fundamental to the sequence of events which trigger the onset of tinnitus and then help perpetuate it (Figure 3).

Understanding our patients’ tinnitus requires assessment of altered sensory input, emotional influences and central injuries, and not just of central generators. Acceptance of the central generation of tinnitus has not significantly altered my approach to its treatment.

When we come to treat a patient who has tinnitus, the same three routes are available for us to influence the central generators as may have helped trigger it in the 1st place:

1. We may improve or manipulate the various sensory inputs acoustically, physically, electrically, surgically, with drugs and eventually, we hope, with hair cell regeneration.

2. We may use psychological approaches which may be at a conscious level with explanation, understanding and cognitive therapy (de-concerning); at an unconscious level with music, other sounds, tinnitus retraining therapy (de-
conditioning); and by reduction of emotional associations with counselling and/or drugs.

3. We may approach the central nervous system directly by surgery, by electrical or magnetic stimulation or with centrally-acting drugs, hormones and dietary factors.

At the present time we usually have to settle for ameliorating tinnitus or simply reducing its impact upon our patient’s life. However, our goal is to cure our patients of tinnitus.

In my search for better treatments, I have enlisted the help of research librarian Sally Wheater and together we have set up and maintain as an EndNote library a database of citations which includes “tinnitus” in the title and/or abstract. Not only do we search existing databases of peer reviewed journals, but also other sources including newspaper and magazine articles, advertisements, conference papers, posters and proceedings. This database includes abstracts but not the full articles. It is available on CD to anyone who wants it. Each month an update of all new articles is e-mailed to anyone who asks to be on our e-mailing list.

In May 2009, there were 7889 citations of which 6082 were from peer reviewed journals. The number of articles about tinnitus has increased steadily from 63 in 1980 to approximately 430 in 2008.

The number of peer-reviewed articles with “therapy, therapeutic or treatment” in the title or keywords has increased from 26 in 1982 to 133 in 2008. Many of these are focused on tinnitus as a complication of treatment and fewer than half on treatment of tinnitus. Drug therapies and trials have been described most frequently and the drug most commonly investigated has been lidocaine, with more than 130 citations. However, the number of drug trials being reported is decreasing, especially trials of drugs which have a central action. There were only six in 2008. Since 2000 the total number of articles on psychological therapies including counselling, cognitive therapy and deconditioning processes (mainly tinnitus retraining therapy) have doubled. They are often included as part of a total package of care. The most rapidly increasing group is that of articles describing repetitive transcranial magnetic stimulation, which was only first described for tinnitus treatment in 2003. In 2007, there were 26 citations from 12 centres.

When we look outside the tinnitus literature we tend to focus on that of chronic pain because of the analogies between chronic pain and tinnitus. However the chronic pain literature is immense and not always comparable. Chronic itch has many similarities to tinnitus, the database is modest and those working in the field also monitor the chronic pain literature as we do and we should monitor the chronic itch literature.

Our tinnitus literature is nowhere near as big as that of chronic pain but, nevertheless, it has become very extensive. We are now burdened with an information overload. The amount of knowledge available to us is increasing but our ability to monitor it and use it to treat our desperate patients may not be improving at a comparable rate. In my view, we need better methods to identify and share useful information which is being overlooked. We need to identify and facilitate research which is focussed on finding better treatments. We have to be prepared to look outside the square.
The papers in this conference will help improve our understanding of how tinnitus generation is initiated and perpetuated, how we can better manage a patient with tinnitus, and the prospects for discovering complete cures. Those contributing have been looking outside the square.

Thank you for sharing your expertise. Welcome to Tinnitus Discovery.

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Personality and perception of tinnitus

D Welch, P J D Dawes

Abstract

Our objective was to develop understanding of the role of personality in the perception of tinnitus in the general population. As a theoretical basis for this, we combined elements of a general model of signal detection with the ideas of ignition (development) and promotion (neural transmission) of tinnitus, and considered plausible roles for personality factors within this conceptual framework. We interviewed a birth cohort of 970 people aged 32 years sampled from the general population. On the basis of questioning, we divided them into three groups, those without tinnitus, those with occasional tinnitus, and those who experienced tinnitus most of the time. We also established how annoying or distressing the tinnitus was. We assessed personality using the Multidimensional Personality Questionnaire.

Tinnitus was experienced rarely by 38.2% and half the time or more by 6.8% of those studied. Men and women did not differ in the amount of tinnitus reported, but women were more likely to find it annoying. People from lower socioeconomic backgrounds were more likely to report tinnitus. People with tinnitus were more socially withdrawn, reactive to stress, and alienated, and less self-controlled. People who were more annoyed by tinnitus were more socially withdrawn, and men were more stress reactive and alienated. Our interpretation of the findings is that personality influences the persistence of tinnitus by influencing the tendency to be aware of it. Consideration of personality factors may improve the ability to tailor tinnitus therapies, and the concept of awareness may benefit treatment outcomes by showing tinnitus sufferers a means of internalising the locus of control over their symptoms.

Despite the varying aetiologies and experiences associated with tinnitus, it is a percept and thus may be supposed to depend upon two components: firstly, the necessary physiological or pathological conditions to provide a stimulus to the auditory cortex, and secondly the personal tendency to report a sound as present.1

Personality describes a set of behavioural traits which display continuity through the life course.2 It is well established that personality depends upon genetics and environment3, and it has been shown that it has a predictive role in mental health and health risk taking.4 In other words, personality reflects both underlying physical and physiological states and overt behaviour, and has been shown to be a pervasive influence in people’s lives. It has been linked with tinnitus in clinical groups, where presence and/or degree of tinnitus, have been associated with higher anxiety, depression, and neuroticism.5-9

Given that tinnitus is a perceptual experience, and that personality may influence it, increased understanding of the association between the two is of interest. We asked a general population sample birth cohort about the amount of tinnitus they experienced, and how distressing they found it. We also assessed personality, and developed profiles describing the different types of personality associated with the different aspects of tinnitus reported. The purpose of this research was to determine whether personality factors differed with these different experiences of tinnitus. The research
and findings on which this talk was based are reported in full in the Ear and Hearing Journal.

**Method**

**Participants**—Participants were members of the Dunedin Multidisciplinary Health and Development Study, a longitudinal investigation of health and behaviour in a birth cohort. The study members were born in Dunedin, New Zealand between April 1972 and March 1973. Of these individuals, 1037 children (91% of eligible births; 52% male) participated in the first follow-up assessment at age 3, which constituted the base sample for the remainder of the study. Cohort families represented the full range of socioeconomic status in the general population of New Zealand’s South Island and were mainly white of European descent. Follow-ups were done at ages 5, 7, 9, 11, 13, 15, 18, 21, 26, and most recently at age 32 years when we assessed 972 (96%) of the 1015 study members still alive. 970 of these answered the questions about tinnitus. The Otago Ethics Committee granted approval for each phase of this longitudinal study. Study members gave informed consent before participating.

**Assessments**—Tinnitus was assessed at age 32 by asking two questions adapted from the questionnaire used by Davis et al. The first question asked “In the last 12 months, when you are awake and it is quiet, have you experienced tinnitus (ringing, whistling or buzzing) in your head and ears?”, and had five response options: never, rarely, about half the time, most of the time, and all of the time. The option ‘rarely’ may include people who had experienced only transient tinnitus lasting less than five minutes. The second question, asked only of those who had experienced tinnitus was “How annoying or upsetting is it?”, to which four replies were possible: not at all, slightly, moderately, and severely.

Amount of tinnitus experienced and annoyance caused by tinnitus was then both compared with personality as measured by the Multidimensional Personality Questionnaire (MPQ). The MPQ is widely used. Its scales have all been established as having high internal reliability, and validity with reference to other personality measures is also high. It provides a description of personality via yes/no questions which represent scales. These scales define first-order factors which are organised under three second-order factors, or ‘superfactors’. The superfactors are listed and their constituent scales described in Table 1.

<table>
<thead>
<tr>
<th>MPQ Scale</th>
<th>Characteristics of a high scorer</th>
</tr>
</thead>
<tbody>
<tr>
<td>Positive Emotionality</td>
<td></td>
</tr>
<tr>
<td>Achievement</td>
<td>Strives for excellence; works hard; persistent.</td>
</tr>
<tr>
<td>Social Closeness</td>
<td>Affectionate; likes and relies upon other people.</td>
</tr>
<tr>
<td>Social Potency</td>
<td>Likes to control others; wants to stand out; likes to assume leadership roles.</td>
</tr>
<tr>
<td>Well Being</td>
<td>Content and happy with life; finds experiences interesting and pleasurable.</td>
</tr>
<tr>
<td>Negative Emotionality</td>
<td></td>
</tr>
<tr>
<td>Aggression</td>
<td>Enjoys hurting others, physically and emotionally; likes to witness suffering in others.</td>
</tr>
<tr>
<td>Alienation</td>
<td>Sees others as opponents and anticipates betrayal; feels disadvantaged because of others’ treatment.</td>
</tr>
<tr>
<td>Stress Reaction</td>
<td>Emotionally oversensitive, moody and irritable; experiences nervousness, worry, and anxiety.</td>
</tr>
<tr>
<td>Constraint Superfactor</td>
<td></td>
</tr>
<tr>
<td>Harm Avoidance</td>
<td>Prefers tedium and discomfort to risking physical danger. Does not enjoy activities with potential for injury.</td>
</tr>
<tr>
<td>Self Control</td>
<td>Plans and organises activities; cautious; not spontaneous.</td>
</tr>
<tr>
<td>Traditionalism</td>
<td>Adopts a conventional morality and believes that this should be imposed more firmly upon others.</td>
</tr>
</tbody>
</table>

Table 1. Multidimensional Personality Questionnaire superfactors and descriptions of subscales
Results

Basic descriptive findings—The amount of time spent with tinnitus in the last twelve months was compared between men and women. There was no association between the proportion of time spent with tinnitus and sex (chi-squared=2.786, p=0.248).

The 437 participants who experienced any tinnitus were asked how annoying or upsetting they found it. Men and women differed, (chi-squared=11.169, p=0.004) in that men were more likely to report no annoyance from tinnitus, while women were more likely to report slight annoyance.

Among those 437 who experienced any tinnitus, the amount of tinnitus suffered was compared with the annoyance experienced. Those who experienced tinnitus more often tended to find it more annoying (chi-squared=19.955, p<0.001).

The amount of tinnitus experienced by different childhood SES groups was compared. Those with lower SES during childhood were more likely to experience tinnitus at age 32, and more likely to experience it more of the time (chi-squared=19.085, p=0.001).

Personality and amount of tinnitus—The amount of tinnitus was associated with differences in several personality traits. Social Closeness was lower in groups with more tinnitus (F(2,946)=13.242, p<0.001). Stress Reaction (F(2,946)=9.144, p<0.001) was higher in the groups with more tinnitus. Alienation was higher in those with more tinnitus (Wald=12.206, p<0.001). Self Control was lower in groups with more tinnitus (F(2,946)=4.752, p=0.009), and marginal evidence in traditionalism was marginally lower in groups with more tinnitus (F(2,946)=2.677, p=0.069). In all personality variables for which effects were significant, the “tinnitus rarely” group scored between the other two groups implying graded associations between tinnitus and personality (Figure 1).

Personality and annoyance of tinnitus—or those who experienced tinnitus, we compared the personality traits of those who were more or less annoyed by it. We also ran analyses wherein we controlled for time spent with tinnitus to allow consideration of tinnitus-related annoyance independent of the amount of tinnitus experienced, and found the same effects. The amount of annoyance caused by tinnitus showed an interaction effect with sex so analyses were conducted separately for men and women (Figure 2). Social Closeness and Wellbeing were lower with more tinnitus distress in both sexes (F(2,424)=3.471, p=0.032; and Wald=6.579, p=0.010 respectively), but for Stress Reaction; men with more tinnitus annoyance scored higher, but women did not (for women: (F(2,203)=0.171, p=0.843), and for men: F(2,220)=12.440, p<0.001)); and higher Alienation was associated with tinnitus-related distress in men but not women (for women: (Wald=0.056, p=0.813), and for men: (Wald=14.722, p<0.001)) (Figure 2).
**Figure 1:** Mean standardised score on each MPQ subscale at age 32 for those who experienced tinnitus never (triangles), rarely (open squares), and half the time or more (filled squares).

**Figure 2:** Mean standardised score on each MPQ subscale at age 32 for those who experienced tinnitus and who found it not at all (filled diamonds), slightly (open circles), and moderately or severely (filled circles), annoying. Separate graphs have been plotted, and data standardised, by sex, because sex by tinnitus annoyance interaction effects were detected for alienation and stress reaction.
**Discussion**

The findings presented here were in a general-population sample of young adults, and showed that 7% experienced tinnitus at least half of the time, while a further 38% had less frequent episodes during the past year. Our findings support earlier reports of tinnitus prevalence in young adults\(^{15}\), and we add a description of personality profiles for those who experience tinnitus (Figures 1 and 2). These findings inform us that, in a non-clinical group, tinnitus has an associated pattern of personality traits which may impact on both the response to treatment, and the likelihood of seeking it.

The personality effects reported can reasonably be expected to be less than those described for clinical groups, who have suffered tinnitus for longer, who are on average older, and for whom tinnitus may have adversely affected their psychological health. However, the severity of tinnitus increases over time in about a quarter of people\(^{15,16}\), so the personality traits observed in this non-clinical group may extend to sufferers of worse tinnitus, and our findings may be useful for clinicians and researchers dealing with tinnitus sufferers.

**Personality and time spent with tinnitus**—A recent hypothesis suggests that there are two aspects to the development of tinnitus. These are ‘ignition’, the development of an abnormality (possibly injury) in the auditory system; and ‘promotion’, whereby the effect of that abnormality is enhanced and transmitted to the auditory cortex\(^ {17,18}\). The cortical representation of a stimulus does not, however, mean that a person is aware of that stimulus. We propose a third aspect, ‘awareness’, based on the psychophysical principal that sensitivity to (or intensity of) a signal, and placement of a response criterion (bias) to report the presence of the signal both contribute to its detection; this has been labelled the Theory of Signal Detection\(^ 1\) (Figure 3).

In the context of tinnitus, the level of the signal would depend upon a combination of the ignitor and promoting activity. The criterion placement would reflect the person’s predisposition to experience a given level of cortical activity as tinnitus; someone predisposed to do so would tend to place the criterion further to the left, and someone predisposed not to would tend to place it further to the right. For example, a noise induced injury to the cochlea may lead to cortical activity as a consequence of both the degree of injury and promoting central events—e.g. the increased base firing rate of unstimulated central connections. The criterion placement in relation to tinnitus would be the reason why two people with the same history of injury and the same level of promotion may have awareness of differing amounts of tinnitus. The two people may have different criterion placement because of personality differences.
We demonstrated an association between adult personality, and the amount of time for which tinnitus was experienced at age 32. The findings were that greater experience of tinnitus was associated with higher Negative Emotionality, lower Constraint, and lower Positive Emotionality. This common personality profile for tinnitus sufferers may reflect the idea that, for identical physiologies and pathologies, two people may still have differing awareness of tinnitus due to the placement of the criterion as described theoretically above. Those with this personality profile are less close to other people, more mistrustful, and more reactive to stress than others.

These features of their personality may tend to increase awareness of, and thus to report experiencing, tinnitus. We think it more likely that the experience is because personality affects awareness of tinnitus, than being because personality affects the promotion of the signal.

Tinnitus-sufferers were also lower in Constraint than others, and particularly the Self-Control subscale. An explanation for this finding can be made by considering the process of ignition; people who are less careful may be more likely to have damage to their hearing due to exposure to noise. That this trait differs from the others is supported by the finding that Constraint was not associated with the amount of annoyance caused by tinnitus (discussed below). That is, if the decision that tinnitus is annoying depends upon similar criteria to the decision that tinnitus is present, then one would not expect Constraint to influence it, because its influence is on behaviour which influences ignition rather than criterion placement. That Constraint was not associated with tinnitus annoyance supports this interpretation.

There are two other possible explanations of our findings: that tinnitus and personality are both reflections of underlying physiological factors which are expressed in both behavioural and neurological ways, or that tinnitus has a causal effect on personality development. Since it is known that tinnitus is common (prevalence ranging from 6-
29% in the normally hearing) in childhood\textsuperscript{19}, and we did not investigate tinnitus then, we cannot comment definitely upon the direction of effects.

**Personality and annoyance caused by tinnitus**—The annoyance or distress caused by tinnitus was associated with differences in personality whether or not we controlled for the proportion of time that tinnitus was present. Greater tinnitus-related annoyance was associated with lower Wellbeing and Social Closeness; and in men but not women, with higher Stress Reaction and Alienation.

The finding of lower Wellbeing in those who report more tinnitus-related annoyance is expected given previous research\textsuperscript{20}, and may well be an effect of being annoyed by tinnitus rather than causal of it. The finding of reduced Social Closeness – reflecting a preference for relative personal isolation – among those who report more tinnitus-related annoyance is interesting.

One explanation for this might be cognitive dissonance; the psychological discomfort experienced when one’s ideals and actions conflict. In this case, cognitive dissonance may arise when a person with strong self-reliance (i.e. low Social Closeness) experiences an unpleasant sensation (i.e. tinnitus) which is self-generated. A second possibility is that, spending more time alone, the relative impact of the tinnitus as noise is greater.

The findings that tinnitus-related annoyance was associated with higher Stress Reactivity and Alienation in men but not women may be explained by considering the finding that men with tinnitus were less likely to report annoyance than women (Table 2), which itself reflects the general finding that men tend to minimise reporting when faced with somatic symptom\textsuperscript{21}, and that men have been found to be less annoyed by noise than women.\textsuperscript{22} Given this, and bearing in mind that effects did not change even when controlling for the amount of tinnitus experienced, the most likely explanation is that for a man to report annoyance, he had to be more Stress Reactive to begin with, whereas a woman’s reporting of annoyance associated with tinnitus occurs irrespective of her Stress Reactivity.

The same argument may apply to Alienation, because people scoring high on the Alienation subscale must agree with statements like “Many people try to push me around”, or “I feel that life has handed me a raw deal”. In other words, given that men are less likely to complain of annoyance, the ones who do may be those who generally tend to complain or to think that life treats them unfairly.

**Strengths, limitations, and further research**—These findings were in a general population sample selected in infancy and with a very high (96%) retention rate and should thus generalise readily to any Western population. The cohort under study was aged 32 at the time of tinnitus assessment, and was thus younger than those who experience most tinnitus. It is possible that the personality factors identified here as being associated with tinnitus may differ in older people and/or those with more severe tinnitus. Future research in this cohort may contribute to this issue, as more people develop tinnitus with age. Further cross-sectional research may also contribute by comparisons between sufferers of more severe tinnitus (e.g. clinical groups) and the present findings.
Conclusion

We have shown that, in a general population sample in their early thirties, people who experience tinnitus tend to be less close to others socially, more negatively emotional, and less constrained than those who do not. That we were able to demonstrate effects of personality when using such a general measure of tinnitus suggests that the effects reported here may be expected to be present for different types of tinnitus.

We think it most likely that different personality traits affect the bias to report tinnitus present for a given internal state, and that they also influence the likelihood of tinnitus ignition as a result of noise. An unknown in tinnitus research and treatment is the determination of the relative inputs of physiology/pathology and psychology to the experience of tinnitus itself; tinnitus is difficult to study in that the boundary between the two is blurred due to the subjective, and yet apparently sensory, nature of the condition. The present findings show differences between the personality profiles of those who do and do not report tinnitus, and is a beginning to the differentiation of these two components.

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References:


Noise-induced hearing loss and tinnitus
PR Thorne

Abstract
Excessive sound exposure is a significant cause of temporary and permanent hearing loss and tinnitus. This presentation reviews the nature of the pathology of NIHL, the pathogenesis injury and, briefly, considers how this relates to tinnitus generation. Classically, injury is localised to sensory cells and primary afferent neurones innervating inner hair cells (IHC). There is increasing evidence of changes to lateral wall tissues, particularly loss of spiral ligament fibrocytes and stria vascularis atrophy, which may be a more generalised pathology than previously indicated. Changes to sensory and neural structures are correlated to loss of sensitivity and tuning, however, the consequences of the lateral wall alterations are not established but may be a deficit in potassium homeostasis and the endocochlear potential (driving potential to sensory cells).

Mechanisms of cell death include oxidative stress, glutamate excitotoxicity and initiation of apoptosis. Chronic noise generates a cochlear inflammatory response which may exacerbate injury and the relevance to tinnitus of chronic inflammation and irritation of cochlear structures, needs to be considered. Clearly many noise effects are pathological but we need to consider that many may also reflect physiological adaptations to excessive stimulation. For example, noise activation of efferent pathways is known to influence hair cell sensitivity; the ear can be conditioned to excessive sound; and there are molecular mechanisms that regulate the sensitivity of the cochlea in noise. That these mechanisms are “protective” around high level physiological sound is important and the possibility that sensitivity to NIHL may relate to the individual efficacy of these adaptive processes needs to be considered.

In terms of tinnitus, these pathological and physiological changes to the cochlea in response to loud sound temporarily and permanently alter the pattern and nature of input to the central nervous system which are manifest in changes in the Cochlear Nucleus, Inferior Colliculus and Cortex.

Tinnitus is substantially associated with noise exposure and noise-induced hearing loss\(^1\text{,}^2\) and thus the noise-induced cochlear injury is likely a significant pathology associated with the generation of tinnitus. This brief review describes the pathology and mechanisms of cochlear injury in noise-induced hearing loss, drawing predominately on animal studies.

The predominant site of injury to the auditory system from excessive noise exposure is the cochlea of the inner ear (Figure 1). The cochlea houses the auditory sensory organ, the organ of Corti containing the auditory sensory cells (inner and outer hair cells) and their primary afferent neurons (the type I and II spiral ganglion neurons, respectively). Adjacent to the organ of Corti, on the lateral wall of the cochlea, is the spiral ligament and the stria vascularis, two structures that collectively are important for the maintenance of the cochlear fluids which bath the sensory cells and neurons and provide the correct electrochemical environment for sensory cell function. Most
important is production and regulation of the endolymph, a unique extracellular fluid with a high potassium content (approximately 150mM) and large positive voltage (80-100mV compared with perilymph), which covers the apical surface of the organ of Corti. This high voltage is essential for sensory cell function as it provides the driving force for ion (potassium) flow through the hair cell transduction channels.

![Cross-section of the mouse cochlea showing the fluid spaces, scala vestibuli (SV) and scala tympani (ST) containing perilymph and scala media (SM) containing endolymph, the sensory organ the organ of Corti (OC) and the lateral wall with the spiral ligament (SL) and stria vascularis (*). Marker 1mm.](image)

**Figure 1:** Cross-section of the mouse cochlea showing the fluid spaces, scala vestibuli (SV) and scala tympani (ST) containing perilymph and scala media (SM) containing endolymph, the sensory organ the organ of Corti (OC) and the lateral wall with the spiral ligament (SL) and stria vascularis (*). Marker 1mm.

**Pathology**—Cochlear injury from noise exposure has been well identified and characterised with respect to the parameters of noise exposure and also the hearing loss. Damage or loss of sensory cells, particularly the outer hair cells (OHC) are now well established as the characteristic injury from noise exposure. Injured sensory cells may survive but with permanently distorted or fused stereocilia (sensory hairs), although loss of stereocilia appears to herald the eventual loss of the sensory cell. The vulnerability of these cells to noise injury is seen across species and the hair cell pathology is also observed in human cochleae. The primary afferent neurons, or more specifically the boutons of the type I neurons innervating inner hair cells (IHC), are also targets in noise-induced hearing loss.

Swelling and degeneration of these nerve terminals is observed after noise exposure, but no direct effect of noise exposure has been observed on the type II neurons innervating the OHC. More recently Kujawa and Liberman have described more extensive neuronal degeneration with little other cochlear injury indicating that the neural consequences of noise exposure may be more extensive. Direct injury of the supporting cells of the organ of Corti appears to be a consequence of very intense, impulse or long duration noise exposures. Often when there is injury to the supporting structures there is complete loss of the integrity of the organ of Corti.
The injury to the sensory and supporting cells is evident following the exposure and the degeneration or loss of these cells will continue for some time after the exposure has ceased. As the hair cells degenerate they are replaced by expansion of the surrounding Deiters’ and pillar supporting cells which form a “phalangeal scar” in the surface or reticular lamina of the organ of Corti.4, 6

More recently attention has centred on pathological changes in the lateral wall tissues of the cochlea as well as the fibrocytes in the spiral limbus, a region medial to the sensory cells of the organ of Corti. There is evidence of swelling and atrophy of the stria vascularis, loss of the fibrocytes within the spiral ligament and the spiral limbus.7, 9 Interestingly, all these structures are involved in the maintenance of the ionic composition of the cochlear fluids, particularly endolymph, suggesting some more generalised disturbance of ion homeostasis may occur as a consequence of the cochlear injury. Injury to the lateral wall and supporting tissues is now recognised as a more significant cochlear pathology following noise exposure than previously realised.

Changes occur in the central auditory nuclei which more likely are secondary to the peripheral injury (eg. Zhang and Kaltenbach10). However, some studies have suggested that the functional and structural alterations in the central nuclei and auditory cortex may be more extensive than can be expected by the degree of peripheral injury (eg Ryan et al.11). Cochlear injury and auditory function after noise exposure may also be modified by further noise exposure in the post-noise period.12

**Relationship to hearing loss**—There is good evidence that cochlear hair cell loss or permanent stereocilia abnormalities on surviving hair cells are correlated with the permanent hearing loss.7, 13 However, there may be some functional change without significant sensory cell loss, although this may well be correlated with subtle sensory cell injury or other changes in the cochlea, such as to lateral wall tissues and neurons.

Recent evidence8 of continuing neuronal degeneration following noise exposure but in the absence of hair cell loss and changes in auditory thresholds indicates that there may be subtle cochlear injuries that are not manifest as changes in thresholds, a routine clinical test of auditory function.

Temporary hearing loss has been shown to be correlated with reversible changes to stereocilia stiffness and swelling of the primary type I afferent neurons14, and may also correlate to structural or molecular changes to the transduction channels (tip links). Significant metabolic and molecular changes are also observed in the cochlea following acute noise exposures that may represent a responsiveness of the cochlea to noise exposure, and it is not unreasonable to assume that not all the hearing loss is going to be correlated to structural abnormalities. Indeed it is becoming more apparent that the acute hearing loss may occur without any evidence of overt structural changes and may thus represent an adaptation to the noise exposure.

**Mechanisms**—There have been substantial advances in our understanding of the mechanisms of the cochlear injury following noise exposure. Some injury to the organ of Corti, including the more subtle pathologies such as stereocilia fracture and loss, from high intensities and impulse noise exposures is a consequence of direct mechanical damage.15
However, there is good evidence that the hair cell injury from lower level and chronic exposures has a more metabolic genesis and most likely is due to oxidative stress and the excessive production of Reactive Oxygen Species (ROS) and free radicals. These free radicals are molecules with an unpaired electron and in excessive amounts interact with other stable molecules, such as proteins, DNA and lipids, to cause cellular damage and degeneration. ROS may include oxygen free radicals, such as superoxide, which may be produced in the cochlea from excessive mitochondrial activity in hair cells and changes in cochlear blood flow (see Henderson et al. for more detail). These interact with hair cell molecules to cause cellular degeneration leading to cell loss.

There is strong evidence for an increase in various ROS molecules after noise exposure and treatment of the cochlea with free radical scavengers or antioxidants or block of oxidative enzymes can reduce the amount of cochlear injury. The neuronal injury is likely the consequence of glutamate excitotoxicity. Glutamate is the main neurotransmitter at the IHC/primary afferent neuron synapse. Excessive release of glutamate during noise exposure, possibly coupled with poor clearance of glutamate in the synaptic region by surrounding supporting cells, leads to prolonged stimulation of the afferent synapse, neuronal swelling and degeneration. This effect on the afferent terminals and the subsequent hearing loss can be blocked by glutamate receptor antagonists (Gordon and Thorne unpublished observations), supporting an excitotoxic mechanism for the neuronal noise-induced injury. Oxidative stress may also be a cause or contributor to the neuronal injury.

Degeneration of cochlear tissues results from a combination of necrotic and apoptotic processes. Activation of a variety of apoptotic triggers have been identified in cochlear tissues following noise exposure and this may also be a consequence of increased intracellular calcium associated with the uptake or release in hair cells during the chronic hair cell stimulation.

The mechanism of the changes to the stria vascularis, the spiral ligament and spiral limbus have not been established. As these areas are involved in ion homeostasis, the structural and degenerative changes may be a consequence of a metabolically-induced abnormality in ion handling by these tissues. During sound stimulation there is a movement of potassium ions from endolymph, through the hair cells and into the extracellular space. This potassium is removed from the extracellular space by the epithelial supporting cells in the organ of Corti and the fibrocytes of the lateral and medial wall tissues to be recycled back to the stria vascularis.

During noise exposure there may be excessive release of potassium into the extracellular space which may not be removed adequately leading to the oedema and degeneration observed in these supporting tissues. It is true that the functional consequences of the changes in the stria vascularis and fibrocytes are not well established. However, the cochlea may cope adequately with this lateral wall degeneration whilst performing in quiet but there may be a more significant effect during physiological noise exposure if the recycling pathway for potassium and other metabolites has been impaired by the noise-induced degeneration of these supporting structures. This has yet to be established.
The cochlea also mounts a significant inflammatory response following noise exposure. Infiltration of inflammatory cells including macrophages as well as activation of inherent macrophages in the cochlea occurs in the period after acute noise exposure. Whilst this inflammatory response is likely part of the reparative processes in the cochlea following noise, it is conceivable that they may exacerbate or contribute to the ongoing injury, especially chronic injury. This needs to be further investigated.

Adaptation to noise—The response of the cochlea to noise exposure is complicated by the fact that its reaction is not purely “passive” as it clearly adapts to excessive sound exposure (eg. Housley et al.).

Exposure to a non-traumatic noise or to hypoxia provides some protection to the ear from a subsequent traumatic noise exposure (eg. Canlon and Fransson). This phenomenon, known as sound conditioning may relate to mobilisation of inherent protective processes, particularly oxidative enzymes and antioxidants. Noise exposure also upregulates the expression of a variety of proteins including protective (eg Yamamoto et al.) and repair proteins. The cochlea receives a descending or efferent innervation that arises in the Superior Olivary Complex in the brainstem and innervates both the OHC and the boutons of the type I neurons innervating IHC. Activation of these pathways appears to dampen the cochlear response to noise and hence may also provide some “protection” from noise exposure.

The role of such a variety of active reactions of the cochlea to excessive sound exposure in the human response to noise exposure has not been established or well considered. However, variations in these across the population could affect the characteristic susceptibility of individuals to noise-induced hearing loss.

Consequences for tinnitus—Clearly there are considerable pathological changes in the cochlea that are associated with noise exposure and noise-induced hearing loss that could underly the tinnitus observed after noise. The changes to the hair cells, neurons and supporting structures will lead to significant alterations in functional input to the central nervous system and alter the pattern of driven and standing activity in central auditory nuclei. Many of the functional changes in the central auditory pathways following noise exposure have been well characterised (eg cochlear nucleus, inferior colliculus and cortex) and will be discussed by others in this symposium. However, mostly these central changes have been correlated to the substantial hair cell and neuronal loss. How injury or loss of some of the supporting and accessory structures such as the lateral wall tissues are manifest centrally has not been established.

Important to the consideration of the relationship of cochlear noise-induced injury and tinnitus is that the cochlear pathologies, and indeed the adaptive reaction to noise, are very different among individuals and also show substantial variation with different noise exposures. There is an implicit assumption that the injury is of hair cell, and to some extent neuronal origin, but clearly there are many other pathologies which would not be distinguishable in humans using standard clinical functional assessment methods such as the pure tone audiogram. The evidence of neuronal injury without any change in auditory thresholds, in animals, further indicates that the pure tone audiogram will not provide a suitable index of all cochlear injury.
Injury to the cochlea after noise may also not be static as indicated by the ongoing degeneration that can occur (eg. Kujawa and Liberman\(^8\)) and the impact of a continued inflammatory reaction in the ear from chronic cochlear injury on auditory function is also not known. Thus it is important to acknowledge that the response of the cochlea to noise exposure is complicated and varies across individuals. This may account for substantial differences in the extent to which noise may influence the generation of tinnitus across individuals.

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Acoustic shock disorder (ASD)
M Westcott

Abstract
Acoustic shock disorder (ASD) is an involuntary response to a sound perceived as traumatic (usually a sudden, unexpected loud sound heard near the ear), which causes a specific and consistent pattern of neurophysiological and psychological symptoms. These include aural pain/fullness, tinnitus, hyperacusis, muffled hearing, vertigo and other unusual symptoms such as numbness or burning sensations around the ear. Typically, people describe acoustic shock as feeling like they have been stabbed or electrocuted in the ear. If symptoms persist, a range of emotional reactions including post traumatic stress disorder, anxiety and depression can develop.

Call centre staff using a telephone headset are vulnerable to ASD because of the increased likelihood of exposure, close to their ear(s), of sudden unexpected loud sounds randomly transmitted via the telephone line. In the early 1990s, coinciding with the rapid growth of call centres in Australia, increasing numbers of employees were reporting ASD symptoms. A similar pattern was being noticed overseas. As a result, a number of audiologists, scientists and occupational health experts began to research ASD.

A study of ASD symptoms in 103 call centre operators exposed to 123 acoustic incidents is reviewed. The proposed neurophysiological mechanism of ASD is discussed, in particular tonic tensor tympani syndrome (TTTS) and temporomandibular disorder (TMD). An understanding of TTTS provides insight into the neurophysiological basis of tinnitus and hyperacusis escalation, in association with high levels of emotional trauma and anxiety. Audiological assessment, diagnosis, rehabilitation and workplace management of ASD is discussed.

The potential severity and persistence of ASD symptoms have significant clinical and medico-legal implications. With the rapid growth of call centres around the world, professionals providing tinnitus and hyperacusis therapy are increasingly likely to encounter some or all of the cluster of ASD symptoms in their clients.

Acoustic shock is an involuntary response to a sound perceived as traumatic (acoustic incident), which causes a specific and consistent pattern of neurophysiological and psychological symptoms. The degree of trauma is influenced by the psychological context of the workplace and/or environment where the acoustic incident exposure occurred. Acoustic shock symptoms are usually temporary, but for some the symptoms can be persistent, escalate and result in a permanent disability. The term acoustic shock disorder (ASD) is used to identify this persistent symptom cluster.

An acoustic incident is any sound that is perceived as threatening, usually a sudden/unexpected/loud sound heard near the ear. The sound is rarely loud enough or present for long enough to cause a noise induced hearing loss. Examples include explosions, telephone faults, scream in the ear.

Background—Call centre staff using a telephone headset are vulnerable to ASD because of the increased likelihood of exposure, close to their ear(s), to an acoustic incident randomly transmitted via the telephone line. In the early 1990s, coinciding...
with the rapid growth of call centres in Australia, increasing numbers of employees were reporting acoustic shock symptoms.2 A similar pattern was being noticed overseas.3,4

In a more general clinical population, any clients who have developed tinnitus and hyperacusis, particularly following exposure to a sudden unexpected loud sound, or associated with a highly traumatic experience, may report at least some of these symptoms.5

**ASD symptoms**—ASD causes a specific and consistent pattern of neurophysiological and psychological symptoms. Initial symptoms include a severe startle reaction, often with a head and neck jerk, and a shock/trauma reaction with symptoms of disorientation, distress, shakiness, crying, headache, and/or fatigue. A severe ASD can lead to Post Traumatic Stress Disorder (PTSD). Other symptoms can include pain/blockage/pressure/typanic fluttering in the ear; pain/burning/numbness around the ear/jaw/neck; tinnitus, hyperacusis and phonophobia; mild vertigo and nausea; headache; and subjective muffled/distorted hearing. ASD generally does not result in a hearing loss, although if present it tends not to follow the typical high frequency pattern of a noise induced hearing injury but affects low and mid frequency sensorineural hearing.1,2

Typically, people describe acoustic shock as feeling like they have been stabbed or electrocuted in the ear. The symptoms are involuntary, unpleasant and frightening; they can range from mild to severe; and be of short, temporary duration or persistent. If symptoms persist, a range of emotional reactions including trauma, anxiety and depression can develop.

As ASD symptoms are subjective, they are easily misunderstood, misdiagnosed or not believed. An inadequate understanding of the symptoms often exacerbates anxiety, and can lead to confusion and distress. The long term symptoms of severe ASD are consistent with severe hyperacusis, or category 4 according to the Tinnitus Retraining Therapy (TRT) system of classification. Some of the most severe cases of hyperacusis seen in my clinic are those with ASD.

**Acoustic shock study review**—Milhinch and Doyle2 carried out the first large scale study of ASD, using data from a number of audiological clinics and from the files of a large call centre operator. A total of 103 people, including 91 females and 12 males, were exposed to 123 acoustic incidents in the period 1994 to 1999.

Pain was the most frequent symptom, reported by 95%. Of these, 81% reported ear pain, 11% pain in the neck or jaw, and 7% facial pain. Tinnitus was reported by 50%, usually accompanied by other symptoms, but in 6% it was the only symptom. Loss of balance was reported by 48%. The most distressing and durable symptom tended to be hyperacusis, reported by 32%.

Other symptoms reported included headaches (32%), facial numbness (9%), a burning feeling in the ear or face (5%), tingling (3%), a feeling of pressure or fullness in the ear (11%), an echo, or hollow feeling in the ear (18.4%) and muffled/distorted hearing (18.4%).

In some cases all symptoms resolved within a few hours or days. In other cases, symptoms persisted for months or indefinitely. In the long term, 10% developed a
range of emotional reactions including anxiety, depression, hypervigilance, anger and feelings of vulnerability.

Repeated acoustic incident exposure exacerbated an individual’s vulnerability to ASD, as well as the degree and persistence of their injury. Pre-existing stress/anxiety, as well as fear of repeated incident exposure, also appeared to increase vulnerability to ASD, with a ripple effect observed amongst other staff members.² Call centre staff are therefore particularly vulnerable: the workplaces are often large, open plan environments with high levels of ambient noise, requiring the operator to turn up the volume of their headset, increasing vulnerability to acoustic incident exposure.

Additionally, the workplace environment is potentially stressful: the job requirements are often competitive, monitored and repetitive, with the calls made frequently unwelcome.

What causes ASD symptoms?—The initial physiological symptoms of acoustic shock are considered to be a direct consequence of excessive, involuntary middle ear muscle contractions. While the stapedial reflex is an acoustic reflex triggered by high volume levels, the tensor tympani reflex is a startle reflex,³ which is exaggerated by high stress levels. The tensor tympani muscle contracts immediately preceding the sounds produced during self-vocalisation, suggesting it has an established protective function to loud sounds,⁴ assists in the discrimination of low frequency sounds,⁵ and is involved in velopharyngeal movements.⁶

Tonic tensor tympani syndrome (TTTS)—TTTS was originally described by Dr I. Klockhoff,⁹⁻¹² and has been proposed by Patuzzi, Milhinch and Doyle¹³ and Patuzzi¹⁷ as the neurophysiological mechanism causing most of the persistent ASD symptoms.

TTTS is an involuntary condition where the centrally mediated reflex threshold for tensor tympani muscle activity becomes reduced as a result of anxiety and trauma, so it is continually and rhythmically contracting and relaxing, aggravated by intolerable sound exposure.¹ This appears to initiate a cascade of physiological reactions in and around the ear, which can include: tympanic membrane flutter; alterations in ventilation of the middle ear cavity leading to a sense of blockage or fullness, as well as muffled/echoey/distorted hearing; irritation of the trigeminal nerve innervating the tensor tympani muscle, leading to frequent neuralgic pain; and symptoms consistent with temporomandibular disorder (TMD).

An exaggerated startle reflex and hypervigilance are listed as symptoms of PTSD (DSM-IV, D.5), and individuals with PTSD have been shown to produce heightened autonomic responses (e.g. increased heart rate) to acoustic stimuli that would not be expected to produce a startle response. My clinical observation of over 85 ASD clients shows that once TTTS has become established, auditory hypervigilance and an exaggerated startle reflex can lead to the escalation of hyperacusis, where the range of sounds that elicit this involuntary response increases to include more everyday sounds. These sounds become increasingly intolerable when TTTS symptoms are exacerbated following exposure. Phonophobia, headache, fatigue, anxiety, and depression can result, particularly if an inadequate explanation or diagnosis of TTTS symptoms is not offered.

A subsequent acoustic incident exposure can therefore lead to a highly enhanced startle response, so that repeated acoustic incidents significantly enhance ASD
vulnerability. The persistent pain caused by TTTS can become further exacerbated in a process of central pain sensitisation.

While acoustic shock is usually triggered by an acoustic incident, in some cases, ASD symptoms can develop as a result of cumulative exposure to sustained headset use, without a specific acoustic incident being identified, apparently as a result of triggering the established protective function of the tensor tympani muscle.

**TMD research review**—Ramirez et al.\(^{14}\) aimed to explore the anatomical and physiological connections in TMD patients with secondary aural symptoms and the central and peripheral mechanisms involved. The authors carried out an extensive peer-reviewed literature search, using data from 12, 436 patients in 49 papers, to analyse aural symptoms (otalgia, tinnitus, vertigo, subjective hearing loss and aural fullness) exacerbated by dysfunctional mouth and jaw dynamics. They proposed a range of muscular, bone communication and neural scenarios to explain this relationship, placing emphasis on tensor tympani muscle involvement and trigeminal nerve dysfunction.

According to Ramirez et al., at a peripheral level, TTTS appears to trigger a series of physiological reactions in and around the ear from tympanic membrane tension and alterations in middle ear ventilation. The tensor tympani muscle is innervated by the motor portion of the mandibular branch of the trigeminal nerve, and the authors consider that TTTS can lead to, and in an efferent pathway be caused by, an abnormal stimulation of the trigeminal nerve. This can lead to a chronic irritation of the trigeminal nerve, as well as other cranial and cervical sensory nerves of the ear and periauricular region. Central sensitisation can develop from the resultant chronic pain, leading to an expansion of the perceived peripheral pain and resulting in the typical symptoms of severe TMD.

**Discussion**

The research carried out by Ramirez et al.\(^{14}\) shows the aural symptoms associated with TMD and their neurophysiological consequences are at least partially a consequence of TTTS. These aural symptoms and the typical pattern with TMD of chronic, severe myofascial pain; numbness, tingling and burning in and around the ear; escalation and trigger point development in the neck, shoulder and arm; and central pain sensitisation are identical to those observed in my clients with severe ASD, and support the proposal that TTTS is the neurophysiological mechanism of ASD. However, ASD clients do not generally have temporomandibular joint (TMJ) dysfunction, unless it is part of a secondary escalation pattern. A hypothesis is presented that TMD can develop when TTTS is caused by an ASD, albeit with a different aetiological pathway and without TMJ dysfunction.

ASD is beginning to be recognised as a legitimate and discreet disorder, and can be readily misdiagnosed as TMD stemming from TMJ dysfunction. From a differential diagnosis perspective, TMJ dysfunction can lead to TTTS symptoms and escalate to TMD. While central pain sensitisation is common with TMD caused by TMJ dysfunction, the aural symptoms do not tend to escalate and hyperacusis is not usually present.
With ASD, TTTS is associated with hyperacusis: the symptoms are triggered or exacerbated by exposure to sound perceived as intolerable, and the primary cause is related to an anxiety/trauma response to sound. Clinically, TTTS appears to be triggered by the anticipation as well as the perception of sounds considered to be highly threatening and/or intolerable. There is little known and much to research in understanding this aetiological pathway.

Hyperacusis escalation is common with ASD, so that an increasing range of sounds become intolerable, with a corresponding escalation in TTTS symptoms, potentially leading to TMD. For this reason, a detailed history is essential in tracking the order of development and escalation of symptoms, and their relationship to acoustic incidents/headset use, prior to making a responsible and considered diagnosis of ASD.

**ASD assessment**—Rapid referral for a comprehensive audiological assessment provides reassurance, and can help control an escalation of symptoms and limit the development of hyperacusis. History taking should document immediate and persistent symptoms since the acoustic incident exposure; prior acoustic incident exposures; and prior otological and psychological history. Significant malingering is rare in ASD clients, in my experience. Most clients are bewildered, frightened or angered by their symptoms and desperate to recover.

For clients with severe ASD, listening to sounds via headphones during a hearing assessment can be highly threatening and often leads to a significant increase in symptoms, which can persist for days. I consider that frequent audiological testing should not be carried out for these clients. Suprathreshold audiological testing should be limited and loudness discomfort testing, in particular acoustic reflex testing due to the volume levels required, is contraindicated. Some ASD clients have unfortunately had their symptoms permanently exacerbated as a result of a traumatic response to acoustic reflex testing.

**Diagnosis of ASD**—On examination of the affected ear, the ear canal and tympanic membrane generally appear healthy and normal. ASD symptoms are subjective, so an experienced clinician makes a diagnosis on the basis of a thorough case history noting the pattern of symptoms; their onset, persistence and escalation; and their link with exposure to intolerable (or difficult to tolerate) sounds. If they have developed in association with acoustic incident exposure and/or hyperacusis is present, it is likely that they are a result of TTTS. The symptoms are remarkably consistent.

TTTS symptoms can be readily confused with outer/middle/inner ear pathology, and an ENT specialist opinion is required to exclude this possibility. If severe vertigo is reported, a perilymph fistula needs to be excluded.

**ASD management and rehabilitation**—A clear diagnosis and explanation of TTTS symptoms has considerable therapeutic benefit.

The most distressing and persistent ASD symptoms tend to be aural pain and hyperacusis. Sharp stabbing aural pain and numbness/burning in and around the ear are consistent with trigeminal nerve irritation. If pain levels are severe, treatment for trigeminal neuralgia, TMD and/or referral to a pain management clinic is indicated. Hyperacusis desensitisation therapy and massage of the muscular trigger points around the neck and shoulder will reduce TTTS symptoms, but progress can be slow once symptoms become entrenched.
Referral for psychological/psychiatric treatment of anxiety, depression and PTSD is indicated, as needed.

**Workplace management**—In call centres, rapid referral for ASD diagnosis and management can help control a ripple effect in other staff.

With the identification of ASD, output limiters in headset equipment have been developed to restrict maximum volume levels transmitted down a telephone line. However, ASD continues to occur despite their use. In my opinion, they are of benefit primarily to help reduce the probability of an initial acoustic incident exposure. The dominant factors of an acoustic incident leading to ASD appear related to the sudden onset, unexpectedness and impact quality of loudish sounds outside the person’s control near to the ear(s), rather than to high volume levels alone. If TTTS develops, because of the vulnerability of further escalation to acoustic incidents at lower volume levels, it is impossible to give a 100% guarantee of protection.

With severe ASD, TTTS symptoms can be involuntarily aggravated by the mere placement of a headset over the ears in the workplace. I consider an ASD client should not return to headset or telephone duties on either ear until the symptoms have fully resolved. A graded return to work can then be carried out with handset use initially on the opposite ear.

**Conclusion**

The potential severity and persistence of ASD symptoms have significant clinical and medico-legal implications. With the rapid growth of call centres around the world, professionals providing tinnitus and hyperacusis therapy, as well as general practitioners, ENT specialists, occupational physicians, TMD specialists, neurologists and trauma psychologists/psychiatrists, are increasingly likely to encounter some or all of the cluster of ASD symptoms in their clients.

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Animal models of tinnitus

CL Darlington, PF Smith, Y Zheng

Abstract

Animal models of disorders are typically used to investigate the mechanisms of pathology and to determine potential treatments that can be tested in human clinical trials. In the case of tinnitus, the development of an animal model presents unusual challenges because it is not possible to directly measure the perception of a phantom sound. In the late 1980s, Jastreboff and colleagues developed an animal model based on a conditioned behaviour that was related to the experience of tinnitus in rats, which could be induced either by the administration of salicylate or acoustic trauma. This model, and modifications of it, have provided a means to investigate the mechanisms of tinnitus in animals and to test potential new drug therapies, although relatively little attention has been paid to the latter. Although the injection of salicylate offers an easy way of inducing tinnitus in animals such as rats, it must be remembered that this form of tinnitus is relatively uncommon in humans. Furthermore, salicylate can interact with drugs that are being tested to treat tinnitus. Therefore, unilateral acoustic trauma is probably a better model for inducing tinnitus in animals.

Animal models of human neurological disorders are a standard way of investigating their mechanisms and testing potential treatments. The development of drug treatments in particular tends to rely on animal models, because it is cheaper and safer to test new drugs in animals before testing them in humans. Subjective tinnitus, an auditory disorder in which a person perceives an auditory sensation that does not actually exist, presents a special challenge for the development of an animal model, because it is difficult to determine what an animal, such as a rat, is experiencing. Two major issues for animal models of tinnitus are how to induce tinnitus in a way that is representative of the condition in humans, and how to determine that an animal is experiencing tinnitus. Historically, tinnitus has been induced in animals either by exposure to loud noise (i.e. acoustic trauma) or by injecting them with sodium salicylate. Most studies suggest that acoustic trauma is the leading cause of tinnitus (23.6%), followed by head and neck injuries (17.7%), and that tinnitus resulting from drug exposure is relatively rare (3.4%). Therefore, it would seem that acoustic trauma would also be the best way to induce tinnitus in animals. However, salicylate injections are easy to administer and have been demonstrated to reliably induce what appears to be tinnitus in animals. Therefore, the salicylate model has been used in many animal studies, despite the fact that it may be a less realistic representation of human tinnitus.

The second major issue – how to determine whether an animal is experiencing tinnitus – was confronted by Jastreboff and colleagues in the late 1980s. They were aware from experimental psychology of the powerful relationship between an unconditioned stimulus (UCS) and a conditioned stimulus (CS), and reasoned that if some behaviour related to tinnitus could be controlled by a CS, then it would be possible to use the response to the CS as an indication of whether the animal is experiencing tinnitus. This turned out to be a remarkably powerful idea. Using the simple fact that rats who are water deprived tend to lick at a water spout, they trained...
the rats to associate a mild electric shock delivered through the floor of the chamber (the UCS), with the offset of a high frequency tone resembling tinnitus (the CS; see Figure 1).

Figure 1: Schematic diagram of the experimental setup showing the conditioning chamber where the animal learns the association between the foot shock (UCS) and the offset of the high frequency tone (CS). The water contains sucrose so that the rat is more interested in drinking and therefore does not have to be water deprived to the same extent. When the rat drinks, it breaks a photobeam and the computer logs the animal’s drinking activity. A camera is used inside the chamber to monitor the animal’s activity. During training, a mild footshock is paired with the offset of a high frequency tone delivered through the speakers. After a number of repetitions of this association, extinction begins. The animal still associates the CS with the UCS; therefore, whenever the tone stops the animal stops drinking. However, animals with tinnitus cannot hear the tone offset, and therefore keep drinking.
The rats quickly learned to connect the CS with the UCS, and therefore stopped drinking as soon as the CS occurred. Once the animals had been trained to do this, the UCS was no longer delivered (the ‘extinction phase’ had begun) but because of the powerful effects of Pavlovian conditioning, the rats continued to stop drinking each time the CS was presented. However, animals in whom tinnitus had been induced by an injection of sodium salicylate could not hear the offset of the tone, because it was masked by the tinnitus, and therefore they persisted in drinking during the CS. The comparison of drinking at the water spout, which could be measured automatically, indicated which animals were experiencing tinnitus.

This basic model for determining whether an animal has tinnitus has been modified many times (see Figure 2), and is now used in combination with acoustic trauma to induce tinnitus and auditory brainstem response measurements to quantify hearing thresholds.

![Figure 2: Example of the difference in suppression ratios (SRs) between rats treated with salicylate to induce tinnitus, and rats treated with the vehicle. On the training day, before drug treatment, the SRs are similar. However, on extinction day 1, the salicylate (SA)-treated rats show a significantly higher SR than the vehicle-treated rats. This effect disappears on extinction day 2 because the effects of the salicylate do not last. The SR is calculated as: SR = (Mean time drinking during off-tone) / (Mean time drinking during off-tone + Mean time drinking during tone). Symbols represent means ± 1 SD.](#)

**Figure 2:** Example of the difference in suppression ratios (SRs) between rats treated with salicylate to induce tinnitus, and rats treated with the vehicle. On the training day, before drug treatment, the SRs are similar. However, on extinction day 1, the salicylate (SA)-treated rats show a significantly higher SR than the vehicle-treated rats. This effect disappears on extinction day 2 because the effects of the salicylate do not last. The SR is calculated as: 

$$SR = \frac{\text{Mean time drinking during off-tone}}{\text{Mean time drinking during off-tone} + \text{Mean time drinking during tone}}$$

Symbols represent means ± 1 SD.

**Modifications to the animal model**—There are several reasons to feel that the salicylate model for inducing tinnitus is not the preferred model. Aside from the fact that salicylate-induced tinnitus is relatively rare in humans, its effects in the brain are somewhat different to those caused by acoustic trauma. Whereas acoustic trauma has been associated with neuronal hyperactivity in auditory areas of the brain, such as the
dorsal cochlear nucleus and the auditory cortex, such hyperactivity has only been reported in the inferior colliculus following salicylate administration. Furthermore, salicylate has been shown to have direct effects on the brain in some cases and it is possible that it produces tinnitus by directly affecting auditory neurons in the inferior colliculus.

Salicylate can also interact with some drugs such as baclofen (Zheng, Smith and Darlington, unpublished observations), making it impossible to test them in a conditioned behavioural suppression paradigm. Finally, although salicylate injections have been used to model chronic tinnitus, acoustic trauma produces tinnitus that is long lasting and therefore it much more closely resembles tinnitus in humans.

In the most recent animal models of tinnitus, anesthetized animals (usually rats) have been exposed to unilateral acoustic trauma delivered using a speculum that is inserted into the external auditory canal. The stimulus is often a narrow band noise with a peak intensity of 105 dB centred at 16 kHz for 1 h. Acoustically evoked auditory brainstem responses (ABRs) to clicks and tone bursts are measured before and immediately after the noise stimulus, in order to confirm that the hearing threshold (in dB sound pressure level (SPL)) has increased in the affected ear.

A conditioned behavioural paradigm is then used to confirm whether the animal is experiencing tinnitus. This behaviour does not have to be licking at a water spout, for example, it can be lever pressing. The animal is then tested with a variety of auditory stimuli and it can be shown that the suppression of behaviour is specific to the frequency range of the acoustic stimulus that was used to deliver the noise trauma. This proves that the effect is frequency-specific.

Applications of animal models of tinnitus—Although animal models of tinnitus can be used to investigate the neural mechanisms of tinnitus in order to devise rational therapies, one of the advantages of a reliable animal model is that it can be used to investigate potential drug treatments even if their exact mechanism of action is not yet understood. For example, we have investigated the effects of the anti-epileptic drug, carbamazepine, on tinnitus-related behaviour in rats and found that it does suppress tinnitus in a dose-dependent fashion (Figures 3 and 4). Using a similar model, the anti-epileptic drug, vigabatrin, has also been shown to reduce tinnitus-related behaviour in rats. The system we use currently is entirely automated and controlled by a computer. Therefore, it is possible to test new drugs quickly and cheaply in order to screen candidates for clinical trials in humans.

Animal models are critical for investigating the mechanisms of human neurological disorders. Using animal models of tinnitus, we have shown that tinnitus is associated with a down-regulation of cannabinoid CB1 receptors in the ventral cochlear nucleus (Figure 5). Since CB1 receptors are thought to serve an anti-epileptic function in the CNS, it is conceivable that this decrease in CB1 receptors contributes to epileptiform activity that underlies tinnitus. We are currently testing cannabinoid receptor agonists to determine whether they might have some efficacy against tinnitus.
Figure 3: Increased SRs following salicylate administration on extinction day 1 for the animals to be used in the carbamazepine study. Symbols represent means ± 1 SD

Figure 4: Carbamazepine, at a dose of 15 mg/kg, but not 5 or 30 mg/kg, significantly reduces the SR, indicative of suppressing tinnitus, following salicylate administration. Bars represent means ± 1 SD
Figure 5: Salicylate-induced tinnitus in rats is associated with a significant down-regulation of cannabinoid CB1 receptors in the ventral cochlear nucleus. Bars represent means ± 1 SE

Conclusions

Animal models of neurological disorders are important for the investigation of pathological mechanisms and therefore for the development of novel therapies. Although subjective tinnitus cannot be perceived by the experimenter, the application of Pavlovian conditioning has led to the development of reliable animal models, which are best used in combination with the acoustic trauma method of inducing tinnitus in animals such as rats. Together with the measurement of ABRs and a sophisticated computerized system for measuring an animal’s responses to specific frequencies of sound, it is possible to confirm that an animal has tinnitus and to use this animal model to test new drug treatments.

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Neural synchrony and neural plasticity in tinnitus
LE Roberts, DJ Bosnyak

Abstract
Current research suggests that tinnitus is generated by synchronous neural activity that develops in regions of the auditory cortex that receive diminished input from the ear, owing to hearing loss caused by otological disease, noise exposure, or the aging process. In this paper we summarize this research which comes from animal experiments, psychoacoustic studies of tinnitus, and functional brain imaging of tinnitus patients. We also discuss the role of neural plasticity in tinnitus and consider implications for the treatment of tinnitus by sensory training. The neural synchrony hypothesis provides a framework that can incorporate some elements of earlier models while explaining some phenomena beyond their reach.

In 1995, Jastreboff \(^1\) proposed a comprehensive model of tinnitus that addressed three clinically prominent features of this condition. These were (1) the tinnitus sensation itself, generated by pathology in the inner ear; (2) the ability of the tinnitus sensation to command attention; and (3) the patient’s disturbing emotional reaction to the tinnitus percept. Jastreboff suggested that although elimination of the tinnitus sensation by treatment of cochlear pathology was in most cases not practical, the latter two features of tinnitus were likely modifiable and if treated would benefit the tinnitus patient.

Tinnitus Retraining Therapy (TRT) was devised to foster extinction of attentional and emotional responses by presenting low-level tinnitus-like external sounds that could be filtered out along with the tinnitus by perceptual mechanisms. Studies of TRT and clinical experience have confirmed that emotional responses diminish with time for most tinnitus sufferers, as does the extent to which tinnitus sufferers attend to their tinnitus percept.\(^2\) These are important and practical goals for tinnitus sufferers which bring substantial clinical benefits. Attempts to eliminate the tinnitus sensation, however, have met with less success.

Since Jastreboff’s seminal work much has been learned about the neural basis of tinnitus from research in animal models and psychoacoustic studies of tinnitus in human tinnitus sufferers. Evidence from these studies has converged to give a better understanding of how the sensation of tinnitus may be generated when hearing loss is present, as it is in the large majority of cases. In this paper we briefly outline this evidence and consider its implications for attempts to modify tinnitus sensations through sensory training.

The Neural Synchrony Model of Tinnitus—It is widely recognized that most cases of tinnitus are associated with sensorineural hearing loss caused by injury, otological disease, noise exposure, or the aging process. Even when auditory thresholds are in the normal range (\(\leq 25\) dB HL), tinnitus sufferers give evidence of restricted cochlear dead regions\(^3\) or show threshold elevations in the audiogram on the order of \(\sim 10\) dB in the tinnitus frequency range compared to age matched controls.\(^4\) It is doubtful, however, that tinnitus is generated by irritative processes that persist in the cochlea damaged by hearing loss. Damage to the cochlea caused by lesioning or noise
exposure typically leads, not to an increase in spontaneous activity in auditory nerve fibres, which might be expected from such processes, but rather to a decrease in auditory nerve activity pointing to a reduction of input to central auditory structures. These results suggest that the sensation of tinnitus is generated in most cases not in the ear but by changes that take place in central auditory pathways when the brain is disconnected from the ear by hearing loss. Consistent with this understanding, most pre-existing cases of tinnitus persist after sectioning of the auditory nerve, and tinnitus is a predictable outcome in previously unaffected patients when cranial nerves are sectioned by the removal of acoustic neuromas.

Animal models of hearing loss have begun to give a picture of the changes that occur in central auditory pathways following auditory deafferentation. The understanding supported by these studies is summarized in Figure 1a (from Eggermont and Roberts), which depicts the primary auditory cortex of a cat that has sustained a high frequency hearing loss owing to noise trauma.

The left side of the figure shows the undamaged region including thalamocortical afferents synapsing on input neurons followed by feedforward (i) and lateral (ii) inhibition after one synaptic delay. Feedforward inhibition is functionally dissociable from lateral inhibition and quenches target neurons after their depolarization, which may protect thalamocortical synapses from downregulation when the neuron is driven by uncorrelated inputs from horizontal fibres in the tonotopic map.

Animal studies have shown that when a region of the tonotopic map is disconnected from the ear by cochlear damage (right side of Figure 1a), auditory neurons in the affected region begin to respond preferentially to inputs conveyed by horizontal fibers as their thalamocortical inputs are impaired or lost. As a consequence, the cortical tonotopic map “reorganizes” when the affected neurons begin to express the tuning preference of their neighbours, leading to an over-representation of edge frequencies in the tonotopic gradient (an example is shown in Figure 1b).

It has been proposed that this over-representation of edge frequencies may correspond to the tinnitus percept, which was thought to be confined to the edge of normal hearing. However, this is doubtful not only because of evidence to be presented below, but also because it is not obvious how the activity of the affected neurons would be heard in terms other than their original cochleotopic tuning.

Other changes in the response properties of neurons documented by animal studies of hearing loss are more likely to contribute to the tinnitus percept. One such change is that neurons in cortical and subcortical auditory structures (but not auditory nerve fibers) increase their spontaneous firing rates as inhibition is withdrawn. At the level of the cortex, this effect has been observed to occur across the tonotopic map, including tonotopic regions that are affected by hearing loss (typically high frequency regions) as well as regions that are less affected (typically low frequency regions). Increased spontaneous activity likely contributes to tinnitus, although uncorrelated neural activity may not be sufficient to generate a coherent sound percept. A second change is an increase in the synchronous activity of neurons, which is expressed as an increase in cross-correlated neural firing compared to control animals. This change is more strictly confined to the hearing loss region and appears to reflect synchronous network activity that is forged by correlated inputs and neuroplastic mechanisms in this region, possibly because the quenching effect of feedforward inhibition is lost.
It should be noted that although thalamocortical input to the affected region is damaged by cochlear injury, the output of the synchronously active neurons remains intact. The neural synchrony model of tinnitus proposes that this output (which is conveyed by nerve fibers more numerous than the forward path) is processed by other brain regions and generates the tinnitus percept.

Figure 1: Central effects of hearing loss in the cat. A. Tonotopic map of primary auditory cortex depicting intact thalamocortical input to neurons in a low frequency region (left) and diminished thalamocortical input to a high frequency region affected by hearing loss (right). Neurons in the damaged region begin to express the tuning of their unaffected neighbours via horizontal fibers when their thalamocortical input is lost. Feedforward (i) and lateral (ii) inhibition is depicted in the intact low frequency region. Graphic from Eggermont and Roberts (with permission). B: Tonotopic representation in a normal cat (solid line) and in a cat with high frequency hearing loss induced by noise trauma (open circles). The abscissa is transcortical distance from a reference point near the apex of the basilar membrane. An over-representation of edge frequencies is seen in the hearing impaired cat. Data from Rajan and Irvine (with permission).
This picture of the neural mechanism of tinnitus has implications for the psychoacoustic properties of tinnitus. One implication is that when subjects are asked to rate sounds of different frequencies for similarly to their tinnitus, ratings should not be restricted to the region of the audiometric edge, but should instead span the region of hearing loss, increasing in proportion to the depth of hearing impairment. This result should be obtained for cases of tonal tinnitus as well as tinnitus with wider bandwidths, because audiometric function is similar among these tinnitus types.

Independent studies by laboratories in France, Canada, and New Zealand have confirmed this prediction (see Figure 2). The results support the view of Fowler who suggested that tinnitus consists of a band of frequencies (called here the tinnitus “spectrum”) and not a single pitch even in tonal cases, although the bandwidth in tonal cases may be wider than Fowler thought.

**Figure 2:** Relation of the tinnitus spectrum (Likeness Rating) and the residual inhibition function (RI Depth) to hearing loss in bilateral tinnitus (n = 59 cases). To obtain the tinnitus spectrum, subjects rated the pitch of each of 11 sounds for their likeness to their tinnitus. A rating exceeding 40 corresponded to a sound that was beginning to resemble the tinnitus. Likeness ratings diminished at 12 kHz, likely because these sounds were not well matched for loudness owing to the depth of hearing loss at this frequency. RI was measured following presentation of band-limited noise maskers differing in centre frequency (band pass ±15 % of centre frequency). A rating of -5 corresponded to “tinnitus gone”. From Roberts et al.
A further implication of the neural synchrony model is that post-masking suppression of tinnitus by band-limited noise maskers (called “residual inhibition” or RI in the tinnitus literature) should increase proportionately as the centre frequency of the masking sound enters the tinnitus frequency region. This is because these masking sounds (which are presented at intensities exceeding the hearing threshold and the tinnitus sound) should re-inject feedforward inhibition into the affected regions of the cortical tonotopic map, temporarily disrupting the synchronous activity underlying tinnitus and weakening the tinnitus percept. This prediction has also been confirmed (Figure 2).

It should be noted that RI does not appear to be caused by habituation of the affected neurons to frequencies contained in the masker. On the contrary, these neurons are actually more easily driven by amplitude-modulated sounds presented to the tinnitus frequency region during RI than during tinnitus, possibly because their capture by synchronous network in tinnitus has been disrupted (Figure 3b).

**Figure 3:** Electromagnetic correlates of residual inhibition (RI). Results from a single subject are shown (MEG recording). **A:** Psychoacoustic data (audiogram and the corresponding RI function for this subject). A band-limited (±15% of CF) masker centred at 4 kHz in the audiometric notch region corresponded to the tinnitus sensation and gave good RI in this subject. This masker was used to induce RI in panel **B.** **B:** The brain response
evoked by a 4 kHz 40-Hz amplitude modulated tone (duration 0.5 sec) is shown after 30 seconds of masking when the subject was experiencing RI (top right panel) and when experiencing tinnitus (top left panel, masker switched off). This brain response (called the 40-Hz auditory steady-state response or ASSR) localizes tonotopically to the region of primary auditory cortex and gives a picture of neural activity in this region (the 4 kHz region in this recording). The ASSR is larger in RI compared to tinnitus (Roberts, Weisz, Wienbruch and Bosnyak, 2001; unpublished data). Subsequent research using EEG found that enhancement of the ASSR after masking is specific to tinnitus subjects (N = 14, p = 0.0058) and is not seen in age matched controls (N = 14, p = 0.99). The lower panels show that, unlike the ASSR, the N1 evoked response (which localizes to secondary auditory cortex) adapted after masking (p = 0.007).37

Other brain imaging results that support the neural synchrony model include evidence for (1) a degraded frequency (tonotopic) representation above ~ 2 kHz in the auditory core region of tinnitus patients compared to controls15 (this reorganization resembling that seen in animal models of hearing loss), and (2) increased spontaneous oscillatory brain activity in tinnitus sufferers.16 The latter effect that tracks the laterality of the tinnitus percept and may reflect augmented network underlying this condition.

As described here the neural synchrony model accords an important role to the primary auditory cortex (A1) in the generation of tinnitus percepts. However, neuron response properties including increased spontaneous activity and map reorganization are also altered by hearing loss in subcortical auditory structures,17-18 although neural synchrony in these regions has not yet been studied. Changes occurring in subcortical structures could be projected to A1 and determine some of the effects seen there, as well as some distinct properties of tinnitus including its modulation by somatosensory inputs.19

Alternatively, some of the changes seen in subcortical nuclei could be sculpted by returning output from the auditory cortex, which may recruit a brain network supporting tinnitus percepts and their attentional and emotional consequences.

Functional brain imaging studies have implicated several brain areas in this network20-23 including frontal and limbic areas that may subserve respectively the attentional and emotional aspects of tinnitus described by Jastreboff.

Neuroplastic Remodeling in Tinnitus—A feature common to the neural synchrony model and the wider framework of Jastreboff is a role for neural plasticity in the generation of tinnitus percepts. Although direct evidence is lacking and not easily procured, there are compelling reasons to propose a role for such mechanisms in tinnitus. Spike-timing dependent plasticity24 appears to be general property of cortical neurons, and inhibitory deficits consequent on hearing impairment with their attendant increase in spontaneous firing rates would be expected to facilitate the formation of synchronous networks forged by this mechanism.

Synchronous activity appears to be expressed over cortical distances that exceed those expected from thalamocortical radiations, which implicates temporal coincidence mediated by horizontal fibers as a driving mechanism.10 From the limited data available, it appears that cross-correlated activity develops within hours of hearing loss and grows over time,9 although the limit of this growth is not known. Neural
plasticity has the potential to explain the variability that is seen in tinnitus percepts among affected individuals, with the addition of no new principles. A role for plasticity is also proposed by sound therapies that aim to reverse the neural alterations responsible for tinnitus sensations.

In the last 15 years, much has been learned about how neural plasticity remodels auditory representations in the normal hearing subject. Experience with sound has a profound effect on tonotopic organization and the tuning properties of auditory neurons in the developing brain and after maturity as well. Neural modelling during development appears to be driven largely by the spectrotemporal statistics of the acoustic input, such that neural representations become tuned to the sounds that are present in the animal’s environment.

After maturity, top-down mechanisms begin to play a role, preferentially gating neural plasticity in the auditory cortex for sounds that are important for behavioural goals. However, passive immersion in a distinctive sound environment can still have profound effects on neuron response properties and neural organization in the adult brain, which appear to reflect at least in part changes in subcortical auditory nuclei that are driven unselectively by stimulus input. These broad principles derived from animal studies appear to be applicable to humans as well, although much remains to be discovered about the specific rules that guide remodelling in both domains and the mechanisms that underlie them.

Can these principles be used to devise training procedures that will dampen or eliminate tinnitus? The answer is likely to depend on at least two considerations. First, the principles that describe remodelling in the normal hearing subject need to apply as well to the tinnitus brain. This cannot be assumed, not only because most people with tinnitus have some degree of hearing impairment, but also because the neural mechanisms that underlie tinnitus (whether neural synchrony or some other process) may impede auditory remodelling.

Second, the neurons affected by auditory training in tinnitus subjects must interact in some way with the neurons that are involved in the generation of tinnitus. In this regard the neural synchrony model suggests that the target of auditory training should be to disrupt or “segregate” synchronous neural network activity that underlies tinnitus. Masking sounds that induce RI may achieve this goal by re-injecting feedforward inhibition into the tinnitus frequency region, but the effect is fleeting.

Early results from research relevant to these two considerations are summarized in Figure 4. Figure 4a shows the electrical brain response (EEG) evoked by a 2 kHz pure tone amplitude modulated at 40 Hz. This response (known as the 40-Hz auditory “steady state response” or ASSR, also shown in the upper panels of Figure 3a, localizes tonotopically to A1 and can give a picture of changes occurring in or projecting to this region during auditory training. In Figure 4b, the ASSR is represented by two compass plots in which each arrow reports the response at one of the 128 scalp electrodes (arrow length gives ASSR amplitude, and arrow angle the timing of the response waveform with respect to the stimulus waveform, called ASSR phase).
Comparison of the two compass plots shows that auditory training (7 sessions, to detect single 40-Hz AM pulses of enhanced amplitude) advanced ASSR phase toward the stimulus waveform by 23 degrees, and increased ASSR amplitude. We have found in companion studies \(^3\) that the phase advance (which is statistically robust, unlike the less reliable amplitude enhancement) consolidates within 24-72 hrs of the initial training session, increases progressively with training, does not require attention, and correlates with behavioural performance. However, these effects on ASSR phase appear to be diminished in tinnitus.

While a group of age-matched controls showed the expected phase advance when trained on a 5 kHz 40-Hz AM sound (n = 11, \( p < 0.001 \)), only two of 8 tinnitus subjects did so, resulting in a non-significant group effect overall (\( p = 0.44 \)). These findings suggest that synchronous neural activity underlying tinnitus may obstruct or reset training effects in the region A1 in tinnitus subjects (5 kHz was chosen for study because it is in the tinnitus frequency range).

**Figure 4:** Effects of auditory training on auditory evoked potentials. A: Response evoked by a 2 kHz tone amplitude modulated at 40 Hz (ASSR). The stimulus waveform and the response waveform recorded at electrode Cz are shown, together with the bipolar scalp topography (128 sensors). In inverse modelling, the cortical generators for an ASSR evoked by a carrier frequency of 4100 Hz (AM at 40 Hz) localize medial to those for an ASSR evoked by a carrier frequency of 250 Hz, in the region of A1.\(^{15,33}\) B: Compass plots of the
ASSR before (left panel) and after (right panel) seven sessions of acoustic training. Normal hearing subjects without tinnitus (n = 9) were trained to detect a single 40-Hz AM pulse of enhanced amplitude in a stimulus of 1 sec duration (carrier frequency 2 kHz). A phase shift (advance of the response waveform toward the stimulus waveform by 23 degrees, \( p < 0.001 \)) and an amplitude enhancement (not significant in this study) were observed. The phase advance (a more consistent effect than the amplitude enhancement) is expressed in A1 but may reflect changes of subcortical origin that are present in the thalamic input to this cortical region. The phase shift appears to be largely absent in tinnitus subjects (see text for details).

C: The P2 transient evoked response (latency ~180 ms) increases with training in normal hearing controls and in tinnitus subjects, suggesting normal remodelling expressed in the region of secondary auditory cortex (A2) in tinnitus subjects. The P2 was measured while subjects performed the task (active condition) and when they listened passively to the 40-Hz AM sounds (passive condition).

Ongoing research is determining what distinguishes tinnitus subjects who remodel at 5 kHz from those who do not, and whether subjects who do remodel show an altered tinnitus percept at 5 kHz. The latter could happen, if the neurons that are recruited by training to represent a 5 kHz sound are removed from participation in the synchronous networks that generate tinnitus. A further objective of ongoing research is to determine whether tinnitus subjects will show a normal phase advance when trained on a sound below the tinnitus frequency region, where neural synchrony is believed to be minimal.

It should be noted that the procedure described here represents only one of many approaches that could be taken to auditory training for tinnitus. Other approaches include augmenting cortical representations in the frequency range of normal hearing by training, which may distribute desynchronizing inputs into the tinnitus region via horizontal connections, or distributing lateral inhibition into the tinnitus frequency region by training at the audiometric edge.

It is noteworthy that while the tinnitus and control groups described above differed with regard to the effect of training on ASSR phase, both groups showed an enhanced P2 auditory evoked potential (Figure 4c). This neuroplastic response localizes to generators in secondary auditory cortex (A2) and could distribute reentrant feedback into tinnitus frequency regions, disrupting activity in these regions.

**Conclusion**

Several lines of research suggest that tinnitus is generated by abnormal synchronous activity that develops in tonotopic regions of A1 that have been deprived of input from the ear by hearing loss, and that neural plasticity is involved. Whether this synchronous activity can be segregated by auditory training remains to be determined. While there are principled bases on which to explore this question, it is nevertheless prudent given the resilience of tinnitus to aim for modest goals.

The research we have described evaluates whether auditory training works the same way in tinnitus subjects as it does in the normal hearing brain, and if so, whether training will modify tinnitus percepts at the specific frequencies that are trained.

Auditory training likely involves elements common to other therapeutic approaches that may contribute to clinical improvement, regardless of its direct effect on tinnitus.
sounds. Examples are: discussion with informed and sympathetic staff; knowledge about the causes of tinnitus and its course; reassurance; and investment in a therapeutic process, all of which may foster a positive attitude and give reason for hope. These elements may contribute to reductions in distress behavior and tinnitus intrusiveness that have been reported for widely different treatments including Neuromonics, Tinnitus Retraining Therapy, and transcranial magnetic stimulation. It would of course be desirable if auditory training conferred the additional benefit of attenuating tinnitus sounds.

Research on this question has the potential to inform us about mechanisms of tinnitus as well as about how auditory training modifies representations for sound in the human brain.

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Similarities between chronic pain and tinnitus: what we’ve learned from chronic pain and how it applies to tinnitus

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Abstract

Chronic conditions such as tinnitus and pain share many common elements including the role of physiological, neurochemical, psychological and social elements in their occurrence and perpetuation, as well as the impact they have on the tinnitus/pain in a patient’s life. While symptoms following an injury to the inner ear (tinnitus) or to tissue (chronic pain) usually occur for the duration of the injury and resolve once the damage has healed, the fact that the sensations produced by these sensory systems can continue long after the initial injury has resolved can be very distressing to those experiencing persistent symptoms.

For tinnitus patients, hearing sound when there is no source generating this sound can be confusing, frustrating and upsetting. Similarly, those who experience on-going pain from an injury that has healed or from an area of the body that is no longer there (e.g. phantom pain) can become greatly distressed, anxious or depressed due to the persistent presence of these symptoms. What causes and leads to the perpetuation of symptoms experienced by tinnitus and chronic pain patients are changes to the processing of information within the central nervous system (CNS; i.e. CNS plasticity). When processing within the CNS becomes maladaptive, the usually beneficial effects of sensory processing can become distorted and dysfunctional as seen with changes in the auditory nervous system (i.e. tinnitus) and somatosensory system (i.e. chronic pain).

Similarities between tinnitus and chronic pain can be seen in the physiological and psychological factors that mediate and perpetuate these conditions as well as in the ways they impact those suffering from the effects of chronic symptoms. What has been learned in the study of chronic pain, and chronic pain phenomena such as phantom limb pain, is providing insight into the causes of tinnitus as well as valuable guidelines for the treatment of this often distressing condition.

There are many similarities between chronic pain and tinnitus in terms of the mechanisms that cause these conditions and the role that the person’s perception and interpretation of sensory information has on their subjective experiences. Because a person’s perception of sensory inputs is shaped by their ability to identify, interpret, and attach meaning to the sensation, for those experiencing tinnitus and chronic pain, a great deal of distress can be caused by the difficulty in understanding the purpose of these ongoing sensations. For example, people can become greatly distressed when they have had an injury that has healed but they continue to experience pain or continue to experience pain in a limb that has been amputated. Similarly, the experience of noise in the absence of something producing those sounds can be distressing.

Conditions such as chronic pain and tinnitus are complex in their presentation because the symptoms experienced usually occur in the absence of obvious or directly observable causes. A great deal has been learned in the study of chronic pain and its
related phenomena in terms of the mechanisms that contribute to the cause and maintenance of this condition that can be applied to other chronic conditions such as tinnitus.

Pain—Pain is considered to be the perception of an aversive or unpleasant sensation that originates from a specific region of the body.¹ The study and treatment of pain originated in the traditional medical model wherein pain was the result of injury and therefore attributable to tissue damage. As can be seen in the early attempts to define pain, a causal link between tissue damage and pain was expected and so the definitions were circular in their descriptions. For example, Mountcastle defined pain as “that sensory experience evoked by stimuli that injure or threaten to destroy tissue, defined introspectively by every man as that which hurts.”² Sternbach defined pain as “an abstract concept that refers to 1) a personal private sensation of hurt, 2) a harmful stimulus that signals current or impending tissue damage; and 3) a pattern of responses which operate to protect the organism from harm.”³

What the traditional medical model and early definitions were unable to account for was the complexity of pain and such phenomena as chronic pain and phantom limb pain, as these involved pain that occurred after an injury had healed or without a directly attributable ongoing injury.

The definition by Merskey comes closer to recognizing the complex association between injury and pain as it defines pain as “an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage.”⁴ This definition recognizes the emotional and sensory dimensions of pain⁵ and it considers not only the instances where there is an observable cause to the pain but also those instances where this is not as clear.

Physiologically, pain can be experienced as a result of damage to peripheral tissue detected by nociceptors found throughout the body. Their function is that of an early warning system that signals the occurrence, or potential occurrence, of damage to tissue. These sensors are usually not active unless stimulated by noxious stimuli. Once activated, nociceptors convey the information pertaining to the injured site to the CNS, resulting in the first component of the experience of pain.

The second component of pain involves the processing of this information in relation to the perceptual, emotional and cognitive components of pain. The multidimensional nature of this processing is evidenced by three major dimensions of pain: sensory-discriminative, motivational-affective, and cognitive-evaluative,⁶ which are subserved by physiologically specialized and interacting systems in the brain.

The sensory-discriminative dimension is influenced by the rapidly conducting spinal systems. The powerful motivational drive and unpleasant affect characteristic of pain are subserved by activities in reticular and limbic structures. The latter are influenced primarily by the slowly conducting spinal systems and CNS processes which evaluate the input in terms of past experience, exerting control over activity in both the discriminative and motivational systems.⁶

When pain becomes chronic—Due to the complexity of pain, the many factors that contribute to it are susceptible to breaking down. When such a disruption occurs the invaluable function that pain serves of maintaining an organism may go beyond its beneficent role causing the process to become destructive, resulting in a pathological
chronic pain state with accompanying emotionality. For example, following peripheral tissue or nerve damage, pathologies may develop in which there may be a reduction in pain threshold resulting in pain produced by a stimulus that does not normally provoke pain (allodynia), an increased response to noxious stimuli (hyperalgesia), an increase in the duration of response to brief stimulation (persistent pain), or a spread of pain and hyperalgesia to uninjured tissue (referred and secondary hyperalgesia). The distressing feature of these pathologies is that they persist long after the injured peripheral tissue has healed and therefore serve no valuable function.

Once damaged tissue has healed, the nociceptors that previously conveyed the sensory information regarding the tissue damage would no longer be activated and would therefore no longer convey information regarding pain from the injury. The persistence of pathological pain after the damaged tissue has healed therefore suggests that such pain involves more than mere sensation by nociceptors and that there are changes and alterations in CNS functioning.

These changes in turn influence subsequent somatosensory inputs and are referred to as CNS plasticity, as they are indicative of the nervous system’s ability to be adaptive and changeable. It has been proposed that the role of CNS plasticity in pain involves alterations that result in the development of memories contributing to pathological pain processes. These “pain memories” occur following an injury through the relaying of that injury’s information to the brain via afferent nerve fibres and a response from the brain to the spinal cord via efferent nerve fibres indicating the necessary response.

The mechanisms of this communication have been thoroughly studied with regard to the alteration of the systems involved in the pain episode and the brain’s response regarding the injured site. What has been found is that peripheral injury can produce central changes which are maintained even after the inputs from the injury are removed. While peripheral mechanisms have usually been held responsible for the prolonged sensory disturbances associated with tissue injury, studies have shown that sensitization within the CNS also contributes significantly to this phenomenon. These studies have demonstrated that after injury, there is sensitization of neurons in the dorsal horn of the spinal cord and other areas of the somatosensory pathway.

Central sensitization can be seen by increased spontaneous activity, reduced thresholds or increased responsivity to afferent inputs, prolonged after discharges to repeated stimulation and the expansion of the peripheral receptive field of dorsal horn neurons. For example, in amputees and patients with severed cervical nerve roots, signs of hyperexcitability can be seen in dermatomes adjacent to the denervated limb which may indicate the spread of spinal hyperexcitability from denervated segments to segments immediately rostral and caudal to the denervated zone. The type of reorganization observed in the spinal map of the limb is also seen in the brainstem and cortical areas where re-mapping occurs following injury, as evidenced by the realistic representation of the missing limb and the persistence of a pain that existed in the limb prior to amputation.

Clinical evidence of CNS plasticity in pain: phantom limbs—To many people, amputation is the most common cause of phantom limbs and phantom limb pain but these sensations can also occur following deafferentation without amputation (e.g.
brachial plexus avulsion or spinal cord injuries). For example, patients with severed spinal cords cannot feel or control anything below the level of the break yet they may continue to experience the pain of an ingrown toe nail, have the experience of phantom legs or other body parts, and may experience pain in these phantom areas. What these examples demonstrate is that if an amputated or completely anaesthetized limb continues to be experienced as a source of pain which resembles an old injury, it is likely that the pain is centrally represented and a “pain memory” exists for the sensations of injury/pre-amputation pain.

The experiences that accompany phantom limbs highlight the complexity of this condition and the role the brain plays in the experience of phantom sensations/pain memories. Additionally, the reality of these sensations gives a clear indication why they can be so distressing for those experiencing them. One of the striking features of the phantom limb is its sense of reality to the amputee due to its vivid sensory qualities. These occur in relation to the limbs precise location in space, movement (e.g. in synchrony to the other limb when walking) and details of the limb before amputation (e.g. a ring on a phantom finger or a painful bunion that had been on the amputated foot).

For people who experience pain in their phantom limb, it can range from mildly irritating to very painful, with changes in this pain perception varying from day to day. Such variability is indicative of the complexity of factors influencing the person’s perception of their symptoms.

The implications of the findings from studies on phantom limbs and phantom limb pain are that the processing within the CNS contributes to not only the reality of the symptoms experienced but also to the complexity of these sensations. As described by Flor et al., it is likely that the central changes and re-organization following amputation occurs not only for the areas involved in the sensory-discriminative aspects of pain, but also for those brain regions that mediate affective-motivational aspects of pain. Such re-organization should therefore also be considered in related phenomena such as tinnitus which has been referred to as a phantom auditory sensation and phantom auditory pain.

**How tinnitus is similar to chronic pain**—Like chronic pain, tinnitus is a complex condition not simply explained by damage to structures that make hearing possible. It is a vexing and complex experience that requires consideration of physiological and psychological factors in its occurrence and its impact on the lives of those suffering from it.

Tinnitus is a perception of sound that occurs in the absence of external sound and can be divided into two groups, objective and subjective tinnitus. Objective tinnitus can be observed and measured as it is caused by sound being generated in the body reaching the ear through conduction in body tissues. In contrast, subjective tinnitus is meaningless sounds not associated with a physical sound, heard only by the person experiencing the tinnitus and is more prevalent than objective tinnitus.

Tinnitus in its most common form presents as ‘phantom’ sensations not associated with any physical stimulus arising in the ear canal and is an enigmatic disorder manifest with mysterious symptoms and perplexing neurobiology, in much the same way as chronic pain conditions manifest with perplexing causes and mediators.
Subjective tinnitus can occur in many different forms (e.g. unilateral, bilateral) and, like pain, can be divided into different severities (e.g. mild, moderate, or severe). Mild forms of the condition rarely cause many problems for the person but moderate forms of the condition can interfere with intellectual work and sleep and can be distressing to the person.

Severe tinnitus, like chronic pain, can cause considerable distress as it can have a major effect on many aspects of the person’s life (cognitive, personal, social, sleep, mood, etc). It is the perception of these sounds (e.g. roaring, buzzing, humming, saccades, hissing, ringing), and their persistence, that causes considerable distress for chronic tinnitus patients as the cause(s) remain complex and uncertain.

In addition to similarities in the distress caused by tinnitus and chronic pain, there are similarities in the causes and treatments of these conditions. Similarities between the perception of chronic pain and chronic tinnitus have been outlined by Tonndorf and Moller, who state that they are both continuous subjective experiences that may change in quality and character over time. Both perceptions are under the control of the CNS.

Both the auditory and somatosensory systems possess a well-developed network of efferent fibres that appear to exercise some control over afferent activity and, furthermore, disruption of this network (deafferentation) might explain both perceptions. Efforts to treat both sensations peripherally have met with limited success but they both have the potential to be masked or reduced by sensory stimulation or medication. In addition to these similarities, chronic pain, and some forms of tinnitus, are characterized by hypersensitivity to sensory stimulation. The anatomic locations of the neuronal structure(s) generating the sensations of chronic pain or tinnitus are different from the locations of the structures to which these symptoms are referred (the ears for tinnitus or the peripheral location of injury for pain).

Additionally, there is a strong psychological component to tinnitus and chronic pain which supports the hypothesis that brain areas other than those responsible for sensory perception are involved.

Clinical evidence of CNS plasticity and tinnitus: phantom sound—The role of the CNS’s ability to change (i.e. CNS plasticity) causing symptoms and signs of disorders has become clearer from studies of neuropathic pain, which led to an appreciation of the similarities between central neuropathic pain and tinnitus. Much like the experience of the phantom limb patient wherein they experience pain in a limb that is no longer there, those with tinnitus experience the sensation of sound without external stimulation which has resulted in tinnitus being referred to as belonging to a group of phantom sensations. As a phantom auditory perception, tinnitus occurs in much the same way as phantom pain due to CNS plasticity and the influence that this process has on the perception of sensory inputs and the experiences that occur as a result.

The role of CNS plasticity can be seen in tinnitus as it is caused by changes in the function of the auditory nervous system including shifts in the influence of excitatory or inhibitory events resulting in neural hyperactivity, reorganization of neuronal networks and rerouting of information, as well as changes in tonotopic maps.
this neural hyperactivity may reside, how it can be identified, what initiates it, and how it is sustained constitutes much of the driving force behind efforts to determine the neural basis of tinnitus.\textsuperscript{20}

What has been found is that following peripheral injury, new patterns of brain activity occur including changes in the primary and non-primary auditory pathways which contribute to tinnitus.\textsuperscript{20} It is proposed that the primary pathways sustain the changes and the non-primary contribute to perceived severity and emotionality.\textsuperscript{20}

The result of these changes and alterations between excitatory and inhibitory brain processes could be neural activity flowing between brainstem and the cortex in a self-sustaining process which produces persistent perceptions of tinnitus. As a result of this neural activity, as with chronic pain, it is therefore necessary to take into consideration the importance of addressing the patient’s perceptions when treating the symptoms of their tinnitus.

Conclusions

Similarities between chronic pain and tinnitus have been shown in many studies.\textsuperscript{21,22,23} For example, subjective tinnitus is considered to be a phantom sensation similar to phantom pain as both conditions have symptoms that occur in the absence of observable causes hence they are experiences of abnormalities rather than diseases.\textsuperscript{21}

The similarities between chronic pain and tinnitus can be seen in the description of tinnitus by Saunders who states that damage to the peripheral organ serves as a trigger for tinnitus, which is then sustained by events occurring in the central auditory pathway.\textsuperscript{20} These changes most likely result from maladaptive neuroplastic processes which may extend to non-sensory areas of the brain giving rise to the attentional and emotional aspects of tinnitus.

Such changes, which correlate to tinnitus, include hyperactivity of spontaneous activity in the brainstem and auditory cortex as well as tonotopic representation of frequency in brainstem and cortical areas following inner ear damage.

As with chronic pain, investigation into the causes and impact of tinnitus have revealed that this is a complex and multidimensional experience. Tinnitus is a constellation of neural changes that are becoming increasingly complex with advancing research. This complexity, and the growing array of contributing and associated brain factors, further suggests why tinnitus has not been amenable to a single treatment.\textsuperscript{20}

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Medical evaluation and management of tinnitus

R Goodey

Abstract

Assessment is the first step in management as a patient's tinnitus may be influenced by their first contact with our clinic. At the first consultation a free-flowing history without a questionnaire may give the patient confidence without introducing additional concerns. Referral to a checklist avoids omissions. At a tertiary tinnitus clinic, questionnaires are essential.

Examination should always include microscopic inspection of the ears and assessment of the neck and temporomandibular joints including any tinnitus modulation associated with them. Pure tone audiometry should include 8000 Hz and, in most cases, tympanometry and speech discrimination. Additional investigations may be required for pulsatile tinnitus, other para-auditory noises, or if there is asymmetrical tinnitus and/or hearing loss. Patients with distressing tinnitus attending a tertiary tinnitus clinic require a case history questionnaire, validated questionnaires of tinnitus severity and associated problems, additional audiological tests including high frequency audiometry, otoacoustic emissions, loudness discomfort levels, possibly auditory evoked potentials, plus psychoacoustic measurements of the tinnitus.

Management requires explanation and understanding as a basis for reassurance, plus improvement and/or manipulation of auditory and, where appropriate, somatic sensory inputs. For those who fail to respond to simple steps, cognitive behavioural therapy, masking or deconditioning with music or other sounds should be provided by those with appropriate training. We look to centrally-acting drugs, electrical or magnetic stimulation as direct approaches to further facilitate reversal of the neuroplastic changes inherent in the development and persistence of tinnitus.

In my opening address, I described my perception of the auditory pathways. I see the non-classical as being the more basic, polysensory pathways, operating at a largely unconscious level and greatly influenced by the limbic and autonomic systems. I see the classical auditory pathways as a later development allowing more conscious awareness of sound and the development of auditory communication, including language. I perceive these pathways as both complimentary and competing.

In my opening address I also acknowledged my acceptance of tinnitus generation as a central phenomenon. However, I pointed out that I do not consider that this reduces the significance of altered peripheral stimulation both on the initial triggering of tinnitus generation and on its subsequent persistence. Therefore the model upon which I base my assessment and management of patients with tinnitus has changed very little. It emphasises alterations in auditory and somatosensory (and possibly other) stimulation. It recognises the influence of psychological factors including emotional associations, fear of that which the patient does not understand, and a conditioning process which is largely unconscious. It recognises the occasional role of central injury.

First contact—Assessment of the patient with tinnitus cannot be separated from subsequent management because assessment is part of the management. A patient’s
first contact with a clinician’s office may reduce or increase that patient’s anxiety about their tinnitus and hence the extent of the problem presented to the clinician and its responsiveness to subsequent management. We need to ensure that a patient’s first contact to make an appointment is reassuringly supportive without being misleading and that it is appropriately businesslike. The patient needs to feel immediately that their problem is now being shared.

**First consultation**—This is even more important. The patient must be confident that the clinician is going to know all the facts about their tinnitus and associated stress, and so will be in a position to help the patient to manage it. As clinicians, we must allow time to listen and we must be seen to take careful notes. At the same time we must remain in control of the consultation and ensure we obtain from the patient all the information which may be helpful but which the patient may not see as relevant.

Personally, I find a checklist is a great help: I can allow the first consultation to follow the pattern of an unfolding history of the problem but by referring back to the checklist I can ensure nothing is been overlooked.

Questionnaires are often seen as a better tool to ensure all required information is obtained and possibly to save the clinician’s time as well.

**When to use questionnaires**—The first tinnitus research I was involved in was in the late 1970s with Dr. Pamela Melding and Professor Peter Thorne. The patients were all highly selected with the tinnitus described as “incurable and intolerable”. Each one was assisted to complete a complex series of questionnaires at the first visit. We learned a great deal from analysing these in conjunction with results of comprehensive audiology, clinical examination and the response to intravenous lidocaine.

Questionnaires are now more sophisticated and have been validated in a variety of situations. Questionnaires play an essential role in the assessment of patients who are attending a specialised clinic. Usually they are doing so because their tinnitus has already been identified as severe and distressing.

A case history questionnaire was evolved, following a consensus session at a Tinnitus Research Initiative meeting in Regensburg in 2006 and a subsequent analysis of other questionnaires. This case history questionnaire contains 35 items of which 14 were considered essential, mainly because they were common to most other questionnaires.

Validated questionnaires for measurement of tinnitus severity are discussed elsewhere in this conference. For some tinnitus patients questionnaires may also be used for assessment of depressive symptoms, anxiety, quality of life, and insomnia and may also be used as outcome measures.

**When not to use questionnaires**—Following our research in the 1970s, I subsequently used the same questionnaires in my private practice. The questionnaires were completed by the patient and a registered nurse together before I first met the patient. I thought I was providing a very sophisticated clinical service for all patients with tinnitus. However, it quickly became evident when the patient started their consultation with me, that the questionnaire had raised a whole lot of new concerns and questions which the patient wanted addressed before the consultation got
underway. I realised that for many of these patients the questionnaire had increased the anxiety associated with their tinnitus and had made subsequent treatment more difficult. In my opinion, questionnaires have no place during first contact with patients in a normal practice in which their concern about their tinnitus may be relatively mild. In my opinion, questionnaires belong in specialist tertiary clinics.

**Audiometry**—Almost all patients with tinnitus should have diagnostic pure tone audiometry up to 8000 Hz and most should have speech audiometry and immittance audiometry at the time of their first consultation. However, sometimes tinnitus and any associated hearing and ear symptoms are eliminated by cleaning of the ear. If audiometry is to be carried out before the consultation then the ear canals should be competently checked first, to avoid unnecessary audiometry and associated cost. Sometimes, the onset of tinnitus is clearly related to injury, whiplash or dental treatment and the patient may be reluctant to have an audiogram which they perceive as irrelevant. In most of these I will subsequently insist they have audiometry and especially so if their tinnitus is not symmetrical.

At specialised clinics, high frequency audiometry, oto-acoustic emissions and loudness discomfort are highly recommended, and auditory evoked potentials should be available. Psychophysical measures of tinnitus including loudness match, pitch match, maskability and residual inhibition are all highly recommended, especially if clinical research is involved.

**Examination**—Minimum examination includes:

1. Inspection of the ears under the microscope by an operator experienced in doing so and with facilities to remove any debris.

2. Examination of the neck for its range of movements, tenderness, muscle tension and modulation of tinnitus by traction, compression and lateral pressure (at the very least).

3. Examination of the temporomandibular joints for tenderness, crepitus, and modulation of tinnitus by clenching or biting (at the very least).

In addition:

1. A more general examination is frequently required especially if there are associated symptoms, or if the tinnitus is pulsatile or other para-auditory sounds are suspected.

2. Asymmetrical hearing and/or tinnitus must be investigated in all but the very old.

3. Unless noise intolerance is suspected, I test for residual inhibition following 60 seconds of noise delivered through a 20 gauge suction needle. A good response encourages both me and the patient.

4. I used to administer up to 2 mg per kg of intravenous lidocaine in my office to a high proportion of patients with tinnitus. There were no untoward effects. The response or lack of it was used to categorise each patient's tinnitus and the likelihood they might respond to oral anticonvulsants. Few patients require such powerful drugs and nor are such drugs very effective in modest doses. However, the dramatic temporary relief experienced by many patients
following intravenous lidocaine raised their expectation to a level which made any other management disappointing. A lidocaine test is better reserved for patients who are desperate and for whom all options must be considered. In a modern context more intensive monitoring is required during intravenous lidocaine administration than was used in the past.

**Initial management**—In my earlier presentation at this meeting, I speculated that cure of tinnitus requires a reversal of the neural changes which cause its generation and also the maintenance of this reversal. I identified three broad routes for treating tinnitus: 1) Repair or manipulation of the various sensory inputs in various ways; 2) Approaches which I grouped together as psychological; 3) Direct approaches which can be through the bloodstream/cerebral spinal fluid (drugs, hormones and dietary factors), magnetic or electrical stimulation or changes induced through radiation, surgery or temperature (Figure 1).

<table>
<thead>
<tr>
<th>Sensory inputs:--</th>
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<tbody>
<tr>
<td>➢ Auditory (sound, electrical, drugs)</td>
</tr>
<tr>
<td>➢ Somatosensory (physical, electrical, drugs)</td>
</tr>
<tr>
<td>➢ Other (vision, olfaction, vestibular, taste etc)</td>
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<table>
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<tr>
<th>Psychological:--</th>
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</thead>
<tbody>
<tr>
<td>➢ Explanation &amp; understanding</td>
</tr>
<tr>
<td>➢ Disassociate emotional associations, drugs</td>
</tr>
<tr>
<td>➢ Cognitive therapy etc, “de-concerning”</td>
</tr>
<tr>
<td>➢ TRT etc, “de-conditioning”</td>
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<th>Direct central:--</th>
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<tr>
<td>➢ Drugs, hormones &amp; dietary factors</td>
</tr>
<tr>
<td>➢ Transcranial magnetic stimulation</td>
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<td>➢ Electrical stimulation</td>
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</tbody>
</table>

**Figure 1:** Reversing the neural changes which cause tinnitus generation, and *then maintaining that reversal.*
I acknowledged that at the present time we seldom cure tinnitus and have to settle for ameliorating it or simply reducing its impact. There are a lot of therapeutic options to help us do so. If, like me, you are a front line clinician seeing many patients with tinnitus and with limited time available for each, then you have to adopt a relatively straightforward approach to management and refer on to a specialised tertiary clinic, those patients who are still severely affected by their tinnitus.

After taking a history, examining the patient and reviewing their audiometry, my first step in management is to provide an explanation which is tailored to their tinnitus and the things which triggered it and those which modulate it. Questions are limited to those focussed on mechanisms.

We then work through the various therapeutic options which can be implemented after the consultation, or for which the patient can immediately be referred (especially hearing aids if appropriate (Figure 2)).

I reassure the patient that they do not have to remember all that we have discussed because within a couple of days they will get receive from me a written report summarising all aspects of the consultation and the treatment options suggested. For most patients I suggest that their tinnitus is likely to start subsiding now that they understand its mechanisms and how to modify it. I do not usually make a follow-up appointment on the basis that I do not want the patient to be reminded about their tinnitus if it has ceased being an issue. I make it clear that they are welcome to ring and make another appointment at any time.

<table>
<thead>
<tr>
<th>Reduce aggravating physical &amp; metabolic factors</th>
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<tr>
<td>• loud noise, caffeine, tinnitus inducing drugs, food &amp; drink intolerances, dietary shortages</td>
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<tr>
<th>Reduce or disassociate emotional factors</th>
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<tr>
<td>• Stress, depression, fear and anger, tricyclic medication?</td>
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<tr>
<th>Manage somatosensory modulators</th>
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<tbody>
<tr>
<td>• Skin, jaw joints and neck</td>
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<table>
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<tr>
<th>Sound enrichment (reduce tinnitus to noise ratio)</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Avoid quiet (especially if hearing impaired as increased sensitivity of central auditory systems increases tinnitus and hyperacusis</td>
</tr>
<tr>
<td>• Relaxing environmental sounds</td>
</tr>
<tr>
<td>• Music – orchestral or instrumental, major mode, moderate tempo, no associated words</td>
</tr>
<tr>
<td>• Correct ear disease and hearing loss when possible</td>
</tr>
<tr>
<td>• Hearing aids</td>
</tr>
</tbody>
</table>

Send the patient a written report

*Figure 2.* Therapeutic opportunities following first consultation.
Subsequent consultation and referral to tertiary tinnitus—If a patient does ring to make another appointment but a lot of time has elapsed, then they are asked to re-read my previous report. If it is lost they are sent a new copy to read. They sometimes ring back to say they no longer need an appointment. If they do need to be seen again, then I reassess almost everything and if nothing has been overlooked I usually refer them to a tertiary tinnitus clinic at this stage. I know they will then get a far more intensive (and time-consuming) assessment. If they have hearing aids, these will be reassessed.

Sophisticated interventions such as desensitisation with music and tinnitus retraining therapy will be available. If relief remains disappointing then I and their family doctor may need to assist the clinic arrange additional measures to deal with personal issues which have not been addressed, holistic measures, hypnotherapy, biofeedback and expert help with stress, depression and sleep problems. Occasionally, a patient's tinnitus will continue to dominate their lives and such people may get great benefit from belonging to a tinnitus support group where they are able to share their tinnitus.

Desperate patients—There is a small group of patients in whom tinnitus is intolerable and who become desperate. Those involved in their management have to go back over all the therapeutic options to see if they can be better understood and more usefully implemented. Additional measures include tricyclic drugs and sometimes a very short course of a benzodiazepine drug, provided this is in conjunction with other strategies which can be continued after the benzodiazepine has been withdrawn.

Occasionally, we have to fall back on even more powerful drugs. I tend to use an oral cocktail of nortriptyline, sodium valproate and carbamazepine. By using a cocktail I find that the dose of each is less and that side-effects are less. Usually, I build up the doses very slowly. If desperation makes a slow approach impossible then full therapeutic doses can be implemented quickly but initial side-effects are severe and this is best done in a hospital environment. I feel more optimistic about the potential of these drugs being helpful if there has been an initial excellent response to intravenous lidocaine.

I look forward to the day when an excellent response to intravenous lidocaine predicts a comparable result to well-tolerated oral drugs which are yet to be developed. Even when more effective drugs become available I expect they will need to be used as part of a package of care.

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Promising medications for tinnitus
PF Smith, Y Zheng, CL Darlington

Abstract

The drug treatment of tinnitus has been undermined by a dearth of experimental drug studies in animals using realistic animal models and also of well controlled clinical trials in humans. The rationale for the use of anti-epileptic and benzodiazepine drugs is that tinnitus is related to neuronal hyperactivity in various auditory centres of the brain. However, the experimental evidence for such hyperactivity is not entirely consistent and salicylate- and acoustic trauma-induced tinnitus may have quite different neural substrates.

There are few well controlled clinical trials of the effects of anti-epileptic drugs in human tinnitus and the few that have been conducted suggest that drugs such as carbamazepine and gabapentin have variable effects. Benzodiazepines have been reported to be effective in some cases, but are associated with considerable adverse side effects such as sedation. There is some evidence that antidepressants may be effective in some cases but further trials are needed. Intratympanic administration of gentamicin, steroids or lidocaine have been reported to be beneficial; however, this form of treatment is effective mainly for tinnitus associated with Menière’s disease and is also associated with significant risks.

The lack of a clear direction in the drug treatment of tinnitus has led some patients to try herbal remedies, such as *Ginkgo biloba* extracts. Unfortunately, there is no reliable scientific evidence to support their efficacy. Some of the most promising drug treatments on the horizon may be the NMDA receptor antagonists that are in clinical trials for the treatment of neuropathic pain.

Drug treatments for medical conditions are discovered either serendipitously or by rational design. However, in order for drugs to be developed by rational design, the mechanisms underlying the disorder must first be understood. Unfortunately, to date, tinnitus is a condition for which drug discovery has been dominated by serendipity more than design. There are several reasons for this.

First, tinnitus has several different causes (e.g., acoustic trauma, head and neck injuries, drug ototoxicity), and it is possible that the neural substrates of different kinds of tinnitus are somewhat different. For example, the evidence that tinnitus is associated with neuronal hyperactivity in central auditory brain regions is more convincing for tinnitus caused by acoustic trauma than that caused by exposure to salicylate.

Second, despite the development of several realistic animal models of the disorder, relatively few studies have employed such models in drug studies.

Third, compared to other neurological disorders, relatively few systematic clinical trials have been conducted even for many of the drugs that are currently used to treat tinnitus in humans (e.g. carbamazepine, gabapentin).

**Intratympanic drug treatment**—Intratympanic gentamicin has been used successfully to treat tinnitus associated with Menière’s disease, where the cause of the
tinnitus is peripheral. The rationale behind this therapy is that aminoglycoside antibiotics such as gentamicin are ototoxic and therefore can be used to reduce activity in the affected ear. However, gentamicin can cause permanent ototoxicity and therefore it is critical that a sufficiently long interval (e.g. 7 days) between injections is used.

Intratympanic steroid treatment (e.g. dexamethasone) has also been used to treat tinnitus of peripheral origin. However, tympanic membrane perforation lasted for more than 6 months in 2 patients. Garduno-Anaya et al. studied 22 patients with unilateral Menière’s disease and found that intratympanic dexamethasone relieved tinnitus in 48% of the patients. This was a prospective, randomised, double-blind, placebo-controlled trial and therefore probably represents the most convincing evidence to date. By contrast, Araujo et al., also using a prospective, randomised, placebo-controlled but single-blind trial, reported that intratympanic dexamethasone had no significant effect on severe tinnitus compared to placebo.

Sakata et al. reported that intratympanic administration of 4% lidocaine relieved tinnitus in 81% of a sample of 292 patients. Unfortunately, vestibular symptoms, including vertigo, often developed following the infusion. The muscarinic acetylcholine receptor agonist, carbachol, and the acetylcholinesterase inhibitor, pilocarpine, have also been used intratympanically; however, while they relieved tinnitus in 50% of patients, the benefit was short-lived and tinnitus returned with its original intensity after 12-72 hs.

Intravenous lidocaine—The use of local anesthetics for the treatment of tinnitus dates back to 1937. A number of studies have reported that i.v. lidocaine can alleviate tinnitus, although the mechanism of action is unknown. Unfortunately, few of the data supporting the use of lidocaine comes from large, well controlled clinical trials with placebo controls and double-blind methodology. Kalcioglu et al. attempted to determine if the effects of i.v. lidocaine on tinnitus might be reflected in changes in otoacoustic emissions. However, they found that while some patients reported subjective relief from tinnitus that lasted up to 4 weeks, there were no significant differences in either spontaneous or distortion product otoacoustic emissions.

This raises the issue of whether the positive effects of lidocaine in uncontrolled trials may be due to expectation, i.e. the placebo effect. It is still unclear how lidocaine might work to alleviate tinnitus, although it is possible that since it may work intratympanically, it might produce either vasodilation or sodium channel blockade in the cochlea.

Osmotic regulators and vasodilators—Since Menière’s disease is associated with a hypertension of the labyrinthine endolymphatic fluid, diuretics have been used to treat Menière’s symptoms, including tinnitus. The loop diuretic, furosemide, has been used with some success. However, in a review of the literature between 1966 and 2005, Thirlwall and Kunda concluded that there was insufficient evidence that diuretics have any significant effect on tinnitus or the other symptoms of Menière’s disease. Other drugs that regulate osmotic pressure, such as glycerol and mannitol, have had limited success.
Vasodilators were once thought to be effective, but recent studies have not confirmed their efficacy. However, misoprostol, a synthetic prostaglandin E1 analogue that stimulates vasodilation, has been shown to be effective in about one third of tinnitus patients. Yilmaz et al. using a double-blind, placebo-controlled design, found that misoprostol reduced the loudness of tinnitus in 18/28 patients.

**Benzodiazepines**—If tinnitus is caused by neuronal hyperactivity in auditory areas of the CNS (the ‘sensory epilepsy hypothesis’), it follows that benzodiazepines might reduce this hyperactivity, via their action on the benzodiazepine binding site of the GABA$_A$ receptor. Gananca et al. conducted a retrospective survey of 25 years of the use of clonazepam in the treatment of tinnitus, and concluded that it was at least partially effective in 32% of cases. Shulman et al. have suggested that for tinnitus of central origin, benzodiazepines can provide long-term relief in 90% of cases.

Unfortunately, there are no systematic well-controlled clinical trials of the effects of benzodiazepines on tinnitus. Even if they were effective it is conceivable that they could work either by having a general anxiolytic effect or by reducing neuronal activity by a mechanism not involved in the generation of tinnitus. One major disadvantage of their use is adverse side effects such as sedation and their high dependence liability.

**Non-benzodiazepine anti-epileptic drugs**—As with benzodiazepines, there is a dearth of well controlled clinical trial studies of the effects of anti-epileptic drugs on tinnitus. Most anti-epileptic drugs such as valproate, phenytoin and carbamazepine work by inhibiting voltage-dependent sodium channels; therefore, if tinnitus was caused by neuronal hyperactivity, such drugs might be expected to provide some relief, although many of them have considerable adverse side effects when used to treat epilepsy.

Carbamazepine has been used to treat tinnitus, but other than case studies, only 3 trials have been published. Melding and Goodley reported that 56% of patients that had responded positively to lidocaine experienced relief from tinnitus following carbamazepine treatment. Sanchez et al. also reported that carbamazepine was effective in reducing tinnitus in 58% of patients and abolished tinnitus in 18% of patients. However, Hulshof and Vermeij reported that carbamazepine was less effective than a placebo.

The best studied anti-epileptic drug in relation to tinnitus is gabapentin. Bauer and Brozoski conducted a prospective, placebo-controlled, single blind trial of the effects of gabapentin on 39 patients with tinnitus. They found that the drug was effective in reducing tinnitus in some patients, especially those in whom the condition was related to acoustic trauma. However, more recently, Witsell et al., using a randomised, placebo-controlled, double blind trial, reported that gabapentin had no significant effect. Similar results have recently been reported by Bakhshae et al.

**Anti-spasticity drugs**—Baclofen, a GABA$_B$ receptor agonist, has been used to treat tinnitus, but with little success and substantial adverse side effects. In a randomised, double-blind, placebo-controlled clinical trial, Westerberg et al. found that baclofen was no more effective than placebo; however, 26% of patients withdrew due to the adverse side effects of the drug.
Antidepressants—It has been reported that patients who suffer from tinnitus-related depression often experience relief from tinnitus when treated with antidepressants. Fomer and Shi found that patients who developed depression following the onset of tinnitus exhibited a significant decrease in tinnitus severity and depression, when treated with selective serotonin uptake inhibitors (SSRIs). In a randomized, placebo-controlled, double-blind study, Zoger et al. found that sertraline significantly reduced tinnitus compared to placebo, with modest side effects. However, Robinson et al. found that paroxetine had no consistent effects on tinnitus in patients who were not depressed. Baldo et al. concluded that there is insufficient evidence at this stage to determine whether antidepressants are effective in treating tinnitus.

N-methyl-D-aspartate (NMDA) receptor antagonists—In addition to increasing inhibition in the CNS, another strategy for reducing neuronal hyperactivity associated with tinnitus, is to reduce excitation. The non-selective glutamate receptor antagonist, caroverine, has been reported to relieve tinnitus in 63% of patients. In general, even selective NMDA receptor antagonists have been associated with severe adverse side effects such as psychotic symptoms. However, memantine and a group of NR2B-selective NMDA receptor antagonists have proven to be well tolerated by patients and have been used in the treatment of neuropathic pain. To date, only one clinical trial of memantine has been published, and this showed no effect on tinnitus. Another NMDA receptor antagonist, flupirtine, has been investigated in humans; however, no significant effects on tinnitus were observed.

Ginkgo biloba extracts—Despite the claims that Ginkgo biloba extracts relieve tinnitus, there is no reliable evidence that they have any effect at the doses normally used in humans. The first large, double-blind, placebo-controlled study of the effect of Ginkgo biloba extracts on tinnitus was reported by Drew and Davies. There was no significant effect of the extract compared to placebo. In the most recent randomized, placebo-controlled, double-blind clinical trial, Rejai et al. also found no effect of Ginkgo biloba compared to placebo.

Conclusions

The development of drug treatments for tinnitus has suffered from a lack of rational design partly due to the poor understanding of the neural basis of the phenomenon and the failure to use realistic animal models to test new drug treatments. This, together with a paucity of well-controlled clinical trials, has led to a situation where there is no obvious drug treatment strategy for the treatment of tinnitus and therapy often follows an experimental approach to determine what drug might be effective for an individual patient.

For tinnitus associated with Menière’s disease, where there is a clear peripheral pathology that sustains the disorder, intratympanic treatment with gentamicin, dexamethasone or lidocaine has proven effective in some cases. However, this is an invasive procedure that carries some risk and is unlikely to work for most forms of tinnitus, which are triggered by a peripheral event such acoustic trauma, and then maintained by maladaptive plasticity in the brain. In this latter case, the drug treatment options are usually benzodiazepines and anti-epileptic drugs; however, despite their frequent use, the evidence for their clinical efficacy against tinnitus is not
consistent. In addition, most of these drugs are associated with significant adverse side effects.

Antidepressants have been reported to be effective in some cases; however, whether their efficacy is against tinnitus as opposed to depression associated with tinnitus, remains unclear.

There is no scientific evidence that herbal remedies such as *Ginkgo biloba* extracts are effective in the treatment of tinnitus.

The effective drug treatment of tinnitus still awaits a better understanding of the neural basis of the disorder. The investigation of novel NMDA antagonists, for which there is a clear rational basis, represents a positive development, although the clinical trial results to date have been disappointing.

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Tinnitus management with repetitive transcranial magnetic stimulation

CM Stinear

Abstract

Transcranial magnetic stimulation (TMS) is a safe, painless, non-invasive technique for stimulating human cortex. Repetitive TMS (rTMS) can decrease auditory cortex excitability, and reduce tinnitus symptoms. Evidence to date indicates that patients with pure tone tinnitus respond better to rTMS than those with white noise tinnitus, and that the benefits decline with tinnitus duration. Maintenance doses of rTMS, and combining rTMS with therapy, are promising future directions currently under investigation. Repetitive TMS may play a useful role in the management of selected tinnitus patients.

Tinnitus affects approximately one third of adults at some point in their lives, and up to 15% seek medical treatment for ongoing symptoms. These symptoms are characterised by unwanted auditory perceptions, such as ringing or hissing, in the absence of external sound. Tinnitus can be linked to hearing loss (noise-induced or age-related), exposure to ototoxic drugs, head trauma, Ménière’s disease, and acoustic neuroma, though often no specific cause can be identified.

When linked to hearing loss, tinnitus is associated with increased neuronal excitability in the auditory cortex, and changes to its tonotopic organisation. Cortical excitability can be safely modified using non-invasive techniques, such as repetitive transcranial magnetic stimulation (rTMS). The use of rTMS in tinnitus management is gaining support, and evidence to guide patient selection and treatment planning is reviewed here.

Repetitive transcranial magnetic stimulation—Transcranial magnetic stimulation (TMS) is a safe, painless, non-invasive technique for stimulating the cortex of the brain in humans. Briefly, a capacitor is discharged through an insulated wire coil, which is held against the scalp. This generates a very brief magnetic field, which passes through the scalp and skull, and generates a weak electric current in the underlying tissues. This depolarises cortical neurons and generates action potentials in those that reach firing threshold. When applied to primary motor cortex, the resulting activation of descending pathways can be detected by recording an evoked potential in contralateral muscles with surface electromyography. When applied at weaker, subthreshold intensities, TMS activates intracortical interneurons without producing any descending output.

Repetitive TMS (rTMS) at subthreshold intensities affects intracortical networks and the excitability of the cortex. RTMS delivered at 1 Hz suppresses cortical excitability, making it less responsive to synaptic inputs. Conversely, cortical excitability is facilitated by rTMS at higher frequencies. These physiological effects outlast the period of stimulation by up to an hour.

Repetitive TMS and tinnitus—As tinnitus is often related to hyper-excitability in the auditory cortex, 1 Hz rTMS has been used in an attempt to suppress its excitability. Typical protocols involve delivering subthreshold stimuli at 1 Hz for up to 20 minutes.
(1200 stimuli). More recently, short trains of up to 5 stimuli delivered at up to 20 Hz have been found to effectively reduce tinnitus symptoms in some patients.\(^7\) The 10-20 system used for positioning EEG electrodes can be used to locate the stimulating coil over the auditory cortex.\(^8\) The primary auditory cortex is located deep below the surface of the brain, on Heschl’s gyrus, and is therefore unlikely to be directly affected by TMS. Rather, the effects of rTMS on secondary auditory areas, located on the surface of the brain, appear to drive changes in excitability throughout the auditory network.\(^9\)

Double-blind, sham-controlled trials of 1 Hz rTMS have found meaningful reductions in tinnitus symptoms for most patients in the treatment arm.\(^9\)-\(^11\) However, some trials have found no difference in the effects of real and sham rTMS, probably due to placebo effects and small sample sizes.

**Patient selection and treatment planning**—All patients should be screened for contraindications to rTMS, including intracranial metallic implants, cardiac pacemaker, and a history of seizure. In general, patients whose tinnitus symptoms are less severe or of shorter duration are more likely to benefit from rTMS.\(^12\) However, there is some evidence that the extent of auditory cortex hyperactivity detected with PET imaging is more predictive than clinical history.\(^13\)

Patients with unilateral symptoms are more likely to respond to rTMS than those with bilateral symptoms.\(^14\) Furthermore, those with pure tone tinnitus seem to respond best to 1 Hz rTMS, particularly those with lower frequency symptoms. Conversely, patients with white noise tinnitus respond best to short bursts of higher frequency rTMS.\(^7,\,14\)

For patients with unilateral, pure tone tinnitus, rTMS is typically applied at 1 Hz for 20 minutes (1200 stimuli). The effects of a single dose are generally short-lived, and can also be produced by sham rTMS due to a transient placebo response.\(^12\) Therefore, most clinical trials deliver rTMS 5 days per week, for 1 or 2 weeks. While the resulting improvements in tinnitus symptoms are generally greatest immediately after the treatment period, they can persist for 6 months\(^15\) and up to 1 year.\(^16\)

The effects of follow-up treatments, applied when symptoms began to return, have been described in a case report of a 44 year old male with a 15 year history of disabling tinnitus.\(^17\) Three follow-up treatments applied over the course of 5 weeks produced a significant reduction in symptoms, which was sustained for at least 6 months. This raises the possibility of maintenance rTMS treatment for the ongoing management of tinnitus.

**Future directions**—The suppression of auditory cortex excitability, on its own, is unlikely to promote plastic reorganisation and normalisation of the tonotopic map. RTMS could be used to prime the auditory cortex prior to other behavioural or physiological interventions that promote beneficial cortical reorganisation. The ‘prime then practice’ model has been developed in chronic stroke patients. Facilitating ipsilesional motor cortex excitability prior to motor practice with the paretic upper limb leads to greater functional improvements than motor practice alone.\(^18\) Priming the auditory cortex with rTMS prior to therapy that promotes normalisation of auditory cortex representations could be a useful next step in this area of research.
Variations in the genes that influence neural plasticity, such as those encoding brain-derived neurotrophic factor, can alter the cortical response to rTMS. Genotype may therefore influence patients’ responsiveness to rTMS. Similarly, medications that act on neurotransmitter systems, such as GABA agonists and selective serotonin-reuptake inhibitors, also influence the cortical response to rTMS. Future work will need to consider these factors. The selection of homogeneous groups of patients is likely to strengthen future trials, and will lead to the development of clinical guidelines for the use of rTMS in tinnitus management.

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Cortical electrical suppression of tinnitus and modulation of its related neural activity

JS Zhang, ZL Guan, XG Zhang, H Beydoun, J Zhang, M Seidman, K Elisevich, S Bowyer, Q Jiang, J Moran

Abstract

Tinnitus is a phantom sound that occurs in the absence of external acoustic stimulation. When chronic, tinnitus can be debilitating and have a significant economic impact on society. Recently, our clinician colleagues at Henry Ford Health System and other groups have demonstrated that auditory cortex electrical stimulation (ACES) suppresses tinnitus in patients. However, the large variability in the efficacy of ACES-induced suppression among individuals has hindered its development as a reliable therapy.

For ethical reasons, many issues cannot be comprehensively addressed in patients. Our research team at Wayne State University developed animal models to investigate mechanisms underlying tinnitus suppression through ACES and neuromodulation. In our first animal model, we demonstrated in hamsters that ACES induced complex neural responses (inhibition, excitation and no change) in the dorsal cochlear nucleus and inferior colliculus, structures that have been implicated in the aetiology of tinnitus. In addition, our magnetoencephalographic (MEG) data showed that ACES significantly decreased coherence within the inferior colliculus but slightly enhanced coherence in the dorsal cochlear nucleus.

These results indicate that ACES may have promoted recovery of the lost balance between excitation and inhibition in the neural network. Suppression of tinnitus may involve neuromodulation and gating adjustment at several brain centres. We expect that a greater understanding of the underlying mechanisms of ACES-induced tinnitus suppression will further clinical investigations and stimulate the development of specialized neural prostheses for effective tinnitus suppression.

Abbreviations

AC auditory cortex
ACES auditory cortex electrical stimulation
CF characteristic frequency
DCN dorsal cochlear nucleus
FTC frequency tuning curve
IC inferior colliculus
MEG Magnetoencephalography

Clinical studies have recently shown that stimulation of the auditory cortex (AC) through transcranial magnetic stimulation (TMS) or direct electrical stimulation have acute or longer-lasting suppressive effects.\footnote{1,5,14} This provides a new hope in finding an effective and reliable therapy. In TMS, an electromagnet is placed on the scalp to deliver magnetic field pulses of 100–300 \( \mu \)s in duration and 1.5–2.0 Tesla in strength.

Depending on the stimulation site and the parameters chosen, as well as the types of tinnitus, the efficacy of suppression differs greatly among individuals with 53% to 100% of patients reporting significant suppression.\footnote{6,7} The duration of suppression can
range from several seconds to minutes,\(^2,8\) 20 minutes to 5 days,\(^6,9\) 4 weeks,\(^10,11\) and up to 6 months when applying several sessions of low-frequency repetitive TMS.\(^10,12\)

Tinnitus suppression can occur as long as stimulation is active\(^1\) or up to 48 hours after stimulation.\(^6\) The drawback is that TMS often nonspecifically stimulates an area that is larger than the AC (33–96 cm\(^2\))\(^13\) and has limited penetration into the brain tissue.

To induce more focal and long-term stimulation, direct auditory cortex electrical stimulation (ACES) was introduced\(^1,3,4,14\) using epidural or subdural electrode arrays placed on the AC surface,\(^1,4\) or an intracerebral electrode inserted in Heschl’s gyrus.\(^14\) Compared to TMS treatment, ACES may yield longer (up to 10 months) tinnitus suppression,\(^15\) with the degree of the induced suppression of pure tone tinnitus higher (97%) compared to that with TMS (77%). The effect upon white noise tinnitus is lower (24%) compared to that of TMS (67%).\(^1\) In general, ACES yields better results by a more focal approach (personal communication, De Ridder, 2008). Because ACES has therapeutic advantage over that of TMS and has been shown to be safe, it deserves further investigation.\(^4\)

Following our recent report,\(^14\) our clinician team at Henry Ford Health System (M. Seidman, K Elisevich, S. Bowyer, Q. Jiang and J. Moran) has continued the investigation of ACES-induced suppression of tinnitus in patients whose tinnitus was debilitating and refractory to conventional therapies. Our newly implanted patient was a 56 year old male with a 5 year history of severe disabling tinnitus in the right ear and normal to moderately-severe sensorineural hearing loss (SNHL) bilaterally.

The preoperative assessment included psychoacoustic measures, questionnaires and functional imaging to determine tinnitus loudness and frequency/pitch which matched 80 dB SL and 10,000 Hz. Scores from the Tinnitus Handicap Inventory (THI), Tinnitus Reaction Questionnaire (TRQ), and Beck Depression index were 54, 47 and 13, respectively. Conventional pure tone audiometry indicated his hearing slightly decreased in the high frequencies, bilaterally. He had minimal SNHL through 3 kHz steeply sloping to severe SNHL. Functional MRI and magnetoencephalography (MEG) were performed to target the locus in the AC responding to a sound of predetermined frequency/pitch matching the patient’s tinnitus.

In MEG studies, a single Equivalent Current Dipole localization (ECD) method was used to detect neural activation and coherence in the AC following acoustic stimulation. The results demonstrated that the correlation and goodness of fit were poor, suggesting that the tinnitus-experiencing AC may have a shift in its tonotopic organization from a position that normally responds to a 10 kHz tone presented to the right ear. We also performed coherence analysis on MEG data collected without sound stimulation, during which the patient lay quietly and listened to their tinnitus. MEG results identified an area that was active in the 30 Hz range in the left AC following co-registration of MEG data with the subject’s MRI image (Figure 1). Both data were used to determine the position for implanting an electrode array.

Under general anaesthesia, a small left temporoparietal craniectomy was performed and a quadripolar electrode array (Quad Compact, Medtronic, Inc, Minneapolis MN) was implanted stereotactically using a neuronavigation unit (see details in recent report\(^14\) uploaded with coregistered MEG and MRI images obtained preoperatively (Figure 2). The initial evaluation of tinnitus was conducted 10 days after surgery. Electrical stimuli (0-10 V, pulse width of 60-450 µs, 3-30 Hz) were delivered in a
random manner and the polarity configuration was set to allow changing active
electrode contacts. Following an initial stimulation, the patient reported 50%
reduction of tinnitus loudness. Specifically, his THI, TRQ and Beck depression index
scores decreased from 54 to 36, from 47 to 38 and from 13 to 4, respectively. His
modified somatic perception questionnaire dropped from to 4 to 1. ACES yielded
apparent relief of tinnitus in this patient. Continued investigations are underway to
study the induced tinnitus suppression over time.

Figure 1: Co-registered anatomical MRI image with MEG coherence data. Red areas indicate
highly coherent areas that are active in the 30 Hz frequency range. The scale was from 0 to 1.
Graph of coherence indicates a peak at 30 Hz.

In summary, ACES has yielded promising results and has the potential to become an
important approach in managing tinnitus. However, the existing large variability in
the efficacy of the induced tinnitus suppression among individuals has hindered its
development as a reliable therapy. The mechanisms underlying ACES-induced
suppression are not clear and, to this end, our research team at Wayne State
University (J.S. Zhang, Z.L. Guan, X.G. Zhang and H. Beydoun) has developed an
animal model of this condition. In this initial animal model, we used hamsters to
investigate how ACES modulates neural activity in the inferior colliculus (IC) and
dorsal cochlear nucleus (DCN; Figure 3), the structures that have been implicated in
the aetiology of tinnitus.\textsuperscript{16-18}
Figure 2: MG tonotopic localizations for a 10 kHz tone presented monaurally to the right ear. Results are overlaid on the patient’s MRI image.

Figure 3: Schematic drawing showing that electrophysiological recordings were simultaneously conducted in the right IC and left DCN while electrical stimulation was delivered to the right AC.
Methods

Adult Syrian golden hamsters (n = 10; male; 60-70 days old) were used in this study. Six animals were exposed under anaesthesia (ketamine/xylazine, 100 mg/kg-10 mg/kg, i.p.) to a 10 kHz tone to the left ear at 125-130 dB SPL for 4 hrs. Another four age-matched control animals were similarly anesthetized but not exposed to the intense tone. Body temperature for tone exposure and the subsequent surgical procedures were maintained at 37°C with a thermostatically controlled blanket. The rationale of using this species was that the auditory system resembles that of humans, and our group has successfully used it to study neural correlates of tinnitus.19,20 All procedures were in accordance with the guidelines of the Institutional Animal Care and Use Committee at Wayne State University.

Seven to ten weeks after sound exposure and control treatment, surgery was performed under anaesthesia to expose the left DCN, right IC and right AC. Prior to implantation, each probe was dipped in 3% Di-I solution (1,1'-dioctadecyl-3,3',3'-tetramethylindocarbocyanine perchlorate, Invitrogen) prepared with DMSO to label electrode tracks from insertions. After placement of probes in the brain structures, warm 2% agarose in Ringer’s solution was used to secure the probes to prevent their displacement. For sound delivery, a pair of custom-made hollow earbars was used.

Briefly, a parieto-occipital craniotomy was performed to expose the left DCN by partial aspiration of the overlying cerebellar tissue. A four-shank 16-channel array (A4 x 4-3mm 100-177, 177 µm²/site; impedance = 1.3-3.8 MΩ, NeuroNexus Technologies) was inserted into the DCN. The right IC was then surgically exposed by partial aspiration of the over-hanging visual cortex tissue. A single shank 16-channel probe (A1 x 16-5 mm 100-177) was inserted into the IC along a dorsolateral to ventromedial trajectory at a 30-45° angle off the parasagittal plane in the coronal plane. Finally, the right AC was surgically exposed and a four-shank 16-channel array (A4 x 4-3mm 100-413, 413 µm²/site; impedance = -0.1 MΩ after activation) was inserted into the primary AC (Figure 4), in which neurons usually respond well to pure tones and have sharp tuning properties. The probe in the AC was lowered about 0.8 mm from the AC surface, in order to stimulate the pyramidal cell layer. The probe implanted in the AC for electrical stimulation and electrophysiological recording was mounted on a switching headstage. The probes in the DCN and IC were connected to chronic headstages for recordings only. All headstages were connected to the auditory workstation (Tucker Davis Technologies, System 3) that controlled stimulation and data acquisition. The output was fed into a 40 bit TDT neurophysiology base station that was controlled by an OpenEx software suite. Frequency tuning curves were obtained in response to tone sweeps delivered to the left ear by an electrostatic speaker. The tones bursts were 50 ms in duration and had 30 ms intervals with frequencies ranging from 4 to 30 kHz. Neural signals were preamplified and bandpass filtered (300-1,000Hz). The TDT preamplifier has a fixed gain (255x) with an input range of ±4.5 mV and a 16-bit signal resolution on the AD converters.

For ACES, single charge-balanced, biphasic, square-wave pulses (width = 50 µs), generated from RX7 microstimulator (RX7, TDT) and optically isolated (MS 16, TDT), were randomly delivered at 30 µA and 10 pps for a period of 1 min. A bipolar stimulation mode was adopted through activating adjacent electrode contacts. Multiunit activities were acquired in the DCN and IC before (10 min recording), during (1 min recording), immediately following ACES (10 min recording, termed as P00) and again 30 min afterward (10 min recording, termed as P30). All data were stored in the hard drive for offline analysis. At the end of each experiment, the animal was euthanized and its brain was histologically processed to verify the electrode placement in the IC and DCN (Figure 4).

For data analysis, average activity rate in voltage events/second from each condition (before, during and after stimulation) was obtained by dividing its recording time. Since the current focus was on ACES-induced effects, normalized ratio values were obtained by dividing activity rates recorded during and after ACES by those recorded before ACES, respectively. The types of responses were categorized into excitatory (10% higher than baseline), inhibitory (10% lower than baseline) and no change (<10% compared to baseline activity rate). The percent incidence for each type of response was calculated to determine the effects of ACES on neural activity in the DCN and IC of control and exposed groups, respectively. For two animals, channel-to-channel coherence within and between the DCN and IC was calculated. For each channel, the signal consisted of the time sequence of multunit spike events (snippets) with amplitudes equal to the RMS amplitude of the corresponding spike events. The time resolution of separate events was the inverse of the sample rate (25 kHz). Channel-to-channel coherence was calculated and averaged for frequencies between 5 and 50 Hz to avoid 60 Hz coherence.
artifact. Each 10 min recording was divided into successive segments of 2048 time points (0.79 seconds). These segment coherences were averaged to obtain the final results. For each data segment, coherence was calculated using FFT spectra with the data windowed using a set of 5 prolate spheriodal wave functions.

Figure 4: A. Electrode placement in the right AC. Histology with Di-I showing tracks of electrode probes in the right IC (B) and in the left DCN (C)

Results

The results demonstrated complex ACES-induced responses in the left DCN and right IC during and after electrical stimulation (Figures 5-7). The complexity was reflected by the occurrence of excitation, inhibition as well as no change compared to before ACES.

Figure 5: Examples showing changes in spike activity during, immediately (P0) and 30 min after (P30) ACES. Note ACES-induced suppression (left column), excitation (middle column) and no change (right column). Upper row, snippets; Lower row, normalized data over pre-ACES baseline.
Figure 6: Percent incidence plots showing excitation, inhibition and no change, in response to during-, immediately and 30 min after ACES. Note that ACES induced more suppression in the DCN and IC of noise-exposed animals, compared to controls.

For during-ACES, 5.6%/39.7% (DCN/IC) responses in the controls and 28.7%/51.3% in noise-exposed animals were inhibitory, while 81.7%/56.9% in controls and 43.6%/46.1% in exposed animals were excitatory, respectively (Figure 6). 12.7%/3.4% (DCN/IC) in the controls and 27.7%/2.6% of exposed animals did not show significant changes (Figure 6). The percent incidence of excitatory responses significantly decreased whereas that of inhibitory responses significantly increased in exposed animals compared to controls. However, the ACES-induced excitatory responses were still predominant in exposed animals (compare columns B and C in Figure 7). In addition, as expected from travel distance, the latency of IC responses (average latency = 5 ms) was shorter than that of DCN responses (average latency = 7 ms; Figure 7).

Following ACES, the percent incidence of the induced excitatory responses at the P00 time point was 66.2% and 55.2% in the DCN and IC of controls and 42.6% and 2.63% in exposed animals, respectively. The percent of the induced inhibitory responses was 7.1% and 17.2% in the DCN and IC of controls and 28.7% and 76.3% in exposed animals, respectively. Such a trend was maintained at the P30 time point, with 49.3% and 36.2% responses in the DCN and IC in controls was excitatory whereas 38.3% and 75.0% in exposed animals was inhibitory (Figure 6).

In addition to both induced excitatory and inhibitory responses, there were channels that did not manifest significant changes (Figure 6). Although the responses at during, P00 and P30 time points were mixed, ACES relatively induced more inhibitory responses in the DCN and IC in exposed animals compared to that of controls (see black bars for exposed animals in Figure 6). Such an enhanced inhibition was even more robust in the IC (See Column B in Figure 6).
Types of responses (During ACES)

DCN responses (During ACES)

IC responses (During ACES)

Figure: 7 During-ACES induced excitation, inhibition and no change in DCN and IC activity of control and exposed animals (Column A). Also note that the ACES-induced excitatory responses were significantly more robust than in controls, and that their latencies were longer in the DCN than in the IC (Columns B and C). Bin width = 1.0 ms.

In addition, our results from 2 animals showed that there was a significant decrease in coherence in the IC that was slightly more pronounced in the high frequency domain (Figure 8). In contrast, a slight enhancement of coherence was observed within the DCN and between the DCN and IC recording sites. The average coherence within the DCN was increased from 0.29 to 0.36 and decreased from 0.41 to 0.39 within the IC for this exposed animal. However, the average coherence between all DCN and IC recording sites was unchanged at 0.29 for before and after ACES. The results suggest that ACES tends to induce an increase in coherent activity within the DCN and suppresses coherent network activity within the IC in exposed animals. However, these coherence results only reflect the network activity corresponding to intermittent high amplitude spike events rather than the full spectrum of activity recorded at each structural site.

Figure 8: Examples showing ACES-induced significant suppression of coherence in the IC and slight enhancement in the DCN of hamsters.
Discussion

Our clinical data continued to provide supportive evidence that ACES is a promising approach for tinnitus management. The results from animal experiments demonstrated that ACES is a powerful approach for the modulation of neural activity in the DCN and IC, both which have been implicated in the aetiology of tinnitus. It has also been suggested using electrophysiology, imaging, and immunocytochemistry techniques that hyperactivity serves as the neural correlate for noise-induced tinnitus. Anatomically, the current animal data are in line with our previously reported results and are supported by the known projections from the AC to the DCN and IC.

Physiologically, it is also known that corticofugal modulation can shape the response properties of neurons (frequency, intensity, time & spatial domains) for better signal detection; and to adjust/select subcortical signal processing or lower brainstem responses through gain controls. Therefore, if hyperactivity in the DCN and IC contribute to the etiology of tinnitus, the currently obtained ACES-induced modulatory effects on the DCN and IC may have contributed to the suppression of tinnitus observed in patients.

Along this line, it has been shown that ACES suppresses tinnitus in patients by reducing hyperexcitability in the AC, increasing intracortical inhibition, and modulating the tonotopic map in the AC. In animal studies, cortical stimulation can change the functional organization of the cortex and produce widespread changes in regional synaptic activity within cortical and subcortical structures. Taken together, these lines of evidence favour the notion that ACES-induced suppression of tinnitus may involve simultaneous modulation of neural correlates of tinnitus at different levels.

The results from the current animal experiments demonstrated complex responses in both the DCN and IC of control and exposed animals during and after ACES. These have included excitation, inhibition and no change. The complex nature of ACES-induced responses in both the DCN and IC may be explained by the fact that a variety of neurotransmitters including glutamate, GABA, glycine and acetylcholine are involved in the neural pathways from the AC to the DCN and IC.

As a result of the complex innervations involving direct and indirect neural circuitry, it is expected that different types of neural responses are found following ACES. The current results with more inhibitory responses in exposed animals are consistent with the report that somatosensory stimulation more frequently induces inhibition in the DCN following noise exposure.

The enhanced inhibitory responses may be due to enhanced sensitivity following noise exposure, which may have resulted in the imbalance between excitation and inhibition in neural network. Such imbalance may ultimately develop into those neural correlates of tinnitus giving rise to tinnitus perception. Thus it is possible ACES contributes to the recovery of the lost balance between excitation and inhibition, yielding tinnitus suppression.

The fact that our data demonstrated significant suppression of activity rate in the DCN and IC suggest that ACES may have down-regulated the neural correlates of tinnitus,
contributing to ACES-induced tinnitus suppression in patients. If this is true, it remains to be determined in more detail as to how ACES modulates different neuronal circuitry in both the DCN and IC.

Results from coherence analysis showed a significant decrease in coherence within the IC and a slight increase in the DCN (Figure 8). The preliminary results suggest that suppression of tinnitus may involve different types of modulation of neural interactions with a scaled effect. In other words, ACES-induced modulation in different brain structures may not be necessarily uniform. The results in the IC strongly support previously published reports that hypersynchrony is a neural correlate of tinnitus,\textsuperscript{17,29} whereas the data from the DCN do not. However, coherence is not a measure of change in the rate of spike activity. Rather, in this study it quantifies the synchrony of spike activity between brain sites.

This information suggests that the effects of neuromodulation at different brain centres may vary in order to achieve the needed gating adjustment. Nevertheless, further experiments are needed to develop research in this area, especially the application of coherence to full spectrum data instead of working on snippets. More experience with coherence analysis within and across several brain sites will provide a more complete picture as to how ACES modulates neural interactions within and across different auditory centres.

\textbf{Future prospect}—Our data demonstrate that ACES suppresses tinnitus in patients and modulates neural activity in structures that have been implicated in the aetiology of tinnitus. Given the potential benefit of ACES to suppress tinnitus, we have developed a rat model in order to directly investigate the correlation between behavioural manifestation of tinnitus and electrophysiological data recorded from multiple channels and structures. We will investigate how neural correlates of tinnitus operate in neural network and how ACES modulates neural correlates of tinnitus. The results identify specific routes for targeted modulation of information flow and effective suppression of tinnitus.

There is an immediate need to understand how electrical stimulation using different frequency domains in the AC modulates coherence indices within and across auditory structures. This information will contribute to the optimization of stimulation strategies, to further clinical investigations and to develop specialized neural prostheses for effective suppression of tinnitus.

Finally, a parallel animal and human model should be pursued in order to accelerate research in neuromodulation to suppress tinnitus. Our clinical team at Henry Ford Health System and the animal research team at Wayne State University are working closely to address issues that are directly related to clinical problems while clinical practice benefits from findings obtained from animal experiments.

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Auditory attention and tinnitus
K Wise, D Singh, GD Searchfield

Abstract
Tinnitus may be linked to aberrant, attention-related processes. Reducing tinnitus annoyance may involve decreasing tinnitus’ attention-capturing properties. A broad overview is presented, to introduce the fields of attention and auditory attention toward understanding how they may contribute to tinnitus or be capitalized upon toward tinnitus remediation. A study based in the context of auditory attention and attention is presented, investigating the effect of performing attention tasks on tinnitus; toward linking attention theory with practical, tinnitus treatment. Results showed selective attention domain tests showed better control-group performance than the tinnitus-group for 2, focused attention tasks (p = 0.06 and p = 0.02). No significant difference was noted between tinnitus and control groups for divided attention tasks. Analysis of Comprehensive Attention Battery™ (CAB™) and Tinnitus Handicap Questionnaire scores showed evidence (p = 0.06) for greater tinnitus handicap; hindering selective attention task execution. Findings support the hypothesis that bothersome tinnitus and underlying, aberrant central processes contribute to tinnitus perception and disrupt selective attention processes. Tinnitus questionnaire scores may be predicative, indicating less selective attention resources available to tinnitus sufferers, explaining poorer performance. Attention training for tinnitus sufferers may address such deficits. A follow-up study comparing tinnitus sufferers and controls to determine if repeat administration of an attention battery may confound results, showed practice effects are a concern.

The fields of attention and auditory attention have benefitted from years of research—study invested toward understanding underlying physiology and function associated with a seemingly intangible, human ability—the ability to attend.

To introduce the realms of attention and auditory attention toward understanding how they have evolved; a broad overview is presented. The synopsis is followed by results from two preliminary studies, to demonstrate how attention and auditory attention might potentially contribute to tinnitus perception or severity. It concludes with the concept of attention training toward developing future, tinnitus remediation options.

Definitions: attention and auditory attention—Various levels or domains of attention have evolved toward qualifying and quantifying deficits or improvements in attention; especially when considering results from specialized test batteries or training regimens.

Focused: Capacity for actively attending and providing distinct, clear responses to perceptual stimuli.

Sustained: Capacity for unbroken attention; upholding a constant, deliberate response state.

Selective: Capacity for processing perceptual information attended to, while excluding remaining stimuli present.
**Alternating:** Capacity for quickly changing or alternating attention between perceptual stimuli or tasks.

**Divided:** Capacity for concurrent attending and responding to, numerous stimuli and tasks.

William James is acknowledged for providing us with a well-known historical description of attention:

“Everyone knows what attention is. It is the taking possession by the mind, in clear and vivid form, of one out of what seem several simultaneously possible objects or trains of thought”

(p. 404).

Since Descartes in 1649 felt movements of a particular endocrine gland correlated with the function of focusing human attention as one wished, the field of attention has continued to evolve. Although William James asserted, “Everyone knows what attention is...” cognitive psychologist, Styles, indicated it would be more accurate to state that “Nobody knows what attention is (p. 1),” highlighting continued disagreement among the psychological community regarding arriving at a clear-cut meaning for “attention” and all it may imply.

The concept of auditory attention employs aspects of the various definitions of attention and contemplates how we, as listeners, have the seemingly innate ability to instantaneously attend to specific parts or portions of auditory messages in an auditory scene; while discarding others. As the field of visual attention has received more research focus, comparatively, less is known about auditory attention.

**Selected history and early auditory attention models**—Aircraft pilots held one of numerous, combat roles during World War II, requiring management of multiple information streams; often arriving simultaneously through multimodal inputs. It become clear humans have a limited capacity to act on more than one perceptual information signal—arriving at the same time and/or concurrently to different sensory channels—visual or auditory. Experiments into the capacity of humans to handle and perform under stress began and so too, the science of attention and auditory attention research.

**Selective auditory attention**—Post-war, 1950s gave rise to attention and auditory attention-related research; with some studies forming psychological models and information-processing models for attention and selective auditory attention. Auditory experiments often involved “shadowing”—verbatim repeating/vocalizing of auditory information—or dichotic listening tasks. Tasks were selected to model central, attentional processes as our ears, unlike our eyes, are immobile; suggesting incoming perceptual information was cognitively processed rather than by motor or external processes.

In 1953, Cherry’s “Cocktail Party Problem” was posed, later becoming known as “Auditory Selective Attention”. A shadowing task was used, presenting two different auditory messages (Continuous speech) simultaneously via headphones; with participants required to attend as instructed to various continuous speech samples. The study showed that humans have the ability to selectively attend to one auditory message while effectively ignoring non-target speech information.
**Information processing models of attention**—Broadbent began a trend in how human, cognitive functions such as attention were analysed. To model the “perceptual bottleneck” caused by two, parallel inputs arriving at a sensory station at the same time, Broadbent proposed a selective, early filtering process that would allow certain perceptual information to move forward beyond short-term memory, while other inputs were left behind (See Figure 1.).

Deutsch and Deutsch rejected the concept of peripheral filtering in favour of information processing with “late selection” of a perceptual input stream; asserting there was no “filter”, all perceptual inputs are fully and completely processed. What was selected and attended to was determined by which input stimulus was more important; accounting for “breakthrough” of some information not being expressly attended to, as all inputs are processed (See Figure 1.).

Treisman conducted shadowing experiments during which, she noted some information presented to the non-shadowed ear “broke through” and was attended to. Treisman felt this supported the likelihood of an “attenuator”; processing perceptual information based on a hierarchy (Grammar, sentence structure and ultimately, meaning). If the system is under stress, meaning will not be extracted but more simple features (Speech or non-speech) are perceived (See Figure 1.)

![Figure 1: Comparison of three information processing theories of attention (Adapted from Eysenck and Keane, 2005, p.3).](image-url)

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Early neurophysiological study and attention—Pioneering neurophysiological research established that responses from neurons in the auditory cortex in cats can be greatly modulated by attention.\textsuperscript{20} Electrode sites were prepared under sedation, but cats were unrestrained and un-sedated during data collection. Single-unit responses were robust for stimuli the animal appeared to be attending to such as: Voice, noises from a mouse-toy or hissing (imitating a cat). Un-meaningful clicks, tones or background noise did not elicit significant response(s).\textsuperscript{20}

Foundations—what may influence auditory attention?—All sounds emanate from an “object” or an event; a source or origin. It can be crucial or practical to determine what auditory object is creating a particular sound and its location in the auditory scene.\textsuperscript{21}

Signal frequency and attention—An experiment toward understanding attention as it relates to signal frequency was undertaken, with participants instructed to attend and respond to a target frequency of 1000 Hz.\textsuperscript{22} Results were consistent with other studies investigating frequency and attention; finding that the pattern of attention was similar to a band-pass filter, revealing an “attentional filter” that passes those frequencies within the “attentional band”.\textsuperscript{8, 22}

Signal direction, discrimination and attention—A 1986 study showed no difference for detection rates for a 1000 Hz tone, whether or not the tone emanated from the target loudspeaker direction (Left). Despite attending to the anticipated, target direction, participants could detect the stimulus just as well when it actually came from the non-target loudspeaker direction (Right).\textsuperscript{23} Directional cueing’s role in discrimination was investigated via a two-loudspeaker array with participants encouraged to discriminate between a target, 1000 Hz tone and 800 Hz tone. Participants were cued to expect tones from the left loudspeaker; with tones emanating from the left 75\% of the time.

Results showed cuing the direction of the target tone aided in faster discrimination when the cue was correct and matched the expected loudspeaker. The control condition (Cued to directly in front) revealed slower responses, but not as slow as for incorrect cues. The results suggested directional cuing engages attentional processes aiding in discrimination. And, an auditory event is perceived regardless of location but once a position is chosen, attentional processes may assist with further cognitive processing.\textsuperscript{8, 22, 24}

Auditory scene analysis and auditory streams—Because there are often multiple, sound-rich events occurring simultaneously, we must cognitively divide them; reducing multi-faceted perceptual information into a discrete, mental representation or “auditory object”.\textsuperscript{21} In order to arrive at a cognitive representation of such complex perceptual information, we rely on extracting distinct combinations of auditory features from the sound spectra such as: Frequency, intensity, timbre, direction and timing. Then, sort feature information in conjunction with basic organisational principles based in cognitive psychology.\textsuperscript{21} Auditory scene analysis involves taking in auditory perceptual information and concurrently grouping it toward creating a meaningful mental representation of our environment; to regain discrete portrayals of each distinct item present in everyday surroundings.\textsuperscript{21}
The cognitive unit that corresponds to a solitary acoustic event can be considered an auditory stream. Auditory data informs our mental representations of events in the soundscape through “auditory streams” with the word “sound” reserved for the causal agent; as events can often contain more than one sound. The auditory streams operate to “bunch together” perceptual information, linking similar aspects toward further cognitive processing; to assist in clarifying the particulars associated with an acoustic event.

Gestalt principles—Gestalt principles are defined psychological principles of cognitive grouping. From the various features present in all sensations, the brain creates associations and forms cognitive groupings. Researchers have used Gestalt principles to explain how features of the acoustic soundscape could be cognitively grouped. Below (See Figure 2.) are visual analogues for three well-known Gestalt principles (Not inclusive).

The Principle of Similarity illustrates we are inclined to cognitively group the four, larger circles together, as they appear similar based on their larger size. The Principle of Proximity illustrates because two rows of circles are noticeably closer in proximity, we are inclined to group them as two, individual groups. The Principle of Continuity illustrates that instead of decomposing the image into four, discrete lines, we prefer to see it as a “plus sign” or cross.

![Figure 2: Three Gestalt Principles: Similarity, Proximity and Continuity.](image)

One approach to evaluating an auditory scene could be to use Gestalt principles to group sounds by their perceptual position. But, some researchers assert there is sufficient evidence to suggest that sound localisation is difficult for humans. Other researchers state problems with localizing sound may be conquered when the auditory feature of frequency is employed to aid in finding sounds in the environment; that per research, humans are demonstratively more inclined to group sound stimuli based frequency instead of sound position/location.

Figure-ground—Figure-ground describes another Gestalt principle wherein, Figure refers to an object of interest while ground is essentially the background. This principle is contingent upon contrast; as demonstrated below, the figure or word “legible” gradually improves in readability as the ground or background, is altered for contrast. Auditory attention is important toward emphasizing figure information over ground/background. (See Figure 3.)
Figure 3: Adjustment of contrast between the figure and ground improve legibility of the figure, “legible” (Adapted from Graham, 2008, p.3).

Informational masking—With excitatory masking or “energetic masking” target signals are essentially rendered inaudible at the auditory periphery before arrival at the cortex; due to signal and masker excitatory-pattern interaction at the level of peripheral structures. Informational masking is associated with higher-level, central processes occurring when a listener is unable to disentangle the elements of the target signal from a masker with similar informational content; making it difficult to mentally distinguish between target and masker or “signal uncertainty”. It has been argued that informational masking represents a failure of selective attention; the ability to attend to a target sound and de-attend to similar distracters.

Bottom-up and top-down processing—Bottom–up cognitive processing is set in motion by a stimulus, with attention focused toward an auditory object that perceptually, becomes prominent or “figure”. Afferent information processing is initiated unconsciously and rapidly by shifting attention to sounds featured over “ground” obscurity; e.g., a dog barking. Top-down, efferent-channel cognitive processing is more deliberate and complex, involving conscious shifting/focusing of attention intentionally on relevant, auditory objects requiring higher central function such as memory. For example, halting conversation as attention has been drawn to the doorbell then, deciding to open the door as it was remembered a friend would be visiting.

Mechanisms—centres involved in auditory attention—A functional magnetic resonance imaging (fMRI) study, investigated central structures activated when participants selectively attended to auditory stimuli and intermittently shifted attention when cued. Results revealed extended temporoparietal, superior parietal and frontal cortex activation and included frontal eye field/premotor cortex. Different portions of the brain were activated with top-down and bottom-up attention shifts: E.g., intraparietal sulcus and superior parietal lobule versus the ventromedial prefrontal cortex, respectively.
Measurement—objectively gauging auditory attention—The following methods have been used to either determine which brain centres appear to contribute to auditory attention or to quantify auditory attentional performance.\(^3,7\)

- Event-related potentials (ERP)
- Single-unit, multi-unit local field potentials (LFP)
- Electroencephalography (EEG)
- Magnetoencephalography (MEG)
- Functional magnetic resonance imaging (fMRI)
- Positron emission tomography (PET)
- Comprehensive Attention Battery\(^\text{TM}\) (CAB\(^\text{TM}\))

**Study 1**

A study was undertaken to determine if abnormal cognitive processes and tinnitus percept-focus associated with bothersome tinnitus, would reveal less attentional resources are available; resulting in compromised performance on selective attention tasks (Focused and divided attention).\(^2\) Psychophysical Tuning Curve (PTC) data was compared between participants with tinnitus and hearing loss and otologically-normal individuals, to gain insight into cochlear damage possibly associated with tinnitus.

**Methods**

Participants were recruited and randomly assigned to 3 groups: Tinnitus Sufferers (N = 16), controls (No tinnitus, but matched for hearing loss, N = 14) and otologically-normal participants (N = 17). Tinnitus-group participants identified as having bothersome tinnitus per questionnaire data and psychoacoustic tinnitus measures received audiometric assessment and were compared for performance on the Comprehensive Attention Battery\(^\text{TM}\) (CAB\(^\text{TM}\)); investigating performance on selective attention domain tasks. Tinnitus and control-group participants were compared via plotting Psychophysical Tuning Curves (PTCs) \(^2, 3\).

**Results**

PTC data was inconclusive for predicting cochlear damage associated with tinnitus. Selective attention assessments revealed the control group consistently performed better than the tinnitus group in two CAB\(^\text{TM}\) tasks examining selective attention (p = 0.06 and p = 0.02, respectively). No significant difference was noted between tinnitus and control groups for the CAB\(^\text{TM}\) divided attention task. Linear regression analysis for CAB performance and Tinnitus Handicap Questionnaire scores revealed \(R^2 = 0.22\); was also supported by multiple regression analysis, showing some evidence (p = 0.06) for greater tinnitus handicap hindering selective attention task performance.\(^2,3\)
Figure 4: Graph of performance for selective attention tests as compared between tinnitus and control groups.

- **AVMTm**: Auditory-visual Multiprocessing (AVMTm) Test
- **AVMTtot**: Combined scores for all AVMTm trials
- **DRTm**: Shift Discriminate Reaction Time (DRTm) Test
- **DRTtot**: Combined scores for all DRTm trials
- **STRa**: Stroop Interference Cancellation (STRa) Test
- **STRtot**: Combined scores for all STRa trials

Figure 5: Graph of Tinnitus Handicap as a function of performance on the Shift Discriminate Reaction Time Test (DRTm), a selective attention task.
Study 2

A follow-up study, conducted to investigate possible learning effects; to determine if tinnitus improvements potentially associated with auditory training tasks may actually be contributed to by re-administration of the CAB™.

Methods

Otologically-normal participants (N = 16) were recruited and assigned to control-group 1. The control group underwent baseline CAB™ administration and a final CAB™ re-administration, following a ten-day time period. The ten-day time period was inserted between pre and post measures, to simulate timing associated with a potential attention training program. Tinnitus sufferers and a second control group (No tinnitus but matched for hearing loss) are currently being assessed for effects of re-administration of the CAB™.

Results

Mean reaction time for all CAB™ tasks was 3.47 ms for baseline CAB™ administration, for control group 1. Post ten-day re-administration revealed a significant (p = 0.001) reduction in mean CAB™ reaction time to 3.16 ms. Mean hit rate (Correct responses) for all CAB™ tasks for the baseline CAB™ administration revealed 89% correct. CAB™ re-administration hit rate significantly improved to 97% correct (p = 0.010).

Figure 6: CAB™ baseline and re-administration scores: Mean task reaction time improvement.
Discussion

Study 1—The hypothesis: Bothersome tinnitus and underlying, aberrant central processes contributing to its perception disrupt selective attention processes; is supported. Tinnitus questionnaire scores may point to a lack of selective attention resources available to tinnitus sufferers, explaining poorer performance. Attention training for tinnitus sufferers may aid in rehabilitation.

Study 2—A significant “learning effect” was demonstrated with re-administration of the CAB™. 3 This is important to consider, when using the CAB™ to measure improvements following attention-focused training. 3 Potential effect(s) of re-administration of the CAB™ on tinnitus sufferers and a second, hearing loss-matched control group are still under investigation.

Potential directions—Attention training programs have been developed to address cognitive deficits following mild brain injury. 29 Attention Process Training (APT), was designed to address problems with attention and concentration that although often present, frequently went undetected or were misdiagnosed; incorrectly associated with memory difficulties. 9

A second version (APT-II) was subsequently developed and investigated the effects of administration on three participants with mild traumatic brain injury (MTBI) through ten weeks of individualized APT-II training, supported by six-seven weeks of instructive sessions. Results revealed enhanced attention and task speed for all participants, with restored mental abilities secure following six weeks cessation in APT-II training. 10

As tinnitus appears to compete with selective attention affecting cognitive ability, and attention training as a tinnitus treatment option continues to be considered and developed, perhaps an established training program such as the APT-II designed for
cognitive injury could have utility.\textsuperscript{10,30} It is clear that more research needs to be undertaken in the realm of attention and auditory attention as they pertain to: Tinnitus, tinnitus-related attentional deficits and potential training programs addressing both conditions.

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Tinnitus assessment
GD Searchfield, C Jerram

Abstract
Tinnitus is usually assessed clinically through a combination of history taking, audiological tests, psychoacoustic matching and questionnaires. It is thought that measurement of tinnitus is important to; understand mechanisms underlying tinnitus; evaluate treatments and change over time; demonstrate its characteristics; provide evidence for legal claims; guide treatment selection and implementation; and to categorize or subtype. There has been a recent upsurge of interest in attempts to subtype tinnitus. This interest is recognition that many tinnitus treatments are only effective in some types of tinnitus. It has been argued that there is a need for consensus on a common set of measures to be used in tinnitus assessment and evaluation of tinnitus treatments. In order for such measures to be successful internationally and to enable valid comparison of treatment outcomes from research undertaken in different countries they must be insensitive to cultural differences and measure dimensions important for tinnitus sufferers. Additional barriers to the acceptance of universal assessment include the existence of several widely used measures and parochialism.

We suggest a potential clinical framework for categorising tinnitus based on rating multiple assessment results across a three dimensional matrix of 1) psychology 2) psychoacoustic matching and 3) physiological. The matrix is intended to allow different assessment measures to be incorporated for a universal index of tinnitus. The present study focuses on the use of questionnaires in the psychological dimension. Three widely used questionnaires, the Tinnitus Handicap Questionnaire (THQ), the Tinnitus Handicap Inventory (THI) and the Tinnitus Severity Index (TSI) from patients attending a specialist tinnitus clinic in New Zealand are compared with international data. All three questionnaires were valid in the New Zealand context, but there were some differences in the data that suggested sensitivity of these questionnaires to different cultural settings.

Assessing a phantom perception—People with tinnitus are often treated as a homogenous group. This approach fails to recognise that tinnitus occurs for many reasons and from different injuries. There is a need for clinicians to be able to categorise tinnitus similar to how psychologists and psychiatrists identify types of mental illness. Even with a well established diagnostic manual, such as the Diagnostic and Statistical manual of Mental Disorders (DSM, http://dsmivtr.ord/), subtyping some conditions can be difficult. Although a difficult proposition, a method to categorise tinnitus using different assessment tools would be very valuable for researchers and clinicians.

Because there is no definitive objective physiological measure of tinnitus, its measurement must consider multiple domains. This is a challenge faced by other sensory complaints such as pain. Lund and Lundeberg (2006) recommend 3 dimensions for pain assessment 1) sensory-discriminative 2) affective-motivational and 3) cognitive-evaluative. We suggest 3 dimensions for measuring tinnitus 1) physiological 2) psychoacoustical and 3) psychological. These “3P’s” in turn
contribute to categorisation based on 1) audiometry and diagnostic measures 2) psychoacoustic matching and 3) questionnaires (Figure 1).

**Figure 1:** Three-dimensional model of tinnitus assessment

**Table 1:** A potential classification system based on site of lesion leading to tinnitus (Based on Zenner and Pfister, p19)

<table>
<thead>
<tr>
<th>Classification</th>
<th>Pathogenic model example</th>
</tr>
</thead>
<tbody>
<tr>
<td>Objective</td>
<td>Venous</td>
</tr>
<tr>
<td>Subjective</td>
<td>Middle ear pathology</td>
</tr>
<tr>
<td>Conductive</td>
<td></td>
</tr>
<tr>
<td>Sensorineural</td>
<td></td>
</tr>
<tr>
<td>Type I</td>
<td>OHC</td>
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<tr>
<td>Type II</td>
<td>IHC</td>
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<tr>
<td>Type III</td>
<td>Transmission</td>
</tr>
<tr>
<td>Type IV</td>
<td>Strial</td>
</tr>
<tr>
<td>Central</td>
<td></td>
</tr>
<tr>
<td>Primary</td>
<td>Traumatic Brain Injury</td>
</tr>
<tr>
<td>Secondary</td>
<td>Phantom</td>
</tr>
</tbody>
</table>

**Physiological categorisation**—A physiological based categorisation attempts to relate the cause of an individual’s tinnitus to a specific physiological mechanism and/or site of lesion. Tests widely used at present include audiometric measures of hearing sensitivity, middle ear status, hair cell function and central processing. The ability to identify physiological events related to tinnitus which can be objectively
recorded and quantified would be invaluable for tinnitus classification. Hearing loss is an important predictor for the incidence of prolonged tinnitus\textsuperscript{11} and pitch does appear to relate to the frequency region of hearing loss in many cases.\textsuperscript{12,13}

Considerable effort has been spent identifying mechanisms underlying tinnitus and developing pathological models (Table 1). An objective tinnitus measurement has been sought for both clinical and research use for some time.\textsuperscript{13} However otoacoustic emissions (OAEs), electrocochleography (ECoG), auditory brainstem response (ABR), late auditory evoked potentials (LAEPs) and imaging techniques are still limited in their ability to differentiate tinnitus from control groups. So, at present, our ability to categorise solely on a physiological basis is very limited.

**Perceptual categorisation**—In the absence of an objective measure of tinnitus researchers and clinicians have used psychophysical assessments of tinnitus qualities.\textsuperscript{1, 14, 15} A common core of measures (Tinnitus pitch, loudness, Minimum masking level and residual inhibition) have existed as a defacto standard since at least 1981.\textsuperscript{16} The measures provide information about the characteristics of the tinnitus. However tinnitus is the perception of sound, it is not in itself a sound. Tinnitus does not necessarily follow standard rules of sound perception.\textsuperscript{17} Although some of the neural networks activated with sound transduction are likely to be active in tinnitus generation they may not be activated in the same manner as sound. Psychoacoustical measures, such as loudness, do not always correlate strongly with self reported tinnitus impact.\textsuperscript{15} Psychoacoustical and psychological evaluations appear to measure different dimensions of tinnitus, so both are needed to characterise tinnitus.

**Psychological categorisation**—Most models of tinnitus generation recognise the important contribution that non-auditory centres make to tinnitus.\textsuperscript{18} It has been argued that without activation of emotional centres (such as the limbic system) tinnitus would be easily habituated.\textsuperscript{19} As a consequence, measuring the psychological effects of tinnitus is crucial to a complete assessment. The psychological dimension could include evaluation from a psychologist and/or use of standardised questionnaires. There are many self-report questionnaires to measure the psychological and social handicap of tinnitus. Patient ratings of tinnitus loudness, annoyance, sleep disturbance and interference with daily activities from self report questionnaires have been used to investigate patterns of tinnitus incidence and treatment outcome.\textsuperscript{20} The Tinnitus Handicap Questionnaire (THQ\textsuperscript{4}) Tinnitus Handicap Inventory (THI\textsuperscript{5}) and Tinnitus Severity Index (TSI\textsuperscript{6}) are three popular questionnaires developed in, but used widely outside of, North America.

The THI consists of 25 items comprising 3 subscales, measuring functional, emotional and catastrophic effects of tinnitus. Scoring for the THI consists of a three point rating scale; “Never” (0) “Sometimes” (2) and “Always” (4). Scores are summed for a total possible score of 100. The THQ consists of 27 items with responses to items (eg. “I do not enjoy life because of tinnitus”) scored on a 100-point rating scale from “Never” (0) to “Always” (100). A total average score is calculated, as well as average scores for each of the three factors. The 12-item TSI measures the effect of tinnitus on work and social activities and overall quality of life. The 12 items of the TSI are totalled for a single severity index. There have been 2 versions of the TSI the original\textsuperscript{6} using 3 and 4 point scales and a modified version\textsuperscript{21} using a primarily a 5 point scale, with one 4 point and two 3 point questions.
The present study represents an evaluation of the THI, THQ, TSI as potential candidates for inclusion as measures for the psychological dimension for the proposed 3-axis matrix. The study attempts to answer the following questions: (i) Are tinnitus questionnaires designed for the USA valid for New Zealand? (ii) How highly are data from different questionnaires correlated to each other?

Method

The methods used in this study were approved by The University of Auckland Human Participants Ethics Committee.

Copies of the THQ, THI, and TSI were administered by post to tinnitus clients approximately one week prior to their attending The University of Auckland’s Hearing and Tinnitus Clinic. Clients also completed a history questionnaire which recorded several self-reported client variables using numerical rating scales. These scales were: tinnitus duration; degree of awareness of tinnitus; how often people experienced tinnitus; the degree to which they considered their tinnitus to be a problem; and the subjective loudness of their tinnitus. Participants were instructed to complete the questionnaires prior to attending the clinic, and to bring them to their appointment. The study sample consisted of 146 males and 111 females (mean= 58.8 years, ranging in age from 15 to 88 years) no selection criteria were applied. The participants included those experiencing tinnitus as a minor complaint through to those with persistent chronic tinnitus as their primary complaint. All participants underwent puretone audiometry on attendance at the clinic.

Analysis—Client data was analysed using SPSS 12.1 for Windows software. To determine whether the domains of tinnitus measured by the questionnaires were sensitive to different cultural groups, factor structure for each was determined for the present study data and compared to that reported elsewhere in the literature. Principal components factor analysis with varimax rotation was used to define the factor structure. In addition, for THQ and THI responses the original factor structures were compared with results in the literature from different populations. To determine whether the individual item content of the questionnaires measured the same domains of tinnitus, correlation analysis was performed between item mean scores for the three questionnaires. Finally, questionnaire factor scores were tested for correlation with data from the subjective rating scales to evaluate clinical relevance.

Results

THQ—The factor analysis of the THQ results in the population studied showed there were four factors which each explained over 5% of the variance (Table 2). Factor 1 (Emotional and behavioural tinnitus effects) consisted of 14 items (40.3% of variance) and was identical to that of Kuk, et al.\(^4\) Factor 2 (Tinnitus effects on hearing) consisted of 6 items (11.7% of variance) and differed from Kuk et.al\(^4\)’s equivalent by the dropping of items 5 and 10, and inclusion of item 7. Factor 3 (Social effects) consisted of items 5, 10 and 15 (5.9% of variance). Factor 4 (outlook on tinnitus) consisted of items 8 and 12 (5.1%). Factor 4 had poor internal consistency. Subscale means for the THQ were significantly correlated to each other and to the total mean score (Table 3) the correlations being of higher magnitude in the present study than the equivalent found by Kuk et al\(^4\).
Table 2: Factor analysis of participant responses to the THQ. Items with Factor loadings of 0.5 or over are highlighted in bold

<table>
<thead>
<tr>
<th></th>
<th>factor 1</th>
<th>factor 2</th>
<th>factor 3</th>
<th>factor 4</th>
</tr>
</thead>
<tbody>
<tr>
<td>THQ1</td>
<td>0.64</td>
<td>0.21</td>
<td>0.21</td>
<td>-0.17</td>
</tr>
<tr>
<td>THQ2</td>
<td>-0.22</td>
<td>0.35</td>
<td>0.11</td>
<td>0.03</td>
</tr>
<tr>
<td>THQ3</td>
<td>0.2</td>
<td>0.77</td>
<td>0.32</td>
<td>0.04</td>
</tr>
<tr>
<td>THQ4</td>
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<td>0.81</td>
<td>0.29</td>
<td>0.11</td>
</tr>
<tr>
<td>THQ5</td>
<td>0.25</td>
<td>0.33</td>
<td>0.65</td>
<td>-0.2</td>
</tr>
<tr>
<td>THQ6</td>
<td>0.1</td>
<td>0.86</td>
<td>0.18</td>
<td>-0.06</td>
</tr>
<tr>
<td>THQ7</td>
<td>0.37</td>
<td>0.66</td>
<td>0.31</td>
<td>-0.07</td>
</tr>
<tr>
<td>THQ8</td>
<td>0.25</td>
<td>-0.07</td>
<td>0.22</td>
<td>0.59</td>
</tr>
<tr>
<td>THQ9</td>
<td>0.7</td>
<td>0.2</td>
<td>0.24</td>
<td>0.09</td>
</tr>
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<td>THQ10</td>
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<td>0.83</td>
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<td>THQ12</td>
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<td>0.01</td>
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<tr>
<td>THQ13</td>
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<td>0.08</td>
<td>0.22</td>
<td>0.31</td>
</tr>
<tr>
<td>THQ14</td>
<td>0.73</td>
<td>0.18</td>
<td>0.27</td>
<td>0.19</td>
</tr>
<tr>
<td>THQ15</td>
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<td>0.1</td>
<td>0.51</td>
<td>0.22</td>
</tr>
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<td>THQ16</td>
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<td>THQ17</td>
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<td>0.09</td>
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<td>THQ18</td>
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<td>0.25</td>
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<td>0.1</td>
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<tr>
<td>THQ19</td>
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<td>0.07</td>
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<td>THQ20</td>
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<td>THQ21</td>
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<tr>
<td>THQ22</td>
<td>0.85</td>
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<td>-0.04</td>
<td>0.15</td>
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<tr>
<td>THQ23</td>
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<td>0.82</td>
<td>-0.06</td>
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<td>THQ24</td>
<td>0.74</td>
<td>0.027</td>
<td>0.13</td>
<td>0.19</td>
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<tr>
<td>THQ25</td>
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<td>-0.03</td>
<td>-0.03</td>
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<tr>
<td>THQ26</td>
<td>0.21</td>
<td>0.08</td>
<td>-0.04</td>
<td>-0.12</td>
</tr>
<tr>
<td>THQ27</td>
<td>0.8</td>
<td>0.32</td>
<td>-0.04</td>
<td>-0.05</td>
</tr>
</tbody>
</table>

Table 3: Correlation (Pearson r) matrix for THQ total and factor mean scores

<table>
<thead>
<tr>
<th>Factor 1</th>
<th>Factor 2</th>
<th>Factor 3</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Factor 1</td>
<td>0.63</td>
<td>0.70</td>
<td>0.86</td>
</tr>
<tr>
<td>Factor 2</td>
<td>0.72</td>
<td>0.88</td>
<td>0.92</td>
</tr>
</tbody>
</table>

The original Kuk et al.\textsuperscript{4} factor structure was then used to compare data to previous studies.\textsuperscript{22, 23} Mean overall THQ overall and subscale factor scores mean scores were lower than those found by Bosceau-Faure et al.\textsuperscript{23} but higher than Newman et al.\textsuperscript{5} The differences were significantly higher for the NZ subjects than for Kuk et al.\textsuperscript{4} (p<0.01, Table 4). Data was not available to assess statistical significance against Bosceau-Faure et al.\textsuperscript{23} and Newman et al.\textsuperscript{5} In all studies internal consistency (Cronbach’s alpha) was high for the overall questionnaire and the first two factors, but not for the third factor. These results indicate that the original factor structure of the THQ is statistically reliable across different population samples, but sensitive to differences in item responses across separate cultural groups.
THQ total and factor mean scores were tested for correlation with scores on several rating scales relating to subjective characteristics. Average hearing thresholds for the range 250-8000 Hz in both ears was significantly correlated to overall and factor means (Table 5) as they were in Kuk et al.\(^4\) Factor mean scores were correlated to duration of tinnitus, and Factor 3 scores correlated to the degree to which tinnitus represented a problem (Table 6). All three factors and the total score were correlated to tinnitus loudness.

**Table 4:** Comparison of THQ factor scores, standard deviation, range and internal consistency (Cronbach’s alpha) across several studies.\(^4\), \(^22\), \(^23\)

<table>
<thead>
<tr>
<th></th>
<th>Present study</th>
<th>Kuk et al</th>
<th>Bosceau-Faure et al</th>
<th>Newman et al</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Total score:</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>mean</td>
<td>44.8</td>
<td>32.4</td>
<td>52.7</td>
<td>36.9</td>
</tr>
<tr>
<td>SD</td>
<td>35.3</td>
<td>22.1</td>
<td>20.2</td>
<td>22.2</td>
</tr>
<tr>
<td>Range</td>
<td>30.2-61.9</td>
<td>20.6-64.5</td>
<td>10.7-89.6</td>
<td>2.2-87.9</td>
</tr>
<tr>
<td>alpha</td>
<td>0.96</td>
<td>0.94</td>
<td>---</td>
<td>---</td>
</tr>
<tr>
<td><strong>Factor 1:</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>mean</td>
<td>45.1</td>
<td>31.2</td>
<td>48.6</td>
<td>33.6</td>
</tr>
<tr>
<td>SD</td>
<td>28</td>
<td>26</td>
<td>26.3</td>
<td>25.1</td>
</tr>
<tr>
<td>Range</td>
<td>30.2-61.9</td>
<td>20.6-58</td>
<td>0-100</td>
<td>0.94-5</td>
</tr>
<tr>
<td>alpha</td>
<td>0.94</td>
<td>0.95</td>
<td>---</td>
<td>---</td>
</tr>
<tr>
<td><strong>Factor 2:</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>mean</td>
<td>45.5</td>
<td>28.4</td>
<td>59</td>
<td>34.4</td>
</tr>
<tr>
<td>SD</td>
<td>28</td>
<td>25.8</td>
<td>27.2</td>
<td>28.9</td>
</tr>
<tr>
<td>Range</td>
<td>30.2-61.9</td>
<td>24.3-43.8</td>
<td>0-100</td>
<td>3.9-13</td>
</tr>
<tr>
<td>alpha</td>
<td>0.95</td>
<td>0.88</td>
<td>---</td>
<td>---</td>
</tr>
<tr>
<td><strong>Factor 3:</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>mean</td>
<td>55.8</td>
<td>46.4</td>
<td>60.8</td>
<td>52.9</td>
</tr>
<tr>
<td>SD</td>
<td>29.5</td>
<td>21.1</td>
<td>18.3</td>
<td>22</td>
</tr>
<tr>
<td>Range</td>
<td>38.6-57</td>
<td>47.4-64.5</td>
<td>0-100</td>
<td>12.5-95</td>
</tr>
<tr>
<td>alpha</td>
<td>0.41</td>
<td>0.47</td>
<td>---</td>
<td>---</td>
</tr>
</tbody>
</table>

**Table 5:** The correlation (Pearson’s r) between THQ total and factor means and average hearing thresholds for the frequencies 0.5-4 kHz for the Present study and Kuk et al.\(^4\)

<table>
<thead>
<tr>
<th></th>
<th>Present study</th>
<th>Kuk et al</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>R average</td>
<td>L average</td>
</tr>
<tr>
<td>THQ 1</td>
<td>0.289</td>
<td>0.184</td>
</tr>
<tr>
<td>THQ 2</td>
<td>0.441</td>
<td>0.398</td>
</tr>
<tr>
<td>THQ3</td>
<td>0.402</td>
<td>0.323</td>
</tr>
<tr>
<td>THQ TOT</td>
<td>0.325</td>
<td>0.315</td>
</tr>
</tbody>
</table>
Table 6: The correlation (Pearson’s r) between THQ total and factor means and self-reported complaints

<table>
<thead>
<tr>
<th></th>
<th>long</th>
<th>aware</th>
<th>often</th>
<th>problem</th>
<th>loud</th>
</tr>
</thead>
<tbody>
<tr>
<td>THQ tot</td>
<td>0.15</td>
<td>0.03</td>
<td>0.13</td>
<td>0.22</td>
<td>0.44</td>
</tr>
<tr>
<td>THQ F1</td>
<td>-0.1</td>
<td>0.08</td>
<td>-0.07</td>
<td>-0.06</td>
<td>0.45</td>
</tr>
<tr>
<td>THQ F2</td>
<td>0.25</td>
<td>0.131</td>
<td>-0.03</td>
<td>-0.05</td>
<td>0.47</td>
</tr>
<tr>
<td>THQ F3</td>
<td>0.15</td>
<td>0.05</td>
<td>-0.11</td>
<td>0.29</td>
<td>0.27</td>
</tr>
</tbody>
</table>

THI—The THI factor structure is shown in Table 7. Factor item listings are shown in Table 8, along with the listings for Baguley & Anderson. Unlike the THQ, the factor structures differ considerably between studies. Mean total scores were also compared between data from the present and previous studies in Table 8. Total mean score for the present study is similar to that obtained by Zachariae et al. and Baguley & Anderson. All three means were considerably higher than that obtained by Newman et al, (1996). In all studies, internal consistency for total mean score were high (alpha> 0.8). For individual THI items, response endorsement rates were not significantly different across studies.

Table 7: Factor analysis of participant responses to the THI. Items with Factor loadings of 0.5 or over are highlighted in bold. The results from Baguley and Andersson are shown for comparison.

<table>
<thead>
<tr>
<th>THI item#</th>
<th>Component 1</th>
<th>Component 2</th>
<th>Component 3</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Present Baguley</td>
<td>Present Baguley</td>
<td>Present Baguley</td>
</tr>
<tr>
<td>1</td>
<td>0.37 0.63</td>
<td>0.64</td>
<td>0.35 0.42</td>
</tr>
<tr>
<td>2</td>
<td>-0.54 0.47</td>
<td>0.12</td>
<td>0.86 0.57</td>
</tr>
<tr>
<td>3</td>
<td>0.73 0.64</td>
<td>0.26</td>
<td>0.08</td>
</tr>
<tr>
<td>4</td>
<td>0.57 0.67</td>
<td>0.29</td>
<td>0.17</td>
</tr>
<tr>
<td>5</td>
<td>0.65 0.74</td>
<td>0.23</td>
<td>0.11</td>
</tr>
<tr>
<td>6</td>
<td>0.64 0.56</td>
<td>0.31</td>
<td>0.07</td>
</tr>
<tr>
<td>7</td>
<td>0.47 0.44</td>
<td>0.47</td>
<td>-0.13 0.52</td>
</tr>
<tr>
<td>8</td>
<td>0.25 0.59</td>
<td>0.08</td>
<td>0.11 0.46</td>
</tr>
<tr>
<td>9</td>
<td>0.38 0.71</td>
<td>0.11</td>
<td>0.72</td>
</tr>
<tr>
<td>10</td>
<td>0.65 0.77</td>
<td>0.32</td>
<td>0.17</td>
</tr>
<tr>
<td>11</td>
<td>0.48 0.46</td>
<td>0.53</td>
<td>0.11</td>
</tr>
<tr>
<td>12</td>
<td>0.61 0.81</td>
<td>0.31</td>
<td>0.35</td>
</tr>
<tr>
<td>13</td>
<td>0.53 0.65</td>
<td>0.33</td>
<td>0.09</td>
</tr>
<tr>
<td>14</td>
<td>0.73 0.75</td>
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<td>0.52</td>
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<td>15</td>
<td>0.18 0.57</td>
<td>0.83</td>
<td>0.09</td>
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<td>0.36</td>
<td>-0.01</td>
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<tr>
<td>17</td>
<td>0.65 0.69</td>
<td>0.17</td>
<td>0.48</td>
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<tr>
<td>18</td>
<td>0.25 0.67</td>
<td>0.08</td>
<td>0.14</td>
</tr>
<tr>
<td>19</td>
<td>0.38 0.11</td>
<td>0.72</td>
<td>0.65</td>
</tr>
<tr>
<td>20</td>
<td>0.3 0.59</td>
<td>0.58</td>
<td>0.23</td>
</tr>
</tbody>
</table>
Table 8: Mean score, standard deviation, and cronbach alpha values across several studies.5,24,25

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Total score (SD)</td>
<td>42.7 (26.2)</td>
<td>25.5 (20.5)</td>
<td>40.0 (22.3)</td>
<td>44.1 (22.8)</td>
</tr>
<tr>
<td>Alpha</td>
<td>0.91</td>
<td>0.93</td>
<td>0.93</td>
<td>0.81</td>
</tr>
</tbody>
</table>

TSI—Comparison of TSI data for the present study with that reported previously, required raw mean scores to be converted to percentages since the present study used a 3 point rating for 7 items and 4 point rating as originally developed6 as opposed to the 5 point (9 items) 4 point (1 item) and 3 point (2 item) scale developed later.21, 26, 27 Total mean percentage score was lower for the present study (58.7%) than that for Folmer & Yong-Bing27 (75.3%) and Folmer & Griest26 (63.5%). Individual item means for the present study and Folmer & Griest26 were not significantly different. Factor analysis of the TSI data revealed two factors. Factor 1 consisted of items 1, 2, 3, 4, 5, 7, 10, 11 and 12 and explained 59.3% of the variance (Table 9). This represented physical and emotional effects of tinnitus. Factor 2 consisted of items 6, 8 and 9 and 10, and explained 15.2% of the variance, representing the effects of tinnitus on activities. All TSI items were significantly correlated to the “Loud” subjective variable, with item 12 “Does your tinnitus interfere with sleep?” being correlated to “Problem”. TSI scores were not correlated with hearing thresholds.

Table 9: Factor analysis of TSI data. Items with factor loadings greater than 0.5 are highlighted

<table>
<thead>
<tr>
<th>TSI item #</th>
<th>Component 1</th>
<th>Component 2</th>
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</thead>
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</tr>
<tr>
<td>2</td>
<td>0.48</td>
<td>0.63</td>
</tr>
<tr>
<td>3</td>
<td>0.52</td>
<td>0.7</td>
</tr>
<tr>
<td>4</td>
<td>0.4</td>
<td>0.77</td>
</tr>
<tr>
<td>5</td>
<td>0.6</td>
<td>0.47</td>
</tr>
<tr>
<td>6</td>
<td>0.9</td>
<td>0.2</td>
</tr>
<tr>
<td>7</td>
<td>0.89</td>
<td>0.07</td>
</tr>
<tr>
<td>8</td>
<td>0.81</td>
<td>0.23</td>
</tr>
<tr>
<td>9</td>
<td>0.64</td>
<td>0.52</td>
</tr>
<tr>
<td>10</td>
<td>0.23</td>
<td>0.73</td>
</tr>
<tr>
<td>11</td>
<td>0.09</td>
<td>0.68</td>
</tr>
<tr>
<td>12</td>
<td>0.38</td>
<td>0.53</td>
</tr>
</tbody>
</table>
**Questionnaire comparison**—The THQ total mean score was significantly correlated (p<0.01) with THI (Pearson r = 0.373) and TSI (r = 0.6) scores as were TSI and THI scores (r = 0.45).

**Discussion**

This study aimed to contribute to the quest to develop “core” universally applicable measures of tinnitus. Data from the THQ, THI and TSI, three widely used questionnaires, were evaluated for comparison with each other, and individually compared to international results. In all three cases questionnaire structure reliability and internal consistency were high across studies.

The relevance of a questionnaire in a particular setting can be measured by comparing the data with other measures. THQ factor mean scores were correlated to the duration of the tinnitus, extent to which tinnitus was considered a problem and tinnitus loudness. In the New Zealand context an element of tinnitus handicap (THQ) correlated with hearing loss. Use of a questionnaire that incorporates some communication questions may assist clinicians in treatment options in cases of mild hearing loss (e.g. sound generators vs. hearing aids). The THI subscales and THQ Factor 3 scores were correlated to the degree to which tinnitus represented a problem. All three THI subscales and the total score were correlated to tinnitus loudness. The THI subscales were not correlated to audiometric thresholds. All TSI items correlated with tinnitus loudness and a question asking about sleep was correlated with the extent tinnitus was a problem.

For a questionnaire with established construct validity in one country but showing statistical alterations in structure in another country, the effect could be viewed as a form of cultural bias that reduces the questionnaire’s potential as an international standard measure. The THQ used with New Zealand subjects showed essentially the same factor structure as the original American questionnaire. The New Zealand THI data had a factor structure that differed substantially from the original. This sensitivity of the THI to changes in internal structure reduces comparability of results from subscales and brings into question its suitability as a standard. At present there is insufficient data in the literature to determine whether factor components of the TSI alter in structure across studies.

**Conclusion**

In the context of ascertaining whether an international standard for measurement of tinnitus is possible, this study has demonstrated the sensitivity of individual questionnaires to cultural differences, even across populations where English is the common language. Interpretation of results obtained in different centres using the same questionnaire should be interpreted with caution. Based on the present study and the available comparative literature, the THQ, THI and TSI could form the basis of a core set of standard measures. The THQ, in particular, remained structurally robust and internally consistent. An international standard measurement of the effect of tinnitus is a noble goal. Because tinnitus has a physiological cause, is perceived differently by individuals, and has varying psychological impact we believe that a thorough evaluation of tinnitus must include at least 3 dimensions 1) psychological 2) perceptual and 3) physiological.
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Sound therapies and instrumentation for tinnitus management

GD Searchfield, H Cameron, S Irving, K Kobayashi

Abstract

Stimulation of the auditory system with sound is a common tinnitus management approach used by Audiologists. Sound, or “acoustic”, therapy can be implemented using different instruments (e.g. hearing aids, sound generators, MP3 players) and different intensities of sound (e.g. partial or total masking, low level sound for habituation). The sounds recommended for tinnitus management vary in their temporal, spectral and emotion-evoking characteristics.

In a review of the literature we consider the spectral and temporal characteristics of stimuli commonly advocated for tinnitus relief and report sufferers’ perception of treatment sounds relative to tinnitus. The results of a pilot study of sound therapy preference are reported. Three sounds (music [Vivaldi Spring], rain, broadband noise) were presented over a short duration to 19 participants. For each condition we measured Minimum Masking Level (MML), Desired Level (DL), change in tinnitus annoyance and change in masker annoyance. Preferred masker type was assessed by comparing the percentage of participants who chose each type. This comparison showed that rain was the most preferred therapeutic sound stimulus.

The results of this and several other studies suggest that stimuli that vary over time are preferred over constant noise for tinnitus treatment. It is possible that time-varying sounds are more representative of the normal acoustic scene. The sound of greatest benefit to individuals varies, and therefore subjective impressions of therapeutic sound should be considered. Preference may vary according to individual psychology, attention and emotional aspects of the sound.

Tinnitus, sound, or acoustic therapy, involves using an external sound to reduce or eliminate the perception of tinnitus or modify individuals’ emotional reaction to it. Sound therapy can therefore be considered to consist of both physical (e.g. frequency, intensity) and psychological (e.g. relaxation) effects. Tinnitus Retraining Therapy (TRT), hearing aids, and masking use sound in some way.

Sounds that have been found to be effective therapeutically include; broadband and narrowband noise, both related and unrelated to tinnitus pitch; music, which is typically orchestral; nature and water sounds. Sound therapy can have immediate effects; reducing tinnitus audibility (e.g. masking) which can also potentially lead to habituation to the underlying tinnitus (e.g. TRT). Sound stimulation may also reverse or alter the abnormal cortical reorganisation thought to be responsible for tinnitus.

The effectiveness of different sounds for masking have been investigated in several studies. Broadband noise and narrowband noise of different centre frequencies have been compared to determine the importance of masker frequency content. The majority of research considering the effectiveness of different sound types has concentrated on the spectrum and intensity of sound to be used. Dynamic sounds, such as music and rain, which vary in intensity over time, have only...
recently been investigated. It appears greater benefit is obtained in reducing tinnitus annoyance is achieved using dynamic sound, compared to non-dynamic sound.\textsuperscript{2,3,8,16} Total masking, partial masking and masking at a mixing point between the sound and tinnitus have all been trialled without universal acceptance of one model of sound presentation.\textsuperscript{15} The participant’s desired level, a balance between tinnitus effectiveness and comfortable listening, is another potential intensity for applying sounds. In this study we consider the effectiveness of dynamic and constant sounds for masking at the participant’s desired level.

It is important not to confuse the term ‘masking’, as used in tinnitus masking, with the masking used in audiological testing. In audiology, masking occurs largely at the level of the cochlea, due to interaction of travelling waves of the stimulus and masker. Tinnitus is a neural representation of a sound, not a physical sound wave, meaning masking cannot occur at the level of the cochlea. Instead, tinnitus masking must occur through another, central, mechanism. This conclusion is supported by many studies showing that masking of tinnitus does not behave like the masking of an external sound.\textsuperscript{2}

For some individuals, their tinnitus may be masked more successfully with narrowband noise centred at their tinnitus pitch, but for others there is no frequency relationship. In conventional masking, a pure tone will not successfully mask a broadband sound, but, in tinnitus masking, some studies have found that presentation of a pure tone will mask tinnitus that is perceived as a broadband sound.\textsuperscript{2}

Feldman\textsuperscript{11} first proposed categories of different masking patterns of tinnitus, with respect to the intensity required to mask tinnitus for masker frequencies. These were; Congruence, where masking of a small sensation level across the frequency range could mask tinnitus; Distance, where a high masker sensation level was required across the frequency range to mask tinnitus; Persistence, where no sound, no matter what intensity or frequency, could mask tinnitus; Convergence, where a high level of low frequency or low level of high frequency sound would mask tinnitus; and Divergence, where a low level of low frequency sound, or a high level of high frequency sound would mask tinnitus.\textsuperscript{11}

Similar categories of tinnitus masking patterns were observed by Tyler.\textsuperscript{17} It has proven difficult to predict which therapeutic sound will be most beneficial for the individual based on factors such as audiogram or tinnitus pitch match. Another important difference between the two masking types is that for conventional masking, presenting the masker to the ear contralateral to the signal presentation will not result in masking of the signal. However, for some individuals, contralateral masking is successful at masking unilateral tinnitus. Furthermore, unilateral masking has also been successful in masking bilateral tinnitus in some individuals.\textsuperscript{2,15,18}

Mitchell, Vernon and Johnson\textsuperscript{19} found that masking curves for tinnitus were not related to either audiogram shape, as participants with similar audiograms showed variation in their masking curves, or tinnitus perception, with those with similar reported tinnitus sensations also showing different masking curves.

Two common explanations for the neural mechanisms of tinnitus masking have been proposed. The first is the “line busy” explanation.\textsuperscript{2} This theory argues that the neural activity caused by the masking stimulus results in the neural pathway being ‘busy’,
similar to an engaged telephone line, so the neural response from the signal is not able to be processed. The second explanation is that the neural activity that results from processing of the masking stimulus suppresses any other activity in the same pathway. Another variation of this concept is that complex sound competes with tinnitus for cognitive resources.

Henry et al. defined relief from tinnitus by masking as any decrease in annoyance from the perception of tinnitus that occurs immediately and only while the masking sound is presented. Annoyance in this definition includes any negative emotional reaction to tinnitus, such as anxiety, frustration, or anger. Just as tinnitus can cause annoyance, so too can the sound prescribed to reduce it. In choosing a sound to treat tinnitus, a balance must be met between its effectiveness in affecting the tinnitus and the sound’s effect on the listener.

Acceptable masking occurs when the sound used to mask tinnitus is more tolerable than the tinnitus itself. Vernon, et al. looked at factors that influenced acceptance of masking as a treatment for tinnitus. These authors concluded that for masking to have an effect, two conditions must be met: 1) the sound must either cover or partially cover the perception of tinnitus, 2) the masking sound must be more agreeable to the patient than their tinnitus sound.

To obtain benefit from masking, the stimulus must be at a low enough intensity to not interfere with hearing and communication. It must also be at a level where it is non-intrusive, and able to be ignored. Unfortunately, there is no known method for predicting the most effective masking stimulus for an individual.

The sounds traditionally used for tinnitus therapy have been various bandwidths of noise with little variation in sound pressure as a function of time. The use of dynamic sound as a tinnitus masker has undergone limited evaluation. Henry et al. conducted a study to evaluate the benefit of different nature, water and air sounds, (‘E-Nature’, ‘E-Water’ and ‘E-Air’). The effectiveness of the different sounds was assessed via stimulus and tinnitus annoyance rating scales, and by measuring the sensation level which produced the greatest relief from tinnitus.

Two sounds, E-Nature and E-Water, provided a significantly greater reduction in annoyance compared to the other sounds. The authors concluded that this difference occurred due to the dynamic nature of the E-Nature and E-Water sounds, and hypothesised that the peaks in amplitude over time in their two dynamic stimuli may have acted in a similar manner as the brief impulses of noise, resulting in residual inhibition for a short time. This study also indicated the large amount of individual variation in the success of tinnitus maskers. Of the 21 participants, 11 participants (52%) found that at least one of the sounds did not provide any tinnitus relief, and four (19%) found that at least one of the sounds increased their tinnitus annoyance. This emphasises the importance of identifying particular sounds which provide greatest amount of tinnitus relief on an individual basis.

Music is another dynamic sound that has proven successful for tinnitus treatment. Therapy from music is thought to be successful due to music’s ability to induce relaxation and divert attention. The emotion evoked by music depends on certain of its elements. These elements include; tempo, mode, key, melody and
rhythm. The most important of these for evoking happiness or sadness are tempo and mode.\textsuperscript{23,25,26}

Hann et al.\textsuperscript{16} investigated the short term effect of music on tinnitus, specifically looking at the effect of tempo and mode. In this study, four pieces of orchestral music were compared to broadband noise and silence. The results showed that two musical pieces were most effective for providing relief of tinnitus. These were Bach’s ‘Air on a G string’ and Vivaldi’s ‘Spring’. Both pieces were played in the major mode\textsuperscript{16}. This study supported the use of music in tinnitus treatment, and highlighted the potential benefit of using dynamic stimuli, consistent with the findings of Henry et al.\textsuperscript{8} The importance of selecting a masker that is not annoying to the tinnitus patient was also highlighted. Hann et al.\textsuperscript{16} found a relationship between ratings of stimulus annoyance and ratings of tinnitus annoyance, with the stimuli with lower annoyance ratings resulting in lower tinnitus annoyance ratings.

Although there has been limited investigation into the use of music as a sound therapy for tinnitus, the studies that have been performed suggest that, for many individuals, music could be beneficial at reducing tinnitus annoyance\textsuperscript{16,27}. However, as with other potential stimuli for masking, there is some individual variation in success, and type of music that will be the most effective\textsuperscript{16}. These studies, along with previously mentioned studies looking at the use of broadband, narrowband and dynamic sound for tinnitus masking, highlight the importance of considering individual preference when deciding the most appropriate treatment for tinnitus.

Previous studies have highlighted the need to consider the type of noise used for masking, due to the high degree of individual variation in frequency content of optimal masking noise.\textsuperscript{13,20} The results of Henry et al.\textsuperscript{8} and Hann et al.’s\textsuperscript{16} studies highlight the benefit that could be obtained if tinnitus treatment devices were able to present sound other than broadband or narrowband noise. Hann et al.\textsuperscript{16} compared several pieces of music to noise, Henry compared noise to nature sounds. The current study compares one noise recording, a natural sound (rain), and music.

**Methods**

The methods outlined in this section were approved by The University of Auckland Human Participants Ethics committee.

**Participants**—A total of 19 participants were used in this study, aged between 24 and 68 years, with a mean age of 54 years. There were 12 males and 7 females. All participants had continuously present tinnitus, no greater than a moderate hearing loss across the majority of frequencies, and no unilateral hearing loss or significant asymmetry in hearing loss. Persons with mixed or conductive hearing loss were excluded.

**Equipment**—A Welsh Allyn\textsuperscript{TM} otoscope was used first to evaluate the external ear canal. All audiometry and tinnitus pitch and loudness match assessments were conducted in a sound treated booth (ANSI S3.1 -1997). A Grasson-Stradler GSI-61 Clinical Audiometer was used, with E.A.R\textsuperscript{TM} tone TM insert earphones and adult sized tips, to obtain air-conduction results from 250 to 8000 Hz and for speech audiometry. Sennheiser HDA 200 headphones were used for high frequency audiometry (10 000 to 16 000Hz). For bone conduction testing a B-71 bone vibrator was used. Tympanometry was performed using a Grasson Stradler GSI-33 Middle Ear Analyser. Masking testing was administered using a Dell Optiplex GX280 computer, with sound output connected to a PA5 Programmable Attenuator, by Tucker-Davis Technologies, played through the HDA 200 headphones.

The masking sounds used in the experiment were music, rain and white noise tracks. The music track was a commercially available version of “Spring” by Vivaldi in the major mode was used in the music condition. This particular piece of music was chosen based on the aforementioned study by Hann et
which found this music to result in a significant reduction in tinnitus annoyance. The rain track was a recording of rain, obtained using a Sony DAT recorder and a hand held Analyser Type 2250 (Brüel and Kjøhl). The white noise track was generated using Cool Edit Pro 2.0.

Audiometry—Audiometry was performed for each participant, including pure tone audiometry from 250 Hz to 8000 Hz and high frequency audiometry at 10000, 12500, 14000 and 16000 Hz. Inter-octave frequencies of 1500, 3000 and 6000 Hz were tested if there was a difference in threshold between octaves of greater than 15 dB. Bone conduction audiometry was conducted from 500 to 4000 Hz if necessary, in order to determine the type of hearing loss. Tympanometry was performed bilaterally, to check the status of the middle ear system.

Baseline tinnitus annoyance and loudness rating—Following the pitch and loudness match testing each participant completed two rating scales a “Tinnitus Loudness Scale” (“Not audible” (0), to “Medium Loudness” (5), to “Very Loud” (10)) and a Tinnitus Annoyance Rating Scale (“Not Annoying” (0), to “Medium, Annoyance” (5), to “Very Annoying” (10)). Participants were instructed to mark a point anywhere along the continua that corresponded to how annoying they perceived their tinnitus to be at that point in time.

Comparison of masker stimuli and different presentation locations—For each sound type the participant’s threshold was found in 0.1 dB steps, using an ascending technique based on the Modified Hughson-Westlake technique. The amplitude was then increased to a level at which the sound covered the participant’s tinnitus, using a similar procedure and was then recorded as the minimum masking level. The threshold was then subtracted from this level to give the minimum masking sensation level. The preferred level of sound for masking was then assessed using the same procedure, and recorded as the desired level. The desired sensation level was obtained by subtracting the threshold from the desired level.

Rating scales—Following each masking presentation participants completed two rating scales while they listened to the masking stimulus at their desired level. The first was a rating of tinnitus annoyance (see above). The second was the Stimulus Annoyance Scale (“Not Annoying” (0), to “Medium Annoyance” (5), to “Very Annoying” (10)). The order of presentation was randomised using random number tables generated by Microsoft Office Excel 2003. Following the presentation of all the masking stimuli, each participant was asked which of the three sounds they preferred for masking their tinnitus, i.e. which they found the most effective.

Data and statistical analysis—The data for each participant was recorded using Microsoft Office Excel 2003. Correlations were calculated between each of masker annoyance; change in tinnitus annoyance; minimum masking level and desired sensation level.; and a multivariate general linear model was used to determine differences between masker types, subjects and their interaction for each of the output measures.

Results

Across all measures, results varied significantly across participants (F(5, 47) = 3.64; p < .05) and masker (F(15, 147) = 4.44; p < .001), suggesting that there was a high degree of individual variation, and that the maskers were of varying efficacy. No interaction was seen between participant and masker [F(10, 96) = .393; p = .94], suggesting that listeners were affected by each masker in a similar way. The effects of interest are described in more detail below.

In order to determine the tolerability and efficacy of each sound as a masker, subjects were asked to set each sound at a level that they would like it to be presented in order to manage their tinnitus. The chosen intensity, in dB SL, was recorded and compared across participants (Fig.1). Statistical analysis showed a strong effect of masker type (F(3, 51) = 16.8; p < 0.001), with Music set to the highest level, followed by Rain, and the lowest levels set for the Noise masker. Individual differences between subjects were also marginally significant (F(1, 51) = 3.7; p = .06).
The minimum intensity required to mask tinnitus (minimum masking level or MML) was also measured for each of the three masking sounds (Fig. 2). Despite large individual variation (F(1, 51) = 7.26; p = .01), there was a significant difference between the MMLs of each masker type (F(3, 51) = 19.12; p < 0.001). Consistent with the high correlation between MML and DSL (r(55) = .943; p < .001), Music had the highest MML, followed by Rain and then Noise.

**Figure 1:** Participant desired sensation levels for each masker type. Each greyscale shade represents one participant

**Figure 2:** Minimum masking levels for each masker type. Greyscale shades indicate participant
In order to ascertain the relationship between the level that was required to mask the tinnitus (MML) and the level which was chosen for each masker by the participant for tinnitus management, the MML was subtracted from the DSL to get the Level Discrepancy (Fig. 3). Positive values indicate that the desired sensation level was higher than the MML, suggesting that the masker would be effective at its desired level.

Negative values indicate that the desired level was not intense enough to mask the tinnitus. Once again, there was a degree of individual variation ($F(1, 51) = 3.2; p = .07$). Of the three maskers, Rain was the most effective and accepted masker, as evidenced by the small amount of negative values, as well as the larger positive values. The latter indicates that the participants could turn the masking sound down from its desired level and masking would still be achieved.
Masker Annoyance was recorded on a VAS (0 = not annoying, 10 = very annoying) for each masker type. Masker annoyance did not vary across listeners (F(1, 51) = .32; p > .05), but there was a significant difference between the annoyance of each masker (F(3, 51) = 7.88; p < .001), with the Rain masker considered generally less annoying than the other maskers, and Noise being statistically more annoying than the other maskers.

One of the key factors in assessing the efficacy of a tinnitus masker is its ability to relieve the symptoms of tinnitus. In this study, a VAS was used to determine tinnitus annoyance when each masker was used (Fig.5.). Tinnitus annoyance and masker annoyance showed a correlation (r(55) = .366; p < .01). There was also a significant effect of masker in the tinnitus annoyance rating (F(3, 51) = 10.95, p < .001). The Rain masker showed the lowest tinnitus annoyance rating, suggesting that it was the most effective at decreasing patients’ annoyance to their tinnitus.

**Figure 4:** Visual analogue scale scores of masker annoyance. Greyscale shades signify different listeners
**Figure 5:** Annoyance of tinnitus with the use of each masker. Greyscale shades indicate participant.
The change in tinnitus annoyance compared to the baseline rating was also compared for each masker type (Fig. 6.). Statistical analysis showed that the reduction in tinnitus annoyance was not participant-dependent \[ F(1, 51) = 1.96; p = .167 \], but that it was related to masker type \[ F(3, 51) = 4.22; p = .01 \]. In particular, the Rain masker caused a greater decrease in tinnitus annoyance from baseline (mean: -2.07), compared to Music (mean: -1.63) and Noise (mean: -1.63).
Aside from measures reported above, participants were also asked which of the three masking noises they would prefer to use to manage their tinnitus (Fig. 7.). Rain was chosen as the preferred stimulus by the greatest percentage of participants (58%), followed by Noise (26%), and then Music (16%).

**Discussion**

This study compared the effectiveness of three different sounds, music, rain, and white noise, as tinnitus maskers. For each masker type, minimum masking level (MML), desired sensation level (DSL), the level discrepancy between the two, the masker annoyance, change in tinnitus annoyance from baseline, and preferred masker type were determined. Previous research has found a relationship between ratings of masker annoyance and tinnitus annoyance, with lower masker annoyance ratings resulting in lower tinnitus annoyance ratings, although the causal nature of this relationship was not defined\(^\text{16}\).

A high degree of individual variation in perceived annoyance from the different stimuli was found in the present study, which supports the importance of considering individual perception when selecting a sound to used for tinnitus masking.\(^\text{16}\)

The fact that the level discrepancy was most positive for the Rain masker suggests that it was pleasant (or at least more tolerable) to more participants, as they chose to play it louder than the minimum level required to mask their tinnitus. The same applies to Music, but listeners found that, although pleasant in the majority of cases, it was harder to mask their tinnitus consistently, as the peaks and troughs due to the melody offered variable masking levels. This may have caused participants to require
a louder overall signal to reach the mixing point with their tinnitus. It was also more
difficult for participants to determine the mixing point when music was the stimulus,
compared to white noise and rain.3,27

This result for music is consistent with results discussed above for MML. A likely
explanation for the lower DSL for white noise, which is consistent with reports from
individual participants during testing, was that due to the high perceived annoyance
that most participants had for the white noise, many felt they would need to turn this
sound down in order to be able to listen to it to manage their tinnitus. This finding
supports the argument that states that for a masking annoyance to be accepted it
cannot be perceived negatively.17,22

It was not surprising that white noise was the least preferred masking stimulus, as this
sound was rated as significantly more annoying than the other two. However, results
showed that fewer participants preferred music as a masker than white noise (3
compared to 5 respectively). Many participants reported that they would not like to
use music as a sound therapy due to the peaks and troughs mentioned above, but also
because it captured attention too much with its increases and decreases in intensity.
However, one participant did report this capturing of attention as a reason for
preferring music over the other sounds. Music may also not have been preferred due
to the higher MML for this particular masking sound.

Henry et al.8 and Hann et al.16 found dynamic sounds to be more effective as maskers
compared to narrowband and broadband noise. It is therefore likely that the rain was
sufficiently dynamic to offer satisfactory masking, but did not add too great a
cognitive load, therefore providing substantial masking with a tolerable sound, which
is emphasised by its larger decrease in tinnitus annoyance from baseline and its lower
level of masker annoyance.

Therefore, it is possible that both perceived masker annoyance and intensity of sound
required to mask tinnitus was involved in selection of the most preferred masking
sound, and emphasises the importance of customising each masking sound to the
client when providing a therapeutic sound.

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Tinnitus perception and the effects of a self-programmable hearing aid on hearing fluctuation due to Ménière’s disease

C McNeill, A Taylor

Abstract
Fifty patients with Ménière’s disease rated their subjective perception of tinnitus loudness just before measuring their own hearing thresholds 3 times a day for a period of 8 weeks. Tinnitus loudness was recorded using a subjective scale 1-5. Hearing thresholds were recorded in dBHL as tested in-situ at 14 frequency bands using a portable programmer connected to custom fitted hearing aids. This equipment allowed the hearing aids to be automatically programmed to the given hearing loss at the end of each self-hearing testing. Patients were given the option to continue wearing the hearing aids and portable programmer to adjust for hearing fluctuation.

Data analysis showed no correlation between changes in tinnitus loudness perception and hearing fluctuation. Usage of self-programmable hearing aids caused a positive impact on tinnitus perception in this group: 20% reported not hearing their tinnitus while wearing their aids, 69% perceived their tinnitus softer and 11% did not notice any changes. Importantly, none of the patients in this group noticed an increase in tinnitus loudness while wearing optimally fitted hearing aids.

Tinnitus is one of the symptoms afflicting patients with Ménière’s disease along with fluctuating hearing loss, ear fullness and episodic vertigo. Hearing loss is known to impact on tinnitus severity. It was assumed that patients with Ménière’s disease would perceive their tinnitus as more severe when hearing thresholds were worse.

Hearing aids are recommended as treatment to reduce tinnitus perception. A survey amongst a group of 126 patients with tinnitus due to various aetiologies who wore optimally fitted hearing aids showed that 31% did not perceive tinnitus while wearing suitable amplification and 42% noticed a significant reduction in tinnitus loudness when with their hearing aids on.

Fluctuating hearing however, complicates the fitting of hearing aids in patients with Ménière’s disease but this maybe overcome by using a self-programmable hearing aid.

This study investigated the correlations of hearing fluctuation with changes in tinnitus loudness and the effects of a self-programmable hearing aid on tinnitus perception in a group of patients with Ménière’s disease.

Methods
Ethics—Ethical approval for this project was obtained from Macquarie University Ethics Review Committee (Human Research).

Subjects and protocol—Fifty participants were chosen if they had a hearing loss in at least one ear due to Ménière’s disease as diagnosed by a specialist in otolaryngology. Diagnostic criteria were based on Gibson’s 10 points scale with score equal or greater than 7.6 or the AAOHNS level of “Certain Ménière’s”. They also needed to be conversant with technology and prepared to test their own hearing at home several times a day for a period of at least 8 weeks. They were all given the option to wear the hearing aid(s) during the period of the study as long as those inexperienced with amplification followed a strict acclimatization protocol. The subjects who had not received amplification before were...
explicitly instructed to start by wearing the hearing aid for only one hour, building up an extra hour daily and not to wear the new instrument outside their homes or in a noisy environment for at least the first week. At the end of data collection they were given the option to continue wearing the hearing aids and portable programmers.

**Instruments and procedures**—The Widex Senso Diva and Inteo range of hearing instruments were selected because they interface with a portable programmer (SP3 and IP5) and allow in-situ unaided hearing threshold measurement (Sensogram™) through the hearing aid set in test mode. Sensogram results are used to automatically program the hearing aid for the given hearing loss, according to proprietor fitting algorithm.

Participants were fitted either monaurally or binaurally, according to their hearing loss, with hearing aids selected according to the available range for each individual’s needs. Participant’s preferences were considered when more than one hearing aid style was suitable. Ear impressions were taken by the audiologist and custom hearing aids (ITC or CIC) or ear moulds for BTE style were made by Widex Australia.

Hearing aid fitting protocol was based on the Widex proprietor’s procedure using the expanded Sensogram. The audiologist performed the fitting in the clinic, using the Widex Compass software connecting the hearing aid to a desktop computer via the Noah Link interface and the proprietor’s fitting protocol was followed using the “expanded Sensogram”. This protocol comprises of measuring the hearing thresholds in-situ (through the hearing aid) followed by the feedback test. The Widex Compass software automatically programs the hearing aids based on these two measurements. Fine-tuning of the hearing aids was performed as required, following the proprietor’s software guide.

**Self-hearing test and hearing aid programming using the portable programmer**—Participants were instructed to connect their hearing aids to the portable programmer device and to measure their own hearing thresholds performing an expanded Sensogram (Figure 1). The Widex IP5 interfaced to the hearing aid and set on “test mode” produces 14 frequencies of narrow band noises in 5dB steps. The instrumentation allows for accurate hearing test providing the hearing aid receiver and ear canal are free of debris and the procedure is conducted in a quiet environment.

![Figure 1: Photograph showing participant using portable programmer](http://www.nzma.org.nz/journal/123-1311/4040/)
Data collection—Hearing thresholds were measured in dBHL at 14 frequencies using 5 dB steps. Subjective perception of tinnitus loudness (without the hearing aid) using a scale 1-5 was recorded immediately prior to measuring the hearing thresholds in a quiet environment. Data was collected 3 times a day over a period of 8 weeks and recorded (Table 1).

Table 1: example of records obtained by one participant over a 24 hour period. *Meaning of grading 1-5 for tinnitus loudness perception: 1= no tinnitus, 2= mild tinnitus, 3 = moderate tinnitus, 4 = severe tinnitus, 5 = extreme tinnitus

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Self-tested hearing thresholds and grading of tinnitus loudness perception, as provided by participants, were entered into a Microsoft Excel spreadsheet and exported into SPSS software for analysis.

Results

Sixty-nine ears were fitted with hearing aids and the data from every ear provided by each of the 50 participants were displayed in a graph.

Only one single tested frequency out of the total 14 was used from each individual ear to facilitate the analysis of hearing fluctuation over time. The chosen frequency was the one presenting the greatest dB change for each individual’s ear.

Correlation between tinnitus loudness and hearing fluctuation—Graphs from the results of 69 ears were obtained showing hearing thresholds and tinnitus loudness fluctuation over time from data provided by the 50 participants. Observation of these graphs did not suggest any consistent relationship between hearing fluctuation and changes in perception of tinnitus loudness. An obvious feature of these results was the variability over participants. The coefficients varied from negative to positive.

Figures 3, 4 and 5 are examples showing hearing fluctuation at one single frequency and changes in tinnitus perception (Y-axis) over time (X-axis).

Y-axis shows hearing thresholds (dBHL) as measured at the frequency (Hz) with greatest hearing fluctuation for that individual ear, together with the variation of tinnitus perception graded 2, 3, 4 or 5 over the number of measurements (observations).

To increase visualization on the Y-axis, for the purpose of these graphs, tinnitus grading was re-classified as 10, 20, 30 and 40 (for subjective ratings of 2, 3, 4 and 5 respectively) and as zero (for participant’s ratings of 1).
The numbers on the X-axis represents each time a measurement was obtained (observations) by the participant during data collection.

Correlations are described at the bottom of each graph. A positive correlation indicates a tendency for hearing thresholds to worsen as tinnitus loudness increases, while a negative correlation means an improvement in hearing as tinnitus perception decreases.

**Figure 2:** Frequencies that presented the greatest hearing threshold fluctuation (X-axis) for right and left ear of individual participants (Y-axis).
Figure 3: Participant 12’s hearing thresholds and tinnitus fluctuation in the right ear during data collection. Correlation between hearing threshold and tinnitus was –0.229
Figure 4: Participant 14’s hearing fluctuation in the right ear and no changes in tinnitus during data collection.
Figure 5: Participant 15’s hearing and tinnitus fluctuation in the left ear during data collection. Correlation between hearing threshold and tinnitus was 0.202

Statistical analysis—The relationship between tinnitus loudness perception and hearing threshold fluctuation over time was further analysed by computing cross-correlations between tinnitus grading and hearing threshold at one single frequency over all observations. These were calculated separately for each tested ear of every subject.

Participants who did not report any variation in tinnitus perception during the study period were excluded from this analysis. First order differences were used to remove the effects of systematic changes over time in either of the measures on the correlations.

As noted in the analysis, there was a great variability of the results over participants. The means, medians, minima and maxima for the distribution of each cross-correlation are shown Table 2.
Table 2: means, medians, minima and maxima for the distribution of each cross-correlation

<table>
<thead>
<tr>
<th>Statistics</th>
<th>Cross Correlation</th>
</tr>
</thead>
<tbody>
<tr>
<td>2 Max threshold variation in</td>
<td>N</td>
</tr>
<tr>
<td>the left ear with Tinnitus</td>
<td></td>
</tr>
<tr>
<td>Mean</td>
<td>12701</td>
</tr>
<tr>
<td>Median</td>
<td>09601</td>
</tr>
<tr>
<td>Std. Deviation</td>
<td>229047</td>
</tr>
<tr>
<td>Minimum</td>
<td>-570</td>
</tr>
<tr>
<td>Maximum</td>
<td>627</td>
</tr>
<tr>
<td>5 Max threshold variation in</td>
<td>N</td>
</tr>
<tr>
<td>the right ear with Tinnitus</td>
<td></td>
</tr>
<tr>
<td>Mean</td>
<td>02042</td>
</tr>
<tr>
<td>Median</td>
<td>00019</td>
</tr>
<tr>
<td>Std. Deviation</td>
<td>216036</td>
</tr>
<tr>
<td>Minimum</td>
<td>-429</td>
</tr>
<tr>
<td>Maximum</td>
<td>564</td>
</tr>
</tbody>
</table>

These results do not suggest any consistent relationship between changes in tinnitus loudness perception and hearing threshold fluctuation for this group of patients with Ménière’s disease.

**Effects of hearing aids on tinnitus loudness perception**—Participants who chose to continue wearing the hearing aids after data collection were further contacted through a survey. A follow-up questionnaire reached 49 of them and 44 replied to a multiple-choice question. They were asked to rate their tinnitus loudness perception while wearing the hearing aids as either: a) louder, b) softer, c) the same, d) cannot hear it.

Figure 6 is a summary of the results. The majority of respondents (69%) reported hearing their tinnitus softer; 20% cannot hear it at all; 11% perceived no changes and none noticed the tinnitus any louder with their hearing aids on.

This survey indicated that optimally fitted hearing aids are useful as a tinnitus management tool for approximately 90% of these participants with Ménière’s disease.
**Conclusion**

This study demonstrated that tinnitus loudness in patients with Ménière’s disease may be reduced by self-programmable hearing aids in spite of any lack of correlation between tinnitus loudness perception and fluctuating hearing loss.

Self-programmable hearing aids were shown to be effective to address the complications of fitting hearing aids for individuals with fluctuating hearing losses. Optimally fitted hearing aids also showed to reduce tinnitus perception for patients with Ménière’s disease and the positive effects observed is no different than for other individuals with hearing loss and tinnitus due to other aetiologies.

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**References:**

Combination open ear instrument for tinnitus sound treatment

L Carrabba, G Coad, M Costantini, L Del Bo, O Dyrlund, S Forti, G Searchfield

Abstract

Tinnitus is a serious condition reducing the quality of life for a large number of people. In 2006 Del Bo et al.\(^1\) reported promising results by integrating open ear amplification with Tinnitus Retraining Therapy (TRT).

The aim of this study was to clinically evaluate the efficacy of sound stimulation delivered by a new open ear combination prototype hearing instrument integrated with TRT. This instrument consists of an amplification system with state-of-the-art signal processing and an advanced sound generator. This combination has been chosen to obtain optimal compensation for the subjects hearing loss and provide the most effective sound enrichment for use in e.g. TRT.

The sound generator included a number of unique features; a white noise sound generator with flexible frequency shaping capabilities and manual level control; a random amplitude modulation feature and an environmental steering feature. The amplitude modulation feature was designed to make the noise signal less monotone, while the environmental steering feature ensures that the noise signal is only applied in certain quiet situations.

The study was designed as a multi center study with 30 tinnitus patients (10 female and 20 male with mean age of 59 years) falling within Jastreboff’s tinnitus category 1 and 2.\(^2\) The mean of hearing loss (PTA) was 25 dB HL. After fitting the instruments TRT was administrated for 6 months and the effect of the treatments was evaluated using the Structured Interview\(^3\) and THI self-administered questionnaire\(^4\) after 3 and 6 months.

The evaluation results show that significant improvements are obtained within 3 months for the THI and all VAS scales. Between 3 and 6 months the improvements are not significant.

Methods

This instrument consists of an amplification part with advanced signal processing such as multi band wide dynamic rage compression, digital feedback suppression and noise reduction, and an advanced sound generator part. This combination instrument has been developed to obtain optimal compensation for the subjects hearing loss and provide the most effective sound enrichment for use in TRT.

The sound generator included a number of unique features; a white noise sound generator with flexible frequency shaping capabilities, manual control of the noise level, a random amplitude modulation feature and an environmental steering feature. The amplitude modulation feature was designed to make the noise signal less monotone and more pleasant to listen to, while the environmental steering feature ensured that the noise signal was only applied in certain quiet situations. Figure 1 shows a block...
diagram of the signal processing in the instrument. The noise level of the sound generator was adjusted individually to the subjects. First the thresholds for the noise signal was determined and then level was adjusted to the desired value in order to set the mixing point, which also could be adjusted by the patient itself via the volume control.

![Combi instrument block diagram](image)

**Figure 1:** Combi instrument block diagram

The study was designed as a multi centre study including the following sites: Fondazione Ascolta e Vivi, Milan, Italy; University of Naples, Italy; and University of Auckland, New Zealand.

30 tinnitus patients falling within Jastreboff’s tinnitus category 1 and 2. Subjects with mild and moderate medium sloping unilateral or bilateral hearing losses (Figure 2). 10 female and 20 male with mean age of 59 ± 11.167 years were included. All had suffered from tinnitus at least 6 month and patients with Ménière’s disease and middle or external ear disease were excluded.

After fitting the instruments Tinnitus Retraining Therapy (TRT) was administrated for 6 months and the effect of the treatments was evaluated using the Structured Interview and THI self-administered questionnaire. Two patients dropped out during the study.
Results

Assessment results were collected at fitting and after 3 months and 6 months. Table 1 shows the mean questionnaires’ scores (± standard deviation)

Table 1: Mean questionnaire scores ± standard deviation

<table>
<thead>
<tr>
<th></th>
<th>THI</th>
<th>Annoyance (VAS)</th>
<th>Intensity (VAS)</th>
<th>Life effect (VAS)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Initial</td>
<td>46.15 ± 18.268</td>
<td>6.90 ± 2.205</td>
<td>6.83 ± 1.838</td>
<td>5.39 ± 2.670</td>
</tr>
<tr>
<td>3 months</td>
<td>36.58 ± 23.342</td>
<td>3.78 ± 2.317</td>
<td>4.68 ± 2.547</td>
<td>3.26 ± 2.289</td>
</tr>
<tr>
<td>6 months</td>
<td>32.16 ± 23.670</td>
<td>3.34 ± 2.331</td>
<td>4.62 ± 2.311</td>
<td>2.72 ± 2.865</td>
</tr>
</tbody>
</table>

Figure 3 and 4 show the development in the 6 months time screen of THI score and structured Interview VAS score regarding ‘annoyance’, ‘intensity’ and ‘tinnitus effects on patients’ life’. Improvements between the initial scores and after 3 months are statistically significant (THI: \( p = 0.001 \); annoyance and intensity: \( p < 0.001 \); life effect: \( p = 0.009 \)). After 6 months, all differences are significant (THI: \( p = 0.001 \); annoyance and intensity: \( p < 0.001 \); life effect: \( p = 0.002 \)). There are not any significant differences between 3 and 6 months results.
Figure 3: THI self-administered questionnaire score

Figure 4: Structured Interview Score (VAS scales)
Discussion

The evaluation results show that significant improvements are obtained within 3 months for the THI and all VAS scales. Between 3 and 6 months the improvements are not significant.

Based on the evaluation results an open fitting combination prototype instrument seems to be an effective solution for sound enrichment in TRT for moderate sloping hearing losses, with a promising reduction of therapeutical time in comparison with sound therapy delivered by wearable sound generators or open hearing instruments.

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References:

Perceptual training of tinnitus
K Jepsen, M Sanders, GD Searchfield, K Kobayashi

Abstract
It is currently believed that the majority of tinnitus is a consequence of changes in the central auditory pathways following peripheral injury. This change in brain function is known as plasticity. Another form of plasticity occurs as a process of learning or training. The basis of perceptual training is that by exposure to sensory stimuli we learn, usually improving perceptual skills. Perceptual training usually requires the listener to be actively involved in listening and responding to training sounds, unlike sound therapy used in masking or habituation therapies where listeners are passively exposed to sounds.

In this paper various forms of perceptual training are introduced before describing Frequency Categorisation Training (FCT) and Frequency Discrimination Training (FDT) and their effects on Tinnitus and late Auditory Evoked Potentials (AEP). Twenty participants underwent assessment and training. Assessment of tinnitus handicap rating and the P1-N1-P2 AEP complex (64 channel BioSemi EEG system) at pitch match, one octave below pitch match and at 750 Hz were undertaken before and after participants completed FCT or FDT for 21 training sessions over 3 weeks. Auditory training was carried out daily in the participants own home using a Palm Tungsten E2 PDA. The THI score reduced by 5.11 points following FDT. Ability to ignore tinnitus improved by 1.36 points following FCT on a ten point rating scale. An increase in average absolute N1 amplitude was seen following FCT, compared to decrease seen following FDT. Similar changes were not observed for the P2 waveform.

The results of this study suggest that short-duration perceptual training can contribute to a reduction in tinnitus perception and that these perceptual changes can be observed as changes in auditory evoked potentials.

In the somatosensory system, sensory discrimination training has been shown to induce cortical plasticity and reduce phantom limb pain.1 Auditory discrimination training has been shown to produce changes in cortical tonotopy and that tinnitus is likely a consequence of neuroplastic changes in the auditory cortex. It has also been suggested that auditory training could be used to reverse such plastic changes, potentially reducing tinnitus severity.2,4 It has been suggested that FDT for frequencies just above or below the tinnitus pitch could reduce cortical over-representation of tinnitus pitch frequencies by expanding the cortical representation of the (non tinnitus) trained tones.2,3

Flor et al5 trained 12 patients with chronic tonal tinnitus. Patients who completed more training sessions (27 – 44 sessions) were significantly more likely to experience a reduction in tinnitus severity than those who competed fewer training sessions (13 – 23 sessions). However a positive effect of training near to tinnitus pitch was not found. This lead the authors to suggest that the effect of FDT may not be as specific as previously assumed, and that non-specific factors such as attentional changes may have contributed to results.5 Herraiz et al.6 trained 27 patients with high frequency
tinnitus for tones close to their tinnitus pitch using a simple discrimination task consisting of recording the number of tones compared to noise stimuli. Compared to wait list controls significant reductions were seen amongst the training group for both a visual analogue scale and THI score.6

Another form of auditory training requires a process of categorisation rather than discrimination between sounds. Frequency categorisation training (FCT) refers to auditory training in which a listener learns to identify a particular subset of sounds belonging to a selected frequency range from a number of tones similar in frequency. As the listener gets better at identifying sounds from the subset that they are listening for, the sounds become recognised as members of the same category and are thus perceptually ‘categorised’.7 FCT using non-speech stimuli produces a reduction in discrimination ability for stimuli within the training category.7 In contrast, FDT using similar stimuli has been shown to produce an increase in discriminability for trained stimuli.7 Guenther et al.8 assessed the effect of FCT compared to FDT for narrowband noise stimuli in normal hearing individuals and hypothesised that FCT and FDT would have opposite effects on the cortical representation of trained tones. Using fMRI to assess cortical changes associated with FCT compared to FDT8, it was found that FCT led to a reduction in the amount of activation elicited by the training stimuli, whereas FDT led to an increase in cortical activation for trained tones.

Frequency categorisation training and tinnitus—The tinnitus sensation may arise at a cortical level from tonotopic expansion of frequencies close to the tinnitus pitch, from increases in neural synchrony, or from increases in spontaneous firing rate (SFR) – all changes which may give rise to an increase in fMRI signal.9,10 It is thus possible that the reductions in cortical activation seen following FCT8 may be a mechanism which could also lead to a reversal of one or more tinnitus-associated cortical changes. We hypothesised that FCT would alter tinnitus sensation as well as affecting auditory evoked potentials representing activation of the central auditory pathways.

Methods
The methods described in this study were approved by The University of Auckland Human Participants Ethics Committee.

Participants—Twenty-four participants were recruited from a volunteer database. Participants were randomly assigned to undertake either frequency categorisation training (group FCT) or frequency discrimination training (group FDT) for a 3 week period. Participants were required to be under the age of 65, have relatively symmetrical hearing between ears, and have no more than a moderately severe (threshold less than or equal to 70 dB HL) hearing loss between 250 – 8000 Hz.

Test procedures—Participants attended two 2 - 3 hour test sessions, one prior to, and one after completing three weeks’ auditory training (either FCT or FDT) in their own homes using a hand held personal digital assistant (PDA) device. CAEPs were measured at both test sessions. At the initial assessment participants underwent normal and high frequency audiometry, tympanometry, tinnitus pitch matching using a tinnitus likeness assessment11, and measurement of their just noticeable difference (JND) at pitch match. In addition, participants completed four questionnaires (a Tinnitus Sample Case History questionnaire12, the THI13, the THI-1214, and questionnaire containing six numerical rating scales.15 Participants then completed three weeks of either FCT or FDT at pitch match and returned for a second appointment. At the second appointment all questionnaires except the tinnitus history questionnaire were reassessed.

Measurement of cortical auditory evoked potentials—For both test sessions CAEPs were recorded in response to narrowband noise stimuli with centre frequencies (CFs) corresponding to the initial
tinnitus pitch match (‘tinnitus’ pitch), the frequency one octave below initial tinnitus pitch match (‘control’ pitch), and at 750 Hz (‘common’ pitch). All recordings were obtained using a 64 (+2 external) channel Biosemi ActiveTwo system (Biosemi, Amsterdam, Netherlands) referenced to the CMS-DRL ground (2 kHz sampling rate; 400 Hz bandwidth). Data was recorded as continuous EEG and analysed offline. The two additional external electrodes were placed near the eye at LO1 and LO2. Stimuli were 500 ms (10 rise/ fall time) samples of narrowband noise (1/18 octave bandwidth) with different CFs created by filtering white noise with a fourth order butterworth bandpass filter presented at the participants most comfortable listening level. All sounds were delivered by a Dell Optiplex GX620 desktop personal computer using Presentation® software (version 12.2, Neurobehavioural Systems) via a TDT HB6 headphone buffer to ER 3A insert earphones (Etymotic Research Inc., Elk Grove Village, Illinois). Stimuli were presented in sets. A set comprised of 75 stimuli of the same CF presented with an interstimulus interval of 2000 ms. Six sets of stimuli (two sets of each test frequency) were presented to each participant in random order to reduce the effect of participant state on the P1-N1-P2 response for any particular stimulus. Because each different test stimulus was tested twice, a total 150 stimuli at each test frequency were presented.

Auditory training—Auditory training was carried out daily in the participants own home using a Palm Tungsten E2 PDA (CPU 200MHz, memory 32MB, display 320x320 Transflective TFT, SD card 1GB) and Philips SHE7850/97 headphones. Data from each training session (date of training, start time, stop time, percentage correct) were recorded to the SD card in a binary file format that could be accessed at the end of training. The training software was programmed using LabVIEW 8.0 PDA Module. The trainer set up the auditory training parameters individually before the patient used the device. The graphical user interface was quite instinctive so patients could start training immediately at home. Participants were asked to complete a minimum of 21 training sessions on consecutive days over a three week period. If the period between assessments was greater than 21 days, participants were allowed to train for more than 21 days.

Figure 1: Stimuli used in the FCT programme. Modified from Guenther et al. 7 (page 2903).

Frequency categorisation training—Participants were trained to identify sounds belonging to a training region which extended two JNDs above and below the pitch match frequency. Stimuli were 500ms in duration and separated by 750ms. There were two types of training trial: ‘listening trials’ during which participants heard example sounds from the training region but did not have to respond behaviourally, and ‘identification trials’ in which they were required to identify one sound from a list of sounds as belonging to the training region.
During an identification trial participants made their selection by pressing the corresponding button on the PDA screen. During a listening trial, participants were played four stimuli which were randomly selected from a group of nine sounds spanning the training region in evenly spaced 0.5-JND increments. In contrast, during an identification trial, participants were played a short list of sounds containing two, three, or four sounds in succession, of which only one came from within the training region.

The other sounds presented were randomly selected from a set of 18 sounds within the ‘‘band edge’’ regions (see Figure 1). These 18 sounds were made up of nine sounds evenly spanning the lower band edge region in 0.5-JND increments, and nine similarly spaced sounds spanning the higher band edge region. A control region was selected one octave below pitch match. There was no overlap between the training and control regions, and no sounds from the control region were presented during training.

Each training period was made up of ten subsessions containing 30 identification trials, to make a total of 300 trials per training session. Participants received feedback as to whether their responses were correct. The number of sounds presented per identification trial increased over the ten subsessions, with a two-sound list used in the first three subsessions, a three-sound list used in the fourth to sixth subsessions, and a four-sound list used in the last four subsessions. A training session usually took 30 – 40 minutes to complete, depending on response time.

**Frequency discrimination training**—Participants were trained to discriminate between sounds belonging to a training region which extended two JNDs above and below the pitch match frequency. The number of times a participant heard each training sound was approximately the same as during FCT.

Training involved participants listening to pairs of stimuli and indicating whether they thought the two stimuli were the same or different in pitch by pressing the ‘‘same’’ or ‘‘different’’ button on the PDA screen. The two stimuli were 500ms in duration and were separated by a brief 250 ms burst of white noise. Once they had made their selection, participants received feedback about the correctness of their response. A training session comprised ten subsessions, each of which consisted of 30 trials, to make a total of 300 trials per training session.

Within each subsession 10 trials involved pairs of stimuli that were the same (‘‘same’’ trials) and 20 involved pairs that were different (‘‘different’’ trials). Task difficulty was increased throughout the training session by decreasing the number of JNDs separating pairs of different stimuli. During the first three subsessions the stimuli presented in a different trial were separated by 2 JNDs. Throughout the fourth to sixth subsessions stimuli presented in a different trial were 1.5 JND apart, in the final four subsessions different stimuli were just 1 JND apart.

Data analysis of Cortical auditory evoked potentials—The continuous EEG data was re-referenced in Brain Electrical Source Analysis software (BESA; MEGIS Software GmbH, Gräfeling, Germany) to an average reference. Trial epochs were 1500 ms in duration including a 500 ms pre-stimulus interval. Epochs containing amplitude deflections greater than 100 µV were rejected prior to averaging. This resulted in a minimum acceptance of 70% (average 88%) of the trial epochs. Averaged segments were digitally filtered using a 1 Hz (6dB/octave, forward) high pass and 30 Hz (24dB/octave, symmetrical) low pass filter.

The amplitudes and latencies of the P1-N1-P2 complex were identified in the averaged data for each subject as well as in the Grand Mean Average (GMA) waveforms using algorithms that search for peak reversals within predetermined latency windows. The N1 peak was determined as the most negative voltage reversal occurring at Cz during the interval 75–150 ms post stimulus onset and the P2 peak as the most positive voltage reversal at Cz between 100–250 ms post stimulus onset.

Ocular artifacts (blinks, saccades, and smooth movements) within the EEG were defined using the automatic artifact correction method available in BESA 5.2. An adaptive artifact correction technique was then used to correct the EEG for ocular artifacts so that trials containing artifacts that were not eye blinks could be identified and eliminated. Trials with amplitudes greater than 100µV were automatically rejected; however the rejection threshold was adjusted on a per participant basis as individual trials containing amplitudes close to the rejection criteria were visually inspected for artifacts. All averages were run with artifact correction turned off to obtain AERP waveforms contaminated with ocular artifacts.

Ocular artifacts were then corrected following averaging using a surrogate method in which the brain activity was modelled by a fixed dipole model.
Results

Hearing thresholds—For both groups the average audiogram for the left and right ear was normal at 250 and 500Hz, sloping to a slight to mild hearing loss between 1000 – 8000Hz and then to a moderate to moderately severe hearing loss between 10 000 – 16 000 Hz.

Auditory training—Participants in the FCT group completed an average of 19.0 (standard deviation [SD] 5.3) training sessions and those in the FDT group an average of 19.6 (SD 4.8). The minimum average percentage of correct responses per training session (i.e. averaged over the entire training period for each individual) for the two training groups was 51.7% in the FCT group and 63.5% in the FDT group.

Questionnaires—No participants noted an improvement in tinnitus according to the question ‘Do you feel your tinnitus is the same, better or worse than prior to training’. The average baseline THI, THI-12 and different numerical rating scale scores for the two groups are shown in table 1. In the FCT group there were 6 participants with slight, 5 with mild, and 2 with severe tinnitus according to THI severity grading. This compared to 3 participants with slight, 2 with mild, and 4 with moderate tinnitus in the FDT group. Table 1 also shows the average change in the score of each questionnaire at the completion of training.

Table 1: Average pre training, average post training and average change in THI, numerical rating scale (mean), and the individual numerical rating scale scores for the two groups. Measures of subjective tinnitus severity that were analysed further using ANOVA are shaded in grey. Standard deviations are shown in brackets next to each value. (NRS: Numerical rating scale)

<table>
<thead>
<tr>
<th></th>
<th>Group FCT (n = 11)</th>
<th>Group FDT (n = 9)</th>
<th>Change (post–pre)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pre training</td>
<td>Post training</td>
<td>Average change (post–pre)</td>
</tr>
<tr>
<td>THI score</td>
<td>26.18 (20.21)</td>
<td>27.09 (17.97)</td>
<td>0.91 (7.61)</td>
</tr>
<tr>
<td>NRS (mean) score</td>
<td>-0.07 (0.90)</td>
<td>0.31 (1.01)</td>
<td>-3.95 (0.50)</td>
</tr>
<tr>
<td>NRS 1 score</td>
<td>3.17 (0.79)</td>
<td>2.88 (1.00)</td>
<td>-0.28 (0.75)</td>
</tr>
<tr>
<td>NRS 2 score</td>
<td>5.59 (1.78)</td>
<td>5.56 (1.78)</td>
<td>-0.23 (1.33)</td>
</tr>
<tr>
<td>NRS 3 score</td>
<td>2.31 (1.93)</td>
<td>2.18 (1.93)</td>
<td>-0.59 (2.71)</td>
</tr>
<tr>
<td>NRS 4 score</td>
<td>2.42 (2.60)</td>
<td>4.82 (2.60)</td>
<td>-0.95 (2.71)</td>
</tr>
<tr>
<td>NRS 5 score</td>
<td>6.09 (2.26)</td>
<td>4.73 (3.00)</td>
<td>-1.36 (1.96)</td>
</tr>
<tr>
<td>NRS 6 score</td>
<td>5.68 (2.51)</td>
<td>5.09 (2.83)</td>
<td>-0.59 (1.95)</td>
</tr>
</tbody>
</table>
Following a Pearson bivariate correlation matrix to evaluate inter-item correlation three measures of subjective tinnitus severity were analysed pre and post training; the THI; numerical rating scale (mean); and numerical rating scale 5 ('how easy is it for you to ignore your tinnitus at present?').

Repeated measures ANOVA revealed there was a marginal difference in the pre and post training THI score between the two training groups (F(1,17) = 2.07, p = 0.17) (see Figure 2). While FCT produced an average increase of 0.91, FDT produced an average reduction of 5.11 in THI score.

![Average THI score pre and post training](image)

**Figure 2:** Average pre and post training THI score for the two training groups. Error bars represent one standard error of the mean.

For numerical rating scale (mean) repeated measures ANOVA failed to identify any time or group by time interactions at p < 0.20. There was no change in subjective rating of tinnitus severity according to numerical rating scale (mean) score following auditory training for either of the two groups.

Finally for numerical rating scale 5, which assessed how difficult it was for each participant to ignore their tinnitus, repeated measures ANOVA revealed there was a marginal difference in the pre and post training numerical rating scale 5 score between the two training groups (F(1,17) = 3.40, p = 0.08). Numerical rating scale 5 score reduced by 1.36 points following FCT, but increased by 0.56 points following FDT (see Figure 3), meaning that at the completion of training on average participants in the FCT group found it slightly easier to ignore their tinnitus but participants in the FDT group found it very slightly harder to ignore their tinnitus.

**Cortical auditory evoked potentials**—Pre and post training cortical auditory evoked potentials were recorded from 20 participants (11 FCT, 9 FDT) who completed more
than 6 auditory training sessions. Of these 20 sets of data, those for two participants, one in the FCT and one in the FDT group, had to be excluded from analysis due to poor quality post training recordings. The pre and post training GMA waveforms for the remaining 10 FCT and 8 FDT participants were evaluated.

![Average numerical rating scale 5 score pre and post training](image)

**Figure 3:** Average pre and post training numerical rating scale 5 score for the two training groups. Numerical rating scale 5 asked ‘how easy is it for you to ignore your tinnitus’.

**N1 results**—GMA waveforms at Cz for each recording condition, pre and post training are shown in Figure 4. The greatest change we saw was for the common pitch stimuli, where an increase in average N1 amplitude was seen following FCT, compared to decrease seen following FDT (see Figures 4, 5 and 6). For the comparison of the change in N1 amplitude after the two different types of training, a mixed ANOVA was performed. Time (pre-training and post-training) and Frequency (common, control, and tinnitus frequency) were treated as repeated measures, and training (FCT and FDT) as a between subjects factor. No significant effects were found, but there were marginal (p<0.2) effects of test frequency overall and a marginal three-way interaction between time, frequency, and training. On this basis, we carried out tests of simple effects within the interaction.
Figure 4: Pre and post training GMA waveforms in response to i) common pitch (750 Hz) ii) Control pitch and iii) Tinnitus pitch stimuli. Pre training waveforms are shown in red, and post training waveforms in blue. Left hand panel: Data for the FCT group (note the increase in N1 amplitude post training); Right hand panel: Data for the FDT group (note the decrease in N1 amplitude in the post training waveform).
Figure 5: Average pre and post training absolute N1 amplitude in response to different stimulus frequencies for the two training groups. Error bars represent one standard error of the mean.

The difference between average N1 amplitude at Cz for training stimuli (tinnitus pitch stimuli) compared to control and common pitch stimuli pre and post training was evaluated in a manner similar to that used by Guenther et al. 2004 (8). When the difference between absolute average N1 amplitude for tinnitus versus common stimuli (i.e. \[|\text{tinnitus post} - |\text{common post}| - |\text{tinnitus pre} - |\text{common pre}|\]) tinnitus vs control and control vs tinnitus were compared pre and post training a reduction in N1 amplitude was seen following FCT and an increase was seen following FDT (see Figure 7).
Figure 7: Comparison of the difference between average N1 amplitude for various test stimuli pre and post training. Error bars represent one standard error of the mean.

P2 results—No consistent change in P2 amplitude was observed.

Discussion

Subjective assessments of tinnitus severity—Although none of the participants self-reported significant improvement in tinnitus, a group by time effect was noted for THI score which demonstrated a marginal difference in the pre and post training THI score between the two training groups. While FCT produced an average increase of 0.91, FDT produced an average reduction of 5.11 in THI score. No significant change in the mean numerical rating scale score was noted for either training group, but when numerical rating scale 5 (‘how easy is it for you to ignore your tinnitus at present?’) was analysed separately repeated measures ANOVA revealed there was a marginal difference in the pre and post training numerical rating scale 5 score between the two training groups. Numerical rating scale 5 score reduced by 1.36 points following FCT, but increased by 0.56 points following FDT.

FDT seemed to reduce THI score marginally more than FCT, but participants were marginally more likely to find it easier to ignore their tinnitus following FCT than following FDT. The latter finding is important because Tinnitus Retraining Therapy (TRT), an established therapy for tinnitus, aims to facilitate habituation to tinnitus, in part by attempting reclassify the tinnitus sensation into a category of ‘neutral’ signals in the hope that once habituation is achieved tinnitus patients will no longer be bothered by, despite being aware of, their tinnitus. Being better able to ignore one’s tinnitus could possibly help reclassify the tinnitus sensation as a more neutral signal thus aiding habituation. With this in mind, it could be possible that a treatment programme combining FCT with some form of counselling to help patients manage their tinnitus using more cognitive methods may provide greater benefit with regard to
subjective tinnitus severity than using either FCT or cognitive based methods in isolation.

**Cortical auditory evoked potential amplitudes**—The main AEP related finding from this study involved the pre and postraining comparison of the difference between average AEP amplitude at Cz for training versus control or common pitch stimuli. This comparison was similar to that used by Guenther et al\(^8\) in their fMRI study investigating the effects of FDT and FCT on cortical representation in normal hearing listeners.\(^8\)

Guenther et al.\(^8\) found that the amount of cortical activation produced by training compared to control pitch stimuli was reduced following FCT and increased following FDT. To test this hypothesis, the pre and post training difference between absolute average AEP amplitude for control versus common pitch stimuli was also compared (i.e. \(|\text{control post} - \text{common post}| - |\text{control pre} - \text{common pre}||\)). This comparison revealed that, as for the tinnitus versus common pitch comparison, a reduction in both N1 and P2 amplitude was seen following FCT combined with an increase following FDT.

Although the results showed only a marginal effect, FCT and FDT appeared to have an equal, but opposite, effect at the common frequency (750 Hz). FCT showed a slight decrease at tinnitus pitch. The combined effect was similar to Guenther et al.\(^8\), although the effect was due primarily to changes in response to the common frequency, rather than tinnitus pitch.

If the changes seen could be proven to be robust and repeatable, one explanation for the results obtained may be that learning induced by the auditory training programmes had generalised to the control pitch but to a lesser degree to the common pitch. Following FCT common pitch stimuli may have become more behaviourally relevant than training pitch stimuli, which became perceptually ‘categorised’, thus producing an increase in neural synchrony in response to common pitch tones. In contrast common pitch tones may have become less behaviourally relevant compared to training pitch tones following FDT, producing a reduction in neural synchrony for common pitch stimuli.

It was hypothesised that FDT would produce an increase in N1 and/or P2 amplitude for tinnitus pitch stimuli. Previous studies\(^18,19,20\) assessing the effect of auditory training in normal hearing listeners have found that the P2 waveform in particular is sensitive to cortical remodelling elicited by FDT. Behavioural results from other FDT studies\(^24\) have found that the time course of perceptual learning can differ depending on training frequency and may be related to pre training perceptual ability for the trained frequency.

Potentially the fact that neural synchrony is already enhanced for regions at and surrounding the tinnitus pitch may mean that the time course for increased neural synchrony is longer for tinnitus pitch stimuli in subjects with tinnitus. Relating this back to our AEP data perhaps the reason we failed to demonstrate the expected increase in N1 and/or P2 amplitude for tinnitus pitch stimuli following FDT was that a high level of pre training neural synchrony already existed for these frequencies. The same hypothesis cannot be used to explain why no change AEP amplitude was seen for tinnitus pitch stimuli post FCT. If an increased level of baseline neural
synchrony indeed exists for frequencies surrounding the tinnitus pitch, one would expect that it would be possible to produce a reduction in neural synchrony with FCT.

As yet no studies have assessed the effect of FCT on AEPs in normal hearing listeners. As such it may be that even in normal hearing listeners FCT does not produce a reduction in N1 or P2 amplitude for trained stimuli. Alternatively the presence of tinnitus i.e. the perception of a continuous sound at the training pitch may maintain a level of neural synchrony that would otherwise be reduced in the absence of tinnitus.

None of theories outlined above can be used directly to explain why no change in absolute AEP amplitude was seen for control pitch stimuli. If indeed the changes in N1 and P2 amplitude for common pitch stimuli were produced due to alterations in behavioural relevance for untrained frequencies then it would be expected that similar (but opposite) changes in P1 and N1 amplitude would have been seen for control pitch stimuli. One way to explain the results seen for control pitch stimuli in the current study may relate to the fact that control pitch stimuli were exactly one octave below training stimuli and thus in some related in pitch to the trained frequency. In contrast, the common pitch stimulus was always 750 Hz and had no octave relationship to training stimuli. Possibly the pitch relationship between control and tinnitus stimuli meant that control pitch stimuli maintained their behavioural relevance at the completion of auditory training.

**Summary**

The results of this study contribute to emerging evidence that auditory training may be a useful means to reduce the perception of tinnitus. Although the proof for changes in the underlying physiology of such improvement from evoked potentials is tentative we believe that there is sufficient evidence for the training techniques, and means to measure outcome of training, to be explored further.

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Hyperacusis: a clinical perspective on understanding and management

M Westcott

Abstract

Hyperacusis is an abnormal intolerance of everyday sounds, which most others tolerate well. It can develop in the belief that ears/hearing may be damaged by intolerable sound exposure; or pre-existing tinnitus/hyperacusis may be aggravated. Once hyperacusis has developed, it has the potential to escalate so that more sounds become perceived as intolerable. Hyperacusis can result in severe lifestyle restrictions; constant environmental monitoring; and difficulty in managing high levels of anxiety in relation to sound exposure.

Tonic tensor tympani syndrome (TTTS) study TTTS has been proposed as causing the neurophysiological symptoms underlying acoustic shock disorder. TTTS symptoms are also common in tinnitus and hyperacusis clients, and a study was carried out to identify the range and incidence of TTTS symptoms in this client group. Any physiological symptoms experienced (excluding tinnitus and hyperacusis increase/kindling) following exposure to sounds perceived as very difficult to tolerate were documented. 37% (19/51) of tinnitus clients reported TTTS symptoms after exposure to intolerable sounds. 29% reported two or more symptoms. 94% (48/51) of hyperacusis clients reported TTTS symptoms, which develop or are exacerbated by exposure to intolerable sounds. The most common TTTS symptoms were aural pain and fullness, which can explain the pain experienced by hyperacusis clients in response to everyday sounds. Research into central pain sensitisation provides an insight into the process of pain escalation in these clients.

Hyperacusis desensitisation Hyperacusis desensitisation can be achieved using a Tinnitus Retraining Therapy (TRT) approach, with the addition of cognitive behavioural therapy (CBT) strategies to manage auditory hypervigilance, reframe inaccurate beliefs about hyperacusis and provide a framework for phonophobia management. These strategies will be discussed.

Conclusion The high incidence of TTTS symptoms in tinnitus and hyperacusis clients warrants acknowledgement and explanation to facilitate effective therapy. TRT and CBT strategies are complementary in a hyperacusis desensitisation program.

Hyperacusis is an abnormal intolerance to ordinary, everyday sounds. Misophonia is a strong dislike of sound and is widespread - almost everyone has a sound they have disliked at some time. Intrusion intolerance is an abnormal aversion to sounds, often made by other people, intruding into an individual’s “own space”, e.g.: the sounds of other people eating, neighbour’s music, noise from a nearby factory etc.

Phonophobia occurs when people fear being exposed to a certain sound in the belief that it may damage their ears/hearing; make their tinnitus/hyperacusis worse; lead to uncontrollably high levels of anxiety, panic or distress. Intrusion phonophobia occurs when people fear being exposed to a certain sound in the belief that it may lead to
uncontrollably high levels of annoyance/distress and they will be unable to control their emotional reaction following exposure.

Hyperacusis can range from mild through to severe. Phonophobia is usually present with significant levels of hyperacusis.

**What can cause hyperacusis?**—Pre-existing tinnitus, misophonia and high levels of anxiety are factors that can predispose towards the development of hyperacusis and phonophobia. Hyperacusis can develop with a number of conditions affecting the auditory pathway (including acoustic shock disorder, Menière’s Disease, otosclerosis, perilymph fistula and Bell’s Palsy), psychiatric disorders, neurological injuries and disorders (including head injury and migraine), adverse reactions to some medications, autistic spectrum disorders, myalgic encephalomyelitis (chronic fatigue syndrome), fibromyalgia and Lyme Disease.

**How does hyperacusis develop?**—When hyperacusis develops, everyday sounds begin to appear unnaturally prominent and increasingly louder. Following exposure to some or many of these sounds, a temporary increase in tinnitus (if present) and/or hyperacusis may be noticed, and escalating sensations in the ear may develop, such as ear pain, a fluttering sensation or an intermittent fullness. This reaction can generalise to include more and more sounds. As a result, people with hyperacusis may come to believe that their ears are no longer able to physically tolerate these sounds and/or that these sounds are causing damage to their ears or hearing and should be avoided. The escalating anxiety about the effects of exposure to these sounds can lead to the development of phonophobia.

When intrusion hyperacusis develops, everyday sounds begin to appear unnaturally prominent and increasingly louder. Following exposure to some or many of these sounds, high levels of anger and irritability can occur. This reaction can generalise, and the escalating anxiety about the emotional reaction following exposure to these sounds can lead to the development of intrusion phonophobia.

**How does hyperacusis affect people?**—People with severe hyperacusis generally don’t tolerate any loud sounds, many moderate volume sounds, particularly if sudden and unexpected, and may not tolerate some soft sounds. High frequency sounds tend to be tolerated less well and some can become highly intolerant of low frequency vibration. Eggermont showed that enhanced high frequency sound exposure in cats (80 dB, 24 hours a day) caused no physiological noise trauma or hearing loss, but had a dramatic effect on the auditory cortex, with an enhancement in the firing rate for low and high frequency sounds. Some changes occurred at lower volume levels (65 dB, 24 hours a day). These changes were persistent, and Eggermont considers them relevant to hyperacusis.

Severe hyperacusis can have a major impact on people’s lives, severely limiting lifestyle horizons and creating secondary symptoms of anxiety, depression and anger. If hyperacusis has developed from or been exacerbated by exposure to a sudden/unexpected/loud/traumatic sound, symptoms of post traumatic stress can occur as a result of acoustic shock.

Hypervigilance of the auditory environment is common, as people with hyperacusis often feel the need to regularly, and sometimes constantly, monitor their auditory environment to avoid intolerable sounds. They may feel a need to protect their ears...
and sense of hearing from exposure to intolerable sounds. Many hyperacusis clients have an elevated startle reflex.

There is very little understanding of hyperacusis in the community. Hyperacusis, phonophobia and any physical symptoms in and around the ear following exposure to intolerable sounds are involuntary and subjective. Explaining such an abnormal reaction to sound to other people, including at times health professionals, is difficult and people with hyperacusis/phonophobia often feel misunderstood, isolated and accused of malingering.

Proposed mechanism of hyperacusis: Tonic Tensor Tympani Syndrome (TTTS)—The tensor tympani reflex is a startle reflex, which is exaggerated by high stress levels. The tensor tympani muscle contracts immediately preceding the sounds produced during self-vocalisation, suggesting it has an established protective function to loud sounds; assists in the discrimination of low frequency sounds; and is involved in velopharyngeal movements.

TTTS has been proposed as causing the persistent physiological symptoms of acoustic shock disorder, and the aural symptoms of temporomandibular disorder (TMD).

TTTS is an involuntary condition where the centrally mediated reflex threshold for tensor tympani muscle activity becomes reduced as a result of anxiety and trauma, so it is continually and rhythmically contracting and relaxing, aggravated by intolerable sound exposure. This appears to initiate a cascade of physiological reactions in and around the ear, which can include: an abnormal stimulation of the trigeminal nerve innervating the tensor tympani muscle, leading to frequent neuralgic pain and sensations of numbness/burning; tympanic flutter; alterations in ventilation of the middle ear cavity causing aural blockage/fullness and muffled/distorted hearing; and in severe cases, symptoms consistent with TMD.

While similar symptoms of aural pain and blockage without underlying pathology have been noted in tinnitus and hyperacusis clients, they have not been widely acknowledged or investigated.

TTTS study in tinnitus and hyperacusis clients

Any physiological symptoms experienced by tinnitus and hyperacusis clients following exposure to sounds perceived as very difficult to tolerate are documented at my clinic. This study aimed to identify the range and incidence of these symptoms (excluding any tinnitus and hyperacusis increase/kindling). 51 clients attending for hyperacusis therapy and the same number of clients attending for tinnitus therapy were evaluated.

The symptoms identified were either present most of the time and exacerbated by exposure to sounds perceived as very difficult to tolerate; or were triggered by exposure to those sounds. In all cases, outer/middle/inner ear pathology and, if symptoms were unilateral, retrocochlear pathology, were medically cleared.

Results

Group 1—Tinnitus clients (TRT category 0, 1 and 2): Of the 51 tinnitus clients, 19 (37%) reported physiological symptoms after exposure to very difficult to tolerate sounds, and 15 (29%) reported two or more symptoms. Pain in and around the ear was
reported by 14 (27% of total); aural blockage by 11 (22%); muffled hearing by 6 (12%); numbness/burning in and around the ear by 5 (10%); mild vertigo by 4 (8%); and tympanic flutter by 3 (6%).

**Group 2—** Hyperacusis clients (TRT category 3 and 4): Of the 51 hyperacusis clients, 48 (94%) reported physiological symptoms after exposure to very difficult to tolerate sounds and 34 (67%) reported two or more symptoms. Pain in and around the ear was reported by 33 (65% of total); aural blockage by 26 (51%); mild vertigo by 14 (27%); tympanic flutter by 10 (20%); muffled hearing by 9 (18%); headache by 9 (18%); numbness/burning in and around the ear by 4 (8%); and reduced cognitive function/disorientation by 4 (8%).

**Discussion**

Almost all hyperacusis and over a third of tinnitus clients were shown to experience physiological symptoms consistent with TTTS following exposure to very difficult to tolerate sounds. TTTS appears to be triggered involuntarily by the anticipation and perception of sounds considered to be highly threatening and/or intolerable. There is little known and much to research in understanding this aetiological pathway.

TTTS explains why hyperacusis clients report aural pain after exposure to sounds they find intolerable, but others tolerate well. Clients with severe hyperacusis are in frequent or constant pain, which is exacerbated by intolerable sound exposure.

Clinically, these symptoms appear in clients whose anxiety has led to a strong need to protect their ears from sounds they consider likely to cause an escalation in their tinnitus, hearing loss or existing TTTS symptoms. The presence of the symptoms after intolerable sound exposure reinforces the belief that “damage “ has taken place, fuelling further anxiety. This response then generalises to other types of sound and to lower sound volume levels, resulting in the development as well as the potential escalation of hyperacusis. I have observed some clients in my clinic with severe hyperacusis develop TTTS symptoms just by thinking about a loud, unpleasant sound or switching off their white noise generators.

Milder TTTS symptoms are common in general audiology clients, particularly those who express anxiety about the health of their ears or hearing. For example, clients who report symptoms of frequent aural blockage, without underlying pathology or Eustachian tube dysfunction.

**Hyperacusis desensitisation therapy**—A detailed history, where the pattern of development and escalation of sounds changing from being tolerable to intolerable is analysed, creates a pathway for this process to be unravelled. History taking should include identifying stress levels at the time of onset; documenting which sounds are intolerable; physiological symptoms experienced after intolerable sound exposure; prior otological and psychological history; and include screening for anxiety and depression.

The high incidence of TTTS symptoms in hyperacusis clients warrants acknowledgement and explanation to facilitate effective therapy. When symptoms are present, TTTS can be included in an explanation of the peripheral and central auditory pathway, using Jastreboff’s neurophysiological model and following the Tinnitus Retraining Therapy (TRT) protocol. Explaining TTTS to these clients provides
validation, reassurance and helps reduce anxiety, which can have an immediate effect on reducing the symptoms.

Clients are counselled that it does not harm the ear to experience TTTS, and even though the TTTS symptoms can seem as if the ear is being damaged by some sounds, this is not the case. Moderate, everyday sounds are quite safe and do not harm the ear or cause a hearing loss.

For clients with severe hyperacusis, listening to sounds via headphones during a hearing assessment can be threatening and should be carried out with care. I consider that loudness discomfort testing, and in particular acoustic reflex testing due to the volume levels required, is contraindicated. Some hyperacusis clients have unfortunately suffered a considerable escalation in their hyperacusis as a result of a traumatic response to acoustic reflex testing. Tracking a reduction of TTTS symptoms rather than loudness discomfort levels over time can give a non threatening indication of the effectiveness of a therapeutic intervention.

Cognitive behaviour therapy (CBT) strategies can be developed to challenge and reframe irrational thoughts, which exacerbate hyperacusis and promote the development of phonophobia. Clients may need help in recognising destructive thoughts and beliefs. Many irrational thoughts can seem logical, and changing them often seems counter-intuitive.

Examples of irrational thoughts include “my ears must be more sensitive than other people’s because I can hear these sounds more strongly”; “intolerable sounds hurt/increase my tinnitus so they must be damaging my ears”; “my ears must be more sensitive than other people’s because I’ve got tinnitus/noise damage already”; and “I can’t lead a normal life because I have to avoid noisy places/music”.

Hyperacusis clients need to be taught to control excessive environmental sound monitoring and hypervigilance, using cognitive distraction strategies. This involves accepting that intolerable sounds will inevitably be present at some time, and understanding that focussing on them will inhibit desensitisation. Identifying and defining where clients feel safe from intolerable sounds is of benefit in encouraging reduced vigilance in those places, and helps in recognising “boundaries of safety”.

Environmental sound enrichment strategies are recommended to promote hyperacusis desensitisation, following a TRT protocol. Now that MP3 players are readily available, allowing a wide choice of stable, neutral environmental sounds, ear level sound generators are rarely used in my clinic. I have found that clients with severe hyperacusis are rarely able to tolerate sound fed directly into their ears via headphones or ear buds.

The proximity of the sound appears to escalate their subconscious need to protect their ears or hearing to maintain their current tinnitus/TTTS levels. The subsequent and involuntary increase in anxiety has the potential to increase their TTTS symptoms. An alternative is to use headphones worn around the neck, so the sound is still audible, rather than over the ears.

Portable sound enrichment is effective used as a barrier or shield to “protect” against unpredictable or intolerable sounds, allowing clients to feel safe while gently nudging and expanding their “boundaries of safety”. The judicious use of solid and/or
musician’s filtered ear plugs frequently assists in maintaining or facilitating this expansion of lifestyle horizons, with encouragement and support given to gradually reduce their use, aiming to substitute the plugs with portable sound enrichment.

For those clients struggling with severe hyperacusis, consideration can be given to fitting in-the-canal hearing aids programmed to provide amplification of soft sounds to compensate for the effect of occlusion limiting communication ability, reduce tinnitus awareness and support a hearing loss if present; while acting as an electronic filter to loud sounds, by reducing the maximum power output (MPO) to minimum levels and using high levels of compression, particularly for high frequency sounds, to reduce startle to impact sounds. This avoids over-protection of the ears from ear plugs while at the same time providing effective protection to loud sounds. When anxiety levels permit, there is opportunity for desensitisation by gradually increasing the MPO and reducing the compression levels.

Without evaluating and addressing the psychological factors underpinning phonophobia, desensitisation is likely to be limited. Encouraging clients to identify and acknowledge the emotions they feel after intolerable sound exposure helps them to understand and manage them more effectively. Stress/panic/anger can be helped using breathing techniques, imagery and active relaxation strategies.

Grief counselling can support the loss of lifestyle choices and restrictions; career and employment limitations; loss of financial security; and anxiety over being a burden to others. Referral for psychological/psychiatric treatment for anxiety, depression and trauma needs to be carried out when indicated. This is particularly required if pre-existing anxiety and depression were reported, which are likely to have exacerbated the level of emotional reaction subsequent to the development of hyperacusis.

Conclusion

TRT and CBT strategies are complementary in an effective hyperacusis desensitisation program.

The high incidence of TTTS symptoms in tinnitus and hyperacusis clients warrants acknowledgement and explanation to facilitate effective therapy. TTTS provides insight into the development of hyperacusis in an anxious or traumatised client, and understanding and explaining TTTS gives a strong framework for desensitisation. Tracking a reduction of TTTS symptoms rather than loudness discomfort levels over time can give a non-threatening indication of the effectiveness of hyperacusis desensitisation therapy.

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Multidisciplinary team approaches to tinnitus

G Shakes

Abstract

The concept of a multidisciplinary approach to the management of tinnitus is not new, however several recent international publications have argued that tinnitus is best managed by a multidisciplinary team approach.\(^1\)\(^-\)\(^3\) Several obstacles to a team approach have been cited including, lack of interdisciplinary communication and collaboration, inadequate conceptualisation of tinnitus, staff skill mix and varying professional experience, limited referral options or management plans.\(^1\)

Recent Dept of Health, NHS Guidelines, ‘Provision of services for adults with tinnitus: a good practice guide’\(^4\) provides an outline structure and pathway for tinnitus service provision within a public health service. The guidelines propose a matrix of services to improve access and treatment options, in which competent assessment and appropriate triage is critical.

A recent publication by the British Psychological Society\(^5\) for psychologists working in social and health care teams in the UK, provides a current understanding of how teams work and how their effectiveness can be maximised. The review provides practical guidance on effective team working and aims to be relevant for non-psychologists working in teams who want to use psychological principles in their work. It is argued that a premeditated team design based upon relevant research evidence results in several positive outcomes from team working.

An example of a public service multidisciplinary tinnitus team\(^6\) located within a specialist regional Department of Audiovestibular Medicine is outlined and described with reference to the good practice guidelines and working in teams publications. It is proposed that a planned, effective and efficient multidisciplinary team approach to tinnitus is better placed to serve the range of clinical needs of people with tinnitus and also offers several advantages to the professional team.

Tinnitus has been described as an internally generated perception of sound produced involuntarily.\(^7\) Tinnitus is classified as objective tinnitus when it can be heard externally, or as subjective tinnitus when it is only audible to the patient.\(^7\) People experiencing tinnitus have described a variety of auditory experiences including ringing, buzzing, whistling and whooshing sounds.\(^4\) Tinnitus is a symptom not a disease.\(^8\),\(^9\)

Tinnitus prevalence estimates vary, although recent research in New Zealand from a general population sample found that occasional or transient tinnitus was experienced by 38.2% whilst 6.8% of people experienced tinnitus for half the time or more.\(^10\) In one large, longitudinal study of hearing, which also enquired about tinnitus prevalence, 10.1% of adults reported having experienced prolonged spontaneous (>5 minutes duration) tinnitus, with 5% of adults reporting moderately or severely annoying tinnitus.\(^11\) Furthermore, in 0.5% of adults, tinnitus has been described as having a severely disruptive effect on the ability to lead a normal life,\(^1,11\) affecting the ability to sleep, work, concentrate, relax and engage in social interaction.\(^12\) It has been argued that the reaction or response to tinnitus must be considered along with the frequency and duration of tinnitus\(^1,7,13\) when considering tinnitus management.
Proposal of a multidisciplinary team approach—Over recent years several international\textsuperscript{1-3} have proposed a multidisciplinary approach to tinnitus management. Clinicians and researchers have suggested adopting a multidisciplinary team model for tinnitus management and treatment, similar to the model for chronic pain services.\textsuperscript{1,2,6}

An holistic approach is adopted by an American tinnitus treatment programme with the goal of enabling patients to manage the multiple consequences of tinnitus, including psychosocial (depression, concentration difficulty, inability to participate in work and leisure); and physical (sleep deprivation and muscle tension) consequences of tinnitus'.\textsuperscript{2} It is argued that tinnitus involves multiple systems and therefore treatment is provided by a multidisciplinary team.\textsuperscript{2}

The recommendation that tinnitus patients are best served by a multidisciplinary team approach, has gained support among several international authors in recent years as the methods for enhancing care and treatment options for patients are explored\textsuperscript{1,3,4,6} although the idea was first proposed over twenty years ago.\textsuperscript{15-16} It is suggested that a multidisciplinary team approach to tinnitus treatment and management in the UK only exists in a few centres of excellence.\textsuperscript{1,4} It has been argued that tinnitus research has been hindered by the need for interprofessional communication and partnership, resulting in an insufficient understanding of tinnitus as a whole.\textsuperscript{1}

A significant collaborative approach by audiology, clinical psychology and otolaryngology\textsuperscript{1} addressed this discrepancy, by producing a clear, multidisciplinary approach which recommends an amalgamation between neurophysiological and psychological models of tinnitus. However practical obstacles to a multidisciplinary team approach to tinnitus treatment are raised including; ensuring appropriate staffing levels, blend of skills and professional experience.\textsuperscript{1}

Services for adults with tinnitus: a good practice guide—A comprehensive document produced by the UK Department of Health, NHS Guidelines, ‘Provision of Services for Adults with Tinnitus: A Good Practice Guide’\textsuperscript{4} provides an outline structure and pathway for tinnitus service provision. The guide proposes a network or ‘matrix of services’ which gives tinnitus patients a greater access to services and a greater choice of treatment options. The two part guide firstly offers a vision, context and principles for the organisation and delivery of services; and secondly a good practice pathway for the majority of adults with tinnitus which suggests an effective way to deliver care and also outlines practical details of the vision for tinnitus services.\textsuperscript{4}

The guideline for a public health model proposes that adults with tinnitus should receive a free, local, high quality, efficient responsive services, with minimal waiting times.\textsuperscript{4} Within the NHS, the pathway to appropriate tinnitus management is not always as efficient and effective, often characterised by long waiting times for services\textsuperscript{4,6} It is argued that clear referral pathways are required for patients whose clinical needs fall outside the skills of specific team,\textsuperscript{1,6} for example, patients with neurological symptoms or extreme distress should be referred to a specialist centre or service.\textsuperscript{4,6}

A stepped care approach to tinnitus treatment was proposed by the good tinnitus guide. Four separate levels of service provision have been identified for tinnitus
management, depending upon the patient’s clinical presentation. The initial service provider is often the primary care service, or general practitioner. Patients with for example, bilateral tinnitus with suspected hearing loss, or persistent mild tinnitus, may be referred to local audiology services for management by qualified audiologists. Referral to a specialist centres or supra-specialist centre, includes a broader multidisciplinary team. The specialist and supra-specialist centres include a range of healthcare professionals; consultant audiovestibular physicians (AVP), Ear Nose and Throat (ENT) surgeons (or otolaryngologist), audiological scientists, audiologists, hearing therapists, clinical psychologist, nurse, administrators and clerical staff, with access to other specialities. Competent assessment and appropriate triage has been cited, as an essential starting point, critical for early identification of any underlying medical conditions requiring prompt and appropriate management of tinnitus.

Within the stepped care approach, it is proposed that multidisciplinary teams operating within the specialist and supra-specialist centres maintain the team integration by: regular team meetings to discuss patient pathways, discuss referral criteria and audit of outcomes; that professional roles are expanded or redesigned with retraining provided; all professional staff be competent in counselling and psychological support skills; clinical psychologist be skilled in CBT; and provide appropriate supervision for staff offering psychological treatment or counselling. It is therefore important to understand how the tinnitus team works together and why working in a well functioning team has advantages for patients and staff.

**Table 1: Advantages for effective teams (from Onyett BPS Report)**

1. Improve quality of care (reduce time in hospital, better accessibility, enhanced user satisfaction, better acceptance of interventions and improved health outcomes) through achievement of co-ordinated and collaborative inputs from different disciplines
2. Link and integrate information for e.g. life of a client based upon long term relationships with different team members.
3. Speedily develop and deliver services cost effectively while retaining high quality
4. Enable organisations to learn (and retain learning) more effectively, in groups rather than one individual’s own knowledge
5. Time saving activities performed concurrently rather than sequentially
6. Innovation promoted by cross-fertilisation of ideas
7. Collegiality, friendship and emotional support from team increases staff satisfaction and professional stimulation

**Working in teams**—One international multidisciplinary approach to tinnitus management outlines the professionals and treatments offered within a given team or service but does not describe how the team operates or functions. The working relationships between the team members as a whole can also form an influential outcome on the overall service. A recent report on the ways in which psychologists...
work in teams in the UK provides an understanding of how multi-professional teams operate and how effectiveness can be maximised. A fundamental aspect of the working in teams report is that the report is relevant for any team member wanting to use psychological principles at work. The working in teams report therefore supports the tinnitus guidelines recommending that all staff working in teams with tinnitus patients should be competent in counselling and psychological support skills.

Several advantages are identified for well planned and well designed effective teams, as summarised by the working in teams report listed in Table 1: Advantages for effective teams. Several of the advantages listed have also been described within a specialist audiovestibular medicine service. A tinnitus multidisciplinary team—In May 2006 the British Tinnitus Association, in conjunction with North Trent Region, Department of Audiovestibular Medicine, hosted a study day for professionals working with adults with tinnitus, using an interactive demonstration of multidisciplinary team work. A key part of the demonstration was designed to reveal the holistic approach to assessment, planning and treatment or interventions, by illuminating the team communication processes using role plays with actors as tinnitus patients and individual members of the multidisciplinary team.

The result was to offer delegates a unique insight into the performance, dynamics and patient benefits of a multidisciplinary tinnitus team in a specialist centre. The initial medical assessment and rationale for tinnitus assessment by the Consultant Physician in Audiovestibular Medicine has been described in detail and provides the key starting point for all patients referred to the service.

Multi-disciplinary team working in a specialist centre with complex tinnitus patients has identified the difficulties of engaging psychologically distressed patients in tinnitus treatment. Psychologically distressed tinnitus patients may take up considerable time and resources in unproductive treatment or therapy, when their psychological needs are unidentified or not adequately addressed. Alternatively patients may drop out of treatment, which then presents general practitioners with the challenges of managing potentially complex patients. A case example of a skilled multidisciplinary team approach provided for a psychologically distressed patient with a complex tinnitus and balance presentation explained the benefits of a multidisciplinary team approach.

A referral to clinical psychology for assessment identified untreated post traumatic stress disorder (PTSD). A coordinated treatment plan, including concurrent psychological therapy, audiology and physiotherapy was provided. The result was a reduction in the patient’s psychological distress and successful management of tinnitus. The efficient and effective use of rehabilitation resources reduced the time and frequency of appointments with audiology and physiotherapy, as the patient’s psychological needs were met. Furthermore, the successful outcome improved each of the therapist’s sense of efficacy with a complex patient.

Several features of the recommended tinnitus teams in specialist and supra-specialist tinnitus centres, are also documented in other reports of team working, including: having a diverse multidisciplinary team, monthly tinnitus team meetings and additional therapists team meetings; whole person approach of physical and
psychosocial needs,\textsuperscript{1,2,6} joint and parallel collaborative working; research and audit; in-service training, for example, suicide prevention training\textsuperscript{6}; case discussions; location of team members in the same department; understanding of professional roles and skills\textsuperscript{17}; respectful and positive working relationships\textsuperscript{1,6,7} and the tinnitus education group.\textsuperscript{2,4,16}

**Summary of factors associated with an effective team working BPS summary**—In summary the working in teams report argue that there are several factors are associated with effective team working\textsuperscript{5} as shown in Table 2 Factors associated with effective team working below:

**Table 2: Factors associated with effective team working (Onyett BPS)**

1. Clear and achievable objectives
2. Differentiated, diverse and clear roles
3. A need for members to work together to achieve shared objectives
4. The necessary authority, autonomy and resources to achieve these objectives
5. Capacity for effective dialogue this means effective processes for decision making, being able to engage in constructive conflict and if complex decision making is involved, the team needs to be small enough (no larger than eight or nine people)
6. Expectations of excellence
7. Opportunities to review what the team is trying to achieve, how it is going about it and what needs to change
8. Effective leadership

**Conclusion**

A multidisciplinary team approach is one part of a bigger network or matrix of service provision in the treatment of tinnitus, which can have significant benefits for patients and the teams in providing a service for tinnitus patients.\textsuperscript{4} Understanding the appropriate use of services and networks enables accurate referral, assessment and provision of healthcare in tinnitus management. The tinnitus good practice guide\textsuperscript{4} suggests different levels of tinnitus service provision, according to clinical need, not all of which is provided within a multidisciplinary team. The suggestion that tinnitus patients are best treated clinically by being offered management in a multidisciplinary team is partially supported, in specific instances. Appropriate patient referrals to specialist centres, suggests that designing clear, effective, efficient, holistic multidisciplinary teams\textsuperscript{5} will benefit tinnitus patients and the staff working with them.

A framework for best practice offered by a multidisciplinary team has been demonstrated to provide a number of benefits. Whether this model can be adapted or employed in healthcare settings outside the UK NHS, is certainly an area for further
debate, particularly if there are insufficient resources available or small services exist in large geographical areas without a local specialist centre. Healthcare providers might want to explore ways in which the provision of tinnitus management could incorporate a multidisciplinary team approach. Perhaps a national centre of excellence could be considered as a starting point, given the evidence that a multidisciplinary team approach has advantages for particularly complex and distressed patients in specific instances.

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