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Treatments for Tinnitus

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The various forms of treatment for tinnitus that have been tested in properly controlled trials can be classified as pharmacological, acoustic–physical, and psychological. In clinical trials, no pharmacological agent has been shown to have lasting effect on the presence or severity of tinnitus, although there are promising signs in an animal model. Acoustic devices do not seem to influence tinnitus, although appropriately fitted hearing aids may slightly reduce its prominence. Of physical treatments, cortical implantation may hold some promise of being effective for tinnitus suppression in selected cases. A psychological treatment that has emerged as consistently beneficial is cognitive-behavior therapy in terms of affecting overall well-being and reducing level of tinnitus annoyance.

Keywords: tinnitus treatments; acoustic–physical; pharmacological; psychological; combination; evidence

In this article, the focus is necessarily on people seeking help for tinnitus; otherwise they would not be engaging in one or other form of treatment. What moves someone to seek help for tinnitus is a valid topic for research attention (Attias et al., 1995) but is not further discussed here.

Treatments proffered for tinnitus can be grouped into four main classes: (a) pharmacological, (b) acoustic–physical, (c) psychological, and (d) some combination of elements from at least two of these three. Pharmacological and physical treatments principally aim to affect the tinnitus itself, ideally to eliminate it or reduce its prominence to the point that it is no longer troublesome. Acoustic treatments have various claimed purposes, from that of simply masking or partially masking the tinnitus to that of influencing the operation of the central nervous system in some way, thereby reducing or removing the tinnitus phenomenon. Psychological treatments are aimed at influencing the person's way of reacting to the tinnitus, with the goal of making it less intrusive in consciousness or less distressing in effect.

There have been debates, especially with respect to acoustically based versus psychologically based treatment approaches (e.g., Jastreboff & Jastreboff, 2006; McKenna, 2004), about the quality of the theoretical ground on which either sort of approach rests. Because the mechanism (or mechanisms) of tinnitus is (or are) not well understood, there is scope for speculative theorizing that can generate clamor as much as clarity. And lack of convergence on mechanism leaves open the question of whether it is reasonable to expect there is only one. In this article, an attempt will be made to parse the domains of tinnitus both in terms of how it may be understood as a phenomenon and in terms of rationales for treatment.

This article comments on the kind of evidence that has been advanced in support of one or other treatment. Before going to that matter, there is some attention given to current knowledge of mechanism. It is only when mechanism (whether one or several) has been unambiguously identified that there can be hope of convergence with respect to treatment. The discussion of mechanism offered here is aimed at giving a sense of current understanding in the literature; the question of mechanism(s) is by no means settled.

Left out of present consideration are rare forms of experience of sound that are non-neural in origin but rather because of dysfunctions of blood flow around the head leading to audible pulsing. Appropriate surgery has been reported as able to address such a cause and remove the auditory experience (e.g., Duvillard, Ballester, Redon, & Romanet, 2004; Shownkeen, Yoo, Leonetti, & Origitano, 2001). It is a tenable argument

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that cases of this nature should not be considered instances of tinnitus, because although the origin of the sound is not in the person's external environment, the origin is nonetheless acoustic. By contrast, the essence of tinnitus is the experience of sound in the absence of an acoustic source (see Dobie, 2004b, p. 1, for a related argument).

This paramount feature of tinnitus—absence of an acoustic source—has led to use of the term *phantom* to describe the phenomenon (e.g., Jastreboff, 1995), on the analogy with "phantom limb" pain (Salvi, Lockwood, & Burkard, 2000). It has been argued that although this analogy is fine in terms of pathophysiology, use of a term such as *phantom* may not be helpful in the context of clinical treatment of people complaining of tinnitus (Noble & Tyler, 2007). *Phantom* implies that what the person experiences is somehow nonexistent. Part of the argument about (avoiding) use of the term in the clinical context turns on the point that the missing body part associated with a phantom limb pain can be shown to and be seen by others, thus enabling them to appreciate that there is something amiss. Damaged or absent cochlear hair cells—the likeliest origin of tinnitus—cannot be displayed in this way. A component of tinnitus suffering is surely the difficulty of providing a ready way to allow people who do not suffer with tinnitus to appreciate what is wrong. This, in turn, may explain a finding that variation in levels of social support is unrelated to the degree of distress caused by tinnitus (Erlandsson, Hallberg, & Axelsson, 1992; Murphy, 2007). Social support has been shown in many other contexts of ill health to have a moderating influence on the degree of experienced distress.

Mechanism

There is convergence on a view that the mechanism for tinnitus (understood as a phenomenon generated within the auditory nervous system) involves increased spontaneous neural activity caused by reduction in inhibitory tone, for example, following injury, which typically occurs at the level of the cochlea (Brozoski, Spires, & Bauer, 2007; Kaltenbach et al., 2002). In normal conditions, when the system is intact, excitation of any part of it triggers inhibition directed to other areas, an effect that presumably serves to enhance the final signal-to-noise outcome. When a part of the auditory nervous system is blocked or damaged, outputs from other areas are subject to reduced inhibition; hence greater amounts of spontaneous activity can occur in those areas. Brain imaging studies (e.g., Lockwood et al., 1998) suggest there is eventually remapping at the cortical level as neurons previously activated by output from the affected cochlear region become recruited to receive the increased spontaneous activity from other cochlear or retrocochlear regions.

Kaltenbach et al. (2002) reported that one triggering mechanism for increased spontaneous neural activity may be the selective disruption of outer hair cells, as occurs with application of cisplatin, an agent used in the control of tumors and known to cause hearing loss and tinnitus. These authors found that doses sufficient to damage outer hair cells in the cochleas of hamsters, while leaving inner hair cells more or less intact, resulted in greater signs of hyperactivity in the auditory nervous system (dorsal cochlear nucleus) than doses sufficient to damage both sets of hair cells. Kaltenbach et al. refer to the action of salicylates, well known as tinnitus-inducing agents (Jastreboff, 1995), whose mechanism also involves disturbance of outer hair cell function.

Brozoski and colleagues (2007) induced tinnitus in laboratory rats by unilateral exposure to high-level narrowband noise, an energy form that is especially hostile to cochlear hair cells. The presence of tinnitus in exposed versus control animals was confirmed using behavioral conditioning techniques. These authors investigated the effect of increasing the level of the inhibitory neurotransmitter γ-amino butyric acid (GABA) through feeding their animals with the drug vigabatrin, a substance that acts to slow the metabolism of GABA. On the assumption that the injury resulting from noise exposure disrupts the excitatory–inhibitory balance in the auditory nervous system, it was predicted that the tinnitus would be suppressed by ingestion of GABA, and this was, indeed, observed. Brozoski et al. note that vigabatrin (produced for prescription as an anticonvulsant) is not currently in clinical use because of negative side effects.

None of the above-cited groups has a final story on how or where tinnitus forms as a "signal" in the auditory nervous system nor whether, for example, outer versus inner hair cell output imbalance is the only or even the primary mechanism for inducing tinnitus. But it is probably a common enough cause, given the high prevalence of tinnitus as a consequence of, for example, occupational noise exposure.

Forms of Treatment

Pharmacology: Reviews of Evidence

Dobie (1999, 2004a) has undertaken an extensive review of literature, particularly addressing pharmacological treatments, including antidepressants, anticonvulsants,

and sundry other preparations, such as zinc supplement and ginkgo biloba. Dobie also makes reference to other treatment domains listed in the present article. Only studies that Dobie calls "randomised clinical trials" were included in the review, involving such features as random assignment of participants to treatment versus placebo or nontreatment groups and blinding of participants with respect to whether they were in treatment versus nontreatment conditions.

Certain treatment approaches lend themselves to tight experimental designs in which participants are engaged in crossover procedures (exposure to the treatment of interest as well as the nontreatment, thus acting as their own controls) and in which both participants and investigators are blind as to which is the condition of interest and which is the placebo control. Ideal forms of treatment for this sort of design (sometimes labeled a *randomized controlled trial*) would involve, for example, transcranial magnetic stimulation or low-power laser energy (see later). Such procedures rely on the use of impressive-looking machinery positioned relative to a participant's head so as to deliver (or, in the placebo condition, not deliver) otherwise undetectable energies, such that any effect of the condition of interest is unambiguously tested.

A high level of experimental control may be scientifically laudable but is practically unachievable with some types of treatment. If a form of noise masking is being tested, it is hard to imagine a within-subject placebo version of such a treatment. A quasiplacebo condition was used by Erlandsson, Ringdahl, Hutchins, and Carlsson (1987), who compared a wearable noise masker with an inactive device nonetheless said to participants to be delivering electrical current. In tests of drug compounds, as Dobie (2004a) points out, side effects such as drowsiness may alert participants to the difference between the drug being trialed and an inert placebo, reducing the effectivenesss of blinding. A way around such problems is to have separate experimental and control groups and/or to use a "wait-list" control procedure in which a separate control group is subject to no initial treatment, is nonetheless compared with the treatment group on the outcome measures involved in the trial, then is exposed to the treatment to determine if the response on the outcome measures changes to resemble that of the initial treatment group. The wait-list control feature is found with psychological approaches to treatment.

Dobie (1999, 2004a) reports that there is no consistent evidence for the effectiveness of any pharmacological agent in tinnitus treatment but that antidepressants may be able to alleviate sleep problems for some severely distressed patients. Robinson, Viirre, and Stein (2004) reviewed the specific topic of depression and tinnitus and concluded that there is a higher incidence of anxiety and depressive symptoms in people distressed by tinnitus compared with the incidence of such psychopathology in the general population. This is hardly unexpected: In people for whom tinnitus is distressing, anxiety about its presence, and depression about its uncontrollability, will surely be elevated. Robinson et al. (2004) also report on tinnitus as either caused by, as a withdrawal symptom from, or as improved by treatments using antidepressants. A contrast is made between earlier "tricyclic" antidepressants and the more recently manufactured "selective serotonin reuptake inhibitors," with the tricyclates having a broader spectrum in their inhibitory biochemistry.

From the review by Robinson et al. (2004), it is difficult to distinguish between these two groups of compounds in terms of effect on tinnitus (either as producing or diminishing it). Where a beneficial outcome from use of the newer antidepressants is observed, it is in terms of tinnitus being reported as less aggravating, bothersome, or annoying (Folmer, Griest, Bonaduce, & Edlefsen, 2002; Zöger, Svedlund, & Holgers, 2006). In studies involving use of antidepressants, the primary factor being addressed is ongoing depression and anxiety. Not only are pharmaceuticals involved; patients will be receiving psychotherapy of one kind or another. If a combination of treatments designed to improve mood is effective in that regard, it cannot be too surprising that the degree of distress in connection with tinnitus experience is diminished also. There is certainly no basis from the literature for a proposal that distressing tinnitus absent psychological problems such as depression and anxiety will be effectively managed by use of antidepressants (Robinson et al., 2005).

It can reasonably be asked why tinnitus was able to be controlled as to its presence or absence by dosage with GABA in an animal model (Brozoski et al., 2007) but equivalent findings are not reported in the human case. An obvious contrast is that tinnitus was induced under specific and highly controlled laboratory conditions, whereas clinical studies involve participants from a huge spectrum of backgrounds and histories. Mechanism is more clearly definable in the laboratory than in the tinnitus clinic.

Acoustic–Physical Treatments: Review of Evidence

Acoustic treatments include hearing aids and masking devices. The latter may offer temporary relief but

have no influence on the tinnitus as such (Dobie, 1999). Particular attention to "sound therapies" is given in separate articles in this special issue of the journal. Hearing aids have been shown to offer modest reduction in experienced severity of tinnitus (Surr, Kolb, Cord, & Garrus, 1999; Surr, Montgomery, & Mueller, 1985). Although hearing impairment to some degree (from slight to profound) is found in the great majority of people with distressing tinnitus, hearing aid provision will likely be a feasible option only for those with more noticeable levels of hearing impairment.

A range of other physical treatments has been investigated, including low-power laser, acupuncture, surface electromagnetic stimulation, and ultrasound. In his review, Dobie (2004a) reported no beneficial effect from any of these procedures. A subsequent exploratory study used low-powered laser technology (Tauber, Schorn, Beyer, & Baumgartner, 2003) that involved highly defined dose and positioning within the ear canal. Outcomes suggested that this refinement of the laser procedure may have beneficial value, meriting more systematic investigation.

In the case of electrical stimulation, Dobie (2004a) noted that if an implantable electrical device could be realized, it may offer relief for some patients (there are cases of tinnitus suppression among cochlear implant users; Rubinstein & Tyler, 2004; there are also cases of tinnitus exacerbated by implantation; Summerfield et al., 2006). De Ridder et al. (2006) reported promising results of implantation in the auditory cortex of 12 patients. Those with tonal tinnitus showed almost complete suppression of the tinnitus sensation, suggesting that this procedure could be highly effective in selected cases.

Psychological Treatments: Reviews of Evidence

Psychological treatments for tinnitus seek to address the way in which the person responds to the condition, having no bearing on its presence or absence. A substantial analysis of a range of psychological treatments was undertaken by Andersson and Lyttkens (1999), including cognitive-behavior therapy, relaxation, education–information, hypnosis, biofeedback, and stress management–problem solving. The most extensively studied procedure is cognitive-behavior therapy, and this provided more consistent signs of sustained reduction in degree of annoyance caused by tinnitus than any of the others in Andersson and Lyttkens's meta-analysis (very few of which included follow-up assessments).

A recent *Cochrane Review* (Martinez Devesa, Waddell, Perera, & Theodoulou, 2007) confirms that cognitive-behavior therapy applied in tinnitus treatment has positive impact on overall quality of life, making this procedure a suitable candidate for consideration as a management option. Cognitive-behavior therapy forms part of clinical psychology training. Noble and Tyler (2007) suggest that flexible coalitions between clinical audiology and clinical psychology may be an optimal model for tinnitus treatment. Some people worried by the onset of tinnitus may need no more than reassurance, following appropriate audiological assessment; others may continue to be distressed to a point that calls for psychotherapeutic intervention, such as application of cognitive-behavior therapy.

The cognitive-behavior protocol has been developed and successfully offered in self-help form using Internet resources (Andersson, Stromgren, Strom, & Lyttkens, 2002) or as printed text (Kaldo, Cars, Rahnert, Larsen, & Andersson, 2007). These ways of providing such psychotherapy can be argued to be cost-effective; however, outcomes are less substantial than with more usual treatment pathways.

Combined Treatments

Tinnitus retraining therapy (Jastreboff & Hazell, 2004) combines what its authors call "directive counselling" with partial masking. The counseling component has been the subject of some criticism (e.g., Wilson, Henry, Andersson, Hallam, & Lindberg, 1998). Subsequent authors have used a more recent variant of the counseling component (Zachriat & Kröner-Herwig, 2004). The consensus is that the partial masking component of this procedure adds nothing beyond what may be achieved by the counseling component (Goebel et al., 1999; Hiller & Haerkotter, 2005) and that the more recent variant of the counseling component is not as effective as cognitive-behavior therapy in terms of improved general well-being (Zachriat & Kröner-Herwig, 2007).

A different approach to partial masking uses filtered music in combination with a counseling program (Davis, Paki, & Hanley, 2007). Independent assessment of this approach remains to be reported.

Conclusion

Tinnitus looks like it will continue to be part of the landscape and no doubt will continue to attract a very substantial amount of clinical and research

interest, as it increasingly has in the past half century. From the foregoing survey, it can be said that a promising line of physical treatment may emerge from further study of cortical excitation and inhibition. Cognitive-behavior therapy has emerged as the psychological treatment of choice.

Although it is orderly to identify treatment approaches as biochemical, physical, or psychological, it may be more productive to recognize tinnitus as a somatopsychic disorder, that is to say, a bodily dysfunction that has substantial mental consequences. The phenomenology of tinnitus cannot be isolated from its physiology (Noble & Tyler, 2007), and no treatment domain can presently be identified as holding prime position in the therapeutic spectrum. Clearly, if some order of physical procedure sustainably eliminates tinnitus as a "signal," and such procedure is accessible and reliable, that will surely take first prize. As matters currently stand, such an outcome is not in sight. All clinical options, therefore, should remain in play. The advantage of psychotherapy is its accessibility and its potential to benefit the person's overall quality of life.

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