International Tinnitus Journal. 2010;16(1):66-72.

A Cognitive Model of Tinnitus and Hyperacusis; A Clinical Tool for Patient Information, Appeasement and Assessment

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Abstract

Tinnitus and hyperacusis are both aggravating audiological symptoms. Their underlying mechanisms are not fully understood, but the pathophysiology involves a central mechanism rather than a peripheral one. There is no curative treatment. A review of the available research on tinnitus and auditory processing was conducted to connect insights gained from different approaches to the subject; this resulted in the development of a holistic view of both conditions. In this view, the chronic course of the symptoms is pathological and attributed to a stress-related lack of habituation. This article adds to the literature on tinnitus and hyperacusis by presenting a schematic model of the cognitive mechanisms which can be used clinically in patient information sessions which are geared towards provide reassurance and encouraging the development of coping skills. In cooperation with the patient, the model can also help in the identification of underlying pathology. Future aims of study are suggested, elaborating on the role of tinnitus and hyperacusis in normal auditory processing and on the value of insight. Finally, parallels are drawn between tinnitus and positive symptom syndromes in neuropsychiatry and some of its modern visions on their treatment.

Keywords: cognitive, hyperacusis, tinnitus.

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INTRODUCTION

The continuous perception of tinnitus is a common and often serious health problem[1-3] with considerable costs. Hyperacusis, which is the phenomenon of decreased sound toleration, is a known comorbidity. The research on hyperacusis is very scarce, but it has been suggested that its mechanism involves peripheral disruptions or the central sound processing at the subcortical level.[4] Tinnitus and hyperacusis seem to be connected by more than coincidental factors.[5] In 1993, Jastreboff and Hazel regarded hyperacusis as a pretinnitus state.[6]

Tinnitus can be considered as a 'positive symptom disorder' characterized by neural hyperactivity due to the loss of afferent inhibition.[7] Although tinnitus is often related to hearing loss,[7, 8] not all tinnitus sufferers have an audiologically objective perceptive hearing loss and many, but not all, hearing impaired people have tinnitus. This suggests that the auditory pathway does not play a decisive central role in tinnitogenesis alone. It has often been suggested that hearing loss is the basis for tinnitus development but that tinnitus must be a far more complex (top-down) phenomenon than just a reorganization of the auditory cortex after damage to the cochlear hair cells.[9] It is suggested that additional processes such as attention, cognition and fear play a role in tinnitus.[10] Since dysfunctional neuroplastic processes are believed to be involved in the mechanism(s) underlying tinnitus and hyperacusis, human studies have used many different techniques to study the brain in tinnitus patients.[10-16] These studies have shown changes in activity in the right hemisphere, tonotopic map changes in the auditory cortex, structural changes in the thalamus, and the limbic system and subcallosal regions, and the involvement of a frontal and amygdalohippocampal circuit. Although these study results have had important implications for tinnitus modeling, most of them did not match their control groups for hearing loss. Animal studies point at the dorsal cochlear nucleus and inferior colliculus as possible sites for initiating tinnitus.[17, 18]

Although the neuroscientific research on tinnitus still has a lot of methodological problems to overcome, it does reveal that there is an imbalance of excitation and inhibition at almost every level of the auditory pathway.[19] This is caused by adaptive (neuroplastic) activity changes. Early manifestations of these neuroplastic changes are believed to be the diminishing of inhibition and subsequently hyperactivity. Late manifestations are the remodelling of the tonotopic areas.[20]

Despite extensive scientific research conducted on this subject, a solid understanding of the underlying mechanism has not been achieved, nor has a curative treatment been found, although numerous therapeutic suggestions, mostly on the management of tinnitus, have been made. Tyler gives an extensive overview of various possible types of treatments and counselling protocols.[21]

A focus on the functional interactions within and between neural networks in auditory processing can provide a useful perspective into the neural mechanisms underly-ing tinnitus and hyperacusis.[22] In this article we will integrate the data on auditory processing and neuroplasticity related to tinnitus with psychological facts. Based on this, we will describe tinnitus and hyperacusis as maladaptive neuropsychological phenomena and present a schematic model, educational for patients and addressing their overall emotional well-being and need for insight. This could be the basis of a very efficient group intervention and be the starting point for a multidisciplinary procedure.

The neuropsychology of auditory processing

Central auditory processes are defined as all the mechanisms and functions which are responsible for behavioural auditory phenomena.[23] These processes are divided into several parts or steps. First there is a conductive part; this is followed by Auditory Scene Analysis, which is subconscious.[24-27] After that, the application and regulation of emotional values takes place. The next step involves the cortical processes where attentional bias and reasoning lead to the behavioral outcomes related to the initial stimulus.

In anatomical terms: after the auditory nerve enters the brainstem, one part of the axons connects with the reticular formation and cerebellum which is responsible for the arousal and startle-reflex. This leads to an overall neurophysiological state of 'fight-or-flight-readiness'.[28] Another part of the axons connects with the colliculi inferiores through a ventral and a dorsal route.[26] These two separate routes of sound processing create a system of perception ('what') and a system of orientation ('where'). Perception and orientation are parallel processes of auditory stimuli which influence the levels of emotional and cognitive auditory processing.[29]

The 'where' route follows a temporo-fronto-parietal network. The temporal regions are believed to be involved in differential processing and the parietal and frontal areas for task related processes such as attention regulation and motor preparation.[30] Attention regulation results in different aspects of sound perception.[31] Memory usage in the orientation route has been related to the efficient processing of sound stimuli so as to differentiate between what sound is safe and what is not.[32]

The 'what' route starts with the fibres going from the colliculi inferiores through the medial geniculate body of the thalamus to the cortical auditory areas.[28] In Auditory Scene Analysis, sound segregation leads to distinct auditory streams. This is enhanced by information from the 'where' route of processing.[33] Sequential elements on the

streams which are formed are grouped into perceptual units, for identifying changing patterns, such as speech.[34] The analysis of stimulus content depends (amongst other things) on implicit (subconscious) memory.[35-44] The memory processes are used as a template against which incoming sounds are compared.[45, 46]

Memory traces may play a role in perceptual illusions such as the continuity effect.[47, 48] This is the auditory analogue of a completion illusion,[49, 50] the process by which perceptual information is organised in order to form stable representations.[49] Continuity ensures the accuracy of perception and orientation and seems to precede the initiation of attention switches and choices, which is important for social behaviour. The emotional state and motivation which is needed for behaviour to occur, involve limbic structures, including the hippocampal formation and the amygdala. Connected to these limbic structures are the basal ganglia, a series of nuclei which has an afferent part (known as the striatum) which is involved with selective attention[51]. Part of the striatum is the nucleus accumbens (NAc) which regulates the transfer of motivational and emotional signals received from the prefrontal cortex, the amygdala and the hippocampus to adaptive behavioural responses[52]. In doing so, the NAc occupies a central role in a network for (emotional) learning.[22] One aspect of emotional learning is called Latent Inhibition (LI), which is a process of habituation. The habituation of a continued stimulus is obtained by inhibiting subsequent associations based on hippocampal retention (memory) with a pre-exposed sound.[22] Based on a small study with functional magnetic resonance imaging, hyperacusis might be related to the neural network associated with the frontal lobes and parahippocampus[53].

In 2006, Muhlau and collegues found an increase of (right sided) thalamic activity and a significant decrease of the NAc volume[54] in tinnitus patients. This alteration of volume was thought to be directly related to functional changes in brain activity[55,56]. A decrease of NAc activity would result in LI disruption. Preceding chronic (pathological) stress can cause this poorer functioning of the NAc[57,58,59]. The result is disinhibition[60] or lack of habituation. This would then cause a pathological ongoing of completion in stimulus content analysis, consistent with Hallam's habituation theory[61,62].

A recent study was not able to replicate these findings.[11] Instead the results showed a decrease of grey matter in the right inferior colliculus and a decreased grey matter concentration in the (left) hippocampus. The authors suggested that decreased inferior colliculus indicated a compensatory mechanism, because many other research groups found hyperactivity in that area[12, 63, 64]. Furthermore they suggested a direct involvement of the hippocampus in tinnitus pathophysiology.[11]

It is possible that the findings of Muhlau (2006) and Landgrebe (2009) do not conflict, but reflect different stages of stress regulation instead. A review of the research on stress and memory indicates that chronic stress, measured by cortisol levels, has a reducing effect on the activity of the hippocampus[65] and on NAc functionality [57, 58, 59]. There are also indications that the effects of reduced Hippocampus activity by cortisol abolish the cortisol response to the stressor[66]. Thus the damaging effect on the NAc is abolished to recover habituation processes. Content analysis (including continuity effects) and Latent Inhibition depend on the hippocampus to be able to compare new incoming auditory stimuli with consolidated auditory information which has been experienced earlier.[15] They also appear to depend on the hippocampus because of its important role in restoring the balance of the response to psychosocial stress[66] and thus restoring habituation.

The neuropsychology of tinnitus and hyperacusis

Feelings, cognition and knowledge of the world, based on former personal experience, are part of auditory processing and perception in general. They relate, at least in part, to psychological and social functioning. Consequently, hearing loss leads to perceptual and communicational problems, and to a reduction in understanding what is around us[67]; in a social context, this creates the threat of social isolation[68]. It is necessary for the brain to adapt to these threats and to compensate for them. Changes in activity within structures of the auditory pathways after altering peripheral input[19, 20] can be seen as manifestations of adaptive compensations in the central auditory system.

Attention has been neuropsychologically defined as an abstract result of activation in the neural circuits[69]. In speech comprehension, activity in the frontal-temporal network can be seen (poorer signal-to-noise-ratios of spoken sentences)[70]. With specific frequencies missing in hearing (e.g. noise induced hearing loss), sound discrimination inherent in speech comprehension also becomes more difficult. The lack of hearing specific frequencies is, therefore, associated with increased activity in the primary auditory cortex and frontal areas[70, 71]. These activation patterns induce (more) attention.

With severe hearing loss, misperception (and often disorientation) occurs. The patient uses mental effort trying to locate the source of the sound and, in so doing, increases the attention given to it[71]. On the other hand, with less severe hearing loss, one may not be aware of the impairment and, in that situation, stimulus content (e.g. speech) is deduced using implicit memory.[42]

In brief: cochlear damage results in decreased perception and orientation, requiring compensation by attention and memory through elicited (amygdalo)

hippocampal and frontal-temporal network activation[70, 72]. From this perspective hyperacusis is not a pretinnitus state, but a symptom of its own with a distinct role in orientation. In fact, hyperacusis might be a secondary symptom of threat by tinnitus.

Even though the majority of tinnitus sufferers have a certain amount of hearing loss, it is possible to experience tinnitus or hyperacusis without having a higher than normal amount of damage to the inner ear hair cells. To increase the sense of safety by enhancing auditory perception and orientation[73], auditory completion and hypervigilance can also be expected in psychologically threatening conditions. In these conditions, obviously chronic stress is present and a decreased functioning of the NAc[57] and hippocampus[65] is exhibited. Under those circumstances, neuroplastic compensations occur even in normal (age-related) hearing or mild hearing loss. As humans are interactive social creatures, it can be caused by psychological or psychosocial factors.

The relatively complex processes of auditory completion and hypersensitivity are shown in a relatively simple schematic model (see Figure 1).

The figure illustrates the relationship between hearing loss, psychological well-being and cognitive functioning. For tinnitus or hyperacusis to develop, one has to fulfill either one of two conditions (or both); a hearing loss or a chronic (neuro)psychological overburdening.

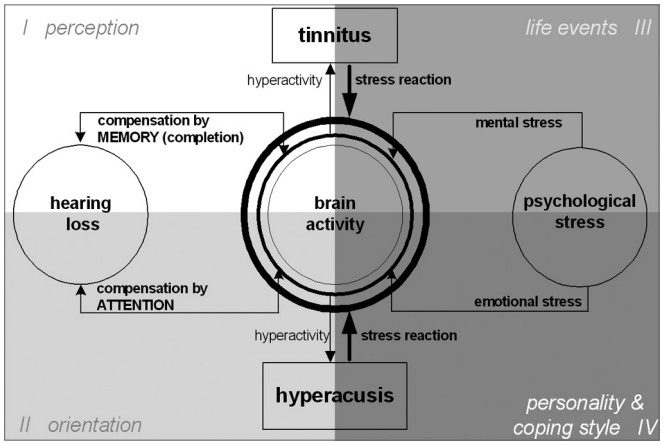

Figure 1. The neuropsychological model of tinnitus and hyperacusis. This schematic model illustrates how attention and memory can be seen as key cognitive instruments affecting auditory behavioural outcomes. The context of increased neural activity is formed by four domains (I - IV) in which human auditory processing takes place: what is where in relation to ourselves versus human psychological functioning in relation to stress toleration and life experiences. The first (innermost) circle of the brain activity shows normal activity, the second (inner) circle reflects the neural over-activity caused by chronic stress, either due to audiological or to psychological threat, which causes the onset of tinnitus or hyperacusis. The thick outer circle reflects the vicious circle of brain hyperactivity caused by reactive stress of tinnitus/hyperacusis, leading to progressive exacerbation of tinnitus or hyperacusis.

First, with a significant hearing loss, brain activity in the auditory areas increases producing a degree of attentional compensation on behalf of *orientation*: a conscious focus on sound is made and there may only be a minor subjective complaint of hearing loss. Instead, there is a growing complaint of fatigue and neuropsychological overload[73]. This is thought to result in hyperacusis, when orientation is diminished, or in tinnitus because of the effort to complete perception. As a result of hearing loss the completion is continuous and, because it is a symptom of sensorial stress, it is perceived itself. If an emotional (stress) reaction then occurs, a vicious circle is created; this explains why some people are bothered by it and others are not.

On the other hand, stress-related brain activity is also a factor in psychopathology, in chronic psychosocial stress and in chronic pain, or it can be a factor related to the use of sedatives or extreme fatigue, or to psycho stimulant withdrawal or brain trauma. As a result of the psychological overload, normal attentional compensation and completion of very small or even normal hearing loss (e.g. at ultra high frequencies) can be perceived as either hyperacusis or tinnitus respectively[74] and the emotional reaction which immediately follows, corresponding with the pre-existing mood, creates a vicious circle leading to chronicity and progression.

Although a stress reaction is a normal reaction to unknown sounds, it can lead to hyper-cortisolism and neural damage to the areas involved in habituation processes. The intensity of stress reactions and neural damage depends strongly on the individual's personality and coping abilities and the degree of appeasement offered by the health care professional who is consulted. The focus of treatment should be on the underlying cause of the neuropsychological stress created during the period before the appearance of the symptom(s). From a neuropsychological perspective, this may either be hearing loss and/or chronic stress due to psychiatric, psychosocial, or somatic suffering; all of which are factors which are open to treatment once they have been identified.

Figure 1 can be clinically used in an informational session focused on giving answers to some of the most distressing questions that tinnitus patients present with. The scheme can also provide a tool for a diagnostic interview, and may lead to answers to the question: 'why'.

Implications for multidisciplinary treatment

We have integrated the neurobiological findings of tinnitus research with neuropsychological views about auditory perception. In the modern world, with epidemic stress or psychopathology on the one hand, and risks of cochlear damage on the other, neuroplastic adaptation can become imbalanced. The neuropsychological approach provides an easy and accessible insight into

the comprehensive and highly complex neurobiological processes. It can be clinically used during an initial information session which is aimed at reducing the stress reaction. This stress reaction is based upon a threatening association[75]. Differentiating between the initial stress and the reactive stress by explaining the model shown in Figure 1 to a group of patients can provide answers to their questions; it is also reassuring because of the move from non-treatable symptoms to controllable underlying problems. It can, therefore, break the reactive vicious circle. Also, the clinical use of Figure 1 presents an opening for the caregiver to help the patient to overcome his/her somatic fixation. Furthermore, this approach also provides rationales for the various treatment options as it suggests that, after gaining insight, successfully treating the underlying pathology and applying relaxation exercises, hyperacusis and tinnitus can be controlled.

The first suggestion for therapy arising from the model is to treat preceding existing hearing loss, if sufficiently present. The suggested treatment for perceptive hearing loss is the fitting of hearing aids with careful adjustment based on a tone audiogram.

Second, the schematic model points to the need for an extensive diagnostic interview and examination of the patient's (neuro)psychological well-being before the onset or sudden aggravation of the tinnitus or hyperacusis; this is especially important if no significant hearing loss is found. Underlying pathology related to emotion (e.g. mourning, depression), impulses (anxiety, anger, frustrations), mental strength (e.g. cerebrovascular accidents, brain trauma, exhaustion, work related burnout) and physical condition (chronic pain, hormonal imbalance) can be expected, most of which can still (and obviously should) be treated. This is an important distinction with counseling as usual, which is aimed at the acceptance of tinnitus or hyperacusis.

Although similar theoretical essences have been outlined elsewhere[76], the schematic model presented in this article is unique in that it can be used to visualize very complex processes in a very efficient educational group session aimed at improving the patient's insight and providing reassurance. What's more, it can also be used diagnostically in cooperation with the patient, giving rise to treatment options for the underlying pathology of which tinnitus and hyperacusis are only the clinical symptoms. In our opinion, education, insight, reassurance and these treatment aims result in getting the better of tinnitus by the patient, which leads to the reduction of tinnitus distress and restored habituation.

RECOMMENDATIONS

Patients with tinnitus or hyperacusis are often in distress and are desperately looking for treatment, information and advice. They most often present at Ear, Nose and Throat clinicians, who have no other therapeutic options than to, at best, offer them psychological counseling. Also in psychiatric clinics, tinnitus or hyperacusis can be the patient's dominant complaint(s), which is often ignored because of the lack of understanding.

Future research could build upon this model to provide a better understanding of tinnitus and hyperacusis and their functions in auditory processing and neuropsychological functioning. In the model, networks of non auditory areas such as the NAc, the hippocampus and limbic structures are believed to be key sites with respect to lack of habituation to a normal neuroplastic phenomenon of auditory scene analysis. The NAc, in particular, has a wide spread of connections with (nearly) all brain areas in which hyperactivity has been shown in tinnitus subjects. Its activity is regulated by dopaminergic and glutamatergic afferents, but is also modified by GABAergic, serotonergic, adrenergic and cholinergic afferents[57]. Focusing on this region could be an extra spur for research into pharmacological or other curative treatments for tinnitus. Furthermore, this particular region has already turned out to be of major importance in neuropsychiatric disorders characterized by positive symptoms[77]. In that field of interest, breakthroughs have been made by experiments with deep brain stimulation (DBS)[78]. Shi (2009) has pointed out that DBS could be promising for tinnitus patients too[79].

Finally, although it is widely accepted that providing information should be one of the first interventions in an intensive multidisciplinary protocol because of the need for appeasement,[21] the information which is taught worldwide differs substantially and no clear consensus exists as to what information should be taught. Research into the effects of educational interventions based on contemporary knowledge is desperately needed. A precise determination could then be made as to what information is required to be able to reassure patients and enhance their sense of control in the face of powerlessness, and how this relates to the subjective perception of tinnitus or hyperacusis[80,81] and habituation or extinction.

ACKNOWLEDGEMENT

We thank Professor G. Borst of the Department of Neuroscience, Erasmus Medical Center, Netherlands, for his expert review of the concept version of this article and his useful recommendations for improvements of the schematic illustration of the model. We also thank reviewers from earlier versions of the article for their recommendations.

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