Sleep Medicine Reviews 17 (2013) 65–74

Contents lists available at SciVerse ScienceDirect

Sleep Medicine Reviews

journal homepage: www.elsevier.com/locate/smrv

THEORETICAL REVIEW

# Tinnitus and insomnia: Is hyperarousal the common denominator?

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ARTICLE INFO

*Article history:*
Received 7 February 2012
Received in revised form
12 April 2012
Accepted 13 April 2012
Available online 30 June 2012

*Keywords:*
Tinnitus
Sleep
Insomnia
Hyperarousal
Depressivity
Anxiety
Somatic symptom severity
Sympathetic nervous system
Hypothalamus-pituitary-adrenal (HPA) axis
Ageing

SUMMARY

Tinnitus is an auditory sensation that is generated by aberrant activation within the auditory system. Sleep disturbances are a frequent problem in the tinnitus population. They are known to worsen the distress caused by the tinnitus which in turn worsens sleep quality. Beyond that, disturbed sleep is a risk factor for mental health problems and distressing tinnitus is often associated with enhanced depressivity, anxiety, and somatic symptom severity. Moreover there is evidence that therapies which alleviate tinnitus-related distress have a positive influence on sleep quality and help interrupt this vicious cycle. This suggests that distressing tinnitus and insomnia may both be promoted by similar physiological mechanisms. One candidate mechanism is hyperarousal caused by enhanced activation of the sympathetic nervous system. There is increasing evidence for hyperarousal in insomnia patients, and animal models of tinnitus and insomnia show conspicuous similarities in the activation pattern of limbic and autonomous brain regions. In this article we review the evidence for this hypothesis which may have implications for therapeutic intervention in tinnitus patients with comorbid insomnia.

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## Background

Sleep disturbances are the second most frequent comorbid condition among tinnitus patients.[1] Tinnitus and insomnia tend to intensify one another, and successful tinnitus therapies often improve insomnia complaints.[2] Studies exploring the relation between tinnitus and insomnia are sparse, however.[1,3–7]

In this review, we put forward the hypothesis that hyperactivity of the sympathetic nervous system might promote the emergence of a distressing tinnitus and at the same time might be the underlying etiologic factor in primary insomnia. In addition, we include data about sleep quality in persons with tinnitus from a survey with 4705 participants and discuss physiological evidence for an association of hyperactivity in the sympathetic nervous system with distressing tinnitus and insomnia, respectively.

### *Tinnitus*

Tinnitus is the perception of a sound that originates from self-generated abnormal neural activity in the auditory system, and that is heard only by the affected person. Tinnitus is rather common,[8] it is considered to be a symptom that is associated with various diseases and that may be a side effect of medications. The majority habituate to their tinnitus, but about 10–20% of the tinnitus patients feel severely handicapped.[9] The prevalence of tinnitus increases with age and more men than women are affected.[8] Although tinnitus is usually accompanied by cochlear impairment,[10] and its perceived frequency is usually located within the frequency range of the hearing impairment,[11] tinnitus is not an automatic consequence of hearing impairment.

Tinnitus complaints can be classified into a category associated with its perception like persistence of awareness, perceived loudness and pitch, and into a category reflecting reactions to this percept, i.e., the distress associated with it. The subjective loudness which is a relevant tinnitus feature for the affected individual can be derived only from subjective rating, because physiological loudness measures deviate substantially.[12] Likewise auditory features are not correlated well with tinnitus-related distress.[13] On the contrary, highly distressing tinnitus is often accompanied by depressive symptoms (depressivity), anxiety, somatic symptom severity, and insomnia.[14–16]

Several ways of interaction between tinnitus and insomnia have been proposed. For example reduced masking by environmental sounds because of low external noise levels during sleep onset may

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doi:10.1016/j.smrv.2012.04.003

increase awareness of the tinnitus sensation. Furthermore anxious focussing on the tinnitus sound and worries about the tinnitus before falling asleep or following awakening during the night might prolong the time that is needed to return to sleep.[13]

*Physiological and anatomical changes associated with tinnitus*

Since tinnitus often persists after auditory nerve transsection,[17] processes in the central auditory system supposedly play a major role in its maintenance even though the primary cause for tinnitus is very likely to be associated with cochlear impairments.[10] As a conscious and often continuous percept, tinnitus is thought to require continued activation of cortical networks engaged in conscious perception.[18] In accordance with that assumption, brain imaging studies reported changes in the activation patterns of cortical and subcortical auditory areas as well as in areas related to emotion and attention (review in[19]). In support of a crucial role of attention for the tinnitus perception is the observation that activity levels in the auditory cortex of tinnitus patients are reduced by cognitive distraction,[20] and that attention to the tinnitus is more pronounced in patients with severe tinnitus-related distress.[21] In addition to activation of the auditory cortex, activation of the amygdala[22] as well as the anterior cingulate and insular cortices were shown (review in[19]). Morphometric alterations associated with chronic tinnitus are gray-matter increases in the auditory thalamus as well as gray-matter decreases in the auditory cortex, and in the subcallosal region including nucleus accumbens[23–25] Cortical gray matter decreases may, however, be a consequence of the hearing loss that is usually accompanying tinnitus and not of the tinnitus itself.[26] Besides that functional connectivity between anterior cingulate cortex and right frontal lobe was found to be correlated with tinnitus-related distress,[18] and functional connectivity was increased between auditory cortex and the amygdala.[27] Finally, magnetoencephalography (MEG) and electroencephalography (EEG) recordings suggest that tinnitus is associated with decreased alpha and increased delta and gamma-oscillatory brain activity.[28–31]

*Tinnitus mechanisms*

The psychoacoustical approach associates the emergence of tinnitus with alterations in the auditory system which are related to a hearing deficit. It concentrates on the psychophysical tinnitus characteristics and is substantiated by a vast body of animal data.[11,32–35] In this context, tinnitus is thought to be heard, if spontaneous neural activity exceeds the level that is perceived as silent or if spontaneous activity is excessively synchronized giving rise to patterned activity. As tinnitus may persist after sectioning of the auditory nerve[17] continuing tinnitus was furthermore supposed to be a consequence of central gain adaptation which is observed following acute hearing loss.[11] Aberrant activation should be present at the cortical level since conscious perceptions such as tinnitus presumably arise cortically (review in[15]). Eggermont and Roberts[36] propose that peripheral damage causes a loss of central inhibition, and that this leads to increased excitation and reorganization in auditory cortex. This is supported by the finding of a down-regulation of inhibitory amino acid neurotransmission in the central auditory pathway of animals in response to cochlear hearing impairment.[37]

According to the thalamocortical dysrhythmia (TCD) model[38,39] loss of auditory input into the thalamocortical auditory feedback loop via the ascending auditory pathway results in excessive thalamic cell membrane hyperpolarization which causes low-frequency oscillations. This kind of activity is normal during delta sleep, but is seen as pathological when occurring continuously and with large scale coherence during wake.[38] Since the ascending auditory system is topographically organized, fast frequency gamma oscillations are thought to arise at the boundary between locations receiving reduced auditory input and locations with normal auditory input. This is thought to be a consequence of reduced lateral inhibition in this area of auditory cortex (edge effect). Changes in oscillatory brain activity are observed in MEG and EEG recordings of tinnitus patients (e.g.,[29–31,40]), and there is direct evidence that certain gamma oscillations relate to the tinnitus percept.[28]

Tinnitus-related distress, i.e., the reaction on the tinnitus percept, appears to be associated with activation of cortical and subcortical areas concerned with the processing of emotions and with brainstem centres of the autonomic nervous system.[41] The neurophysiological tinnitus model[41,42] proposes that tinnitus-related distress is the result of a conditioning process by which a stressful, unconditioned stimulus is linked to the tinnitus signal. This process is supposed to be under control of the amygdala which in turn controls the autonomic nervous system. Activation in this system supposedly prevents habituation to the tinnitus signal.[13] This model is supported by the observation that the amygdala a crucial structure for the emotional evaluation of sensory stimuli as well as for emotional learning and conditioning[43] is highly active during tinnitus induction in animals,[33,44] and that temporarily sedating the amygdala in patients may temporarily reduce the tinnitus.[22] In accord with the neurophysiological model, a decrease of autonomic reactions to the tinnitus during the initial stages of habituation therapy is observed and a decrease in the level of tinnitus-related distress can be achieved without changes in tinnitus loudness.[45,46]

A recent model integrates and extends the major aspects of the neurophysiological and the TCD model. It is based on observations in tinnitus patients of altered activation in the auditory cortex and in striatal areas that belong to a general appraisal network.[24] In this model the cortio-striatal interaction is thought to act as a noise-cancellation system tuning out uninformative background noise by dampening abnormal activity in auditory brain centres. For some as yet unknown reason, tinnitus-related oscillatory brain activity which is thought to arise as described by the TCD model and fed into this system via the amygdala escapes cancellation.

*Current tinnitus therapies*

Essentially, there exist two different therapeutic approaches that are combined in some of the current tinnitus therapies.[47] One approach focuses on the reduction of the tinnitus percept, whereas the other focuses on a reduction of the reaction to this percept. Evaluation of therapeutic success and comparison of different approaches is complicated by the circumstance that there is no universally used measure to scale tinnitus severity. Several measures are currently being used and a modified scaling has just been proposed.[48]

Many tinnitus patients benefit from stimulation of the auditory system either by sound or electrically, and the tinnitus perception is often diminished by the use of hearing aids or cochlear implants ([49] review in[50]). Therapeutic sound comprises either the whole audible frequency spectrum,[47] the region of impaired hearing excluding the tinnitus frequencies (notch filtering[51]), or the frequency spectrum of the sound deprived regions including the tinnitus frequencies.[52] Notch filtering the energy spectrum of auditory stimuli around the individual tinnitus frequency is thought to silence the auditory neurons involved in tinnitus perception by means of lateral inhibition.[51] The alternative approach, stimulating the frequency range affected by the hearing impairment is motivated by the hypothesis that hyperactivity of neurons with characteristic frequencies at the tinnitus pitch is caused by a loss of input resulting in a compensatory increase in gain, and that hyperactivity of these neurons can be reduced by stimulating them.[11] Though auditory stimulation suppresses the conscious perception of tinnitus in a substantial portion of the tinnitus patients, the exact mechanism and consequently the most effective type of stimulation still has to be

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