European Geriatric Medicine 6 (2015) 170–174

Available online at ScienceDirect www.sciencedirect.com

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Hot topic in geriatric medicine

Current perspectives of tinnitus and its therapeutic options

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ARTICLE INFO

Article history:
Received 25 October 2014
Accepted 12 January 2015
Available online 31 January 2015

Keywords:
Tinnitus
Hearing impairment
Vascular anomalies
Ménière's disease
Vestibular
Schwannoma

ABSTRACT

Tinnitus (noise or ringing of the ears) is a common problem with a higher prevalence in industrialized countries. The basic division distinguishes objective tinnitus, often resulting from vascular anomalies, and subjective tinnitus, which is more common and whose etiopathogenesis is not yet fully understood. Risk factors for tinnitus are common cardiovascular risks, elevated BMI, hypertension, dyslipidemia, exposure to excessive noise and hearing disorders in general. An important role is also played by psychological aspects of the individual. Tinnitus can be associated with other diseases of the nervous system such as multiple sclerosis or a vestibular schwannoma. Tinnitus can also be experienced by individuals using various classes of drugs (salicylates, aminoglycoside antibiotics, cytostatics). Treatment of tinnitus is challenging; various therapeutic strategies have been developed such as pharmacotherapy, physical therapy, psychotherapy, and rehabilitation.

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1. Introduction

Tinnitus (from the Latin word tinnitus meaning "ringing"), or noise in the ear, is not perceived as a single disease entity but as a symptom associated with a variety of related medical conditions. Its common definition includes perception of sound without an adequate acoustic stimulus. An exception from this is objective tinnitus whereby the acoustic stimulus can be identified. Tinnitus has been shown to have a variety presentation, intensity, and triggers. It is a relatively frequent condition, which can appreciably impair quality of life and, in its severest forms, may lead to suicidal behavior.

2. Basic classification

Essentially tinnitus is divided into objective and subjective one. Objective tinnitus has been related to vascular anomalies (vascular cause) or to Eustachian tube dysfunction – contraction of the soft palate muscles (mechanical cause). Vascular etiology tinnitus is typically persistent, regular, and synchronous with heartbeat. This may be due to impairment both at the level of the carotid system and that of the vertebrobasilar arterial system, with varying tinnitus intensity on compressing the afferent artery. This group is verifiable (i.e., the tinnitus can be heard) and the condition can usually be managed causally [1–3].

Subjective tinnitus is much more variable including as it does permanent or variable problems of constant or varying intensity and ranging from clear tones through various bruits to noises. Not infrequently, tinnitus intensity reflects the psychological state of the affected individual. In most cases, the tinnitus occurs at frequencies of about 3 kHz; by contrast, the tinnitus in Ménière's disease is usually a low-frequency (about 125–250 Hz) noise [4,5] (Table 1).

Another criterion applicable to tinnitus classification is duration of the problem. Acute tinnitus is usually experienced on exposure to excessive noise and not seldom is associated with sensorineural deafness (perceptive hearing impairment). It is treated with standard vasodilators, corticoid therapy, vitamin therapy or, possibly, and hyperbaric oxygen therapy. In tinnitus sufferers receiving inadequate or late therapy, the condition may progress to a chronic stage. Chronic tinnitus is a condition with complaints lasting 6 and more months. While there are inconsistent data in the literature regarding tinnitus irreversibility with respect to its duration, it is generally accepted tinnitus becomes irreversible after a period of 2 years. However, its chronicity is not always related to the therapeutic effect [6].

3. Incidence, prevalence, and risk factors

In industrialized nations, the reported incidence of subjective tinnitus varies being in the range of 8 to 15%. Population-based

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http://dx.doi.org/10.1016/j.eurger.2015.01.004
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Table 1
Basic classification of tinnitus.

Tinnitus – objective type	Tinnitus – subjective type
Vascular type (arteriovenous malformations, vascular tumors, etc.)	Idiopatic
Mechanical type (Eustachian tube dysfunction, palatal myoclonus, tensor tympani and stapedius myoclonus)	Otologic disorders
	Neurological disorders (sclerosis multiplex, vestibular schwannoma)
	Ototoxic factors
	Metabolic disorders
	Temporomandibular joint disorders

studies designed to assess hearing impairment in adult patients aged 48 to 92 years have reported a prevalence of 8.2% (start of study) with an incidence of 5.7% during 5-year follow-up [7]. Tinnitus prevalence has been shown to increase with age.

Risk factors for developing tinnitus reported in population-based studies include cardiovascular risk factors, exposure to excessive noise, psychological status of the affected individual and hearing disorders in general. Cardiovascular risk factors associated with a higher incidence of tinnitus include increased body mass index (BMI), smoking, hypertension, diabetes mellitus, and dyslipidemia (average incidence 21–24% of the general tinnitus population as against 28–32% in at-risk patients). Exposure to noise has also been related to a higher incidence of tinnitus (average 24% of the general population vs. up to 39% of the exposed population). A clear association has been documented between the psychological state of the individual and tinnitus; in particular, the condition is more often experienced by depressive patients (19% vs. 39.5% of depressive patients). Tinnitus is more often associated with hearing disorders (20% vs. 30–37%). These data are based on a study including over 14,000 respondents with an average tinnitus [8,9] prevalence of 25.3% in the individual subgroups [7,10] (Table 2).

A crucial role in the development of tinnitus is played by ototoxic factors. Drugs proposed to have an otoxotic effect include some antibiotics, salicylates, loop diuretics, and cytostatic agents, usually causing both sensorineural deafness (perceptive hearing impairment) and tinnitus. The effect of drugs is highly dose-related and dependent on the affected individual's sensitivity. Ototoxic antibiotics, in particular aminoglycosides (streptomycin, gentamicin, neomycin), bind to N-methyl-D-aspartate (NMDA) cochleal receptors, which are glutamate-controlled ion channels providing for excitatory neurotransmission but also involved in excitotoxic neuronal injury. Other suggested ototoxic factors include the adverse effect of free oxygen radicals whose rate of production is increased in patients using these antibiotics. The above effect can be prevented by a once-daily dosing schedule and, possibly, N-acetylcysteine administration. N-acetylcysteine has strong antioxidant properties and serves as a precursor for the formation of glutathione. N-acetylcysteine is also a modulator of glutamatergic system and may act as a factor decreasing the toxicity of the above factors.

Table 2
Risk factors for tinnitus.

Risk factors for tinnitus	Compare incidence between general population and risk patients
Cardiovascular risk factors	21–24% vs. 28–32%
Exposure to noise	24% vs. up to 39%
Psychological state (depressions)	19% vs. 39%
Hearing disorders	20% vs. 30–37%

Cytostatic agents, in particular the platinum derivatives bleomycin and vincristine lower the perception threshold at higher frequencies possibly resulting in tinnitus development. Problems usually occur at doses greater than 60 mg/m^2 body surface area. High-dose furosemide has also been shown to be ototoxic.

While the administration of salicylates, unlike other ototoxic factors, does not result in unequivocal damage to ear structures, these drugs do affect and modulate their performance. Salicylates reversibly affect the electromotility of the outer hair cells and can reduce spontaneous otoacoustic emissions. In addition, salicylates can exert an effect on spontaneous activity of the inferior colliculus and the auditory cortex. The search for an association between salicylate administration and development of tinnitus continues to focus on the mechanism of modulation of outer hair cells. Cyclooxygenase inhibition results in tinnitus development via activation of cochlear NMDA receptors [11–14].

4. Association with other diseases

As a symptom accompanying a number of pathological entities, tinnitus has been associated with the following conditions and diseases. Broadly speaking, the term otologic disease refers to problems ranging from those at the level of the ear canal (earwax) through middle ear pathologies (mesotitides, otosclerosis) to hearing impairment related to the inner ear (Ménière's disease and other cochlear disorders). Neurological diseases presented, inter-alia, by tinnitus, include multiple sclerosis, whiplash injury, head injuries or vestibular schwannoma (acoustic neurinoma). Infective causes of tinnitus include meningitides, meningoencephalitides, Lyme disease, and syphilis. It is also worth mentioning the possibility of association with metabolic disorders, thyroid disorders, diabetes and zinc deficiency.

5. Current perspectives of tinnitus etiopathogenesis

Current perspectives of the etiopathogenesis of tinnitus are based both on theories related to the peripheral auditory system and those related to the central nervous system (CNS) responsible for processing acoustic stimuli.

Hair cell electromotility has been implicated in the generation of mechanical very low-pitch detectable sound waves; these acoustic signals are referred to as spontaneous otoacoustic emissions. Whilst normally inaudible, in theory, they may be a source of tinnitus.

Regarding the peripheral auditory system, the currently most widely accepted concept related to tinnitus development is "discordant theory" presuming dysfunction of the outer hair cells and normal function of the inner hair cells. High-intensity noise or other ototoxic factors lead primarily to external hair cell destruction. Normally, the outer hair cells are in contact with the tectorial membrane whereas the internal hair cell cilia are not. Upon outer hair cell destruction, the inner hair cells may come into contact with the membrane causing in their depolarization; subsequent outer hair cell dysfunction results in disinhibition of neurons in the dorsal cochlear nucleus. Hence, spontaneous activity is increased with direct stimulation from the inner hair cells [5].

The finding that normal hearing can be maintained in tinnitus patients can be explained by the fact that partial damage (up to 30%) to the outer hair cells is not associated with hearing

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