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Known frequent comorbidity with still unknown etiology

Tinnitus is an example of comorbidity of unknown pathophysiology that significantly reduces subjective perception of quality of life. The incidence of the disability has an increasing trend. It is not surprising information, when we consider what the risk factors for tinnitus are: hearing loss, sound exposure, stress, anxiety, depression, ototoxic drugs, hypertension, and aging. Almost all of these risk factors belong to the exemplary cases of civilization noxes that are currently not realistic to be eliminated. Therapy therefore focuses on influencing the structures of the hearing aid and the neural network. Besides pharmacotherapy, hyperbaric oxygen therapy has a significant beneficial effect. Their application can substantially eliminate symptoms of tinnitus.

Key words: Tinnitus. Hearing disorders, Hyperbaric Oxygen Therapy

Introduction

Human hearing loss and comorbidities like tinnitus pose serious problems for people's daily life, which in most severe cases may lead to social isolation, depression, and suicide[1]. Tinnitus is a widespread, but poorly understood symptom often seen in hearing loss patients. It is a condition associated with a continuous

[1] D. Gollnast, K. Tziridis, P. Krauss, A. Schilling, U. Hoppe, H. Schulze, *Analysis of Audiometric Differences of Patients with and without Tinnitus in a Large Clinical Database*, „Front Neurol" 2017, nr 8, p. 31.

auditory percept in the ears or head and can arise as a symptom of many different medical disorders[2].

Tinnitus is perceived as ringing, buzzing, beeping, or hissing and is characterized according to various clinical criteria. It can be subjective (perceived by the aected individual) or objective (heard by an observer), continuous or episodic, unilateral or bilateral, or pulsatile (synchronous or asynchronous). It can range from low- to high-intensity sound and can manifest any frequency. Tinnitus can be acute (<3 months), sub-acute (3-6 months), or chronic (>12 months) with a gradual or sudden onset or be associated with other triggers or comorbidities[3].

The prevalence of tinnitus in the general population is assumed to be at around 10-15%[4,5,6]. Men seem to be more often affected then women up to the age of 75, when prevalence is about equal for both genders[7]. About 1-2% of the tinnitus patients state their quality of life being significantly decreased by their phantom percept[8]. Assuming a conservative tinnitus prevalence of 10% (1% of severe tinnitus) for the 425 million adults living within the European Union, tinnitus affects more than 42 million citizens and is experienced as

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[2] W. Schlee, D.A. Hall, B. Canlon, R.F.F. Cima, E. de Kleine, et al., *Innovations in Doctoral Training and Research on Tinnitus: The European School on Interdisciplinary Tinnitus Research (ESIT) Perspective*, „Front Aging Neurosci" 2018, nr 12, p. 447.

[3] B. Vona, I. Nanda, W. Shehata-Dieler, T. Haaf, *Genetics of Tinnitus: Still in its Infancy*, „Front Neurosci" 2017, nr 8, p. 236.

[4] Tamże.

[5] D. Baguley, D. Mcferran, D. Hall, *Tinnitus*, „Lancet", 2013, nr 382, p. 1600–1607.

[6] F. Alhazmi, T. Kay, I. Mackenzie, G.J. Kemp, Sluming V., *An Investigation of the Impact of Tinnitus Perception on the Quality of Life*, „J Phonet Audiol" 2016, nr 2, p. 1000113.

[7] H.J. Hoffman, G.W. Reed, *Epidemiology of tinnitus*, [w:] *Theory and Management*, J.B. Snow (red.), wyd. BC Decker, Hamilton 2004, s. 24-27.

[8] S. Hebert, B. Canlon, D. Hasson, L.L. Magnusson Hanson, H. Westerlund, et al., *Tinnitus severity is reduced with reduction of depressive mood – a prospective population study in Sweden*, „PLoS One" 2012, nr 7, s. e37733.

a severe problem by more than 4 million. Moreover, incidence of new cases is expected to grow over the next few decades[9,10,11].

Risk factors of tinnitus include hearing loss, sound exposure, stress, anxiety, depression, ototoxic drugs, hypertension, and aging. While the association between individual risk factors and tinnitus is not straightforward, tinnitus seems to be correlated with advancing age and hearing loss. Interestingly, only about half of patients with tinnitus have recognized risk factors, which is a reason it has been hypothesized that predisposition to tinnitus is linked with genetic background[12]. Although hearing loss and the prevalence of tinnitus are increasing with age[13], it is still under debate if age-related hearing loss or age-related changes in physiological processes in the central and peripheral auditory system are the source of the increasing prevalence of tinnitus[14,15].

Multidisciplinary approach, etiology and forms of tinnitus

The condition is very common and of varying severity, but the fundamental mechanisms of tinnitus are still incompletely understood. Although not all individuals are unduly troubled,

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[9] W. Schlee, D.A. Hall, B. Canlon, R.F.F. Cima, E. de Kleine, et al., *Innovations in Doctoral Training*..., dz. cyt.

[10] A. McCormack, M. Edmondson-Jones, S. Somerset, D. Hall, *A systematic review of the reporting of tinnitus prevalence and severity*, „Hear Res" 2016, nr 337, s. 70-79.

[11] D.M. Nondahl, K.J. Cruickshanks, T.L. Wiley, B.E. Klein, R. Klein, et al., *The ten -year incidence of tinnitus among older adults*, „Int J Audiol" 2010, nr 49, s. 580-585.

[12] B. Vona, I. Nanda, W. Shehata-Dieler, T. Haaf, *Genetics of Tinnitus*..., dz. cyt..

[13] A.H. Lockwood, R.J. Salvi, R.F. Burkard, *Tinnitus*, „N Engl J Med" 2002, nr 347, s. 904-910.

[14] A.R. Møller, *Epidemiology of tinnitus in adults*, [w:] *Textbook of Tinnitus*, A.R. Møller, B. Langguth, D. DeRidder, T. Kleinjung (red.), wyd. Springer, New York 2011, s. 29-37.

[15] D. Gollnast, K. Tziridis, P. Krauss, A. Schilling, U. Hoppe, H. Schulze, *Analysis of Audiometric*..., dz. cyt.

many find the disorder life-changing. In cases with severe tinnitus, mental disorders, and symptoms such as anxiety, depression, insomnia, and concentration problems can impair quality of life often to a level that leads to sick leave and disability pension[16,17].

Tinnitus is essentially made up of two components, the phantom perception of a sound in the ears or head, and the degree of emotional reaction to that percept. Clinically, tinnitus can be very heterogeneous with respect to the perceived sound characteristics (e.g., tonal vs. broadband noise), its localization (in one or both ears, in the head, etc.), its time course (continuous, intermittent, fluctuating), its modifying factors (e.g., reduction by masking), and its comorbidities (hyperacusis, depression, insomnia)[18]. Tinnitus can co-occur with several medical-otological disorders such as presbycusis, though etiology is unknown for the majority of tinnitus patients. In rare cases tinnitus indicates a serious underlying pathology such as vascular troubles, vestibular schwannoma, or otosclerosis. In most cases however subjective tinnitus is a benign symptom. In many patients co-morbidities exist such as anxiety, depression, insomnia, and concentration problems, all of which severely impair quality of life. In 1-3% of cases tinnitus causes severe health problems, with a wide range of effects on daily life functioning. Evidence corroborates that the aversive psychological reactions, such as cognitive problems, negative emotions, and dysfunctional attentional processes are of main importance in leading to a severe tinnitus condition[19].

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[16] W. Schlee, D.A. Hall, B. Canlon, R.F.F. Cima, E. de Kleine, et al., *Innovations in Doctoral Training*..., dz. cyt.

[17] E. Friberg, C. Jansson, E. Mittendorfer-Rutz, U. Rosenhall, K. Alexanderson, *Sickness absence due to otoaudiological diagnoses and risk of disability pension: a nationwide swedish prospective cohort study*, „PLoS ONE" 2012, nr 7, s. e29966.

[18] D. de Ridder, A.B. Elgoyhen, R. Romo, B. Langguth, *Phantom percepts: tinnitus and pain as persisting aversive memory networks*, „Proc Natl Acad Sci USA" 2011, nr 108, s. 8075-8080.

[19] T.E. Fuller, H.F. Haider, D. Kikidis, A. Lapira, B. Mazurek, et al., *Different Teams, Same Conclusions? A Systematic Review of Existing Clinical Guidelines for the*

Tinnitus is not a priority for any individual discipline. A change in its scientific understanding and clinical management requires a shift toward multidisciplinary cooperation[20]. Any disorder of the brain, especially to the auditory system, can cause tinnitus. Hearing loss in particular increases the likelihood of experiencing chronic tinnitus. Among young adults, the most common cause of tinnitus is noise exposure. Among the elderly, age-related hearing loss (presbycusis) is the most common cause of tinnitus, although the impact of early cochlear insults could sum with aging to accentuate tinnitus. Other tinnitus etiologies include cardiovascular and cerebrovascular disease, medications, head/neck trauma and injury, and hyper- and hypothyroidism. Often, the etiology of tinnitus is considered idiopathic, as 40% of patients report "no known events" associated with their tinnitus onset. Importantly, if a person with tinnitus has hearing thresholds "within normal limits" (i.e., within 25 dB hearing loss), there may still be evidence of auditory damage, such as cochlear dead had thresholds 25 dB or better in both ears up to 8 kHz but varying degrees of impairment at higher regions[21, 22, 23] or elevation of hearing thresholds in the tinnitus frequency range[24],[25]. Importantly, many individuals who

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Assessment and Treatment of Tinnitus in Adults, „Front Psychol" 2017, nr 8, s. 206.

[20] W. Schlee, D.A. Hall, B. Canlon, R.F.F. Cima, E. de Kleine, et al., *Innovations in Doctoral Training*..., dz.cyt.

[21] J.A. Henry, L.E. Roberts, D.M. Caspary, S.M. Theodoroff, R.J. Salvi, *Underlying mechanisms of tinnitus: review and clinical implications*, „J Am Acad Audiol" 2014, nr 25, s. 5-22.

[22] N. Weisz, T. Hartmann, K. Dohrmann, W. Schlee, A. Noreña, *High-frequency tinnitus without hearing loss does not mean absence of deafferentation*, „Hear Res" 2006, nr 222, s. 108-114.

[23] L.E. Roberts, *Neural synchrony and neural plasticity in tinnitus*, [w:] T*extbook of Tinnitus*, A.R. Møller, B. Langguth, D. DeRidder, T. Kleinjung (red.), wyd. Springer, New York 2011, s. 103-112.

[24] Henry J.A., Roberts L.E., Caspary D.M., Theodoroff S.M., Salvi R.J., *Underlying mechanisms*..., dz. cyt.

[25] L.E. Roberts, G. Moffat, M. Baumann, L.M. Ward, D.J. Bosnyak, *Residual in-*

claim to have tinnitus with "normal hearing" in the conventional audiometric range (125 to 8000 Hz) often have elevated thresholds at frequencies above 8000 Hz. One quarter (8/32) of the tinnitus cases studied by Roberts et al.[26] had thresholds 25 dB or better in both ears up to 8 kHz but varying degrees of impairment at higher frequencies[27]. Furthermore, population studies revealed that individuals with tinnitus on average suffer from stronger hearing loss in high frequencies; patients with low-pitched tinnitus (below 1,500 Hz) show stronger low frequency hearing loss than patients with middle- or high-pitched tinnitus[28].

Even if current etiologic models assume a complex interplay of various factors, several lines of evidence indicate that hearing loss is the most relevant etiologic factor for tinnitus development[29,30]. First, epidemiological studies have identified hearing loss as a major risk factor for tinnitus[31]. Second, induction of hearing loss in animals induces reliably increased neuronal activity and synchronicity[32, 33] as well as behavioral evidence of tinnitus[34]. Third,

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hibition functions overlap tinnitus spectra and the region of auditory threshold shift, „J Assoc Res Otolaryngol", 2008, nr 9, s. 417-435.

[26] L.E. Roberts, G. Moffat, D.J. Bosnyak, *Residual inhibition functions in relation to tinnitus spectra and auditory threshold shift*, „Acta Otolaryngol Suppl", 2006, nr 556, s. 27-33.

[27] J.A. Henry, L.E. Roberts, D.M. Caspary, S.M. Theodoroff, R.J. Salvi, *Underlying*..., dz. cyt.

[28] D. Gollnast, K. Tziridis, P. Krauss, A. Schilling, U. Hoppe, H. Schulze, *Analysis of Audiometric*..., dz. cyt.

[29] B. Langguth, P.M. Kreuzer, T. Kleinjung, D. de Ridder, *Tinnitus: causes and clinical management*, „Lancet Neurol" 2013, nr 12, s. 920-930.

[30] S.E. Shore, L.E. Roberts, B. Langguth, *Maladaptive plasticity in tinnitus – triggers, mechanisms and treatment*, „Nat Rev Neurol" 2016, nr 12, s. 150-160.

[31] H.J. Hoffman, G.W. Reed,, *Epidemiology of tinnitus*..., op. cit.

[32] S.E. Shore, L.E. Roberts, B. Langguth, *Maladaptive plasticity*..., op. cit.

[33] J.J. Eggermont, L.E. Roberts, *The neuroscience of tinnitus*, „Trends Neurosci", 2004, nr 27, s. 676-682.

[34] J.G. Turner, *Behavioral measures of tinnitus in laboratory animals*, „Prog Brain Res" 2007, nr 166, s. 147-156.

the tinnitus spectrum of most tinnitus patients is clearly related to their pattern of hearing loss[35, 36]. If, for example, somebody experiences tinnitus at 4 kHz at the left ear, typically a hearing loss at 4 kHz on the left ear can be detected in the audiogram[37].

Management strategies

During the last decades, efforts have been made to better understand tinnitus pathophysiology and provide specialized treatments to patients. A large number of management strategies including various assessment and treatment procedures exist and have evolved but lack empirical support. For example, there is no evidenced treatment or licensed pharmacological therapy to eliminate the tinnitus percept. The Cochrane Library lists 10 completed systematic reviews on different tinnitus treatments, all of which reported small numbers of studies of variable quality. These facts combined makes it difficult for healthcare professionals to decide what is best for which tinnitus patient. This is evidenced by the discrepancy between scientific and clinical perspectives on the management of tinnitus and the actual day-to-day practice in European healthcare settings; tinnitus patient care is fragmented and ad hoc. To date there has been no overview of the number of existing clinical practice guidelines for tinnitus, the details included, their comparability, or their purpose. Clinical practice guidelines are defined as systematically developed statements to assist practitioner and patient decisions about appropriate health

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[35] A. Norena, C. Micheyl, S. Chery-Croze, L. Collet, *Psychoacoustic characterization of the tinnitus spectrum: implications for the underlying mechanisms of tinnitus*, „Audiol Neurootol" 2002, nr 7, s. 358-369.

[36] M. Schecklmann, V. Vielsmeier, T. Steffens, M. Landgrebe, B. Langguth, et al., *Relationship between audiometric slope and tinnitus pitch in tinnitus patients: insights into the mechanisms of tinnitus generation*, „PLoS One" 2012, nr 7, s. e34878.

[37] B. Langguth, M. Landgrebe, W. Schlee, M. Schecklmann, V. Vielsmeier, et al., *Different Patterns of Hearing Loss among Tinnitus Patients: A Latent Class Analysis of a Large Sample*, „Front Neurol" 2017, nr 20, s. 46.

care for specific clinical circumstances. They have the benefit of simplifying and standardizing assessment and treatment options for clinicians and patients[38].

Research

The heterogenity of tinnitus is a major challenge for tinnitus research. Even if a complex interaction of many factors is involved in the etiology of tinnitus, hearing loss has been identified as the most relevant etiologic factor[39].

In a retrospective study, Gollnast et al.[40] compared 37,661 patients with sensorineural or conductive hearing loss with or without tinnitus. Tinnitus characteristics were determined in terms of signal type and signal level measured in decibel hearing level by comparison of the internal tinnitus with external sound from the audiometer. Three types of signals were possible: broadband noise, narrow band noise (1/3 octave bandwidth), and pure tones between 0.25 and 8 kHz. Tinnitus loudness was determined by increasing the signal level above hearing threshold slowly in steps of 1 dB and asked the subjects for a comparison with their tinnitus percept. The results of the pure tone audiometry comparisons showed significant differences in tinnitus patients compared to non-tinnitus patients. In young patients, they generally found lower hearing thresholds in tinnitus compared to non-tinnitus patients. In adult patients, differences were more heterogeneous: hearing thresholds in tinnitus patients were lower in low frequency ranges, while they were higher at high frequencies. Furthermore, lower thresholds were more often found

[38] T.E. Fuller, H.F. Haider, D. Kikidis, A. Lapira, B. Mazurek, et al., *Different Teams*..., dz. cyt.

[39] B. Langguth, M. Landgrebe, W. Schlee, M. Schecklmann, V. Vielsmeier, et al., *Different Patterns*..., dz. cyt.

[40] D. Gollnast, K. Tziridis, P. Krauss, A. Schilling, U. Hoppe, H. Schulze, *Analysis of Audiometric*..., dz. cyt.

in conductive hearing loss patients and could rarely be detected in sensorineural hearing loss patients. In speech audiometry, only conductive hearing loss patients with high-pitched tinnitus showed lower thresholds compared to non-tinnitus patients' thresholds. The results of this study may point to a biologically plausible functional benefit on hearing thresholds in hearing loss tinnitus patients.

Animal models are important because they allow for the comparison of neural changes between animals that express behavioral evidence of tinnitus and animals that do not, when their preceding experience has been the same. Two neural correlates of tinnitus identified by this approach are: (1) an increase in the spontaneous activity of neurons in central auditory pathways, and (2) an increase in cross-correlated or synchronous activity among the aected neurons. Homeostatic plasticity and spike-timing-dependent plasticity, respectively, are thought to underlie these two neural correlates, although as will be noted later, these mechanisms may interact to produce tinnitus behavior. These forms of plasticity are also believed to underlie increased sound -driven neural and behavioral responses that have been observed in noise-exposed animals, suggesting hyperacusis, which is reported subjectively by about 40% of tinnitus patients[41].

The proposed mechanisms of action

Most broadly, there are two types of tinnitus - objective tinnitus and subjective tinnitus. Objective tinnitus refers to the perception of acoustic vibratory activity that is generated mechanically within the body. Objective tinnitus can have its origin in vascular, muscular, skeletal, or respiratory structures. These "body sounds" (somatosounds) have an internal acoustic source. The most common somatosound is pulsatile tinnitus that fluctuates in

[41] L.E. Roberts, *Neural plasticity and its initiating conditions in tinnitus*, „HNO", 2018, nr 66, s. 172-178.

synchrony with the heartbeat. Somatosounds can also be nonpulsatile, such as the spontaneous contraction of middle ear muscles or the Eustachian tube. Information about diagnosing and identifying objective tinnitus is available elsewhere. By far the majority of patients have subjective tinnitus that is not associated with an identifiable sound source. Tinnitus of this type is assumed to be caused by or associated with damage to the auditory system, that is, "sensorineural" tinnitus or tinnitus with a neurophysiologic origin. The histopathologies or cellular changes that presumably give rise to subjective tinnitus can exist anywhere between the cochlea and auditory cortex, although the majority of cases are triggered by or associated with cochlear damage. Tinnitus is sometimes only heard when in quiet environments; however, in some cases, tinnitus is perceived constantly and can become very bothersome, interfering with concentration, sleep, and daily activities. Some individuals experience tinnitus that can even be heard in fairly intense background noise[42].

It is generally accepted that people with subjective tinnitus may experience two kinds of symptoms: one is the hearing of a sound that does not come from the environment and the other experience is a form of distress or suffering. These two kinds of symptoms are not directly related and an individual who experiences a weak tinnitus sound may nonetheless experience severe suffering. Others may experience a strong sound but suffer little or not at all. It seems likely that these two expressions of tinnitus have different pathologies and may engage different circuits in the brain. Recent studies indicate that changes in connections in many parts of the brain play an important role in causing the symptoms of tinnitus mentioned above. The networks formed by these connections consist of cortical and subcortical areas that serve auditory as well as other functions. Understanding the

[42] J.A. Henry, L.E. Roberts, D.M. Caspary, S.M. Theodoroff, R.J. Salvi, *Underlying mechanisms...*, dz. cyt.

abnormalities in these networks and their dynamic interactions (connectivity) is of utmost importance for understanding different people's experience of tinnitus[43].

Clinical heterogeneity of tinnitus is paralleled by heterogeneity in tinnitus pathophysiology. Recent pathophysiological models assume that tinnitus emerges as a clinical symptom as result of abnormal activation of different overlapping and interacting brain networks[44]. Abnormally activated networks in tinnitus patients include the auditory, attention, salience, and distress networks. The activation pattern varies from patient to patient and reflects the individuals' symptoms. As an example, distressed and not distressed tinnitus patients differ in their activation of the cortical stress-related network[45].

In any case, the question remains why in some patients with hearing loss subjective tinnitus is developing at all. In a recent study, Krauss et al.[46] put forward a model for the physiological improvement of hearing thresholds, which as side effect also explains the development of tinnitus. The model was based on the idea that the auditory system tries to compensate for a hearing loss by means of stochastic resonance at the receptor level. Stochastic resonance refers to the phenomenon that weak signals that are sub-threshold for a given sensor still can be detected and transmitted by that sensor if noise (internal or external) is added to the sensor input, both in technical and physiological

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[43] M. Meyer, B. Langguth, T. Kleinjung, A.R. Møller, *Plasticity of neural systems in tinnitus*, „Neural Plast" 2014, nr 2014, s. 968029.

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[46] P. Krauss, K. Tziridis, C. Metzner, A. Schilling, U. Hoppe, et al., *Stochastic resonance controlled upregulation of internal noise after hearing loss as a putative cause of tinnitus-related neuronal hyperactivity*, „Front Neurosci" 2016, nr 10, s. 597.

systems[47,48, 49, 50]. Authors further assumed that hearing loss leads to an unequal distribution of spectral input into the auditory system, with a reduced input from the affected spectral ranges. Their model proposes that the impaired hearing thresholds within those frequency channels may be improved again (at least to a certain degree) by means of stochastic resonance. Obviously, for stochastic resonance to work, internal noise has to be generated within the auditory system and fed back to the receptor level[51]. Authors proposed that this internal noise is reflected in neuronal hyperactivity. If the hearing loss is permanent, the neuronal hyperactivity that enables stochastic resonance to compensate for increased thresholds may subsequently cause neuronal plasticity along the auditory pathway and finally may lead to the development of a phantom percept, i.e., subjective tinnitus. In that sense, the model views tinnitus as a side effect of a mechanism within the auditory system that seeks to optimize signal transmission at the receptor level. If this model would be true, we would expect that, in tinnitus patients, initial hearing loss should be compensated to a certain degree, resulting in overall better hearing thresholds in tinnitus patients compared to non-tinnitus patients with comparable damage in the auditory system[52].

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[50] L. Gammaitoni, P. Hänggi, P. Jung, F. Marchesoni, *Stochastic resonance*, „Rev Mod Phys", 1998, nr 70, s. 223.

[51] P. Krauss, K. Tziridis, C. Metzner, A. Schilling, U. Hoppe, et al., *Stochastic resonance*..., dz. cyt.

[52] D. Gollnast, K. Tziridis, P. Krauss, A. Schilling, U. Hoppe, H. Schulze, *Analysis of Audiometric*..., dz. cyt.

The neurophysiological model proposed by Jastreboff suggests that tinnitus would be the result of the interaction of auditory and nonauditory pathways. The limbic and the autonomic nervous systems would act as determinants of the condition called tinnitus[53, 54, 55]. Thus, the association of tinnitus with unpleasant or dangerous situations, for example, would contribute to the perception of it and to increased annoyance. The presence of this symptom causes negative repercussions on the patient's quality of life such as anxiety, depression, emotional instability, sleep disorders and behavioral changes, which in turn reflect on the individual's performance of daily and professional activities[56, 57, 58, 59].

Hearing loss and tinnitus

Although specific events associated with the onset of tinnitus may vary, the great majority of patients with tinnitus have some degree of hearing loss indexed by the audiogram. This suggests that tinnitus, associated with different specific etiologies, impacts a final common path, irrespective of the degree or pattern

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[53] A.R. Teixeira, A.H. Lessa, L.P. Rosito, C.Z. Neves, C.D. Bueno, et al. *Influence of factors and personal habits on the tinnitus perception*, „Rev CEFAC", 2016, nr 18, s. 1310-1315.

[54] P.J. Jastreboff, *Phantom auditory perception (tinnitus): mechanisms of generation and perception*, „Neurosci Res" 1990, nr 8, s. 221-254.

[55] P.J. Jastreboff, J.W.P. Hazell, *A neurophysiological approach to tinnitus: clinical implications*, „Br J Audiol" 1993, nr 27, s. 7-17.

[56] A.R. Teixeira, A.H. Lessa, L.P. Rosito, C.Z. Neves, C.D. Bueno, et al. *Influence of factors*..., dz. cyt.

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[58] D.M.B.M. Ferreira, A.N.R. Júnior, E.P. Mendes, *Caracterização do zumbido em idosos e de possíveis transtornos relacionados*, „Braz J Otorhinolaryngol, 2009, nr 75, s. 249-255.

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of impairment in peripheral or central auditory pathways. When individuals with tinnitus are asked to rate sound frequencies between 5 and 12 kHz for similarity or "likeness" to their tinnitus, the resulting likeness ratings scores mirror the pattern of hearing loss. This finding suggests that tinnitus is generated by aberrant neural activity taking place in frequency regions deafferented by hearing loss. However, paradoxically some young patients with normal hearing thresholds experience tinnitus while some older individuals with significant hearing loss do not experience tinnitus. Nonetheless, when these two groups are compared with their appropriate controls (i.e., young individuals without tinnitus and older patients with tinnitus), in both cases hearing thresholds above 2kHz were ~10dB greater in the groups with tinnitus, suggesting a relationship to audiometric function. Moreover, normal audiometric function per se is unlikely to detect inner hair cell loss or auditory nerve damage[60].

It has often been observed that tinnitus percepts and the circumstances associated with their onset are to some degree variable between individuals. While it is important to acknowledge this variability and memorable unique cases that may be related to medical disease, it is nonetheless true that most chronic tinnitus sufferers describe their tinnitus asacontinuous tonal, ringing, or hissing sound, and thath hearing impairment measured by the clinical audiogram up to 8 000Hz is present in up to 90% of cases. Nonetheless, tinnitus without audiometric hearing loss needs to be explained, as does the absence of tinnitus in many individuals where such hearing loss is present. Recent research findings suggest that some if not many of these cases could reflect cochlear changes that are not detected by threshold measurements. Deafferentation resulting from cochlear pathology is known to activate forms of neural plasticity in auditory pathways that appear

[60] J.A. Henry, L.E. Roberts, D.M. Caspary, S.M. Theodoroff, R.J. Salvi, *Underlying mechanisms*..., dz.cyt.

to underlie tinnitus percepts and associated conditions including hyperacusis and impaired auditory temporal processing[61].

Henry et al.[62] summarized basic understanding of the neurophysiological changes in the auditory system likely to be responsible for tinnitus. Based on their review nearly all forms of cochlear damage decrease the neural output from the cochlea that is sent to the central auditory system. This decreased output is readily detected as a reduction in the amplitude of the acoustic nerve compound action potential. Cochlear destruction and noise damage initially cause a reduction in spontaneous discharge rates in the cochlear nucleus. However, beginning approximately seven days following cochlear damage, neurons in the dorsal part of the cochlear nucleus respond by increasing (up-regulating) both spontaneous and sound-evoked neural activity. This increase in activity occurs over much of cochlear nucleus, but it tends to be centered near regions tuned to the cochlear damage. Moreover, increases in spontaneous rate in the dorsal part of the cochlear nucleus were correlated with behavioral evidence of tinnitus. This up-regulation in spontaneous activity is thought to be caused by an alteration in the normal balance between excitatory and inhibitory nerve transmission brought about by loss of inhibition (disinhibition), which leads to an increased firing rate. These changes occur because of plastic central nervous system changes based on experience and/or loss of central auditory system input due to damage. In essence, pathology in the cochlea and reduced auditory nerve activity can result in increased and/or bursting neural activity in response to plastic compensatory changes within central auditory structures that attempt to restore homeostasis[63].

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[61] L.E. Roberts, *Neural plasticity*..., dz. cyt.

[62] J.A. Henry, L.E. Roberts, D.M. Caspary, S.M. Theodoroff, R.J. Salvi, *Underlying mechanisms*..., dz. cyt.

[63] Tamże.

Selected treatment modalities

Tinnitus is not likely generated by a single neural source but is rather a network phenomenon involving several brain structures, neural transmitters, and receptor types in a cascade of changes initiated in most cases by hearing impairment. As such, it is unlikely that a single curative treatment can be found, short of reversing or compensating for hearing loss. Many patients who receive a hearing aid or cochlear implant for hearing loss report that their tinnitus has also improved. One implication for drug treatments is that drugs that have multiple effects on synaptic processes (therapeutic "shotguns") may prove to be more effective at disrupting network behavior and reducing tinnitus than pharmaceuticals that have more specific action profiles[64].

The most commonly used treatment is vasoactive treatment in order to improve blood flow to the microvessels, or drugs specifically for the inner ear and the equilibrium apparatus. If cervical spine changes are identified, rehabilitation and treatment should also be targeted in this direction. Alternative methods used in treatment of tinnitus are for example laser therapy for tinnitus treatment, as well as hyperbaric oxygen therapy.

Hyperbaric oxygen therapy

Based on above-mentioned mechanisms the modality for the treatment of sudden sensorineural hearing loss and tinnitus is hyperbaric oxygen therapy. Hyperbaric oxygen therapy is a therapeutic approach where the patient is exposed to 100% oxygen at pressures higher than ambient (1 ATA). This leads to an increased blood oxygen level, which than can penetrate to ischemic areas more deeply than under normobaric conditions[65]. Normal-

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[64] J.A. Henry, L.E. Roberts, D.M. Caspary, S.M. Theodoroff, R.J. Salvi, *Underlying mechanisms*..., dz. cyt.

[65] A.B. Parabucki, I.D. Bozići, M. Bjelobaba, I.C. Lavrnja, P.D. Brkić, et al. *Hyperbaric oxygenation alters temporal expression pattern of superoxide dismutase 2 after*

ly 97% of the oxygen transported from the lungs to the tissues is carried in chemical combination with hemoglobin or red blood cells, and the remaining 3%in a dissolved state in plasma. Under hyperbaric conditions, it is possible to dissolve sufficient oxygen, i.e., 6 vol.% in plasma, to meet the usual requirements of the body. In this case oxyhemoglobin will pass unchanged from the arterial to the venous side because the oxygen physically dissolved in solution will be utilized more readily than that bound to hemoglobin[66, 67].

Hyperbaric oxygen therapy is a standard therapy for decompression sickness, gas embolism and CO poisoning. Hyperbaric oxygen therapy is also effective for gas gangrene, anaerobic infection, diabetic foot, Burger's disease and other oxygen-deficient conditions. In addition, hyperbaric oxygen therapy has been proved effective in the healing of chronic wound, such as radiation -induced soft tissue necrosis. Meanwhile, many studies reported the therapeutic or preventive effect of hyperbaric oxygen therapy in various kinds of inflammatory or immune-mediated diseases, such as systemic lupus erythematosus, atherosclerosis, collagen -induced arthritis, Crohn's disease, ulcerative colitis and atopic dermatitis, although these diseases are not included in the current indication of hyperbaric oxygen therapy[68].

Hyperbaric oxygen therapy has been used successfully in the management of sudden sensorineural hearing loss and tinnitus based on the concept that hyperbaric oxygen therapy increases the pO_2 in the inner ear; improves hemorrheology and

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cortical stab injury in rats, „Croat Med J, 2012, nr 53, s. 586-597.

[66] K.K. Jain, *Textbook of Hyperbaric Medicine*, wyd. Hogrefe and Huber Publishers, Göttingen 2009.

[67] E. Králová, Z. Krajčovičová, V. Meluš, R. Zigo, *Hyperbaric oxygen therapy in the treatment of tinnitus and hearing disorders in musicians*, „University review" 2016, nr 10, s. 41-44.

[68] E. Králová, Z. Krajčovičová, V. Meluš, R. Zigo, *Hyperbaric oxygen therapy...*, dz. cyt.

contributes to improved microcirculation, lowers the hematocrit and whole blood viscosity, and also improves the erythrocyte elasticity[69, 70]. In addition, research has shown a potential advantage of hyperbaric oxygen therapy carried out in hearing loss to be increased oxygen pressure (pO_2) in the blood and thus, through diffusion, raised pO_2 in the inner-ear fluids, which supply the sensory and neural elements in the cochlea. Hyperbaric oxygen therapy induces cell metabolism in the inner ear, even if the blood supply is insufficient[71, 72, 73, 74]. Based on these finding can be assumed correlation reducing the subjective perception of the intensity of tinnitus in patients with sudden sensorineural hearing loss treated with hyperbaric oxygen therapy.

Effect of hyperbaric oxygen therapy on tinnitus

Porubsky et al.[75] analyzed the effectiveness of hyperbaric oxy-

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[69] H. Suzuki, H. Koizumi, J. Ohkubo, N. Hohchi, S. Ikezaki, et al., *Hearing outcome does not depend on the interval of intratympanic steroid administration in idiopathic sudden sensorineural hearing loss*, „Eur Arch Otorhinolaryngol", 2016, nr 273, s. 3101-3107.

[70] K.K. Jain, *Textbook*..., dz. cyt.

[71] E. Sevil, S. Bercin, T. Muderris, F. Gul, M. Kiris, *Comparison of two different steroid treatments with hyperbaric oxygen for idiopathic sudden sensorineural hearing loss*, „Eur Arch Otorhinolaryngol" 2016, nr 273, s. 2419-2426.

[72] K. Lamm, C. Lamm, W. Arnold, *Effect of isobaric oxygen versus hyperbaric oxygen on the normal and noise-damaged hypoxic and ischemic guinea pig inner ear*, „Adv Otorhinolaryngol" 1998, nr 54, s. 59-85.

[73] B. Fattori, S. Berrettini, A. Casani, A. Nacci, A. de Vito, et al., *Sudden hypoacusis treated with hyperbaric oxygen therapy: a controlled study*, „Ear Nose Throat J" 2001, nr 80, s. 655-660.

[74] R. Zigo, Z. Krajčovičová, V. Meluš, E. Ralausová, D. Schlenkerová, et al., *Overenie vplyvu hyperbarickej oxygenoterapie v liečbe náhlej sezorineurálnej straty sluchu*, „Otorinolaryngologie a foniatrie" 2017, nr 66, s. 28-34.

[75] C. Porubsky, P. Stiegler, V. Matzi, C. Lipp, A. Kontaxis, et al., *Hyperbaric oxygen in tinnitus: influence of psychological factors on treatment results?* „ORL J Otorhinolaryngol Relat Spec" 2007, nr 69, s. 107-112.

gen therapy in the context of accompanying factors. They randomized 360 patients suffering from tinnitus into 2 hyperbaric oxygen treatment protocols (group A: 2.2 bar for 60 min bottom time and group B: 2.5 bar for 60 min bottom time once a day for 15 days). All patients were asked to fill in a questionnaire (social and medical history, tinnitus characteristics, pre-hyperbaric oxygen therapy duration of tinnitus, prior therapy, pretreatment expectation, accompanying symptoms). A subjective assessment of the therapeutic effect was obtained. Twelve patients (3.3%) experienced complete remission of tinnitus, in 122 (33.9) the intensity lessened, and 44 (12.2%) had a subjectively agreeable change of noise characteristics. No change was found in 157 cases (43.6%) and 25 (6.9%) experienced deterioration. There was no statistically significant difference between groups A and B ($p > 0.05$). Out of 68 patients with a positive expectation of hyperbaric oxygen therapy effects, 60.3% stated that the tinnitus had improved whereas only 47.2 and 19%, respectively, out of patients who underwent therapy with an indifferent ($n = 271$) or negative expectation ($n = 21$) reported an improvement. The influence of subjective expectation on the outcome was statistically significant ($p < 0.05$). Based on their results, the therapeutic effects of hyperbaric oxygen therapy on subjective tinnitus may be substantially influenced by psychological mechanisms.

Furthermore, Lamm et al.[76] evaluated more than 50 studies with a total of 4 109 patients suffering from idiopathic sudden hearing loss, acoustic trauma or noise-induced hearing loss and/or tinnitus, hyperbaric oxygen therapy was administered as a secondary therapy, i.e. following unsuccessful conventional therapy. If the onset of affliction was more than 2 weeks but no longer than 6 weeks, one half of the cases showed a marked hearing gain (in at least 3

[76] K. Lamm, H. Lamm, W. Arnold, *Effect of hyperbaric oxygen therapy in comparison to conventional or placebo therapy or no treatment in idiopathic sudden hearing loss, acoustic trauma, noise-induced hearing loss and tinnitus. A literature survey*, „Adv Otorhinolaryngol" 1998, nr 54, s. 86-99.

frequencies of more than 20 dB), one-third showed a moderate improvement (10-20 dB) and 13% showed no hearing improvement at all. 4% no longer experienced tinnitus, 81.3% observed an intensity decrease and 1.2% an intensity increase of their tinnitus condition. 13.5% remained unchanged. If hyperbaric oxygen therapy was administered at a later stage, but still within 3 months following onset of affliction, 13% showed a definite improvement in hearing, 25% a moderate improvement and 62% no improvement at all. 7% no longer suffered from tinnitus, 44% reported an intensity decrease, a similar percentage noticed no change and 5% a temporary deterioration of their tinnitus condition. If the onset of affliction was longer than 3 months up to several years, no hearing improvement can be expected in the majority of patients; however, one third of the cases reported an intensity decrease of tinnitus, 60-62% reported no change and 4-7% noticed a temporary intensity increase. In conclusion, it may be deduced that hyperbaric oxygen therapy is recommended and warranted in those patients with idiopathic sudden deafness, acoustic trauma or noise-induced hearing loss within 3 months after onset of disorder.

Conclusion

Although much progress has been made in understanding the pathophysiology, tinnitus remains a scientific and clinical enigma. Tinnitus is a symptom rather than a distinct disease, and its multivariate manifestations can be subtyped according to various dimensions such as its etiology, time since onset, perceptual characteristics (i.e., pitch, loudness, location, and temporal dynamics), perceived emotional distress, and comorbidities[77].

The key to development of new treatments is a better understanding of the pathology of the disorder. Recent years have seen important progress in the understanding of pertinent aspects of

[77] W. Schlee, D.A. Hall, B. Canlon, R.F.F. Cima, E. de Kleine, et al., *Innovations*..., dz. cyt.

the neuropsychology and neurobiology of subjective idiopathic tinnitus but many questions remain unanswered in that rapidly burgeoning field of neuroscience. The anatomical location of the pathology that causes the phantom sound is not completely known nor is it known what changes in the brain are directly or indirectly associated with distress or suffering. Recent advances in neuroscience and clinical medicine have introduced new models and frameworks that help elucidate the mechanisms underlying the pathology of subjective tinnitus[78].

In summary, it is now clear that tinnitus is a pathology involving synaptic plasticity. The origin of tinnitus can occur either at the level of the synapses between inner hair cells and the auditory nerve, within the auditory nerve itself, or from central auditory system structures. Long-term maintenance of tinnitus is likely a function of a complex network of structures in the central auditory system and nonauditory systems. While much has been learned, much remains to be learned. The ultimate goal of tinnitus mechanisms research is to develop a cure. This goal is particularly challenging because different forms of tinnitus may relate to specific pathophysiologies. We know that anything that can cause hearing loss can also cause tinnitus, including noise exposure, ototoxicity, traumatic brain injury, and so on. No single origin of tinnitus has yet been identified; thus, it is unknown if each cause of tinnitus results in different forms of tinnitus generation, each of which may require a different therapeutic cure. However, it is also known that, in all cases of tinnitus, the tinnitus neural signal is transmitted through the auditory pathways with conscious perception involving complex processing between sub-cortical structures, the auditory cortex, and higher pathways. There is thus hope that a single cure can be found that would target a common mechanism[79].

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[78] M. Meyer, B. Langguth, T. Kleinjung, A.R. Møller, *Plasticity of neural*..., dz. cyt.

[79] J.A. Henry, L.E. Roberts, D.M. Caspary, S.M. Theodoroff, R.J. Salvi, *Underlying mechanisms*..., dz. cyt.

Acknowledgements

This publication was created in the frame of the project "Completion of the technical infrastructure for the development of science and research at Alexander Dubček University of Trenčín through Hyperbaric Oxygen Therapy", ITMS code 26210120019, based on the Operational Programme Research and Development and funded from the European Social Fund.

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Szum w uszach: znane schorzenie współwystępujące z nadal nieznaną etiologią

Szum w uszach jest przykładem schorzeń współwystępujących o nieznanej patofizjologii, które w sposób znaczący zmniejszają subiektywne postrzeganie jakości życia. Częstość występowania tego rodzaju niepełnosprawności rośnie. Nie jest to informacja zaskakująca, gdy bierze się pod uwagę czynniki ryzyka związane z szumem w uszach: utrata słuchu, ekspozycja na dźwięk, stres, lęk, depresja, leki ototoksyczne, nadciśnienie i starzenie się. Niemal wszystkie wspomniane czynniki ryzyka należą do przykładowych przyczyn chorób cywilizacyjnych, których obecnie nie da

się wyeliminować. Terapia jest zatem ukierunkowana na wpływ na struktury aparatu słuchowego oraz sieci neuronowej. Oprócz farmakoterapii znaczącym efektem odznacza się także hiperbaryczna terapia tlenowa. Ich zastosowanie może w znacznym stopniu wyeliminować objawy szumu w uszach.

Słowa kluczowe: szum w uszach, zaburzenia słuchu, hiperbaryczna terapia tlenowa