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Psychiatric approach to tinnitus

Psihijatrijski pristup tinitusu

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Summary

Tinnitus is a symptom with a significant incidence in the general population, usually of unclear etiology, that can cause serious difficulties in people's daily functioning, significantly impair the quality of life, and have a negative impact on mental health.

The paper aims to present a brief overview of current knowledge about this frequent and unpleasant phenomenon, including epidemiology, etiology, clinical presentation, diagnosis, and treatment.

The paper highlights contemporary theories of tinnitus that link damage to the peripheral organ of hearing and the consequent neuronal changes involved in the subjective experience, which are the target sites for treating psychological disorders associated with tinnitus. The psychiatric approach to tinnitus is aimed not so much at reducing the sound intensity as at reducing the negative experience of this phenomenon and preventing the development or worsening of existing psychological disorders.

The results of previous researches indicate numerous therapeutic options for treating tinnitus, including drugs, cognitive-behavioral therapy, and neuromodulation techniques with promising results.

Key words: neuromodulation, neuroplasticity, psychiatry, tinnitus

Sažetak

Tinitus je simptom sa značajnom pojavnošću u općoj populaciji, najčešće nejasne etiologije, koji može izazvati ozbiljne poteškoće u svakodnevnom funkcioniranju, značajno narušiti kvalitetu života i dovesti do negativnog utjecaja na psihičko zdravlje.

Cilj rada je prikazati kratki pregled dosadašnjih spoznaja o ovome, često neugodnom fenomenu, uključujući epidemiologiju, etiologiju, kliničku sliku, dijagnostiku i liječenje.

U radu su istaknute suvremene teorije tinitusa koje povezuju oštećenje perifernog organa sluha i posljedične neuronske promjene uključene u subjektivni doživljaj, koje su ciljno mjesto liječenja psihičkih smetnji udruženih s tinitusom. Psihijatrijski pristup tinitusu usmjeren je, ne toliko na smanjenje zvučnog intenziteta, koliko na smanjenje negativnog doživljaja ovoga fenomena i sprječavanje razvoja ili pogoršanja postojećih psihičkih poremećaja.

Rezultati dosadašnjih istraživanja ukazuju na brojne terapijske mogućnosti liječenja tinitusa, uključujući lijekove, kognitivno-bihevioralnu terapiju, te neuromodulacijske tehnike s obećavajućim rezultatima.

Ključne riječi: neuromodulacija, neuroplastičnost, psihijatrija, tinitus

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Introduction

The appearance of noise lasting longer than five minutes in one or both ears in the absence of external sound sources is called tinnitus. In terms of duration, it can be permanent and constant, or recurrent, with episodes occurring at least once a month. According to the American Academy of Otolaryngology-Head and Neck Surgery, tinnitus can be divided into primary and secondary.[1] The primary is of an unknown cause, and the secondary is always associated with an ear disease or some other systemic illness. Both external, middle, and inner ear diseases can lead to tinnitus. Middle ear inflammation, otosclerosis, and Meniere's disease are some of the most common ear disorders that can provoke tinnitus. Systemic diseases, such as atherosclerosis, cervical spine spondylosis, and anemia, may also be associated with the development of tinnitus. Secondary tinnitus is always characterized by a clear organic cause of the disorder.[2]

Tinnitus can be divided into subjective and objective types based on causative factors. Subjective tinnitus is a sound that cannot be explained by the existence of a real source of sound and is mainly associated with hearing loss. Objective tinnitus is much less frequent and is associated with sound formation near the ear due to cardiovascular abnormalities and disturbances in musculoskeletal structures. Objective tinnitus is always secondary to a clearly defined organic cause.[3] Subjective tinnitus may manifest as a sniffing, buzzing, whistling, or ringing sound. Sometimes, there may be polymorphic exchanging characteristics, and if several types of sound phenomena are present simultaneously, we can talk about complicated tinnitus. If objective tinnitus is synchronous with heart function, there will be a sense of pulsation. On the other hand, an occasional onset is a characteristic of tinnitus due to muscular-skeletal disorders.[4]

The mechanisms of sound perception are very complex; therefore, it is difficult to clarify the origin of tinnitus as a phantom sound phenomenon. The primary auditory cortex is responsible for processing auditory information. It is located bilaterally, approximately in the upper temporal lobes, and takes part in the spectro-temporal analysis of the sounds, determining the frequency and time of the stimulus.[5] The associative cortex in the parietal and frontal lobes is responsible for final sound processing and is associated with the emotional and cognitive experience of sound. The auditory cortex has a crucial but ambiguous function in hearing. Multiple sounds are simultaneously transduced during the hearing process, and it is the responsibility of the auditory system to determine which components form the sound link. A disorder in any part of the complex auditory pathway may be a possible cause of tinnitus.[6]

The absence of different sounds usually amplifies the severity of tinnitus, therefore, it is most noticeable during sleep onset. This important physiological function is impaired since the symptoms worsen in silence and before sleep. Tinnitus may lead to the development or worsening of anxiety and depression disorders. On the other hand, psychological stress is often associated with increased intensity or the occurrence of tinnitus.[7]

The fifth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5) does not describe tinnitus as a separate entity, and the eleventh edition of the International Classification of Diseases (ICD-11) describes it as a non-specific symptom of hearing disorders but not as a separate disorder.[8] In 2021, the international multi-disciplinary group proposed a new definition of tinnitus as a conscious sense of sound or composite noise without an appropriate external sound source. As a disorder, tinnitus is defined only if there is an impairment of emotional, cognitive, and working functions, leading to subjective suffering.[9] This definition of tinnitus is more associated with affective disorders than with a disorder in the perception of reality. Some experts have characterized vertigo and tinnitus as hallucinations, vertigo as a spatial hallucination, and tinnitus as an acoasm. It is important to distinguish tinnitus from simple auditory hallucination.[10,11] It should be stressed that patients do not attribute any deeper psychological meaning to the sound caused by tinnitus, as they do with hallucinations. Tinnitus may provoke newer enhance existing psychological issues such as anxiety, addiction, and depression disorders. Some patients will require psychiatric treatment to overcome their tinnitus burden.[12,13]

Epidemiology

Tinnitus is a common disorder, affecting about 15% of the general population[5]. The severity of negative subjective experiences varies from slightly disturbing to extremely disturbing. Its incidence and prevalence are relatively stable worldwide, though not precise due to the wide range of intensities and polymorphic phenomena, as well as the complex and poorly understood etiology.[14]

Smokers, people exposed to chronic noise and stress, nightshift workers, and patients with various bodily comorbidities are more affected.[15] Gender prevalence studies are contradictory.[13,16] The likelihood of developing tinnitus significantly increases with ages. The general prevalence in the

population older than 65 is about 13.5%, but tinnitus occurs at all periods of life, and even children are not spared.[17] In a Polish study of 15,199 students aged seven to 12. 6% reported having tinnitus lasting five minutes or longer at least once in their lives, with no gender difference.[18]

Etiology

The cause of objective tinnitus is usually easily discovered by hearing sounds from nearby vascular and musculoskeletal structures. In subjective tinnitus, it is difficult to talk about the reason, therefore, we will talk about the reluctant phantom sensation that causes the patient's negative experience. Tinnitus may occur as a multi-level disorder. The occurrence of tinnitus is primarily related to the disturbance of the external, middle, or inner ear or the impairment or loss of hearing. The next level is in the central nervous system, which can be in the neuronal circles of the auditory and extra-auditory regions of the brain stem and cortex.[19]

The exact changes in neural activity that lead to the perception of the creation and maintenance of this phenomenon remain unknown.[20] There are several hypotheses about the formation of tinnitus, including the leading theory of degeneration at the level of sensory cells in the inner ear and the theory of neuroplasticity, which includes the impaired regulation of impaired excitation parts of the central nervous system included in the sense of sound.[21,22]The theory of neuroplasticity is followed by the theory of cortical reorganization, which recognizes changes in the structure of cortical circuits as an important factor in the psychological experience of tinnitus.[23]

Risk factors

Numerous studies have found several factors of risk associated with tinnitus, such as age, impaired or lost hearing, exposure to sound stress, psychological disorders, psychosocial stress, temporomandibular joint disorders, inflammatory diseases, head and neck trauma, ototoxic medications, viral infections, long-term smoking, and lack of sleep. Tinnitus occurs more frequently in people with comorbidities such as hyperlipidemia, asthma, osteoarthritis, rheumatoid arthritis, and thyroid dysfunction, and usually in patients with damaged hearing apparatus on all levels, from the external auditory canal, eardrum membrane, middle ear ossicles, to internal ear structures.[13] Although several risk factors for tinnitus have been discovered, not everyone with these factors experiences tinnitus or an equally profound sense of subjective suffering. Subjective psychological experience is of crucial importance for the sensation of tinnitus-related discomfort.[24]

As we live during the COVID-19 pandemic, it is important to emphasize the connection between viral infections and tinnitus. Viruses can harm the cells of the inner ear or the brain region involved in the sense of hearing in various ways. The varicella-zoster virus and human immunodeficiency virus are two examples of viruses that can directly damage neuronsorvascular endothelium,[25] whereas SARS-Co-V2 has been shown to cause hyper coagulability as one of the mechanisms of damage to peripheral hearing organs and central nervous system structures.[26]

There have been few studies on audio vestibular symptoms in SARS-CoV2 patients, but more patients with these problems can be expected in the future, especially after treatment in intensive care units with ototoxic drugs such as chloroquineanda-minoglycosides.[27] Preliminary results of the Italian study by Viola et al. on a small sample of 185 patients who were infected by COVID-19 at the beginning of the pandemic were published; 23.2% of patients had a different intensity and duration of tinnitus during and after the disease.[25]

Disorders within the Central Nervous System

Modern theories associate tinnitus with disorders of the central nervous system. The occurrence of tinnitus is explained by the spontaneous activation of neurons after peripheral damage to the organs of hearing and consequential regulatory disorders between the limbic system and the central auditory cortex.[17]

The dominant theory put forth by Jastreb offdates back to 1990, according to which damage to the peripheral organs of the hearing leads to changes in the audio vestibular path, which further leads to spontaneous hyperactivity in the brain stem and auditory cortex.[19,28] Subsequently, reorganizational changes in the brain stem and cortical areas occur as a neuroplastic response, which can enhance the subjective feeling of tinnitus. It is assumed that the displeasure of tinnitus induces a reorganization of neuronal activity that strengthens neural connections in brain areas related to the emotional experience.[29]

Changes in the central nervous system remain the target areas of the investigation of tinnitus etiology, but still without a full explanation. The complexity of the pathophysiology of tinnitus and its consequential changes in neuroplasticity and subjective experience is partly explained by the difficulties in treating this complex disorder. The failure to treat only one

cascade in this complex enchanted circle explains the persistence of tinnitus even after the treatment of hearing disorders. As mentioned earlier, future treatments must simultaneously focus on several aspects of tinnitus pathophysiology.[30] Research shows that changes in neuronal activity are not limited only to the auditory system but also include non-auditory brain areas such as the frontal lobe, which is involved in emotional experiences.[31,32]

Genetics

The number of genetic studies of tinnitus is increasing. A large Swedish cohort study from 2019 using data from the National Register between 1964 and 2015 suggests that tinnitus is related to genetic factors in 32% of cases. This study did not find a link between common environmental factors and the development of tinnitus.[33]There is a link between several genes and polymorphisms related to oxidative stress and the inflammatory response to tinnitus.[6]

The genetic research on tinnitus is focused on gene sequencing, bioinformatics analysis, and understanding gene regulation through epigenetic mechanisms. Examples of genes that may be associated with the occurrence of tinnitus are genes for pro-inflammatory cytokines and growth factors, whereas the example of epigenetic regulation of gene expression involved in the development of tinnitus may be the significant difference detected in the ratios of the methylation of cytosine gene sections for the brain-derived neurotrophic factor (BDNF) between the control group and patients with tinnitus.[24]

The function of BDNF is to inhibit neuronal damage and promote neuronal regeneration after a lesion, whereas damage to the auditory nerve system is a major pathophysiological cause of tinnitus. Changes in BDNF expression in patients with tinnitus could reflect the repair process following injuries to parts of the brain involved in hearing sensation. The genetic basis for the development of tinnitus requires further research.[34]

Clinical presentation

Tinnitus is a highly heterogeneous disorder with no significant success in classifying its clinical presentation. Problems related to tinnitus are described in a continuous gradation with no apparent boundaries. These patients may perceive a soft background or a loud external noise. The intensity of interference and functional limitations may vary from case to case, while approximately 20% of patients are severely disabled in everyday functioning.[12]

Subjective tinnitus is usually called tinnitus, and objective tinnitus is often called somatosound. Most tinnitus sounds resemble sniffing, buzzing, whistling, or ringing. Tinnitus patterns are stored in the auditory memory and are associated with limbic system emotions. Patients with tinnitus have significantly higher levels of depression and anxiety on psychological scales. Other mental disorders, such as alcohol abuse, dysfunction, and social function, including suicide, are more frequent among these patients.[35]

In objective tinnitus, physical movements such as turning the eyes, clenching the jaw, or applying pressure to the head and neck can change the frequency or intensity of the ringing. Objective tinnitus may be caused by neck muscle contractions or clenching of the jaw, which may disappear during sleep but usually returns within a few hours.[4] The occasional onset of tinnitus is characteristic of disorders in the nasopharynx or middle ear muscle contractions. Pulsatile somatosounds associated with heartbeats in objective tinnitus may result from carotid or vertebrobasilar stenosis.[13]

Tinnitus can be induced after noise exposure, and this acute-induced tinnitus duration ranges from minutes to weeks. If tinnitus lasts over two years, it is considered permanent and irreversible. Chronicity of tinnitus is associated with a negative treatment response.[3]

Diagnosis

The diagnosis of tinnitus is based on anamnestic data, such as duration, location, and sound frequency. It is primarily important to distinguish objective from subjective tinnitus. To rule out possible cardiovascular causes of occasional present-day tinnitus, it is critical to determine whether it is caused by a pulse wave. Stimulating and mitigating factors such as neck movements, swallowing, and head position speak in favor of a musculoskeletal etiology.[36]

Imaging radiological methods such as magnetic resonance imaging and computerized tomography are used to determine the etiology of cardiovascular and musculoskeletal diseases. The simultaneous appearance of other symptoms with tinnitus, such as loss of hearing, vertigo, pain, and ear discharge, indicates an otologic etiology.[19] Neurological damage may also cause tinnitus and should be excluded. It is important to assess the effects of tinnitus on patients, their mental experience, and their association with mental disorders such as anxiety, depression, and insomnia. Risk factors for tinnitus development, such as noise exposure, changes in diving or airplane travel pressure, infection, and ear or central nervous system

injuries, may indicate the level at which tinnitus developed.[35]

Unilateral tinnitus that has been present for six months or more or in the presence of a hearing disorder should have an extensive audiological assessment. If an objective cause is not found, we can discuss subjective tinnitus as an idiopathic phenomenon lacking objective diagnostic tools.[36]

Treatment

Different methods are currently used to treat tinnitus: medications, cognitive-behavioral therapy, neurofeedback therapy, and neuromodulation. There are now no strong guidelines or specific treatments for tinnitus. The current recommendations aim to treat mental comorbidities, including insomnia, anxiety, and depression, and improve microcirculation to lessen suffering.[37] Anticonvulsants, local anesthetics, antiarrhythmics, antihistamines, antidepressants, antipsychotics, anxiolytics, calcium channel blockers, diuretics, vasodilators, and vitamins are among the medications used to treat tinnitus.[38] So far, no medicinal product has been authorized by the Food and Drug Administration for the specific treatment of tinnitus.[39]

The use of anticonvulsants is based on the assumption of hyperactivity in brain pathways involved in sound sensation, but Cochrane's study has led to the conclusion that anticonvulsants are ineffective in treating tinnitus and have a significant risk of numerous adverse reactions that were manifested by as many as 18% of patients.[40]

Lidocaine, a local anesthetic and antiarrhythmic, is effective in treating tinnitus but carries a high risk of dangerous side effects.[41]

Antihistamines affect microcirculation, and studies have been conducted to prove their efficacy in treating tinnitus. Betahistine, a potent histamine H3 receptor antagonist and histamine H1 receptor agonist used to treat Meniere's disease, does not treat tinnitus successfully.[42]

The use of antidepressants in tinnitus treatment is widespread and particularly effective in treating comorbid symptoms of anxiety, depression, and insomnia but less significant in reducing the feeling of an unpleasant sound phenomenon. Several studies have found that tricyclic antidepressants and selective serotonin reuptake inhibitors effectively reduce tinnitus-related subjective suffering,[43] while trazodone has shown no effect.[44]

Sulpirideas an antipsychotic was noted in the treatment of tinnitus, which significantly reduced the tinnitus sensation in the study by Lopez-Gonzalez et al. by 56%, in addition to hydroxyzine by 86%.[45]

The most frequently prescribed medicines for tinnitus-related problems are benzodiazepines, which are significantly effective in treating anxiety disorders and insomnia. The occurrence of tinnitus after discontinuation of benzodiazepine therapy for anxiety supports the theory of disturbed neuronal circuits as an important cause of the occurrence of tinnitus and the increase in its intensity. Clonazepam has been shown to significantly reduce the subjective intensity of tinnitus.[32]

Calcium channel blockers, diuretics, and vasodilators reduced tinnitus intensity but had a negative impact due to numerous adverse cardiovascular reactions.[39]

Vitamin B preparations often alleviate tinnitus problems, but different studies confirm and deny their effectiveness.[46,47]

Tinnitus treatment can be divided into two categories based on the goal of therapy. The first is to reduce or eliminate the perception of tinnitus. The second focuses on the effect of tinnitus on the patient's life and aims to reduce anxiety and the consequent development of low mood, anxiety, and insomnia. Cognitive-behavioral therapy in tinnitus treatment focuses on changing the patient's negative experience by correcting negative and unrealistic beliefs and behaviors. Cognitive-behavioral therapy aims not to reduce negative experiences but to help create more positive thoughts in this situation.[48]

Numerous neuromodulation techniques that have emerged during the past twenty years have been recognized as promising new approaches for treating tinnitus. Neurofeedback, transcutaneous stimulation of the vagus nerve, transcranial electrical stimulation, and repetitive transcranial magnetic stimulation are new and promising treatment options.[2] Some of these techniques have demonstrated encouraging findings regarding prospective treatment and understanding the pathophysiology of various conditions within the brain's structure, which may be included in the pathophysiology of tinnitus. To obtain considerable and long-lasting improvement in tinnitus therapy, neuromodulation techniques become an effective therapeutic option.[8]

Conclusion

Tinnitus is a significant burden due to an unpleasant subjective experience that can limit everyday functioning and exacerbate mental disorders such as depression, anxiety, and insomnia. Tinnitus etiology is very complex, but it is always associated with hearing impairment, whether it is a disturbance in the peripheral organ, the audio vestibular nervous pathway, or brain areas related to

hearing sensation. After hearing damage, changes occur in neuronal circles important for the emotional and subjective sense of tinnitus.

The personal experience of tinnitus has a wide range of intensities, ranging from almost inconspicuous to incapacitating with a strong sense of suffering. Psychological structure and psychic comorbidities play an important role in the experience of tinnitus.

The treatment today is aimed at reducing sound perception and subjective suffering. Benzodiazepines and antidepressants play a special role in reducing emotional suffering. Cognitive-behavioral therapy aims to correct negative beliefs and unacceptable behaviors associated with tinnitus. Neuromodulation techniques have also been developed as an alternative treatment, with promising results. Numerous studies on the efficacy of various treatments have been conducted, some of which could be more consistent. Further studies are necessary to shed light on the development's causes and determine the appropriate therapeutic approaches.

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