



International Journal of Current Research and Academic Review

ISSN: 2347-3215 Volume 3 Number 5 (May-2015) pp. 122-126

www.ijcrar.com



Some aspects on etiology, diagnosis and treatment of Tinnitus

Sonya Varbanova, Dimitar Konov*, Spiridon Todorov and Venelin Marinov

Department of Otorhinolaryngology Audiology, Medical University– Sofia, 15,
Bul.Akademik Ivan Gueshov, 1431 Sofia, Bulgaria

*Corresponding author

KEYWORDS

Tinnitus,
Auditory
perception,
Pulsating noise

A B S T R A C T

The main goal of the study is defined tinnitus by an auditory perception in the absence of an external source of sound. Tinnitus is not a disease in itself but rather a reflection of something else that is going on in the hearing system or brain. This condition provides the distinctive possibility of extracting neural coding of perceptual representation, code of phantom perception, in this case auditory. There are many theories as to what causes tinnitus; therefore, there are many treatment options for tinnitus. This study attempts to recognize the etiology and the most appropriate treatment of tinnitus.

Introduction

Subjective sound is defined as a phantom hearing perception; the sensation of sound in the absence of sound or an abnormal hearing phenomenon, not connected to any external source of stimulation. The sensation may be restricted to the ears, or it might be experienced all over the brain.

The adaptation to the subjective noise is defined as an adapting process of the central nervous system. In this way, it decreases or eliminates the perception of stimuli, which are constant, or variable. Around 75% of the patients adapt to their subjective noise. If

this process is disrupted, the subjective noise can lead to heavy states of depression.

Epidiology: Ear noise most often occurs in the ages between 51 and 75. It is described that 12% of men over the age of 65 and 7% of the women in the same age group (64% of these patients) have normal hearing. It is widely accepted that around 10% of the population experiences subjective ear noise. (Coles, 1996; Toker *et al.*, 2005)

Tinitology is first defined during 1987, and it aims at studying the mechanisms of origination of subjective noise in the ear.

Characteristics of the noise properties

The noise can be defined as: objective, subjective and pulsating. It is experienced in one or both ears (one-sided or two-sided) or in the head. The noise can be constant or variable, differing in frequency and magnitude, connected or not connected with hearing loss. The subjective sensation can be associated with paraesthesia of the somato-sensory nervous system, and sometimes even with central neurological pain.

Pulsating noise

Arterial pulsating noise: can be caused by a series of arterial malconditions, such as arteriosclerosis, AV malformations, high blood pressure, glomus tumors, anomalies of the carotid artery, as well as the persisting stapedial artery.

Venous pulsating noise: this can be caused by anomalies, such as dehiscence of the bulbous venae jugularis, thrombosis of the sinus sigmoideus or pseudo-brain tumors.

Non-vascular subjective noise is the third kind of pulsating ear noise, caused by: muscular myoclonus, dysfunction of the Eustachian tube, blockage of the external ear canal.

Subjective noise

This is represented by different sound pictures. Moller (2000) accepts that quiet to average subjective noise is most probably generated in the cochlea, while the strong, unbearable noise – in the central nervous system, as pain. These generators in the central nervous system are most probably a consequence of reorganization of the nerve paths. They can result from real incoming sources of irritation, or in their absence, most commonly from the peripheral part of the hearing analyzer. The topic organization

of the cochlea is analogical to that of the central nervous system. The high-frequency hearing loss, at a peripheral level, can decrease the inhibiting function of the hearing nervous system, as a result of which, the arousal of the neurons increases, which is experienced as a subjective noise.

Patients with sensorineural hearing loss have reported tinnitus auris. These complaints are expressed strongly by patients, exposed to noise and vibrations. Koltenbach and Alman (2000) describe the spontaneous activity of the dorsal cochlear nucleus. The damaged cochlea with a variable decrease in hearing sends changed and chaotic signals along the hearing path to the central nervous system. These changes lead to abnormal interactions between the hearing and other central nervous paths. The decreased nervous hearing stimulations, on the other hand, lead to changes in the dorsal cochlear nucleus, which lessens the spontaneous activity in the central hearing system. These changed conditions of the hearing path confuse the central nervous system, which generates its own noise, due to the lack of normal signals. These new signals are perceived as noise.

Simpton and Davies (2000) accept that the noise level is connected with the dysfunction of the serotonin transmitters. Serotonin is a neuro-transmitter, which participates in modulating the paths of serotonin, which control the moods and emotions of people, and influences the origination of hallucinations, also owed to the plastic alterations in the central nervous system.

Etiology of the subjective tinnitus auris

- Permanent or sudden noise damage (after an explosion, a shot, or lengthy exposure to the influence of enormous noise levels;
- Head injuries with brain damage;

- Incorrectly positioned wisdom teeth;
- A side effect from certain medications, such as: antibiotics, aminoglycosides, diuretic substances, aspirin or chininum, after over-dosing, or an allergic reaction;
- Substance abuse: alcohol or other psychotropics;
- High blood pressure
- Hyperfunction of the thyroid gland, and other endocrinological alterations;
- Alterations in the inner hearing cells;
- Anomalies in the afferent nervous system;
- Loss of part of the GABA-inhibition neurons;
- Alterations in the GABA-genes, responsible for lysosomal membrane proteins, connected with the glucolipid metabolism (which leads to combined cortical hearing phantom sounds, pure tones or white noise.)
- Hearing loss due to ageing and noise injury, leading to damage in the cochlea. The damaged cochlea with a variable decrease in hearing sends changed, random signals along the hearing path to the central nervous system. These changes lead to abnormal interactions between the hearing and other central nervous paths.

Materials and methods

In this study, we have tracked patients with subjective ear noise, divided in groups, according to:

- age;
- etiology of tinnitus;
- additional medical states;
- Labor conditions.

Group 1: Patients with an origination of subjective noise after acute noise trauma:

- with normal hearing – 13 patients. Noise tolerance – 7 (according to our own adapted scale for self-assessment, and endurance to the subjective noise)
- with decreased hearing – 17 patients (average hearing loss: through air conduction for 500, 1000, 2000 and 4000 Hz.), the main loss ratio being 37 dB, and noise-tolerance – 6.

These are patients up to the age of 35, without any previous ear diseases; the reason for the complaint being a night out at a noisy club, a concert, and mass-celebrations with fireworks and loud music.

Group 2: Patients with stress-noise, with normal hearing, normal otoacoustic emissions and a lack of anamnesis for any previous ear diseases. These subjects (13 patients) report poor understanding of speech in critical conditions and have a noise-tolerance level of 2.

Group 3: Patients with diseases of the sound-conducting part of the hearing analyzer, accompanied by conductive or combined hearing loss and subjective noise. (23 patients, aged 15 to 51) Average hearing loss: 42 dB (through air conduction for 500, 1000, 2000 and 4000 Hz.); noise tolerance – 7.

Group 4: Patients with noise-admittance hearing loss and strong, disturbing tinnitus – 41 people, divided in two sub-categories:

- 19 patients with chronic ailments of general character – high blood pressure, diabetes, artrosis alterations of the spine – average hearing loss: 47 dB (through air conduction for 500, 1000, 2000 and 4000 Hz.), and a noise tolerance level of 4
- 22 patients exposed to chronic noise damage – average hearing loss: 63 dB (through air conduction for 500, 1000,

2000 and 4000 Hz.), and a noise tolerance level of 3.

Clinical diagnosing for pulsing noise is particularly difficult, and requires a broad spectrum of common medical and audiological examinations, to define and objectively determine the kind of tinnitus with maximum accuracy, as well as its duration and in some cases, the origin of the subjective noise. The necessary examinations include:

- 1. anamnesis;
- 2. Otolaryngological status check;
- 3. General medical examinations – to verify the cardiovascular, kidney, endochrynological, metabolic, collagen diseases and others.
- 4. Audiometric examinations – tonal and speech audiometry, tympanometry and impedancemetry, OAE, Brainstem Evoked Response Audiometry, electronystagmography. We created our own self-assessment scale for subjective noise, for noise tolerance levels (0 – 10).
- 5. Blood criteria: Full blood test, and an examination of the thyroid hormone levels.
- 6. Imaging examination: CAT-scan and MRI.

In this way, we can determine the kind and degree of hearing loss, the topic of the lesion, and we can exclude retro-cochlear damage, Morbus Meniere, secondary hydrops of the labyrinth, as well as interaction with other diseases.

Results and Discussion

We applied different treatment to the patients in the groups described above, according to the etiology of their conditions, as well as non-medical therapy, with different time spans.

A. The patients with acute acoustic trauma were treated with cortical steroids in decreasing doses, vessel-expanders, de-swelling substances, and vitamins of the ‘B’ group. The treatment was conducted for 10 days, on an inter-vein basis, and 15 days per os. In the check examination on the tenth day, we noted an improvement in the hearing, from 37 dB to 23 dB, and an influence in the noise from level 7 to level 10.

B. In treating the patients of the second group, we used the following medicaments: Dexametazone; Benzodiazepines and a silencing of the noise by listening to pleasant music (analogical to tinnitus retraining therapy). The results after 10 days of training were: a positive influence on the stress levels, but with the noise persisting, with an intensity level of 8. The continuation of the treatment for a further 20 days did not lead to any substantial changes.

C. The patients in this group were treated etiologically, according to their basic disease, by additional therapy of cortical steroids and vitamins. As a result of this treatment, we observed correlation between the dynamics of the main inflammatory disease of the middle ear and the degree of the hearing and noise damage. The changes on the tenth day were: an improvement in hearing, from 42 to 21 dB, and a decrease in noise to level 9.

D. In treating the patients of this group, the first sub-division was treated with medicaments, such as vessel-expanding substances and vitamins, and non-medicament therapy, with tinnitus-masker or the use of hearing aids for permanent hearing loss. The result is a change in objective noise from 4 to 6, for the patients with hearing aids, which experience better social interaction with the improvement in

their hearing, and therefore, better tolerance of their own noise. The patients of the second sub-division were treated intensively with corticosteroides in decreasing doses, vessel-expanders, de-swelling substances, and group 'B' vitamins. Ten days later, the therapy was continued with benzodiazepines and tinnitus masker. We registered an unsatisfactory change in relatively few of the patients.

Treatment

With a mild effect:	With a moderate effect:
Lidocaine	Carbamazepine
Benzodiazepines	Ginko-Biloba
Antidepressants	Gabapentine
Electrical stimulations	Vessel-expanders
Enoxaparine	Vitamins
	Minerals
	Dexametazone

Non-medicament therapy applied: tinnitus-masker, hearing aids, tinnitus retraining therapy and others.

We recommend, if there is a persisting subjective noise in the ear:

1. Avoid loud sound, noise and vibration;
2. Avoid nervous stimulants, such as caffeine and nicotine, even in small quantities;
3. Avoid ototoxic drugs, lengthy dosage of aspirin, nonsteroid antinflamatory, quinine and its derivatives, and ototoxic antibiotics, some diuretics, chemotherapy with cis-platinum;
4. Patients with subjective ear noise should keep to certain physical regimes and dieting.

Conclusion

We found significant differences between tinnitus sufferers. In summary there already exists a huge market for a tinnitus drug, which will further grow and which is

currently still untapped. Similarities between tinnitus and other CNS disorders suggest synergistic effects. Recent advances in tinnitus research indicate, that the problems which might have hampered the field in the past, are solvable in the near future.

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