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Tinnitus Therapy Based on High-Frequency Linearization Principles – Preliminary Results

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The aim of this work is to present problems related to tinnitus symptoms, its pathogenesis, hypotheses on tinnitus causes, and therapy treatment to reduce or mask the phantom noise. In addition, the hypothesis on the existence of parasitic quantization that accompanies hearing loss has been recalled. Moreover, the paper describes a study carried out by the Authors with the application of high-frequency dither having specially formed spectral characteristics. Discussion on preliminary results obtained and conclusions are also contained.

Keywords: tinnitus, sensorineural hearing loss, tinnitus masking, Tinnitus Retraining Therapy (TRT), signal quantization, dithering technique, high-frequency linearization, ultrasound dither noise

1. Pathogenesis of tinnitus

The aim of this work is to elucidate problems related to the pathogenesis of tinnitus, treatment methods and research carried out by the Authors.

Jastreboff (1995; 2004) defines tinnitus as the sensation of sound without any stimulation of the organ of hearing by external acoustic signals. Jastreboff's division into subjective and objective tinnitus, also called somatic sounds, has widely been accepted (Jastreboff, 1995). Objective tinnitus is the perception of a somatic sound which arises due to abnormalities or pathologies in structures adjacent to the cochlea or the within head and neck. As a result, the cochlea becomes mechanically stimulated. Such sounds can be registered and heard by other people.

Subjective tinnitus is caused by the nervous system activity and is not associated with cochlea stimulation by any external stimulus (Bartnik *et al.*, 2001; 2002; Lalaki *et al.*, 2011). These sounds cannot be heard by other people or registered by any acoustic equipment. Only the affected patient can hear them. They result from perceiving a neural signal that reaches the cerebral cortex via the auditory pathway and is generated otherwise than by an external, mechanical stimulation of the cochlea. Patients describe their subjective auditory sensations as: chirping, whistling, squeaking, murmur of the sea, hum of the wind, kettle whistling, pulsation and many other. These sounds can be heard in one ear or both, between the ears, inside the head, with the same or different volume, with constant or changing intensity and duration.

Another classification was proposed by Zenner and Pfister (1999) who in addition to objective and subjective tinniutus discerned also neurosensory and central type of tinnitus, with regard to the topographical level affected.

1.1. Aetiology of objective tinnitus

Objective tinnitus can be caused by various conditions associated with generating somatic sounds. Somatic sounds can be classified (Herraiz, 2005) as either vascular or mechanical. Objective tinnitus can be either pulsatile or non-pulsatile. Marsot–Dupuch (2004) stated that non-pulsatile tinnitus, often of venous origin, is predominantly bilateral, constant, occurring both at rest and effort, and described by the patients as "buzzing of a fly". Pulsatile tinnitus is of vascular origin and often unilateral. Its fre-

quency is synchronized with the heartbeat, and its intensity may be related with physical effort. Another cause of objective tinnitus may be an intracranial tumor, which results in characteristic unilateral tinnitus, combined with unilateral, progressive hearing loss. Carotid artery stenosis can also lead to a vascular hum. Similar symptoms can occur in patients with large aneurysms or as a result of external pressure on the carotid vessels. Mechanical tinnitus is predominantly non-pulsatile and its cause is easier to diagnose.

Objective tinnitus can be alleviated by treating the underlying condition. Both vascular and mechanical tinnitus may require surgical treatment. In some cases, pharmacological treatment can be administered that e.g. reduce muscle tension. In cases of objective tinnitus, noise generators or auditory discrimination training are generally not prescribed. However vascular and mechanical tinnitus may be treated by using the auditory training (mostly TRT).

1.2. Aetiology of subjective tinnitus

Aetiology of subjective tinnitus is not entirely clear as yet. Scientists agree that this type of tinnitus is a phantom perception of a neuronal signal, generated non-acoustically as a consequence of abnormal nervous activity, interpreted by the nerve centers as sound. None of the hypotheses to explain abnormal activity of the nervous system that underlies subjective tinnitus has been sufficiently supported to constitute the basis for the development of causal treatment methods. Apparently, that there is no mechanism of tinnitus pathogenesis common for all its types (Jastreboff, 1990). This suggests that the so-called noise generator that sends abnormal nervous impulses may be situated at different points of the auditory pathway. Bartnik *et al.* (2001) suggest that the mechanism of pathological signal generation and its perception in the auditory cortex should be analyzed separately. This is because only the appearance of the pathological activity in the auditory pathway causes reaction, i.e. the signal is detected, amplified, perceived, sustained, and finally interpreted as tinnitus, which triggers the emotional and defensive reaction to the noise. Jastreboff stated that the process by which tinnitus emerges, can be divided into three stages; generation; detection; perception and evaluation. Generation occurs usually at the periphery (although it may be central) and in the majority of cases can be associated with disorders occurring in the cochlea or the cochlear nerve. The process of detection occurs at the level of the subcortical centres, while perception and evaluation of tinnitus-related activity occurs in the auditory cortex, with considerable and significant participation of the limbic system, the prefrontal cortex and several other cortical areas (Jastreboff, 1990).

It seems that the compensation mechanism plays a significant role in tinnitus perception. Minor peripheral disorders of the organ of hearing trigger a number of pathological processes in the central auditory pathway. The system of hearing attempts to compensate for the defects and limited data flow. Consequently, sensitivity is increased of the centers that participate in detection and perception of sound. This may result in tinnitus and hyperacusis.

Hypotheses on aetiology and pathogenesis of tinnitus most frequently quoted in the literature have been summarized below.

1.3. Cochlea as the generator of tinnitus

It is assumed that in up to 80% of cases tinnitus is generated in the cochlea as a result of pathological lesions in the inner ear (Bartnik *et al.*, 2001; Eggermont, 1990; Jastreboff, 1996). Tinnitus in most cases accompanies sensorineural hearing loss, associated with e.g. acoustic trauma, age (presbyacusis), ototoxic damage or Meniere's disease.

Tinnitus is often diagnosed along with sensorineural hearing loss with the use of an audiogram (Hazell, 1984). It should be mentioned here that sensorineural hearing loss very often occur due to exposure to excessive noise (Augustyńska *et al.*, 2010; Czyżewski *et al.*, 2007; Dudarewicz *et al.*, 2010; Kotus, Kostek, 2008; Kotus *et al.*, 2010). This is also accompanied by volume equalization, which results from the loss of non-linearity in the cochlea micromechanics. Therefore, it has been suggested that outer hair cells participate in tinnitus pathogenesis, as they constitute the basis for cochlea non-linearity (Bartnik *et al.*, 2002). Tinnitus can also occur when hair cell damage is so minute that it cannot be detected on the pure-tone audiogram (Bartnik *et al.*, 2002; 2007).

1.4. Neurophysiological tinnitus bases

1.4.1. Efferent system

The cochlea micromechanics includes a feedback loop between the afferent and efferent systems. The afferent system carries nervous impulses from the cochlea to the CNS (see Fig. 1). When the auditory fibers are stimulated, the feedback loop becomes active and efferent nerve fibers become stimulated, causing outer hair cell hyperpolarization and afferent response inhibition. The efferent system is believed to inhibit cochlea activity by limiting its response to acoustic stimuli. Thus, it protects the sensory cells of the organ of Corti. Moreover, it is believed that the feedback between the basilar membrane and the outer hair cells, as well as the contractile activity of outer hair cells, generate weak sound waves, directed back to the auditory tract. This phenomenon is known as otoacoustic emission.

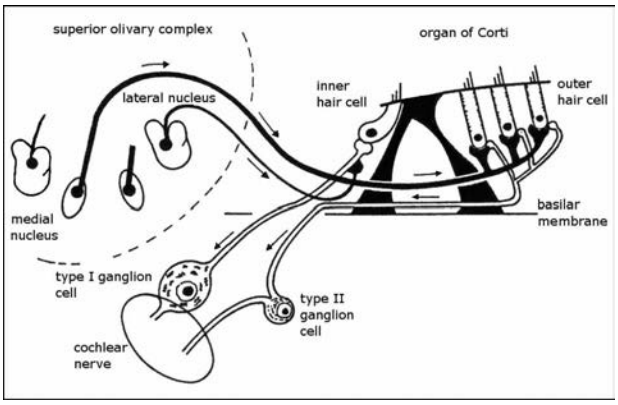

Fig. 1. Afferent and efferent innervation of the organ of Corti (Śliwinska–Kowalska, 2005).

Activity of type II afferent nerve fibers that lead from the outer hair cells carries the data on the sound wave captured by the organ of hearing. These data, processed in the brainstem, affect the activity of the efferent system, which in turn influences the activity of the outer hair cells. Consequently, impaired activity of the outer hair cells on a given area of the basilar membrane indirectly inhibits the activity of the efferent fibers. This, in turn, reduces the inhibiting effect of type I afferent fibers, causing hyperactivity of the functioning inner hair cells. Such modifications in the inner ear and the remaining elements of the auditory pathway, without any natural mechanisms of stimulus selection, may lead to tinnitus.

Unbalanced activity of type I and type II afferent fibers

The majority of factors that damage the cochlea cause primarily outer hair cell degeneration and progressive imbalance (disharmonic damage) of the outer and inner hair cell afferent system (Jastreboff, 1990; 1995). Within a given area of the basilar membrane, outer hair cells are damaged, while the respective inner hair cells stay intact. In these areas, the tectorial membrane may collapse due to the dysfunction of the outer hair cells. This results in reducing the space between the tectorial membrane and the cilia of the still active outer hair cells and may lead to inner hair cell activation, causing abnormal afferent activity and tinnitus.

Cases of patients with tinnitus and no apparent hearing loss have been reported, as well as cases of patients with a significant hearing loss and no tinnitus. It should be noted that pure-tone audiograms represent mainly inner hair cell activity and that dispersed damage of up to 30% of outer hair cells does not significantly affect the threshold of hearing (Fabijańska *et al.*, 1999). The condition of the outer hair cells can be determined with the use of evoked otacoustic emissions and distortion product otoacoustic emissions. When a patient suffers from a symmetrical damage of both outer and inner hair cells, tinnitus may not occur. Another patient, with an identical audiogram, may experience tinnitus if asymmetrical damages are present, which causes unbalanced activity of type one and type two afferent fibers.

Gate control theory

According to Tonndorf's (1987) theory of gate control, the two types of afferent fibers cooperate to produce subjective sensations. A stimulus from one type of the system "closes" the gate for stimuli from the other type on relay neurons in the brainstem and spinal cord. Moreover, the stimulus regulates data transmission to higher levels of the CNS. When one type of the hair cells is damaged, the gate balance between the stimuli carried by afferent fibers leading from these cells is shifted in one direction. This way, damage of the outer hair cells might open the gate for the stimuli from the inner cells and create the sensation of noise. It is also possible that the balance between these two systems is influenced by the efferent system. In that case, an analogy could be found with the pain control system.

1.4.2. Interrelated spontaneous activity of auditory fibers

One of the theories on the genesis of tinnitus associates it with spontaneous nervous activity and increased discharge frequency in the nervous fibers. Some experiments on animals disprove this theory. After exposing laboratory animals to a loud noise or high doses of ototoxic drugs, spontaneous activity of the acoustic nerve fibers was reduced (Bartnik *et al.*, 2001). Therefore, a suggestion was raised that the sensation of noise is probably associated with time-domain rather than the frequency of discharge in the auditory nerve (Moller, 1995).

Normally, the increased volume of sound causes increased synchronization of the nerve fiber activity, although it never reaches the maximum value. The level of synchronization determines whether a given stimulus is interpreted as sound or not. Pathological processes, however, may synchronize nervous activity without stimulation by external sound and cause tinnitus. Moller (1995) states that such factors may include mechanical damage (e.g. in the course of a surgery), where myelin sheath is injured and the affected nerve loses its electrical insulation. Thus, nervous impulses may be transmitted between the fibers. Another damaging factor could be the pressure on the nerve, exerted by a tumor or arterial loop pulsation, etc.

1.4.3. Influence of the non-classical auditory pathway

The extralemniscal system is the non-classical auditory pathway, which carries out a less specific analysis

of sound and its activation is related to phenomena of sound distortion and hyperacusis. These pathways are active in infancy but are rarely stimulated acoustically beyond the age of 20 years old. An exception to this is the activation that occurs in some severe cases of tinnitus (Herraiz, 2005). The afferent neurons of this pathway lead mainly to the association cortex. The suggestion to link tinnitus with the extralemniscal system was proposed because evoked potentials registered in the brainstem show no lesions even in severe cases of tinnitus. The presence of other symptoms such as hyperacusis, anxiety or depression is also associated with the participation of the non-classical (extralemniscal) auditory pathways.

1.4.4. Deafferentation and sensitization of the central neurons

Deafferentation, or interruption of the stimuli transmission from the cochlea to the CNS, causes major change within auditory representation areas, where nervous impulses are received that code the frequency and volume of sound in the areas that border with the damaged zone (Bartnik *et al.*, 2001). This has been showed in research carried out on animals with damaged hearing due to acoustic trauma, where increased sensitivity to electrical stimulation was observed (Gerken, 1976). Without stimulation or input, the inhibition systems switch off and stimulation systems become dominant that increase the activity of the hearing centers. As a result, neurons react to low-level stimuli, which would normally remain below the reaction threshold (Bartnik *et al.*, 2001). Such stimuli usually originate in the areas that border with the damaged zone in the cochlea. As a result of CNS remapping, the area of the auditory cortex expands, respectively to the expanded cochlear representation for frequencies that border with the damage.

1.4.5. Influence of the sympathetic system

Numerous patients report that fatigue, stress or conditions related with sympathetic activity aggravate tinnitus. Papers exploring the possible causes of tinnitus stress the presence of significant sympathetic innervation of the cochlea (Bartnik *et al.*, 2001; Skarżyński *et al.*, 2000). If sympathetic fibers can stimulate fibers of the cochlear nerve, it is also possible that the tonic activity of sympathetic fibers may create auditory sensations without external acoustic stimulation (Meikle, 1995).

1.5. Tinnitus – analogy to quantization systems

There is a number of theories and hypotheses on the genesis of tinnitus and many of them are based on various pathologies within the inner ear or higher levels of the auditory pathway. The most popular ones include disorders of motor, electromechanical or biochemical activity of the auditory cells or disproportional (disharmonic) damage of the outer and inner hair cells. Disharmonic damage means that the outer hair cells are affected, while the inner hair cells remain intact. This type of damage is quite common, because outer hair cells are more liable to become damaged than the inner hair cells (Bartnik *et al.*, 2002; 2007). Audiological tests show higher hearing threshold (as regards low-volume sounds), normal hearing of loud sounds and normal or even lower threshold of acoustic discomfort, which may point to hyperacusis. In such cases, neuron activation occurs in response to higher-volume signals, according to a threshold system of a higher activation level. This type of hearing loss, also characterized by the presence of volume equalization, is classified as cochlear sensorineural hearing loss.

Czyżewski *et al.* (2002) see tinnitus as the development of an additional mechanism of threshold quantization of weak acoustic stimuli, caused by the increased activation level of nerve cells. Thus, the difference between the hearing threshold and normal hearing is interpreted as the quantization threshold. In audiology, theories that aim at clarifying this phenomenon do not directly embrace the mechanisms of signal quantization, which takes place according to the threshold characteristics in a given transmission system. In the proposed theory (Czyżewski *et al.*, 2002), human hearing is modeled by a system that includes a "telecommunication" channel – the ear and the auditory nerve fibers – and a receiver – the auditory cortex. In such a channel, a number of phenomena can be observed, such as filtration, occurrence of interference and quantization, which may contribute to tinnitus generation. Similar processes take place in digital transmission channels.

1.6. Treatment of tinnitus

Despite numerous clinical studies on tinnitus treatment, no completely successful method has been designed yet. This results from the complexity of the issue and insufficient understanding of the tinnitus pathogenesis. Most of the mechanisms described above are only hypotheses. According to Skarżyński *et al.* (2000), this variety of hypotheses proves that there are numerous causes of tinnitus. As a result, there are also many different treatment methods and, notably, their effectiveness is often uncertain.

Presently, the most popular method is *Tinnitus Retraining Therapy* (TRT) (Jastreboff, Hazell, 2004). It is utilized for auditory habituation understood as retraining the brain. It was developed by Jastreboff in late '80s (Jastreboff, 1990) who introduced the neurophysiological model of tinnitus.

This model assumes that tinnitus results from abnormal activity on all levels of the auditory pathway and of some subsystems of the central nervous sys-

tem other than the auditory system (Jastreboff, Hazell, 1993; Jastreboff, 1995). The method makes use of the brain plasticity, the ability of the central nervous system (CNS) to modify neuronal connections, as well as filter and select signals. With this ability, the brain can manage the large amounts of data which it constantly receives. According to Bartnik *et al.* (2001), the TRT method aims to transform tinnitus into a neutral sensation and to make patients less aware of their tinnitus. TRT should lead to habituation of reactions and habituation of tinnitus perception. The therapy consists of therapeutic meetings and, predominantly, auditory training. This form of therapy is time-consuming and requires good cooperation between the patient and the attending physician.

Sound applied in TRT is supposed to reduce the contrast between the tinnitus noise and the background noise (Jastreboff, 1996). Sound can be supplied using different devices, e.g. from a CD player, from the radio or from noise generators. If noise generators are used, the generated noise should not cover tinnitus but only limit the contrast in the auditory pathway. Appropriately set generators, which should be applied to both ears, are used for app. 8 hours a day. The therapy lasts 14–18 months or even longer. It seems that noise generators can be used not only to make the phantom perception less bothersome, but also to stimulate auditory habituation. Tonotopic organization of the cortex changes according to the properties of signals supplied by the auditory nerve. It is thought that generators provide a new type of stimulus, which induces tonotopic reorganization of the auditory cortex. Therapy utilizing generators can make tinnitus less annoying by way of auditory training. In this case the auditory cortex is stimulated by the correct signal, and the perception of tinnitus is switched off.

2. Utilizing sounds near the upper audible frequency limit and ultrasounds

Broadband noise shows good masking properties. However, using this type of noise in tinnitus treatment has several disadvantages. Broadband noise is not a natural sound, therefore it might be annoying or unpleasant when applied for longer periods of time. Moreover, components of this noise effectively mask useful sounds as well, thus affecting the understanding of speech or hearing soft sounds. Therefore, in some applications the range of the masking noise is reduced to e.g. frequencies above the speech level. This solution has been applied in the UltraQuiet therapy (Goldstein *et al.*, 2001). This technique employs sounds in the 10 to 20 kHz range, and music is used to modulate them. The signal, processed as described, is emitted by a bone conduction transducer at a level of 6 dB SL for 30–60 minutes, twice a week. This method is based on the so-called residual inhibition, thanks to which the intensity of tinnitus after the masking noise is reduced. This method differs from conventional masking, as tinnitus is masked by noise whose frequency does not overlap with the tinnitus frequency or at least this overlapping is not necessary.

Research on the effectiveness of this treatment carried out by Goldstein *et al.* (2001; 2005) and Lenhardt (2003) typically used frequencies of 10–20 kHz, although in some cases the range was broadened to 6–20 kHz or 20–26 kHz. The authors concluded that the method was most successful in patients with the hearing threshold of 50 dB or lower in the range of 10–14 kHz. As a result, some patients reported reduction or disappearance of the disturbance caused by tinnitus for a period of a few minutes up to a few days of the moment when masking stopped. Some patients did not experience symptom alleviation due to the treatment. The authors stress that one of the most important advantages of this method is a relatively short time of treatment and fast results as compared to TRT. These results have been criticized by Tucker (2010), who pointed to such issues as a small number of patients, lack of randomization and lack of blind tests.

According to Lenhardt (2003), tinnitus masking is possible with the use of audible sounds of high frequencies and ultrasounds of low frequencies. Mechanisms of reception and perception of high audible sounds and ultrasounds are identical. The only difference is that for ultrasound detection an intermediary centre is essential, namely the brain. The role of the brain in demodulating ultrasounds is to map the ultrasounds on the first few millimeters of the basilar membrane. The basis of this phenomenon is the brain resonant frequency, which, according to the author's calculations, should be between 11 kHz and 16 kHz. The exact value depends on the individual cranial geometry. The brain ultrasound demodulation theory suggests that the sound produced by brain resonance can be transmitted to the ear via fluid channels. By applying ultrasound noise by bone conduction, cochlear masking should be obtained in frequencies that correspond specifically to the resonant frequency of the brain. The research (Lenhardt, 2003) shows that ultrasound masking can suppress audio thresholds in the range of 8 kHz to 12.5 kHz by 2–29 dB.

Scientists of the Gdańsk University of Technology reached similar conclusions in research, where the ultrasound noise in the range of 16–30 kHz was used (Czyżewski *et al.*, 2006). In presence of ultrasound dither noise applied by bone conduction, audio threshold suppression was observed around 8 kHz by applying 15 dB in pure-tone air conduction audiometry. The authors state that when applying a sufficiently high ultrasound dither volume, the masker starts to function as a classical tinnitus masker that uses audible sounds.

The above examples show that it is justified to use ultrasound maskers in tinnitus treatment. It should be stressed that even though noise is applied in frequencies higher than the audible frequency, it may be

perceived like an air-conducted sound in the range of 8–16 kHz. The research (Lenhardt *et al.*, 1991) shows that pitch perception depends on the highest audible frequency in the examined ear. This means that pitch associated with ultrasound in one ear can be different than in the other one due to asymmetrical hearing loss. This is referred to as ultrasonic diplacusis, or double hearing. The mechanism of ultrasonic pitch detection is, according to Lenhardt (2003), strictly linked with the brain resonant frequency transmitted to the inner ear. Thus, the basilar membrane carries stimuli to the still functional hair cells responsible for high frequency detection, which in turn leads to sound perception.

In some cases, tinnitus masking with the use of ultrasounds may be difficult to perform. This is because apart from the masking effect, which is beneficial, additional perception of audible sounds that originate from the ultrasound masker may be bothersome.

3. Application of the high-frequency linearization in tinnitus treatment

As described above, the pathogenesis of tinnitus may be modeled using an analogy to the digital transmission channel. When the amplitude of the quantified signal is close to that of the quantization threshold, the spectrum of the processed signal shows significant harmonic interference. The quantization error may have a value as high as the quantization threshold itself. As a result of interpreting the hearing loss as the increase in the quantization threshold, this threshold may appear on a level that is a very high in comparison to the full scale of hearing dynamics. Thus, the effects related to the auditory signal quantization become even more significant.

The hearing loss-related quantization is very specific, because it involves introducing a dead zone to the quantizer characteristics (Czyżewski *et al.*, 2006). The analyses show that if the level of a signal does not enter the dead zone, the output signal of the quantizer has a zero value at a given moment. If the signal enters the dead zone, a correct quantizer output value is obtained. Nevertheless, it has been shown that such quantization causes severe frequency distortions of the signal. The signal spectrum includes additional components, which can be heard, according to the phantom pain theory, if they are above the hearing threshold. According to the phantom pain theory, the threshold curve of the auditory cortex does not necessarily have to become deformed with the changes of the threshold curve in the ear. The difference between these two curves is the dead zone, where phantom pain can be present. In the case of tinnitus, phantom sound perception occurs.

In digital sound processing, methods have been developed of noise elimination that is generated in course of threshold quantization. These methods are known as the dithering technique. Dithering consists in adding broadband noise of a very low level to useful acoustic signals, in order to prevent spontaneous noise generation that results from the threshold characteristic. These issues were analyzed in the paper by Czyżewski *et al.* (2002), who demonstrated that the above interpretation of tinnitus genesis, based on the digital sound processing, and the resultant utilization of the dither technique may be useful in the tinnitus treatment.

The relevant papers and analyses (Czyżewski *et al.*, 2002; Klejsa, 2005) show that when dither noise is applied, tinnitus masking effect may be more significant. Moreover, providing hair cells with stimuli triggers their activity and, consequently, stimulates the auditory nerve fibers. The resulting activity of the fibers resembles to some extent a low-level spontaneous emission.

It seems that obtaining the latter effect only, namely auditory stimulation, would be most beneficial, because it could result in tinnitus reduction by way of limiting the phantom perception. Consequently, the treated patients would not have to be constantly exposed to the audible masking signal. If broadband noise is added to the useful sound signal before quantization, the noise component is clearly distinguishable in the signal spectrum after quantization. Consequently, the dither applied in digital tracks has strictly defined spectral characteristics (Lipshitz *et al.*, 1992). The noise characteristics is designed to have frequencies associated with lower perception in humans. Research carried out at the Gdańsk University of Technology showed that applying dither in the frequency range of 16–30 kHz made it possible to significantly lower the level of spectral components associated with frequency distortions, introduced due to the presence of a dead zone. Even though the spectrum of a signal after dithering includes a prominent dither component, it remains beyond the audible ranges (Czyżewski *et al.*, 2002).

4. Research

In order to verify the results of the analyses and research summarized above, a series of tests has been carried out on patients with tinnitus of different aetiology. The aim of the research was to determine the effectiveness of ultrasound dither treatment in these patients.

Before the research, medical history of each patient was taken to determine the aetiology of tinnitus, its type and duration, as well as factors that influence the sensation. Moreover, the medical data has been re-analyzed to exclude the diagnosis of pathologic causes such as tumor and other extracochlear or central nervous system conditions. In the research conducted, a set of devices was applied that had been constructed at the Gdańsk University of Technology and that enable ultrasound transmission to the organ of hearing by bone conduction. Elements of the measurement set-up have been shown in Fig. 2.

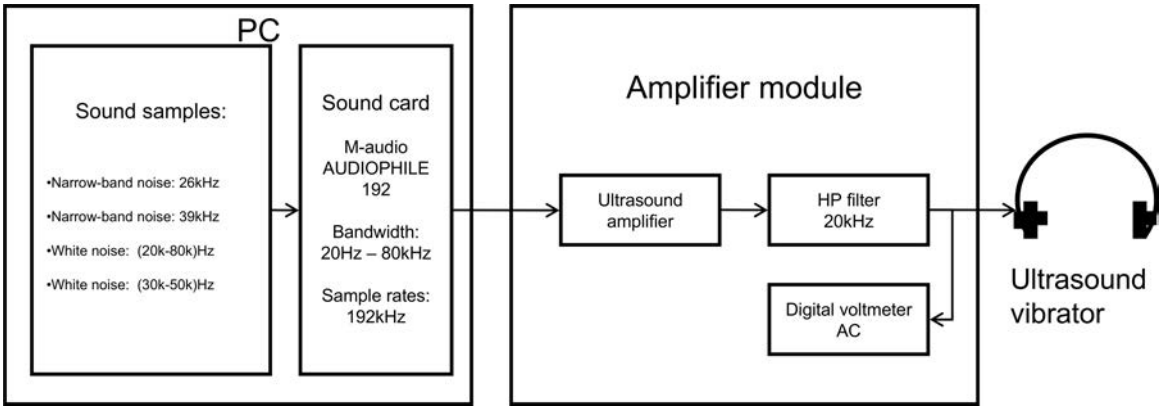

Fig. 2. Block diagram of the measurement set-up.

In the research presented, sound samples were used, generated with the use of the AdobeAudition software. Sound samples were prepared in such a way that white noise in the range of 20–80 kHz was generated and then the required ranges were isolated with the use of a band-pass filter with the Kaiser window of 180 dB. Tests performed used white noise filtered in the following ranges:

- 20–80 kHz,
- 30–50 kHz,
- narrow-band noise with the centre frequency of 26 kHz,
- narrow-band noise with the centre frequency of 39 kHz.

The obtained samples were saved in the WAV file format.

The study included the following steps:

1. Otoscopy.
2. Determining the threshold of air and bone conduction with the use of the Interacoustics AD 229E audiometer.
3. Examining otoacoustic emissions with the use of the Scout Sport device.
4. Examining the auditory brainstem response (ABR).
5. Presenting the ultrasound samples – the patients asked to describe subjectively the influence of the sounds on their tinnitus and their perception of the dither (ultrasound noise).
6. Determining the threshold of air conduction in presence of the ultrasound noise.
7. Examining otoacoustic emissions in presence of the ultrasound noise.
8. Examining the ABR in presence of the ultrasound noise.

The above steps can be grouped into three stages:

- Stage 1 (steps 1–4): the aim was to determine the audiological profile of the patients, in order to be aware of specific features of patients' hearing. During this stage, the researchers tried to limit the group of study patients to those with whom the probability of dithering treatment success would be the highest.
- Stage 2 (step 5): treating the patients with ultrasound dither noise of the characteristics described above in order to check the effectiveness of such treatment in tinnitus alleviation.
- Stage 3 (steps 6–8): checking the effect of the ultrasound dither noise treatment on the hearing test results.

The research included six patients. Each of them suffered from tinnitus of different aetiology. Patient No. 1 suffered from unilateral sensorineural hearing loss on the level of 60 dB HL. The patient was unable to detect the presence of ultrasound dither noise and the experienced no detectable effect of the treatment. The remaining patients (No. 2–6) had normal hearing or bilateral sensorineural hearing loss on the level of up to 40 dB HL. Patients 2 and 3 were able to detect ultrasound dither noise but experienced no effect of the treatment on their tinnitus.

Results for patient No. 4 supported the observations described in the literature (Hazell, 1984). In this patient tinnitus was successfully masked in presence of ultrasound dither noise. When the ultrasound sound sample was presented to this patient, it was loud enough to mask the perception of tinnitus. However, the patient stressed that the masker was just as annoying as tinnitus, although the sound differed. It should be emphasized that only one of the 4 presented samples had the masking effect on the patient's tinnitus. The remaining samples were audible but had no effect on the sensation of tinnitus.

The best results were obtained for patients No. 5 and 6, where the dithering effect occurred. Because test results for both of these patients were similar, below quoted are only results of audiometry, otoacoustic emission and ABR tests for patient No. 5. Due to time limitations, in all patients, tests were repeated in presence of ultrasound dither noise only in the ear that was more severely affected or that showed better therapeutic results.

Patient No. 5 suffers from bilateral tinnitus, more severe in the right ear. During sound sample presentation, the patients reacted positively to different types

of ultrasound dither noise. In this patient, both dither noise in the range of 20–80 kHz, and the narrow-band noise with the centre frequency of 26 kHz and 39 kHz led to complete eradication of tinnitus. It should be noted that ultrasound dither noise was not audible to the patient. This effect could be compared to the application of dither in digital transmission channels. When the dither effect was obtained, audiometry was repeated. Air conduction threshold marked by letter "M" in Fig. 3 shows that the results obtained in this test were practically identical to those obtained in the test carried out without the application of dither. The differences were within the measurement error.

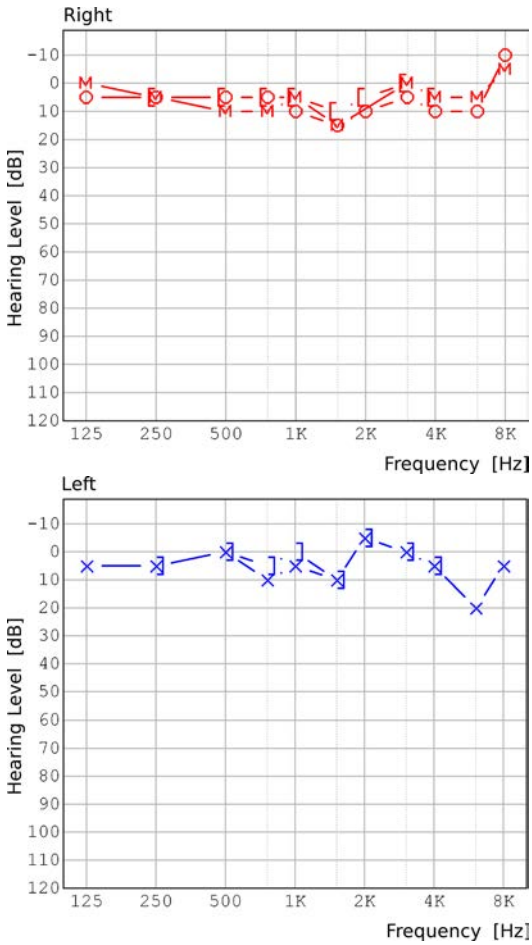

Fig. 3. Pure-tone audiogram of patient No. 5. Symbols: (o, x) – air conduction threshold; ([,]) – bone conduction threshold with masking; (M) – examination of air conduction threshold in presence of ultrasound dither noise.

Similar observations can be made after analyzing the distortion product otoacoustic emission results presented in Fig. 4. The presented DP-Gram shows that dither has practically no effect on the obtained result. The transient-evoked otoacoustic emission (TEOAE) and spontaneous otoacoustic emissions (SPOAE) showed similar results. The latter included noise components of dither, but they were independent of the patient and observable also when the vibrator was placed near the otoacoustic emission probe only.

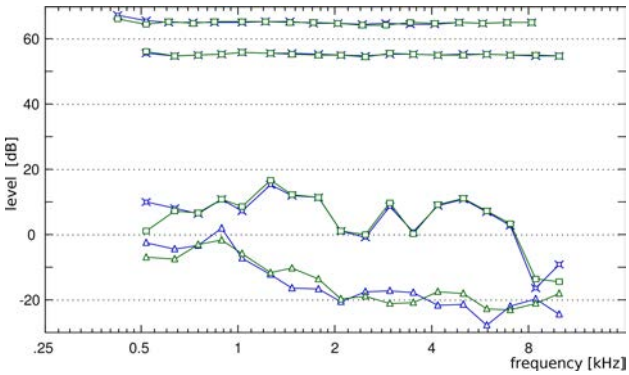

Fig. 4. Distortion product otoacoustic emissions in the right ear of patient No. 5. Symbols: (□) – DP-Gram in presence of ultrasound dither noise, (o) – DP-Gram without ultrasound dither noise.

Obtaining the ABR results in presence of ultrasound dither noise was difficult, as major electromagnetic interference was produced by the bone vibrator, utilized to present ultrasound dither noise. This conclusion was reached because switching the vibrator off, without placing it on the mastoid process, resulted in appearance of significant artifacts in the registered response when no stimulus was provided from the headphones to the ear. To minimize the effect of interference, values of the registered response were averaged. As a result, Fig. 5 shows no change in the hearing threshold determined by ABR and the wave V latency analysis in presence of dither.

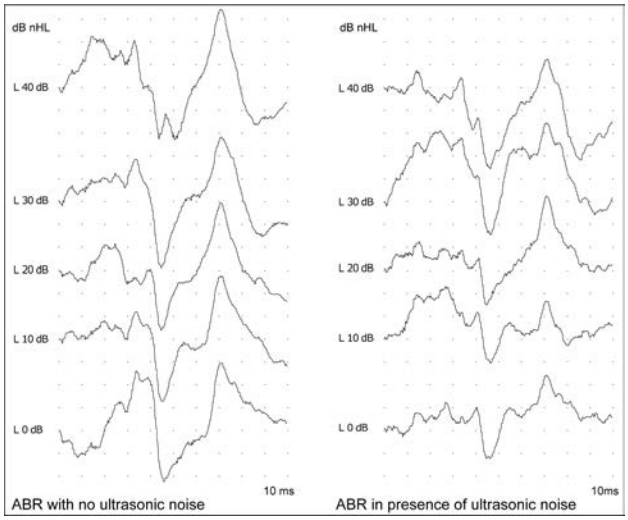

Fig. 5. Examining the auditory brainstem response (ABR) in patient No. 5 with the use of clicks in the right ear.

The observation results of the influence of ultrasound dither noise on tinnitus have been summarized for patients No. 4, 5 and 6 in Table 1.

In the case of patient No. 4, only one sound sample showed tinnitus masking properties. The masking signal, however, was unpleasant for the patient. The patient was able to perceive the remaining samples, yet they had no effect on tinnitus.

Table 1. Effect of the ultrasound noise (dither) on tinnitus, RE – right ear, LE – left ear.

Type of applied dither noise	Patient No. 4	Patient No. 5	Patient No. 6
20–80 kHz	Dither detection with no effect on tinnitus in RE and LE	*Disappearance of tinnitus with no detection of dither in RE*	Dither detection with no effect on tinnitus in RE and LE
30–50 kHz	*Tinnitus masking with the use of dither in RE and LE*	Dither detection with no effect on tinnitus	*Disappearance of tinnitus with no detection of dither in RE*
26 kHz	Dither detection with no effect on tinnitus in RE and LE	*Disappearance of tinnitus with no detection of dither in RE*	Dither detection with no effect on tinnitus in RE and LE
39 kHz	Tinnitus aggravation in RE and LE	*Disappearance of tinnitus with no detection of dither in RE*	Dither detection with no effect on tinnitus in RE and LE
General remarks		The effect of dither significantly lower in LE or no effect	No effect of dither when applying the signal into the LE

In the two remaining patients (No. 5 and 6), tinnitus was eliminated and dither was not audible. It should be emphasized that tinnitus was not experienced and ultrasound dither noise was inaudible only when the dither was applied by bone conduction. When the level of the applied signal was lowered or switched off, tinnitus reappeared immediately. To confirm the effect of the treatment, a double-blind test was performed.

5. Conclusions

According to the authors' observations, ultrasound dithering can be used as one of the methods for the treatment of tinnitus. However, it is not always effective. The obtained effects vary depending on both the presented signal and the side to which it is applied. For some patients, the ultrasound dither noise did not affect tinnitus perception while for the others it masked, aggravated or eradicated this symptom.

For both patients who experienced the dithering effect, the air conduction threshold curves were symmetrical and did not exceed 30 dB HL, with traits of sensorineural hearing loss. Another feature, common for both patients, was the fact that tinnitus appeared as a result of mechanical injury to the head. Nevertheless, as the number of examined patients was limited, it is impossible to draw definite conclusions as to the ultrasound therapy. Moreover, the therapeutic effect was obtained after a short presentation of the stimuli (up to a few minutes). It should though be accounted that the patients reacted positively to this method. Some time later one of them "lost" tinnitus, and the other reported that tinnitus disappeared for approximately 90 minutes after the ultrasound dither noise had been applied. Other patients didn't perceive tinnitus for some minutes or dozen of minutes.

However, the effect of a long-term exposition to ultrasound dither noise requires further research. It is necessary to check whether residual inhibition, that reduces the intensity of tinnitus when dither is no longer applied, can be achieved with this type of ultrasound dither noise.

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