

Tinnitus Suppression with Threshold and Subthreshold Sound Stimuli

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Abstract: In this preliminary report, we present the results from our investigation of 34 tinnitus patients for tinnitus suppression with frequency-specific sound stimuli within the auditory spectrum. Of this number, 22 (64.7%) experienced suppression, 5 (14.7%) had partial suppression, and 7 (20.6%) were nonresponders. Suppression of peripheral tinnitus may result when mechanosensitive outer hair cells are recruited by sound stimuli that can remain at subthreshold level. The suppression mechanism is possibly explained by the electromodel of the auditory system. This physiological model could be the basis of tinnitus suppression therapy in which a low-intensity, frequency-specific and tinnitus-suppressing sound stimulus is introduced instead of a wide-band masking noise.

Key Words: electromodel of auditory system; subthreshold sound stimuli; tinnitus suppression therapy

Sound treatment for subjective tinnitus remains a challenge to otolaryngologists. Audible masking noise has become a widely accepted form of treatment [1,2]. Its usefulness has been demonstrated through masking devices and hearing aids. A masking device, however, is not always accepted by a tinnitus patient, as it introduces yet another noise, and listening to it can be unpleasant. An improvement has been achieved with the concept of tinnitus retraining therapy in which masking noises are maintained at a reduced level [3,4]. Here, audible noises that do not mask tinnitus are an intended and integrated part of the treatment strategy. The recent description of UltraQuiet therapy promises a further improvement with the application of sounds outside the audible spectrum [5]. The aim of this technique is the production of long-term residual inhibition, possibly as a result of plastic changes in the brain.

In this report, we present the preliminary results of responses to inaudible sounds within the normal audi-

tory spectrum. The basis of this approach was the electromodel of the auditory system [6]. Following the electromodel of the auditory system, a mechanism exists that is driven by mechanosensitive outer hair cells (OHCs), that operates close to and below threshold level, and prevents tinnitus by suppressing inner hair cells (IHCs). In tinnitus sufferers, this natural and inherent suppression mechanism is suspected to be faulty. Some argue that remaining functional OHCs could be recruited with sound stimuli and that these sound stimuli would possibly be effective just below threshold level, as this is the level at which OHCs may reduce IHC activity. Understanding these physiological events at the receptor level should then translate into better management of tinnitus in affected patients, as we propose in tinnitus suppression therapy.

SUBJECTS AND METHOD

Over a period of 6 months, we conducted a study of 34 consecutively selected patients who experienced tinnitus, attended our Tinnitus Research Clinic, and agreed to participate in this study. Patients with a conductive hearing loss, suspected retrocochlear pathology, and pulsatile tinnitus did not take part in this study. We also excluded patients with a severe hearing loss. These patients were suspected to have insufficient

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functional OHCs, which were regarded as essential for suppression.

The 34 subjects included 20 male and 14 female participants. Their average age was 55.4 ± 11 years. Unilateral tinnitus was present in 22 patients and bilateral tinnitus in 12. The character of the tinnitus varied from a ring to a hiss, whistle, buzz, and cicada-like sounds. One patient described his tinnitus as a click. Of the 22 unilateral tinnitus patients, 14 were affected in the left ear. Of the 12 bilateral tinnitus patients, 7 thought the sound in the right ear was worse, and 5 thought the left ear noise was worse. Spontaneous otoacoustic emission was absent in all patients.

The audiological assessment consisted of a pure-tone audiogram, tympanometry, speech audiometry, and assessment of spontaneous otoacoustic emissions (Madsen Celesta, Madsen Electronics, Denmark). Brainstem au-

diometry was performed to exclude retrocochlear pathology (Biologic Navigator, Chicago, IL). Hearing was considered normal when the hearing level in all frequencies tested (250 Hz–8 kHz) remained below 20 dB. A hearing loss was regarded as mild when the average hearing level at three neighboring frequencies was between 20 and 40 dB and as moderate when the average was between 40 and 65 dB.

Matching with a pure tone was used to assess the frequency and the intensity of tinnitus. The level of tinnitus masking also was determined with a pure tone at the tinnitus frequency.

For suppression, the worst ear was tested when bilateral tinnitus was present. A pulsed pure tone was applied to the tinnitus-affected ear. The pulse had a frequency of 2 Hz and a rise time and falloff time of 125 msec, with a sound-free interval of 125 msec. The

Table 1. Results of Introduction of Pulsed Suppression Tone

Patient	Gender	Age (yr)	Laterality	Side Affected	Hearing Level	Tinnitus Freq.	Suppression Freq.	Inhibition (sec)	Level of Response
1	F	80	U	L	Mo	487	634	60	RE
2	F	59	B	R	Mi	746	801	—	P
3	M	58	B	L	Mi	923	4,641	—	P
4	M	66	U	L	Mo	987	2,099	—	RE
5	F	48	U	R	Mo	1,070	—	—	NO
6	F	49	U	R	Mo	1,106	—	—	NO
7	M	53	B	L	Mi	1,500	1,706	—	RE
8	F	64	B	L	Mi	1,733	3,710	—	RE
9	M	49	U	L	Mi	1,750	—	—	NO
10	F	49	U	L	Mi	1,986	2,411	—	RE
11	F	51	U	R	Mi	2,000	5,030	—	RE
12	F	57	U	R	Mi	2,000	5,592	—	RE
13	F	58	U	R	Mo	2,049	2,112	30	P
14	F	39	U	L	Mi	2,222	2,245	—	P
15	M	79	B	L	Mo	2,250	2,753	—	RE
16	M	57	B	R	Mi	2,314	2,349	—	RE
17	F	75	U	R	Mi	2,750	2,998	—	RE
18	M	50	B	R	Mi	2,750	—	—	NO
19	M	54	U	R	Mi	2,750	3,944	—	P
20	F	62	U	L	N	3,000	5,081	—	RE
21	F	49	U	R	Mi	3,139	7,568	—	RE
22	F	53	U	L	N	3,500	4,014	—	RE
23	M	66	B	L	Mi	3,944	7,454	—	RE
24	M	41	U	L	Mi	4,407	7,516	—	RE
25	M	58	B	R	Mi	5,346	5,682	27	RE
26	M	27	U	L	N	5,426	8,224	—	RE
27	M	61	B	R	Mi	5,500	6,372	—	RE
28	M	61	B	R	Mo	5,511	8,746	—	RE
29	M	43	U	L	N	7,710	9,000	—	RE
30	M	68	U	L	Mi	7,812	8,700	43	RE
31	M	47	U	L	Mi	8,000	—	—	NO
32	M	48	B	R	Mo	9,063	—	—	NO
33	M	52	U	L	N	9,109	—	—	NO
34	M	53	U	L	Mo	10,000	8,658	—	RE

B = bilateral; L = left; Mi = mild hearing loss; Mo = moderate hearing loss; NO = nonresponder; P = partial responder; R = right; RE = responder; U = unilateral. Note: In no patient did we find spontaneous otoacoustic emissions.

introduction of a pulsed suppression tone resulted from preliminary tests that have shown less effective suppression when a continuous tone was applied. This latter finding may have been due to adaptation. The stimulus was limited to and did not exceed the 90-dB hearing level. In four patients who responded well to suppression, the contralateral ear was stimulated with a low-level (5- to 10-dB sound level [SL]) narrow-band noise (Table 1). Three patients had bilateral tinnitus.

The initial step was to introduce a pulsed pure tone at a frequency the same as that of the tinnitus frequency until complete masking was achieved. In consecutive small steps, the intensity of the stimulus then was reduced until tinnitus again became audible. This was followed by an increase in the stimulus frequency until tinnitus once more became inaudible. A gradual increase in the stimulus frequency and a gradual decrease in the stimulus intensity were continued until a level was reached at which neither the applied stimulus nor tinnitus could be heard.

RESULTS

The results are shown in Table 1. Of our 34 tinnitus patients, 22 (64.7%) experienced suppression: That is, tinnitus was not audible, and the introduced sound stimulus simultaneously was not heard. While the sound stimulus remained inaudible, five patients (14.7%) experienced reduction in the intensity of their tinnitus. In seven (20.6%) tinnitus could not be suppressed, and even masking was unable to influence the intensity of the tinnitus. Four selected patients (see Table 1) had experienced suppression of their tinnitus with contralateral low-level narrow-band noise stimulation.

The majority of patients experienced return of their tinnitus as soon as the suppressing stimulus was switched off. However, residual inhibition was experienced by some of the patients. It was variable and lasted from 27 to 60 sec (see Table 1). The suppression frequency was always found to be higher than the tinnitus frequency, but no clear relationship emerged regarding the frequency difference. In some cases, the suppression frequency was very close to the tinnitus frequency, and in others it was widely separated. Also, the suppression frequency never was found at the maximum hearing loss frequency.

The majority of the audiograms were consistent with a sensorineural hearing loss mainly affecting the high frequencies. The hearing losses were classified as mild ($n = 20$) to moderate ($n = 9$). Five patients had normal hearing in the frequency range of 250 Hz to 8 kHz. Figure 1 is a representative audiogram in which the tinnitus frequency, suppression frequency, and suppression

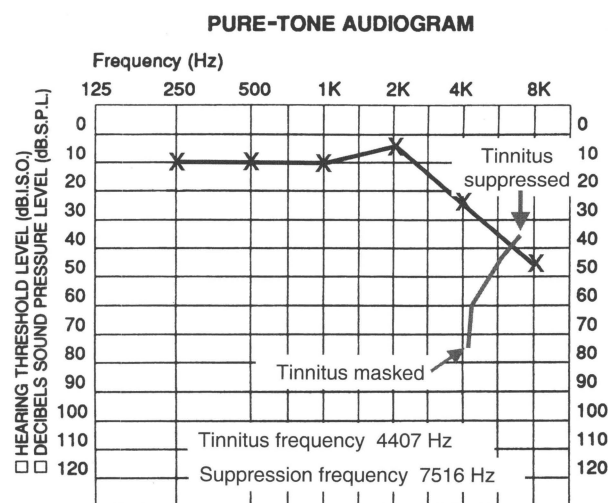


Figure 1. Representative audiogram of a tinnitus patient (patient 24). The suppression frequency is basal to the tinnitus frequency. Suppression was effective below threshold level. The tinnitus frequency and suppression frequency did not coincide with the frequency of maximum hearing loss.

level are indicated. The tinnitus frequency invariably was found apical to the maximum hearing loss.

DISCUSSION

An important relationship has long been suspected to exist between IHC and OHC function. It also is possible that a disturbance of this relationship could lead to the phenomenon of tinnitus. A number of studies have highlighted the importance of this inter-hair cell relationship. Penner [7] suggested that tinnitus results from a local increase in the firing rate due to loss of normal suppression. Penner could demonstrate, in subjects with tinnitus, that two-tone forward-masking patterns are decidedly different in the normal region and the tinnitus region. However, she did not speculate regarding the origin of this suppression, although the subjects investigated must have had an OHC loss due to noise exposure.

Rajan [8,9] believed in a protective function of the crossed olivocochlear bundle, which drives OHCs. He found that the temporary threshold shift, which is observed after exposure to loud sounds, is significantly reduced when the crossed olivocochlear bundle is simultaneously stimulated.

Le Page [10] believed in an excitatory drift in the operating point of IHCs controlled by the OHCs. Degradation of OHC activity leads to a phantom acoustic input to the central nervous system (i.e., tinnitus). Tinnitus in this model is due to reduction of the population of OHCs, these being unable to generate suppression of

IHCs because of lack of suppressive displacement of the tectorial membrane.

Patuzzi [11] emphasized the role of the endocochlear potential in sensitizing IHCs and producing excessive firing of the auditory nerve, leading to tinnitus. The role of the OHCs is to regulate the endocochlear potential, which may be achieved through a current shunt by OHCs. Failure of this OHC regulation can lead to tinnitus.

The electromodel of the auditory system also regards the inter-hair cell relationship as an important function of the auditory system. As a principle of this model, IHCs that may detect electrical potentials from the tectorial membrane are primarily regarded as electroreceptors, and OHCs are primarily regarded as mechanoreceptors [6]. Gain or sensitivity of IHCs is regulated by mechanosensitive OHCs that produce a positive summing potential and determine the magnitude of IHC suppression. Diminished OHC function may lead to diminished IHC suppression close to the threshold level. Loss of OHC function thus can result in an auditory neuron discharge and tinnitus.

Sensitivity regulation of IHCs, therefore, seems to be an important and inherent mechanism in the cochlea that operates close to the threshold level and regulates gain of IHCs. A disturbance of this mechanism can result in tinnitus. The hypothesis presented by the electromodel of the auditory system was an attractive concept that inspired the design of this study, as it could lead to a simple clinical application. The results of this study support this hypothesis, but how could its mechanism be explained?

Pierson and Møller [12] found at one recording location two sources of cochlear microphonics that were 180 degrees out of phase. One maximal amplitude of microphonics is created more basally and originates from OHCs. The other maximal amplitude of microphonics is created more apically and originates from IHCs. The maximal amplitude of the traveling wave coincides with the maximum of OHC microphonics. However, this is not the location of the maximum of IHC microphonics at the same frequency; these are more apical. In other words, the perception of a pure tone, say 4 kHz, is not at a location that corresponds to the maximal amplitude of the traveling wave.

Offutt [6] reinterpreted these data from Pierson and Møller. His results are summarized in a diagram (Fig. 2) that shows the tonotopic location of cochlear events at threshold level: The response of IHCs occurs apical to where suppression is produced by OHCs. Tinnitus is found at the location of IHCs that lack suppression, and the frequency of tinnitus is probably the same as the tuned frequency of the IHCs. This is apical to the OHCs tuned to the same frequency.

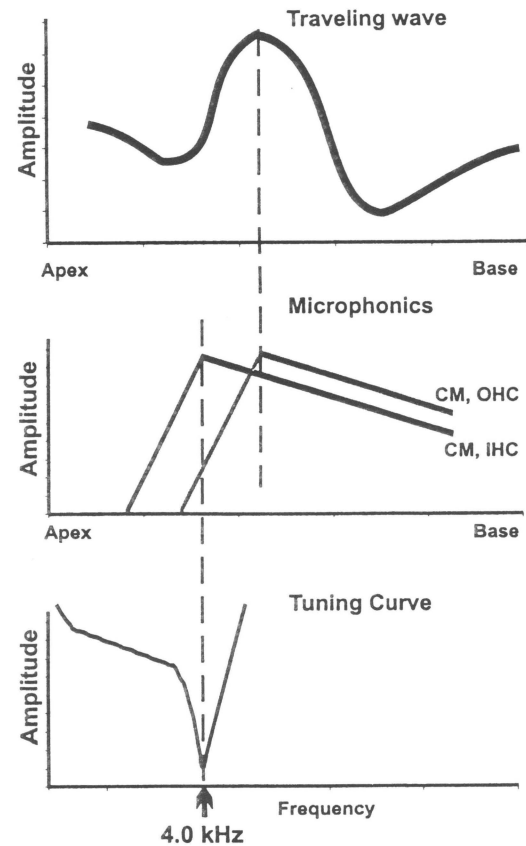


Figure 2. Tonotopic location of cochlear event. A pure tone presented at a frequency of 4 kHz is probably detected at a location apical to the maximal amplitude of the traveling wave. The maximal amplitude of the traveling wave coincides with the maximal amplitude of the cochlear microphonics of the outer hair cells (*CM, OHC*) but not with the maximal amplitude of cochlear microphonics of the inner hair cells (*CM, IHC*). Tinnitus can occur when inner hair cells lack suppression. (Redrawn with permission of the author from G Offutt, *The Electromodel of the Auditory System*. Shepherdstown, PA: Go-Lo Press, 1984.)

In our study, a frequency higher than that of the apparent tinnitus was used to administer the suppression stimulus. On the basis of the electromodel, this relationship was expected to maximize stimulation of any residual OHCs. Possibly the residual OHCs were insufficient to achieve complete suppression in the five patients (14.7%) who had only partial tinnitus reduction. The seven (20.6%) who did not respond to this treatment may have had tinnitus of a central origin.

We have used a pulsed pure tone for suppression. This selection was made on the basis of preliminary tests that have shown less effective suppression with a continuous tone. We speculated that the latter situation may have been attributable to adaptation. A similar outcome was observed when a narrow-band noise was

applied to the tinnitus-affected ear, and we were unable to suppress below threshold level. This may be explained by the phenomenon of the critical bandwidth, whereby a narrow-band noise is perceived as being louder because it has a much lower threshold than does a pure tone in patients with a sensorineural hearing loss [13].

No consistent interval was found between the tinnitus frequency and the suppression frequency. This lack of correlation may have been due to various configurations of the hearing loss or might have been due to a variability of available functional OHCs at different locations. It also is possible that the variability was due to octave confusion, which was present in a number of our patients. Despite the lack of correlation, we observed a frequency dependency for tinnitus suppression. The suppression frequency invariably was found basal to the tinnitus frequency. It also was not found at the point of maximal hearing loss, as might have been expected.

We excluded for this study patients with a severe hearing loss. The rationale was to include only patients who had a chance of responding. If a patient did not have sufficient functional OHCs that could be recruited, no positive effect would be obtained from stimulation. We also were looking for stimulation at safe intensity levels. In all our patients who responded to suppression, the stimulus levels were within the normal speech range and well below a 65-dB hearing level.

The absence of spontaneous otoacoustic emissions (SOAEs) confirms the presence of dysfunctional OHCs. That is not surprising in a sensorineural hearing loss. However, SOAEs were also absent in five tinnitus patients with normal hearing, four of whom were older than 50 years; the absence of SOAEs in this age group can be expected [14]. Nevertheless, this absence must have a bearing on the functionality of OHCs, as not a single tinnitus patient in our study demonstrated SOAEs.

In four patients, contralateral suppression—but with stimuli beyond threshold and starting stimulation at the tinnitus frequency with a narrow-band noise—was tried and was successful. This is not unexpected with audible noise bands and pure tones [15]. One explanation is that contralateral stimulation activates the crossed olivocochlear bundle, which drives OHCs, increasing their microphonics and positive summing potential: This would exert some suppression effect on IHCs.

The presented results demonstrate a suppression mechanism for peripheral tinnitus using subthreshold stimuli. Application of this technique is restricted, and it might not be appropriate when changes in the central auditory pathway have developed in tinnitus patients. Therefore, a reasonable step would be to determine whether tinnitus suppression plays a complementary role in an already existing treatment modality that addresses the central component of tinnitus. Such a treat-

ment modality is tinnitus retraining therapy, which has at its core the detachment of the emotional response from tinnitus. This is achieved through a process of habituation by focusing on the so-called detection level of tinnitus and the so-called level between detection and reaction [3,4].

The detection level is addressed with the introduction of an audible wide-band masking noise, chosen in the belief that masking tinnitus is frequency-independent. The findings in our study contradict this, however. Our findings indicate that a frequency-specific sound should be used, one that is different from the tinnitus frequency. This is indeed supported by findings that suggest the initiation of cortical reorganization with a sound stimulus that differs from the tinnitus frequency and is driven into the enlarged cortical representation of the tinnitus frequency. The aim is to reduce tinnitus representation at the cortical level [16].

Taking into consideration that tinnitus suppression is not frequency-independent, we can now change the detection level of tinnitus by the introduction of a low-intensity, audible, frequency-specific, and tinnitus-suppressing signal instead of a wide-band masking noise. This would have the advantage of demonstrating immediate tinnitus reduction and would facilitate habituation.

Tinnitus suppression therapy is an approach based on a physiological model at the tinnitus detection level and a neurophysiological model at the level between detection and reaction. It promises greater efficiency in the management of tinnitus.

SUMMARY

OHCs suppress IHCs close to threshold level. Loss of functional OHCs can lead to peripheral tinnitus. In some patients, functional OHCs can be mobilized with a frequency-specific and tinnitus-dependent sound stimulus just below threshold level. The mechanism may be explained by the electromodel of the auditory system. This physiological model could be the basis of tinnitus suppression therapy, which promises to become a valuable addition to current treatment options.

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