CLINICS

CLINICAL SCIENCE

Cortical activity in tinnitus patients and its modification by phonostimulation

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OBJECTIVE: The goal of this study was to observe spontaneous cortical activity and cortical activity modulated by tinnitus-matched sound in tinnitus patients and healthy subjects with no otoneurologic symptoms.

METHOD: Data were prospectively collected from 50 tinnitus patients and 25 healthy subjects. Cortical activity was recorded in all subjects with eyes closed and open and during photostimulation, hyperventilation and acoustic stimulation using 19-channel quantitative electroencephalography. The sound applied in the tinnitus patients was individually matched with the ability to mask or equal the tinnitus. The maximal and mean amplitude of the delta, theta, alpha and beta waves and the type and amount of the pathologic EEG patterns were noted during each recording. Differences in cortical localization and the influence of sound stimuli on spontaneous cortical activity were evaluated between the groups.

RESULTS: The tinnitus group exhibited decreased delta activity and increased alpha and beta activity. Hyperventilation increased the intensity of the differences. The tinnitus patients had more sharp-slow waves and increased slow wave amplitude. Sound stimuli modified the EEG recordings; the delta and beta wave amplitudes were increased, whereas the alpha-1 wave amplitude was decreased. Acoustic stimulation only slightly affected the temporal region.

CONCLUSION: Cortical activity in the tinnitus patients clearly differed from that in healthy subjects, i.e., tinnitus is not a ''phantom'' sign. The changes in cortical activity included decreased delta wave amplitudes, increased alpha-1, beta-1 and beta-h wave amplitudes and pathologic patterns. Cortical activity modifications occurred predominantly in the temporal region. Acoustic stimulation affected spontaneous cortical activity only in tinnitus patients, and although the applied sound was individually matched, the pathologic changes were only slightly improved.

KEYWORDS: Tinnitus; Cortical Activity; Neurotology.

Pawlak-Osińska K, Kaźmierczak W, Kaźmierczak H, Wierzchowska M, Matuszewska I. Cortical activity in tinnitus patients and its modification by phonostimulation. Clinics. 2013;68(4):511-515.

Received for publication on September 19, 2012; **First review completed on** October 20, 2012; **Accepted for publication on** December 23, 2012

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INTRODUCTION

Tinnitus can originate anywhere along the audiologic pathway. In some cases, the source has remained unknown, which has led to the hypothesis that tinnitus is a phantom perception that is analogous to phantom pain (1,2). The role of cortical control is interesting from both scientific and therapeutic points of view. Changes in cortical activity can both elicit and suppress tinnitus. Thalamocortical dysrhythmia due to increased theta and gamma activity could be responsible for tinnitus (3). When coexisting with hearing loss, tinnitus might be the result of neuronal hyperactivity provoked by reduced peripheral input (4). This hyperexcitability is suspected to be located in parts of the auditory cortex that represent intact hearing frequencies (5). The left temporal gyrus is overactivated, independent of tinnitus laterality (6). Suppression of tinnitus was positively correlated with activation of the left and right temporal gyrus and the parahippocampal-hippocampal interface (6). In addition, increased spontaneous alpha power in the auditory cortex occurred in suppressed states. (7). Sound stimuli are believed to modulate the centrally generated sensations that result in tinnitus inhibition (2). Where tinnitus and sound interact, however, is not known. The effects of sound might be psychogenic, and long-term sound application could be harmful.

No potential conflict of interest was reported.

DOI: 10.6061/clinics/2013(04)12

Table 1 - Global differences in EEGs between the healthy subjects and tinnitus patients.

Condition	Number of tests performed	Number of significant tests	Significance
Basic recording	228	6	0.9734
Eyes open	228	3	0.9969
Acoustic stimulation	228	10	0.5910
Photostimulation	228	7	0.8870
Hyperventilation	**228**	**34**	**0.0000**
Pathologic patterns	**152**	**26**	**0.0000**

Number of tests performed: measurements of mean and maximal amplitudes of cortical activity gathered from each EEG electrode (together = 228 results to compare); number of significant tests: number of tests showing a significant difference ($p<0.05$) between the healthy and tinnitus groups based on Student's t-test; significance: results of Student's t-test for the whole analyzed condition. Significant differences between groups with regard to hyperventilation are explained in the Discussion (line 11).

In the present study, we compared spontaneous and sound-modulated electroencephalography (EEG) recordings between tinnitus patients and healthy controls.

MATERIALS AND METHODS

The tinnitus patient group consisted of 50 individuals (24 women and 26 men) aged 20 to 63 (mean: 42.5) years with tinnitus who were not selected according to any tinnitus factors. The subjects' tinnitus was subjective, located in the outside ear and the VIII-th nerve, according to the exclusion criteria. The exclusion criteria included suffer from external, middle or internal ear diseases or malfunction of the VIII-th cranial nerve; additionally, subjects with focal signs of central nervous system disturbances, head and neck injuries and epilepsy were excluded. Additional diagnoses accompanying tinnitus were as follows: cervical spondylosis (35 patients), hyperlipidemia (21 patients), arterial hypertension (16 patients), atheromatosis (11 patients), hormonal disturbances (thyroid = 9 patients; sexual = 9 patients), hyperglycemia (7 patients), arrhythmia (5 patients), allergy (4 patients), depression (3 patients), occupational exposure to noise (2 patients), nicotinism and coronary disease (1 patient each). The features and outcomes of tinnitus were as follows:

a) localization: right ear = 11 cases; left ear = 15 cases; bilateral = 24 cases;

b) nuisance (minimal = 1, serious daily trouble = 10): 1-4 = 11 cases; 5-8 = 24 cases; 9-10 = 15 cases;

c) effect of masking: present = 6 cases; lacking = 44 cases;

d) time result: permanent tinnitus = 35 cases; interrupted tinnitus = 15 cases; and

e) psychological effect: disturbed concentration = 32 cases; disturbed sleeping = 7 cases; nervousness = 11 cases.

The tonal audiometry results from tinnitus patients revealed the mean hearing losses as follows (with standard deviation (SD) in dB): 500 Hz = 33 (18.1) right ear, 30 (16.8) left ear; 1000 Hz = 28 (21.6) right ear, 25 (22.1) left ear; 2000 Hz = 30 (23.9) right ear, 20 (24.6) left ear; and 4000 Hz = 38 (25.3) right ear, 25 (27.3) left ear. None of the patients had received previous therapy for tinnitus.

In every patient, monopolar 19-channel EEG recordings (Neuron-Spectrum 4/P, Neurosoft Company) were obtained under the following conditions: with eyes closed and open and with hyperventilation, photostimulation and acoustic stimulation. The EEGs were recorded and digitized with sampling at 500 Hz, with a high-pass filter of 0.50 Hz and low-pass filter of 35.0 Hz. In every patient, tinnitus was measured before EEG was obtained to estimate the pitch and intensity. In the entire group, the tinnitus intensity and pitch ranged from 10 to 85 dB (mean 43.8 dB, SD 19.91) and from 125 to 8000 Hz (mean 2052.5 Hz, SD 1697.1), respectively. A sound that matched the frequency and intensity of the tinnitus and that varied for each individual patient was presented through air conduction, with earphones applied to the ear that experienced the tinnitus or binaurally when the tinnitus was located in the head. Acoustic stimulation was performed in a silent room for 2 minutes. Hyperventilation was induced by taking 40 deep breaths per minute for 3 minutes. Photostimulation was applied using stroboscopic flashes lasting 7 seconds, with a 5-second pause, at frequencies in the following sequence: 1, 3, 7, 10, 15, 20, 25, 30 and 50 Hz.

The control group consisted of 25 healthy subjects (12 women, 13 men) aged 18-61 (mean: 40.4) years old with no otoneurologic symptoms. Hearing based on tonal audiometry was normal (not more than a 15-dB loss) in both ears. EEG recordings were obtained in the same manner as in the tinnitus group; acoustic stimulation was the same in every case, and a 1000-Hz, 40-dB stimulus was presented.

Ethics

The procedures were conducted in accordance with the ethical standards of the responsible committee on human

Table 2 - Basic recording with eyes closed: differences in cortical activity between the healthy subjects and tinnitus patients.

	Tinnitus group		Control Group		Significance of a Levene's test	Variance in 2 groups	statistics t	Df	Significance of t test (2-tail)	95%	CI
Variable	Mean	SD	Mean	SD						min	max
Aav (D) for F3Fpz	**13.8**	**5.7**	**17.8**	**11.8**	**0.051**	**Equal**	**-2.00**	**73**	**0.050**	**-8.0**	**0.0**
Aav (Bl) for F4Fpz	**4.16**	**1.30**	**3.56**	**1.04**	**0.192**	**Equal**	**2.01**	**73**	**0.049**	**0.00**	**1.20**
Aav (D) for T3Fpz	**15.74**	**6.07**	**18.80**	**5.54**	**0.986**	**Equal**	**-2.12**	**73**	**0.038**	**-5.94**	**-0.18**
Aav (Bl) for T4Fpz	**6.44**	**2.37**	**5.20**	**1.55**	**0.028**	**Different**	**2.71**	**67.46**	**0.008**	**0.33**	**2.15**
Amax (Bh) for T4Fpz	**23.3**	**11.5**	**19.3**	**5.1**	**0.009**	**Different**	**2.08**	**72.41**	**0.041**	**0.2**	**7.8**
Aav (Al) for T6Fpz	**18.2**	**8.4**	**13.7**	**9.0**	**0.815**	**Equal**	**2.16**	**73**	**0.034**	**0.3**	**8.7**

Abbreviations: Aav: average amplitude; Amax: maximal amplitude; 95% CI: 95% confidence interval; SD: standard deviation. Rhythms: A = alpha, B = beta, D = delta. Cortical regions: T = temporal, P = parietal, F = frontal, O = occipital, C = central.

Table 3 - Differences in cortical activity during acoustic stimulation between the healthy subjects and tinnitus patients.

	Tinnitus Group		Control Group		Significance of Levene's test	Variance in 2 groups	statistics t	df	Significance of t test (2-tail)	95%	CI
Variable	Mean	SD	Mean	SD						min	max
Amax for O1Fpz	115.9	44.0	93.7	34.1	0.158	Equal	2.21	73	0.030	2.2	42.2
Am (Al) for O1Fpz	74.7	33.8	56.3	32.5	0.624	Equal	2.26	73	0.027	2.2	34.8
Aav (Al) for O1Fpz	24.7	13.0	18.4	12.0	0.365	Equal	2.00	73	0.049	0.0	12.4
Am (Al) for F8Fpz	26.7	12.2	18.8	12.7	0.529	Equal	2.59	73	0.011	1.8	13.9
Aav (Bl) for F8Fpz	4.54	1.64	3.76	0.97	0.053	Equal	2.19	73	0.032	0.07	1.49
Amax for T6Fpz	103.8	44.7	79.7	39.8	0.421	Equal	2.28	73	0.026	3.0	45.1
Average for T6Fpz	22.0	8.2	17.6	9.1	0.806	Equal	2.12	73	0.037	0.3	8.6
Am (Al) for T6Fpz	57.9	24.1	41.1	27.4	0.813	Equal	2.72	73	0.008	4.5	29.1
Aav (Al) for T6Fpz	18.6	8.9	13.6	9.7	0.909	Equal	2.24	73	0.028	0.6	9.5
Am (Bl) for T6Fpz	25.8	9.8	20.9	9.7	0.981	Equal	2.04	73	0.045	0.1	9.6

Abbreviations: Aav: average amplitude; Amax: maximal amplitude; 95% CI: 95% confidence interval; SD: standard deviation. Rhythms: A = alpha, B = beta, D = delta. Cortical regions: T = temporal, P = parietal, F = frontal, O = occipital, C = central.

experimentation (institutional or regional) and with the Helsinki Declaration of 1975, which was revised in 1983.

RESULTS

Global differences were observed in the EEG recordings between the healthy subjects and tinnitus patients (Table 1). The responses to hyperventilation were significantly different. Therefore, we analyzed the type and localization of the significant changes. Spontaneous cortical activity with eyes closed revealed differences predominantly in the mean amplitude of the beta and delta waves between the groups. In the tinnitus patients, the power of the delta activity was decreased, whereas the power of the beta activity was increased (Table 2). Changes in the cortical activity under both the eyes-open and eyes-closed conditions were located strictly in the temporal regions. Tinnitus patients under acoustic stimulation showed amplitude changes in both the alpha and beta waves in the right temporal and frontal regions and the left occipital cortex (Table 3). Acoustic stimulation was followed by a decline in the beta power in the parietal region and an increase in the alpha power in the occipital and temporal regions. The mean and maximal amplitudes of the beta and alpha waves changed, and there were no regions in which the cortical activity remained the same (Table 4). Pathologic patterns were observed in the EEG recordings from tinnitus patients. Both the number and amplitude of the patterns described above were greater in the tinnitus patients than the healthy subjects (Tables 1 and 5).

Table 4 - Type and localization of significant differences during acoustic stimulation between the healthy subjects and tinnitus patients.

Variable	Regions
Aav Bh	Fp2Fpz, F4Fpz, F7Fpz, T6Fpz
Amax Bh	T6Fpz
AavB1	Fp2Fpz, F4Fpz, C4Fpz, F7Fpz, T6Fpz,
AmaxB1	F8Fpz, T6Fpz
AavA1	F4Fpz, P3Fpz, O1Fpz, O2Fpz, F7Fpz, F8Fpz, T3Fpz, F4Fpz, T5Fpz, T6Fpz
AmaxA1	P3Fpz, O1Fpz, O2Fpz, F8Fpz, T5Fpz, T6Fpz

Abbreviations: Aav: average amplitude; Amax: maximal amplitude; 95% CI: 95% confidence interval; SD: standard deviation. Rhythms: A = alpha, B = beta, D = delta. Cortical regions: T = temporal, P = parietal, F = frontal, O = occipital, C = central.

When acoustic stimulation matched the pitch and intensity of the tinnitus in tinnitus patients, the total maximal power of the cortical activity increased in the frontal, central, parietal and temporal regions but only bilaterally in the central cortex (Table 6). Changes were detected in the mean amplitude of beta-1, beta-h, alpha-1 and delta waves and in the maximal amplitude of delta waves (Table 6).

DISCUSSION

EEG activity differed significantly between the healthy subjects and tinnitus patients. Changes in cortical activity were most frequently observed in the bilateral temporal lobes. Because these areas are involved in normal hearing, the pathology in tinnitus cases was suspected to be localized only in this region. This finding supports the hypothesis that tinnitus is not a ''phantom'' phenomenon but rather results from inappropriate cortical auditory perception, usually hyperactivity or activity, without any auditory input (8,9). The migration of tinnitus cortical generators over time is possibly represented by changes in activity in the auditory cortex, motor area, insula and prefrontal lobes (10).

The changes in cortical activity in the tinnitus patients included decreased delta wave amplitudes and increased beta-1, beta-h, and alpha-1 wave amplitudes. According to Vanneste (10), high-frequency waves are associated with

Table 5 - Significant differences in pathological EEG patterns and their localization in the tinnitus patients compared with the healthy subjects.

Variable	Regions
A peak	Fp1Fpz, P4Fpz, T4Fpz, T5Fpz,
Amount sharp w-slow w	Fp2Fpz, CzFpz, P3Fpz, P4Fpz, O1Fpz, T6Fpz
A slow wave	C3Fpz, CzFpz, P3Fpz, O1Fpz, F8Fpz, T3Fpz, T4Tpz, T5Fpz
A sharp w-slow w	C3Fpz, C4Fpz, P3Fpz, T6Fpz
Amount peak-slow w	T3Fpz, T6Fpz
Amount slow w	T6Fpz
A peak-slow w	T6Fpz
Legend: A: amplitude; w: wave (s)	

Abbreviations: Aav: average amplitude; Amax: maximal amplitude; 95% CI: 95% confidence interval; SD: standard deviation. Rhythms: A = alpha, B = beta, D = delta. Cortical regions: T = temporal, P = parietal, F = frontal, O = occipital, C = central. W = wave.

Table 6 - Significant differences between EEG recordings with eyes closed and EEG recordings during phonostimulation in the tinnitus patients.

		Eyes closed		Phonostimul.		Significance of Levene's test	Variance in 2 groups	Statistics t	Df	Significance of t test (2 tail)	95%	CI
Group	Variable	Mean	SD	Mean	SD						min	max
Tinnitus group	Amax for Fp1Fpz	47.6	20.7	68.0	57.5	0.002	Different	-2.37	61.51	0.021	-37.7	-3.2
	Amax for FzFpz	5.1	12.3	17.3	27.2	0.000	Different	-2.89	68.33	0.005	-20.6	-3.8
	Amax for C3Fpz	1.54	3.74	3.42	4.36	0.001	Different	-2.31	95.82	0.023	-3.49	-0.27
	Amax for C4Fpz	6.0	9.6	15.1	19.8	0.026	Different	-2.92	70.93	0.005	-15.3	-2.9
	Amax for CzFpz	2.24	3.48	4.16	3.87	0.047	Different	-2.61	96.90	0.011	-3.38	-0.46
	Amax for PzFpz	15.1	7.4	21.5	15.6	0.004	Different	-2.63	69.98	0.010	-11.3	-1.6
	Amax for T6Fpz	7.5	10.1	13.3	14.2	0.053	Equal	-2.36	98	0.020	-10.7	-0.9
	Average for Fp2Fpz	11.26	4.12	13.86	6.99	0.063	Equal	-2.27	98	0.026	-4.88	-0.32
	Amax (D) for P3Fpz	16.9	7.5	21.0	11.8	0.156	Equal	-2.08	98	0.040	-8.0	-0.2
	Amax (D) for T6Fpz	11.38	4.25	13.52	6.26	0.024	Different	-2.00	86.22	0.049	-4.27	-0.01
	Aav (D) for Fp2Fpz	12.94	5.31	15.84	8.19	0.039	Different	-2.10	84.02	0,039	-5.64	-0.16
	Aav (Al) for F3Fpz	18.12	6.22	15.96	3.65	0.009	Different	2.12	79.17	0.037	0.13	4.19
	Aav (Bl) for F4Fpz	21.9	10.4	26.7	12.2	0.648	Equal	-2.12	98	0.036	-9.3	-0.3
	Aav (Bh) for C3Fpz	21.7	6.4	25.3	9.2	0.245	Equal	-2.24	98	0.027	-6.7	-0.4
	Aav (Bh) for O1Fpz	30.6	18.8	39.7	19.2	0.737	Equal	-2.38	98	0.019	-16.6	-1.5

Abbreviations: Aav: average amplitude; Amax: maximal amplitude; 95% CI: 95% confidence interval; SD: standard deviation. Rhythms: A = alpha, B = beta, D = delta. Cortical regions: T = temporal, P = parietal, F = frontal, O = occipital, C = central.

tinnitus volume. Cortical changes were noted predominantly during hyperventilation, suggesting that physical activity with increased breathing could intensify tinnitus. The altered cortical activity during hyperventilation spread to other brain lobes, including the bilateral frontal and occipital, right prefrontal and central and left parietal regions.

The ''epileptic theory'' of tinnitus was supported by the pathologic EEG patterns. These abnormal patterns were present in the tinnitus patients significantly more frequently than in the healthy subjects. First, these patterns were located in the temporal lobe at point T6 on the 10 to 20 EEG montage system. From this site, they spread to the central and parietal regions bilaterally. The number of ''sharp wave-slow wave'' patterns and the amplitudes of the slow waves, peaks and ''sharp wave-slow wave'' increased.

An important finding of the present study was the influence of sound on cortical activity. The masking of tinnitus is a popular treatment method. Therefore, during audiometry, a sound that matched and therefore masked an individual's tinnitus was used as the acoustic stimulation for EEG. In the tinnitus patients, sound application changed spontaneous activity in the prefrontal, frontal, parietal, temporal, central and occipital cortices on the left or right, depending on the region. The amplitude of the beta-1, beta-h and delta waves was increased, and the amplitude of the alpha-1 wave was decreased; sound stimulation emphasized this pathology in the tinnitus patients. Two other observations regarding the alpha-1 and delta waves indicated a tendency to return to normal after acoustic stimulation. Sound stimulation, however, inconsistently affected cortical activity; it did not appear to modify the same waves that were observed to be abnormal in a particular lobe. Moreover, the temporal region, which is considered a potential origin of tinnitus, was modified only very slightly.

Zeng (7) observed the intensification of alpha power, followed by tinnitus suppression, when external stimulation was applied via a cochlear implant to the apical part of the cochlea. The sound delivered was specific, i.e., a low rate less than 100 Hz and softer than the tinnitus, unlike in the present study, in which the acoustic stimulation was individually selected according to the subject's tinnitus. Weisz (11) reported desynchronization of the rhythm between 6 and 12 Hz (theta, alpha) after sound stimulation and discussed the inhibitory effect of alpha activity on tinnitus sensation.

Thus, the usefulness of sound therapy for tinnitus patients is not clear. Additionally, the healthy subjects, in contrast with the tinnitus patients, did not demonstrate any changes in spontaneous cortical activity during acoustic stimulation. Thus, while the dyssynchrony-synchrony theory of tinnitus is supported by cortical activity, concomitant sensory, biophysiologic, metabolic-electrophysiologic and neurochemical aspects are also involved (12).

EEG is an effective method for evaluating cortical modification after sound stimulation, but there are some limitations to this method. Changes in the power of cortical rhythms can be influenced by the psychologic state, e.g., meditation can enhance the process of habituation to background stimuli (also acoustic, with no task imposed), and depression can reduce perception accuracy (13,14). Furthermore, we do not know what happens to cortical activity after the cessation of acoustic stimulation, e.g., EEG changes after conventional laser stimulation last for 15 minutes (15). To further investigate the usefulness of sound therapy for tinnitus, studies should be undertaken to observe neural activity on EEG and positron emission tomography simultaneously.

In summary, the present study demonstrated the following findings:

1. Cortical activity in tinnitus patients is different from that in healthy subjects, and it is not a ''phantom'' sign;

2. The alterations in cortical activity in the patients with tinnitus, which were observed predominantly in the temporal regions, include decreased delta wave amplitudes

and increased alpha-1, beta-1 and beta-h wave amplitudes demonstrate a pathologic pattern;

3. Significant alterations in spontaneous cortical activity induced by acoustic stimulation were only observed in the tinnitus patients, and despite individual matching of the applied sound, the pathologic changes only slightly improved;

4. The usefulness of sound therapy for tinnitus should be considered together with the results of other objective studies, e.g., positron emission tomography; and

5. EEG is an effective method for evaluating cortical modification after sound stimulation, but some psychological limitations to this method should be taken into consideration.

AUTHOR CONTRIBUTIONS

Pawlak-Osińska K conceived the idea and wrote the manuscript. Kaźmierczak W prepared the manuscript for publication. Kaźmierczak H conducted the literature review. Wierzchowska M collected and prepared the data. Matuszewska I obtained the EEG recordings.

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