The post- noise exposed condition of the normal hearing workers with tinnitus and hyperacusis developed by the chronic occupational noise exposure

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Research article

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Abstract

Background: Noise pollution has been called an invisible killer. It has been a critical issue for the people working in the noisy environments especially in industry and education. This study was conducted to evaluate the differences of neuronal activity between groups, an experimental group is profession in occupational noise environments and a control group is who did not, and all of the subjects had tinnitus or hyperacusis. We used the electroencephalography data from 17 patients. The two experimental subjects were a tinnitus patient and a hyperacusis patient. The fifteen control subjects were seven tinnitus and eight hyperacusis patients and all of the subjects had normal hearing.

Results: We compared the brain activity for three states among the groups: after noise-induced state and no sound exposure state for the two experimental subjects, and no sound exposure state for the control group. The activity of the auditory cortex in the experimental group after noise exposure were significantly increased in gamma ($p = 0.002$) and decreased in delta and theta band. In other brain areas, the rates of the delta, theta, beta 1~3 and gamma for the control group were higher than the experimental subjects for both with or without noise exposure states.

Conclusions: Through this study, it was suggested that the professions of tinnitus and hyperacusis with normal hearing in occupational noise environment could be maintain their pathological states by abnormal hyper-activation of the primary and secondary auditory cortex alone.

Background

Tinnitus, perception of hearing ringing, buzzing or hissing sound without external sounds, is a typical and chronic symptom of permanent hearing loss [1,2]. Sometimes when people are exposed to a loud noise, like noise from public transportation, transient threshold shift (TTS) of hearing can occur in normal condition of healthy people and subjective tinnitus may also possible to develop temporarily [3,4]. Occupational noise exposure such as loud noise and chronic noise exposure could develop permanent threshold shift (PTS) which is belong to causative factors of permanent hearing loss to workers, and it is so called as noise-induced hearing loss which is major cause of chronic subjective tinnitus [3,5,6]. In these unexpected and unpreventable situations from noise, transient or permanent tinnitus is a well-known major symptom of noise exposure.

In the case of hyperacusis, they experience physical symptoms and uncomfortable feelings such as migraine and pain when exposed to general living noise of low-intensity, and these sensations are the main symptoms of hyperacusis [7–10]. Because noise is an invisible, unpredictable and so powerful energy source, these subjective hearing disorders, tinnitus and hyperacusis, are becoming worse and a crucial issue in an occupational noise environment [11].

Regarding occupational noise environments, there have recently been studied with a large number of subjects, of hundreds to thousands, regarding clinical effects of the occupational noise exposure, e.g.,

construction [12–14], industry [15], comparisons of the four occupations (cf. education, music, industry and other occupational noise environment [16], and obstetric wards [17]. In usual circumstances of these working environments, occupational noise is a usual condition of long-term and higher than 80dB of noise exposure, continuously generated from the working environments in every day and whole time of the working hours [12]. The consequent chronic noise exposure in occupational noise environment physically damage to the hearing of workers and critically affects their susceptibility in the noise-induced stress and their quality of life [18,19]. The audiometry results, especially in the industry and education, were revealed that the workers who suffered from inner ear disorders were significantly higher than other occupational groups [16].

Since 1970's, several research group have attempted to study evaluating the hearing and clinical pathology status of the central nervous system for the professions in the certain occupations of the chronic noise environments, e.g., tractor operator [20], industry professions including construction [21–25], traffic police officer [18], veteran [26], aviation pilot [27], and other occupational noise environments [28–30].

The subjects participated above the studies had been also undergoing low ability performance in an attention task [18,26], enhancement of (auditory) sensory processing in a silent condition [18], and a disorder of central auditory processing in non-speech condition of the noise-exposed and normal hearing subjects [21].

Not only noise environmental occupations but also central pathologic status of tinnitus and hyperacusis has been studied via neuroimaging. Among of them, the researches related to auditory resting state of tinnitus represented pathological brain states of the patients and resting state of electroencephalography (EEG) were assessed through spectrum analysis and connectivity [31–38]. Based on the above studies, resting state of quantitative EEG was used as assessment tool, and we evaluated activity of the auditory/non-auditory brain area whose locations were designated based on the 10–20 montage and anatomical location (see, Fig. 1).

Because most of central processing problems of tinnitus and hyperacusis patients were developed by peripheral hearing loss, hearing researches also have been conducted in certain environments in which otologic disorders frequently occurs. According to the previous reports, in over twenty different construction industry professions, hearing was statistically significantly worse [12], and condition of chronic occupational noise exposure and that of duration were also significantly associated with subject's hearing [13,14]. Also, in the study of normal hearing workers, the occupational noise index of the workers in obstetric wards was significantly related to tinnitus and auditory fatigue which induced by sound [17].

In the previous reports, regarding cellular level of noise-induced condition, neuronal activity was showed the fast gamma pattern with spiky in the temporal and auditory cortex in animal models [38–42].

Comprehensively, above the cohort and/or clinical trials in human and the in-vivo researches of the animal models, we carried out this study with hope that the study could evaluate the pathophysiologic differences previously reported (e.g. high gamma pattern), and the relation of these differences in neuronal activity and the clinical pathology symptoms (e.g. tinnitus and hyperacusis) caused by occupational noise exposure in normal hearing workers. Also, we intended to suggest that how their default modes of tinnitus and hyperacusis subjects working in a noisy environment differ from those patients who are not exposed to occupational noise.

Methods

Participants

The EEG data from the two experimental subjects from a previous study (EB Bae, 2019) were included in this study. We compared the EEG data between the experimental group (N=2) and the control group (N=15). Of the two experimental subjects, one had tinnitus and the other had hyperacusis; thus, we selected patients with the same disorders as a positive control group from the previous researches database. EEG data of 17 subjects in total were used from completed clinical trials which introduced in the previous studies.

Because the two subjects had normal hearing, we selected EEG data from patients who had the same normal hearing from these approved research databases. In the first study, 7 out of 80 subjects had normal hearing; the mean score for right ear hearing was 8 (±4) dB and 8.9 (±4.9) dB for left ear hearing. In the second study, the control EEG data were from 8 out of 9 subjects who had normal hearing; the mean score for right ear hearing was 5 (±3.6) dB and 6.1 (±3.9) dB for left ear hearing (Table S1). Thus, the EEG data from a total of 17 subjects, 2 in the test group and 15 in the control group, all with an otologic disorder, were used in this study. In total, the EEG data from eight tinnitus cases were used. One case was for the test group and seven cases were for the control group. From the hyperacusis database, one case was used for the test group, and eight cases were used for the disorder control group (Table 1).

Experimental subjects

The experimental subject with tinnitus has been working at a noisy construction site with an extremely loud booming sound that could cause hearing loss in healthy people, such as a metal banging sound or sound from heavy equipment. Even if his bilateral hearing thresholds were within normal range, see Table 1, tinnitus was developed because of chronic exposure to an extremely noisy working environment during working hours for a long duration [16,43].

Although the noise level of the working environment was not enough to cause hearing loss in this subject, it is thought that tinnitus, which is commonly found in hearing loss patients, is caused by chronic exposure to loud noises [12]. The tinnitus sound got louder on the day he worked, and he also complained that his tinnitus remained even on his off-day. During much of his working hours, he was exposed to high random frequencies and high intensity noise; thus, he was defenseless exposed to the sound and had to

listen to the noise throughout his working hours. As a result, he experienced auditory trauma from the occupational noise in his working environment [3,26,44-46].

Another otologic disorder is chronic hyperacusis. Hyperacusis has different symptoms than those of tinnitus in that the condition cannot be recognized without an external noise [8]. Tinnitus is a ringing sound usually in the hearing damaged ear that occurs all the time without any external noise [1,9,47]. However, hyperacusis symptoms in normal hearing usually occur only when patient heard a sound in a noisy environment. Sound or noise is a necessary condition to provoke symptom of hyperacusis. In the second experimental case, the female patient was aware of her physical symptoms herself when she was exposed to only a noise louder than her uncomfortable level (UCL).

She was a teacher in a girl's high school. Most of her unpleasant sounds came from the working environment. The sounds that provoked her symptoms were piano, food plate scraping, stereo sound, and speaker sound in the playground, and she also got symptoms when teenage girls would suddenly shout loudly. These sounds are unpredictable, high frequency, loud and can cause hyperacusis symptoms. The UCL was measured by pure-tone audiometry, and the mean threshold was 84.3 (±5.0) dB. She had the same UCL on the right and left ear. This UCL was a higher intensity than that of the other hyperacusis controls whose average thresholds were 76.3 (±19.5) dB (Table S1.). Each patient's noise condition is described in the supplementary data (SI 1.).

Electroencephalography test

The same procedure was used as in a previous study (prev. ref). EEG data were recorded from the two experimental subjects and 15 controls. Two reference electrodes were located each on the right and left ear, and we used the average reference montage. EEG was recorded in a sound- and electrically-shielded booth. While recording the EEG for 5 minutes, no sound was induced except for case 2 with hyperacusis. Post-processing of the EEG data included baseline correction, eye movement and other artifact rejection, interpolation of bad channels, and averaging using Independent Component Analysis methods.

Analysis

Comparisons were done among the two noise conditions in the experimental subjects and the control group. A total of three groups were used: the no sound exposed state (NS) group, the after noise induced condition (aNI) group, and the positive control group (see figure 2).

The neuronal power density of each group was represented by brain topography. The color scale bar of the gamma band was normalized to 20% of the maximum thresholds, and the gamma-theta ratio was normalized to 300%.

Neuronal activity was evaluated by the amplitude and frequency rates. Brain areas were grouped by bilateral auditory and non-auditory cortex; statistically, a minimum of four channels were used for auditory cortex (see, figure 1, 2). Non-parametric analysis was done by two-independent test. Moreover,

Kruskal Wallis test was done among the three groups. All the statistically results presented in this study were obtained by SPSS v.23, IBM.

Using Low-Resolution Brain Electromagnetic Tomography (LORETA) program, we compared the activity of the whole brain area among the noise induced states of the two experimental subjects, the no sound exposed state, and the positive control group.

Results

Brain Topography

Figure 3-A, B shows the neuronal power density results of the experimental subjects. Figure 3-A shows the neuronal power density for the mild temporal hyper-activated states (MTHS) for the NS exposed state. Figure 3-B shows the neuronal power density for the severe temporal hyper-activated state (STHS) for the aNI condition. In Figure 3-A, the bilateral auditory cortices had a weaker hyperactivity evident by the absence of pointed waveforms when there was no speech stimulation and noise exposure. The gamma wave intensity of the neurons was dramatically increased after noise exposure. In the positive control group, abnormally high oscillations were observed in general, while in the two experimental subjects, the gamma band was observed only in the auditory cortices before and after noise exposure.

Neuronal activity comparisons

Neuronal activity was evaluated comparing the neuronal power density and the rates of the neuronal frequency between the three groups. In Figure 4-A, in the bilateral auditory cortex, the neuronal power of alpha 2, beta3 and gamma bands for the aNI group was significantly higher than the positive control group. The percentage of delta and theta bands was significantly different between the positive control group and the aNI group. In Figure 4-B, in other brain areas, the neuronal power was significantly reduced between the NS group and the positive control group in the delta, theta, alpha, beta2, and beta3 bands. In contrast to the power, the percentage rates for seven frequency bands except for the alpha2 band were significantly higher in the control group than in the experimental groups.

LORETA analysis

Figure 5 shows the frequency analysis results with sLORETA. Frequency comparison was done between the aNI group and the control group and between the aNI group and the NS group. As a result of subtracting the NS from the aNI using sLORETA, the left auditory cortex had a positive score (red to yellow), and all other areas were minus (skyblue to blue). When the control was subtracted from the aNI, the result was positive on the left side and little difference on the right side.

Discussion

Considering that neuronal power dramatically changed in only the bilateral auditory cortex, after noise-exposed in silent state, and did not change in other brain areas (see, Fig. 3-A and B), the auditory cortex of the experimental subjects seems to be separate from the surrounding areas and acts differently in the subjects. Abnormal spiky signals were only observed in the primary and secondary auditory cortex areas in the previous study, and the original signals were assumed to come from cochlear nerve [48,49].

In Fig. 4-A, the percentage rates of delta and theta band in the aNI condition of these noise industry professions were significantly decreased compared to the NS and control groups. It means that the inhibitor function of delta and theta were not properly working when exposed to noise. Gamma, beta3 and the phase coupling ratio of delta and theta also increase at the same time due to noise. This suggests that the main and original functions of the auditory brain area might be sensitized to chronic occupational noise exposure and that auditory cortex separately and hysterically act by auditory stimulation and eventually could be develop into physical symptoms and disorders, see Fig. 1, e.g. tinnitus and hyperacusis. If auditory stimulus causes abnormal neuronal activity, this physical condition may be classified as a wide range of auditory trauma and in this respect, this results are similar with [50] that minor damage could developed hyperactivity of the auditory cortex in tinnitus and/or hyperacusis. According to calculation of recovery time curve, if someone exposed to 100dB of noise to 17minutes, more than 8 hours of recovery time was expected and in case of occupational chronic exposure, 2 hours of 105dB of noise exposure may lead to 40-50dB of TTS, it would need about one and a half day (33.3 hours) of recovery time [51,52] it is known that the recovery time needs to be more than 15 minutes after noise exposure, and the recovery time can be different based on the noise intensity and exposure time [46,51,52]. For long-term auditory fatigue by noise trauma, auditory recovery was thought to take a long time [53].

Comparing the intensity of the overall brain area activation, the firing strength of the inhibition band in the noise industry professions tends to decrease (Fig. 4-B, NS-NI), and the alpha2, beta, and gamma bands show a statistically significant increase (Fig. 4-A). It is interpreted that the theta and delta bands that inhibit the gamma and beta3 activity are decreased and that the spiky abnormal beta and gamma activity due to sound stimulation persist for a long time [39–42]. However, power strength of the gamma band has not changed, whether noise exposed or not. Contrary to other brain areas, the gamma band in the auditory cortex was significantly increased between the aNI status and the NS condition and between the aNI status and the control of disorders.

In contrast to the experimental subjects, the intensity of the brain activity in the control group was generally weak overall brain area (Fig. 4-A left, B left), and the inhibition activity of the delta and theta bands were significantly higher proportion in the control auditory cortex while gamma band was lowered than noise induced state of the experiment subjects. It suggests that the results of our control group, tinnitus and hyperacusis patients who are non-occupational noise exposed, supported previously reported results. The results are that the auditory cortex of hyperacusis patients with tinnitus did not show hyperactivity in auditory resting state [32]. Unlike other tinnitus and hyperacusis subjects (control group),

occupational chronic noise exposed subjects showed highly activated solely bilateral auditory cortex. Applying neural plasticity theory to our results, auditory hyperactivity (temporal hyperactivity) could increase the hyperactivity of other brain areas if the subject is exposed to work environment noise from months to decades during working hours every day [37,50,54].

Our results also provide following clinical view same with previous reported in [17,18,21]; 1. The results showed that still strong and enhanced gain in the auditory cortex even in the silent condition. 2. Our subjects who have been long-term exposed to occupational noise with normal hearing has persisted symptoms of tinnitus and hyperacusis in no-sound condition. 3. The workers, our subjects, in the occupational noise environments had tinnitus and hyperacusis caused by chronic sound exposure.

From above the results, it is recommended that treatment may be approached differently in general cases of tinnitus and hyperacusis and in noise industry professions because central neural processing and clinical neuro-pathologic symptoms might be different. Previously reported studies, [55] showed that sound enriched environments reduced effects of hearing loss in the case of noise-induced hearing loss. However, in the case of the normal hearing experimental subjects in this study, sound using therapy may temporarily worsen the symptoms. Considering recovery time of TTS, recovery time is related to noise exposure duration and noise intensity, however it is determined directly by TTS thresholds rather than exposure time or noise intensity [52]. To sum it all up, we suggest routine check-up for hearing through hearing conservation program during working period in noise environments, and we recommend that noise industry professions work as far away from noise sources as possible, or minimize the period they are exposed to noise. By further minimizing noise exposure, it is thought that there will be improvement [3,4,56].

Limitation and Future work

This study, which analyzed resting EEG, shows that the activity of the cerebral cortex changes before and after sound exposure but brain activity at the time of sound stimulation is unknown. This is a study on abnormal and active states in the absence of sound stimuli after noise exposure. Despite the differences in sex, age, noise working environment, and symptoms, these common patterns identified in this study by tinnitus and hyperacusis seem to be an impact of clinical significance and should not be underestimated. Because noise environments causing tinnitus are different from the noise environment in which hyperacusis occurs, with this study, it is difficult to evaluate the common mechanism of these two disease groups when the same conditions are applied in a specific noise environment.

Furthermore, it is expected difficult to reveal this common mechanism in a separate clinical trial in an occupation group. In a prospective study, to identify the implications of this study, it is recommended that patients be screened as a group of workers in each several different noise work environment or occupations and be selected who has tinnitus and hyperacusis symptoms with normal hearing.

Conclusion

The results of this study are clinically meaningful in the following two perspectives: The first is the finding of the first affected area in the central region of tinnitus and hyperacusis caused by noise through simple EEG. Second, for noise environmental professionals, it is important that they differ from normal neural activity patterns seen in normal hearing tinnitus and hyperacusis. In general, for tinnitus and hyperacusis patients, the activity of various parts of the brain including the auditory cortex is high, whereas in the two subjects who worked in noise environment professions, abnormal cortical beta3 and gamma bands occurred in only the auditory cortex and lasted for a long time. This is interpreted to be due to the fact that the delta and theta bands are rapidly reduced at the same time with noise exposure, and inhibition of the beta and gamma bands is not achieved. This is the first attempt to distinguish subtypes of tinnitus and/or hyperacusis according to an onset mechanism using EEG. And also our results may help to prevent permanent hearing loss or chronic tinnitus and hyperacusis [57] for the professions in the occupational noise environment by a regular inspection of simple EEG. If a more research with large number of subjects is done in the future, the results that we reported may be useful for establishing a marker that distinguish tinnitus and hyperacusis of occupational noise exposure in normal hearing from general tinnitus and hyperacusis.

Declarations

Ethics approval and consent to participate

The tinnitus study was approved by the Institutional Review Board of the Seoul National University Bundang Hospital on August 29, 2016 (IRB No.: B-1607-355-004), and the hyperacusis study was approved in April 2017 (IRB No.: B-1612-373-001). The written consent was obtained from each of all participants.

Consent for publication

The author obtained written consent from two experimental subjects.

Not applicable to control group.

Availability of data and materials

The datasets are not publicly available. The data are available when request data from the corresponding author.

Competing interests

None.

Funding

Not applicable.

Authors' contributions

EBB designed this study, derived and analyzed data, and drafted the manuscript.

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Supplementary data

Yes

Abbreviations

Transient threshold shift (TTS)

Permanent threshold shift (PTS)

Electroencephalography (EEG)

Uncomfortable level (UCL)

No sound exposed state (NS)

after noise induced condition (aNI)

Low-Resolution Brain Electromagnetic Tomography (LORETA)

Mild temporal hyper-activated states (MTHS)

Severe temporal hyper-activated state (STHS)

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Table

Table 1. Demographic data of the experimental subjects and 15 control of the two otologic disorders.

ıts	Age	Age	Side	Duration (yr)	VAS intensity	VAS distress	Noise exposure environments	Noise induced symptoms
	M	54	B	4.5	6	10	Laboring at construction sites	Louder tinnitus
	F	26	R	10	7	7	High school teacher	Hearing sounds of ear muscle contraction Hearing noises in the ear

ɔl						
	M:F	Age	Duration	VAS intensity	VAS distress	C.C
	6:1	45.7±15	4.96± 7.91	7.3± 0.8	7.1± 1.3	Tinnitus
	4:4	31.5± 11.4	2.9± 2.8	7.4± 1.6	7.7± 1.4	Hyperacusis
ɔontrol						
	10:5					Otologic disorder

Figures

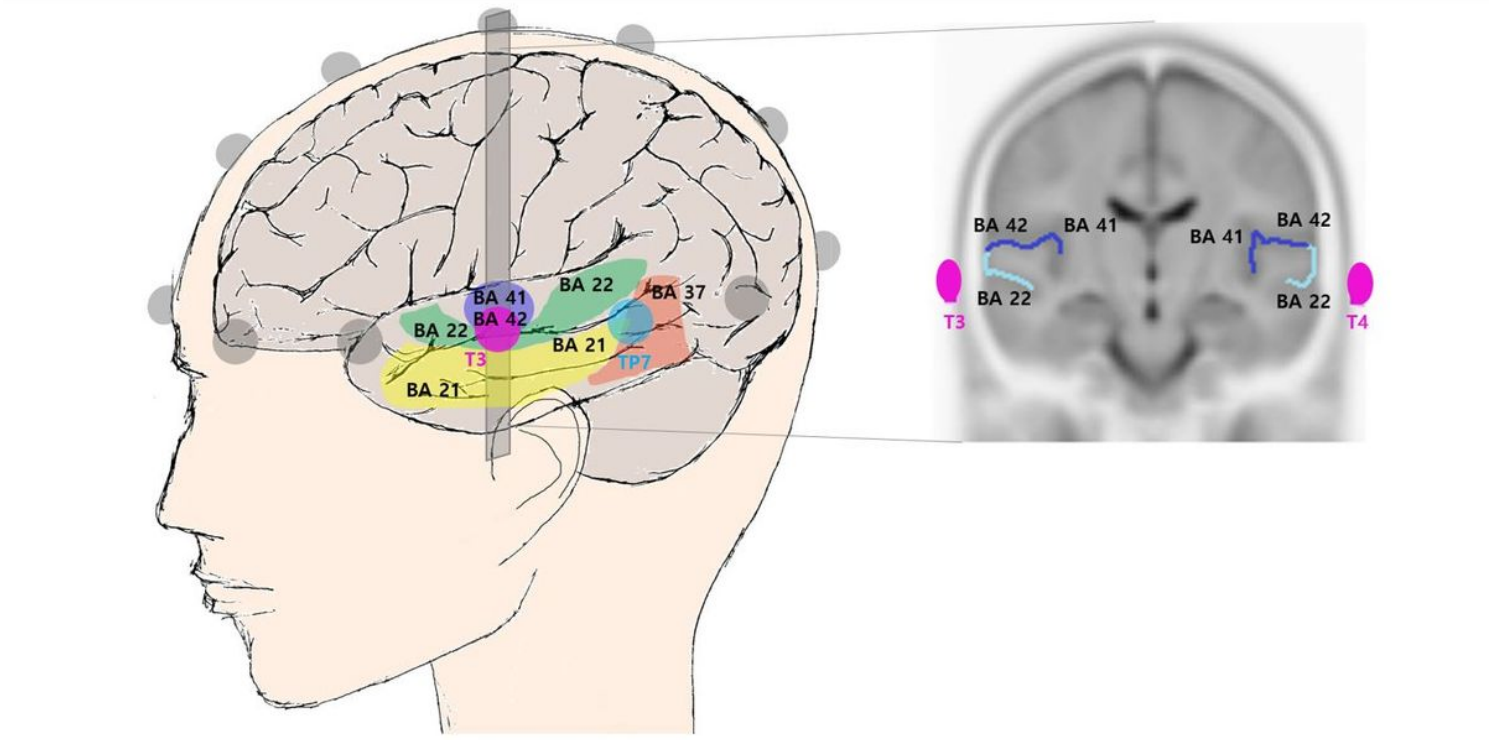

Figure 1

Electrodes representing cortical area. Left: Location of 31 electrodes on the scalp and cortex Right: Temporal electrodes near auditory cortices in neuroanatomical view. The figure was drawn by EB.B. Reference information: [BOOK] Atlas of neuroanatomy and neurophysiol_ Netters et al. Cerebral Hemispheres/Telencephalon, Waxman SG. Clinical Neuroanatomy, 28e; 2017 Sagittal view of the brain http://umich.edu/~cogneuro/jpg/Brodmann.html https://en.wikipedia.org/wiki/ : brodmann area 21, 22, 37, 41, 42 MRI scan of Brain: sLORETA

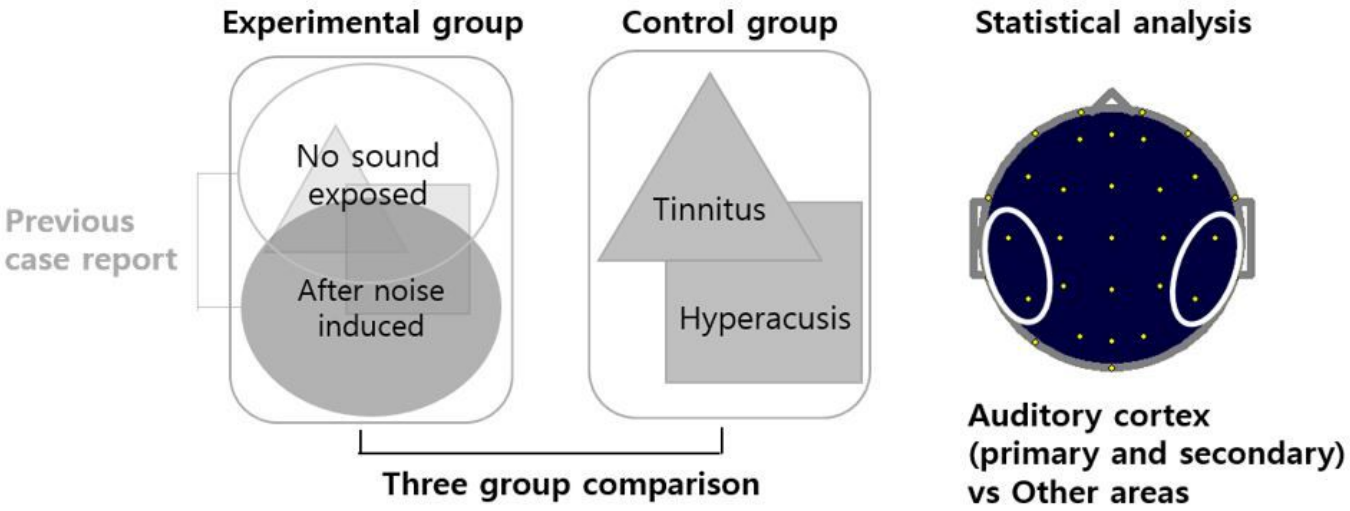

Figure 2

The diagram for analyzing procedure.

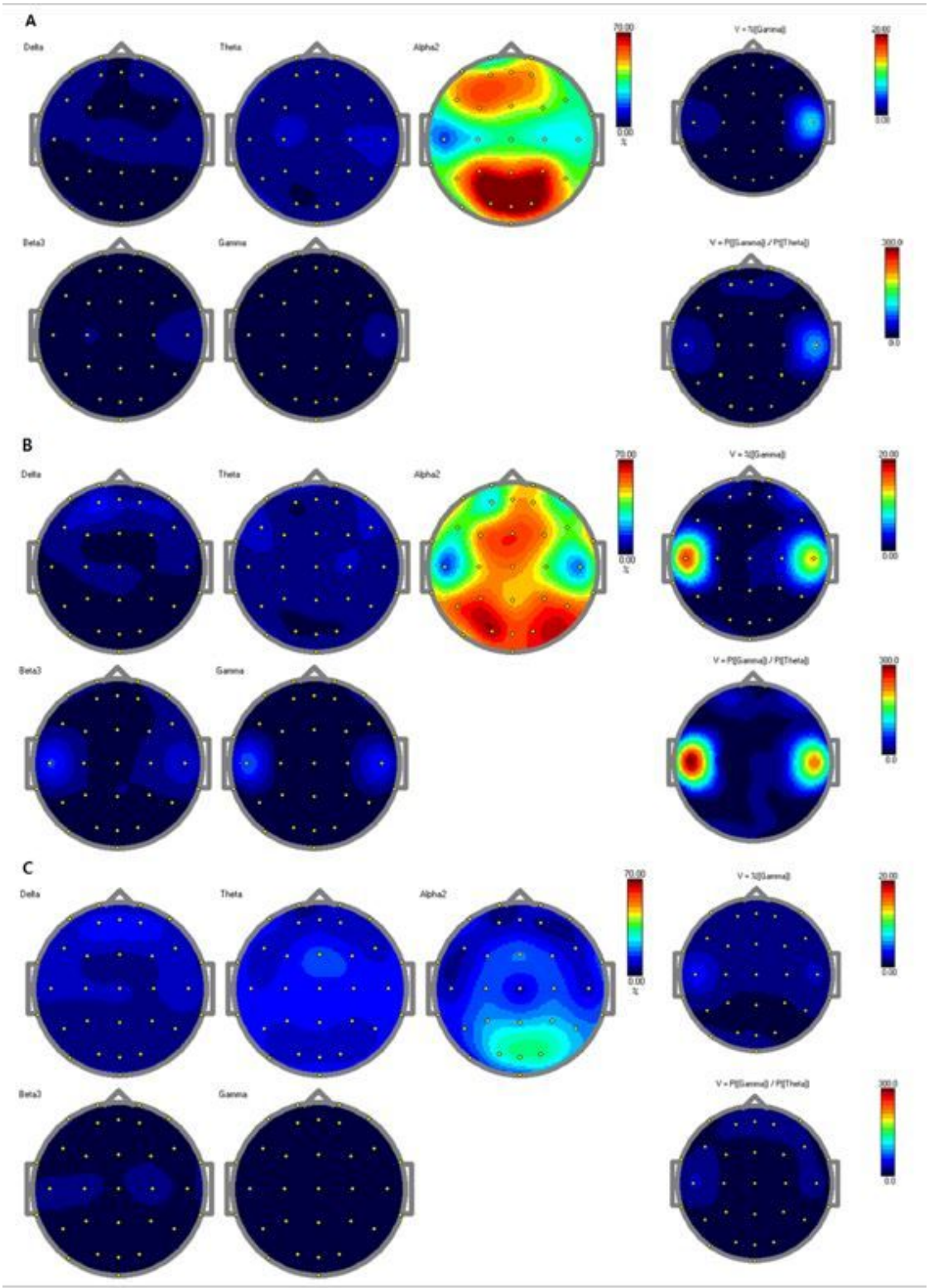

Figure 3

The brain activity of the two subjects and the control represented on the brain topography. A: resting state, no sound exposed condition in the two subjects. B: resting state after speech sound induced, (no listening) condition. C: Otologic disorder control (tinnitus and hyperacusis, n=16).

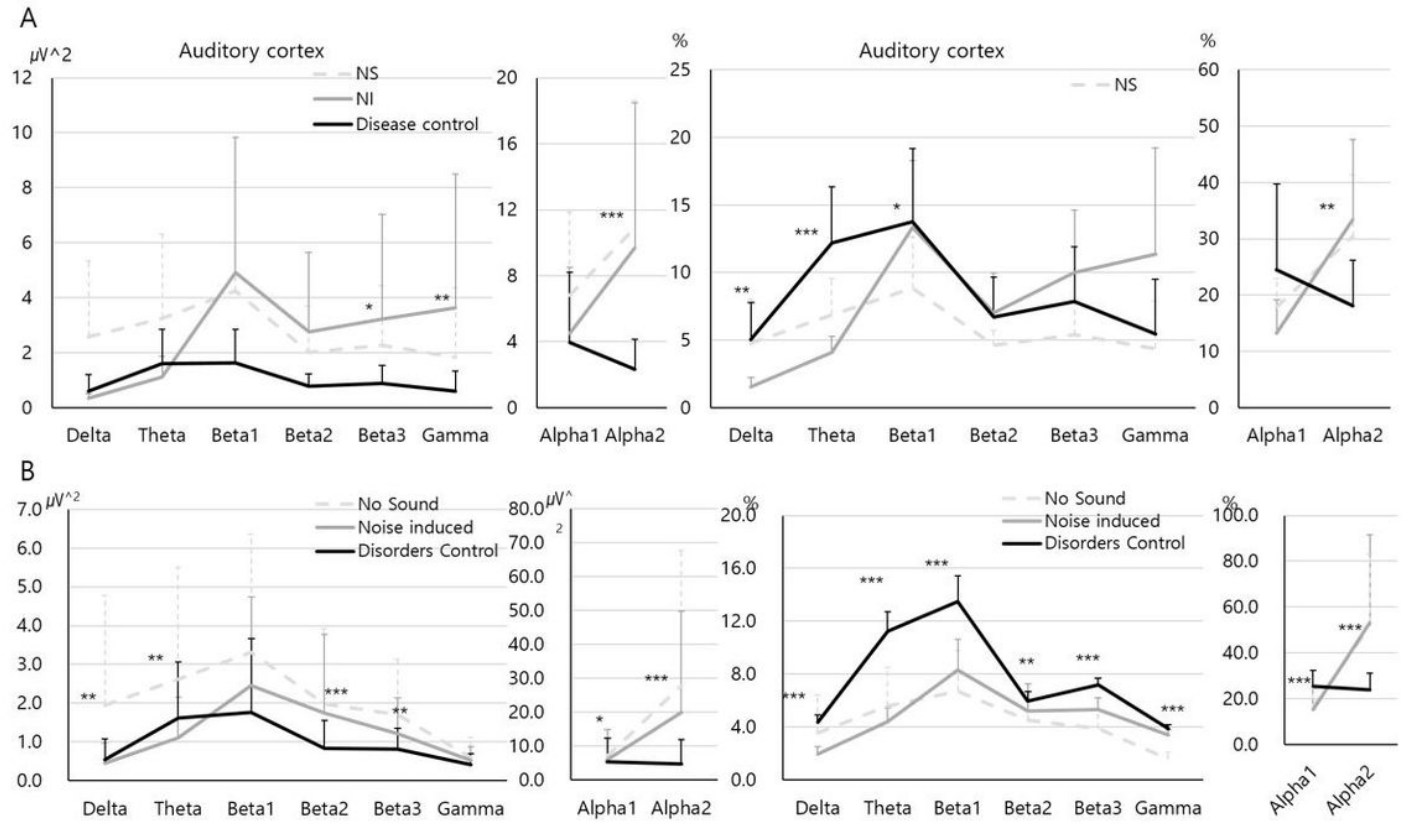

Figure 4

Neuronal activity was presented by the power and frequency rate. NS: no sound exposed condition, aNI: after noise induced state, Disorders control: normal hearing tinnitus and hyperacusis (n=16). A: auditory cortex (T3, T4, TP7, TP8) B: Other brain areas (27 channels). Significance: p<0.05*, p<0.01, p<0.001*****

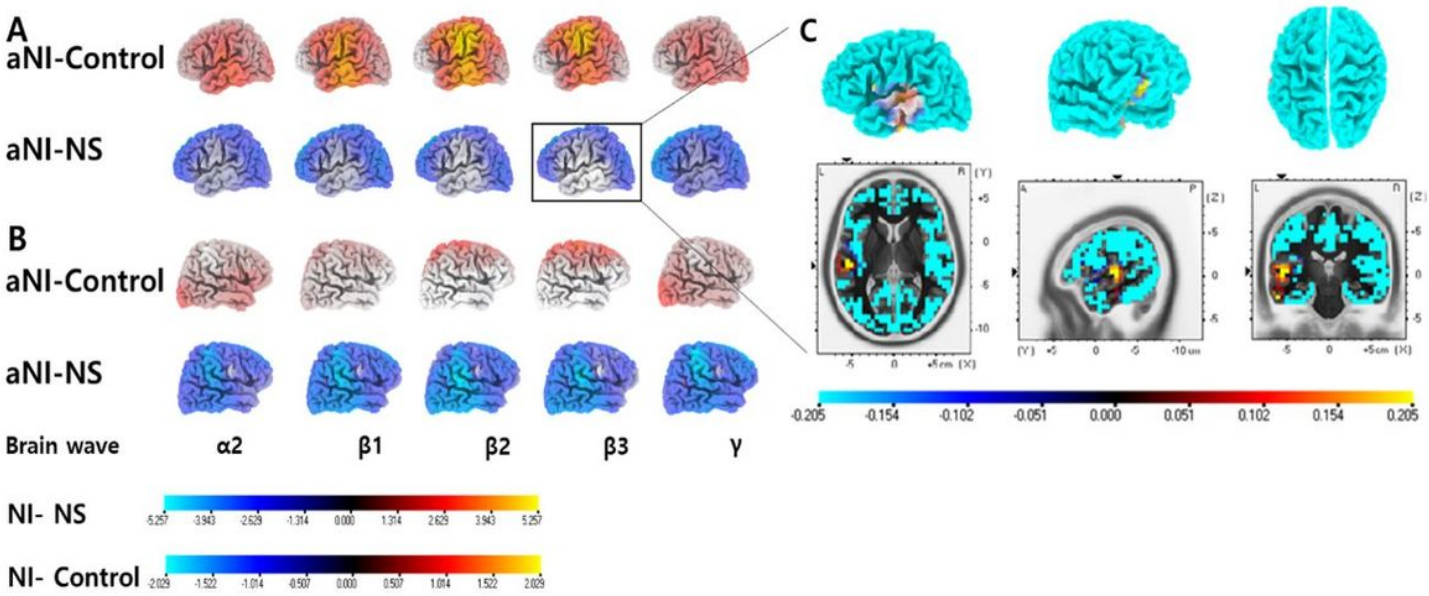

Figure 5

LORETA power density. A: Left side of the cortex. B: Right side of the cortex. C: Threshold of the right side of the cortex modulated focusing on BA22, 41 in the beta 3 band. aNI-NS: (after noise induced condition) – (No sound exposed state), $p<0.00000$ aNI-Control: (after noise induced condition) – (Disorder control), $p=0.00020$

Supplementary Files

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- BMCneuroscin17SupplementaryDataEBB.docx