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Tuning the tinnitus percept by modification of synchronous brain activity

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Abstract. *Purpose:* Tinnitus, the perception of sound without the presence of a physical stimulus, provides the opportunity to study neural codes of percepts without simultaneous processing of stimuli. Previously, we have found that tinnitus is associated with enhanced delta- and reduced tau-power in temporal brain regions. By operantly modifying corresponding aspects of spontaneous EEG activity, the aim of the present study was to corroborate the assumption that tinnitus should be reduced if patterns of ongoing synchronous brain activity are normalised.
Methods: In response to different variants of neurofeedback, a total of twenty-one patients produced significant changes in EEG frequency bands.
Results: Simultaneous alteration of both frequency bands was strongly related to changes in tinnitus intensity matched before and after the intervention ($r = -0.74$). In those two patients with the greatest modulatory success, the tinnitus sensation resided completely in response to the treatment. Comparing the neurofeedback-treated patients with a group of patients trained with a frequency discrimination task ($n = 27$), the tinnitus relief in the neurofeedback group was significantly stronger.
Conclusions: This study supports the notion that altered patterns of intrinsic ongoing brain activity lead to phantom percepts and offer new routes to the treatment of tinnitus.

Keywords: Tinnitus, EEG, neurofeedback, perceptual coding

1. Introduction

Subjective tinnitus, or the perception of ringing or buzzing sounds without any acoustic signal is a condition associated with maladaptive injury-related reorganization within the auditory system. The term 'maladaptive' signifies that it can have negative consequences for those affected, by being either bothersome or even dramatically reducing the quality of life. It is a wide-spread phenomenon with an estimated 5–15% of the people affected in industrialized countries (Axelsson & Ringdahl, 1989). Our research seeks to combine electrophysiological approaches that measure cortical synchronized activity of relatively large neuronal ensembles (EEG/MEG) with tools that are frequently used in tinnitus research reflecting psychoacoustic and affective aspects of the tinnitus percept. Using this approach we focus mainly on the question how ongoing oscillatory activity may be involved in generating tinnitus. Understanding the mechanisms of tinnitus is however not only of potential clinical value, but presents a unique opportunity for the study of neural coding of perceptual processes given that there is no confound from simultaneous input or processing of a physical stimulus.

The primary cause for tinnitus to emerge appear to be a deprivation of neurons in the auditory system from their normal afferent input, usually a consequence of damaged hair cells in cochlear regions (Shiomi et

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al., 1997; Weisz et al., 2006). It is thought that this deafferentation triggers functional reorganization in the central auditory system, which then produces neural activity sensed as tinnitus (Muhlnickel et al., 1998). The relationship of the tinnitus percept to the pattern of cochlear damage is illustrated by the correspondence between the audiogram and the subjectively-rated composition of tinnitus frequencies (Norena et al., 2002). Because it is impossible to experimentally induce tinnitus in humans, research on the neural mechanisms underlying this phantom percept is essentially correlational: i.e. measuring subjective aspects of tinnitus and neuronal activity around the same time. In our contribution to this special issue we present a study, in which we took a different approach, which relied on the operant modification of central neural mass synchronisation and then tested consequent changes in tinnitus perception. To do this, a specific neurofeedback strategy was devised to modify brain activity previously found to be abnormal in subjects with chronic tinnitus. Results from this work should yield more "experimental" support to the role certain brain oscillations play within the tinnitus framework.

At least three central nervous phenomena have been related to tinnitus all of which are reconcilable with the central notion that a reduced afferent input leads to a diminished ability of these neurons to inhibit normally subthreshold excitatory input: Tonotopic map reorganization (Muhlnickel et al., 1998), altered synchronised cortical activity (Weisz et al., 2005) and enhanced spontaneous firing rates in various structures of the auditory pathway (see Eggermont & Roberts, 2004 for a review). Animal research leaves little doubt that central neurons will retune their receptive fields when deprived from their input, becoming sensitive to input from neighbouring frequencies. The cortical representations of these lesion-edge frequency regions become subsequently expanded. In humans, Mühlnickel and colleagues (1998) observed corresponding distortions of the tonotopic map in auditory cortex in relation to reported tinnitus severity. However, hearing loss may cause several changes on a central level among which map reorganization could be a by-product not causally linked to tinnitus. The observation that tinnitus appears immediately after a noise trauma, whereas map reorganization requires time, would suggest that reorganization cannot be a requirement for tinnitus to develop. However, functional as opposed to structural reorganization has fast and slow components (Elbert & Rockstroh, 2004) and both may support similar changes in the ongoing dynamics of neural mass activity. Top-down modulation has been implicated in the fast, near instantaneous map reorganisation. Given the continualness of the tinnitus sensation, we suggest that the key to understanding tinnitus lies in patterns of the spontaneous neuronal activity (i.e. that is not evoked by an external event) of auditory and non-auditory regions. Provided a sufficient amount of neurons are synchronously activated – a reasonable assumption in case of a conscious percept – and oriented in a similar direction, then non-invasive methods such as EEG and MEG could be suitable in capturing the abnormal activity underlying tinnitus.

Using magnetoencephalography (MEG), we have recently observed such abnormal spontaneous activity patterns in subjects with chronic tinnitus (Weisz et al., 2005). The differences were characterized by enhanced power in the delta range (1.5–4 Hz) and reduced power in the alpha frequency band (8–12 Hz). Given that both effects were most pronounced in temporal brain regions, we will refer to the 8–12 Hz activity as *tau activity* (Lehtela et al., 1997). The level of abnormality was strongly correlated with the magnitude of the tinnitus-related distress, especially in right temporal and left frontal areas. Hypotheses about the functional significance of such brain rhythms extend from epiphenomena such as the “noise” of the brain machinery to core processing units such as perceptual binding and object segmentation. The generators of the MEG *tau rhythm* have been localized in the supratemporal auditory cortex (Hari & Salmelin, 1997). Reduction of *tau* is a normal cortical reaction to sound presentation (Lehtela et al., 1997). The work by Weisz et al. (2005) extends this finding by showing that dampening of *tau* may be more generally related to the sound perception itself, as no real physical sounds were presented that needed to be processed.

The presence of enhanced delta conforms with propositions made by Llinas and colleagues (2005), who ascribe this activity in pathological conditions to a thalamic deafferentation followed by overinhibition. The hyperpolarized thalamic neurons then trigger so-called low-threshold calcium spike (LTS) bursts at approximately 4 Hz (Jeanmonod et al., 1996), which should be observable on a cortical level as slow wave oscillations. Yet on a cortical level it seems unlikely that tinnitus related slow wave activity is due to bursting activity. After inducing tinnitus via a noise trauma Norena & Eggermont (2003) observed an increase of the spike number within a burst immediately following the trauma, which however ceased after a few hours. Importantly, the mean Inter Stimulus Interval between

bursts did not differ between pre- and post-trauma. Thus it is not clearly shown in the case of tinnitus that slow wave activity is indeed of thalamical origin or if it could reflect excitability variations on a purely cortical level. A further contributing factor could be that slow waves reflect to some extent top-down modulation of auditory cortical activity, since slow oscillations have been implicated in the synchronization of activity of distant brain regions (Sauseng et al., 2005). The coordinated involvement of extra-auditory brain regions seems likely considering the potentially adverse affective effects of tinnitus and the fact that a majority of tinnitus sufferers report their tinnitus to be modifiable depending on the attentional and emotional state. The view that tinnitus involves a network of brain regions, is supported by correlations of frontal and temporal abnormal spontaneous activity patterns with the tinnitus-related distress (Weisz et al., 2005, Schlee et al., 2006). Overall, it appears that a tinnitus-related neuronal network is made up of at least two parts, with one coding perceptual features of the tinnitus represented (more in temporal areas) and the other being responsible in forming an emotional representation and drawing attention to the tinnitus sensation (more in frontal and limbic brain regions).

After showing that an abnormal spontaneous activity pattern is present in tinnitus, it is still uncertain as how far this condition indeed is a fundamental element underlying tinnitus rather than being an epiphenomenon of selective hearing loss. To tackle this question an experimental manipulation of the ongoing oscillatory brain activity is needed while audiometric properties remain unchanged. Neurofeedback has proven to be an effective tool for modifying electrophysiological properties of spontaneous and evoked brain activity in the past (e.g. Nowlis & Kamiya, 1970; Elbert et al., 1984, Fuchs et al., 2003). In these cases, distinct characteristics are extracted from the physiological signal and instantaneously fed back in the form of a simple signal to the patient. The patient is rewarded if this feedback signal is successfully modulated upon command. Operant conditioning, and the information processing itself, have been presented as mechanisms underlying successful modification of the brain responses (summary see Elbert & Rockstroh, 1984).

If the persistence of tinnitus is causally related to abnormal alterations of ongoing synchronous brain activity, then successful neurofeedback leading to a normalization of the tinnitus-specific brain activity should result in an alleviation of symptoms. Such a finding would not only advance our understanding of perceptual coding in synchronous neural mass activity, it could potentially offer new ways for the treatment of tinnitus. In contrast to the previous method of using neurofeedback to treat tinnitus, which was thought to reduce the distress by enhancing parieto-occipital EEG-alpha, we used the temporally-generated *tau rhythm* (8–12 Hz) and slow-waves in the delta (3–4 Hz) range for feedback. We measured the signals from frontocentral (instead of posterior) sites, since it is known that the orientation of electrical generators within the auditory cortex project to these frontal sites (Pantev et al., 1995). Given that the deprived auditory regions putatively shift to slow-wave oscillatory mode and that the normal *tau* activity is reduced, the goal is to attenuate the power in the delta-band and enhance *tau* waves. In order to evaluate the relationship between the two frequency bands and tinnitus we used three different neurofeedback protocols (ten sessions at 30 minutes each): the goal of the first was to augment the ratio of tau/delta, the second was to reduce isolated delta-power and the third was to enhance tau power.

The outcome of this neurofeedback group was compared to a group that received auditory frequency discrimination training (FDT), targeted to achieve map normalisation, i.e., a reduction of the hearing-loss induced reorganisation. FDT has been inspired by the idea that essentially map reorganization drives the tinnitus appearance (Mühlnickel et al., 1998). In a previous study by Flor et al. (2004) the essential assumptions of FDT in reducing tinnitus could not be confirmed and only an effect of the duration of participation turning out to be related with tinnitus distress reduction. The present comparison with FDT data serves to control for unspecific effects, such as attention, of the expected tinnitus reductions induced by our neurofeedback approach.

2. Methods

2.1. Participants and procedure

Twenty-one patients with chronic tinnitus were recruited through advertisements in the local newspaper and through self-help groups. Patients gave written informed consent to participate and the study was approved by the Konstanz University Ethical Review Board. The participants were between 31 and 62 years old, with an average age of 48 years (standard error $+/-$ 2.1). There were 9 females and 12 males. The mean tinnitus duration was 8.7 years (SE $+/-$ 1.6).

The sample had a mean tinnitus intensity of 25 dB HL (SE $+/-$ 2.61; matched via 1 kHz tone, see below) and a mean distress level of 26.5 points (slight distress) on the Tinnitus Questionnaire.

Neurofeedback training consisted of 10 individual sessions lasting 30 minutes each, distributed across four weeks. EEG (Eldith GmbH, Germany) was recorded from four electrodes attached to fronto-central positions (F3, F4, FC1, and FC2) and referred to the right mastoid. We assigned patients to one of the following three feedback protocols: (1) reward for increments of the ratio between tau (8–12 Hz) and delta power (3–4 Hz; (2) reward for increments of power in the tau band; (3) reward for reduction of power in the delta band. The particular target measure was fed back to the patient in the form of a cartoon fish that slowly moved from left to right on a computer monitor. The patient sat in front of the monitor and was asked to move the feedback signal in a vertical direction while it continued horizontally with constant speed. In each session the fish started at a baseline (a horizontal line in the middle of the screen) calculated from the current spectral power. The height of the fish represented the amount of the EEG activity. For instance, in protocol (1), the stronger the amplitudes of the tau/delta frequency bands were, the higher the fish was located above baseline. Contrary to protocol (1) and (2), the patients of protocol (3) had to move down the fish in order to reduce the amount of delta waves. When the patient reached an individually adjusted threshold, this was indicated by a sunshine-symbol. The patient obtained no particular instructions on how to manipulate the moving target, except that it could be done only by mental activity. The first eleven patients accepted for the study received protocol (1), the next five protocol (2) and the last five protocol (3). Before and after each training session, five minutes of resting EEG (via the four fronto-central electrodes) were recorded. Besides the different protocols, the design of the study was similar for every participant. Patients coped well with the feedback task. In the majority of cases, patients reported to feel comfortable about performing the task, with the exception of those who did not benefit from the training after several sessions.

Tinnitus status was assessed by the following procedures:

1. The intensity of the tinnitus was compared to a self-report to a 1000 Hz pure tone at the audiometer both before and after training (in 20 of the 21 patients). Therefore, the therapist started with a 1000 Hz pure tone at the hearing level of the patients and changed the intensity in an ascending manner in 3 dB steps. The patient was instructed to stop the therapist if the intensity of the pure tone is concordant with the intensity of the tinnitus. Then, the therapist repeated the procedure by adding 10 dB to the first level and continuing in a descending manner. Again, the patient had to give a sign if the level of the tone corresponded to the level of his tinnitus. If these two obtained values differed, the therapist noted the average value. The intensity values are specified in decibels above hearing level (dB HL). Patients were not provided with feedback about the values they reached.
2. Tinnitus-related distress was surveyed using the Goebel-Hiller questionnaire (TF, Goebel & Hiller, 1998), a validated German questionnaire which patients filled out before and after training as well as every week in between. Follow-ups were conducted at 6 weeks and 6 months. The TF questionnaire provides us with a total distress score but also with ratings on diverse subscales, such as emotional distress, cognitive distress, intrusiveness, hearing problems, sleeping problems and somatic complaints. The total score ranges from 0 to 84 points. Scores fall into a distress category of slight (0 to 30), moderate (31–46), severe (47–59) or very severe (60–84).

In order to estimate the specific effects of the neurofeedback training on tinnitus, we compare the results from the neurofeedback training with results from a training approach, called frequency discrimination training (FDT). We conducted the FDT in our laboratory with comparable settings as in the neurofeedback group. It consisted of ten sessions at 120 minutes. Twenty-seven patients with chronic tinnitus took part in the FDT. The patients (4 women) from the FDT group are on average 53 years old (standard error: 2.02, range from 24 to 65 years) and have tinnitus duration of 9.1 years (SE: 1.48, range from 1 to 32 years). Patients had to differentiate two pure tones with the distance between the tones decreasing with increasing success. The frequency range of the training tones lay in the hearing loss range thus feeding the deprived zones of the tonotopic map with behaviorally relevant input. Furthermore, patients wore a hearing aid, which enhanced frequencies in the hearing loss region.

2.2. Data analysis/statistics

Raw data of five minutes of resting, eyes-open EEG (128 Hz sampling rate; online filter 0–60 Hz) were

online-corrected for eye artefacts. Spectral power from the EEG activity from the four electrodes (F3, F4, Fc1, Fc2) was subsequently calculated in BESA using a mean Fast Fourier Transformation (FFT). This procedure slides a 512 point window across the data (hamming windowed with 50% overlap), performs an FFT, and averages the amplitude values of all obtained windows. The power spectrum was normalized by dividing each power value by the average overall power. The frequency bands of interest were defined as follows: Delta (3–4 Hz) and Tau (8–12 Hz).

3. Results

Patients demonstrated considerable ability to regulate the amplitudes of their brain waves by means of the neurofeedback training procedure. As a whole, the group was able to enhance the tau/delta ratio across all ten sessions an average of 71% (range of changes from -32% to 325%; mean tau/delta ratio pre: 0.56; mean tau/delta ratio post: 0.85; $t(20) = -3.34$, $p = 0.003$, paired t-test, two-sided). Overall, the intensity of the tinnitus decreased substantially, from a mean of 25 dB (SE: $+/-$ 2.61, range of 8 to 52 dB) before training to 16.9 dB (SE: $+/-$ 2.96, range of 0 to 49 dB) after the tenth session. The intensity remained below baseline at the first six week follow-up (mean 20 dB; SE: $+/-$ 3.34, range of 2 to 51 dB) and again at six months (17.4 dB; SE: $+/-$ 3.17, range of 0 to 36 dB). A repeated measures ANOVA with the within-subject factor of "time" (pre, post, follow-up 1, follow-up 2) revealed a significant change with $F\,(1.5, 19.7) = 3.85$, $p = 0.049$ due to a significant comparison between the baseline and post-training tinnitus intensity (after the tenth session) with F (1, 13) = 16, p = 0.008, but no other significant comparisons. Tinnitus-related distress was also noticeably reduced from a mean of 26.5 (SE: $+/-$ 3.35, range of 6–68) at the beginning to 19 points (SE: $+/-$ 3.3, range of 6 to 68) at the end of the last session on the Tinnitus Questionnaire. After six weeks the sample showed a slight increase in the distress score (20 points, SE: $+/-$ 4.14, range of 3 to 71) that persisted up to six months (20.5, SE: $+/-$ 5.38, range of 3 to 66). A repeated measures ANOVA with the between-subject factor of "group" (neurofeedback vs. frequency discrimination) and the within-factor of "time" (pre, post and 6 month follow-up) revealed a significant group*time interaction (F (2, 72) = 3.73, $p = 0.008$, see Fig. 1).

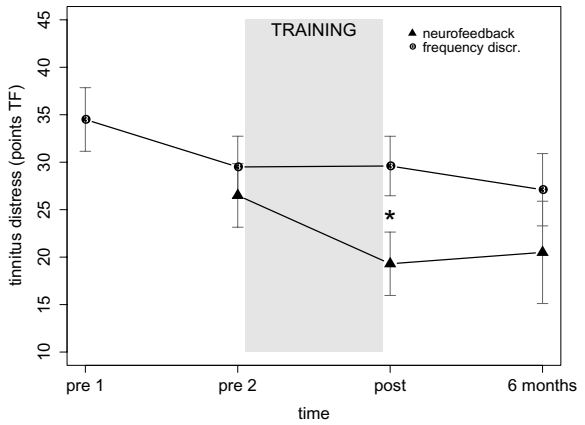

Fig. 1. Mean distress values ($+/-$ 1 standard error) for the frequency discrimination group (circles) at the initial interview (pre 1), one week before training onset (pre 2), after the training period (post) and six months follow-up and the neurofeedback group (triangles; values for "pre 1" are nonexistent). The grey bar displays the training period. The asterisk indicates a significant difference between the groups *after* the training period, whereas there is no significant difference at pre 2 and six months follow-up between the two training groups. Tinnitus distress is measured via a German Tinnitus Questionnaire (TF) and comprises values from 0 to 84 points, with high values indicating severe distress. Both samples have slight to moderate initial distress values (0–46).

One noteworthy outcome is that the patients who successfully modified their oscillatory brain activity were the ones who benefited the most (in terms of an intensity reduction), as indicated by a striking correlation between training success (operationalized as the ratio between tau/delta ratio after versus before the training) and the change in matched tinnitus intensity (ratio of intensity post versus intensity pre training; Fig. 2; $r = -0.74$ ($t(18) = -4.69$, $p < 0.001$). Patients were unaware of the dB levels and the experimenter did not know the patients' training success rate; thus, both were blinded with respect to this association. The influence of the single frequency bands on the intensity reduction is almost additive, but the correlations of tau change with intensity change ($r = -0.29$), and accordingly delta change with intensity change ($r = 0.37$), are not statistically significant ($p > 0.05$).

Concerning the distress, there is neither a significant correlation between the change of tau/delta ratio and distress change ($r = 0.22$, n.s.), nor between each single frequency band with the distress change.

Analyzing and reporting changes of the tau/delta ratio as a whole however does not definitely reveal a simultaneous change in both bands – tau and delta – as desired. A change of the tau/delta ratio could also only reflect changes in one band. Therefore, it is important to determine the predictive parameter. Patients

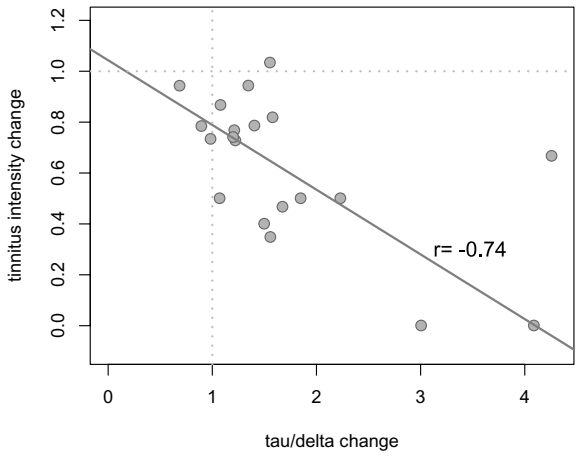

Fig. 2. Correlation between the change in tau/delta power (x-axis; displayed as the ratio between the tau/delta ratio after the training divided by the tau/delta ratio before the training) and the change in tinnitus intensity (y-axis; intensity after the therapy divided by the intensity before therapy). Values in tau/delta change above 1 (dashed line) indicate a high normalization (= enhancment of tau and/or reduction of delta), whereas slight values in tinnitus intensity reduction (under the dashed line) indicate large reduction. Two patients with large normalization show a tinnitus change of zero, indicating no tinnitus at post-training. The drawn through line is the regression line with the regressor of tinnitus intensity reduction and the predictor of tau/delta change. These analysis are independent from the different feedback protocols.

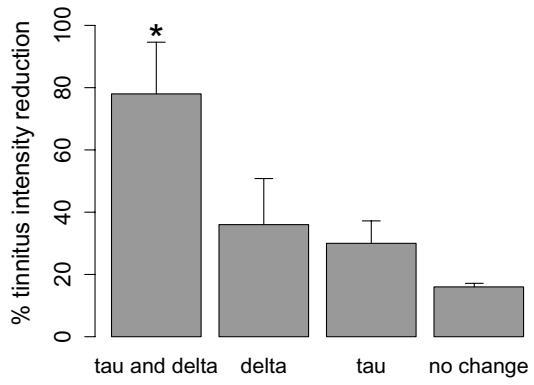

Fig. 3. Barplot of tinnitus intensity reduction depending on which frequency band could be modulated. This analysis is independent of the three different feedback protocols. The whole training group is divided into four clusters depending on the value of tau change pre-post training and delta change pre-post being above or under the median of changes. The left cluster comprises patients with values *above* the median of tau change and simultaneously *under* the median of delta change. These patients are referred to as the most successful ones as they were able to alter both bands in the desired direction. At the same time, they had the greatest tinnitus intensity reduction. The reduction is significantly higher than in subjects managing to alter delta (but not tau, second cluster from the left), or in the ones managing to change tau (but nor delta, third cluster) and finally those subjects who neither enhanced tau nor reduced delta (fourth cluster). The reduction in the fourth cluster is about 16%, indicating a placebo effect.

are classified in four groups based on their ability to modify tau and delta irrespective of the feedback protocol. This analysis shows that participants who modified both bands simultaneously showed the strongest tinnitus reduction or even abolishment of the tinnitus sensation. Their tinnitus intensity reduction was at 78% and was therefore significantly greater compared to the other three groups (Fig. 3; comparison with the "no change" group: $t = 2.72$, $p = 0.017$). Patients who only managed to change one band did not reduce their tinnitus significantly (tau: 30%; delta: 36% reduction) compared to the patients who showed no change at all. The latter group also reduced its tinnitus intensity by 16%, thereby representing an estimate of the placebo influence present in our treatment.

4. Discussion

The results of this experimental study suggest that changes in the oscillatory properties of cortical networks are fundamental to the tinnitus sensation; i.e., a normalization of abnormal rhythms is likely to lead to an alteration in tinnitus perception. Compared to the frequency discrimination training, the neurofeedback training shows significantly stronger effects on the tinnitus reduction. The FDT approach has had little success to alleviate tinnitus, a finding which is in agreement by the failure of a similar approach attempted by Flor et al. (2004). While treatment expectancy produces a weak reduction from 34 to 29 points on the Tinnitus Questionnaire before the actual start of the training (pre 1 to pre 2), no further reduction was produced by the discrimination training as opposed to the feedback training procedure. The result of the neurofeedback approach, that normalization of abnormal oscillatory rhythms reduces tinnitus, is consistent with and extends the earlier findings from our group that used magnetic source imaging (Weisz et al., 2005, 2006). In two patients with the greatest training success, the tinnitus sensation disappeared completely in the course of the training. Furthermore, results show that the alteration of both bands is the most successful approach for the tinnitus intensity reduction. This fact is an indicator for the coupling of tau and delta to some extent in the cell assemblies underlying tinnitus. The maladaptive oscillatory pattern has to be disrupted in order to alleviate tinnitus.

Even though this outcome has been predicted on the basis of our earlier findings (Weisz et al., 2005), the

exact mechanism by which changes in delta and tau promote the generation of an auditory phantom perception is still far from being understood. The work by Lehtala et al. (1997) demonstrates that tau is the normal idling brain rhythm present during silence. Therefore, the perception of tinnitus appears to induce a block of tau activity even in the absence of a physical sound source. Basic sensory perceptions are thought to arise from synchronized activity between neurons within and between cell assemblies coding relevant features of the to-be-perceived object, which is most likely reflected in fast (gamma) oscillations (Engel & Singer, 2001). Following hearing damage, this activity could be induced in circumscribed regions of the tonotopic map by a deafferentation of thalamocortical fibers leading to reduced inhibitory capacities within deafferented areas of the auditory cortex. In this view, delta would constitute a driving mechanism upholding synchronized activity within a cell assembly representing the tinnitus sound. This activity could also be boosted by long-range connections to limbic areas, which is also expressed in enhanced slow wave activity. By reducing delta activity and reestablishing the normal idling rhythm in the auditory cortex, we therefore disrupt the functional architecture that sustains tinnitus with continuously-enhanced gamma activation. Unfortunately this notion is speculative at this stage, as our sampling rate did not permit a sophisticated anaylsis of the gamma band and its potential relations with slow wave activity. However, it is substantiated by a recent study of Weisz et al (in press) who showed a) slow-wave related enhancements of gamma activity, b) an overall enhancement of gamma in tinnitus and c) a close match of tinnitus laterality and hemispheric dominance of $\sim$55 Hz oscillatory activity.

There are at least two limitations of the present study. First, results indicate that alterations of the distress level are not correlated with changes of the brain activity in the predicted direction, in contrast to the findings of Weisz et al. (2005), although the mean distress level can be reduced by about 26%. This result is mainly due to very low pre-training distress levels and therefore little room for improvement or reduction in symptoms. Apart from this, Weisz et al. applied a distributed inverse solution to their MEG data, therefore measuring with greater spatial resolution, enhancing the probability of finding meaningful associations.

A further limitation of the present study includes the form of the feedback protocol. From the post-hoc analysis we conclude that the simultaneous alteration of both frequency bands is necessary for changes in tinnitus loudness, but the protocols include only one of the two or the ratio of tau/delta, and even the ratio can only be modified by changing one band. To optimize our results we need protocols that will provide the information about tau and delta separately on the screen, for instance a two-dimensional feedback, with the magnitude of tau represented on the x-axis and the magnitude of delta represented on the y-axis. In this case, the patient has to move the symbol in the quadrant of high tau and low delta values. These ideas are currently being implemented in our laboratory.

Nonetheless, the strong relationship observed between success in normalizing the deviant brain activity and a change in the loudness of rated tinnitus raises the possibility that neurofeedback procedures can be developed into useful tools for the treatment of tinnitus. To date, neurofeedback in the treatment of tinnitus has been applied mainly to induce relaxation by training *posterior* alpha (Gosepath et al., 2001; Schenk et al., 2005), but no attempts have been made previously to normalize deviant and regionally specific brain wave activity. In contrast, the present approach focuses specifically on regional brain activity that appears to be abnormal in tinnitus sufferers. Individual adjustment of feedback parameters will likely improve the efficacy. Prior to the shaping of brain self-regulatory activity, a multi-channel recording could determine where and in what frequency bands deviant activity prevails. It seems promising to test such a training module as a means to ameliorate tinnitus in future research.

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