

NEUROFEEDBACK FOR TREATING TINNITUS

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Increasing evidence confirms the theory that tinnitus sensations are linked to plasticity in central nervous system structures. On a cortical level this has been shown for the auditory cortex. In earlier work, we have discussed distortions of the tonotopic map a candidate mechanism. But more recent work from our laboratory suggests that this link is not direct and indicates that abnormal changes in ongoing spontaneous activity patterns could be causally related to tinnitus perception and its associated distress. Using MEG and magnetic source imaging, we uncovered relationships between tinnitus and enhanced slow wave and concomitant reduced alpha wave activity over temporal and frontal areas. Modification of this pattern via EEG-neurofeedback significantly reduced the matched tinnitus intensity. Participants who consistently altered their oscillatory patterns profited from the treatment even to the extent that the tinnitus was completely abolished. Tinnitus distress in these patients was of mild to moderate intensity. Extending research to patients with the full range of distress, we observed a tight relationship between the subjective report and the coupling of distinct brain regions (via phase coherence of single-trial steady state responses). In particular, severe distress was perfectly correlated ($r=.97$) with network activity involving the right parietal and the anterior cingulate cortex. Based on this breakthrough in the neuroscience of tinnitus, we now propose to implement a treatment protocol that is apt to allay tinnitus distress by direct intervention into this newly uncovered tinnitus distress network. We will train a total of three groups (15 patients each) to investigate the effectiveness of this therapeutic measure and compare it to the previous treatment approaches using neurofeedback with tinnitus patients. One group will be given a training to reduce phase synchrony between brain regions representing the tinnitus distress network while they listen to an amplitude-modulated version (eliciting a steady-state response) of their tinnitus sound. A second group will get an optimised treatment of neurofeedback to enhance posterior alpha. As comparison to those, we will have a control group to train phase synchrony between brain regions that are unlikely to be involved in the tinnitus distress network.

Novel therapeutic approaches are arising from progress in the behavioural and neurosciences. Recent observations suggest that tinnitus distress results from a coupling of distinct brain regions, triggered by transient or permanent alterations in auditory input. Even in cases, where hearing is restored, this network of neural activities remains connected, unless actively modified, i.e., the tinnitus results from a chronic self-sustaining neural network phenomenon. We propose reorganisation of this network by means of neurofeedback, a technique that directly informs the patients when brain activities are modified in the desired direction. In case of success, our approach could be easily implemented on a large scale in clinical settings for outpatients. Manufacturing specific devices, costs of installations would be low. In addition, findings from our research will stimulate progress of other interventions – such as transcranial magnetic stimulation (TMS) – that may have the potential to interrupt neuronal connectivity in the "distress network". While the development of optimal therapeutic strategies remains a long-term goal, the presently proposed comparison of different approaches will lead quickly to first modules that can assist distressed tinnitus sufferers. We plan to transfer our algorithms to an open source project which allows fast dissemination to interested scientists and clinicians working with tinnitus patients.