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Case Report: Impaired Memory and Disorientation Induced by Delta Band Down-Training Over the Temporal Brain Regions by Neurofeedback Treatment

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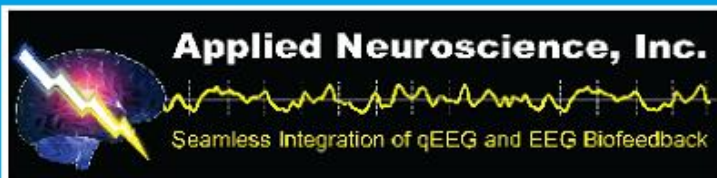
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LETTER TO THE EDITOR

Case Report: Impaired Memory and Disorientation Induced by Delta Band Down-Training Over the Temporal Brain Regions by Neurofeedback Treatment

To the Editor:

We describe the case of a patient who developed memory impairment and disorientation while undergoing neurofeedback (NF) treatment designed to lead to the down-training of the delta band over the temporal brain regions. These symptoms recovered following the reversal of the NF protocol.

The subject (A.B.), a 65-year-old male suffering from tinnitus for some 20 years, was referred for NF treatment. At the age of 45 years A.B. had been subjected at the time of war to intensely loud noise, and since then had suffered from severe hearing loss and tinnitus in both ears as well as an associated mildly symptomatic posttraumatic stress disorder. At the age of 62 years he became somewhat forgetful, misplaced objects, forgot the names of people, and was diagnosed as having mild cognitive impairment. At that time it was noted that he was fully orientated to time, place, and person and was functioning normally. The patient was not treated with psychotropic drugs, claiming that “they did not help him much in the past.”

At the initial clinical evaluation an EEG was recorded for 3 min with eyes closed and 3 min with eyes open. Measurements were performed using Deymed Truescan 32 (Deymed Diagnostic, Payette, ID). The NeuroGuide

software (Applied Neuroscience, Inc., St. Petersburg, FL) was utilized for analyzing the tracing. The main QEEG findings (both with eyes closed and with eyes open) included the following:

1. Absolute power: Deviation from age matched norms of approximately $-3 SD$ of frontal alpha.
2. Relative power: Deviation from age matched norms of approximately $-3 SD$ of posterior alpha, $+2.5 SD$ of temporal-parietal delta.

In an effort to determine the most effective therapeutic approach to the patient's tinnitus, we reexamined current knowledge relating to this condition. In summary, subjective tinnitus is the false perception of sound in the absence of an acoustic stimulus. It is a common, often intractable, and frequently distressing symptom. This perception may be described as ringing, buzzing, cricket-like, hissing, whistling, or humming. It is usually bilateral with lateral dominance to one ear. Chronic hearing loss is one of the causes of this multietiological phenomenon (Lockwood, Salvi, & Burkard, 2002).

The QEEG of patients with tinnitus demonstrates a diversity of alterations from the norm, and one may find abnormalities

in one or more of the frequency bands, ranging from relatively localized areas up to extensive cortical involvement (Shulman, Avitable, & Goldstein, 2006). A prominent finding is the higher than normal delta power. This finding may result from the deprivation of input to the auditory system, which may alter the spontaneous activity of groups of thalamic neurons leading to their hyperpolarization, and this may in turn trigger a bursting activity at slow frequency (i.e., delta band; (Dohrmann, Weisz, Schlee, Hartmann, & Elbert, 2007). Another phenomenon reported in tinnitus patients is a reduction of alpha power around 10 Hz in recordings from the temporal regions (*the so-called tau rhythms described in magnetoencephalography measurements*), which was hypothesized to be associated with a normal reaction to sound (Hari & Salmelin, 1997). Therefore it was assumed that because these patients continually hear sounds (associated with the tinnitus), alpha power will be reduced (Dohrmann et al., 2007).

Based both on these observations and the QEEG findings in this patient, we decided to down-train the delta band over the left and right temporal areas. The patient was treated twice weekly. Each session included 15 min of down-training of 1–4 Hz over one of the temporal areas (either left or right) compared to the ipsilateral ear, and then another 15 min of down-training over the other temporal lobe (T3 and T4 according to the 10–20 system). Due to the sensitivity of the temporal lobes and to avoid undesirable side effects, we used a referential montage, enabling us to train each temporal lobe separately. We used Deymed TrueScan 32 for the neurofeedback treatment (the amplifier of this system utilizes AC and not DC current). In the first two sessions there was no clinical improvement and no side effects. During the following three sessions the patient reported a significant improvement in the subjective feeling of tinnitus of the left ear but not of the right ear. At the sixth session, the patient's wife reported with much distress that A.B. had developed a severe decline in memory and orientation compared to his condition prior to the NF intervention. He would repeatedly ask questions such as,

“where should I put the cup, what day is it today, and what city are we in?” When his children had come to visit he had not recognized them and had asked who they were.

Apart from the significant deterioration in memory and orientation, there were no other complaints related to health, vital signs were stable, and neurological and physical examinations were unchanged. His sleep pattern was normal and appetite was preserved. Suspecting that the memory and orientation impairments had been induced by the NF treatment, we decided to up-train the delta band over the temporal lobes, and this was achieved by one session using the correction protocol. Two days later the patient's wife reported that the impairment of memory and orientation had disappeared, stating that A.B. had “come back to himself.” We thus decided to discontinue NF treatment. At a follow-up visit 1 month later the patient's clinical status remained stable.

DISCUSSION

The presentation of this case raises a number of theoretical and practical issues.

Although the temporal association between NF treatment and the development of cognitive symptoms in this patient is not necessarily a proof of a casual relationship, the fact that by reversing the protocol both memory and orientation returned to normal makes it feasible that these symptoms were actually undesirable side effects induced by NF treatment.

A number of mechanisms may be suggested to explain how the down-training of the delta band had caused such adverse effects. One explanation relates to regional brain metabolic changes. Cook, O'Hara, Uijtdehaage, Mandelkern, and Leuchter (1998) examined the correlation between EEG frequencies and metabolism, the latter reflected by local PET measurements. They reported a positive correlation for both delta and beta bands and a negative correlation for theta and alpha bands with regional brain metabolism over wide cortical areas. Therefore we may postulate that the down-training of the delta band had

influenced temporal lobe metabolism leading to impaired memory and orientation. Another possible mechanism suggested by recent studies associates the delta band to certain memory functions. One such study (Mormann et al., 2008) reported the existence of independent delta and theta rhythms in the human medial temporal lobe, suggested to be produced by autonomous generators. The authors propose that these rhythms play an important function in the connection between the entorhinal and hippocampal areas. Thus, at least theoretically, the down-training of the delta band may adversely affect the independent delta rhythm in the human medial temporal lobe, leading to memory impairments. Yet another explanation could be associated with the well-known role of theta activity in memory processing (Klimesch, 1996; Klimesch, Freunberger, Sauseng, & Gruber, 2008). Although we aimed at down-training the delta band (1–4 Hz), due to filter properties we may have also down-trained the 5 Hz. It should be noted that some actually consider the 4 Hz frequency to be part of the theta band (Niedermeyer & Lopes da Silva, 2005). Therefore down-training of the lower theta band could also possibly be related to the induced memory impairments just reported.

This case report suggests that the down-training of slow waves (1–4 Hz) is not always harmless and that precaution is needed when treating the electrical activity of the temporal lobes by means of NF in patients with cognitive impairment. Finally, there seems to be a lack of reports of adverse effects related to NF treatment. In an article titled, “First, Do No Harm: Adverse Effects and the Need for Practice Standards in Neurofeedback” (Hammond & Kirk, 2008), the authors pointed out the discrepancy that exists between the many reports of treatment related events appearing in various group-lists in the Internet, and the lack of proven scientific evidence regarding the side effects of NF treatment. We suggest that after 40 years of experience with NF, the scientific community should aim to advance our

knowledge regarding the possible negative effects of NF treatment.

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