The linear-model formalism introduced here to explain the performance of conventional beamformers for correlated (brain or artifact) sources can be easily expanded to more than two sources, which will be done in our future study.

References


18. TRANSCRANIAL INFRARED BRAIN STIMULATION MODULATES EEG ALPHA POWER

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strongest activation was between 8-10 min (77-97 J/cm²) after TIBS started, and the Alpha power density diminished rather quickly during the recovery period, 2-3 min after TIBS stopped.

Fig. 1. The subtraction of averaged time-frequency map between TIBS and Placebo at electrode Fp2. (N=20).

Fig. 2 shows several rendered cortical maps and 3D tomographic images at 6, 8, 9, 10, and 11 min after TIBS onset. These t-maps showed TIBS-induced significant changes in Alpha-band power relative to baseline. Moreover, (i) this change manifested after approximately 2 minutes, (ii) the neuromodulation propagated from anterior to posterior cortical regions, (iii) TIBS modulated ipsilateral activity at the anterior cortex, while also modulating bilateral activity at the posterior cortex. (iv) TIBS modulated an ipsilateral, fronto-parieto-occipital network and also a contralateral, parieto-occipital network at the Alpha frequency throughout the 11-min stimulation period.

Fig. 2. 3D views of t-maps (p<0.01) between TIBS and placebo conditions based on Alpha-band (8-13 Hz) power density at 6, 8, 9, 10, and 11 min after right-side prefrontal TIBS was initiated. EEG data were averaged over 20 subjects.

5. Discussion and Conclusion

We performed a placebo-controlled experiment to record EEG data from 20 human subjects before, during, and after prefrontal TIBS. A gradual and strong dose-dependent increase of EEG power in the Alpha band was observed, suggesting that electrophysiological signals in cortical networks are modulated by the transcranial application of coherent infrared light.
Case Summary: A 61-year-old man with treatment-resistant progressive ME/CFS was referred for consultation. His condition, likely virally induced decades before, was characterized by recurring periods of extreme fatigue, which had become unrelenting over the prior two years, impairing many dimensions of his life. Expert immunological and neurological work-ups were negative. Fibromyalgia had been ruled out. The patient failed many medically advised approaches, including empirical antidepressant trials of Fluoxetine and Bupropion, an extensive course of acupuncture and a gluten-free diet. Presenting complaints: For the prior two years his life was severely circumscribed due to severe, persistent fatigue, with inability to exercise. He social life was curtailed as he focused his limited energy on teaching and his family. His sleep was intact, without difficulty initiating or maintaining asleep. He was not depressed. On the Montgomery-Asberg Depression Rating Scale (MADRS) his score was 5 with notable lassitude. On the Patient Health Questionnaire (PHQ-9) his score was 3 with notable low energy nearly every day. Childhood Medical History: At age of 2 the patient had serious neurological problems including weakness of lower extremities with abnormal gait. He was suspected of having polio and endured a very stressful quarantine in the hospital for several weeks. No diagnosis was confirmed and his condition resolved with normal development. Adult Medical History: In his early 20’s while travelling abroad in a third world country he had acute illness with high fevers and delirium. No diagnosis was confirmed, though viral illness was suspected. His problems with intermittent severe fatigue problems began within the following year.

3. Methods

We considered his ME/CFS as a treatment-resistant, chronic, neuro-inflammatory syndrome, likely related to his earlier infections. Limited genetic analysis through Genomind, Inc. revealed COMT Val/Val variant implying that the patient might have better response to dopaminergic agents and/or neuromodulation [3]. The patient failed trials of Vyvanse (long-acting amphetamines) and Concerta (long-acting methylphenidate), but Provigil (modafinil) 200 mg provided partial relief of fatigue. Further analysis revealed brain-derived neurotrophic factor (BDNF V66M) variants of Met/Val. In light of the failure of all previous treatment modalities, we decided that neuromodulation was a valid treatment approach. TDCS was chosen as a safe off-label neuromodulation treatment option, allowing cumulative, ongoing treatment to target persistent chronic neuro-inflammation. Patient was trained with tDCS in the office and then treatment was self-administered at home daily. The protocol was: anode (left dorsolateral prefrontal cortex), cathode (right dorsolateral prefrontal cortex), 2 mA/min, 20 minutes, 40 mA total dose, using 1.5 cm diameter electrode pads. Patient reported significant benefit early in the day during the first month of daily tDCS. After four weeks, maintenance tDCS sessions were increased to twice daily. Over the next few months the patient experienced full clinical recovery. Follow-up quantitative EEG (qEEG) and neurophysiological testing was done 14 months after initial presentation; pre- and post- comparison studies provide insight into possible treatment biomarkers that may help to explain this patient’s full and unexpected recovery.

4. Results

Patient reported significant benefit early in the day during the first month of daily tDCS. We increased tDCS sessions to twice daily (6 AM and 12 Noon). This provided consistent benefit, though we needed to lower modafinil to 100 mg due to insomnia. Over the next few months, he experienced full recovery with improved exercise tolerance and a return to physical exercise several times each week. During this period, we stopped modafinil, but he felt less well, so it was resumed at 100 mg daily.

Follow-up: We repeated neuropsychological studies 14 months after initial presentation. The patient had been in full remission for almost a year. Before treatment, patient demonstrated normal motor speed, normal reactive variance with no commission errors, but high omission errors (14.29%, selective attention) as compared to the normative database. After treatment, all behavioral measures were within the normal range.

Evoked Reaction Potentials: Pre-treatment visual ERP findings (156 ms, -12.6 mV) improved substantially at post-treatment (180 ms, -13.2 mV).

Heart Metrics: Heart-rate variability (25 ms) and total power (287 ms²) were both low at pre-treatment. Post-treatment heart-rate variability improved significantly (35 ms); total power improved substantially (511 ms²).

We performed qEEG analysis to assess brain wave states that might explain his full and unexpected recovery (see Fig. 1 showing Pre vs. Post qEEG after one year of maintenance tDCS). Before treatment, qEEG showed higher than normal incidence of alpha in both temporal lobes (7-9 Hz), higher than normal incidence of alpha in the right frontal lobe (7-9 Hz). No significant changes were noted. At follow-up, in full recovery, the qEEG showed

Abstracts / Brain Stimulation 10 (2017) e46–e83
e69

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Proceedings #19. Maintenance TDCS: A Case of Full and Durable Recovery From Myalgic Encephalomyelitis/Chronic Fatigue Syndrome (ME/CFS)

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1. Abstract

A middle-aged man with progressive myalgic encephalomyelitis/chronic fatigue syndrome (ME/CFS) was referred for neuromodulation. His condition, likely virally induced decades ago, was characterized by recurring periods of extreme fatigue, lasting months at a time. Medication trials provided partial benefit. Transcranial direct current stimulation (tDCS) was chosen as a neuromodulation treatment, allowing cumulative, ongoing treatment to target ongoing inflammation [1]. Patient was trained with tDCS in the office and then treatment was self-administered at home daily. The protocol was: anode (left dorsolateral prefrontal cortex), cathode (right dorsolateral prefrontal cortex); 2 mA/min, 20 minutes, 40 mA total dose, using 1.5 cm diameter electrode pads. Patient reported significant benefit early in the day during the first month of daily tDCS. After four weeks, maintenance tDCS sessions were increased to twice daily. Over the next few months the patient experienced full clinical recovery. Follow-up quantitative EEG (qEEG) and neurophysiological testing was done 14 months after initial presentation; pre- and post- comparison studies provide insight into possible treatment biomarkers that may help to explain this patient’s full and unexpected recovery.

2. Introduction

Chronic neuro-psychiatric diseases are the third leading cause of disability worldwide [2]. Individuals who experienced adverse childhood events, including medical illness or developmental trauma, have higher risk for altered immunity and stress response increasing the risk of medical illnesses later in life. Myalgic encephalomyelitis/chronic fatigue syndrome (ME/CFS), like other early immune activation syndromes, is poorly understood and subsequently lacks reliable and effective treatment options. Finding efficacious, durable, and efficient interventions that might increase chance of full and lasting recovery are a clinical priority.