Evaluation of the effect of TDCS on TMS-EEG responses in patients with disorders of consciousness

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Introduction
Due to life-saving medical advances, a more commonly occurring clinical issue is the diagnosis and treatment of disorders of consciousness (DOC). One recently developed intervention option has been non-invasive transcranial direct current stimulation [1]. While this approach has had some success, there are also several patients who show no change in behavioural state after stimulation. This dichotomy of patient responders may be better understood by investigating the mechanism behind the tDCS intervention. The combination of transcranial magnetic stimulation and electroencephalography (TMS-EEG) has been an important diagnostic tool in DOC patients [2,3]. The resulting neural complexity, guided by underlying mechanisms of bistability are able to uniquely classify DOC patients [4]. We therefore examined the neural response using TMS-EEG both before and after tDCS in a group of DOC patients.

Methods
Seven patients (3 diagnosed as in a minimally conscious state and 3 with unresponsive wakefulness syndrome; 4 female; mean age 34.7) participated in the study. The anodal portion of the tDCS was applied over the left dorsolateral prefrontal cortex with the cathode over the right supra-orbito frontal area for a total duration of 20 minutes [1]. 400 individual TMS pulses were applied to the premotor cortex using patient-specific MRI-guided neuronavigation to ensure stimulating structurally intact brain regions and consistent localisation both within and across sessions [5].

Analysis focused on two key measures of bistability in the TMS-EEG response: slow wave activity, and high-frequency suppression. Given the inherent variability in responses across individual (yet consistent patterns across sessions), we qualified the slow activity as the minimum activity across all channels in 3 time windows (baseline, -400 to -200 ms; early, 0 to 200 ms; and later, 200 to 400 ms). High-frequency suppression was measured after spectral decomposition as reduced activity between 20 and 50 Hz over similar time windows. We also looked at the relationship between slow activity and high-frequency suppression. Last, we detected and analysed individual slow waves in the resting state EEG recordings before and after tDCS.

Results
None of the seven patients showed a reliable behavioural change after tDCS. We did however find that the overall evoked slow activity was significantly reduced following tDCS intervention. While no single channel was significantly reduced at the group level, the minima across channels at a variety of percentile thresholds showed significant reduction (55% to 95% minimal activity). As expected, we also found a significant positive correlation between the strength of the slow activity and the amount of high-frequency suppression. However, there was no significant pre-post tDCS difference in high-frequencies.

In the resting state EEG, we observed that both the incidence of slow waves (measured as inter-wave interval) and the positive slope of the wave were significantly affected by tDCS at the group level. No changes were found for either wave amplitude or duration.

Discussion
Taken together, these results suggest that the tDCS intervention can reduce the slow wave activity component of bistability but this may not directly affect high-frequency activity. Given any lack of behaviour effects in this subset of patients, we hypothesise that while reduced slow activity may be necessary for recovery of neural function, especially consciousness, this alone is insufficient.

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References


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