[8] Poreisz C, Boros K, Antal A, Paulus W. Safety aspects of transcranial direct current stimulation concerning healthy subjects and patients. Brain Res Bull 2007;72:208–14.
[9] Hahn C, Rice J, Macuff S, et al. Methods for extra-low voltage transcranial direct current stimulation: current and time dependent impedance decreases. Clin Neurophysiol 2013;124:551–6.
[10] Lagopoulos J, Degabriele R. Feeling the heat: the electrode-skin interface during tDCS. Acta Neuropsychiatr 2008;20:98–100.

Integration of Cortical Brain Stimulation and Exposure and Response Prevention for Obsessive-compulsive Disorder (OCD)

Dear Editor:

Individuals diagnosed with obsessive-compulsive disorder (OCD) reliably evidence deficits in cognitive control [1] and recent findings suggest hyperactivity of the pre-supplementary motor area (pre-SMA) mediates deficits in cognitive control in OCD [2]. Six studies have attempted to treat OCD with low frequency repetitive transcranial magnetic stimulation (LF-rTMS) of the pre-SMA [3,4]. Much like the psychosocial treatment of choice, exposure and response prevention (ERP) [5], however, many OCD patients do not respond to TMS and symptom remission is rare [3,4]. While LF-rTMS of the pre-SMA is effective for symptoms related to incompleteness (e.g., symmetry) it has limited effectiveness for symptoms related to harm avoidance (e.g., responsibility/checking) [6]. Integration of rTMS and ERP may mitigate shortcomings of each individual treatment and improve treatment efficacy.

Here we present the effects of integrated LF-rTMS and ERP for a patient who showed minimal response to psychopharmacology.

AC was a 52-year-old, middle-class, Caucasian, male. After providing written informed consent, AC completed a pre-treatment Mini International Neuropsychiatric Interview, which revealed that he met DSM-IV-TR criteria for OCD, Recurrent MDD, and Social Anxiety Disorder. AC reported that his OCD began in childhood and became clinically significant during early adulthood. At the time of intake he reported that he spent an average of 1–3 h obsessing and 3–8 h ritualizing per day. AC reported that his OCD interfered with personal relationships and, while he was able to maintain full time employment, his OCD interfered with his professional life.

Upon presenting for treatment, AC was prescribed fluvoxamine, quetiapine, temazepam, and clonazepam. AC reported minimal response to medications. AC discontinued clonazepam at the end of the first week of treatment. AC remained on stable doses of all remaining medications for the duration of the study. AC had no history of evidence-based psychotherapy.

AC was enrolled in the active TMS arm of a non-randomized pilot intervention study. AC received active rTMS but was blinded to the TMS treatment; meaning, he was told that he may receive real or sham (placebo) TMS as part of the study. LF-rTMS treatment strategies mimicked previously reported methods [7,8]. LF-rTMS was delivered to the pre-SMA along the sagittal midline (50% of the distance between the Fz and FCz). 1 Hz pulses were delivered to the pre-SMA using a figure-8 coil for 20 min per session (1200 total pulses) at 110% of resting motor threshold. LF-rTMS was delivered immediately prior to ERP for the duration of the 3-week integrated TMS and ERP period for a total of 15 rTMS treatments (weeks 2–4).

ERP strategies mimicked those described by Foa and colleagues [9]. Psychoeducation and hierarchy development were completed one week prior to TMS and ERP. During the integrated TMS and ERP period, 90–120 min therapist assisted exposure sessions were conducted each weekday [15 sessions (weeks 2–4)]. Finally, 8, weekly, 45 min, ERP-only maintenance sessions were completed (weeks 5–12). An emphasis was placed on *in vivo* exposure, but imaginal exposures were also included. Complete response prevention was encouraged and homework was assigned daily.

AC's pre-treatment (week 1) Yale-Brown Obsessive-Compulsive Scale (YBOCS) score was 25, suggesting severe OCD. AC's YBOCS increased following education and planning (week 2 YBOCS = 28). AC's YBOCS decreased after the first week of TMS + ERP (week 3 YBOCS = 22) but drastically decreased after 2 weeks of TMS + ERP (week 4 YBOCS = 13). After the final week of TMS + ERP, AC's YBOCS reflected a 54% reduction from pre-treatment (week 5 YBOCS = 11) (Fig. 1). YBOCS reductions were maintained through the 8-week ERP maintenance phase and AC's final YBOCS reflected a 64% decrease in obsessions and compulsions (week 12 YBOCS = 9).

Consistent with clinician-rated YBOCS scores, AC's pre-treatment self-reported OCD was severe [Dimensional Obsessive-Compulsive Scale (week 1 DOCS-total = 36)]. Pre-treatment DOCS scores suggested symmetry obsessions and compulsions were AC's primary symptoms (DOCS-symmetry = 13). AC also reported significant contamination/washing (DOCS-contamination = 8) and responsibility/checking (DOCS-responsibility = 10) symptoms. DOCS scores followed patterns of change that were similar to those seen for YBOCS scores. At the end of the TMS + ERP phase, AC's self-reported OCD severity was reduced by 50% (week 5 DOCS-total = 18) and reductions were maintained through the ERP maintenance phase (week 12 DOCS-total = 19). Importantly, AC reported roughly equivalent reductions across all symptom dimensions (week 12 DOCS-symmetry = 6, DOCS-contamination = 4, DOCS-responsibility = 5).

AC's pre-treatment General Anxiety Disorder Scale (GAD-7) and Patient Health Questionnaire (PHQ-9) scores were above clinical cut-offs and were suggestive of severe anxiety (week 1 GAD-7 = 20) and moderately-severe depression (week 1 PHQ-9 = 18). AC's GAD-7 and PHQ-9 scores were below clinical cut-offs following the TMS + ERP phase (week 5 GAD-7 = 8 and PHQ-9 = 7). Reductions in anxiety and depression were maintained through the ERP maintenance phase (week 12 GAD-7 = 7 and PHQ-9 = 5).

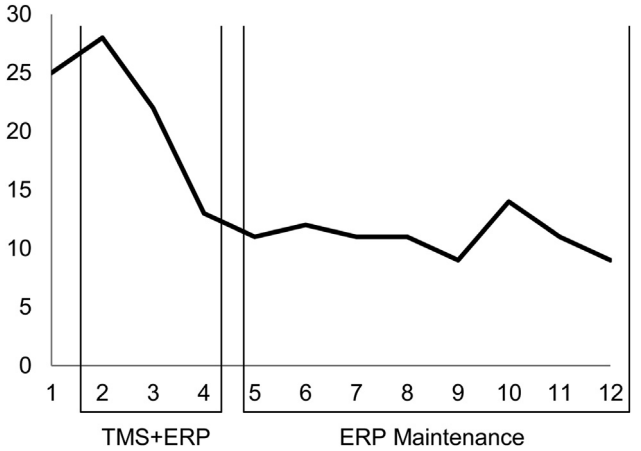

Figure 1. Change in Yale-Brown Obsessive-Compulsive Scale (YBOCS) scores over the course of treatment. YBOCS was completed at the beginning of each week. As such, scores at any given week reflect the effects of previous weeks.

Anecdotally, AC doubted his diagnosis of OCD and the relevance of ERP at the beginning of treatment. AC's compliance with response prevention and willingness to complete difficult exposures dramatically increased between the first the third weeks of treatment. For example, during the weekend after the second week of TMS + ERP, AC assigned himself and completed exposures at the top of his hierarchy. Afterward he was both proud of his achievements and surprised at his willingness to do that which he considered reckless two weeks prior.

The case of AC highlights the potential of integrated TMS and ERP and the need for additional research into treatments that integrate brain stimulation with psychotherapy, particularly for disabling and treatment resistant forms of psychopathology such as OCD. The present case of integrated TMS + ERP was effective for all obsessive-compulsive symptom dimensions and resulted in large and rapid symptom reductions. This suggests that the inclusion of ERP with TMS may mitigate shortcomings of LF-rTMS of the pre-SMA for OCD and suggests that the addition of TMS may improve the speed of ERP. Well-controlled research will be necessary to establish if TMS and ERP have additive effects and to determine how these interventions may synergistically augment neural functioning to affect manifest symptoms.

Acknowledgments

The authors would like to thank Dr. Ron Acierno, Ph.D. for his assistance with this case study.

The authors have no known conflicts of interest associated with this publication and there has been no significant financial support for this work that could have influenced its outcome.

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Received 30 May 2014
Available online 18 July 2014
http://dx.doi.org/10.1016/j.brs.2014.06.010

References

[1] Chamberlain S, Blackwell A, Fineberg N, Robbins TW, Sahakian BJ. The neuropsychology of obsessive compulsive disorder: the importance of failures in cognitive and behavioural inhibition as candidate endophenotypic markers. Neurosci Biobehav Rev 2005;29:399–419.
[2] de Wit S, de Vries F, van den Heuvel O, et al. Presupplementary motor area hyperactivity during response inhibition: a candidate endophenotype of obsessive-compulsive disorder. Am J Psychiatry 2012;169:1100–8.
[3] Berlim Marcelo T, Neufeld NH, Van den Eynde F. Repetitive transcranial magnetic stimulation (rTMS) for obsessive–compulsive disorder (OCD): an exploratory meta-analysis of randomized and sham-controlled trials. J Psychiatr Res 2013;47:999–1006.
[4] Blom R, Figee M, Vulink N, Denys D. Update on repetitive transcranial magnetic stimulation in obsessive-compulsive disorder: different targets. Curr Psychiatry Rep 2011;13:289–94.
[5] Schruers K, Koning K, Luermans J, Haack MJ, Griez E. Obsessive–compulsive disorder: a critical review of therapeutic perspectives. Acta Psychiatr Scand 2005;111:261–71.
[6] Mantovani A, Shubeck J, Gowatsky J, Simpson HB, Greenberg B. P 247. Incompleteness and harm avoidance in obsessive compulsive disorder: different response to transcranial magnetic stimulation. Clin Neurophysiol 2013;124:e182–3.
[7] Mantovani A, Simpson H, Fallon B, Rossi S, Lisanby SH. Randomized sham-controlled trial of repetitive transcranial magnetic stimulation in treatment-resistant obsessive–compulsive disorder. Int J Neuropsychopharmacol 2010;13:217–27.
[8] Oliveira Gomes P, Brasil-Neto J, Allam N, Rodrigues de Souza E. A randomized, double-blind trial of repetitive transcranial magnetic stimulation in obsessive-compulsive disorder with three-month follow-up. J Neuropsychiatry Clin Neurosci 2012;24:437–43.
[9] Foa E, Liebowitz M, Tu X, et al. Randomized, placebo-controlled trial of exposure and ritual prevention, clomipramine, and their combination in the treatment of obsessive-compulsive disorder. Am J Psychiatry 2005;162:151–61.

Skin Lesions Induced by Transcranial Direct Current Stimulation (tDCS)

For several years, at the Institute Guttmann Neurorehabilitation Hospital (Barcelona, Spain), we have been applying Transcranial direct current stimulation (tDCS) for the management of Neuropathic pain after spinal cord injury. TDCS has been established as useful therapeutic option for patients with neuropathic pain [1]. Several recent studies demonstrate its efficacy, good tolerance and minimal side effects [2,3]. Our accumulated experience spans to having treated more than 100 patients, using always the same standardized protocols. Direct current is delivered with a battery-driven, constant current stimulator (NeuroConn, Ilmenau, Germany) and two surface sponge electrode pads (7 × 5 cm, 35 cm^2) soaked with a saline solution (0.9% NaCl; 308 mosm/l). The anode is placed over C3 or C4 (EEG 10/20 system) aiming to target the motor cortex, and the cathode over the contralateral supraorbital area [4,5]. The electrodes were fastened into position by using two rubber polyester headbands (70 cm × 3 cm). For patients with asymmetric pain, the anode is placed contralateral to the afflicted body part, while for patients with symmetric pain, the anode is placed over the dominant hemisphere. A constant current of 2 mA intensity is applied in daily sessions of 20 min (Current was ramped-up for 15 s until it reached 2 mA; and finally the device was turned-off with a ramp-down of 15 s) during a period of 2 weeks (from Monday to Friday; total of 10 treatment sessions). After each session and patient, the material was replaced and cleaned with soap and water.

Here we report on three cases of skin burns during this tDCS treatment. All three affected patients were men with little hair on their scalp. The three were stimulated with the same parameters getting values of impedance of 3–4.5 kΩ. Their skin's surface was not cleaned with alcohol because the impedance levels were correct. All reported a usual tingling and itching sensation under the electrodes, but none experienced significant discomfort or pain. Following our standard operating procedures, constant electrode's moistness, electrode position and impedance levels were controlled and stable during stimulation sessions.

All patients showed a mild redness of the skin under the central part of the electrodes after the tDCS session but it disappeared after few minutes without discomfort. However, in all three instances, the skin lesions occurred under the cathode (supraorbital region)