Clinical Psychology and Special Education
2020, vol. 9, no. 3, pp. 105–126.
DOI: 10.17759/cpse.2020090308
ISSN: 2304-0394 (online)

Клиническая и специальная психология
2020. Том 9. № 3. С. 105–126.
DOI: 10.17759/cpse.2020090308
ISSN: 2304-0394 (online)

Case Series of Transcranial Direct Current Stimulation as an Augmentation Strategy for Attention Bias Modification Treatment in Adolescents with Anxiety Disorders

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This article presents the results of a case series to assess the feasibility, acceptability, and clinical promise of transcranial Direct Current Stimulation (tDCS) as an augmentation strategy in clinic referred adolescents. Attention Bias Modification Treatment (ABMT) is a computer-based attention-training protocol designed to reduce rapidly deployed attention orienting to threat and thereby reduce anxiety symptom severity. Studies of ABMT reveal overall small to medium effect sizes. Advances in the neural underpinnings of attention to threat and attention-training protocols suggest the potential of tDCS of the dorsolateral prefrontal cortex (dlPFC) as a novel augmentation strategy to enhance ABMT's efficacy (ABMT + tDCS). However, tDCS has never been tested in a sample of adolescents with anxiety disorders. Six adolescents with a primary anxiety disorder completed all four ABMT + tDCS sessions. Adverse effects were mild and transient. Adolescents and parents independently reported fair to excellent levels of satisfaction. Impairment ratings of the primary anxiety disorder significantly decreased. Further, electrophysiological data recorded via electroencephalography (EEG) suggested decreases in neural resources allocated to threat. These findings support the feasibility, acceptability, and clinical promise of tDCS as an augmentation strategy in adolescents with anxiety disorders, and provide the impetus for further investigation using randomized controlled designs in larger samples.

Keywords: anxiety, adolescents, attention bias modification, transcranial direct current stimulation, neuromodulation, electroencephalography (EEG), event related potential (ERP).

Funding. The reported study was funded by the National Institutes of Health (R25 GM061347, UH2 MH101470, and R01 MH119299). The content is solely the responsibility of the authors and does not necessarily represent the official views of the National Institutes of Health.

Acknowledgements. The authors gratefully acknowledge Dr. Greg Hajcak for his contributions to this study.

For citation: Vaclavik D., Bechor M., Foster A., Gralnik L.M., Bar-Haim Y., Pine D.S., Bikson M., Silverman W.K., Reeb-Sutherland B.C., Pettit J.W. Case Series of Transcranial Direct Current Stimulation as an Augmentation Strategy for Attention Bias Modification Treatment in Adolescents with Anxiety Disorders. Klinicheskaia i spetsial'naia psikhologiia=Clinical Psychology and Special Education, 2020. Vol. 9, no. 3, pp. 105–126. DOI: 10.17759/cpse.2020090308

Introduction

Despite the established efficacy of cognitive behavioral therapy (CBT) and antidepressant medications for the treatment of anxiety disorders in adolescents, treatment protocols are lengthy, and up to one-half of anxious adolescents do not respond well to treatment [15; 53; 59]. This highlights the need for alternative, brief, and efficacious treatments. In this case series, we present data on Attention Bias Modification Treatment (ABMT) augmented with transcranial Direct Current Stimulation (tDCS) as an alternative, brief treatment for anxiety in adolescents.

Behavioral and neuroscience research extensively documents that adolescents with anxiety disorders show heightened attention to threat such as angry faces [3; 17; 59]. Neural regions corresponding to attentional processes develop rapidly during adolescence, suggesting a developmental window of opportunity for delivering treatments that target these neural regions subserving attention to threat. The most commonly used paradigm for assessing attention to threat has been through behavioral responses (i.e., reaction times) to a visual probe dot-probe task. Research using behavioral responses to the dot-probe task provides support for an association between attention bias to threat and anxiety (for reviews see [24; 37]); however, behavioral responses calculated from reaction times are a distal measure of attention, evidenced by poor internal consistency and test-retest reliability [10; 29; 45; 48]. Event-related potentials (ERPs), the neural activation in response to a discrete event, are a common measure for assessing the chronometry of neural activity in relation to attention [54] and provide a more proximal measure of attention to threat than traditional behavioral reaction time measures [36].

Evidence supports ERP components P1, P2, and P3 as potential neural measures of attention to threat [2; 19; 41; 54; 57]. P1 and P2 represent early stage processes involved in attention orienting and detection of threat [27; 31], whereas P3 represents later stage processes involved in the strategic regulation of attention (i.e., attentional control [18; 26; 49]).

ABMT is the translational treatment implication of heightened attention to threat that shows promise for reducing anxiety and its disorders (e.g., [21; 32; 35], including in adolescents [34; 39; 42; 44]. Despite the promise of ABMT, effect sizes on changes in attention to threat and anxiety symptom severity are usually small to medium in samples of children and adolescents with anxiety disorders (for reviews see [32; 34; 37; 46]). These small to medium effect sizes highlight the critical need for novel therapeutics that could be used as augmentation strategies to reduce attention to threat and enhance the anxiety reducing effects of ABMT.

Advances in the neural underpinnings of attention to threat and attention training suggest that tDCS of the dorsolateral prefrontal cortex (dlPFC) may enhance the effects of ABMT [14; 24; 25]. tDCS is a noninvasive technique that facilitates spontaneous neuronal activity and plasticity in specific areas of the brain by applying electrical current to corresponding regions of the scalp [7; 13]. Heightened attention to threat signals corresponds to perturbations in amygdala-prefrontal cortex (PFC) circuitry. The amygdala

facilitates vigilance through rapid threat processing, whereas the ventro- and dorsolateral PFC (vlPFC and dlPFC) facilitate regulation of attentional deployment to threat [20; 22; 23; 56]. ABMT targets neural circuitry subserving attention to threat [9; 11; 55; 60], and tDCS of the dlPFC enhances neuronal activity and plasticity [7; 28; 50]. Thus, it is reasonable to expect that tDCS of the dlPFC during ABMT (ABMT + tDCS) would enhance ABMT's effects.

Data from two studies support this expectation [14; 24]. In Clarke et al., 77 nonreferred college students with trait anxiety in the middle quartiles of a sample distribution were randomly assigned to either anodal tDCS of the dlPFC or sham stimulation while completing a single ABMT session [14]. Participants who received active stimulation showed significant changes in attention to threat in the expected direction compared to participants who received sham stimulation. In Heeren et al., 56 nonreferred college students with elevated trait anxiety were randomly assigned to anodal tDCS of the dlPFC, cathodal tDCS to inhibit activity of the dlPFC, or sham stimulation while completing a single ABMT session [24]. Compared to participants who received cathodal or sham stimulation, participants who received anodal tDCS of the dlPFC displayed significant reductions in attention to threat. Together these data from single sessions in nonreferred samples show that stimulation of the dlPFC enhances ABMT effects on attention to threat. However, neither of these studies used a sample of clinic-referred adolescents with anxiety disorders, used a standard multi-session protocol of ABMT, or reported on anxiety outcomes.

The purpose of the present case series was to examine the feasibility, acceptability, and clinical promise of tDCS of the dlPFC as an augmentation strategy for a standard multi-session ABMT protocol in referred adolescents who met criteria for anxiety disorder diagnoses. We hypothesized that participants and their parents would find the treatment acceptable and that participants would experience reductions in anxiety severity and attention to threat, as indicated by lower amplitudes on P1, P2, and P3 ERP components during the presentation of angry faces on the dot-probe task. Such data lay the groundwork for further testing and clinical administration of ABMT + tDCS.

Research Program

Participants

For study inclusion, participants had to be between the ages of 13 and 17 and were required to meet criteria for a primary Diagnostic and Statistical Manual of Mental Disorders-4th Edition (DSM-IV [1]) diagnosis of Social Phobia/Anxiety Disorder (SOP) or Generalized Anxiety Disorder (GAD) using the Anxiety Disorders Interview Schedule for Children (Child and Parent Versions; ADIS-IV:C/P [52]). Exclusionary criteria were (1) organic mental disorders, psychotic disorders, pervasive developmental disorders or attention deficit hyperactivity disorder, (2) severe risk of suicide, (3) serious and uncorrected vision problems, (4) physical disability that would interfere with ability to perform the dot probe task, (5) left-handedness or ambidexterity, (6) current psychotropic medication, and (7) seizure disorder or recent traumatic brain injury.

Participants were recruited from a university-based outpatient clinic specializing in the treatment of child and adolescent anxiety in a large urban area. Ten families on the clinic's waitlist were identified and contacted by telephone to share information about this study. The parents of all 10 adolescents expressed interest in participating. One adolescent declined due to distance and travel time, and three adolescents declined without providing a reason. These four adolescents also declined clinical services unrelated to a research study (i.e., cognitive behavioral therapy) and never came to our clinic for an intake assessment. The remaining six families consented to participate (i.e., parents provided informed consent and adolescents provided assent) and completed the pretreatment assessment. Age, sex, and diagnoses for each participant are provided in Table 1. Four participants identified as white Hispanic/Latino and two identified as non-Hispanic white.

Table 1

Demographic, diagnostic, and symptom data

Sex	Age	DSM-IV-TR Diagnoses		ADIS Impairment Ratings				PARS		SCARED				CSQ	
				Parent	Parent	Child	Child			Parent	Parent	Child	Child	Parent	Child
		PRE	POST	PRE	POST	PRE	POST	PRE	POST	PRE	POST	PRE	POST	POST	POST
F	14	SAD	SAD	6	4	6	4	14	12	10	10	60	37	31	16
F	16	GAD	GAD	5	-	7	4	17	14	37	13	42	44	32	26
F	15	SAD	SAD	4	4	4	-	13	10	5	12	21	24	29	28
M	16	GAD	No Dx	-	-	7	-	22	9	26	15	46	34	31	32
F	16	SAD	SAD	6	8	7	7	23	19	53	60	68	56	19	18
F	14	SAD	SAD	8	6	7	7	25	24	45	39	41	52	29	18

Notes. SAD – Social Anxiety Disorder; GAD – Generalized Anxiety Disorder; No Dx – diagnosis was not endorsed by neither parent nor child. ADIS – Anxiety Disorders Interview Schedule for Children; PARS – Pediatric Anxiety Rating Scale; SCARED – Screen for Child Anxiety Related Emotional Disorders; CSQ – Client Satisfaction Questionnaire. F – Female; M – Male. PRE – pretreatment; POST – posttreatment.

Procedures

This study was conducted as approved by the Institutional Review Board. Participants were compensated for their time completing the assessments. Participants completed a pretreatment assessment to determine anxiety diagnosis and severity, as well as to obtain a baseline measure of attention bias to threat. Participants then completed four weekly treatment sessions (ABMT + tDCS; described below), and a posttreatment assessment conducted one week after treatment sessions finished, all at the university clinic. Acceptability, feasibility, and satisfaction were measured by the (1) number of eligible families who consented to participate, (2) number of adolescent participants who completed the study, (3) adverse effects and acclimation to stimulation, and (4) adolescent

and parent ratings of satisfaction with treatment. Neural resources allocated to nonthreatening and threatening stimuli (i.e., attention to threat) were measured using event related potentials (ERPs) during an emotional faces dot-probe task [5; 19; 54]. A multi-informant anxiety outcome assessment was conducted, which included clinician-, parent-, and adolescent self-ratings of anxiety symptom severity and impairment.

Attention Bias Modification Treatment (ABMT). The ABMT protocol (TAU-NIMH ABMT initiative) was the same as has been used in previous studies of ABMT in children and adolescents [42; 43]. The training protocol is identical to the attention to threat measurement (i.e., dot-probe) task with three exceptions: (1) a different set of faces was used during treatment than during measurement; (2) each block consisted of 160 trials instead of 240 during measurement, presenting 120 angry–neutral and 40 neutral–neutral combinations, with two consecutive blocks completed per treatment session; and (3) the probe replaced the neutral face on 100% of trials to establish a training contingency between neutral face location and probe location. On average, completion of the training protocols took participants 15 minutes.

Transcranial Direct Current Stimulation (tDCS). tDCS was administered using a Soterix 1X1 tDCS Limited Total Energy (LTE) Stimulator while adolescent participants completed the ABMT protocol at each session. Direct current was transferred via two 5 cm x 5 cm conductive silicone electrodes each within a disposable 5 cm x 7 cm saline-soaked (6 ml per side) sponge pouch. The anodal (active) electrode was secured over the left dlPFC, localized using F3 on the 10/20 international EEG system, and the cathodal (reference) electrode was secured over the contralateral supra-orbital area [24]. The intensity of tDCS was 1 mA with a 30 sec ramp up/down time. Stimulation began two minutes before the initiation of the ABMT protocol to allow for a ramp up period and acclimation to stimulation before training began. Stimulation was administered continuously for 20 minutes after ramp up. Once the full intensity (1 mA) was reached and adolescents had two minutes to acclimate, the ABMT protocol began. In cases where adolescents reported adverse effects/discomfort that did not abate during the two-minute period, we extended the acclimation period until discomfort ceased or an additional five minutes had passed. At the end of the additional five minutes, the ABMT protocol was initiated and tDCS was administered at 1 mA or the maximum intensity tolerable to the adolescents (e.g., 0.8 mA). Participants were instructed that they could discontinue at any time.

Measures

Anxiety Diagnosis, Impairment, and Symptom Severity. Youth anxiety disorders were assessed using the Anxiety Disorders Interview Schedule for Children, Child /Parent Versions (ADIS-IV: C/P [51]). The ADIS was administered independently to parent and adolescent. Anxiety-related impairment was assessed using a severity/impairment rating scale as reported by each informant, scored from 0 to 8. A severity/impairment rating ≥ 4 was used as a clinical cutoff of impairment for diagnosis. Youth anxiety symptom severity was assessed using clinician ratings on the Pediatric Anxiety Rating Scale (PARS [47]) and adolescent and parent ratings on the Screen for Child Anxiety Related Emotional Disorders – Child/Parent Versions (SCARED-C/P [8]). All anxiety measures were administered by trained and supervised psychology doctoral students.

tDCS Adverse Effects. Adverse effects were assessed using the tDCS Adverse Effects Questionnaire [12], a clinician-administered rating scale that assesses the severity of ten adverse effects on a 4-point likert scale (1 – "absent", 2 – "mild", 3 – "moderate", 4 – "severe"). It was administered at each session immediately before stimulation, to obtain a baseline measurement, and immediately after the end of stimulation, to compare to baseline and determine if adverse effects were related to stimulation (1 – "none", 2 – "remote", 3 – "possible", 4 – "probable", 5 – "definite"). A score ≥ 3 indicated an adverse effect of stimulation.

Client Satisfaction. Satisfaction with treatment was assessed using the Client Satisfaction Questionnaire-8 (CSQ-8 [30]). Adolescents and parents independently rated the quality of and satisfaction with treatment on a 4-point likert scale (1 – "poor/quite dissatisfied", 2 – "fair/indifferent or mildly dissatisfied", 3 – "good/mostly satisfied", 4 – "excellent/very satisfied"). Overall satisfaction ratings were categorized as ''poor'' (8–13), ''fair'' (14–19), ''good'' (20–25), and ''excellent'' (26–32).

Attention to Threat. Consistent with prior research, the angry/neutral faces dot-probe task (TAU-NIMH ABMT initiative; http://people.socsci.tau.ac.il/mu/anxietytrauma/research/) was used while EEG/ERP data were collected to obtain a measure of attention to threat [6; 19; 54]. In the task, participants are presented with 240 trials. In each trial, a white fixation cross appears in the center of the screen for 500 milliseconds (ms), followed by a pair of faces of the same actor for 500 *ms*, arranged vertically (one above the other). In each trial, the faces display one of three combinations: neutral-angry, angry-neutral, or neutral-neutral. This is immediately followed by a visual probe ("<" or ">") replacing either the top or bottom face. Participants are asked to respond as fast as possible and indicate the orientation of the probe by clicking the left or right mouse button (left for "<" and right for ">") using their dominant hand. The probe remains on the screen until participants respond, and the next trial starts immediately. Angry-face location, probe location, probe type, and actor are fully counterbalanced.

Electrophysiological Recording and Processing. Participants were fitted with a 64-channel Geodesic Sensor Net (Electrical Geodesics Inc., Eugene, OR) previously soaked in a non-toxic, potassium chloride solution for 10 minutes. The raw EEG signal was amplified using an high-impedance EGI NetAmps 400 amplifier and sampled at 1000 Hz. Impedance values were checked and adjusted to be below 50 kΩ prior to data collection. EEG was recorded continuously during the completion of the dot-probe task and was referenced to Cz after artifact rejection. Continuous raw EEG was processed using EEGLab [16] and ERPLab [33]. Offline, data were resampled to 512 Hz and filtered using a high pass filter of .1 Hz and a low pass filter of 30 Hz. Data was segmented into epochs with a 200 ms baseline period and 500 ms post-face stimulus onset period. Data were baseline corrected to the average voltage during the 200 ms prior to stimulus onset. Epochs were processed for artifacts using a voltage threshold of ± 75 μV. Remaining epochs were visually inspected for ocular and motor artifacts. Of the trials not rejected, individual bad channels were identified and replaced using spherical spline interpolation. Individual subject averages were constructed separately for neutral-neutral (NN) and neutral-threat (NT) stimuli.

Stimulus-Evoked ERP Components. ERP neural activity was time-locked to the onset of the face stimuli. Specific components of interest were P1, P2, and P3. In line with previous studies, P1 and P2 components were examined at midline occipital sites (Oz or E37 [4; 19; 38; 40]), and the P3 component was examined at frontal sites (Fz or E4 [18]). ERP latency windows were determined by visually inspecting grand average waves. Peak and mean amplitudes for P1 (0-100 ms), P2 (150-250 ms), and P3 (280-400 ms) were generated separately for NN and NT trials in ERPLAB and data were imported into SPSS version 22.0 [53] for final statistical analysis. In addition, for each ERP component at pretreatment and again at POST, we computed ΔNT-NN scores as the difference between the amplitudes for NT and NN trials. ΔNT-NN scores indicate the relative allocation of neural resources when a threat stimulus is present (NT trials) versus when only neutral stimuli are present (NN trials). We examined pretreatment to posttreatment changes in ΔNT-NN scores for each component.

Results

Feasibility, Acceptability, and Satisfaction

All contacted parents expressed interest in participating. All six remaining adolescents completed all four treatment sessions as well as pretreatment and posttreatment assessments. Treatment satisfaction ratings by adolescents and their parents appear in Table 1. Five parents rated satisfaction as Excellent and one as Fair. Three adolescents rated satisfaction as Excellent and three as fair.

Adverse Effects and Dosage Toleration

Figure displays rates of specific adverse effects at each of the four sessions reported in the tDCS Adverse Effects Questionnaire. Excluded adverse effects that were also assessed but not endorsed include: neck pain, scalp pain, burning sensation, and sudden mood change. Following stimulation, the most common adverse effects were mild skin redness, itching, and sleepiness. In session one, three adolescents displayed mild redness, two reported mild itching, one reported mild tingling, and two reported mild sleepiness. In session two, two adolescents displayed mild redness, one reported mild itching, and one reported mild headache. In session three, two adolescents displayed mild redness and one reported mild sleepiness. In session four, three adolescents displayed mild redness, one reported mild itching, one reported mild headache, and four reported mild sleepiness. Moreover, one adolescent reported moderate tingling and a moderate hot sensation in session one. No adolescent reported a severe adverse effect. Adolescents stated that adverse effects decreased during stimulation, and attributed sleepiness to boredom with the procedures.

All adolescents required additional acclimation time beyond the allocated two minutes in at least one session, mostly in earlier sessions, meaning that they requested stimulation be lowered from 1 mA, prior to stimulation being ramped up to full intensity (1 mA). In one session, two adolescents did not reach full intensity after an additional five minutes; after those five minutes, the ABMT protocol was initiated and stimulation was administered at the maximum tolerable intensity.

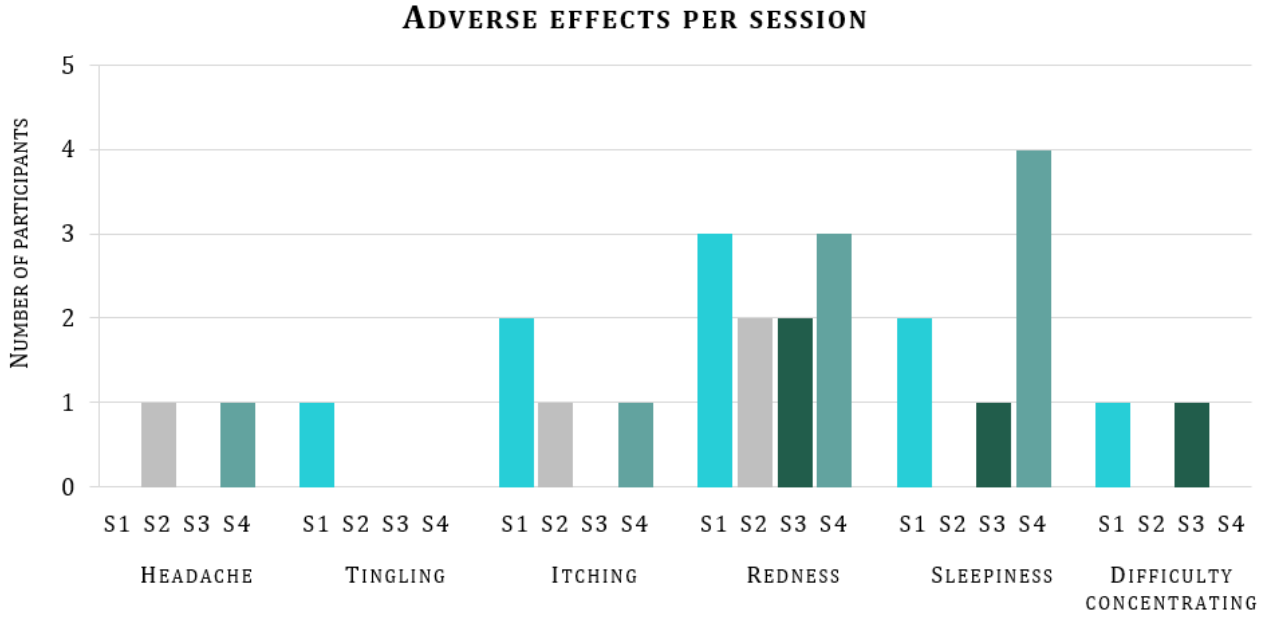

Figure. Distribution of endorsed mild adverse effects per session and participant

Notes. S1, S2, S3, S4 – Sessions 1-4. Figure includes adverse effects that were endorsed for at least one participant at least once. The Adverse Effects Questionnaire was administered before stimulation to determine baseline, and immediately after stimulation. It was considered an adverse effect if it was endorsed immediately after stimulation and not present (or mildly present and increased in intensity) before stimulation (sensations present prior to stimulation were considered to be unrelated to stimulation).

Attention to Threat

Peak and mean amplitudes for ERP components are presented in Table 2. Most effects were not statistically significant, as we expected in this small feasibility study with six cases. We therefore focus our presentation of results on effect sizes. For the P1 component peak amplitude, we found a medium (d = .47) pretreatment to posttreatment decrease on neutral-threat (NT) trials (ns), a large (d = 1.58) pretreatment to posttreatment increase on neutral-neutral (NN) trials ($p < .05$), and a large (d = 4.17) pretreatment to posttreatment decrease in ΔNT-NN (pretreatment: NT > NN; POST: NN > NT; $p < .05$). For the P2 component peak amplitude, we found a large (d = 1.97) pretreatment to posttreatment decrease on NT trials ($p < .05$), a large (d = 1.27) pretreatment to posttreatment increase on NN trials ($p < .10$), and a large (d = 1.73) pretreatment to posttreatment decrease in ΔNT-NN (ns). For the P3 component peak amplitude, we found a small (d = .16) pretreatment to posttreatment increase on NT trials (ns), a small (d = .19) pretreatment to posttreatment decrease on NN trials (ns), and a large (d = 1.46) pretreatment to posttreatment increase in ΔNT-NN (pretreatment: NN > NT; POST: NT > NN; $p < .05$).

For P1 and P2 components mean amplitude, we found medium to large (ds = .61–1.99) pretreatment to posttreatment changes on NN and NT trials (ns), in the same direction as changes in peak amplitude, and large (ds = 1.60–1.77) pretreatment to posttreatment decreases on ΔNT-NN (ns). For P3 mean amplitude, we found small

(*d*s = .21–.35) pretreatment to posttreatment changes on NN and NT trials (ns) and a large (*d* = 1.27) pretreatment to posttreatment increase on ΔNT-NN (pretreatment: NN > NT; posttreatment : NT > NN; p < .05).

Table 2

Peak and mean amplitudes for ERP components

Component	Trial	Pretreatment	Posttreatment	*t*	*d*
Peak Amplitude					
P1 (*M, SD*)	NT	0.95 (0.92)	0.45 (1.19)	0.75	0.47
	NN	-0.20 (1.34)	2.83 (2.36)	-3.61*	1.58
P2 (*M, SD*)	NT	1.32 (0.98)	-1.06 (1.40)	2.85*	1.97
	NN	-0.13 (1.21)	1.55 (1.43)	-1.49	1.27
P3 (*M, SD*)	NT	1.95 (3.12)	2.36 (1.97)	-0.40	0.16
	NN	2.80 (3.49)	2.31 (1.17)	0.45	0.19
Peak Amplitude ΔNT-NN					
P1 (*M, SD*)		1.15 (0.42)	-2.38 (1.12)	2.90*	4.17
P2 (*M, SD*)		1.44 (1.88)	-2.61 (2.73)	2.11	1.73
P3 (*M, SD*)		-1.56 (2.22)	0.96 (1.0)	-3.59*	1.46
Mean Amplitude					
P1 (*M, SD*)	NT	-0.25 (0.10)	-0.77 (1.20)	1.05	0.61
	NN	-1.80 (0.50)	-0.60 (0.69)	-2.11	1.99
P2 (*M, SD*)	NT	-0.61 (0.63)	-1.87 (1.69)	1.86	0.99
	NN	-1.35 (1.42)	-0.40 (0.70)	-1.76	0.85
P3 (*M, SD*)	NT	0.82 (2.61)	1.29 (1.65)	-0.55	0.21
	NN	1.20 (2.95)	0.35 (1.67)	0.81	0.35
Mean Amplitude ΔNT-NN					
P1 (*M, SD*)		1.55 (1.15)	-.71 (1.39)	2.20	1.77
P2 (*M, SD*)		0.74 (1.13)	-1.48 (1.61)	2.10	1.60
P3 (*M, SD*)		-0.63 (2.34)	1.71 (1.13)	-3.11*	1.27

Notes. M – mean; *SD* – standard deviation. NT – Neutral-Threat trials; NN – Neutral-Neutral trials. *N* = 5 (one participant did not complete posttreatment EEG due to a scheduling conflict); *df* = 4; * – *p* < .05.

Anxiety-Related Impairment and Symptom Severity

Participants' demographic and clinical characteristics are presented in Table 1, and pretreatment to posttreatment differences in anxiety-related impairment and symptoms severity are presented in Table 3. Most effects were not statistically significant, as we expected in this small feasibility study with 6 cases. We therefore focus our presentation of results on effect sizes. Mean adolescent impairment ratings on anxiety diagnoses decreased from pretreatment (M = 6.50, SD = 1.38) to posttreatment (M = 2.67, SD = 3.08), with a large effect size (Cohen's d = 1.60). Mean parent impairment ratings on primary diagnosis decreased from pretreatment (M = 6.17, SD = 1.60) to posttreatment (M = 4.83, SD = 2.86), with a medium effect size (Cohen's d = .58). Four of six parents rated the impairment of adolescents' primary diagnosis lower at posttreatment than pretreatment.

Table 3

Mean scores and pretreatment to posttreatment differences for primary diagnoses' impairment ratings, SCARED, and PARS scores

	Pretreatment	**Posttreatment**	***t***	***df***	***p***	***d***
ADIS Impairment-P (*M, SD*)	6.17 (1.60)	4.83 (2.86)	1.40	5	.221	0.58
ADIS Impairment-C (*M, SD*)	6.50 (1.38)	2.67 (3.08)	3.01	5	.030*	1.60
SCARED-P (*M, SD*)	29.33 (19.19)	24.83 (20.29)	0.93	5	.397	0.23
SCARED-C (*M, SD*)	46.33 (16.40)	41.17 (11.91)	1.01	5	.360	0.36
PARS (*M, SD*)	19.00 (5.02)	14.67 (5.79)	2.43	5	.059+	0.80

Notes. ADIS – Anxiety Disorders Interview Schedule for Children. P – parent; C – child/adolescent. M – mean; SD – standard deviation. SCARED – Screen for Child Anxiety Related Emotional Disorders; PARS – Pediatric Anxiety Rating Scale. N = 6; * – $p < .05$; "+" – $p < .10$.

Mean anxiety severity on clinician ratings, adolescent self-ratings, and parent ratings decreased from pretreatment to posttreatment; these decreases were large for clinician ratings (Cohen's d = .80) and small for adolescent self-ratings (Cohen's d = .36) and parent ratings (Cohen's d = .23). For all six adolescents mean clinician-rated PARS ratings decreased from pretreatment (M = 19.00, SD = 5.02) to posttreatment (M = 14.67, SD = 5.79), and for one-half of parents and adolescents mean anxiety ratings on the SCARED-C/P decreased from pretreatment (P: M = 29.33, SD = 19.19; C: M = 46.33, SD = 16.40) to posttreatment (P: M = 24.83, SD = 20.29; C: M = 41.17, SD = 11.91).

Discussion

This case series presents preliminary data on the feasibility and acceptability of tDCS augmentation of ABMT in adolescents with anxiety disorders. Of the 10 families recruited, all parents agreed for their adolescent child to participate, and six adolescents agreed to

participate. Adolescents tolerated treatment well and adverse effects were mild and transient. Additional acclimation time to stimulation was required for all adolescents in at least one session, and two adolescents did not reach full stimulation intensity in one session. This suggests that while adolescents with anxiety disorders can tolerate stimulation at 1 mA, tDCS protocols may need to be modified to allow for a longer ramp up period and flexibility in intensity of stimulation when working with this population. Overall, both parents and adolescents rated treatment satisfaction in the excellent to fair range, supporting the acceptability of the treatment.

With regard to anxiety reduction effects, anxiety-related impairment ratings decreased from pretreatment to posttreatment, with medium to large effect sizes. Anxiety symptom severity reduction effect sizes on rating scales ranged from large on clinician ratings to small on parent and adolescent ratings, and scores were in the moderate range for several adolescents at posttreatment. These effects, while in the expected direction, were largely not statistically significant. The direction of these effects however suggests that four sessions of tDCS augmented ABMT (ABMT + tDCS) at 1 mA may be sufficient for some but not all adolescents with anxiety disorders. Lengthier and/or higher intensity monotherapy protocols have been used in past research, including up to eight ABMT sessions and 2 mA tDCS intensity. In future studies, it will be important to investigate whether additional treatment sessions and/or higher intensity tDCS may lead to greater anxiety reduction effects, and characteristics of adolescents with anxiety disorders who are most likely to benefit from tDCS augmentation of ABMT.

With regard to the target of treatment, attention to threat, tDCS augmentation of ABMT led to reductions in neural resources allocated to threatening stimuli and increases in neural resources allocated to neutral stimuli in components associated with rapidly deployed attention orientation. This is indicated by pretreatment to posttreatment reduction in the P1 and P2 components on NT trials and pretreatment to posttreatment increase in the P1 and P2 components on NN trials, as well as pretreatment to posttreatment changes in the relation between NT and NN. At pretreatment, participants allocated relatively more neural resources at rapidly deployed attention stages when threat stimuli were present, whereas at posttreatment participants allocated relatively more neural resources when only neutral stimuli were present. tDCS augmentation of ABMT also led to reductions in neural resources allocated to neutral stimuli and increases to threatening stimuli in the component associated with later strategic attention regulation and effortful disengagement from threat. This is indicated by pretreatment to posttreatment reduction in the P3 component on NN trials and pretreatment to posttreatment increase in the P3 component on NT trials, as well as pretreatment to posttreatment changes in the relation between NT and NN. At pretreatment, participants allocated relatively more neural resources at a later stage when only neutral stimuli were present, whereas at posttreatment participants allocated more neural resources when threat was present.

Conclusion

Overall, these preliminary data support the feasibility, acceptability, and clinical promise of tDCS augmentation of ABMT as a treatment for adolescents with anxiety

disorders. It further supports the promise to engage the treatment target by increasing attention orientation in the presence of neutral stimuli and increasing attention regulation when a threat was present. We urge caution in interpreting these results, as most effects were not statistically significant in this small feasibility case series. Future research is encouraged to investigate the optimal dosing parameters to produce adequate treatment response and examine the efficacy of tDCS augmentation of ABMT in larger samples of adolescents with anxiety disorders using a randomized controlled design.

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Vaclavik D., Bechor M., Foster A. et al. Case Series of Transcranial Direct Current Stimulation as an Augmentation Strategy for Attention Bias Modification Treatment in Adolescents with Anxiety Disorders
Clinical Psychology and Special Education
2020, vol. 9, no. 3, pp. 105–126.

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Клиническая и специальная психология
2020. Том 9. № 3. С. 105–126.

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2020. Том 9. № 3. С. 105–126.

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В данной статье представлены клинические случаи прохождения курсов транскраниальной микрополяризации (tDCS) с целью оценки обоснованности, приемлемости и клинических перспектив данного метода в качестве поддерживающей стратегии у подростков, направленных в клинику. Терапия модификации искаженного внимания (Attention Bias Modification Treatment, ABMT) – это компьютерный протокол тренировки внимания, разработанный для снижения непроизвольного переключения внимания на источники кажущейся угрозы и, соответственно, для уменьшения тяжести симптомов тревоги. Исследования эффективности ABMT демонстрируют его умеренную общую результативность. Достижения в исследованиях нейронных механизмов внимания к угрозам и протоколов тренировки внимания указывают на высокий потенциал транскраниальной микрополяризации применительно к дорсолатеральной префронтальной коре в качестве новой поддерживающей стратегии повышения эффективности ABMT (ABMT + tDCS). Однако метод tDCS никогда не проверялся на выборке подростков с тревожными расстройствами. Шесть подростков с первичным тревожным расстройством прошли все четыре сеанса ABMT + tDCS. Побочные эффекты были легкими и временными. Подростки и родители независимо друг от друга сообщали о среднем или высоком уровне удовлетворенности результатами терапии. У подростков, прошедших курс ABMT + tDCS, значимо снизились первичные симптомы тревожного расстройства. Кроме того, электрофизиологические данные (в частности, результаты ЭЭГ) свидетельствовали об уменьшении нейронной активности, вызванной ситуацией воспринимаемой угрозы. Полученные результаты подтверждают обоснованность и перспективность применения tDCS в качестве поддерживающей стратегии у подростков с тревожными расстройствами, что дает импульс для дальнейших исследований контрольных групп с более крупными выборками.

Ключевые слова: тревожность, подростки, модификация искажения внимания, танскраниальная микрополяризация, нейромодуляция, электроэнцефалография, событийно-связанные вызванные потенциалы.

Финансирование. Настоящее исследование поддержано Национальным институтом здоровья США (R25 GM061347, UH2 MH101470 и R01 MH119299). Содержание

исследования является исключительной ответственностью авторов и может не являться отражением официальной позиции Национального института здоровья.

Благодарности. Авторы благодарят доктора Грега Хайака за его вклад в настоящее исследование.

Для цитаты: Вацлавик Д., Бихор М., Фостер А., Гральник Л.М., Бар-Хаим Я., Пайн Д.С., Биксон М., Сильверман В.К., Риб-Сазерленд Б.С., Петтит Дж.В. Клинические случаи курсового применения транскраниальной микрополяризации как поддерживающей стратегии при терапии модификации искаженного внимания у подростков с тревожным расстройством [Электронный ресурс] // Клиническая и специальная психология. 2020. Том 9. № 3. С. 105–126. DOI: 10.17759/cpse.2020090308

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Vaclavik D., Bechor M., Foster A. et al. Case Series of Transcranial Direct Current Stimulation as an Augmentation Strategy for Attention Bias Modification Treatment in Adolescents with Anxiety Disorders
Clinical Psychology and Special Education
2020, vol. 9, no. 3, pp. 105–126.

Вацлавик Д., Бихор М., Фостер А. и др. Клинические случаи курсового применения транскраниальной микрополяризации как поддерживающей стратегии при терапии модификации искаженного внимания у подростков с тревожным расстройством
Клиническая и специальная психология
2020. Том 9. № 3. С. 105–126.

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Received: 18.04.2020

Accepted: 10.09.2020

Получена: 18.04.2020

Принята в печать: 10.09.2020

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