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REHABILITATION TECHNOLOGY (R HARVEY, SECTION EDITOR)

Transcranial Direct Current Stimulation for Motor Recovery Following Brain Injury

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Abstract

Purpose of Review To discuss the potential use of transcranial direct current stimulation (tDCS) to improve motor behavior after brain injury.

Recent Findings Despite evidence that tDCS can improve motor function following brain injury, meta-analysis studies have largely failed to find conclusive support for tDCS as a viable treatment. In part, these inconsistencies arise from widespread variability in individuals' responsiveness to tDCS because of biological and experimental factors.

Summary Properly designed smart clinical studies are still needed to determine the optimal stimulation parameters and combinations of tDCS. However, some patterns of "best practice" have begun to emerge: (1) pairing tDCS concurrently with high-intensity motor training as opposed to before, after, or in the absence of physical practice, (2) repeating sessions of stimulation in close succession over a single administration, (3) administering stimulation during more acute periods of recovery over chronic states, and (4) utilizing modeling techniques based on individual anatomy to tailor electrode placement and optimize current flow.

Keywords Brain injury · tDCS · Neurorehabilitation · Motor recovery

Introduction

There has been a recent rise in popularity of using transcranial direct current stimulation (tDCS) to modulate cortical excitability in hopes of improving behavior. Here, we will discuss the potential use of tDCS for neurorehabilitation to improve motor behavior after brain injuries. We begin with a review of how tDCS induces neuroplastic changes. Next, we summarize how neurophysiological changes associated with motor recovery following brain injury and motor learning can inform treatment. Finally, we coalesce the successes and failures that have been documented using tDCS to treat brain-injured patients into recommendations for practical application.

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Mechanisms of tDCS

Evidence suggests tDCS is a form of non-invasive brain stimulation (NIBS) that can elicit functional and morphological changes of the underlying cells it targets. It is thought to slightly depolarize/hyperpolarize cell membranes depending on the polarity of the stimulation and can affect cellular excitability from minutes to hours [1–4, 5•, 6]. In vitro and in vivo animal studies using direct current stimulation (DCS)–induced changes in synaptic plasticity such as modulations in spontaneous neuronal activity [7•, 8–10], neuronal evoked potentials [11••, 12, 13, 14•], and neuronal paired-pulse plasticity [12]. DCS also increased expression of BDNF, Ca^{2+}, and other genes that play a role in long-term potentiation (LTP)/long-term depression (LTD) [15–17]. DCS-induced effects rely on calcium-dependent mechanisms [14–16], NMDA receptors [18], and BDNF and TrkB receptor pathways [11, 19], and are modulated by other types of LTP-inducing protocols [20]. Other cell types besides neurons are affected by DCS; DCS increased Ca^{2+} surges in astrocytes, cells with a crucial role in NMDA-dependent plasticity [14•].

In addition to functional changes, DCS elicits morphological changes. DCS can influence orientation of neuronal processes and their growth direction [21], increase spine density

[22•], and increase oligodendrocyte-specific progenitor cells [23]. Individual cells can be affected by orientation of the electrical field, distance from the current, and their own morphology. Hence, the orientation of the electrical field influences different cell types and different compartments within the same cell [24••].

In humans, there is similar evidence that tDCS modulates cortical excitability. tDCS can modulate motor evoked potential (MEP) amplitudes and cerebello-brain inhibition (CBI) as assessed by transcranial magnetic stimulation (TMS), [2, 5•, 25–29] spectra changes at different frequency bands in electroencephalogram (EEG) [30], blood oxygenation level–dependent (BOLD) changes assessed by functional magnetic resonance imaging (fMRI) [31–33], and GABA and glutamatergic concentrations assessed by magnetic resonance spectroscopy (MRS) [34, 35]. Pharmacologically, NMDA receptors [36] and Ca^{2+} are underlying mechanisms for the lasting effects of tDCS [1, 2, 5•].

Overall, the literature suggests tDCS uses direct electrical currents to modulate both the functional strength and morphology of neuronal and non-neuronal synapses. Substantial evidence links these effects to LTP-like and LTD-like mechanisms. Generally, anodal tDCS is believed to enhance cortical excitability of the targeted brain area whereas cathodal tDCS diminishes it. However, it is important to note that this is an oversimplification of what happens with an electrical current in a complex structure as the human brain.

Neurophysiological Changes in Motor Recovery and Motor Learning

The motivation behind using tDCS in neurorehabilitation largely rises from two bodies of literature: first, research on the neurophysiological changes associated with motor recovery after brain injury and second, neurophysiological changes associated with learning new motor tasks. A basic understanding of any overlapping mechanisms that drive recovery and learning can guide tDCS strategies for neurorehabilitation.

Neurophysiological studies of upper limb motor recovery in chronic (> 6 months post injury) stroke patients using TMS have found higher motor thresholds and reduced MEP amplitudes in ipsilesional motor cortex (iM1) as compared to contralesional M1 (cM1) and healthy controls, and are often correlated with measures of hand dexterity and function [37, 38]. In healthy individuals, the two motor cortices exert mutual inhibition at rest and prior to movement (i.e., interhemispheric inhibition—IHI). However, chronic stroke patients have stronger IHI imbalances (from cM1 to iM1) both at rest and prior to movement onset that are correlated with more severe motor impairments [39•, 40, 41]. Interhemispheric imbalances are also present in other motor areas such as the dorsal premotor (dPM) cortex where more unbalanced contralesional PMd (cPMd) influence on iM1 is present in patients with greater clinical impairment [42–44]. Similarly, stroke patients with right hemisphere neglect have shown pathologically exaggerated intrahemispheric influence from left contralesional posterior parietal cortex (cPPC) to left cM1 that was related to severity of neglect [45].

In studies following stroke recovery in acute stroke patients (< 3 months after injury), iM1 thresholds and corticomotor excitability of the upper limb were initially reduced but over time increased with improved upper limb impairment and function [46•, 47–49]. Interestingly, in acute stroke patients, premovement IHI was initially normal following injury and later became abnormal. This emergence of the abnormal pattern from acute to chronic recovery was inversely correlated with motor recovery suggesting that interhemispheric imbalances might be a consequence of other underlying recovery processes rather than the cause of poor motor recovery [50].

Neurophysiological changes following motor recovery in traumatic brain injury show a similar profile to stroke. Moderate to severe chronic TBI patients show higher motor thresholds [51, 52], higher MEP variability [52], and reduced MEP amplitudes in the paretic upper limb as compared to the non-paretic side, changes that were found to be related to the severity of diffuse axonal injury (DAI) and motor impairment [51, 53•]. In acute cases of mild to moderate TBI, patients had initially higher motor thresholds, higher MEP variability, and reduced MEP amplitude that then showed a trend to return to normal levels in the chronic state [52, 54].

Overall, both stroke and TBI patients show neurophysiological evidence of cortical reorganization after injury and normalization of these corticomotor changes with recovery. This suggests motor and functional recovery in brain injury patients might be linked to the restoration of normal inter- and intracorticomotor patterns. Based on this theory of restoring normal corticomotor patterns after brain injury, several strategies for neurorehabilitation have emerged to either upregulate excitability of ipsilesional motor areas (e.g., M1, dPM, PPC), downregulate excitability of contralesional motor areas, or do both simultaneously to ameliorate the paretic side motor dysfunction. However, it is important not to assume that all behaviors follow the same recovery pattern as the upper limb. For example, in contrast to upper limb motor recovery post stroke, in swallowing motor recovery, corticomotor excitability of laryngeal cM1 in acute stroke patients was initially reduced, but over time increased along with improved swallowing impairment and function [55]. Moreover, an important caveat to this body of work is that it is limited to stroke and TBI patients for whom it is possible to obtain MEPs (i.e., mild to moderate motor deficits). Thus, it remains unclear what neurophysiological recovery may look like for patients with more severe motor deficits lacking MEPs.

Similar neurophysiological changes happen with motor learning as seen with motor recovery following brain injury. Motor learning in neurorehabilitation is to either relearn to

perform a task the same way as it was done before injury (recovery) or to implement a different strategy from that used before injury (compensation). Although recovery and compensation likely involve different pathophysiological mechanisms, both are associated with an increase in corticomotor excitability. Changes in the plasticity of synaptic connections of M1 are widely believed to play an essential role in learning and memory [56•]. In both animals and humans, there is evidence of increased M1 excitability following motor learning [57–64], as well as functional and structural changes to a distributed brain network that connects M1 with the dPM, PPC, cerebellum, somatosensory cortex, supplementary motor area (SMA), and other motor areas [65–67].

Based on studies demonstrating increased excitability of motor areas following motor learning, a body of research has emerged attempting to use tDCS to modulate M1 excitability and/or its connection with other motor areas to augment motor learning and retention. Notably, several studies have demonstrated application of tDCS during motor learning can boost performance in healthy humans: sequential visual isometric pinch task [11••, 27, 68•], finger sequencing [2, 69–71], reaching adaptation [29, 72, 73], walking adaptation [74], balance [75], and even functional motor tasks such as the Jebsen-Taylor Hand Function Test (JTT) [76•].

Utilizing tDCS to Augment Motor Recovery in Neurorehabilitation

Converging evidence that motor recovery and learning engage overlapping neurophysiological mechanisms and tDCS can improve motor learning in healthy individuals has made tDCS an appealing strategy for augmenting motor recovery in brain-injured patients. Here, we identify some successes and failures associated with using tDCS over various motor areas to treat motor syndromes such as hemiparesis, hemineglect, and dysphagia.

Hemiparesis

The most studied application of tDCS in brain injury patients is hemiparesis of both the upper and lower limbs. The most commonly studied electrode montages are anodal tDCS applied over ipsilesional M1 (anodal-iM1), cathodal tDCS applied over contralesional M1 (cathodal-cM1), and bihemispheric-M1 (i.e., anode and cathode are placed simultaneously over iM1 and cM1, respectively).

In hemiparesis of the upper limb, tDCS has been used to improve arm and hand function. In some studies of chronic stroke patients, even a single session of anodal-iM1, cathodal-cM1, or bihemispheric-M1 stimulation during motor training improved upper motor limb function measures such as JTT, pinch force, and reaction time [77–81] and was associated with enhanced cortical excitability and reduced intracortical inhibition of iM1 [77, 78]. Studies with multiple sessions (ranging from 5 to 20) of tDCS over M1, applied prior to or concurrently with motor training, have elicited immediate and long-lasting behavioral improvements of JTT, Upper Extremity Fugl-Meyer (UE-FM) Scale, Action Research Arm Test (ARAT), Wolf Motor Function Test (WMFT), Modified Ashworth Scale (MAS), Stroke Impact Scale (SIS), Medical Research Council (MRC) Scale, Barthel Index (BI), National Institutes of Health Stroke Scale (NIHSS), and muscle strength with effects ranging from weeks up to a year in acute and chronic stroke patients [82–97]. Some of these behavioral changes were also accompanied and/or correlated with increased iM1 cortical excitability [96, 98••], reduced IHI imbalance [98••], increased fMRI activation in ipsilesional and decreased in contralesional motor areas [85, 91], and increased fractional anisotropy of descending motor tracts of iM1 [94]. Though largely less examined, studies targeting other motor areas like ipsilesional PM (iPM) have also shown that repeated anodal stimulation over iPM with concurrent motor training can improve UE-FM Scale, MAS, MRC Scale, and BI, [99–101] behavioral changes that were accompanied with increased iM1 cortical excitability [99]. Surprisingly, only one study has explored the effects of tDCS to treat upper limb hemiparesis in TBI. Pairing multiple sessions of bihemispheric-M1 with concurrent motor training improved UE-FM Scale, SIS, Box and Block Test, and Purdue Pegboard Test in chronic TBI patients, changes that were largely sustained 6 months post intervention [102].

In contrast to these promising findings, other studies have presented contradictory results. In a single session of bihemispheric-M1 applied concurrently with motor training, although chronic stroke patients showed reduced IHI imbalance, there was no improvement in UE-FM Scale [103]. In other studies where anodal-iM1, cathodal-cM1, or bihemispheric tDCS was administered for multiple sessions either in the absence of [104], prior to [105], or concurrently with motor training [106–109], tDCS failed to elicit improvements in UE-FM Scale, MAS, Motricity Index (MI), NIHSS, and Box and Block Test beyond sham stimulation, and in some patients even worsened performance [109].

Meta-analysis studies have largely failed to validate the effectiveness of tDCS for improving upper limb function following a stroke. [110] This may be due to the vast variability in patient demographics and experimental protocols, as well as a lack of large robust randomized controlled trials. However, some patterns supporting improved effectiveness have emerged. Namely, multiple stimulation sessions in close succession with concurrent motor training are suggestive of better outcomes [111]. Additionally, efficacy is influenced by dose-related parameters relating to electrode size, charge density, and current density [112].

In hemiparesis of the lower limb, tDCS studies have focused on improvement of gait and balance. In acute and chronic stroke patients, a single session of anodal-M1 or bihemispheric-M1 tDCS, with concurrent motor training, has acutely enhanced paretic ankle control, Five Times Sit-to-Stand (FTSTS) Test, Timed Up and Go (TUG) Test, lower limb force production, postural stability, and walking speed [113–117] and simultaneously increased iM1 and decreased cM1 cortical excitability [118]. Another single-session study, targeting ipsilesional SMA during motor training, showed improved 10-Meter Walk Test (10MWT) and TUG Test [119]. Similarly, studies with multiple sessions (ranging from 10 to 14) of anodal-iM1 tDCS applied concurrently with motor training have elicited immediate and long-lasting behavioral improvements of Lower Extremity Fugl-Meyer (LE-FM) Scale, Lower Extremity Motricity Index (LE-MI), Functional Ambulatory Category (FAC), 6-min Walking Test (6MWT), 10MWT, TUG Test, and SIS [120•, 121, 122], and increased iM1 cortical excitability [120•].

In contrast, some single-session studies of either anodal-iM1 or bihemispheric-M1 applied before [123], after [124], or concurrently with motor training [125, 126] have failed to show behavioral improvements in spatial-temporal gait parameters and MAS nor elicit cortical excitability changes in iM1 or cM1. Additionally, one study with repeated sessions of anodal-iM1 with concurrent motor training failed to improve 6MWT or 10MWT [127].

However, in spite of some conflicting evidence, a systemic review with meta-analysis supports tDCS in improving gait speed after stroke, with multiple sessions being more effective than single sessions to improve functional outcomes [128].

Hemineglect

In hemineglect, tDCS montages are based off a similar concept as hemiparesis studies with either increasing activity of a hypoactive cortical region affected by the stroke (i.e., the right hemisphere in the case of neglect) or reducing cortical hyperactivity of the corresponding cortical region in the contralateral left hemisphere. Hence, tDCS studies on neglect have exclusively studied montages of either anodal applied over ipsilesional right posterior parietal cortex (anodal-iPPC), cathodal applied over contralesional left PPC (cathodal-cPPC), or simultaneously placed anode and cathode over iPPC and cPPC, respectively (bihemispheric-PPC).

In acute and chronic stroke patients, a single session of tDCS of anodal-iPPC, cathodal-cPPC, or bihemispheric-PPC, even without therapy, has shown significant improvement of Line Bisection Test (LBT), Star Cancellation Test (SCT), and Neglect Subtest of Test Battery for Attentional Performance (TAP) [129–131]. In other studies where multiple sessions (ranging from 10 to 15) of tDCS were applied either prior to or concurrently with motor training, both anodal-iPPC and cathodal-cPPC improved performance on the Motor-Free Visual Perception Test (MVPT), LBT, SCT, Modified BI, and Behavioral Inattention Test (BIT) [132, 133•]. In contrast, one study applying bihemispheric-PPC for multiple sessions (unpaired with therapy) failed to show improvements in BIT [134].

Though no meta-analysis study has focused solely on the efficacy of tDCS in the treatment of hemineglect, other meta-analyses including tDCS and other NIBS protocols have found moderate-quality evidence supporting the effectiveness of NIBS techniques for the treatment of neglect especially when combined with therapy [135, 136].

Dysphagia

Unlike upper and lower limb muscles, cortical representations of swallowing muscles have more bilateral cortical innervations [137]. Though cortical representations of swallowing muscles are asymmetrical (showing a swallowing dominant side unrelated to handedness) [137, 138], research studies have exclusively explored stimulating either iM1 or cM1. Currently, only four studies have specifically investigated the effects of tDCS in post stroke dysphagia.

In each of these studies of either acute, subacute, or chronic stroke patients, repeated sessions (ranging from 5 to 10) of anodal tDCS over iM1 or cM1, paired with concurrent motor training, were found to improve Functional Dysphagia Score (FDS) and Dysphagia and Outcome Severity Scale (DOSS) either immediately following treatment [139, 140•, 141] or at a 1–3-month follow-up [140, 142].

Although no meta-analysis studies have focused solely on the efficacy anodal tDCS in the treatment of dysphagia, other meta-analyses including tDCS and other NIBS protocols have found a small but significant effect of NIBS on post stroke dysphagia with slightly better effect size for stimulation of cM1 over iM1 [143, 144].

Limited and Conflicting Evidence for tDCS in Neurorehabilitation

Some consistent patterns have emerged implicating a common thread of "best practice" across syndromes: Pairing tDCS concurrently with motor training, repeated sessions of stimulation in close succession, and early administration during more acute periods of recovery show the best efficacy. A noteworthy limitation in the tDCS literature on the treatment of brain injury is the bias toward the study of stroke recovery. Few studies have examined the use of tDCS as clinical intervention for motor recovery in TBI patients.

Additionally, despite promising evidence that tDCS can serve as an adjunct to maximize motor recovery in brain injury patients, there have been some studies showing inconsistent

and even contradictory effects such that meta-analysis studies have failed to find conclusive support for the use of tDCS as a viable treatment [110]. Factors relating to both biological and experimental variability are major culprits that underlie some of these inconsistencies across the literature [145••].

Biological Factors

Even in healthy participants, there is a fair amount of heterogeneity in responsiveness to tDCS due to a variety of biological factors. These biological factors can be subdivided into factors that affect inter- (across) and intra- (within) subject variability. Inter-subject variability is affected by factors that are due to an individual's constant traits, such as age [146], gender [147], anatomical variability [148, 149], and genetics [11••, 150, 151]. Intra-subject variability is affected by factors that are due to an individual's current state, such as hormonal cycles (menstrual cycle and circadian rhythms) [152, 153], prior history of brain activation [154], sleep deprivation [155], attentional focus [156, 157], alcohol/drug use [158], and fluctuations in resting brain activity [159].

In brain injury patients, these same biological factors are further complicated by additional heterogeneity introduced by the brain lesion itself. For example, brain injury patients may have structural brain changes related to the underlying pathology (e.g., brain atrophy) that can alter the current distribution [160]. In a tDCS modeling study based on individual MRI anatomy, patients with skull damage (after TBI) were found to have significant changes in their current distribution which altered the efficacy of tDCS application and in some cases even lead to unfavorable neurophysiologic changes [161, 162]. Brain injury patients are also likely to be on concurrent medications affecting the nervous system (e.g., antidepressants, antipsychotics, anxiolytics, analgesics, anticonvulsants) and have other comorbidities that may alter their neurochemistry. Overall, these trait and state biological factors introduce significant challenges in standardizing current flow and intensity across individuals.

Experimental Factors

In addition to individual variability introduced by biological factors, there are several other experimental factors relating to the administration of tDCS that influence its efficacy: electrode montage and type, dosage, timing, and endpoint measures.

Electrode montage, skin preparation, and type of electrodes are all critical for the spatial distribution and direction of the electric current. The diversity of tDCS montages (unilateral or bilateral, and over various motor areas), type (bipolar or high-definition tDCS [163]), and polarity (anodal or cathodal) can induce varied effects on the brain. Even when targeting a particular motor region, neighboring and connected areas can be affected as well. Not surprisingly, research has shown that different montages have distinct effects on a motor behavior—i.e., targeting M1 preferentially influenced speed and retention of motor learning [68•], whereas targeting the cerebellum promoted accuracy and acquisition [164].

A second experimental factor is the dosage of stimulation, including both intensity, duration, and number of sessions and their relative spacing—all of which influence the longevity, magnitude, and even direction of the effects of tDCS [5]. To a certain extent, longer bouts of stimulation elicit longer-lasting aftereffects [6], increasing stimulation intensity can increase the magnitude and improve the consistency of aftereffects [2, 28], and consecutively repeated sessions of stimulation can have cumulative effects [83, 165–167]. However, linearly increasing each of these parameters does not necessarily increase their effects in a linear way. In some cases, increasing intensity, duration, and number of sessions and session spacing can engage homeostatic mechanisms that shift or even reverse the direction of their effects [28, 167, 168], mechanisms that may even further interact with biological factors as well.

A third critical factor is the timing of stimulation and time of stimulation relative to the time of injury. Studies have varied application of tDCS, and here timing will pertain to two independent dimensions: timing of stimulation relative to motor training and timing of stimulation relative to acuity of injury. With motor training, studies have varied application of tDCS to either occur prior to motor training (as a priming effect) [34, 35, 89, 93, 105, 169, 170], immediately following motor training (to target consolidation) [171], concurrently [2, 31, 68, 164, 172–174], or in the absence of any motor training [104, 175]. However, evidence in humans indicates that timely co-application of tDCS with motor training is associated with the largest and most consistent behavioral gains [104, 176]. Similarly, in animal studies, applying DCS over M1 with concurrent activity was crucial to induce LTP [11••], and a lack of concurrent activity resulted in no aftereffects. With regard to the time of injury, most studies have focused on studying brain injury patients in a chronic state of recovery, though there is evidence to suggest that tDCS applied during more acute stages of injury may be more effective for recovery [92, 120, 177–179]. Special caution is needed to avoid use of tDCS too early in the recovery process as there is the possibility it could aggravate the injury. In an animal model of stroke, tDCS applied 1 week following the injury was associated with better recovery than when applied only 1 day after injury [180].

A final critical experimental factor is the variability of endpoint measures across the literature which limits direct comparisons across studies. To efficiently move the field forward, a consensus on specific and appropriate endpoint measures that span the model of disability (including metrics assessing recovery at the level of body function, activity, and

participation) is needed. Having a comprehensive combination of appropriate endpoint measures will help identify which experimental factors are most relevant for neurorehabilitation and what biological factors may preclude certain individuals from benefiting from tDCS as a treatment.

Modeling techniques that use a patient's MRI to simulate current flow offer one effective approach to improve and individualize the use of tDCS in neurorehabilitation. For one, modeling techniques can be used to optimize electrode placement and intensity to improve uniformity of current flow across individuals [181, 182]. Second, current distribution models can be applied to past data sets and can identify study confounds and distribution patterns that are associated with better behavioral outcomes.

Conclusions

Substantial evidence supports that tDCS can induce both functional and structural changes of the cells it targets depending on their orientation and type, and these effects are linked to LTP-like and LTD-like mechanisms. Similar to motor learning, motor recovery is associated with increased corticomotor excitability and other types of cortical reorganization, mechanisms that can enable functional restoration following brain injury. In some cases, neurophysiological and behavioral restoration following motor training has been boosted with concurrent application of tDCS. Despite promising evidence that tDCS can improve motor syndromes following brain injury (e.g., hemiparesis, hemineglect, and dysphagia), meta-analysis studies have largely failed to find conclusive support for tDCS as a viable treatment. Variability in biological and experimental factors between studies explains, in part, these inconsistencies in the literature. Nonetheless, some patterns of "best practice" have begun to emerge: (1) pairing tDCS concurrently with high-intensity motor training, (2) repeating sessions of stimulation in close succession over a single administration, (3) administration during more acute periods of recovery, and (4) tailoring electrode placement with modeling techniques to optimize electrical distribution patterns. Properly designed smart clinical studies are needed to determine the optimal stimulation parameters and combinations (i.e., with medications, behavior, etc.) of tDCS to verify its true efficacy in clinical settings.

Compliance with Ethical Standards

Conflict of Interest The authors declare that they have no conflicts of interest.

Human and Animal Rights and Informed Consent This article does not contain any studies with human or animal subjects performed by any of the authors.

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