

Comparison and Affecting Factors of Three tDCS Montages in Motor Recovery of Chronic Stroke Patients: A Resting-State EEG Study

Chunfang Wang (✉ chfwang@tju.edu.cn)

Tianjin People's Hospital Tianjin Union Medical Center <https://orcid.org/0000-0001-6201-3980>

Ying Zhang

Tianjin Union Medical Centre

Yuanyuan Chen

Tianjin University

Peiqing Song

Tianjin University of Sport

Hongli Yu

Hebei University of Technology

Changcheng Sun

Tianjin Union Medical Centre

Jingang Du

Tianjin Union Medical Centre

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Abstract

Objective: We aimed at exploring the modulation of tDCS on spontaneous cortical activity through the changing of EEG rhythms to different tDCS montages and the interaction between cortical responses and variability factors of stroke individuals.

Methods: 19 stroke subjects underwent 4 tDCS sessions with 3 different tDCS montages (anodal (atDCS), cathodal (ctDCS) and bilateral (bi-tDCS)) and sham stimulation in a single-blind, randomized, controlled crossover design. We acquired resting-state (eyes closing and opening alternately) EEG data before and after tDCS, and calculated the spectral power of each frequency band. Paired-samples T test was applied to examine the difference of spectral power between pre- and post-stimulation of each montage. Three-way repeated measures analysis of variance with lesion hemispheres, stimulation montages and locations were carried out to investigate tDCS effects of different lesion, montages, and channel locations, and the interaction. Further, the effects of tDCS over time were analyzed applying three-way repeated ANOVAs as well with post trials, lesion hemispheres and channel locations separately to each montage. Finally, linear and quadratic regression model were used separately to describe the association between clinical factors of stroke patients and change of spectral power.

Results: We found that induced effect of tDCS was limited to the alpha rhythm of opening-state. atDCS increased the alpha power especially alpha1 (8-10 Hz) in local and distant areas of mainly frontal and partial. bi-tDCS affected the alpha power as well, but in a smaller area which mainly focused on alpha2 (10-13 Hz). ctDCS and sham had no effect on alpha rhythm. No significant difference of alpha band was found over the observed time range after the stimulation over. Results further showed that the quadratic model can better characterize the relationship between clinical factors and the tDCS effects of alpha rhythm than linear model. The changing of alpha especially alpha2 in contralateral hemisphere induced by atDCS was related to time since stroke, and alpha2 in ipsilateral hemisphere induced by bi-tDCS to motor impairment level.

Conclusion: Our results provide electrophysiological evidence that different tDCS montages in stroke subjects modulate rhythmic cortical activity of alpha band in different ways, and the effects maintained for at least 30 minutes. The tDCS modulation effect was related to clinical factors, especially the time since stroke and the level of motor impairment. These findings are of great significance for the knowledge on modulation effect to stroke patients and for therapeutic application of motor recovery following stroke.

1 Introduction

Transcranial direct current stimulation (tDCS) - a noninvasive brain stimulation (NIBS) technique modulating the local field potential in neural tissue and cortical excitability has been widely used in recovery post stroke including motor rehabilitation and evidenced its behavioral and neurophysiological effects by numbers of previous studies [1–4]. However, despite its increasingly application in experimental and clinical settings, the results remain variable, some studies failed to show a positive response to the stimulation in some kinds of stroke patients [5–8]. Many researchers have thus tried to clarify the sources of variability affecting tDCS's efficiency. Factors like current amplitude of stimulation, placement of electrodes, polar of stimulation electrodes and so on are proved to be possible suspects [8, 9]. However, the precise mechanism of how the above factors affecting the stimulating results remains largely unclear.

Among the above mentioned sources of variability, one of the key considerations in using tDCS to improve motor performance after stroke is how the stimulation modulates cerebral cortex [9, 10]. Polarity of the electrodes is proved to be especially critical in individuals with stroke due to the spread of functional reorganization in the post-stroke brain [10]. Given the hypothesis that rebalancing interhemispheric interactions and/or restoring excitability in the ipsilesional hemisphere is thought to be beneficial for post-stroke motor recovery [11, 12], present studies show three montages of tDCS position modes to regulating the excitability of cerebral cortex in poststroke patients: upregulating excitability of the ipsilateral hemisphere through posing the anode tDCS (atDCS) on it; down regulating excitability of the contralateral hemisphere through posing the cathode tDCS (ctDCS) on it; upregulating the ipsilateral cortex and downregulating the contralateral cortex at the same time [13–15]. Some studies have found that the excitability or suppression to the brain is not a “one size fits all” approach to recovery following stroke [1, 10, 15]. It may be related to stroke states like motor impairment level or stroke period (acute, sub-acute or chronic). But the interaction between regulation montages and the above factors is not clarified clearly.

Previous studies have proved that particular QEEG indices like the delta/alpha power ratio (DAR) and (delta + theta)/(alpha + beta) power ratio (DTABR) are sensitive to some cerebral pathophysiology following stroke and can inform clinical decision-making, including the efficacy of acute reperfusion therapies and outcome prognostication [16]. Some research has found electrophysiological changes in EEG oscillations of healthy people over rest and task states following tDCS over motor related cortex. Ardolino et al. [17] reported that cathodal stimulation of the motor cortex increased the power of delta and theta frequency bands. Mangia et al. [18] found an increase in alpha and beta power of spontaneous EEG during and after atDCS stimulation over postero-parietal cortex. In some motion imagination experiments, event-related desynchronization (ERD) of mu rhythm increased post anodal stimulation over the left primary motor area and decreased post cathodal stimulation [19]. Results from finger motion task showed an increase of ERD in low alpha and beta bands in sensorimotor related regions after atDCS but not ctDCS [20]. The above all focus on healthy people and we found few studies investigate EEG changes after tDCS in stroke patients.

In this study, we aimed at exploring the modulation effects of three different tDCS montages and the association between tDCS effects and variability of clinical factors in stroke individuals. First, we assessed the induced effects of different tDCS montages on spontaneous cortical activity through the changing of power of EEG rhythms. Then, we also investigated the duration effects thirty minutes since the end of tDCS stimulation. Finally, we tried to find out whether the clinical status (including time since stroke, location of stroke, level of motor impairment and so on) impact these effects.

2 Methods

2.1 Participants

19 patients (15 males, 4 females, mean age 56.5 ± 8.90 years, range 40–67 years, 9 right-hemisphere lesion, 10 left-hemisphere lesion) at least 3 months after subcortical cerebral infarction were included in this study. All of the patients were diagnosed with ischemic stroke according to MRI. Basic information of subjects was shown in Table 1. All of participants were informed of all aspects of the experiment including the possibility of minor adverse effects related to tDCS, such as transient sensations of itching, burning and prickling on the scalp and

signed an informed consent before the experiment started. The study was approved by the ethics committee of Tianjin Union Medical Centre.

Table 1
Participant characteristics in this study

Subject	Gender	Age(y)	Hand	Hemisphere	Cite	Time (/m)	FM	MBI	MAL:AOU	MAL:QOM
1	Male	63	Right	Right	BG	21	55	90	38	58
2	Female	60	Right	Right	BS	9	57	80	116	116
3	Male	39	Right	Left	BS	8	59	100	57	101
4	Male	63	Right	Left	BS	13	41	70	2	4
5	Male	58	Right	Right	BG	13	20	90	1	1
6	Male	64	Right	Left	BG	20	57	100	74	114
7	Male	56	Right	Right	BG	21	50	90	7	7
8	Female	67	Right	Left	BG	21	10	65	0	0
9	Male	47	Right	Left	BG	11	57	90	74	96
10	Female	63	Left	Left	BG	6	64	100	113	115
11	Male	61	Left	Right	BG	21	61	100	150	145
12	Male	61	Right	Right	BG	24	44	100	23	23
13	Male	56	Right	Left	BG	26	60	100	90	90
14	Male	59	Right	Right	BG	5	63	100	120	121
15	Male	44	Left	Left	BG	5	46	85	39	32
16	Male	46	Right	Left	BG	5	17	80	0	0
17	Male	40	Right	Right	BG	5	63	100	112	133
18	Female	68	Right	Left	BG	4	53	85	29	37
19	Male	58	Right	Right	BG	5	7	35	0	0

Age (year, y); Hand: Dominant hand; Hemisphere: Hemisphere affected by stroke; Cite = Cite of lesion; BG: Basal ganglia; BS: Brain stem; Time = Time following stroke (/month, m); FM: Fugl-Meyer scores; MBI: Modified Barthel Index; MAL: Motor activity log; AOU: Amount of use; QOM: Quality of movement

2.2 Experimental design

This was a single-blind, randomized, controlled crossover experiment, which consisted of four within-subject experimental sessions: three active conditions (anodal, atDCS; cathodal, ctDCS and bilateral, bi-tDCS) and a sham condition. Sham stimulation was used as control in the experiment to isolate the effects solely due to current stimulation and not due to the placebo and somatosensory effects that could arise from tDCS application. We generated a random table using block random method through Matlab program to determine the implementation order of atDCS, ctDCS, bi-tDCS and sham condition. Patients performed the four sessions as the order shown in

the randomized table and were blinded to the condition. The interval of each two of the four conditions was at least 1 week.

Each session contained four blocks: Baseline of Electroencephalogram (EEG), tDCS, Electrodes placing for EEG and EEG of post-stimulation. All of the four blocks were conducted in a quiet and electrically shielded room. Patients were asked to sit and relax on a comfortable chair during the experiment. In the baseline block, patients were required every 2 minutes to open or close their eyes according to a voice prompt produced by E-prime software. This process lasted for 12 minutes (6 trials with 3 eyes closing state and 3 eyes opening state). EEG signals were collected at the same time (Block 1). After that, we conducted one of the four tDCS montages (atDCS, ctDCS, bi-tDCS or sham) according to the random table for 20 minutes (Block 2). Then, electrodes of EEG acquisition were placed on the brain, which lasted for 10 minutes (Block 3). Finally, patients were asked to open and close their eyes alternately (per 2 minutes) again for 20 minutes (10 trials with 5 eyes closing state and 5 eyes opening state). EEG signals were collected meanwhile (Block 4). Figure 1 shows the experimental design of each session.

2.3 Transcranial direct current stimulation

Direct current was transferred by a saline-soaked pair of surface sponge electrodes (5 cm*7 cm) and delivered by a specially developed battery-driven constant current electrical stimulator (Neuroconn, made by German). The impedance of electrode was kept below 1 kΩ when the DC stimulator working. Target area of stimulation was the primary motor cortex (M1). The C3 or C4 according to the international standard 10–20 EEG system was defined to be the position of the M1. Placement of electrodes depended on the stimulation montage and lesion side of stroke patients (left or right hemisphere). For anodal stimulation (atDCS), the anode electrode was placed over the M1 of ipsilesional side and the cathode electrode was placed over the lateral supraorbital as reference electrode. For cathodal stimulation (ctDCS), the cathode was placed over the M1 of contralateral hemisphere and the anode was placed over the lateral supraorbital as reference electrode. For bilateral stimulation (bi-tDCS), the anode was placed over the M1 of ipsilesional side and the cathodal was placed over the M1 of contralateral hemisphere. The placement of sham stimulation was consistent with atDCS.

Patients were asked to seat in a chair quietly during the stimulation. For real stimulation, the current was ramped up over 8 s, held constant at 1.75 mA (current density: 0.5 A/m²) for 20 minutes and finally ramped down over 8 s. During the sham condition, the electrodes were located in the same positions as in the anodal stimulation, but the current was supplied only for the first 43 s (8 s ramp up, 30 s of DC stimulation and 8 s ramp down). This procedure ensured that the subjects felt the tingling sensation at the beginning of the stimulation [18].

2.4 EEG recording and processing

Recording

Resting-state EEG with eyes closed and opened was recorded in a quiet and electromagnetic shielding room. Participants were instructed to stay awake and avoid movement during the acquisition. EEG was recorded using Neuroscan EEG system made by US. 62 electrodes were placed on the scalp according to the International 10–20 position system. A pair of vertical electro-oculogram (VEOG) and a pair of horizontal electro-oculogram (HEOG) were also recorded to remove ocular artifacts. All of the electrode impedances were kept below 10 kΩ. The EEG

signal was amplified with a band pass of 0.1–70 Hz and sampled at 1000 Hz. The forehead was set as ground and linked earlobes were set as reference electrodes.

Spectral power analysis

Firstly, EEG data was desampled into 250 Hz and filtered by a 0.25–45 Hz bandpass filter. Then, Independent component analysis (ICA) was used to remove eyes artifacts. 100 seconds (10–110 seconds) of EEG signals of eye closing state or eye opening state of each trial (120 seconds) were selected for the following spectrum analysis separately. We conducted all the above process through EEGLAB tool box of Matlab software.

Each channel of each trial dataset (100 seconds) applied the following method to calculate the spectral power. A digital FFT-based power spectrum analysis (Welch technique, Hamming windowing function) was used to compute power spectrum density (PSD) average value of each EEG trial separately with NFFT = 1024, window = 256 and 50% overlap [21]. Then, spectral power of delta (1–4 Hz), theta (4–8 Hz), alpha (8–13 Hz), alpha1 (8–10 Hz) and alpha2 (10–13 Hz) beta1 (13–20 Hz) and beta2 (20–30 Hz) rhythms was calculated according to the frequency bands. We calculated average power of the three trials of Block 1 (eye-closing and eye-opening separately) as pre-stimulation parameters and average power of the five trials of Block 4 (eye-closing and eye-opening separately) as post-stimulation parameters. As for the same stimulation montage, patients with different lesion side (left or right) were conducted stimulation over different locations, we normalized the 62 channels to ipsilesional hemisphere, contralateral hemisphere and central zone.

2.5 Statistical analysis

To explore the effects of stimulation, paired-samples T test was applied to examine the difference of spectral power between pre- and post-stimulation. Each frequency band of 62 channels, two states and four tDCS montages were conducted separately. Since measures at different scalp locations, states and frequency bands are correlated to each other, we corrected the multiple comparisons using the Benjamini and Hochberg method of False Discovery Rates (BHFR) for each of the four conditions.

The effects of tDCS over time were analyzed applying three-way repeated measures analysis of variance (ANOVAs) with 5 post trials, lesion hemispheres and locations (5*2*62) separately to each montage. Before the ANOVAs, Mauchly's test of sphericity was used to test the covariance matrix sphericity. If the spherical assumption was not satisfied, Greenhouse-Geisser (G-G) method was used to adjust the degree of freedom (DF) to reduce the probability of Type I error.

Linear and quadratic regression model were used separately to describe the relation between clinical factors of stroke patients and spectral power changing. The demographics age, gender and lesion hemisphere were used as co-variables. Clinical factors includes time after stroke, level of motor impairment reflected by activity of daily living- Modified Barthel Index (MBI) and motor function evaluation scale - Fugl-Meyer scores (FM) and Motor activity log (MAL). The change of alpha power was described by the ratio of post-stimulation to pre-stimulation. The regression analysis was conducted separately for each montage.

3. Results

3.1 Post-stimulation effects

No significant difference was found between alpha power of post and pre stimulation on eye-closing state. Thus, these analyses focused solely on eye-opening state. The result of comparison between post- and pre-stimulation was shown in Fig. 2. Among the alpha power of eye-opening state, ctDCS and sham montages showed no significant difference between post- and pre-stimulation in any channels. For atDCS montage, compared with alpha power pre-stimulation it had a significant increase ($p < 0.05$) post stimulation in pre-frontal, frontal, central and parietal lobe of ipsilesional side and frontal and front-central region of contralateral side. Specifically, the increasing was mainly contributed by alpha1 band, all of the channels except temporal of ipsilesional side, pre-frontal of contralateral side and occipital of both sides showed significantly difference ($p < 0.05$) in the increasing of alpha1power. The increasing of alpha2 power was only focused on frontal and frontal-central region of both sides ($p < 0.05$). For bi-tDCS montage, only alpha 2 power of post stimulation showed increasing to pre stimulation, And the significant difference was focused on front-central, central and center-parietal region of ipsilateral side and some central and temporal region of contralateral side ($p < 0.05$).

3.2 tDCS effects of different montages and over time

For atDCS, repeated ANOVAs' results showed that there was a main effect in within-subject factor of location [$F(1.8,28.8) = 8.2, p = 0.002^*$] indicating a difference in different location. No other significant difference was found with Time [$F(2.0,32.2) = 0.84, p = 0.441$], Lesion [$F(1,16) = 0.30, p = 0.59$], Time*Lesion [$F(2.0,32.2) = 1.19, p = 0.32$], Location*Lesion [$F(1.8,28.8) = 0.57, p = 0.56$], Time*Location [$F(4.7,74.4) = 0.66, p = 0.64$], Time*Location*Lesion [$F(4.7, 74.4) = 0.64$]. None of the other three montages showed any significant main effect or interaction effect.

3.3 Association between tDCS and clinical facotrs

The regression analysis showed that the changing of alpha power (ratio of post-stimulation to pre-stimulation) induced by atDCS or bi-tDCS was related to the time since stroke and Fugl-Meyer scores separately. The quadratic regression model was better suited to model the variation trend than linear regression model both for atDCS and bi-tDCS. No correlation was found between the changing of alpha power and clinical factors after ctDCS and sham stimulation.

Figure 3 showed scatter plots and fitted curves of representative channels with clinical scale on the abscissa and alpha power's ratio of post- to pre-stimulation. Table 2 showed the channels at which the clinical factors could predict the power change of alpha band using quadratic regression model under the significant level of $p < 0.05$. For atDCS, the alpha changing ratio of central and partial area in contralateral side could be predicted by the time since stroke using quadratic regression model, and the alpha2 of front-central, central and center-parietal region of central and contralateral could be predicted by the time as well. Stroke subjects with 3 to 6 months and longer than 20 months since stroke showed higher alpha power increase than other subjects, indicating higher response to the montage of atDCS. For bi-tDCS, the alpha2 changing ratio of frontal and frontal-central area in ipsilateral could be predicted by Fugl-Meyer scores using quadratic regression model. The model showed that power of alpha2 increased the most in moderately impaired subjects with respect to mild and severe impairment.

Table 2

the statistical results of the channels at which the clinical factors could predict the power change of alpha band using quadratic regression model under the significant level of $p < 0.05$.

Stimulation Montage	Frequency Band	Channel	R ²	F	Sig
atDCS	alpha	C4	0.58	3.52	0.03
atDCS	alpha	P2	0.69	5.77	0.01
atDCS	alpha	P6	0.60	3.87	0.02
atDCS	alpha2	FZ	0.62	4.22	0.02
atDCS	alpha2	F2	0.60	3.90	0.02
atDCS	alpha2	FCZ	0.63	4.42	0.01
atDCS	alpha2	FC2	0.63	4.40	0.01
atDCS	alpha2	FC4	0.56	3.36	0.04
atDCS	alpha2	CZ	0.54	3.09	0.05
atDCS	alpha2	C2	0.63	4.44	0.01
atDCS	alpha2	C4	0.59	3.73	0.03
atDCS	alpha2	C6	0.57	3.40	0.04
atDCS	alpha2	CPZ	0.56	3.32	0.04
atDCS	alpha2	CP2	0.62	4.33	0.02
atDCS	alpha2	P2	0.64	4.68	0.01
bi-tDCS	alpha2	F3	0.60	3.84	0.02
bi-tDCS	alpha2	FT7	0.58	3.60	0.03
bi-tDCS	alpha2	FC3	0.60	3.92	0.02
bi-tDCS	alpha2	C5	0.56	3.25	0.04

4 Discussion

This study aimed at investigating the tDCS effects of different montages on cortical activity of chronic ischemic stroke patients. We focused on spectral power changing after three kinds of tDCS montages (atDCS, ctDCS and bi-tDCS) and the difference of after-effects among them. We are also interested in the after-effects over time and the relation between changing of alpha and clinical factors of stroke patients. There were four important findings from our study: (1) the tDCS effect was limited to the alpha rhythm of opening-state. (2) atDCS increased the alpha power especially alpha1 (8–10 Hz) in local and other areas. bi-tDCS affected the alpha power as well, but in a smaller area and mainly focused on alpha2 (10–13 Hz). ctDCS had no effect on alpha rhythm. (3) No significant difference of alpha band was found over the observed time range after stimulation. (4) The change of alpha especially alpha2 in contralateral hemisphere induced by atDCS was related to time since stroke, and alpha2 in ipsilateral hemisphere induced by bi-tDCS to motor impairment level.

Alpha band of EEG has been proved by several studies to be a brain rhythm involved in several cerebral functions, ranging from sensory-motor processing to memory formation [22, 23]. Ischemic stroke showed an attenuation of normative, faster activity, particularly in the alpha band (8–12 Hz) [24, 25]. Alpha band of stroke patients was found to be locally reduced in brain regions critical to observed motor or cognitive behavioral deficits. The decrease of alpha band synchrony was found related to cognitive and motor deficits in post-stroke patients [26]. Some study showed that motor recovery could be predicted by increased alpha-band functional connectivity in motor-related areas [27]. So we supposed that the increasing of alpha band may be beneficial to stroke recovery.

Previous studies investigating the changing of cortical activity after tDCS through rs-EEG power spectrum analysis were mainly focused on healthy people and showed response difference among stimulation montages. Some research showed increase of alpha band after atDCS in healthy people, but not after ctDCS, which is similar to our result in stroke subjects. Notturno et al. [20] found a higher low alpha band power post- than pre-atDCS over motor related regions, but not for ctDCS. Spitoni et al [28] explored the tDCS effect over the right posterior parietal area in healthy subjects and found that the effect was limited to the alpha band, and atDCS significantly affected the alpha band whereas the ctDCS did not elicit any modifications. This is consistent with our finding in stroke patients. However, the impact was shown in eye-closing but not in eye-opening state, which is different with this study. We hypothesized that this might be related to sleepiness with eye-closing state in some patients. Besides that, the difference of area of stimulation target may be another impact factor. For bilateral tDCS, studies are mainly focused on its rehabilitation efficacy on stroke patients [29–32]. A reduction of inter-hemispheric imbalance was found after a long-term effect of tDCS associated with physical therapy according to the analysis of motor evoked potential (MEP) [30]. We found no reports about EEG power spectrum following the bi-tDCS montage. In our study, both atDCS and bi-tDCS modulated alpha band, but atDCS preferred low-band alpha and bi-tDCS preferred high-band alpha. Studies have shown that different alpha components correspond to different cognitive processes. Low-band alpha rhythm was supposed to be related to anticipatory attentional processes and high alpha would indicate task-specific visuo-motor processes according to some task-related ERD/ERS study [33]. We speculated that these different changes on alpha rhythm induced by the two montages may imply that they work in different ways.

There are also some inconsistent reports with our results. Besides alpha band, some study showed power changing in other frequency band after tDCS [17, 34, 35]. In some study rs-EEG power spectrum analysis showed no difference comparing baseline with post stimulation in any of the tDCS conditions (one-hemispheric tDCS or bi-lateral tDCS) over dorsolateral prefrontal cortex in healthy subjects [36, 37]. Confounding results may be due to the difference in stimulation target, current density, time of duration and participants.

For the stimulus target area of tDCS, recent studies showed that brain stimulation leads not only to local changes of cerebral activity under the stimulated region, but also to distant changes in inter-connected brain regions throughout the brain [34, 38, 39], which is consistent with our results. Besides the local target area, we found that alpha power of some distant areas including frontal and parietal showed an increase following atDCS and bi-tDCS. Besides that, the influence of atDCS was more widespread associated with bi-tDCS.

For the duration effect of tDCS, some study reported increased alpha power during and after atDCS which persisted for 12 minutes without attenuation [18]. Spitoni et al [28] reported that the strongest change of alpha power occurred in the first 2 min after the atDCS ended, and the effect diminished systematically and was effective for approximately 8 min. We missed the first 10 min EEG information immediately after stimulation

because of placing electrodes. So only the 10–30 min EEG signals after stimulation were analyzed in the present study. Although alpha power didn't change in the 10–30 min after stimulation, they keep higher level than that of pre-stimulation, indicating that the effect maintained for at least 30 minutes with no significant attenuation.

For the clinical factors affecting modulation results, previous studies have found that tDCS stimulation efficacy may vary with time after stroke, nature and location of stroke and level of motor impairment [9, 29, 40]. We found that the change of alpha especially alpha2 in contralateral hemisphere induced by atDCS was related to time since stroke, and alpha2 in ipsilateral hemisphere induced by bi-tDCS to motor impairment level. Regression analyses confirmed that individuals' response of alpha power change to atDCS could be predicted from their time after stroke. Stroke subjects with 3 to 6 months and longer than 20 months since stroke showed higher alpha power increase than other subjects, indicating higher response to the montage of atDCS. For bi-tDCS, alpha band power increased the most in moderately impaired subjects with respect to mild and severe impairment, implying that subjects with moderate motor impairment were more susceptible to this kind of montage. Plasticity processes were variable with different phases or degree of stroke. tDCS effects may interact with these processes. Studies have suggested that patterns of neural recovery may differ for individuals based on the severity of their stroke [41–42]. The quadratic regression model was better suited to model the variation trend than linear regression model both for atDCS and bi-tDCS, indicating a complicated relation among clinical factors and EEG parameters. The result may help explain the variable rehabilitation efficacy to individuals with different clinical stroke features using tDCS.

6 Conclusions

Our results provide electrophysiological evidence that different tDCS montages in stroke subjects modulate rhythmic cortical activity of alpha band in different ways, and the effects maintained for at least 30 minutes. The tDCS modulation effect was related to clinical factors, especially the time since stroke and the level of motor impairment. These findings are of great significance for the knowledge on modulation effect to stroke patients and for therapeutic application of motor recovery following stroke.

There are some limitations in our study. Present study only found the changing of alpha power induced by tDCS, longitudinal analysis was need to verify the correlation of motor recovery and changing of alpha power. Besides that, although we did not find any difference in spectral power between pre- and post-stimulation on ctDCS montage, it does not mean ctDCS has no effect on cortical activity or stroke patients. We plan to try other methods like network connectivity or non-linear dynamic analysis to explore the performance of cortical electrical activity after ctDCS and other montages in our next work.

Declarations

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Authors' contributions

CW conducted the study and analyzed the EEG data with assistance from YZ; YC and CS supervised the complete study, including the edition of table and figure; PS recruited participants and organized their data. HY conducted

the statistical analysis; JD sponsored this study and supervised the final manuscript. All authors read and approved the final manuscript.

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Availability of data and material

The datasets used and analyzed during the current study are available from the corresponding author on request.

Ethics approval and consent to participate

All of participants were informed of all aspects of the experiment including the possibility of minor adverse effects related to tDCS, such as transient sensations of itching, burning and prickling on the scalp and signed an informed consent before the experiment started. The study was approved by the ethics committee of Tianjin Union Medical Centre.

Consent for publication

Not applicable

Declarations of interest

The authors declare that they have no competing interests.

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Figures

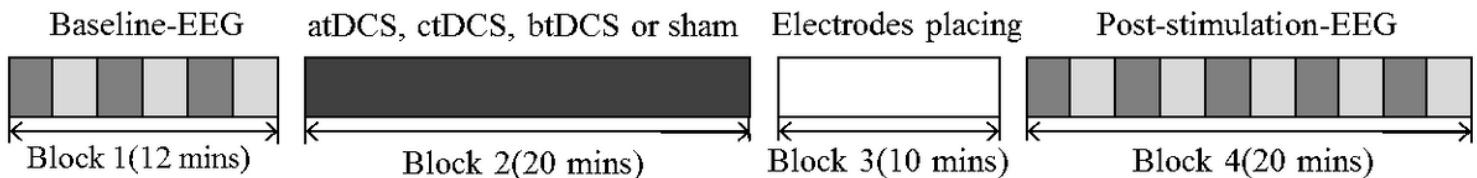


Figure 1

Experimental design of each session. Dark gray blocks represent eyes-close state; Light gray blocks represent eyes-open state. atDCS: anodal transcranial direct current stimulation; ctDCS: cathodal transcranial direct current stimulation; btDCS: bilateral transcranial direct current stimulation; Block 1: EEG signals are collected for 12 minutes which contains 6 trials with 3 eyes closing state and 3 eyes opening state. EEG signals were collected. Block 2: real (atDCS, ctDCS or btDCS) or sham tDCS is delivered for 20 minutes. Block 3: Electrodes of EEG acquisition were placed on the brain, which lasts for 10 minutes. Block 4: EEG signals are collected for 20 minutes which contains 10 trials with 5 eyes closing state and 5 eyes opening state.

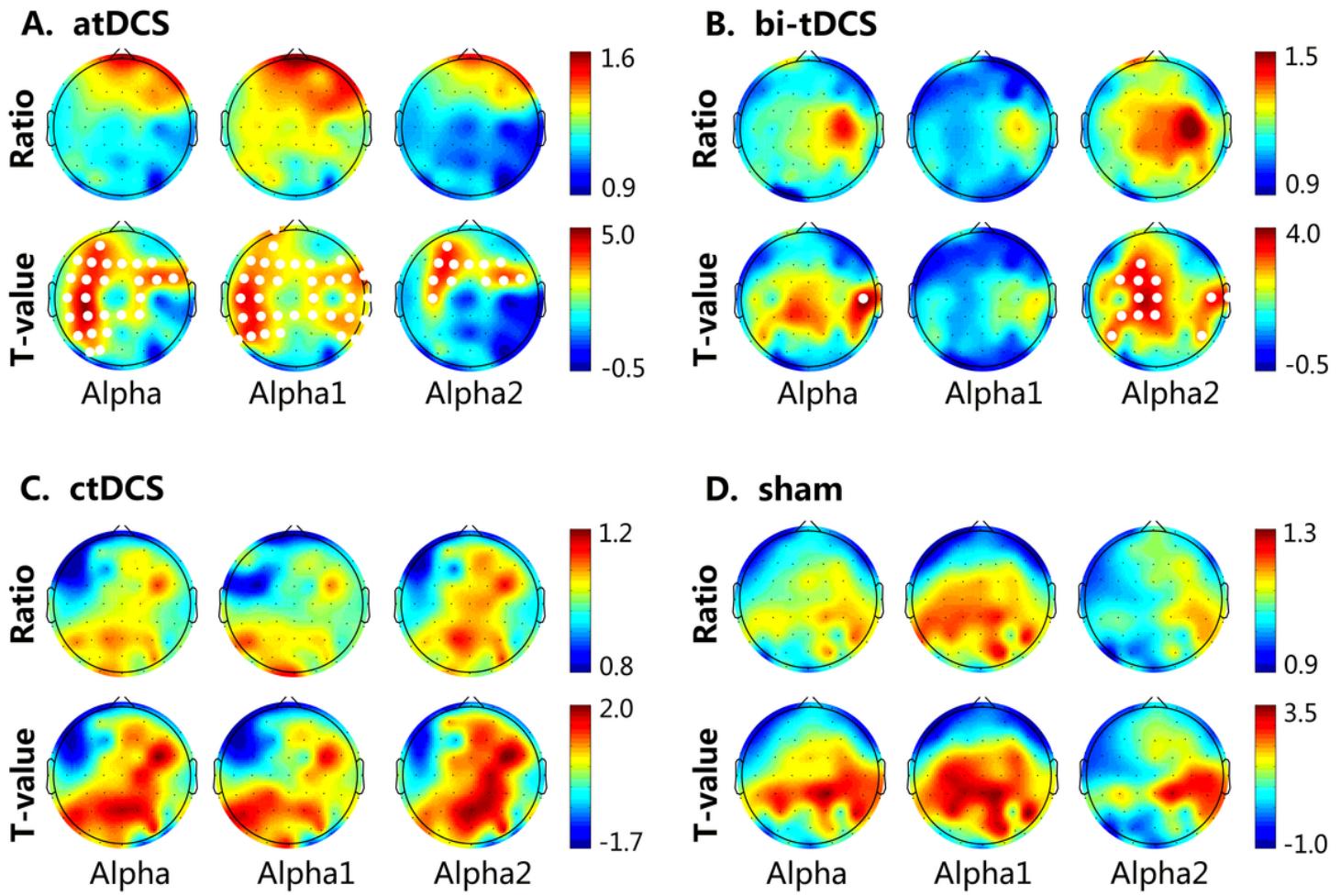


Figure 2

Cortical activity changing of alpha (8-13 Hz), alpha1 (8-10 Hz) and alpha2 (10-13 Hz) frequency band induced by anodal transcranial direct current stimulation (atDCS), cathodal (ctDCS) and bilateral (bi-tDCS)) and sham stimulation. For the topographical maps, Ratio represents the spectral power ratio of post- to pre-stimulation. T-value represents the statistic t value of the paired-samples T test. The white dots represent channels that have significant difference ($p < 0.05$) between post- and pre-stimulation. The 62 channels were normalized to ipsilateral hemisphere (left side), contralateral hemisphere (right side) and central.

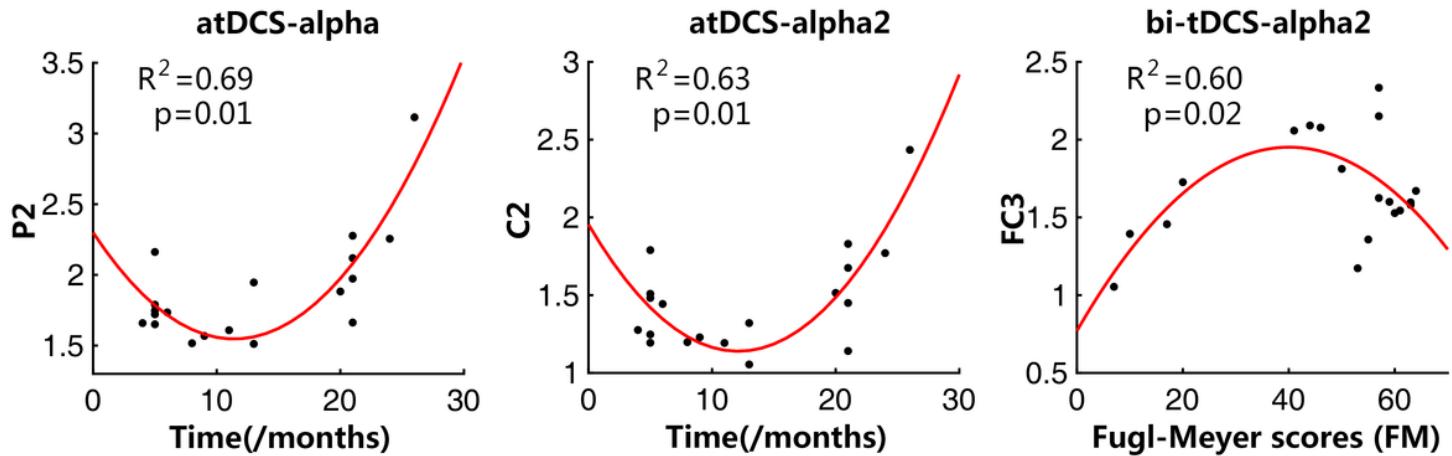


Figure 3

Scatter plots and fitted curves of representative channels with clinical scale on the abscissa and alpha power's ratio of post- to pre-stimulation. We applied quadratic fitting to these scatters with co-variables of age, gender and lesion hemisphere. The coefficient of determination R² and p-value of Fisher's F-test were shown in the plots.