Contralesional Cathodal versus Dual Transcranial Direct Current Stimulation for Decreasing Upper Limb Spasticity in Chronic Stroke Individuals: A Clinical and Neurophysiological Study

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Background: Different transcranial direct current stimulation (tDCS) paradigms have been implemented to treat poststroke spasticity, but discordant results have been reported. *Objective:* This study aimed to determine the efficacy and persistence of dual tDCS (anode over affected motor cortex [M1] and cathode over contralateral M1) compared with cathodal tDCS (cathode over contralateral M1) on upper limb (UL) functional, behavioral, and neurophysiological measures in chronic poststroke individuals. *Subjects and Methods:* Ten subjects with UL spasticity (7 men; mean 62 years; 8 ischemic stroke; years from event: 2.3 years) were enrolled in a cross-over, double-blinded study. Cathodal and dual tDCS, both preceded by 1 week of sham stimulation 1 month before real stimulation, were applied with 3 months interval. Stimulating paradigm was 20 minutes for five consecutive days in each block. Evaluations were performed before (T1), after real or sham treatment (T2), and after 1 (T3), 4 (T4), and 8 weeks (T5). Functional, behavioral, and neurophysiological tests were performed at each time. *Results:* Both tDCS paradigms decreased spasticity, increased strength, and ameliorated behavioral scales. Cathodal tDCS was superior to dual tDCS in reducing UL distal spasticity immediately after treatment (T2: cathodal > dual: $P = .023$) and provided a higher and longer lasting reduction at proximal districts (T3: cathodal > dual: $P = .042$; T4: cathodal > dual: $P = .028$; T5: cathodal > dual: $P = .05$). These findings are supported by an H-reflex modulation (overall time effect $P > .002$). *Conclusions:* Cathodal tDCS is slightly more effective than dual tDCS in reducing distal UL spasticity in chronic poststroke subjects. A modulation of spinal inhibitory mechanisms, demonstrated by H-reflex modifications, supports this finding. **Key Words:** Hemispheric imbalance—motor rehabilitation—non-invasive brain stimulation—ischemic stroke.

Introduction

Increased hand and wrist muscle tone is one of the major problems in the management of people with chronic hemiparesis and may seriously impair dressing, washing, and other activities of daily living[1] Upper motor neuron syndrome in stroke patients comprises symptoms such as muscle weakness, loss of manual dexterity, increased reflexes, and muscle hypertonia. The overall tone increase and reduced mobility produces a series of complications, such as rheological changes in muscles and connective tissues, fibrosis, and subsequent stiffness of tendons and joints.[1]

Several studies have reported that muscular injections of botulinum toxin type A decrease tone in hand

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hypertonic muscles, with improvement in the use of the upper limb (UL) and decrease of complications.[2-5] However, the dosage of the toxin cannot always be sufficient to treat extensive and severe hypertonia in upper and lower limbs. Rehabilitation, assisted arm training, and treatments directed to the connective components should also be considered.

Recent studies demonstrated that non-invasive brain stimulation (NIBS) of the motor cortex (M1) can induce motor function improvement by either diminishing excitability of the unaffected hemisphere with low-frequency repetitive transcranial magnetic stimulation (rTMS)[6-9] or cathodal transcranial direct current stimulation (tDCS),[10,11] or by enhancing excitability of the affected hemisphere by high-frequency rTMS[9] or anodal tDCS[12-17]. The rationale of this approach rests on the assumption that a motor deficit may arise from both a reduced output from the affected hemisphere and a disproportionate (i.e., uncontrasted) inhibition from the unaffected hemisphere,[18,19] that is, the "interhemispheric competition model."[20,21] Combining the two stimulations may potentiate the effects of anodal stimulation to the lesional hemisphere[22] through additional modulation of interhemispheric interactions[23] via cathodal stimulation to the contralesional motor cortex (cM1).[10] Indeed, promising outcomes on motor performance after dual tDCS have been reported both in healthy individuals[21] and in poststroke subjects,[24-27] based on behavioral outcome measures. On the other hand, a single case report[28] described, mainly with nonconventional rating scales, a reduction of spasticity after dual tDCS in a poststroke subject.

The aim of the present study was to determine which stimulation paradigm (cM1 cathodal tDCS versus dual tDCs) provides the greater and longer-lasting reduction of UL spasticity in a population of chronic poststroke individuals, quantified with specific clinical scales. Neurophysiological measures were recorded to investigate the mechanisms underlying possible effects. Cathodal tDCS was preferred over anodal based on previous reports of cathodal greater efficacy in reducing spasticity.[29]

Subjects and Methods

People with UL spasticity consequent to ischemic stroke were enrolled in the study. Inclusion criteria were age >18 years; first ever ischemic stroke at least 9 months before enrollment; no treatment of the affected limb with any botulinum toxin serotype or with phenol, alcohol, or surgery after the acute event; no contraindications to tDCS (cranial implanted metallic devices, epilepsy); UL spasticity defined as a grading ≥1 at the Modified Ashworth scale (MAS). Exclusion criteria were severe cognitive impairment, pacemaker implant, orthopedic pathology of the UL and metallic implants within the brain. The experimental protocol was approved by the local Ethic Committee and all participants provided informed consent.

Study Design

The study was a blinded cross-over study. All participants underwent a 1-week sham tDCS 4 weeks before the first block of experiment. Subjects were then randomly assigned, through a computer-generated list, either to cathodal tDCS or dual tDCS as the first type of stimulation. The second block (cathodal or dual according to the first experiment) was run 3 months after conclusion of block one (Fig 1). Participants were blinded on the stimulation sequence. To ensure blinding, tDCS was applied by a medical personnel not involved in the assessments. Active treatment consisted in cathodal tDCS applied over the cM1 for 20 minutes for 5 consecutive days; dual tDCS consisted of anodal stimulation of the affected M1 and cathodal stimulation over cM1 with the same schedule.

Clinical evaluations were performed before study beginning (T1), immediately after real or sham tDCS treatment (T2), and after 1 week (T3), 4 weeks (T4), and 8 weeks (T5) after real tDCS by an examiner blinded to the experimental condition (Fig 1).

Stimulation Paradigm

tDCS was delivered through two rubber electrodes encased in sponge soaked in a saline solution (TransQE, IOMED, Salt Lake City, UT; surface area 25 cm^2). For cathodal stimulation, the cathode was positioned on the projection of the hand knob of the unaffected primary M1 as identified by TMS stimulation, with a figure-of-eight coil (Magstim Rapid^2, Withland, UK) evoking a minimal twitch from the relaxed contralateral first dorsal interosseous muscle. The anode was placed on the skin overlying the contralateral supraorbital region. Stimulation was applied using a constant current. Current was initially increased in a ramp-like fashion over 10 seconds to reach the stimulation intensity of 1 mA. Sham stimulation was performed applying the same current for 30 seconds at the beginning and ending of stimulation. For dual tDCS, cathode was positioned over the cM1 and anode over M1, and schedule and stimulation parameters remained the same.

Outcome Measures

Clinical scales included functional and behavioral scales to assess treatment efficacy on spasticity and its correlates. The Medical Research Council scale for testing muscle strength, MAS,[1,28] finger flexion scale (Bhakta), Postural Assessment Scale for Stroke Patient, and Action Research Arm test scores were collected at each evaluation. In addition, the European Stroke Scale (ESS), Hamilton Rating Scale for Depression (HRSD), and Barthel Index

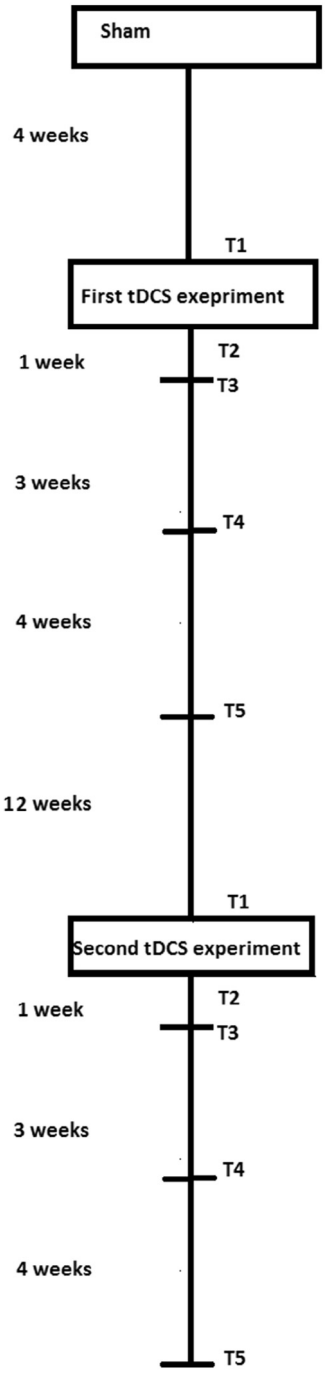

Figure 1. *Experimental design.*

were administered. Wrist and finger flexors muscle tone was evaluated with the use of Ashworth scale, having the plegic hand fixed at the wrist while stretching fingers. Passive range of motion (ROM) was assessed and graded with the aid of a digital goniometer to measure ROM at the wrist. The goniometer was synchronized with a video polygraphy (Micromed System, Brain Quick, Italy).[1,30]

A neurophysiological examination was performed to assess spinal excitability before and after sham and before and after tDCS, and after 1, 4, and 8 weeks from the active stimulation (Fig 1). Evaluation included distal motor nerve conduction velocity from abductor digiti minimi by ulnar nerve stimulation and H-reflex responses from flexor carpi ulnaris elicited by 1 Hz stimulation of the ulnar nerve at the elbow, averaging the H wave peak-to-peak amplitude and the mean latency of the responses[31] from seven responses. Cortical silent period, an interruption of voluntary muscle contraction by transcranial stimulation of the contralateral M1,[32] was measured.

Statistical Analysis

Statistical analysis was run using SPSS 20.0 (IBM SPSS Statistics for Windows, IBM Corp., Armonk, NY). Individual clinical and electrophysiological values were analyzed with a general linear model for repeated measures for each stimulation paradigm. Outcome differences between the two treatments were calculated by using "stimulation paradigm," "time," and "clinical test" as between-subjects factor; interaction between "time × stimulation paradigm," "stimulation paradigm × test," and "time × test × stimulation paradigm" were calculated. Post hoc comparisons were performed with the Bonferroni correction. The alpha level chosen for statistical significance was set at .05.

Results

Subjects

Ten subjects were enrolled (7 men; mean age 62 years, range 44-80 years; 8 ischemic stroke). They presented with mixed cortical and subcortical lesions, mainly in the territory of middle cerebral artery. Mean time from stroke was 2.3 years (range 9 months to 4 years). Most individuals were on cardiovascular medications (antiplatelet and antihypertensive treatments), but none on drugs interfering with tDCS (e.g., gamma-aminobutyric acid [GABA]ergic). Indeed, to avoid possible confounders, drug regimen was maintained stable throughout the study.

Sham Stimulation

No significant changes in any of the clinical scales were observed.

Cathodal tDCS

Mean values and standard deviations (SDs) of test results are reported in Table 1.

Cathodal tDCS induced an overall improvement over time, with a time effect significant for all tests ($P = .0001$, F (7): 32.26).

Behavioral Measures

The HRSD showed an improvement over time (F (7): 5.2; $P = .029$), with an improvement immediately after stimulation that gradually waned off at last follow-up visit.

Table 1. *Clinical scales' values at different time points are reported for cathodal and dual tDCS stimulation*

		Cathodal	SD	Dual tDCS	SD	*P* value			Cathodal tDCS	SD	Dual tDCS	SD	*P* value
MRC hand	T1	1.4	1.3	1.9	1.6		European Stroke Scale	T1	66.0	12.5	66.7	11.9	
	T2	2.3	1.2	3.1	1.2	0.023		T2	69.0	13.2	67.2	11.9	
	T3	2.1	1.3	2.5	1.5			T3	69.0	13.2	67.1	11.7	
	T4	1.8	1.3	2.1	1.4			T4	68.0	12.6	66.9	11.9	
	T5	1.7	1.3	2.0	1.5			T5	68.0	13.1	66.9	11.9	
MRC wrist	T1	1.3	1.1	1.6	1.0		Bhakta fist	T1	12.5	6.1	2.6	1.5	
	T2	2.2	1.1	2.6	1.0			T2	10.0	5.2	3.1	1.2	
	T3	2.0	1.2	2.0	1.0			T3	10.0	5.3	2.8	1.4	
	T4	1.7	1.3	1.7	1.0			T4	11.5	5.8	2.8	1.4	
	T5	1.7	1.3	1.7	1.0			T5	12.0	5.9	2.8	1.4	
MRC elbow flexion	T1	2.0	1.2	2.6	1.0		Bhakta fist	T1	3.0	1.5	2.6	1.5	
	T2	2.9	1.2	3.1	0.7			T2	4.0	1.1	3.1	1.2	
	T3	2.6	1.3	2.7	1.0	0.042		T3	4.0	1.3	2.8	1.4	
	T4	2.3	1.2	2.7	1.0	0.023		T4	4.0	1.4	2.8	1.4	
	T5	2.3	1.2	2.7	1.0	0.05		T5	4.0	1.4	2.8	1.4	
MRC shoulder abduction	T1	2.6	1.0	2.6	1.0		Action research arm test	T1	6.5	5.0	5.4	4.8	
	T2	3.2	0.9	3.1	0.7			T2	8.5	6.4	7.9	6.1	
	T3	3.1	0.9	2.7	1.0	0.063		T3	8.5	6.4	7.4	6.1	
	T4	2.9	0.9	2.7	1.0			T4	7.5	5.6	6.4	5.4	
	T5	3.1	0.6	2.7	1.0			T5	7.0	5.4	6.0	5.2	
MAS finger flexion	T1	3.1	1.1	3.2	1.1		Hamilton Rating Scale for Depression	T1	7.0	6.8	9.3	6.5	
	T2	2.2	1.0	2.5	1.0			T2	7.0	6.9	11.0	7.1	
	T3	2.3	1.2	2.9	1.2			T3	7.0	6.8	10.9	7.1	
	T4	2.5	1.1	3.1	1.2			T4	7.0	6.8	10.7	7.2	
	T5	2.5	1.2	3.1	1.2			T5	7.0	6.8	10.6	7.3	
MAS wrist flexion	T1	2.9	0.7	2.9	0.7		Barthel Index	T1	67.5	14.0	69.1	13.4	
	T2	2.0	0.8	2.0	0.6			T2	72.5	14.7	72.5	14.2	
	T3	2.1	0.9	2.5	0.8			T3	70.0	14.8	71.1	14.5	
	T4	2.2	0.8	2.7	0.8			T4	70.0	15.3	68.8	12.9	
	T5	2.4	0.7	2.9	0.7			T5		15.3	68.6	12.9	
MAS elbow flexion	T1	2.9	1.0	3.2	0.8		Postural assessment scale	T1	21.5	5.0	24.6	5.1	
	T2	2.1	0.7	2.4	1.0			T2	23.5	4.9	26.9	5.0	
	T3	2.2	0.8	2.9	0.8			T3	23.5	5.0	25.9	5.3	
	T4	2.4	0.7	3.1	0.7			T4	23.5	5.1	25.2	5.1	
	T5	2.5	0.8	3.2	0.8			T5	23.5	5.1	25.2	5.1	
MAS shoulder abduction	T1	2.3	0.8	2.6	0.7								
	T2	2.0	0.0	2.1	0.9								
	T3	2.0	0.0	2.5	0.8								
	T4	2.2	0.4	2.6	0.7								
	T5	2.2	0.4	2.6	0.7								

Abbreviations: MAS, modified Ashworth scale; MRC, Medical Research Council scale; SD, standard deviation; tDCS, transcranial direct current stimulation; T1, baseline; T2, immediately after stimulation; T3, after 1 week from stimulation; T4, after 4 weeks from stimulation; T5, after 8 weeks from stimulation.

Note reduction of finger spasticity with cathodal stimulation immediately after tDCS (T2) and a higher and longer lasting effect of cathodal tDCS at the elbow (T3, T4, T5).

P values refer to statistically significant differences between the two stimulation paradigms at different time points.

Barthel Index had a time effect (F (7): 5.2; $P = .029$), with an improvement at immediate testing after tDCS (and after 1 week, as did the Action Research arm test (F (7): 5.1; $P = .03$). No time effect emerged for ESS. Lastly, the postural assessment scale had an effect over time (F (8): 47.32; $P = .0001$), with an improvement immediately after stimulation and after 1 week, and after 4 and 8 weeks (compared to baseline).

Bhakta followed a time-dependent improvement (F (8): 7.09; $P = .009$), more marked immediately after treatment and after 1 week, which gradually faded off over time.

Spasticity Measures

For overall Ashworth score, the time effect was statistically significant (F (12): 84.707, $P = .001$).

Immediately after active treatment, Ashworth score for finger flexors, wrist flexors (Fig 2), and elbow flexors and extensors (T1 > T2: $P = .008$) dropped dramatically. All participants reported subjective decrease of discomfort in the hypertonic muscles and a sensation of suppleness in the hand and forearm muscles. After 1 week from cathodal active treatment, Ashworth score decreased although a persistent benefit was still present, which was marked more distally. After 4 weeks from active treatment, Ashworth score for finger and wrist flexors still showed benefits, which lasted to the eighth week.

The finger flexors showed a marked reduction of spasticity with an Ashworth change of .8 ± .4 ($P = .001$) (Fig 2, Table 1).

A similar trend over time was observed for Medical Research Council (F (8): 12.6; $P = .002$), with an effect that was concentrated over the first weeks after stimulation (Fig 3).

Concerning range of passive motion, time effect was statistically significant (F (12) = 81.457, $P < .001$). Post hoc comparisons showed a significant difference of ROM between baseline and immediately after stimulation ($P < .01$) (20° versus 50°, respectively), and between baseline and the second week ($P < .05$) (20° versus 40°, respectively). There were no differences between baseline and muscle tone of the wrist flexors after 4 weeks (P = NS).

Neurophysiological Measures

Motor nerve conduction, motor action potential amplitude and latency, and cortical silent period remained unchanged during the course of the experiment (please refer to Table 2 for mean values and SD). A time effect for H was detected ($P < .001$). A slight decrease of H wave latency was detected after stimulation over time immediately after and after 2 weeks from stimulation ($P < .002$).

Dual tDCS

Mean values and SDs of test results are reported in Table 1.

The time effect was statistically significant for all tests ($P = .0001$, F (7): 32.26).

Behavioral Measures

The HRSD had a time effect (F (7): 5.2; $P = .029$). A marked improvement emerged after stimulation in comparison to baseline with gradual effect reduction. Barthel Index did not show a time effect (F (7): 3.06; $P = .094$), with improvement only immediately after stimulation. The Action Research arm test displayed a time effect (F (7): 5.1; $P = .03$), with an immediate effect that persisted over

Table 2. *Neurophysiology results are reported for cathodal and dual stimulation.*

Variables	tDCS	T0	T1	T2	T3	T4	T5	*P* value (GLM model)
Motor nerve conduction (ms)		55 (6)	54 (7)	57 (5)	54 (8)	55 (7)	56 (7)	NS
CMAP latency (ms)	Cat	3.5 (1.3)	3.6 (2)	3.9 (1.7)	3.6 (1.4)	3.5 (2)	3.5 (3)	NS
	Dual	3.4 (1.5)	3.6 (2.5)	3.8 (1.7)	3.5 (1.7)	3.5 (2)	3.4 (1.2)	NS
CMAP amplitude (mV)	Cat	11 (2)	12 (2)	11 (2)	12 (1.8)	11 (2.1)	11 (2)	NS
	Dual	11 (2.5)	12 (2)	11 (2)	11 (2.85	11 (2.1)	11 (2.7)	NS
H wave latency (ms)	Cat	29.4 (3.2)	25 (6)	26.4 (.9)	28.9 (3.8)	27 (4.6)	31.2 (4.1)	$P < .002$
	Dual	29.7 (4)	25.1 (6)	27.4 (1.3)	29 (4)	29.3 (4.5)	29.2 (4.1)	$P < .01$
H wave amplitude (uV)	Cat	790 (120)	325.1 (247.7)	543.8 (267.1)	288.2 (262)	450.7 (242.3)	264.3 (70.3)	$P < .001$
	Dual	680.7 (134.7)	340.1 (229.8)	498.7 (230.6)	390.2 (47.8)	386.7 (256.4)	289.3 (100.2)	$P < .05$
Silent period healthy hemisphere (ms)	Cat	35.1 (14.5)	33.1 (15.5)	34.8 (19.9)	28.9 (19.5)	36.3 (21)	33.3 (19.1)	NS
	Dual	34.3 (11.7)	34.6 (17.9)	35.2 (24.5)	27.9 (15.8)	32.1 (19.4)	29.3 (21.4)	NS

Abbreviations: CMAP, compound motor action potential; GLM, general linear model; NS, not significant; tDCS, transcranial direct current stimulation; T1: baseline; T2: immediately after stimulation; T3: after 1 week from stimulation; T4: after 4 weeks from stimulation; T5: after 8 weeks from stimulation.

Note H wave modifications immediately after treatment, with a major effect for cathodal stimulation.

P values refer to the time overall factor.

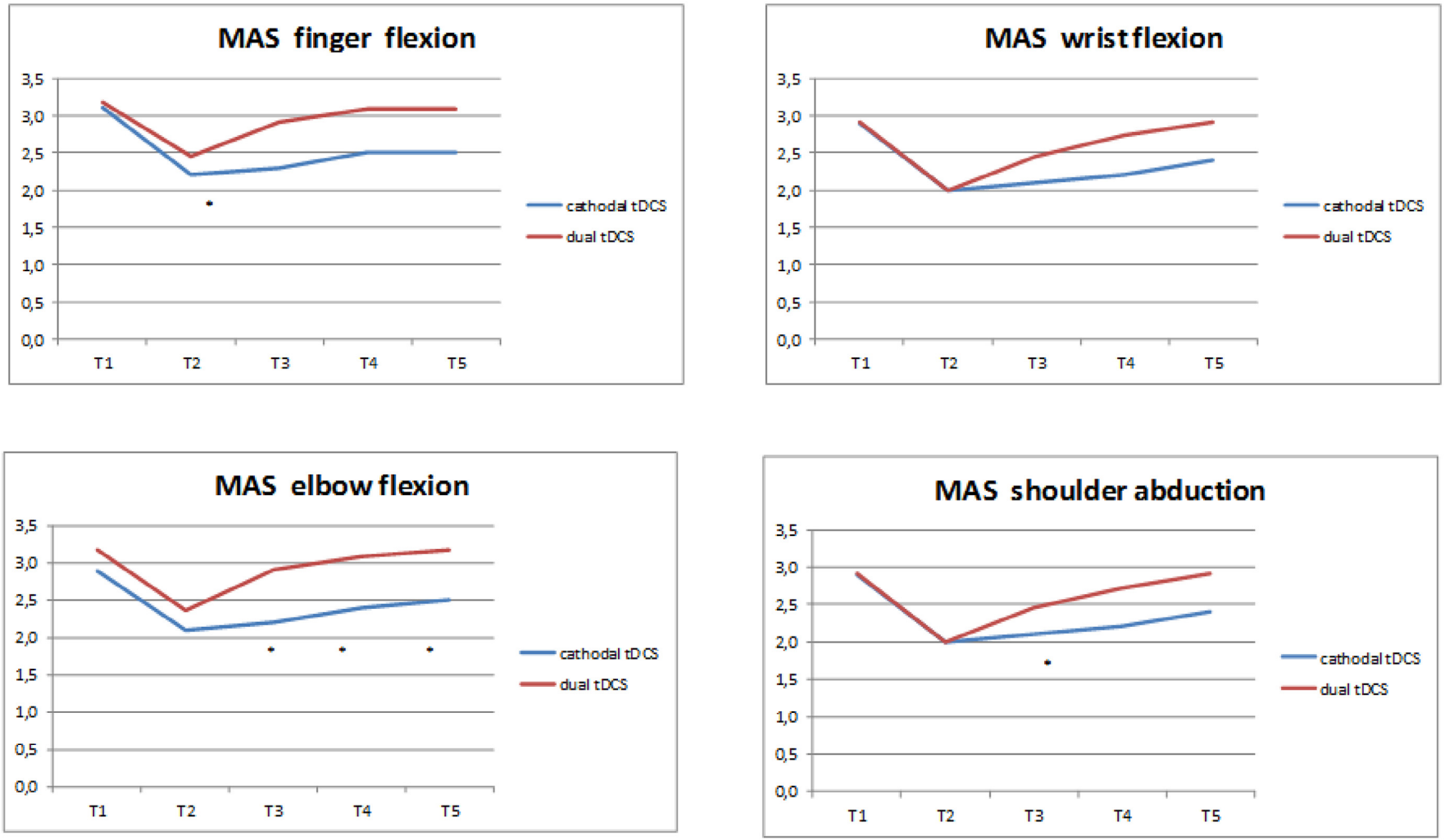

Figure 2. *MAS modification over time for finger, wrist and elbow flexion, and shoulder abduction. Asterisks mark significant statistical differences. Abbreviation: MAS, modified Ashworth scale.*

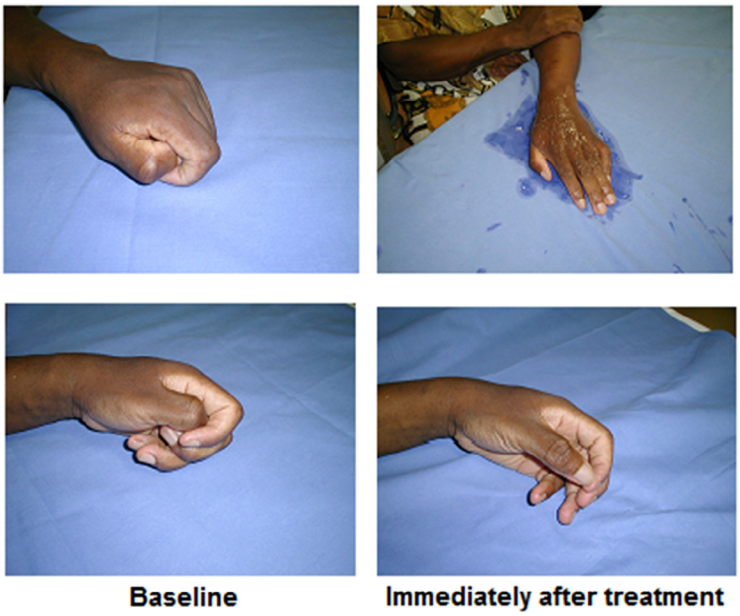

Figure 3. *Clinical findings in a treated subject before and at conclusion of dual tDCS stimulation paradigm. Abbreviation: tDCS, transcranial direct current stimulation.*

time up to 8 weeks). Lastly, the postural assessment scale showed a time effect (F (8): 47.32; $P = .0001$), with a marked improvement immediately after stimulation that gradually diminished over time. No modifications were detected for ESS.

No time effect emerged for the Bhakta test (F (9): 3.54; $P = .073$), which improved only immediately after stimulation (Table 1).

Spasticity Measures

Time effect was statistically significant (F (8): 12.6; $P = .002$). Spasticity as measured by the MAS showed a time-dependent reduction only for elbow extension (F (8): 7.9; $P = .009$). Distal segments did not benefit immediately except just after 1 week from stimulation completion, whereas the effect on the elbow was more marked with a clear time dependency and a higher effect directly after stimulation. Muscle strength showed a time effect [F (8): 12.6; $P = .002$]. Improvement was immediate, with an effect lasting up to 1 week after tDCS for finger, wrist, and elbow flexion and shoulder abduction.

Concerning range of passive motion, time effect was statistically significant (F (12) = 81.457, $P < .001$). Post hoc comparisons showed a significant difference of ROM between baseline and immediately after stimulation ($P < .01$) (20° versus 50°, respectively) and between baseline and the second week ($P < .05$) (20° versus 38°, respectively). There were no differences between baseline and muscle tone of the wrist flexors after 4 weeks (P = NS).

Neurophysiological Measures

Motor nerve conduction, motor action potential amplitude and latency, and cortical silent period remained unchanged during the course of the experiment (please refer to Table 2 for mean values and SD). A time effect for H was detected ($P > .01$). A slight decrease of H wave latency was detected after stimulation over time immediately after stimulation ($P < .05$).

Effects Comparison between Cathodal and Dual tDCS

The two stimulation protocols produced overall comparable results in terms of evaluation scores improvement and modification of parameters over time. The only exception was a higher reduction of finger spasticity with cathodal stimulation immediately after tDCS (T2 cathodal > dual: $P = .023$) and a higher and longer lasting effect of cathodal tDCS at the elbow (T3 cathodal > dual: .042; T4 cathodal > dual: .028; T5 cathodal > dual: $P = .05$). A tendency for MAS improvement at the shoulder emerged for cathodal tDCS (T3 cathodal > dual: $P = .063$). H-reflex modulation persisted slightly longer for cathodal tDCS (T2 cathodal > dual: $P = .05$).

Discussion

The present results provide evidence of the substantial comparable efficacy on functional and behavioral UL parameters of cathodal tDCS applied over the unaffected M1 and dual tCDS (anode over lesional primary M1 and cathode over the contralateral one). The only exception was for the MAS, which showed a slightly higher and longer lasting benefit with cathodal tDCS.

The novelty of this finding is that neurophysiological data support the results: we observed a modulation of the H-reflex after stimulation.

H-reflex is a monosynaptic spinal reflex that measures alpha motor neurons excitability, which is one of

the neurophysiological signatures of spasticity in association with decreased presynaptic and reciprocal inhibition and reduced facilitation of Ia nerve fibers. H-reflex is considered a reliable and objective measure of spasticity.

The rationale for decreasing cM1 excitability, possibly coupled with an induced increase of lesional motor area excitability in dual tDCS, is backed by the neurophysiological phenomenon of imbalance in primary M1 excitability. This phenomenon causes a relative under-excitability in the stroke-affected hemisphere and a relative over-excitability in the contralesional hemisphere, with worse clinical outcomes for patients with greater imbalance.[33] Rebalancing of cortical excitability in patients with stroke has been associated with improved UL function.[6,33-36] To potentiate this rebalancing mechanism, dual tDCS has been deployed, in which ideally activation of the ipsilateral affected motor area is contemporarily coupled with inhibition of the hyperfunctioning contralateral cortex.[37] This rationale has informed several dual tDCS studies which have reported transient changes in motor evoked potentials (MEPs) and improved hand motor performance in healthy controls and stroke patients.[21,25-27,37] However, for standard tDCS protocols (e.g., 1 mA, 20 minutes), the assumption that a bilateral montage induces neurophysiological results on the M1 in a simple summative fashion (i.e., anode induced excitability increase plus cathode decrease) does not appear to hold. A recent study[38] reported a lack of efficacy of dual tDCS compared with anodal or cathodal tDCS in modifying hand MEPs in healthy subjects or to improve motor performance in the paretic limb of chronic stroke patients, concluding that dual tDCS is less effective than either anodal or cathodal tDCS.

To date, a single report focuses on dual tDCS efficacy in reducing spasticity, described as incidental find in patients undergoing stimulation to increase motor learning.[28] Indeed, whereas we did not recognize significant differences on the outcome measures of strength and behavioral parameters, spasticity was the only differential outcome. Following cathodal tDCS, UL spasticity was greatly reduced immediately after stimulation, with a stronger and longer lasting effect.

This clinical finding is supported by the H-reflex amplitude decrease and latency increase. H-reflex is modulated by inhibitory reciprocal neurons, which are conversely under control of inhibitory descending fibers. Cortical neuromodulation appears thus to act on inhibitory components through the cortico-reticular spinal tract, strengthening the already known neuroplastic changes induced by tDCS.

Our results are in contrast to previously published ones,[25] reporting comparable benefits of anodal, cathodal, and bihemispheric tDCS: in fact, the authors used as evaluation tool a performance test, the Jebsen–Taylor Test (JTT), in which different hand tasks are scored, omitting other assessments. It is possible that our data could pick up more subtle nuances, given the wider array of tests we used.

Both stimulation paradigms had an overall equal effect on muscle strength improvement. Upper motor neuron weakness is determined by a reduced firing frequency of involved motor units,[39,40] whereas spasticity, a condition in which stretch reflexes that are normally latent become obvious, rests on the monosynaptic inhibitory input to antagonist alpha motoneuron when Ia fibers surrounding intrafusal muscle spindles are stretched. Upper motor neuron firing rate is likely to be equally enhanced by both stimulation types: tDCS modulates action potential threshold of neurons underlying the electrode.

On the other hand, spasticity, through its local monosynaptic circuits controlled by polysynaptic descending pathways, seems to be more influenced by a reduction of contralateral excitatory control than by the combination of anodal/cathodal stimuli. According to computational models of tDCS electric field distribution,[41] unilateral tDCS exerts its effect only over the stimulated hemisphere, opposed to the bi-hemispheric spread of dual tDCS. The bilateral activation with opposite polarities of descending fiber bundles, such as the corticospinal and rubrospinal pathways, which have a facilitatory effect on flexor reflexes,[42] could determine a subtractive effect of descending volleys, thus explaining the less satisfactory result of this type of stimulation.

A limitation of our work is the small sample size, which hampered us from performing a more detailed statistical analysis, for example, stratifying for lesion severity. Indeed, given the wealth of data on predictive factors of response to tDCs, we felt such a result to be redundant.

Conclusions

We provide evidence of the slightly higher efficacy of cathodal tDCS over the unaffected hemisphere in reducing UL spasticity in chronic stroke people compared with dual tDCS. This finding is supported by a modulation of the H-reflex, pointing to an effect of neuromodulation on spinal inhibitory circuits. The development of new non-invasive brain stimulation techniques and the wealth of data warrant an ongoing research by the scientific community to ascertain their efficacy and applications.

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