Transcranial Direct Current Stimulation Improves Naming Reaction Time in Fluent Aphasia
A Double-Blind, Sham-Controlled Study

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Background and Purpose—Previous evidence suggests that anodal transcranial direct current stimulation (A-tDCS) applied to the left hemisphere can improve aphasic participants’ ability to name common objects. The current study further examined this issue in a more tightly controlled experiment in participants with fluent aphasia.

Methods—We examined the effect of A-tDCS on reaction time during overt picture naming in 8 chronic stroke participants. Anode electrode placement targeted perilesional brain regions that showed the greatest activation on a pretreatment functional MRI scan administered during overt picture naming with the reference cathode electrode placed on the contralateral forehead. A-tDCS (1 mA; 20-minute) was compared with sham tDCS (S-tDCS) in a crossover design. Participants received 10 sessions of computerized anomia treatment; 5 sessions included A-tDCS and 5 included S-tDCS.

Results—Coupling A-tDCS with behavioral language treatment reduced reaction time during naming of trained items immediately posttreatment (Z = 1.96, P = 0.025) and at subsequent testing 3 weeks later (Z = 2.52, P = 0.006).

Conclusions—A-tDCS administered during language treatment decreased processing time during picture naming by fluent aphasic participants. Additional studies combining A-tDCS, an inexpensive method with no reported serious side effects, with behavioral language therapy are recommended. (Stroke. 2011;42:819-821.)

Key Words: anomia ▪ brain stimulation ▪ recovery ▪ stroke ▪ tDCS

Recently, our group demonstrated how anodal transcranial direct current stimulation (A-tDCS) can enhance the effect of behavioral aphasia treatment.1 Ten patients, with varying types and severities of chronic aphasia, received computerized aphasia treatment coupled with A-tDCS to the left frontal lobe. In 4 of these patients, A-tDCS significantly amplified the effect of the aphasia treatment compared with sham tDCS (S-tDCS). Inspection of the data suggested that good responders primarily had left frontal lobe damage; for those patients, stimulation occurred closer to the perilesional rim compared with the remaining patients whose damage was more posterior. Although the effect of A-tDCS varied across patients, this study yielded overall positive results, warranting further research.

The current study improved on the previous study in the following ways: (1) in addition to blinding both participants and clinicians who scored pre- and postnaming tests, clinicians who administered the tDCS protocol and the computerized treatment were also blinded to stimulation type; (2) instead of including a broad range of aphasia types and lesion sites, all participants had fluent aphasias with posterior cortical or subcortical lesions; and (3) maintenance testing was extended from 1 to 3 weeks posttreatment.

The final difference between the present study and our earlier work was the selection of a posterior rather than anterior focus for stimulation. Although Baker et al1 targeted the left frontal lobe with A-tDCS, recent work2-4 suggests that increased left hemisphere activation in both anterior and posterior regions supports treatment-assisted improvement in naming among aphasic participants. Therefore, anode electrode placement here targeted perilesional brain regions showing the greatest activation on a pretreatment functional MRI scan during overt picture naming.

We maintained the design feature whereby A-tDCS was compared with a placebo (S-tDCS) administered in a crossover design. Each participant received 10 sessions of computerized aphasia treatment, 5 of which included A-tDCS and the other 5 S-tDCS.

The participants in this study had relatively good scores on the assessment used to chart naming improvement, limiting naming accuracy as a measurement of treatment-related change. Therefore, reaction time (RT) was chosen as the dependent measure because we expected it would be sensitive to treatment-related changes among participants whose anomia, in most cases, was mild.
Methods

Participants ranged in age from 53 to 79 years (mean, 68.13 years; SD, 10.40); time poststroke ranged from 10 to 150 months (mean, 58.38 months; SD, 44.60). A computerized anemia treatment coupled with tDCS was administered during 2 treatment phases. Each treatment phase lasted 1 week with 3 weeks separating the 2 treatment phases (A-tDCS versus S-tDCS). The number of treatment sessions (5 consecutive days for each treatment phase), length of stimulation (20 minutes per session), and stimulation intensity (1 mA) was modeled after previous research. A computerized naming assessment of both trained and untrained words was conducted 6 times for each treatment phase (A-tDCS and S-tDCS): twice at baseline, twice immediately after the final treatment session for each phase, and twice at 3 weeks after completion of each treatment phase.

During both A-tDCS and S-tDCS, the anode electrode was placed over the predesignated area of the scalp overlying the left posterior cortex. The reference cathode electrode was placed on the right forehead. To ensure blinding, in-house software switched the tDCS on and off without intervention from the participant or experimenter. For S-tDCS, the stimulator was turned off after 45 seconds; for A-tDCS, stimulation was maintained for 20 minutes.

The self-administered computerized treatment consisted of a spoken word–picture matching task occurring concurrently with the application of tDCS. Beginning 5 minutes before the start of tDCS, this treatment was modeled after tasks used in previous studies that have resulted in improved naming accuracy in participants with aphasia. Separate word lists were used for each treatment phase.

Results

After A-tDCS, the median group change in RT for trained items was −455.57 ms (interquartile range, −672.08 to −393.93) immediately posttreatment and −430.06 ms (interquartile range, −511.63 to −346.83) at 3 weeks posttreatment. Comparable reductions in RT after S-tDCS were −281.17 ms (interquartile range, −516.54 to −241.77) immediately posttreatment and −265.86 ms (interquartile range, −328.25 to 228.62) 3 weeks posttreatment (Figure). To minimize the effect of outliers, all RT values greater than ±2 SD from the mean were removed and the mean recalculated. A Wilcoxon signed rank test (1-tailed) revealed greater reduction in RT during correct naming of trained nouns after A-tDCS compared with S-tDCS immediately on treatment completion (Z=1.96, P=0.025) and also at 3 weeks posttesting (Z=2.52, P=0.006). On immediate posttesting, 7 of 8 participants experienced greater reduction in RT after the A-tDCS compared with the S-tDCS. At 3 weeks posttesting, all 8 participants experienced greater reduction in RT after A-tDCS compared with S-tDCS. The probability that of 16 comparisons, 15 occurred in a direction that favored A-tDCS (7 for immediate posttesting and 8 for 3-week follow-up) was calculated (P=0.0002; based on binomial distribution).

Discussion

This study included 8 participants with fluent aphasia and revealed greater treatment-related reduction in RT during naming of trained items after A-tDCS compared with S-tDCS immediately after treatment completion as well as at 3-week follow-up testing. This treatment effect was not accounted for by unspecific arousal differences (ie, changes in blood pressure, heart rate recordings, etc), differences in comfort level (ie, scalp sensations), or treatment order.

Differences in stimulus generalization were absent because RTs were similar for untrained items after both treatment conditions. However, the receptive treatment task did not include overt naming, suggesting that greater response generalization occurred after A-tDCS than S-tDCS. This relates to the work of Fritsch and colleagues, who found that the effect of A-tDCS on motor learning is stimulus-driven because A-tDCS in the absence of behavioral training did not improve task performance. Specifically, their study revealed A-tDCS induces secretion of brain-derived neurotrophic factor (BDNF), a protein crucial for new learning. It is possible that increased BDNF secretion in perilesional areas promoted improved naming performance among our participants. Based on Baker et al and the current data, it is reasonable to propose that positive treatment effects may be further enhanced and maintained by coupling language stimulation with A-tDCS applied to the left hemisphere.

The current findings warrant further investigation to evaluate the effect of A-tDCS on aphasia recovery. Clearly, more research is needed to understand factors such as stimulus
generalization, brain plasticity associated with A-tDCS, the necessary time course of stimulation, and perhaps most importantly, the ecological validity of this method. Our hope is that in the future, research such as this may aid aphasia recovery.

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Disclosures
None.

References
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