

The ontogenetic model for CVA rehabilitation with transcranial stimulation

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Abstract

Currently, transcranial stimulation for CVA treatment is based on the interhemispheric rivalry model. This model has proven to have many anomalies, necessitating a new paradigm. Spontaneous recovery from post-CVA hemiplegia has an ontogenetic pattern. We reanalyzed the 2008 longitudinal London study and found that cortical disinhibition is the mechanism for ontogenetic CVA recovery. We propose that transcranial stimulation with 10 Hz rTMS or anode electrical microstimulation can produce CVA recovery similar to spontaneous recovery. (*Acta Med Colomb* 2022; 47. DOI: <https://doi.org/10.36104/amc.2022.2466>).

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Introduction

For the last 15 years studies have been done with transcranial stimulation for cerebrovascular accident (CVA) rehabilitation, based on the interhemispheric competition model (1). According to this model, after the CVA, the contralesional hemisphere worsens the function of the injured hemisphere. Thus, the objective of CVA treatment with transcranial stimulation is to increase the excitability of the injured hemisphere or inhibit the contralesional hemisphere (2). However, this model has been questioned from the beginning (3), there is no evidence to date to support its clinical use (4), patients with severe deficit may worsen (5), and its neurophysiological bases are wrong (6). In light of these very significant anomalies, a new paradigm is needed.

Natural or spontaneous CVA recovery is produced through brain plasticity mechanisms (4). Spontaneous recovery from post-CVA hemiplegia has an ontogenetic pattern, first recovering axial-proximal and then distal movement (7, 8). If we understand the mechanisms of ontogenetic recovery, we can design strategies to achieve CVA recovery similar to spontaneous recovery (9).

Ontogenetic CVA recovery, reopening of critical periods and cortical disinhibition

Coupling between the genetically determined brain connectivity and the individual's experiences occurs during the critical neurodevelopment periods (10). Reopening the critical periods causes rejuvenation of brain plasticity. One way of reopening the critical periods is through cortical

disinhibition (11). The most studied critical period is that of ocular dominance. Children with strabismus or congenital cataracts will have normal vision if they undergo surgery during the critical period; otherwise, the children will develop amblyopia. Since cortical disinhibition improves amblyopia (12-16), this mechanism is thought to reopen the critical period of ocular dominance.

The critical periods are rich in brain plasticity, and their reopening has been suggested as treatment for CVAs (10, 17). Ontogenetic CVA recovery is accompanied by an increase in proteins related to the critical periods (8). The London group confirmed the pattern of post-CVA ontogenetic recovery and found that this recovery is related to cortical disinhibition processes at three months (18). Recently, critical motor period reopening following a CVA was shown to occur in the second and third months (19). Cortical disinhibition may also open the critical motor period in patients with CVAs.

Recovery from post-CVA hemiplegia and phased recruitment

The London group proposed that the recovery of patients with severe CVAs is produced by phased recruitment of the contralesional premotor cortex (PMC) and the ipsilesional primary motor cortex (M1) (18). Most of the corticoreticulospinal tract (CRST) originates in the PMC (20). In post-CVA adults, the CRST exerts most of its connectivity on the proximal muscles (21). Most of the corticospinal tract originates from the M1, which is mainly responsible

for distal extremity movement (22). Thus, we speculate that the London group's proposal is in line with the ontogenetic pattern of CVA recovery (9).

Brain stimulation for CVA recovery with an ontogenetic pattern

Repetitive transcranial magnetic stimulation (rTMS) at 10 Hz induces cortical disinhibition (23). Since the application of 10 Hz rTMS improves adult patients with amblyopia (14), it is suggested that this procedure may reopen the critical period of ocular dominance.

We report a patient with chronic post-brainstem CVA hemiplegia, who after two cycles of bilateral 10 Hz rTMS, initially used as treatment for dysphagia, recovered axial-proximal movement and postural control (24). After a third cycle, minimal voluntary distal movement appeared (9). We speculate that our patient's recovery, with an ontogenetic pattern, was triggered by reopening critical periods using rTMS at a frequency which induces cortical disinhibition. However, since this is a report of a single case, we cannot rule out spontaneous improvement or the placebo effect.

There are two situations to keep in mind. First, the response to rTMS depends on the baseline levels of cortical inhibition, which could explain why some patients respond to rTMS treatment and others do not (25, 26). The second is that early disinhibition should be avoided, as this can worsen the CVA's severity in animals (27).

Conclusion

We propose that the ontogenetic post-CVA recovery model is related to reopening of the critical motor period due to cortical disinhibition. We suggest that cortical disinhibition induced by 10 Hz rTMS can reopen this period and allow CVA recovery similar to spontaneous recovery. Since anode electrical microstimulation induces cortical disinhibition (28) and improves amblyopia (16), it may also be used to reopen the critical motor period.

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