



Measuring and inducing brain plasticity in chronic aphasia

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ABSTRACT

Brain plasticity associated with anomia recovery in aphasia is poorly understood. Here, I review four recent studies from my lab that focused on brain modulation associated with long-term anomia outcome, its behavioral treatment, and the use of transcranial brain stimulation to enhance anomia treatment success in individuals with chronic aphasia caused by left hemisphere stroke. In a study that included 15 participants with aphasia who were compared to a group of 10 normal control subjects, we found that improved naming ability was associated with increased left hemisphere activity. A separate study ($N=26$) revealed similar results in that improved anomia treatment outcome was associated with increased left hemisphere recruitment. Taken together, these two studies suggest that improved naming in chronic aphasia relies on the damaged left hemisphere. Based on these findings, we conducted two studies to appreciate the effect of using low current transcranial electrical stimulation as an adjuvant to behavioral anomia treatment. Both studies yielded positive findings in that anomia treatment outcome was improved when it was coupled with real brain stimulation as compared with a placebo (sham) condition. Overall, these four studies support the notion that the intact cortex in the lesioned left hemisphere supports anomia recovery in aphasia.

Learning outcomes: Readers will (a) be able to appreciate the possible influence of animal research upon the understanding of brain plasticity induced by aphasia treatment, (b) understand where functional changes associated with anomia treatment occur in the brain, (c) understand the basic principles of transcranial direct current stimulation, and (d) understand how brain stimulation coupled with aphasia treatment may potentially improve treatment outcome.

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1. Introduction

Improved understanding of plasticity in the adult brain suggests that the human cortex is quite amenable to both functional and structural change (e.g., Butefisch, 2004; Heiss & Thiel, 2006; Meinzer et al., 2008; Nudo, 2003, 2007; Pascual-Leone, Amedi, Fregni, & Merabet, 2005; Saur et al., 2006; Thompson & den Ouden, 2008). Currently, there is no evidence suggesting that plasticity is absent or even reduced following brain damage. In fact, neural sprouting has been shown to be enhanced in the brain regions surrounding the frank cortical lesion (Nudo, 1999; Stroemer, Kent, & Hulsebosch, 1995). For the most part, aphasia treatment research has taken only limited advantage of the recently improved understanding of brain plasticity. Although several reasons may underlie this development, or lack thereof, it is undoubtedly important that much of the recent advances in understanding brain plasticity have been revealed in animal models of motor impairment. For example, utilizing a rat model, Jones and colleagues (as reviewed in the current issue) have shown how early training of the

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spared forelimb following induced motor cortex damage negatively affects later function of the affected forelimb. Related to language impairment and its rehabilitation, it is not clear how these findings by Jones' group can be applied. More generally, it is uncertain how improved understanding of brain plasticity in animal models of motor impairment may relate to aphasia. Nevertheless, it is crucial to emphasize that principles of brain plasticity in animal models probably relate to human recovery of communication abilities. At the very least, improved understanding between training in brain lesioned animals and outcome may fuel research questions that can be applied to recovery from aphasia. For example, is it possible that early treatment that targets relatively spared communication abilities may negatively affect later recovery of less spared language functions? Although the application of animal models to the understanding of brain plasticity associated with aphasia recovery is not so straightforward, neuroimaging studies in humans have so far provided some insight into brain plasticity associated with aphasia treatment (for a brief review see [Crinion & Leff, 2007](#); [Fridriksson, 2010](#)). As importantly, studies have revealed, albeit indirectly, that external brain stimulation can enhance brain plasticity associated with aphasia treatment. In the following paragraphs, I will review recent studies conducted in my lab that have focused on functional brain changes associated with aphasia treatment as well as the use of low current transcranial brain stimulation to enhance aphasia treatment outcome. My aim is to demonstrate how studies of treatment-related brain plasticity (as measured with functional magnetic resonance imaging: fMRI) in persons with aphasia can motivate research that seeks to combine behavioral stimulation with transcranial brain stimulation to enhance treatment outcome and, presumably, brain plasticity.

2. Measuring brain plasticity in aphasia

Several studies have related language impairment to functional brain activity in persons with chronic stroke ([Fridriksson, 2010](#); [Fridriksson, Bonilha, Baker, Moser, & Rorden, 2010](#); [Postman-Caucheteux et al., 2010](#); [Rochon et al., 2010](#); [van Oers et al., 2010](#)). In one such recent study, our group examined if functional brain activation predicts the severity of anomia, an impairment in object naming that is commonly seen in aphasia ([Fridriksson et al., 2010](#)). This study involved 15 stroke survivors with chronic aphasia who underwent fMRI scanning while completing an overt picture naming task. Naming attempts were recorded with a non-ferrous microphone and stored electronically for later scoring by a certified speech-language pathologist. Then, brain activation associated with correct naming was analyzed for each participant and contrasted with naming-related activation among a group of 10 control participants. To appreciate whether particularly high or low cortical activation, as compared to the normal controls, was related to naming ability, each participants' activation map was utilized as a predictor of correct naming in a group analysis. Overall, this analysis revealed that greater activation of both anterior and posterior regions in the left hemisphere was associated with successful picture naming. Specifically, participants who were able to name more pictures during the fMRI scanning had increased left hemisphere activation. Based on this finding, we suggested that improved long-term outcome of anomia among persons with aphasia is mediated via plastic changes (i.e., functional activation changes) in the left hemisphere.

In a related study, we examined functional brain changes associated with behavioral treatment of anomia in 26 persons with chronic aphasia caused by stroke ([Fridriksson, 2010](#)). The specific purpose of this study was to understand where functional brain changes that support treatment-assisted anomia recovery occur in the brain. As a secondary goal, this research also associated structural brain damage with treatment outcome to appreciate whether damage to specific brain regions has a particularly negative effect on anomia treatment. Each participant underwent three hours of anomia treatment per day, five days a week, for two weeks. The anomia treatment protocol consisted of a cueing hierarchy where verbal cues of increasing cueing strength were administered to elicit correct naming of pictures depicting common objects. Participants underwent two fMRI sessions before and two fMRI sessions after the treatment period. As in the study discussed above, participants attempted to name pictures of common objects during the fMRI scanning. To understand treatment-related changes in functional activation, naming-related activity was compared between the first two and last two fMRI sessions for all participants. Then, the change in functional activation was utilized as a predictor of naming improvement (qualified as increase in correct naming). In short, the results revealed a strong association between anomia treatment success and increased cortical activation in the left hemisphere. That is, participants who fared well in treatment also experienced a significant increase in left hemisphere activation suggesting that recovery from anomia in chronic stroke is mediated by the left hemisphere. In addition to examining treatment-related changes in cortical activation, this study used voxel-wise lesion symptom mapping (VLSM) to examine the location and extent of structural brain damage as predictors of anomia treatment outcome. The results revealed that damage to the left posterior middle and inferior temporal lobes is especially detrimental for anomia treatment success. With regard to the number of participants with aphasia and extent of aphasia treatment, this group study is the largest of its kind in which changes in functional brain activation have been related to treatment success, providing strong evidence that treatment-assisted recovery from anomia among patients with chronic left hemisphere stroke is related to increased left hemisphere activation. Moreover, it demonstrates how damage to particular brain regions can not only cause the initial behavioral impairment but also affect the success of treatment targeting the same specific impairment.

3. Inducing brain plasticity in aphasia

Taken together, the two studies reviewed above suggest that long-term anomia status as well as its treatment rely on modulation of the left hemisphere. Based on these findings, we hypothesized that modulation of the left hemisphere in

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