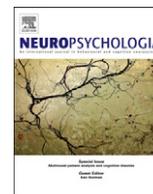




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Therapy-induced neuroplasticity in chronic aphasia

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ABSTRACT

Research on the neural substrate of aphasia recovery has consistently increased since the advent of functional neuroimaging. The evidence from therapy-induced aphasia recovery studies shows that better recovery results from the reactivation of left hemisphere function; still, the specific left hemisphere key areas that sign successful outcome with a specific therapy approach remain to be identified. Nine participants suffering from aphasia received brief and intensive therapy with Semantic Feature Analysis (SFA). Behavioural and neuroimaging data during overt picture naming were obtained prior to and after therapy. This paper reports on a group of participants having benefited from SFA, and two distinct patterns of improvement.

Correlational analysis showed that differences in outcome were not related to lesion size, but were negatively correlated with damage to Broca's area (BA45). Moreover, a group analysis showed that therapy-induced recovery following SFA was characterized by (a) a significant correlation between improvement and activation in the left precentral gyrus (BA4/6) before therapy, and (b) the recruitment of the left inferior parietal lobule, an area known for its role in semantic integration, following therapy with SFA. Individual fMRI analyses showed that although adaptive brain plasticity appeared to operate differently in each patient, best responders to SFA therapy recruited less areas after training compared to participants having shown less recovery who showed a larger number of activated areas sustaining recovery. The results of the present study suggest that a significant activation of BA4/6 could indicate the use of SFA to achieve successful outcome. Also our results suggest that greater SFA improvement in chronic aphasia is associated with recruitment of areas in the left hemisphere.

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1. Introduction

Despite the availability of a wide range of neuroimaging techniques, it is still difficult to predict the outcome of aphasia following a stroke (Pedersen, Vinter, & Olsen, 2004). Many patients will recover rapidly after the lesion in the phases known as acute and sub-acute. However, language recovery is minimal for others

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during the same time period, and a relatively severe aphasia may persist one year after brain insult. According to Wade, Hower, David, and Enderby (1986), one in eight stroke survivors will present with persistent chronic aphasia. In addition, among the patients who initially suffered from aphasia, two in five will still present with aphasia one year after stroke; this represents so-called chronic aphasia (Pedersen et al., 2004). In the past, intensive language therapy was considered to be efficient only with acute and sub-acute patients. However, several recent studies have shown that intensive language therapy is associated with language improvements even in chronic aphasia (Cornelissen et al., 2003; Fridriksson, Morrow-Odom, Moser, Fridriksson, & Baylis, 2006; Fridriksson et al., 2007; Meinzer, Streiftau, & Rockstroh, 2007; Peck et al., 2004; Vitali et al., 2007).

The potential of aphasia therapy to trigger recovery is limited by a number of factors, such as the initial severity (Pedersen, Jorgensen, Nakayama, Raaschou, & Olsen, 1995) and the size and location of the lesion (Basso, 1992). Of these two factors, the role of

lesion size has been examined more extensively. Specifically, a significant relationship between lesion size and aphasia severity has been reported when both cortical and subcortical areas were damaged (Demeurisse & Capon, 1987). However, when the lesion was restricted to cortical areas, aphasia outcome could not be predicted (Demeurisse & Capon, 1987; Lazar, Speizer, Festa, Krakauer, & Marshall, 2008; Naeser, Helm-Estabrooks, Haas, Auerbach, & Srinivasan, 1987). Thus, smaller lesions that include core language areas have shown to induce more severe language impairments than larger lesions elsewhere and are better predictors of aphasia recovery (Naeser & Palumbo, 1994). For example, Naeser et al. (1987) reported that lesions in Wernicke's area and beyond the middle temporal gyrus (Dronkers, Wilkins, Van Valin, Redfern, & Jaeger, 2004) produced more severe and more persistent comprehension impairments than more widespread brain damage in other areas. Greater improvements after therapy were associated with smaller volume loss in the hippocampus, whereas no correlations were detected between therapy-related improvement and lesion size (Meinzer et al., 2010). Parkinson, Raymer, Chang, Fitzgerald, and Crosson (2009) investigated the relationship between lesion size and naming abilities and reported that larger lesions in anterior areas were associated with better naming abilities and greater improvement after therapy, which supported previous findings (Vitali et al., 2007; Zahn et al., 2004). This suggests that the brain mechanisms underlying aphasia recovery are complex and are determined by multiple factors (Lazar et al., 2008) that have not yet been fully explored.

Recently, the advent of functional neuroimaging techniques has greatly contributed to our understanding of therapy-induced brain plasticity in aphasia. Neuroplasticity is a concept that refers to the brain's potential to modify neural circuits. This potential is reflected not only during development (developmental neuroplasticity) but also throughout the lifespan and following brain damage. In the latter case, two types of plasticity have been described: functional reactivation and functional reorganization. Functional reactivation implies a functional recovery of perilesional classical language areas in the left hemisphere (LH) (Cappa, 2000; Heiss, Thiel, Kessler, & Herholz, 2003), and has been associated with better outcomes (Heiss & Thiel, 2006; Rosen et al., 2000; Saur et al., 2006). Functional reorganization consists in the activation of non-classical language areas, either perilesional areas in the LH (Karbe, Kessler, Herholz, Fink, & Heiss, 1995; Léger et al., 2002; Warburton, Price, Swinburn, & Wise, 1999) or right hemisphere (RH) areas homologous to the damaged areas, which can be recruited when there is permanent damage to the LH language processing areas (Crinion & Price, 2005; Musso et al., 1999; Pizzamiglio, Galati, & Committeri, 2001; Pulvermüller et al., 2001; Pulvermüller, Hauk, Zohsel, Neininger, & Mohr, 2005; Rosen et al., 2000; Sharp, Scott, & Wise, 2004; Weiller et al., 1995). Thus, some research groups associate recovery with neuroplastic changes in the LH (Cornelissen et al., 2003; Fridriksson, Baker, & Moser, 2009; Meinzer et al., 2008), whereas others report recovery following functional reorganization in the RH (Crosson et al., 2005; Peck et al., 2004), and still others argue that therapy-induced recovery from aphasia relies upon bilateral neuroplasticity (Fridriksson et al., 2006, 2007).

In view of this disparity, there has been growing interest in the role of both the LH and RH in the recovery from aphasia. A recent review of the literature shows a lack of convergent results of this research, highlighting the roles of both cerebral hemispheres in aphasia recovery (Meinzer, Harnish, Conway, & Crosson, 2011). Although some studies associate language improvement with RH activations (Thulborn, Carpenter, & Just, 1999; Winhuisen et al., 2005), the role of the RH in aphasia recovery remains controversial. For instance, Belin et al. (1996) suggested that the recruitment of the RH was maladaptive and could harm language recovery in the

LH, which could lead to the persistence of linguistic deficits. In line with this perspective, Naeser et al. (2005) used repetitive transcranial magnetic stimulation (rTMS) to inhibit the right pars triangularis. The authors reported improved oral naming in a severe non-fluent global aphasic patient and argued that their findings provided evidence of a maladaptive functional reorganization involving the RH. In another study, Winhuisen et al. (2005) used rTMS on the inferior frontal gyrus (IFG) bilaterally in subacute aphasic patients. As opposed to patients who experienced interference when rTMS was applied to both the left and right IFG, participants for whom the stimulation only triggered interference when applied to the left IFG performed significantly better 10 days after suffering from their stroke. However, in their follow-up study, Winhuisen et al. (2007) reported equivalent improvement in two participants who showed significant bilateral interference of the IFG eight weeks after their stroke. These results suggest that RH activation might not be a negative factor for prognosis and that the RH's potential for adaptive neuroplasticity needs further exploration.

If the role of the RH is still controversial, it is relatively widely acknowledged that the recruitment of the LH seems to be associated with better recovery from aphasia. In this regard, a recent study by van Oers et al. (2010) reported a positive correlation between the activation of the left IFG and improved picture and sentence naming. Accordingly, robust correlations between post-therapy aphasia recovery and significant activations in the LH language areas have been reported consistently (Breier et al., 2004; Cao, Vikingstad, George, Johnson, & Welch, 1999; Crinion & Price, 2005; Heiss & Thiel, 2006; Meinzer et al., 2008), suggesting that better language recovery is associated with LH activations. In line with this view, Baker, Rorden, and Fridriksson (2010) applied transcranial direct-current stimulation on left frontal areas and showed better naming improvements when this was combined with computerized language therapy.

In summary, research on therapy-induced neuroplasticity in aphasia recovery has increased considerably in the last years. Most studies have adopted a multiple single-case perspective; more recently, a few group studies have been published (Fridriksson, 2010; Meinzer et al., 2008; van Oers et al., 2010). On the one hand, multiple single-case studies have the advantage of accounting for individual differences, while on the other hand, research with individuals who share common symptoms (e.g., severe anomia), or group studies, has the advantage of allowing for generalization and may demonstrate the efficacy of the therapy (Basso, 2003). Therefore, in the present study, we used a combination of multiple single-case and group studies to investigate the recovery from chronic aphasia and to better understand both individual differences and the common areas recruited by a specific therapy.

Thus, the purpose of the present study was to identify the neuroplastic changes associated with therapy-induced recovery from chronic aphasia following Semantic Feature Analysis (SFA) therapy (Boyle & Coelho, 1995; Ylvisaker & Szekeres, 1985). Nine participants with moderate to severe chronic aphasia were evaluated during two pre-/post-therapy event-related functional magnetic resonance imaging (fMRI) sessions. In addition, activation maps associated with oral naming performance were obtained before and after three weekly sessions of therapy. SFA sessions lasted for a maximum of six weeks, or until participants were able to name 80% of the trained items in two successive therapy sessions. Given the extended lesions and the severity of aphasia, we hypothesized that (1) successful naming improvement following SFA would be associated with the significant activation of a bilateral network, and (2) the greatest improvements in naming would be associated with a significant reactivation of perilesional areas in the LH.

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