ACUTE AND CHRONIC APHASIA: IMPLICATIONS FOR NEUROPLASTICITY
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Although functions of brain areas are in principle localizable, the localizations of functions are not fixed, which implies that the adult human brain retains the potential for plasticity. With regard to recovery of function, the neural circuitry supporting language functions responds differently to different types of lesions. As an example, cases of low-grade gliomas affecting Broca’s area (Brodmann areas 44 and 45) have been reported in which language function was later supported by areas that are typically not implicated in language, such as BAs 46 and 47. On the other hand, the literature on recovery of language following stroke contains controversies with regard to the issue of reorganization of language substrates in aphasia; there are reports of involvements of both perilesional and contralesional areas. The exact roles of these areas in language recovery after stroke are not clear at the moment, and in particular, the role of the right hemisphere in recovery has been hotly debated for years.

Furthermore, there is evidence indicating that perilesional areas are the substrate of language reorganization following smaller lesions (e.g., see Cao et al., 1999), while larger lesions typically involve contralateral homologous areas. In line with this evidence, some research has reported that language recovers better when recovery occurs within the original network than when it occurs in the contralateral hemisphere (e.g., Karbe et al., 1998). Similarly, research contrasting acute and slow-growing lesions has shown that functional compensation is considerably better following low-grade gliomas than acute stroke (Desmurget et al., 2007). If the hypothesis that brain plasticity is mediated by a gradual learning process, with plastic changes taking place via “supervised learning”, is correct, then the findings on improved aphasic recovery supported by perilesional areas makes sense. Similarly, this hypothesis would explain why acute destruction, which prevents gradual learning, leads to poor recovery (Desmurget, 2007). More importantly, the hypothesis could also explain the data on “shifting” of language function from the right hemisphere to the left hemisphere once learning, i.e. recovery, is relatively complete.

The differences in mechanisms of language recovery after stroke in acute and chronic aphasias have not yet been thoroughly investigated, despite recent methodological advances that allow such research, including the availability of functional neuroimaging and external brain stimulation techniques. Until recently, patterns of language recovery after stroke were typically studied in chronic aphasia (Cramer & Riley, 2008). However, cases of acute aphasia are more informative about language recovery processes. Additionally, studying acute aphasia is important because early intervention is essential. Unlike structural imaging methods (e.g., computerized tomography (CT) and magnetic resonance imagining (MRI)), which lack the potential to capture plastic changes in the brain, functional neuroimaging techniques such as positron emission tomography (PET) and functional magnetic resonance imaging (fMRI) yield insights on preservation of function, thereby contributing to our understanding of the organizational flexibility of the human brain. More importantly, functional neuroimaging allows us to study temporal patterns in recovery of language function in aphasia, which may help to resolve the current controversy on the role of contralesional areas in recovery. One step towards a better understanding of the mechanisms of language recovery after stroke, including the patterns of reorganization and the roles of the perilesional and contralesional areas in these patterns, would be to collect data at different post-stroke temporal points, such as acute, subacute, and chronic.

Recently, Saur and colleagues (2006) have shown that the patterns of reorganization differ at distinct phases of post-stroke recovery of language. More specifically, they have proposed that the acute phase is associated with little perilesional activation; the subacute phase is associated with activation of homologous areas in the right hemisphere, while in the chronic phase, a re-shifting to the language...
areas of the left hemisphere is associated with further language improvements. According to this model, activation of the right hemispheric areas in a chronic aphasic patient indicates a poor recovery.

While Saur et al.'s model nicely explains their data, it requires further testing. It also raises some interesting questions. For example, do changes in activation patterns amongst the three distinct post-stroke temporal points indeed arise from plasticity (as opposed to, for example, arising from resolution of diaschisis (Hillis, 2006), and why does progress in language recovery after stroke seems to require changes in lateralization? How are the mechanisms that support language in aphasic patients who recover well different from the mechanisms in patients with similar lesions who fail to recover?

The answers to questions like these are relevant not only for gaining further insights into the functional architecture of language and into neuroplasticity in general, but also for treatment of aphasia. Given the high incidence of stroke in many countries, its steady increase in causing disability, and a general need for improvement of aphasia treatments, these answers are important.

REFERENCES: