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# The Neurobiology of Language Recovery in Aphasia

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Most individuals with aphasia show recovery of language function despite persisting damage to left hemisphere language zones (Holland et al., 1996). Such recovery is a complex process that is dependent on neurophysiological processes, environmental factors, and other variables. Soon after damage to the neural networks that subserve language, reorganizational processes begin. In addition, most individuals with aphasia receive treatment to facilitate maximal language recovery. Converging evidence from neural imaging studies, clinical studies of aphasic patients, and other data suggest that the primary candidates for recovery (in right-handed, left-hemisphere language dominant patients) include undamaged portions of the language network in the left hemisphere and/or homologous right hemisphere areas (Demeurisse & Capon, 1987; Heiss et al., 1999; Weiller et al., 1995). The issues addressed here concern the effects of treatment on patterns of language reorganization. Does treatment influence the course and extent of neural reorganization? Is it possible that certain treatments could enhance neural reorganization? Could particular treatments prevent or impede recovery of certain processes? It is suggested that research addressing these questions could not only improve our understanding of the neurobiology of language and recovery, but also impact treatment of aphasia.

What influences the extent to which undamaged portions of the left hemisphere or areas in the right hemisphere are recruited for processing language once the system is damaged? More specifically, does the treatment provided influence reorganization of the language system or does it reorganize in a biologically predisposed manner, considering site and extent of lesion and other variables? Given the results of animal studies as well as recovery studies of aphasia, it is highly likely that treatment plays a strong role. Indeed, animal studies have shown that motor learning, tactile stimulation, and auditory stimulation directly influence neural representations of motor, somatosensory, and auditory processing, respectively (Jenkins et al., 1990; Nudo et al., 1996; Recanzone et al., 1993; Van Praag et al., 1999). For example, Greenough et al, (1985) found that in adult rats motor learning results in increases in dendritic aborization and synaptic connections of neurons in the cerebrum and cerebellum. Similarly, Jenkins et al. (1990) reported enlargements of somatosensory areas associated with controlled tactile stimulation in adult owl monkeys. Studies also have shown that rehabilitative training after injury results in enhancement of representational plasticity (Nudo et al., 1996; Xerri et al., 1998). For example, Nudo et al. (1996) trained monkeys to retrieve pellets from small wells (an activity that requires skilled digital use), lesioned the motor cortex, and retrained the motor task. Motor maps derived before and after training showed substantial rearrangement of representations surrounding the lesion. Notably, areas of cortical digital representation were expanded, while wrist and forearm representations were contracted. These findings indicate that experience directly

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shapes physiological reorganization. Thus, it is likely that treatment for aphasia, which is known to improve language processing abilities, influences reorganizational processes.

Does the domain of language or the type of treatment provided influence recovery patterns? For example, does treatment focused on sentence production result in reorganizational processes that differ (at least in some respects) to that that results from treatment focused on naming or word retrieval? Does treatment for sentence production focused on lexical and syntactic properties known to influence normal language processing result in different neurophysiological outcomes than treatment aimed at teaching patients to produce sentences and phrases for communicating in certain functional contexts? Indeed, it is arguable that treatment focused on a particular language domain would result in recruitment of different aspects of the language network. The treatment approach also might result in markedly different outcomes. Thompson and colleagues have shown, for example, that linguistic specific treatment of sentence production deficits in agrammatic aphasia results in generalization to untrained sentences that are linguistically similar to those trained, indicating improved access to the structures and computations required to produce sentences (Ballard & Thompson, 1999; Thompson & Shapiro, 1995; Thompson et al., 1998). Conversely, treatment aimed at teaching certain functional behavior results in increased use of trained responses, but little generalization to untrained responses (Thompson 1989). Because of the relation between behavioral change and brain reorganization that has been noted in the animal literature, it is likely that behavioral outcomes associated with each treatment are reflected in differential reorganization of the language network.

Further, it could be argued that treatment aimed at stimulating access to properties of language that once were fully accessible to the individual with aphasia might enhance neural reorganizational processes; whereas, teaching compensatory strategies which likely by-pass the language network may not result in maximal recovery (and concomitant rebuilding of the underlying neural networks). Providing compensatory treatment, e.g., teaching patients to access limited functional responses, could result in stimulation only of those pathways required to access those responses, but would do little to stimulate reorganization of the language system. Therefore, it appears conceivable that providing treatments that potentially by-pass language processing routines used prior to brain damage may be contraindicated in some cases. On the other hand, widening the scope of treatment by teaching underlying forms rather than surface forms might facilitate rebuilding of the language system. This is not to suggest that compensatory treatments should not be considered as a treatment option for some patients, However, if this postulate turns out to be true, it would move us a long way toward the goal of providing optimal treatment for individuals with aphasia.

Without question research is needed to investigate the questions raised here. Studies are needed that precisely measure changes in neural networks involved in language processing during the course of recovery. Further, specific changes in neural networks associated with certain behavioral changes induced by treatment need to be mapped onto the brain. Finally, research aimed at determining the relative effects of providing different types of treatment for certain language deficits on subsequent reorganization is needed. As advances are made in functional imaging technology and other brain mapping techniques we may begin to answer the questions posed here. The results of such research would not only increase our understanding of the neurobiology of language and recovery, but also would move us closer to understanding the full effects of treatment for aphasia.

## References

Ballard KJ, Thompson CK. Generalization of complex sentence structures in agrammatism. Journal of Speech, Language, and Hearing Research. 1999; 42:690–707.

Demeurisse G, Capon A. Language recovery in aphasic stroke patients: Clinical, CT and CBF studies. Aphasiology. 1987; 1:301–315.

Greenough WT, Larson JR, Withers GS. Effects of unilateral and bilateral training in a reaching task on dendritic branching of neurons in the rat motor sensory forelimb cortex. Behavioral Neural Biology. 1985; 44:301–314.

Heiss WD, Kessler J, Thiel A, Ghaemi M, Karbe H. Differential capacity of left and right hemispheric areas for compensation of poststroke aphasia. Annals of Neurology. 1999; 45:430–438. [PubMed: 10211466]

Holland AL, Fromm V, DeRuyter F. Treatment efficacy for aphasia. Journal of Speech and Hearing Research. 1996

Jenkins WM, Merzenich MM, Ochs MT, Allard T, Guic-Robles E. Functional reorganization of primary somatosensory cortex in adult owl monkeys after behaviorally controlled tactile stimulation. Journal of Neurophysiology. 1990; 63:82–104. [PubMed: 2299388]

Nudo RJ, Wise BM, SiFuentes F, Milliken GW. Neural substrates for the effects of rehabilitation training on motor recovery after ischemic infarct. Science. 1996; 272(5269):1791–1794. [PubMed: 8650578]

Recanzone GH, Schreiner CE, Merzenich MM. Plasticity in the frequency representation of the primary auditory cortex following discrimination training in adult owl monkeys. Journal of Neuroscience. 1993; 13:87–103. [PubMed: 8423485]

Thompson, CK. Generalization research in aphasia: A review of the literature. In: Prescott, TE., editor. Clinical aphasiology. Vol. 18. Boston: College-Hill Publication, Little, Brown and Company; 1989. p. 195-222.

Thompson CK, Shapiro LP. Training sentence production in agrammatism: Implications for normal and disordered language. Brain and Language. 1995; 50:201–224. [PubMed: 7583187]

Thompson CK, Shapiro LP, Ballard K, Jacobs B, Schneider S, Tail ME. Training and generalized production of wh- and NP-movement structures in agrammatic speakers. Journal of Speech, Language and Hearing Research. 1997; 40:228–244.

Van Praag H, Kempermann G, Gage FH. Running increases cell proliferation and neurogenesis in the adult mouse dentate gyms. Nature Neuroscience. 1999; 2(3):266–270.

Weiller C, Isensee C, Rijntjes R, et al. Recovery from Wernicke's aphasia: A positron emission tomographic study. Annals of Neurology. 1995; 37:723–732. [PubMed: 7778845]

Xerri C, Merzenich MM, Peterson BE, Jenkins W. Plasticity of primary somatosensory cortex paralleling sensorimotor skill recovery from stroke in adult monkeys. Journal of Neurophysiology. 1998; 79:2119–2148. [PubMed: 9535973]