Aphasia

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Aphasia, a significant loss or impairment of language functions, typically as a result from an injury to an extended (commonly left-hemispheric) network of cortical and subcortical structures, which are largely served by the middle cerebral artery. Aphasia can follow from different types of brain injuries, such as traumatic, stroke, brain tumor or infections. Aphasia affects 21–38% of acute stroke patients [1-3]. It is associated with significant short- and long-term morbidity, substantial cost of care, and a significant burden to affected individuals and their caregivers [4].

Most commonly aphasia results from an injury to the Broca- or Wernicke-Area, and depending on the cortical localization of the injury to the brain, different forms of Aphasia can develop (Fig.1):

1. **Wernicke Aphasia** (WA), also called fluid, semantic, or receptive aphasia is characterised by severely impaired auditory semantic comprehension and repetition, with fluent speech punctuated by phonological paraphasias and neologisms [5]. Comprehension impairment in WA is apparent at both, the word and sentence level [6]. Traditionally WA's comprehension impairment has been termed the "decoding deficit", with an underlying deficit in acoustic-phonologic decoding. Patients demonstrate an impaired auditory decoding of phonemes whilst auditory analysis of prosody remains intact, thus leading to an apparent dissociation between comprehension in formal testing conditions and contextual comprehension [3]. A decoding deficit is consistent with lesions in the posterior part of the left superior temporal plane, a region associated with phonological processing [7].

2. Broca Aphasia, or expressive aphasia (non-fluent aphasia) is characterized by the loss off or the reduced ability to produce language (spoken or written). Individuals with Broca's aphasia have damage to the frontal lobe of the brain.

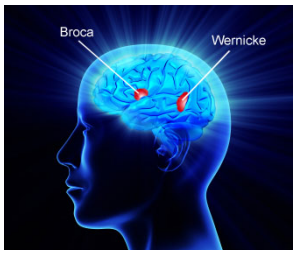

Figure 1: Illustration of the main language areas located in the Broca-, and Wernicke-Area within the left hemisphere

3. Global Aphasia, results from damage to extensive portions of the language areas of the brain. Individuals with global aphasia have severe communication difficulties and may be extremely limited in their ability to speak or comprehend language.

Therapy: The most common treatment of Aphasia to date is speech therapy. However, within current experimental approaches, recovery from symptoms like Aphasia after stroke is believed to depend on three mechanisms: [i] recruitment of lesioned and perilesional left hemispheric regions, [ii] acquisition, unmasking or refinement of language processing ability in contralesional, homologue areas of the right hemisphere, and on the other hand [iii] dysfunctional activation of the contralesional hemisphere that may interfere with language recovery [8]. Accordingly, the role of the right hemisphere in aphasia recovery is controversial; it has been characterised as both a facilitator as well as a suppressor of rehabilitation. It has been shown that increased right hemispheric recruitment is associated with improved language skills [9] whereas loss of inter-hemispheric inhibitory influence from the lesion-affected language areas in the left hemisphere may lead to an excessive and unwanted inhibitory control by the right over the lesioned hemisphere. In this context, transcranial direct current stimulation (tDCS) and Transcranial Magnetic Stimulation (TMS) has been employed to reduce excessive right hemispheric activation thereby improving language performance in post-stroke aphasia patients [10, 11] even after discontinuation of the intervention [12]. These changes have been ascribed to brain stimulation induced changes in cortical excitability [13].

1. Wade, D.T., R.L. Hewer, R.M. David, and P.M. Enderby, Aphasia after stroke: natural history and associated deficits. *J Neurol Neurosurg Psychiatry*, 1986. **49**(1): p. 11-6.
2. Weiskopf, N., C. Hutton, O. Josephs, and R. Deichmann, Optimal EPI parameters for reduction of susceptibility-induced BOLD sensitivity losses: a whole-brain analysis at 3 T and 1.5 T. *Neuroimage*, 2006. **33**(2): p. 493-504.
3. Luria, A.R., *Disturbances of Understanding of Verbal Communication in patients with sensory aphasia*, M.C. B.V., Editor 1976: The Hague.
4. Laska, A.C., A. Hellblom, V. Murray, T. Kahan, and M. Von Arbin, Aphasia in acute stroke and relation to outcome. *J Intern Med*, 2001. **249**(5): p. 413-22.
5. Thompson, C.K., S. Lukic, M.C. King, M.M. Mesulam, and S. Weintraub, Verb and noun deficits in stroke-induced and primary progressive aphasia: The Northwestern Naming Battery(). *Aphasiology*, 2012. **26**(5): p. 632-655.
6. Goodglass, H., E. Kaplan, and B. Barresi, *The Assessment of Aphasia and related disorders (3rd ed.)*2001, Baltimore: Lippincott Williams &Wilkins.
7. Vigneau, M., V. Beaucousin, P.Y. Herve, H. Duffau, F. Crivello, O. Houde, B. Mazoyer, and N. Tzourio-Mazoyer, Meta-analyzing left hemisphere language areas: phonology, semantics, and sentence processing. *Neuroimage*, 2006. **30**(4): p. 1414-32.
8. Hamilton, R.H., E.G. Chrysikou, and B. Coslett, Mechanisms of aphasia recovery after stroke and the role of noninvasive brain stimulation. *Brain Lang*, 2011. **118**(1-2): p. 40-50.
9. Ohyama, M., M. Senda, S. Kitamura, K. Ishii, M. Mishina, and A. Terashi, Role of the nondominant hemisphere and undamaged area during word repetition in poststroke aphasics. A PET activation study. *Stroke*, 1996. **27**(5): p. 897-903.
10. Naeser, M.A., P.I. Martin, M. Nicholas, E.H. Baker, H. Seekins, M. Kobayashi, H. Theoret, F. Fregni, J. Maria-Tormos, J. Kurland, K.W. Doron, and A. Pascual-Leone, Improved picture naming in chronic aphasia after TMS to part of right Broca's area: an open-protocol study. *Brain Lang*, 2005. **93**(1): p. 95-105.
11. Knechtel, L., R. Thienel, and U. Schall, Transcranial direct current stimulation: Neurophysiology and clinical applications. *Neuropsychiatry*, 2013. **3 (1)**: p. 89-96.
12. Barwood, C.H., B.E. Murdoch, B.M. Whelan, D. Lloyd, S. Riek, J.D. O'Sullivan, A. Coulthard, and A. Wong, Improved receptive and expressive language abilities in nonfluent aphasic stroke patients after application of rTMS: an open protocol case series. *Brain Stimul*, 2012. **5**(3): p. 274-86.
13. Stagg, C.J., M. Wylezinska, P.M. Matthews, H. Johansen-Berg, P. Jezzard, J.C. Rothwell, and S. Bestmann, Neurochemical effects of theta burst stimulation as assessed by magnetic resonance spectroscopy. *J Neurophysiol*, 2009. **101**(6): p. 2872-7.