Recovering from acquired childhood aphasia (ACA) – 20 years later, learning about the neuroplasticity of language

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1. Introduction

Language is at the core of human cognitive abilities and the mechanisms associated with neural recovery from aphasia are still largely unknown. Important insights have been reported, such as: i) Significantly better recovery occurs, if the brain lesions leading to aphasia were suffered during childhood (ACA); ii) that *perilesional map expansion* and *transference of language functions to homologue areas in the contralateral hemisphere* are thought to be two main processes sustaining this neuroplasticity [1]. From the study of peri-natal brain lesions of left hemisphere is known that they are often associated with language transfer to the right hemisphere [2–4].

We investigated if potential factors underlying the neuroplasticity of language (such as the developmental linguistic stage at onset of aphasia and size of the lesion) would significantly contribute to determine the recovery strategy. We report the a follow-up study of three cases of left frontal subcortical ischemic strokes about 20 years after onset of aphasia.

2. Method

2.1. Participants

Three young adults (1 female DF, 2 male JAF + JPF) that suffered from a aphasia, due to a left frontal sub-

cortical lesion (age at onset JAF 2,1; DF 2,7; JPF 13,5 years respectively), performed an extensive neurolinguistic testing (age at testing 31,5; 33,4; 26,3 years respectively) applying 20 different neurolinguistic tests.

The activation pattern of the 3 patients were compared to 4 (3 female, 1 male) healthy controls (mean age of 26,3 years and 15 years of formal education).

2.2. MRI

MRI data acquisition was performed on 3T Philips Achieva Scanner. Two fMRI language paradigms, semantic decision paradigm with both visual and auditory presentation were applied. Visual semantic decision paradigm, written in block design; image acquisition was performed in a continuous manner (TR 2000 ms, 160 volumes). Auditory semantic decision paradigm, written in event design; image acquisition was performed in sparse design, in order to present the auditory stimulus without interference of the scanner noise (TR 8000 ms, 120 volumes). Functional data was processed using the BrainVoyager software. Single and group General Linear Models (GLM) were applied to the data. For the visual paradigm activation clusters were corrected for multiple comparisons after the Bonferroni method to an p-level of 0,05. The threshold for the auditory paradigm was chosen less strictly (auditory stimuli $p < 0,000059$, without correction for multiple comparisons), due to the weaker signal of the data of the event related paradigm. For anatomical localization of activation clusters peak-voxel cluster were defined

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Table 1
SEMANTIC DECISION visual stimuli p (Bonf) < 0.05

	Control	DF	JPF	JAF
cluster 1	LEFT BA4	LEFT BA8	LEFT BA3	RIGHT BA 22
cluster 2	LEFT BA6	LEFT BA6	LEFT BA40	RIGHT BA 46
cluster 3	LEFT BA9	LEFT BA6	LEFT BA45	RIGHT BA 6
cluster4	LEFT BA22	LEFT BA9	LEFT BA21	RIGHT BA10
cluster 5	LEFT BA45	LEFT BA39	LEFT BA21	RIGHT BA 6
cluster 6		LEFT BA21	LEFT BA 40	LEFT BA 9
cluster 7		LEFT BA3		LEFT CEREBELLUM
cluster 8		LEFT BA21		LEFT BA 22
SEMANTIC DECISION auditory stimuli $p < 0,000059$				
	Control	DF	JPF	JAF
cluster 1	LEFT BA21	LEFT BA13	LEFT BA39	RIGHT BA32
cluster 2	LEFT BA22	LEFT BA 22	LEFT BA40	RIGHT BA9
cluster 3	LEFT BA 41		LEFT BA22	RIGHT BA24
cluster 4			LEFT BA43	LEFT BA24
cluster 5				LEFT BA32
cluster 6				LEFT BA20
cluster 7				LEFT BA20
cluster 8				LEFT BA39
cluster 9				LEFT BA 22
cluster 10				LEFT BA42
cluster 11				LEFT BA21

with a cluster threshold of 300 voxels for the visual condition and 150 voxels for the auditory condition. All participants performed a T1-weighted anatomical scan. All anatomical data were transformed into the common space of Talairach.

3. Results

In all 3 patients structural MRI showed the sequelae of a left frontal subcortical infarction in a similar localization but of different size. All three patients showed fully functional recovery of language; however JAF performed below one SD in the semantic (food and animals) and the phonological (letters M,P,R) verbal fluency task. JAF, but also JPF, admitted, as the only symptom, some word finding difficulties in the self evaluation form. In Table 1 the respective Brodmann areas of the peak-voxel VOIs are listed. The main pattern of recovery in patients DF and JPF was reorganization of language function in the dominant left hemisphere. The exception is patient JAF who had a partial language transfer. His neuronal network for language is much larger distributed (e.g. involvement of the left cerebellum). The homologue language areas (BA22 and BA46) of the right hemisphere are more activated by the visual stimuli of the language paradigm than their left counterparts, whereas heard language stimuli continue to be processed predominantly in the left hemisphere.

4. Conclusion

The results contrasts to what is known from perinatal brain lesions before language acquisition that are often associated with a complete language shift to the right hemisphere [4]. This suggests that an early commitment of the left hemisphere to language initiates an almost irreversible specialization [5–7]. Only when the left hemisphere is compromised by a sufficient large lesion language functions that are acquired later on tend to be transferred to the homologue areas of the contra-lateral hemisphere. In JAFs case the processing of written stimuli (acquired 4 years after lesion onset) was directed to the right hemisphere. Further research should investigate the underlying factors in the exceptional cases of transference of language function to the opposite brain hemisphere.

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