

Is the emergence of speech errors in chronic post-stroke aphasia a result of ongoing compensatory brain plasticity mechanisms?

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BACKGROUND AND OBJECTIVE

Traditional descriptions of aphasia have ascribed language disturbances to tissue damage, mostly in the left perisylvian cortex (Damasio and Damasio, 1992). This seems the simplest explanation for the impairments that imply impoverishment of the previous language abilities, for instance reduced auditory comprehension or anomia. However, symptoms expressed as repetitive verbal behaviors such as mitigated echolalia (ME) and conduite d'approche (CdA) cannot emanate from fully dysfunctional areas affected by irreversible tissue damage.

CdA is the repetitive and self-initiated approximation to a target word during spontaneous speech or naming tasks (e.g., target word: elephant; Patient response: eli... elipha... eliphan... iliphan... ephe... a... eli... aliphant... ile... elephant!) (Ueno and Lambon-Ralph, 2013). **ME** refers to the echoing of a just heard word/sentence introducing a subtle change (e.g., the examiner asks: are you a doctor?; patient response: Are you a doctor?... I' m not a doctor!)(Berthier, Torres-Prioris, López-Barroso, 2013).

Aim: To explore the functional and structural mechanisms supporting CdA and ME, and their relationship with compensatory plastic changes occurring within the language network.

Hypothesis: ME may emerge from spared dorsal stream when the ventral system is compromised, while CdA may result as an attempt of the ventral stream to compensate dorsal damage

METHODS

Participants: 3 persons with fluent post-stroke aphasia showing CdA (Subject 1), ME (Subject 2) or both symptoms (Subject 3). Subjects were right-handed males and Spanish monolingual. Subject 1: 43 yo; tpo: 29 months; conduction aphasia. Subject 2: 53 yo; tpo: 4 months; transcortical sensory. Subject 3: 51 yo; tpo: 8 months; Wernicle's aphasia.

Language and cognitive assessment: Digit span, Western Aphasia Battery, repetition and naming tasks.

Multimodal neuroimaging: structural magnetic resonance imaging and 18FDG-PET. PET data of the three subject was compared to 25 healthy controls by using utilizando Statistical Parametric Mapping (SPM12). White matter lesion approach: Tractotron and Disconnectome map (BCBtoolkit; www.toolkit.bcblab.com; Foulon et al., 2018).

CONCLUSIONS

- CdA may reflect the compensatory role of the ventral (semantic) language pathway when the dorsal (phonological) pathway is damaged, whereas ME may reflect the compensatory mechanism through the dorsal pathway after damage to the ventral route.
- The coexistence of CdA and ME may occur after incomplete lesions involving both routes
- CdA and ME may be produced in an attempt to overcome phonological and lexico-semantic deficits, respectively.
- CdA and ME can be viewed as a therapeutic opportunity since they represent an active, yet suboptimal, attempt to improve communication.

RESULTS

1. Behavioral results: CdA in Subject 1 was mainly present in repetition and naming tasks, while ME in subject 2 was mainly found in task that requires semantic access such as comprehension and words definition tasks. In subjects 3, instances of CdA and ME were seen in tasks overlapping with the two previous.

2. **PET results:** PET analysis showed significant hypometabolism in perilesional areas of the left hemisphere in the three subjects. Subject 2, in addition, showed a cluster of significant increased metabolic activity in the right hemisphere (Fig. 1).

3. **Tractotron and Disconnectome Map results:** Subject 1 had a disconnection pattern that greatly overlapped with the dorsal language pathway, while Subject 2's lesion location bisected the ventral pathway discontinuing the projection of fibers that run through it. Subject 3 presented a disconnection pattern in-between the two previous ones (Fig. 2).

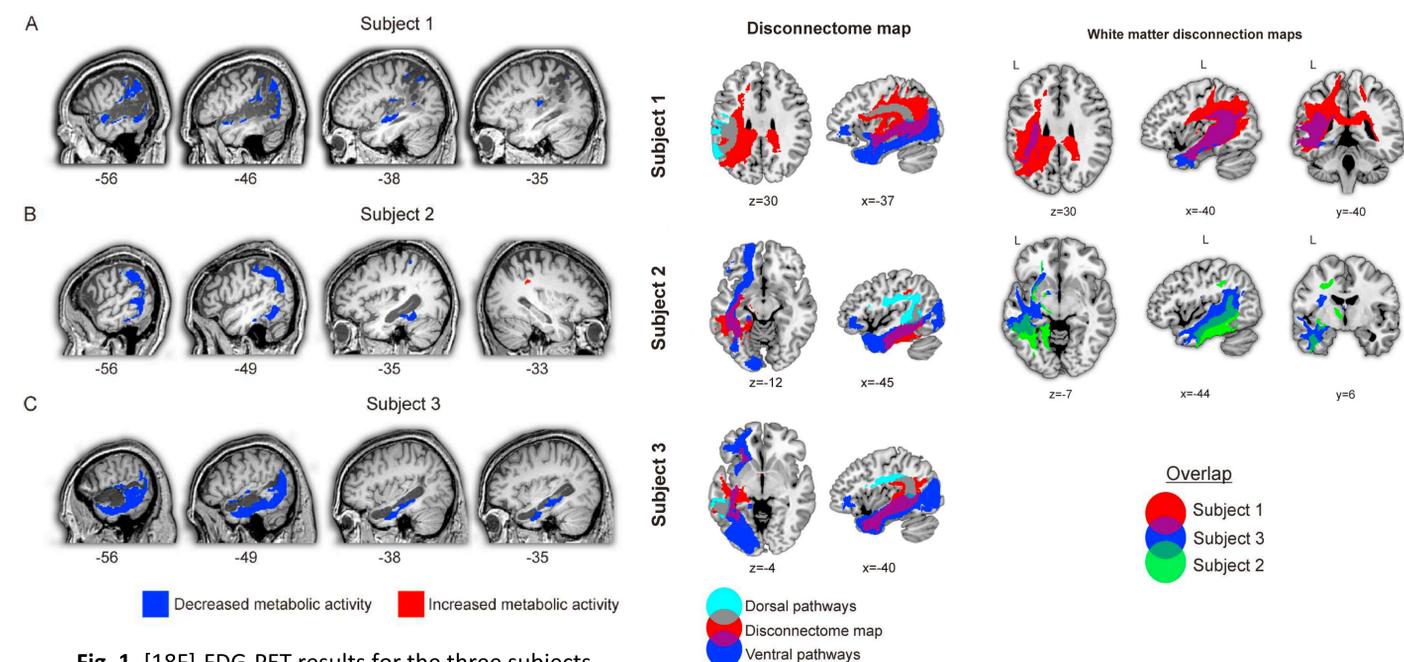


Fig. 1. [18F]-FDG-PET results for the three subjects.

Fig. 2. Lesion-based disconnection analysis Results are shown using a probability of disconnection equal or major than 0.90.

References

Damasio and Damasio. Scientific American. 1997, 267267(3),88-108;Berthier, Torres-Prioris, López-Barroso. Front Hum Neurosci, 2017, 11, 164.;Foulon et al. GigaScience, 2018, 7, 1-17; Ueno and Lambon-Ralph, Front Hum Neurosci, 2013, 7, 422.