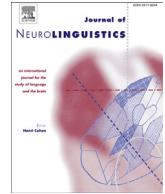




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Functional recovery in subcortical crossed and standard aphasia



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ABSTRACT

There are very few longitudinal studies investigating functional recovery in aphasics affected by subcortical lesions. In addition, no longitudinal follow-up studies exist of patients with subcortical crossed aphasia.

Our aim was to investigate functional recovery in two anomic patients with subcortical aphasia, one crossed aphasia and one standard, uncrossed aphasia due to a similar subcortical lesion. An er-fMRI follow-up paradigm was employed during a picture-naming task. Both patients were scanned prior to speech therapy (T0), after 3 months of anomia-specific rehabilitation (T1) and following 6 months of conventional language rehabilitation (T2). Irrespective of lesion lateralization, fMRI data disclosed a grounding role for homologue naming-specific areas (respectively, LIFG and RIFG) in determining the progressive pattern of behavioural naming recovery throughout different disease phases. Thus, functional recovery, paralleled by improvement in behavioural naming performance, seems to be strictly related to recruitment of homologue areas in the hemisphere opposite to the aphasiogenic lesion.

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1. Introduction

1.1. Crossed aphasia

Crossed aphasia in dextrals (CAD) denotes any aphasic language disorder following a lesion in the right hemisphere of a natural right-hander with a structurally intact left hemisphere and no familiar history of left-handedness, early brain damage or epilepsy (Mariën, Engelborghs, Vignolo, & De Deyn, 2001). This condition in which an aphasic syndrome results from a lesion ipsilateral to the dominant hand (CA) was first reported by Bramwell (1899) in a left-handed stroke patient. However, observations in left-handed aphasia patients during the 1950s (e.g., Goodglass & Quadfasel, 1954; Humphrey & Zangwill, 1952a, 1952b), showed that CA in sinistrals (70–80%) is the rule rather than the exception. By contrast, CA in dextrals (CAD) is an exceptional phenomenon variably estimated to affect .4–2.0% of the aphasic population (Bakar, Kirshner, & Wertz, 1996; Brown & Hecaen, 1976). A number of hypotheses have been advanced to explain this rare condition of atypical cerebral language organization, but the exact pathophysiological mechanisms remain to be elucidated. Brown et al. (Brown & Hecaen, 1976; Brown & Wilson, 1973) suggested that crossed aphasia is the consequence of incomplete lateralization processes of language functions in the left hemisphere during development. More specifically, Annett & Alexander (1996) suggested that crossed aphasia represents a natural variation in functional brain organization due to the presence or absence of one single gene. Finally, crossed aphasia has been attributed to a pathological inhibition of left-hemisphere functioning by the right-hemisphere lesion.

The diagnostic criteria for CAD have been recently reviewed by Mariën, Paghera, De Deyn, and Vignolo (2004). The authors analysed 152 case-reports of right-handed patients with aphasia following a right-hemisphere stroke published since 1975 and proposed a new set of criteria to classify such patients into three different groups: 1) unreliable, 2) possible, and 3) reliable cases. Application of their criteria allowed the authors to individuate 49 reliable CAD patients (32.2%). Importantly, the Mariën et al. (2004) study revealed that CAD following stroke intrinsically differs from standard, uncrossed aphasia in several aspects. First, it is more frequent in men than in women; second, non-fluent cases are not more frequent than patients with fluent speech; third, the relative frequency of the various clinical types is independent of age, i.e. does not show the increase in fluent aphasia with age. Moreover, written language is more severely affected in CAD. An area of controversy is the nature and extent of recovery in CAD. Several authors have reported that the linguistic deficits in this syndrome are transient in nature (Botez & Wertheim, 1959; Zangwill, 1967), whereas others have found the recovery of deficits is comparable to standard aphasia (Hindson, Westmoreland, Carroll, & Bodmer, 1984; Wechsler, 1976; Zangwill, 1979). Differences in lesion size and location may induce, however, different subtypes of CAD (Basso, Capitani, Laiacona, & Zanobio, 1985; Carr, Jacobson, & Boller, 1981; Henderson, 1983). Contrary to earlier views, the results of Mariën et al. (2004) showed that CAD is rarely transient and full language recovery is not the rule.

An interesting aspect of CAD is that it occurs frequently after subcortical stroke (Cappa et al., 1993; Habib, Joannette, Ali-Cherif, & Poncet, 1983; Holmes & Sadoff, 1966; Mariën et al., 2004). In the present study, we investigate the neural correlates of functional recovery in a case of CAD due to a subcortical lesion and compare it to a control patient with a similar subcortical lesion in the dominant hemisphere (uncrossed aphasia).

1.2. Subcortical aphasia

The study of language disturbances after subcortical lesions is in itself a challenge because of the diversity of language symptoms (Cappa & Abutalebi, 1999) that may occur. This is mainly due to the heterogeneous nature of the distinct structures that may be involved (i.e. basal ganglia, different regions of subcortical white matter and the thalamic nuclei) and to their multiple connections with cortical association areas (see for review: Alexander, 1989). For this reason, Cappa and Abutalebi (1999) proposed an anatomic-functional distinction of various types of subcortical aphasia: aphasias due to lesions of the basal ganglia (striato-capsular aphasia), aphasias due to thalamic lesions (thalamic aphasia) and aphasias due to periventricular white matter lesions.

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