fMRI findings in an aphasic patient
with reversed cerebral dominance for language

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Abstract

An 80-year-old right-handed woman with no history of brain damage or familial strain of left-handedness acutely developed aphasia associated with a left hemiparesis following a right hemisphere stroke. Brain MRI showed a posterior insular ischemic infarction extending to the temporo-parietal region of the right hemisphere. Severe overall language disruption (global aphasia) in the acute phase of the stroke rapidly evolved into conduction aphasia, characterized by a neurolinguistic profile of disproportionately severe repetition deficits and markedly distorted phonological skills. In the lesion phase of the stroke, a functional MRI study using a word repetition task was conducted which revealed a consistent pattern of right hemisphere activations. For the first time, right hemisphere language dominance is demonstrated by fMRI in a clear instance of crossed aphasia in a dextral.

Key words : fMRI ; crossed aphasia ; cerebral dominance ; language ; cognition ; stroke.

Introduction

Reversed cerebral dominance for language – i.e. right hemisphere language dominance in right-handers – is extremely rare in dextrals. The incidence of crossed aphasia in dextrals (CAD) – i.e. aphasia following a right hemisphere lesion in right-handers – is variably estimated between 0.38% and 3% (Hécaen *et al.*, 1971 ; Carr *et al.*, 1981) in patients with vascular lesions. Since the first description of crossed aphasia by Byrom Bramwell in 1899, more than 200 etiologically different patients with CAD have been reported (for a review, see Mariën *et al.*, 2004). Continuous attention for this rare phenomenon has substantially improved the understanding of the neurobiological mechanisms underlying the functional organisation of language and related cognitive skills.

One of the most influential views on CAD was advanced in the 1970s by Brown *et al.* (Brown and Wilson, 1973 ; Brown, 1976 ; Brown and Hécaen, 1976). In accordance with the classical concept of acquired childhood aphasia and aphasia in sinistrals, CAD was defined as prototypically nonfluent and transient regardless of the lesion site. Many subsequent studies added evidence to this view, supporting the concept of more diffuse language representation as the consequence of a disrupted lateralisation process (e.g. Urbain *et al.*, 1978 ; Barroche *et al.*, 1979 ; Goldstein *et al.*, 1979 ; Pillon *et al.*, 1979 ; Donoso *et al.*, 1980 ; Barroche *et al.*, 1981 ; Delreux *et al.*, 1989). Since the advent of advanced neuroimaging techniques, the validity of this view has fundamentally been challenged. From the 1980s onwards studies on CAD have documented similar aphasia types, lesion-behaviour relationships and recovery patterns as in uncrossed aphasia (UCA) (mirror-image CAD cases) (e.g. Carr *et al.*, 1981 ; Yarnell, 1981 ; Joanette *et al.*, 1982 ; Henderson, 1983 ; Castro-Caldas and Confraria, 1984 ; Sweet *et al.*, 1984 ; Alexander *et al.*, 1989 ; Mariën *et al.*, 2001 a,b,c). However, in comparison with UCA the number of CAD cases that violate typically expected lesion-behaviour relationships (anomalous CAD cases) is disproportionately high. In this respect, review studies show that less than 13% of the cases of UCA display anomalous lesion-behaviour correlates (Basso *et al.*, 1978). This proportion increases to 35% in CAD (Alexander *et al.*, 1989 ; Coppens & Hungerford, 1998, Mariën *et al.*, 2004).

This study reports detailed clinical, neurocognitive and neuroradiological data of a patient who developed conduction aphasia after a vascular lesion in the typical homologous regions of the right hemisphere (mirror image CAD). To the best of our knowledge, this is the very first patient with an unambiguous diagnosis of vascular CAD in

whom deviant cerebral language dominance is documented by means of an fMRI study.

Case report

An 80-year-old right-handed woman was admitted to the hospital because of an abrupt onset of left-sided weakness and speech and language disturbances. On admission, the patient was alert, well-oriented and co-operative. Clinical neurological examination revealed severe aphasia affecting both expressive and receptive language levels. She was not able to execute simple commands and speaking, reading and writing were impossible. There was a left central facial nerve paresis and a moderate left hemiparesis mainly affecting the upper limb. Tendon reflexes were brisker on the left side of the body and the left plantar response was neutral whereas the right plantar response was flexor. Sensory system functions could not be tested due to aphasia. Co-ordination was impaired in the left upper and lower limbs, due to hemiparesis. Apart from arterial hypertension, medical and neurological history was unremarkable. As demonstrated by a laterality quotient of + 100 on the Edinburgh Handedness Inventory (Oldfield, 1971), the patient was a strong dextral and after careful inquiry no familial history of left-handedness was found. The patient had an educational level of nine years and had retired as a cleaning lady more than 20 years ago. A repeat brain CT scan three days after onset neurological symptoms revealed an ischemic lesion in the vascular territory of the right medial cerebral artery. An MRI of the brain was carried out one month after stroke and confirmed these findings, demonstrating focal ischemic damage to the right posterior insula, the temporo-parietal region and the adjacent white matter. No lesions were found in the left hemisphere.

After remission of global aphasia within the acute phase of the stroke, neurocognitive functions were formally assessed. Language was investigated three weeks postonset by means of the Dutch version of the Aachener Aphasia Test (AAT) (Graetz *et al.* 1992), the Boston Naming Test (Mariën *et al.*, 1998) and a verbal fluency task (unpublished norms). The aphasia profile matched a diagnosis of conduction aphasia. Repetition was severely disrupted while moderate to mild deficiencies were found at the level of verbal auditory comprehension, visual confrontation naming, reading comprehension, reading aloud and writing. Speech errors predominantly consisted of phonematic paraphasias (omissions, substitutions, additions and transpositions of sounds and syllables) and repetitive attempts to self-correction (*conduites d'approches*). Neuropsychological investigations carried out three weeks after onset of neurological symptoms consisted of a selection of standardized tests, i.e. the Coloured Progressive Matrices (Raven, 1983), the Hierarchic Dementia Scale (HDS) (Cole and Dastoor, 1987), the Rey-Osterrieth figure (Osterrieth, 1944) and the Judgment of Line Orientation test (JLO) (Benton *et al.*, 1983). Apart from a Gerstmann syndrome (left-right disorientation, finger agnosia, acalculia and agraphia) no associated neurospychological problems were identified. Orientation, visual memory, constructional, ideomotor, ideational and drawing praxis, gnosis and non-verbal problem solving were intact. Mood and behaviour were normal.

Functional magnetic resonance imaging

MATERIALS AND METHODS

An fMRI study was carried out using a word repetition task that consisted of 16 blocks in which block A (resting condition) and block B (silent repetition task) were repeated eight times. Each block lasted 1 minute, which resulted in a total scanning time of 16 minutes. These blocks were presented in an alternating pattern : AB(1)-AB(2)-…-AB(8). During the whole scanning session the patient was instructed to keep the eyes shut. Block A was a resting condition which required no cognitive or motor action. The patient was instructed to relax during this block. Block B consisted of a covert repetition task of auditory presented Dutch nouns (n = 80) carefully balanced for length (number of syllables), frequency of occurrence in the lexicon and imageability. Every 6 seconds a different noun was presented. The patient was asked not to repeat the auditory stimulus aloud but to slowly repeat the noun just once without moving the articulators. During each block B, 10 nouns were presented. None of the words were used more than once. Generated by the Cubase LE software package (Steinberg, Germany), the stimuli were presented through a custom built air transmission system. The silent fMRI protocol we used, produced a silent gap every 3 seconds, during which the words were presented for optimal intelligibility. In order to minimize motion artefacts, the patient's head was immobilized using foam cushions and a clamp, attached to the headphones she was wearing.

A 1.5T scanner (Sonata, Siemens, Erlangen, Germany), equipped with 40 mT/m gradients and a CP head coil was used. The acquired BOLD-sensitive T2* weighted EPI-images (TE/TR 50/3000 ms ; $3 \times 3 \times 3$ mm), covering cerebrum and cerebellum, were linked to a two-dimensional T1-weighted dataset (SE ; TE/TR 15/700 ; $1 \times 1 \times 1.5$ mm), which was, in turn, linked to a 3D volume T1-weighted dataset (MP-RAGE ; TE/TR 3.76/1700 ms ; $1 \times 1 \times 1$ mm). Every TR was followed by a 3000 ms pause, producing a silent gap, during which the nouns were presented, in order to optimise intelligibility. After preprocessing, including Gaussian spatial smoothing (8 mm FWHM),
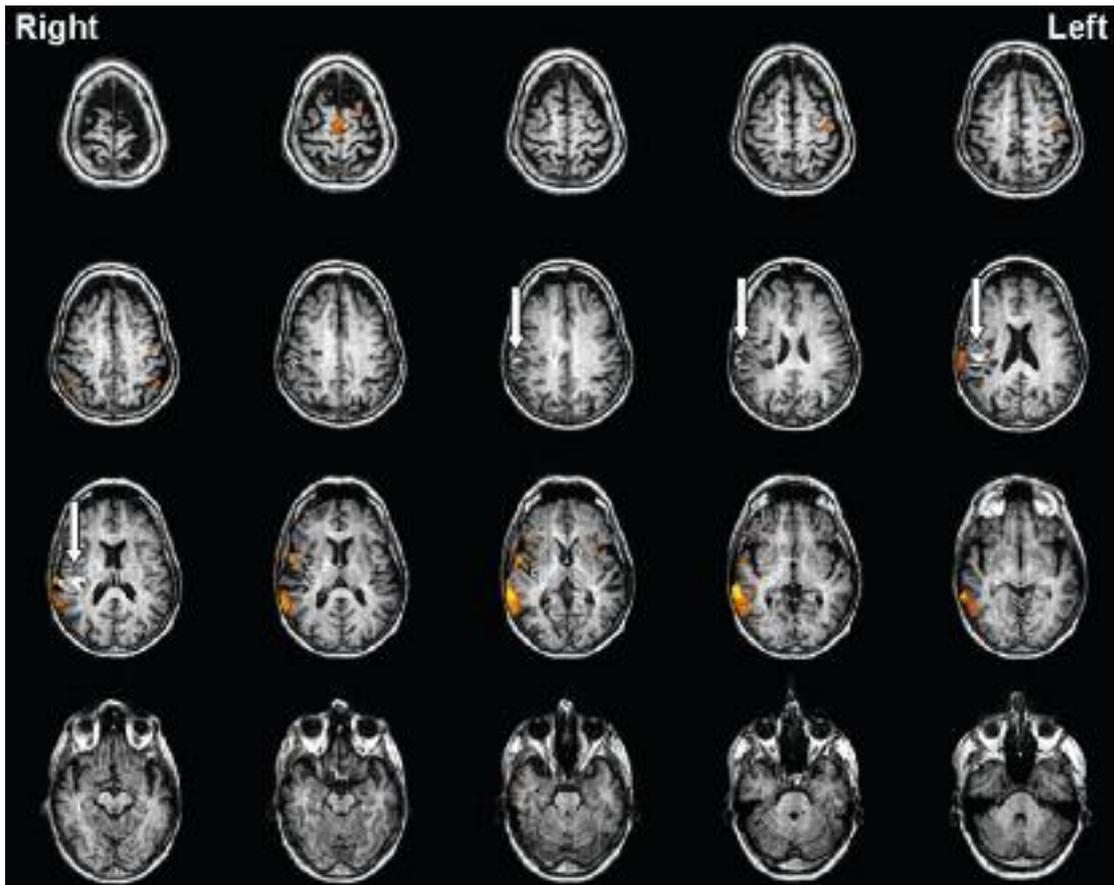
FIG. 1. — Two dimensional visualisation of the sites of activation superimposed on axial slices of the T1-weighted MPRAGE images (at t = 6,2 / radiological convention). See table 1 for details. Note the hypointense lesion (white arrows) in the right semi-oval centre and in the right fronto-temporal subcortical region due to ischemic damage. The hyperintense foci are caused by hemorrhagic conversion of the ischemic lesions situated in the vicinity of the right angular and supramarginal gyri (images in radiological convention). Z-coördinates are (from left to right and from above to below) : 65, 60, 55, 50, 45, 40, 35, 30, 25, 20, 15, 10, 5, 0, -5, -10, -15, -20, -25, -30.

Table 1

Anatomical location, Brodmann classification, x-, y- and z-coordinates, maximum t-value of the sites of activations and cluster sizes (at t = 6,2)

Left cerebrum						
Lobe / Gyrus	*BA*	*x*	*y*	*z*	*t-value*	*Nr of voxels*
Frontal Lobe, Precentral Gyrus	4	-38	-17	48	6, 9	435
Frontal Lobe, Superior Frontal Gyrus	6	-21	1	63	7, 2	285
Parietal Lobe, Inferior Parietal Lobule	40	-48	-53	42	6, 8	409
Claustrum		-33	15	5	6, 2	395
Right cerebrum						
Lobe / Gyrus	*BA*	*x*	*y*	*z*	*t-value*	*Nr of voxels*
Frontal Lobe, Medial Frontal Gyrus	6	2	-14	60	7, 1	1332
Frontal Lobe, Inferior Frontal Gyrus	45	45	21	4	6, 6	456
Frontal Lobe, Inferior Frontal Gyrus	47	50	22	0	6, 3	314
Temporal Lobe, Middle Temporal Gyrus	21	64	-36	2	9, 1	1664
Temporal Lobe, Superior Temporal Gyrus	22	55	2	7	7, 5	567
Temporal Lobe, Superior Temporal Gyrus	22	67	-21	6	7, 1	2221
Temporal Lobe, Superior Temporal Gyrus	38	57	16	-19	7, 4	783
Parietal Lobe, Postcentral Gyrus	40	67	-21	17	7, 4	307
Parietal Lobe, Inferior Parietal Lobule	40	47	-58	39	6, 7	227
Temporal Lobe, Superior Temporal Gyrus	41	50	-30	8	6, 8	153

high pass filtering, linear trend removal, motion correction and slice scan time correction, the brain was warped into standard stereotactic space using the Talairach template. In order to depict cerebral and cerebellar activity, the condition of interest was convoluted with a haemodynamic response function and served as an independent predictor in the general linear model (GLM). BrainVoyager QX (version 1.9 ; Brain Innovation, The Netherlands) was used for all data pre-processing and analysis. The cutoff value for visualisation of activation was set to a t-value of 6,2 coinciding with a false discovery rate of 1%.

<center>RESULTS</center>

Right cerebral hemisphere activations were found in the frontal lobe (BA 6, 45 and 47), the temporal lobe (BA 21, 22, 41 and 38) and the parietal lobe (40). In the left cerebral hemisphere, activations were found in the frontal lobe (BA 4 and 6) the parietal lobe (BA 40) and the anterior claustrum.

Discussion

This patient presented with global aphasia following an ischemic infarction in the right posterior insula, the right temporo-parietal region and the adjacent white matter. During the acute phase of the stroke, global aphasia receded and developed into a taxonomically typical conduction aphasia : formal language assessments revealed an aphasia profile in which repetition was disproportionately affected and phonology markedly disrupted.

The neurolinguistic symptoms in this patient are consistent with the operational criteria for the diagnosis of "reliable" crossed conduction aphasia (Mariën et al., 2004) since in this right-handed patient : 1) a persistent aphasia was objectified by language tests, 2) the structural integrity of the left hemisphere was confirmed by a brain MRI scan, 3) early brain damage or a history of epilepsy was excluded and 4) no family history of left-handedness was found after careful inquiry.

Neuro-anatomical studies of conduction aphasia in right-handers have identified lesions of the left inferior parietal lobe (supramarginal gyrus, BA 40), the left primary auditory cortices (BA 41 & 42), the left insular cortex (BA 13 & 14) and the adjacent white matter (Axer & Berks, 2001 ; Benson et al., 1973 ; Damasio & Damasio, 1980), as well as lesions in the left temporo-occipital region (Bartha et al., 2004) and the left arcuate fasciculus in some cases (Damasio & Damasio, 1980 ; Palumbo, 1992 ; Tanabe et al., 1978). Since the lesion-aphasia relationships in this patient are comparable to those following analogous lesions in the left hemisphere, the anatomo-clinical configurations of this patient match the concept of mirror-image conduction CAD.

Neurocognitive investigations carried out in the lesion phase of the stroke revealed full-blown Gerstmann syndrome (Gerstmann, 1940). This finding suggests that in addition to atypical hemispheric lateralisation of the language functions, reversed hemispheric dominance for at least some of the typical dominant hemisphere functions (calculation, left-right orientation, fingergnosis) existed as well. Indeed, Gerstmann syndrome in right-handers generally follows from destruction of the typical lesion area situated in the left parietal region with the angular gyrus (BA 39) crucially involved (dominant angular gyrus syndrome). In addition, absence of typical non-dominant hemisphere symptoms in this patient such as constructional apraxia or left visuo-spatial neglect might suggest reversion of the 'non-dominant hemisphere functions', with atypical lateralisation of these functions in the left hemisphere.

Right hemisphere language dominance in this mirror-image CAD case is for the first time confirmed by fMRI. Indeed, the language paradigm consisting of a covert repetition task of Dutch nouns predominantly activated classical posterior/receptive language regions atypically lateralised in the right hemisphere : Wernicke's area (BA 22) and the inferior parietal lobe (supramarginal gyrus - BA 40). In the contralateral left hemisphere no homologous activations were found. In addition, activation of the pars triangularis of Broca's anterior speech area (BA 45) was found in the right hemisphere while no activations occurred in the contralateral homologous motor speech region of the left hemisphere. Although the language task predominantly activated eloquent areas in the right hemisphere, small spots of left hemisphere activity occurred in the frontal lobe (BA 4 and 6), the parietal lobe (BA 40) and the anterior part of the left claustrum. It may be speculated that activation of the left claustrum might indicate that covert repetition is closely bound with the formulation of an articulatory plan and that motor speech planning in our patient partly involved the brain areas typically linked with articulatory planning (Wise et al., 1999) in the left hemisphere. However, the small circumscribed spots of left hemisphere activity might alternatively be explained as : 1) the result of neuroplasticity during the restoration of language functions and/or 2) the result of neuroplastic changes induced by aphasia treatment. After a long-standing tradition of lesion-behaviour research in aphasia, the most debated issue regarding the neuroplastic substrates of aphasia recovery and treatment remains the role of the nondominant hemisphere versus the role of intact brain tissue surrounding the aphasiogenic lesion in the language dominant hemisphere. Many clinical and functional neuroimaging studies with PET and fMRI have suggested that recovery of language functions in aphasia is due to a transposition of the destroyed linguistic functions

to the homologue areas in the nondominant, contralateral hemisphere (Weiller *et al.*, 1995). On the other hand, a variety of clinical and neuroimaging studies has demonstrated that recovery may be achieved by novel cognitive strategies for linguistic performances recruited in the uninjured regions adjacent to the aphasiogenic lesion in the language dominant hemisphere (Rosen *et al.*, 2000). Which mechanism determines the restoration of impaired language processing in which temporal succession and to which extent remains to be elucidated (Cappa & Abutalebi, 2008). Current research, however, seems to suggest that both nondominant and perilesional tissue might play a crucial role in aphasia recovery under specific circumstances but that these circumstances are still to be identified (Crosson *et al.*, 2007).

As demonstrated in this patient with reversed cerebral language dominance, fMRI based on a linguistic activation paradigm is a very powerful tool to investigate anomalous lesion-behaviour relationships in atypical clinical populations. As a result, the different combinations of typically dominant and non-dominant cognitive disorders in CAD deserve further prospective research with fMRI, as they offer a unique opportunity to uncover the neurobiological principles which govern the separate lateralisation of cerebral functions.

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