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CASE REPORT

Crossed aphasia. II: Why are deep lesions overrepresented with respect to standard aphasia?

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In this paper we have reviewed the cases of vascular crossed aphasia reported in the literature, in order to check whether deep lesions are really overrepresented in crossed aphasia with respect to standard aphasia. The comparison with a large sample of standard left-hemisphere-damaged aphasics revealed a significantly higher incidence of purely deep lesions in crossed aphasics than in standard aphasics. The overrepresentation of deep lesions in crossed aphasia appears to be contingent on the co-occurrence of aphasia and Unilateral Neglect after right-hemisphere lesion. This suggests an interaction between language and attentional mechanisms in the case of reversed language lateralisation: the overcrowding of these functions in the right hemisphere could make language more vulnerable after right deep lesions.

Keywords: Aphasia – Deep lesions – Hemispheric specialisation

INTRODUCTION

The aim of this paper was to review the cases of crossed aphasics reported in the literature, in order to see whether there is really an overrepresentation of deep lesions, as suggested by Habib *et al.* (1983). We first took into account the hypothesis of Habib and colleagues concerning the special role of deep nuclei in crossed aphasia attributable to their possible lesser degree of lateralisation. Following a different approach, we then discuss whether the distribution of lesions in crossed aphasics is influenced by the crowding of language and spatial abilities within the same hemisphere.

METHODS AND RESULTS

Review of the published cases of deep crossed aphasia

We will start by updating the review of crossed aphasic patients published by Joanette *et al.* (1982), following the strict criteria adopted by those authors that are listed below.

(1) Strong right-handedness with no familial (parents and siblings) sinistrality.
(2) Literacy.
(3) Unilingual.
(4) Absence of childhood brain damage or previous lesions in adulthood.
(5) CT-documented lesion restricted to the right hemisphere.
(6) Thorough language testing at least two weeks post-onset of the disease.

We excluded cases of head injury, neoplasia and infections of the central nervous system because, as pointed out by Boller (1973), such lesions might cause bilateral damage. Considering only deep lesions, eight patients in the literature, and the patient MR (see accompanying paper) fulfilled these criteria. Tables 1A and 1B show the clinical data of the nine patients considered.

In these cases, testing most frequently disclosed the association of a 'left-hemisphere' deficit (aphasia and acalculia) and unilateral neglect (which is generally associated with right-hemisphere lesions). No consistent aphasic pattern was evident apart from the greater impairment of written language and the better preserved repetition described in most "subcortical" cases.

TABLE IA. Clinical data and neuropsychological features of deep crossed aphasics

Case	Age (years)	Sex	M.D.	S.D.	Type of lesion	Side of lesion lesion	Type of asphasia	O/W diss.	L-H functions IMA	OA	AC	Bilateral CAP	R-H functions ULN	S-P	AL
1 MR	67	M	+	+	H	Thalamus, putamen	Broca	w<o	–	–	+	–	+	–	+
2 Carr *et al.* (1981) case No 3	80	M	+	–	H	Deep temporal, internal capsule, thalamus, caudate nucleus, putamen	Tr. s.	w<o	ne	ne	ne	ne	+	ne	–
3 Habib *et al.* (1983)	61	M	+	+	I	Lenticular nucleus, caudate nucleus, internal capsule	W.	w<o	ne	ne	ne	+	+	ne	ne
4 Colombo *et al.* (1984)	67	M	+	+	H	Basal ganglia	W.	w<o	–	–	ne	+	+	ne	ne
5 Basso *et al.* (1985) case No 6	56	M	+	+	H	Basal ganglia, insula, internal and external capsule	W.	w<o	–	–	+	–	pp–	ne	ne
6 Fromm *et al.* (1985) case No 6	64	M	+	ne	H	Lenticular nucleus, internal capsule	Tr.s.	w<o	ne	ne	ne	+	+	ne	ne
7 Fromm *et al.* (1985) case No 11	75	M	+	+	H	Thalamus	A.	w<o	ne	ne	+	ne	+	ne	ne
8 Perani *et al.* (1988) case No 2	67	M	+	–	H	Basal ganglia, internal capsule, thalamus	Tr. Mix.	w<o	+	+	ne	+	–	ne	ne
9 Cappa *et al.* (1993) case No 1	79	F	+	+	I	Lenticular nucleus, periventricular white matter	Tr. Mix.	w<o	+	+	ne	Ne	+	ne	ne

Motor Deficits (M.D., + presence/– absence), Sensory Deficits (S.D., + presence/– absence), Haemorrhage (H), Ischaemia (I), O/W diss. = oral tasks are performed better (>), equal to (=) or worse (<) than written tasks at least in one instance. Broca Aphasia (B), Transcortical Sensitive Aphasia (Tr.s), Wernicke Aphasia (W), Conduction Aphasia (C), Mixed Transcortical Aphasia (Tr.mix.) Amnestic Aphasia (A.); Left hemisphere (L-H), Right hemisphere (R-H), Ideomotor Apraxia (IMA), Oral Apraxia (OA), Constructional Apraxia (CAP), Acalculia (AC), Unilateral Neglect (ULN), Spatial and Perceptual Impairment (S-P), Affective Language (AL), Pathological (+), Normal (-), Not Examined (ne), Position Preference (pp).

TABLE IB. Language patterns of deep crossed aphasics

Case	Speech output	Comprehension	Repetition	Writing
1	Paucity of speech, echolalia, wordfinding difficulties, verbal paraphasias, non-words	Mild	Mild	Mispelt, unrecognisable words
2	Echolalia, jargon, verbal and literal paraphasia, non-words	Poor	Mild	Strings of unrelated words
3	Phonemic and verbal paraphasias, jargon	Good	Mild	Pure jargon
4	Dysarthrias, word-finding difficulties, verbal paraphasias, non-words	Poor	Poor	Unrecognisable words
5	Poor, word-finding difficulties, phonemic paraphasias	Good	Normal	Unrecognisable words
6	Paraphasias, anomias, perseverations	Impaired	Excellent	Impaired
7	Anomias	Mild	Mild	Sloppy, inaccurate or imcomplete
8	Poor, jargon, semantic paraphasias	Severely affected	Mild	Only his signature
9	Severe reduction, anomias, paraphasias, perseverations	Defective	Mild	Severe agraphia

The role of deep structures in crossed aphasia

Habib *et al.* (1983) have suggested that the deep nuclei play a major role in the genesis of crossed aphasia because of their lesser lateralisation. Seemingly, this implies that deep lesions are more common among crossed aphasics than standard aphasics. A greater prevalence of subcortical lesions in crossed aphasia has already been remarked upon (Joanette *et al.*, 1982; Cappa *et al.*, 1993) and questioned (Basso *et al.*, 1985), but never confirmed in a population study. Thus, to test this hypothesis, we compared the incidence of deep lesions in crossed and standard aphasics. In addition to the cases reviewed by Joanette *et al.* (1982), we found further reports of crossed aphasia which met Joanette *et al.*'s criteria listed above. Table II shows the cases reported by Joanette *et al.* (1982), and the new cases (until May 1994). Thirty-six patients (including MR, see accompanying paper) fulfilled our adopted criteria. The excluded cases and the grounds for their exclusion are reported in the Appendix.

TABLE II. Reported cases of crossed aphasia

	Age	Sex	Aphasia	ULN	C,WM	DWM	C+DN	DN
1) Urbain *et al.* 1978	37	F	B	–	+			
2) Denes and Caviezel, 1981	35	M	B	–			+	
3) Carr *et al.* 1981, No2	61	M	G	–	+			
4) Carr *et al.* 1981, No3	80	M	TS	+				+
5) Haaland and Miranda, 1983	67	F	C	–		+		
6) Puel *et al.* 1982	55	M	C	+			+	
7) Assal, 1982	60	M	C	–			+	
8) Habib *et al.* 1983	61	M	C	+				+
9) Henderson, 1983, No2	60	F	W	+	+			
10) Henderson, 1983, No3	61	M	W	+	+			
11) Colombo *et al.* 1984	67	M	W	+				+
12) Kapur and Dunkley, 1984	57	M	B	+			+	
13) Hindson *et al.* 1984	72	M	B	–	+			
14) Basso *et al.* 1985, No1	64	F	B	–	+			
15) Basso *et al.* 1985, No2	53	F	W	+	+			
16) Basso *et al.* 1985, No3	55	M	W	+	+			
17) Basso *et al.* 1985, No4	63	F	W	–	+			
18) Basso *et al.* 1985, No5	49	F	AG	+	+			
19) Basso *et al.* 1985, No6	56	M	W	–				+
20) Basso *et al.* 1985, No7	64	F	B	+	+			
21) Fromm *et al.* 1985 No6	64	M	TS	+				+
22) Fromm *et al.* 1985, No11	75	M	A	+				+
23) Demeurisse *et al.* 1986	65	M	W	–			+	
24) Reinvang, 1987	56	M	TS	+			+	
25) Schweiger *et al.* 1987	59	M	FL	–	+			
26) Castro-Caldas *et al.* 1987, N1	34	M	B	–	+			
27) Castro-Caldas *et al.* 1987, N2	66	M	G	+	+			
28) Perani *et al.* 1988, No2	67	M	TMx	–				+
29) Delreux *et al.* 1989	74	M	B	–	+			
30) Alexander *et al.* 1989, No1	54	M	W	–			+	
31) Faglia and Vignolo, 1990	65	F	G	+			+	
32) Berndt *et al.* 1991	56	F	C	–	+			
33) Cappa *et al.* 1993, No1	79	F	TMx	+				+
34) Cappa *et al.* 1993 No2	56	M	FL	–		+		
35) Cohen *et al.* 1993	72	F	B	–	+			
36) MR	67	M	B	+				+

Cortical lesion (with or without participation of the immediately underlying white matter) (C,WM), Deep white matter lesion in a specific lobe (DWM), Cortical and deep nuclei lesion (C+DN), Deep nuclei lesion (DN), Global aphasia (G), Broca's aphasia (B), Conduction aphasia (C), Wernicke's aphasia (W), Transcortical Sensitive aphasia (TS), Agraphia (AG), Transcortical Mixed aphasia (TMx), Fluent aphasia (FL); Unilateral Neglect (ULN); position preference (pp+). Patients have been classified as ULN + if they had definite Neglect or position preference not accounted for by hemianopia.

We considered a reference sample of 287 standard aphasic patients from Milan University (Laiacona, 1985) who met the criteria used in our list of crossed aphasics. This sample was drawn from a continuous series of 1085 patients tested in the Aphasia Unit of the University of Milan (Neurological Department), in the period 1978–1985 and was studied (courtesy of Prof. A Basso) by one of the authors (M.L.) in her post-doctoral dissertation (Laiacona, 1985). Part of these patients were also studied in the paper by Basso, *et al.* (1987). In the reference sample, all patients had focal left hemisphere damage whose vascular aetiology and site of lesion were documented by CT-scans. Deep lesions in the reference sample included basal ganglia and/or thalamus and/or internal capsule and/or external capsule and/or insula. This cumulative localisation did not allow us to separate cases of deep lesion with or without involvement of the deep nuclei; however, the cerebral cortex was always spared. As a result, the number of deeply damaged patients may have been overestimated in the reference sample. We decided to accept this bias, as it acts against the hypothesis under test. Lesions sparing all of the above structures were defined as 'cortical', even if, strictly speaking, they also involved a certain amount of the underlying white matter. In the standard aphasic sample, the 'cortical lesioned' group also included patients with a single lesion in the white matter of a specific lobe. Similar crossed aphasic patients (numbers 5 and 34 in Table II) with a lesion confined to the white matter underlying the supramarginal gyrus or a periventricular lesion, were included in the cortical group. The incidence of haemorrhage was 15% in the standard sample and 21.6% in the crossed sample, a discrepancy far from statistical significance (Chi-square = 1.09, D.F. = 1, p = N.S.).

In this sample, it appears that pure deep lesions were more common in the crossed aphasic group: 25.0% versus 7.0%.

The incidence of deep lesions in the standard aphasic population can also be estimated from other sources. Thus, as a check on the reliability of our figures, we considered the recent survey of 221 cases by Willmes and Poeck (1993). In this series of patients (see their Table 1) there was a rate of deep lesions of 14.9%. This is a higher figure than that estimated from the Milan sample. However, some of the deep cases from the Willmes and Poeck series also had cortical damage (see their Table 3), whereas mixed cases were not considered 'deep' in either the Milan sample or the crossed group. Therefore, the rate of pure deep lesions observed in the Willmes and Poeck series is likely to be smaller than 14.9% (however, it should be recalled that among crossed aphasics, the incidence of pure deep lesions was 25.0%). Notwithstanding this overestimation of pure deep lesions in standard aphasics, we decided to collapse the German and Milan series of standard aphasics; the resultant incidence of purely deep standard aphasics was 10.4%. The comparison with the crossed group (see Table III) yielded a significant Chi-square (7.064, D.F. = 1, p = 0.008).

TABLE III. Prevalence of standard and crossed aphasics according to the lesion site in different samples.

	Standard aphasics (Laiacona, 1985 + Willmes and Poeck, 1993): n = 508	Crossed aphasics (overall) n = 36
Cortical lesions	263 (51.8%)	19 (52.78%)
Cortical and deep nuclei lesions	192 (37.8%)	8 (22.2%)
Purely deep lesions	53 (10.4%)	9 (25.00%)

For a comment about the patients' classification made by Willmes and Poeck, 1993, see text.

The prevalence of deep lesions with or without a cortical lesion was not higher in crossed aphasic than in standard aphasics (47.22% versus 48.23%; Chi-square <1, N.S.): This finding casts doubt on Habib *et al.*'s (1983) hypothesis that implies a greater representation of deep lesions, irrespective of the presence of associated cortical damage.

DISCUSSION

Habib *et al.* (1983) have suggested that aphasia is seen more often following a right deep lesion than a right cortical lesion because subcortical structures, of an earlier philogenetic origin, are systematically 'less lateralised'. However, their hypothesis is loosely defined and seems to suggest that not only purely deep lesions, but also mixed lesions (cortical and deep) are overrepresented in crossed aphasics. In our survey, deep lesions (with and without cortical involvement) were not overrepresented in crossed aphasics whereas only pure deep lesions were.

To account for the higher incidence of purely deep lesions among crossed aphasics, we have an alternative hypothesis. The figures we have analysed so far were drawn from samples selected for the presence of aphasia. Let us consider the case of standard, left-hemisphere damaged patients: it is well known that a certain percentage of patients with purely deep lesions are not included within samples selected for aphasia, on the grounds that they show no language

disturbances (see e.g. Cappa *et al.*, 1983, 1986). As a result, the percentage of patients with purely deep lesions is *smaller* when subjects are sampled for aphasia than when they are sampled for brain lesions. Unfortunately, satisfactory samples of the latter type are not available from the literature.

Let us now consider crossed aphasics. Here again, our focus is on subjects selected for the presence of aphasia. As for standard aphasics, the percentage of purely deep lesions is influenced by the rate at which patients slip through the sampling net because their language is unaffected. One possible explanation for the overrepresentation of purely deep lesions among crossed aphasics is that fewer subjects with crossed language representation are free from aphasia after a purely deep lesion. In other words, the right hemisphere of subjects with crossed language representation could be equally liable to aphasia either due to cortical or deep lesions, whereas among subjects with standard representation, deep structures are less often necessary to ensure language functions.

Among crossed aphasics it seems feasible that the concentration of both language and attentional functions within one hemisphere (and the consequent sharing of neural resources) would make these functions more fragile. The effect could be a greater incidence of aphasia after purely deep lesions only in anomalous dextral subjects, with language and attention 'crowded' in the right hemisphere.

In this respect, it is of interest to group the crossed aphasics described in the literature according to the presence or absence of unilateral neglect (Table IV).

In our review of 36 crossed aphasics (Table II), purely deep lesions were present in seven of the eighteen patients with Unilateral Neglect (38.9%), whereas in the other eighteen patients without Neglect, deep lesions were present in only two (11.1%). The analysis of the resultant contingency table is shown in Table IV. The degrees of freedom were split according to the method of Kimball (Maxwell, 1961). The proportion of deep lesions was greater in crossed aphasics, but varied according to the presence of Unilateral Neglect. The incidence of deep lesions in crossed aphasics *without* Unilateral Neglect was similar to that of standard aphasics, whereas it was higher in crossed aphasics *with* Unilateral Neglect. An interference between language and *visuo*-attentional mechanisms, represented in the same hemisphere in the case of cross-lateralisation of language, and a possible forced sharing of neural resources could explain the greater vulnerability of language after a purely deep lesion in these cases. The topic of right hemisphere crowding has been discussed so far with reference to early unilateral damage to the left hemisphere that causes a right language representation. In these cases there is a greater vulnerability for those capacities that normally depend on the right hemisphere (Teuber, 1974; Milner, 1974). In our case we hypothesise that right deep structures are crucial for language and visual attention when the latter functions are both subserved by the right hemisphere. This suggests a peculiarity of the intrahemispheric reorganisation of the right hemisphere, possibly on the basis of a more diffuse representation of functions, as suggested by Strauss *et al.* (1990). As can be appreciated from an inspection of Table IV, crossed aphasia can be observed also without the concomitance of Unilateral Neglect and the literature reports cases of 'pure' crossed aphasia without other symptoms normally associated with right hemisphere lesions (e.g. Marshall and Halligan, 1992). Functional overcrowding of the right hemisphere is by no means necessary for the appearance of crossed aphasia. However, the lack of evidence of visuo–perceptual deficits and of Unilateral Neglect in crossed aphasics may derive from different sources. Either the functional localisation is completely crossed, and all the functions normally represented on the right side are

TABLE IV. Analysis of the relationship between lesion site, hemispheric language representation and Unilateral Neglect

	Crossed aphasics with Unilateral Neglect: $n = 18$	Crossed aphasics without Unilateral Neglect: $n = 18$	Standard aphasics (Laiacona, 1985 + Willmes and Poeck, 1993): $n = 508$
Cortical lesions	7 (38.9%)	12 (66.7%)	263 (51.8%)
Cortical and deep nuclei lesions	4 (22.2%)	4 (22.2%)	192 (37.8%)
Purely deep lesions	7 (38.9%)	2 (11.1%)	53 (10.4%)

Overall Chi-square = 15.916, D.F. = 4, $p = 0.003$.
Partition of degrees of freedom: Within crossed aphasics: interaction between lesion site (cortical versus cortical and deep) and presence of Unilateral Neglect: Chi-square = 0.556, D.F. = 1, N.S. Between crossed and standard aphasics: is there a different proportion of cortical lesions versus cortical and deep-lesions? Chi-square = 1.419, D.F. = 1, N.S. Within crossed aphasics: is the proportion of purely deep lesions greater when patients are affected by Unilateral Neglect? Chi-square = 6.877, D.F. = 1, $p = 0.009$. Between crossed and standard aphasics: is the proportion of purely deep lesions greater among crossed aphasics? Chi-square = 7.064, D.F. = 1, $p = 0.008$.

subserved by the left-hemisphere, or these functions are still right-sided, but their neural substrate was spared by the lesion. In the individual patient only a functional activation study with PET or analogous techniques may provide the answer to such questions. Whereas group studies can estimate the general statistical rules governing the clinico–anatomical correlations, they cannot provide all-or-none rules that are valid for single cases. In every case, should our hypothesis be confirmed, the study of interference between language and attentional functions within the same hemisphere could represent an interesting avenue for future neuropsychological research.

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APPENDIX

Fresh cases of crossed aphasia reported in the literature by Joanette *et al.* (1982) until May 1994, not present in our list grouped for the reason of exclusion.

Absence of true aphasia or only transitory symptoms:
- Guard *et al.* 1983
- Perani *et al.* 1988; N.1.

Lack of CT scan:
- [1]Angelergues *et al.* 1962
- [1]Fernandez-Martìn *et al.* 1968 (excluded also for the non-vascular aetiology and for his pre-surgical examination).
- [1]Brown and Wilson, 1973
- Henderson *et al.* 1981 (excluded also for the lack of anamnestic and clinical data).

Non-vascular aetiology:
- Larrabee *et al.* 1982
- Castro-Caldas *et al.* 1986
- Giovagnoli, 1993
- Primavera and Bandini, 1993

Presence of lesion on the left side:
- Alexander *et al.* 1989; N.2
- Sweet *et al.* 1984

Presence of other neurological disorders:
- Loring *et al.* 1990
- Hadar *et al.* 1991

Too poorly educated:
- Roeltgen and Heilman, 1983
- Fromm *et al.* 1985 N.9 (excluded also for a small older lesion affecting the left occipital lobe).
- Castro-Caldas *et al.* 1987, N.3

Handedness not assessed:
- Brust *et al.* 1982 (excluded also because of his poor language examination).

Presence of familial sinistrality:
- Pillon *et al.* 1979 N.1 and N.2
- Assal *et al.* 1981
- Henderson, 1983 N.1
- Fournet *et al.* 1987

Too early examination (less than two weeks) or long-term follow-up:
- Fromm *et al.* 1985, N.8 (excluded also for transitory symptoms).
- Castro-Caldas *et al.* 1987, N.4
- Walker-Batson *et al.* 1988

Speakers languages written in non-alphabetic script:
- Washimi *et al.* 1987
- Hamasaki *et al.* 1987
- Hu *et al.* 1990
- Sakurai *et al.* 1992

Multilinguism:
- Karant and Rangamani, 1988
- Solin, 1989
- Paradis and Goldblum, 1989 (excluded also for non-vascular aetiology).

Acquired aphasia in children:
- Martins *et al.* 1987
- Assal, 1987

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[1]Cases included by Joanette *et al.* (1982) and excluded because they were not CT-documented.