

Transcortical Sensory Aphasia in a Right-Handed Patient Following Watershed Infarcts in the Right Cerebral Hemisphere: A 15-Month Evaluation of Another Case of Crossed Aphasia

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Well-documented cases of crossed (transcortical sensory) aphasia, especially those with longitudinal evaluation, are rare. We report a case of crossed transcortical sensory aphasia following watershed infarcts in the right hemisphere, from the moment of the accident until 15 months afterward. The aphasia type, and the course of recovery, is a “mirror” representation of that seen in cases of uncrossed aphasia. Unfortunately, the data do not permit strong conclusions regarding the lateralization of language in association with praxis and visuospatial abilities. This underlines the need for more well-documented (case) studies to come to a better understanding of the mechanisms through which lateralization occurs. © 1999 Academic Press

INTRODUCTION

Transcortical sensory aphasia is characterized by preserved repetition in addition to impaired auditory comprehension, fluent spontaneous speech with semantic paraphasias, and word-finding difficulties (Alexander et al., 1989a). Reading and writing are usually disturbed in a similar manner (Alexander et al., 1989a). Transcortical sensory aphasia is described after lesions of the anterolateral thalamus (Servan et al., 1995; Graff-Radford et al., 1985), the posterior second and third temporal gyri (Damasio, 1991), the temporo-parieto-occipital junction (Heilman et al., 1981), the inferior temporo-occipi-

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tal region (Kertesz et al., 1982), and the posterior periventricular white matter adjacent to the posterior temporal isthmus (Alexander et al., 1989a).

Aphasia in a right-handed person as a result of a lesion in the right cerebral hemisphere, so-called crossed aphasia, is rare. Its estimated incidence is 1 to 2 % of all right-handed aphasics. (Gloning et al., 1969). The English and French literature scarcely contain 100 well-documented cases of crossed aphasia (Faglia et al., 1990), including 3 cases of transcortical sensory aphasia (for references see Alexander et al., 1989b). Only 34 cases provide sufficient information for "review" (Alexander et al., 1989b).

Well-documented cases, especially those with longitudinal evaluation of the aphasic disturbance, are rare (Rey et al., 1994; Trojano et al., 1994). This article presents the course of the aphasia profile, and some other neuropsychological disturbances, in a right-handed patient following watershed infarcts in the right cerebral hemisphere, from the moment of the accident until 15 months afterward.

CASE REPORT

HSJ, a 70-year-old purely right-handed woman (Edinburgh Handedness Inventory, Oldfield, 1971) with a high school education and no family history of left-handedness, was admitted for evaluation of muscular spasms and loss of skilled fine movements of the left leg. General and physical examination revealed no abnormalities. Neurologic examination only showed absent Achilles tendon reflex. Duplex sonography showed a 70% stenosis of the right internal carotid artery, which later increased up to 90% 6 months later and was confirmed by digital subtraction angiography.

HSJ underwent right-sided carotid endarterectomy. Immediately after the operation HSJ was able to move her arms and legs normally. However, 2 h later she was unable to lift her left arm. Neurological examination revealed full consciousness, palsy of left-sided gaze, left homonymous hemianopia, and left hemiplegia. To our surprise, in the following days an aphasic disturbance was observed. The muscle strength and function of the left leg showed a good recovery in about 3 weeks. The left arm remained paralytic and non-functional, and the hypotonia of the left arm gradually evolved into a slight hypertonia. The hemihypesthesia remained, although HSJ was able to locate firm stimuli. The left homonymous hemianopia in the lower quadrant persisted.

Neuropsychological Assessment

The first week HSJ was unable to produce spontaneous speech, except the word "yes." Auditory comprehension seemed relatively well preserved in uncomplicated functional communication. Naming and repetition were not possible. Production of automatized sequences (the alphabet, counting, days of the week) was possible after prompting the first one or two words. In an

attempt to write, single graphemes were recognizable, the rest was cryptic jargon. Reading aloud and reading comprehension were severely disturbed.

The following 2 weeks HSJ began to speak in short, incomplete sentences. Striking disturbances were word-finding difficulties, semantic paraphasias, and (to a less extent) perseverations. She did not notice these semantic paraphasias and perseverations. She began to succeed with naming: at first HSJ was just able to complete a prompted unfinished sentence, later she could name (pictures of) objects, often helped by a cue, such as by formation of the lips of the first sound. Later on she began to use abstract words instead of specific names or unusual synonyms to evade word-finding difficulties: she replaced the intended sentence "my son lives in Amsterdam" with "my son chooses domicile in Eindhoven, no Eindhoven no." Repetition and automatic sequences were no longer a problem. The reading and writing difficulties persisted. In some instances this patient was not able to recognize a specific grapheme: she then read aloud a sound that as a grapheme showed a strong resemblance with the presented grapheme. In other instances she could not name a specific grapheme: she then helped herself by reciting the alphabet to get to the right sound. Reading aloud words and sentences did not go well. Quite often this patient just made a guess on the strength of the context. Sometimes she read words that were not in the presented text. Furthermore, reading was impeded by the lower quadrant hemianopia. In 9 of 10 instructions, writing on dictation resulted in a neologistic letter or word sequence.

Twenty-seven days postictal, the Dutch version of the Aachen Aphasia Test (AAT) [Graetz et al., 1992 (Huber et al., 1984)] was administered. Spontaneous speech was slow, approximately 60 words/min. It was characterized by abundant word-finding difficulties, many semantic and a few phonological paraphasias, and stereotypy. HSJ made very long, complex sentences with many narrowings and doublings and many cut-offs. In the test setting comprehension was, in contrast with the findings of the first week, moderately to severely disturbed. She scored better on word comprehension than on sentence comprehension. On the word level she often chose pictures with a meaning closely related to the presented word (presented is *bed*, chosen is *pillow* instead of *flowerbed*; presented is *star*, chosen is *sun* instead of *famous singer*). On the sentence level there was, in 3 of 10 items, usually no connection between the presented sentence and the chosen picture (presented is *how does she behave*, chosen is *a girl going upstairs* instead of *a girl helping mother cleaning the dishes*). The "naming" subtest resulted as follows. No mistakes were made in Color naming. Naming single nouns resulted twice in a severe semantic failure (e.g., *sprinkler* instead of *tractor*; *telephone block* instead of *till*) and once in a correct name with a semantically appropriate specification. Naming composite nouns resulted four times in a severe semantic failure (e.g., *paintbrush* instead of *vacuum cleaner*; *1,2,3,4* instead of *roller-skate*) and once in an appropriate but incorrect word

TABLE 1
Percentile Scores on the Aachen Aphasia Test

	4 weeks	5 months	12 months
Token test	45	72	74
Repetition	96	98	97
Writing	35	58	74
Naming	55	79	87
Comprehension	29	75	71

(*flagship* instead of *sailboat*). The “naming situations and actions” subtest resulted twice in evasive responses and twice in semantic disagreement with the intended sentence. HSJ also produced several semantic paraphasias and inaccuracies: she used a pronoun instead of a noun once and produced an abstract word instead of the specifically asked name once. This patient made few mistakes on the “repetition” subtest. She substituted a sound and a word, omitted a word, and displaced a word in a sentence one time each. On the “reading aloud” subtest this patient substituted a sound and a word one time each (both were graphemes on the left side of the page). In a longer sentence she replaced a word with another word that contained a part of the original word (*vain person* instead of *to be vain*). The patient was unable to read one sentence at all. The percentile score on the “reading comprehension” subtest was 29, indicating, as on the “auditory comprehension” subtest, a moderate to severe disturbance. On this subtest the patient scored better on word comprehension than on sentence comprehension. Writing from dictation was more severely disturbed in proportion to the length of the dictated words: substituting one sound evolved into omitting and multiple changing of more than one-third of the original word/sentence to neologistic letter sequences. During this subtest HSJ loudly repeated the dictated word. In 9 of 10 items, writing from dictation resulted in neologistic letter or word sequences (Table 1).

Five months after the stroke the AAT was reassessed. Insofar as the phonological and semantic structures were concerned, spontaneous speech showed no substantial changes: some phonological paraphasias were still heard (“verwangen” instead of “verlamming”) and the patient still made long sentences with many cut-offs. In all subtests, except “repetition” and “reading aloud,” there was a significant improvement. The clear difference between the “repetition” subtests and the remaining subtests persisted. Auditory and reading comprehension were equally disturbed, the disturbance now being slight. Incompleteness and/or semantic inaccuracies still dominated the “naming situations and actions” subtest. The most remarkable disturbances were seen on the “writing from dictation” and “composing from dictation” subtests. When dictated to she sometimes was not able to produce a word or start a sentence at all. When asked to compose words from graph-

emes printed on blocks, she was unable to select all the right graphemes and/or sequence them correctly (when asked to compose *priester*, she composed *riest*; *garage* became *arge*). Classification according to the AAT now resulted in 100% definite aphasia with a transcortical sensory profile of slight severity. The percentile score on the Token Test was 72 (Table 1).

Twelve months postictal the AAT was assessed for the third time. Only a few phonological uncertainties and cut-offs were heard at spontaneous speech. In the "auditory comprehension" and "reading comprehension" subtests no differences were found between the second and third assessment. In the "naming" subtest there was a slight improvement, but still some semantic inaccuracies were found (e.g., *car* instead of *tractor*). A striking improvement was noted in the "writing from dictation" subtest: there was a shift of the percentile score from 58 to 74. In the "composing from dictation" subtest many uncertainties were still present. This time the AAT classification revealed 98.2% definite aphasia with an amnestic profile of slight severity. The percentile score on the Token Test was 74 (Table 1).

Nonverbal neuropsychological examination by means of (neurobehavioral) observation and standardized tests, 35 days postictal, revealed impulsiveness, slight perseverations, insufficient self-evaluation and -correction, neglect of left visual field, mild left-sided visuospatial perception and constructional difficulties, and fatigability together with declining performance within 20 to 30 min. Buccofacial and limb praxes were normal.

Repeated nonverbal neuropsychological examination, 15 months after the accident, showed distinct quantitative improvements of lassitude, intelligence, planning, learning, visuospatial perception and orientation, and visuoconstruction. Striking qualitative findings included persistent inertia, irritation about own (dys-)functioning, anger after being praised, and quite often achievement of the correct response by devious ways. Finger localization, according to Benton et al. (1983), was clearly disturbed. Ideomotor praxis of the right arm and hand, as assessed by the scheme of Poeck (1989), was normal.

Neuroradiological Findings

A preoperative cerebral CT scan appeared to be normal. Postictal it showed two hemorrhagic infarcts in the watershed areas of the right hemisphere and edema in the distribution of the middle cerebral artery (Fig. 1). Four weeks later the edema was resorbed without signs of persistent structural damage and the hemorrhagic component had disappeared. Three months postictal the watershed infarcts were sharply demarcated.

Tc-99m HMPAO SPECT, 35 days after the accident, revealed clear occipital and frontoparietal disturbances in the right cerebral hemisphere (Fig. 2).

Cerebral magnetic resonance imaging (MRI), 26 months after the stroke, showed the well-known watershed infarcts as well as lacunar infarcts in the

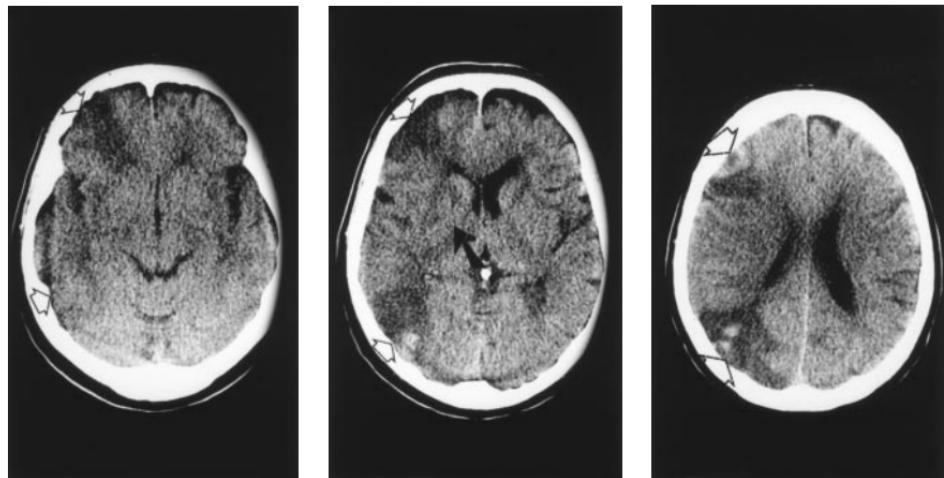


FIG. 1. Computerized tomography slices without intravenous contrast 1 day after the carotid endarterectomy showing hypodensity and swelling in the watershed area between the great cerebral artery territories of the right hemisphere (open arrow). There is some hyperdensity of hemorrhagic transformation. Also swelling and slight hypodensity in the deep territory of the medial cerebral artery (black arrow).

left medial thalamus and right pons (Figs. 3 and 4A). Retrospectively, the lacunar infarct in the left medial thalamus was also visible in the preoperative CT scan (Fig. 4B).

DISCUSSION

HSJ's clinical picture in the first week after the cerebral vascular accident can be fully explained on the basis of infarcts in the anterior as well as in the posterior watershed territories. The edema in the territory of the middle cerebral artery seems to have little importance. The SPECT images correlate with those of the CT scans. This is in agreement with the reports of Primavera and Bandini (1993), Sullivan et al. (1991), and Perani et al. (1988). It also suggests functional integrity of the left hemisphere in the postacute stage.

The improvement of the initial language disturbance, i.e., almost complete mutism (or perhaps transcortical mixed aphasia?), agrees and parallels the improvement in the initially paretic left leg. The language disturbance further evolved via moderate transcortical sensory aphasia (3 weeks postictal) to mild transcortical sensory aphasia (5 months postictal) to amnestic aphasia (12 months postictal). As a rule, recovery of "uncrossed" transcortical sensory aphasia is quite good: often by the 3rd month only anomia and mild circumlocution remain (Kertesz, 1985). The baseline neurological symptoms are best explained by posterior watershed infarct. The improvement of the nonverbal neuropsychological functions parallels the improvement of the language disturbance. Less perseverations and better executive functions

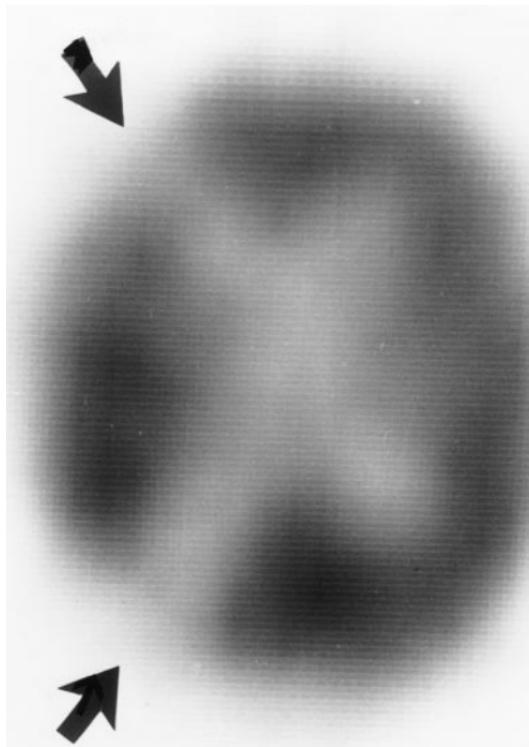


FIG. 2. A SPECT image of a Tc-99m HMPAO blood flow study showing decreased activity in the right temporo-occipital and frontal areas (black arrows).

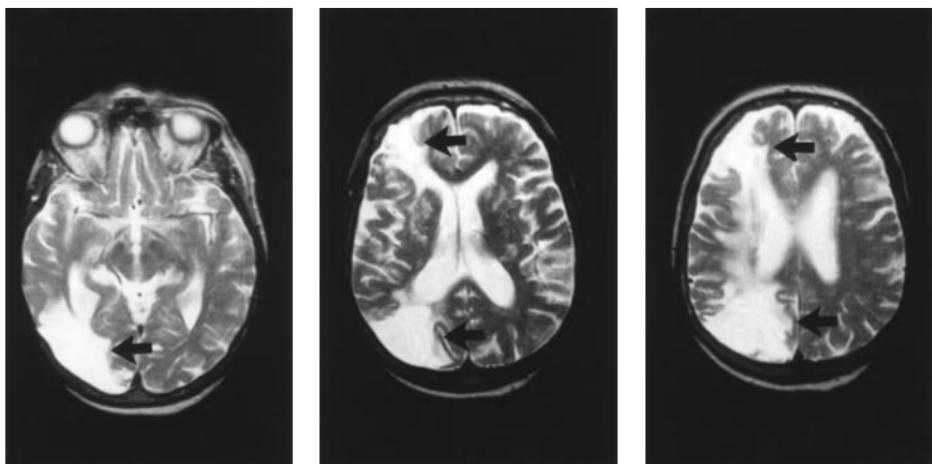
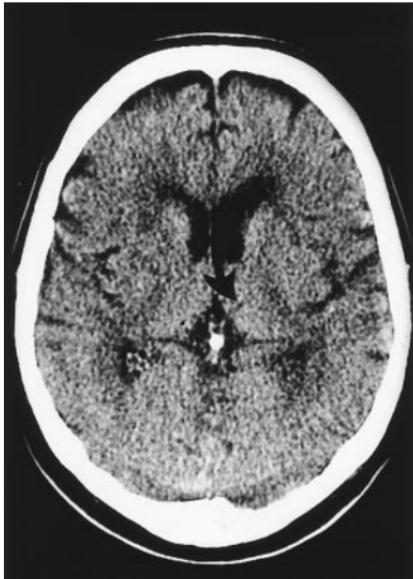


FIG. 3. Some T₂-weighted MR images (turbospin-echo 4000/90 TR/TE) showing high signal intensities in the watershed infarctions of the right hemisphere (black arrows).



(A)



(B)

FIG. 4. (A) T₁-weighted MR-image (spin-echo TR/TE 560/15) with a small area of low signal intensity medially in the left thalamus presumed to be a lacunar infarction. (B) On a preoperative CT scan the left thalamic lesion is visible as a small hypodensity. There is a difference in angulation and slice thickness with the MR scan.

(planning and self-regulation) can be interpreted as signs of improved functioning of the anterior regions of the right cerebral hemisphere (Lezak, 1995). The lasting difficulties of visuoperceptive abilities point to persistent dysfunctioning of more posterior regions. We do not have an explanation for the rapid and successful functional recovery of the anterior infarct, in contrast with the posterior infarct. Bogousslavsky and Regli (1986) have not found a difference in the short-term recoveries among their three groups of patients with watershed infarcts.

In general, lesion studies fail to demonstrate a precise correlation between neuroanatomical lesion site and specific neurolinguistic deficit (Bachman & Albert, 1988). This case report is no exception. As discussed above, the complete neurological picture can be explained by infarctions in the anterior and posterior watershed areas. According to the method of Damasio (1991), the anterior watershed area in our patient corresponds with Brodmann areas 46, 45, 9, and 6. The posterior watershed area corresponds with Brodmann areas

39, 37, 22, and 19, containing the posterior sector of the middle temporal gyrus, the angular gyrus, and the white matter underlying these cortices. In our patient, the right thalamus is spared. Although transcortical sensory aphasia is described after lesions of the thalamus (Servan et al., 1995; Alexander et al., 1989a; Graff-Radford et al., 1985), we do not believe that HSJ's lacunar infarct in the left medial thalamus has a functional meaning. First, SPECT imaging suggests functional integrity of the left hemisphere. Second, the preexisting infarction of the left thalamus on its own has not shown a preexisting language disturbance. So, our patient can be classified in the "mirror image group" of Alexander et al. (1989b), assuming there is complete lateralization of language to the right cerebral hemisphere.

Visuospatial disturbances are associated with lesions in the right cerebral hemisphere (Hamsher et al., 1992; Geschwind & Galaburda, 1985; Mesulam, 1981). This suggests that in our patient the visuospatial disturbances are "uncrossed." This is in agreement with the views of Bryden et al. (1983), Henderson (1983), and Kreindler et al. (1966). In the patient presented here, there are no indications for disturbed buccofacial or ideomotor praxes. This is in accordance with the case described in Rey et al. (1994). Buccofacial apraxia with relatively spared limb praxis is reported as well (Alexander et al., 1989b; Basso et al., 1985).

In the English and French literature scarcely 100 cases of crossed aphasia are reported (Faglia et al., 1990). According to the description of the original author(s), just one case of crossed transcortical sensory aphasia is referred to (case 3 in Carr et al., 1981). The lesion of Carr's patient is located deep in the right temporo-parietal lobe with involvement of the internal capsule, thalamus, caudate nucleus, and putamen. The language disturbance in Carr's patient 19 months after the accident is unchanged compared to 11 weeks postictal. Neuropsychologic examination is not reported. According to Alexander et al. (1989b), another two cases of crossed transcortical sensory aphasia are reported: cases 2 and 3 in Basso et al. (1985). The lesion in patient 2 is located in the inferior and middle gyrus of the right frontal lobe, partially extending to the central and postcentral gyri. The aphasia, just as the mild oral apraxia, improves completely in a few months. The lesion in patient 3 is located in the territory of the right posterior cerebral artery. Neuropsychological examination shows severe unilateral neglect and apraxia. Seven months later the aphasia, apraxia, and unilateral neglect are unchanged. Comparison with our patient is not possible because of insufficient CT-scan information needed for exact localization and insufficient congruence in the applied aphasia and neuropsychological testing.

Our case is unique regarding the location of the lesion (watershed infarcts), the type of aphasia (transcortical sensory), and the length of the evaluation. Unfortunately, the data do not permit strong conclusions regarding the lateralization of language in association with praxis and visuospatial abilities. This underlines the necessity of further documentation of such cases.

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