elderly people: relationship to longitudinal change on cognitive tests. Age Ageing 1996;25:125–129.
6. Lindeboom J. Amsterdamse Dementie Screenings test-ADS-3. Lisse, The Netherlands: Swets en Zeitlinger, 1988.
7. Shulman KI, Gold DP, Cohen CA, Zucchero CA. Clock-drawing and dementia in the community: a longitudinal study. Int J Geriatr Psychiatry 1993;8:487–496.
8. Pliskin NH, Kiolbasa TA, Towle VL, et al. Charles Bonnet syndrome: an early marker for dementia? J Am Geriatr Soc 1996;44:1055–1061.
9. Hughes CP, Berg L, Danziger WL, Cohen LA, Martin RL. A new clinical scale for the staging of dementia. Br J Psychiatry 1982;140:566–572.
10. American Psychiatric Association. Diagnostic and statistical manual of mental disorders. 4th ed. Washington, DC: American Psychiatric Association, 1994.

Disruption of residual reading capacity in a pure alexic patient after a mirror-image right-hemispheric lesion

Article abstract—A 74-year-old woman became a letter-by-letter reader after the occurrence of a left occipito-temporal hematoma. Seven months later, she suffered a second, mirror-image hematoma in the right hemisphere. After this second lesion, her residual reading capacity deteriorated dramatically in terms of both accuracy and reading latencies for words and isolated letters. Our findings support the hypothesis that the right hemisphere contributes to the residual reading capacities of pure alexic patients.

NEUROLOGY 1998;50:286–288

Paolo Bartolomeo, MD, PhD; Anne-Catherine Bachoud-Lévi, MD; Jean-Denis Degos, MD; and François Boller, MD, PhD

Pure alexia is an acquired reading disorder that occurs after posterior left-hemisphere (LH) injury. Patients may not be able to read at all, or they read words in a slow, letter-by-letter fashion. However, they may be able to perform tasks of lexical decision and semantic categorization on words they do not read. These implicit reading abilities might depend on a right-hemisphere (RH) contribution.[1-3] Coslett and Monsul[4] provided evidence for this hypothesis using transcranial magnetic stimulation, which may transiently block the neural function of the underlying brain regions. They stimulated the posterior regions of the RH or the LH of a pure alexic patient and found that stimulation of the RH but not of the LH disrupted residual oral reading. Longitudinal studies of patients with consecutive brain lesions provide another approach to test interhemispheric interactions in reading. Thus, a LH-damaged aphasic patient who was able to read lost this ability after a second RH stroke.[5] To our knowledge, there is no available report of pure alexic patients who suffered a second RH stroke. Here we describe one such patient who became alexic after a LH temporal-occipital lesion and whose residual reading capacities markedly worsened after a second, mirror-image RH lesion.

Case report. A 74-year-old homemaker, strongly right-handed according to the Edinburgh Inventory,[6] suddenly developed a reading disturbance with preserved writing in May 1995. CT showed a hematoma located across the left temporo-occipital sulcus. Goldmann perimetry showed a right paracentral scotoma, which disappeared with IV/4 test. Simple motor reaction times (RTs) to lateralized visual stimuli[7] were moderately slowed for right-sided targets, but in the range of age-matched controls for left-sided stimuli. In December 1995, the patient suffered a second, right-sided hematoma, almost symmetric to the first. The lesion was centered on the middle occipital gyrus, just posterior to the temporo-occipital sulcus (figure). After the RH stroke, the patient became achromatopsic, prosopoagnosic, and object agnosic. Goldmann perimetry showed a small central scotoma with II/4 test. Visual evoked responses with black-and-white pattern were normal for latency and amplitude. She obtained a verbal IQ of 109 on the Wechsler Adult Intelligence Scale-Revised, and correctly performed the screening test and the space perception test of a visuospatial battery,[8] but failed on all the object perception tests. On a test of line orientation judgment,[9] she obtained a corrected score of 25, well within normal limits. She produced a plausible copy of the drawing of a landscape. Her RTs to left-sided targets in the simple motor RT task did not differ from response latencies recorded before the occurrence of the second lesion.

Methods. *Word reading.* A total of 200 lower-case nouns of 4 to 8 letters in length, matched for lexical frequency, were presented free field on a paper strip without time limit. Accuracy and reading time were recorded. The word list was presented twice on separate occasions, after both the first and the second stroke.

Letter reading. The 26 letters of the French alphabet were randomly presented in lower case on paper strips. Accuracy was recorded. Reading latencies for letters were measured for our patient and her 74-year-old, right-handed husband, free from neurologic deficit. Each letter was presented at the center of a computer screen, in lower-case Trip font subtending a visual angle of about 1°30′. Subjects were instructed to name the letter as soon as it appeared on the screen and to respond as fast as possible. Naming latencies were measured from the appearance of the letter to the response onset. Stimuli remained visible until a vocal response was made or 5 seconds had elapsed. The 26-letter set was randomly presented four times for

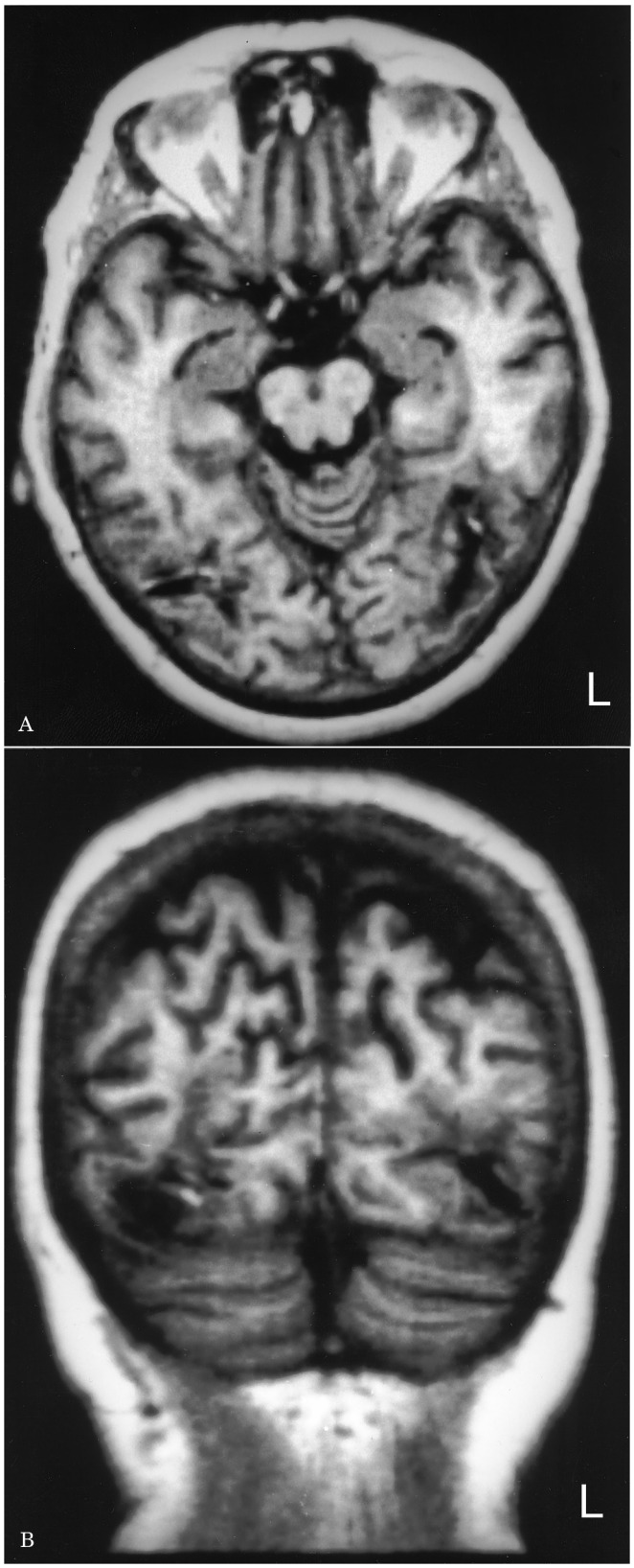

Figure. MRI. Transversal (A) and coronal (B) T1-weighted sections showing the first left hemispheric lesion and the second right hemispheric lesion.

each session. The patient and her husband performed three sessions; after her second stroke, the patient performed two sessions.

Table *Mean correct reading latencies for words (in seconds) following the first and the second stroke*

	Word length (letters)		
	4–5	6	7–8
LH lesion	2.6	3.5	4.8
LH + RH lesions	9.9	15.6	13.9

Reading tests: Initial assessment. This phase of testing took place between July and December 1995. The patient correctly read aloud 398 of 400 words (99%). She showed a linear relationship between word length and reading time ($r = 0.77$), with a slope of 0.50 seconds per additional letter ($t = 24.02$, $p < 0.0001$) (table).

She read 156 of 156 letters (100%) presented for unlimited time. On the time-limited computer presentation, she read 295 of 312 letters (94%). Her husband scored 307 of 312 correct (98%), thus performing in the same range ($\chi^2[1] = 0.41$, $p > 0.5$). However, when reading latency was considered, our patient was about 230 msec slower than her husband (patient: mean RT = 697 msec [SD 98]; control: mean RT = 464 msec [SD 31]; $t[50] = 11.54$, $p < 0.0001$). This finding suggests a subtle deficit of letter identification.[10]

Reading tests: Second assessment. Between January 1996 and March 1997, the patient underwent the same tests as before the occurrence of the RH lesion. In contrast with her near-perfect reading accuracy after the first stroke, she now scored only 156 of 400 correct (39%) with words and 22 of 52 correct (42%) with letters. Word reading latencies were significantly greater than before ($t[504] = -14.50$, $p < 0.0001$). The patient now needed 13 seconds on average to read a single word. She continued to use a letter-by-letter strategy, but now made several letter identification errors. For example, when trying to read the word *rescapé* (survivor), she took 59 seconds to say, "n . . . o . . . s . . . c . . . u . . . p . . . Moscou (Moscow)?" A word-length effect was still present ($r = 0.75$), but with the steeper slope of 2.01 seconds per additional letter ($t = 14.07$, $p < 0.0001$). Mean correct RTs for words of different lengths are displayed in the table. The patient's mean vocal RT to isolated letters was now 1,197 msec (SD 166).

Discussion. This patient with pure alexia showed a remarkable increase in her reading difficulties after a second, mirror-image lesion in the RH. The deterioration of word and letter reading performance concerned both accuracy and response time.

Our patient's increased reading latency is not a consequence of a nonspecific slowing caused by the brain lesions, as she performed in the normal range when responding to left-sided visual stimuli on a simple motor RT task. A low-level visual deficit cannot explain her impaired reading accuracy after the second stroke, given her good performance on copying drawings, on line-orientation judgments, and on the spatial subtests of the visuospatial battery. Moreover, both MRI and visual evoked potentials suggest a substantial sparing of early visual process-

ing in our patient. Thus, the deterioration of reading accuracy after the second stroke seems to indicate that the RH lesion damaged some compensatory reading mechanisms. These putative mechanisms apparently subserved both word and letter identification. After her first lesion, our patient showed a length effect on word reading latencies, possibly in relation to a strategy of serial letter identification. The persistence of a length effect after the second stroke suggests that the patient continued to employ such a strategy. However, serial letter identification was much less efficient after the second lesion, being both slower and less accurate. Our results support the claim[1-3] that the RH plays a role in the residual reading of pure alexic patients. In particular, our findings suggest that, after the LH lesion, our patient used RH-based mechanisms to increase accuracy and speed of letter and word identification, even in the absence of right-sided hemianopia. However, it is unlikely that her residual reading capacities were entirely attributable to the RH because pure alexic patients with isolated LH lesions may show a higher degree of impairment than our patient.[11] Rather, in our patient the RH seemed to cooperate with some LH residual mechanisms. Following this line of reasoning, the slowed but accurate letter identification observed after the LH lesion[10] might have resulted from a relative impairment of a LH-based letter identification mechanism and a compensatory contribution from a RH-based system for object recognition.[3] This object recognition system would process individual letters as familiar visual forms, but would not allow parallel identification of letters in words, thus explaining letter-by-letter reading. The subsequent RH stroke might have lesioned the object-recognition system; as a consequence, letter identification became error prone and much slower than before, causing in turn a deterioration of word reading. The appearance of object agnosia after the RH lesion further supports this interpretation.

Acknowledgments

We thank Dr. Victor Rosenthal for kindly providing us with test material and Dr. Eran Zaidel for stimulating discussion.

From the INSERM Unit 324 (Drs. Bartolomeo and Boller), Paris; LSCP, EHESS, CNRS (Dr. Bachoud-Lévi), Paris; and Neuroscience Department (Drs. Degos, Bartolomeo, and Bachoud-Lévi), Hôpital Henri-Mondor, Créteil, France.

Supported by the European Union grant ERBCHBGCT930344 to Dr. Bartolomeo.

Received April 17, 1997. Accepted in final form June 3, 1997.

Address correspondence and reprint requests to Dr. Paolo Bartolomeo, INSERM Unit 324, Centre Paul Broca, 2ter rue d'Alésia, F-75014 Paris, France.

References

1. Landis T, Regard M, Serrat A. Iconic reading in a case of alexia without agraphia caused by a brain tumour: a tachistoscopic study. Brain Lang 1980;11:45–53.
2. Coslett HB, Saffran EM. Evidence for preserved reading in 'pure alexia.' Brain 1989;89:327–359.
3. Coslett HB, Saffran EM. Mechanisms of implicit reading in alexia. In: Farah MJ, Ratcliff G, eds. The neuropsychology of high-level vision: collected tutorial essays. Hillsdale, NJ: Lawrence Erlbaum Assoc., 1994:299–330.
4. Coslett HB, Monsul N. Reading with the right hemisphere: evidence from transcranial magnetic stimulation. Brain Lang 1994;46:198–211.
5. Heilman KM, Rothi L, Campanella D, Wolfson S. Wernicke's and global aphasia without alexia. Arch Neurol 1979;36:129–133.
6. Oldfield RC. The assessment and analysis of handedness: the Edinburgh Inventory. Neuropsychologia 1971:97–113.
7. Bartolomeo P. The novelty effect in recovered hemineglect. Cortex 1997;33:323–332.
8. Warrington EK, James M. The visual object and space perception battery. Bury St. Edmunds: Thames Valley Test Company, 1991.
9. Benton AL, Hamsher K, Varney NR, Spreen O. Contributions to neuropsychological assessment. Oxford: Oxford University Press, 1983.
10. Bartolomeo P, Bachoud-Lévi AC, Degos JD. A hidden letter identification deficit in a pure alexic patient. Presented at the 14th European Workshop on Cognitive Neuropsychology, Bressanone, Italy, January 1996.
11. Perri R, Bartolomeo P, Silveri MC. Letter dyslexia in a letter-by-letter reader. Brain Lang 1996;53:390–407.