

Alexia without agraphia: a study of a case of verbal alexia without accompanying colour-naming defect

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Summary. A 47-year-old man developed a persistent form of alexia without agraphia as the result of a haemorrhagic intracerebral lesion in the left inferior temporo-occipital region, due to the rupture of an arteriovenous malformation. Surgical evacuation of the haematoma and excision of the malformation did not produce any modification of the reading deficit, which remained unchanged during a 4-year follow-up. The reading deficit was restricted to words and the patient was able to read only letter-by-letter, so that the whole words were reconstructed from the auditory names of the letters. So far, the disconnection explanation is the standard explanation of alexia without agraphia and the present case of verbal alexia may be regarded as being within this overall category. However, this explanation meets with unanswered questions that suggest more flexible interpretations. Neurolinguistic studies have questioned the unique character of alexia without agraphia as a clinical entity and, in contrast to the disconnection hypothesis, support the notion that the different varieties of alexia that are traditionally described represent distinctive syndromes, each with its own clinical features and pathophysiological basis. In this context, the reading properties in this case seem fully compatible with a deficit of the visual word-form system postulated by Warrington and Shallice, that is, the relatively early stage of the reading process through which a word-form or equivalent unit is attained. This system might be lateralized to the left hemisphere, as suggested by the fact that this case, like other cases of verbal alexia, had sustained damage to the left hemisphere and did not show any differential preservation of the reading of concrete words.

Key words: Alexia without agraphia – Verbal alexia – Letter-by-letter reading – Disconnection – Visual word-form system

Zusammenfassung. Bei einem 47jährigen Mann trat eine intracerebrale Blutung infolge Ruptur eines arteriovenösen Angioms der linken basalen Temporookzipitalregion auf. Dies führte zu einer persistierenden Alexie ohne Agraphie. Auch die chirurgische Ausräumung des Hämatoms und der Mißbildung bewirkte keine Veränderung der Leseschwierigkeiten, die während vier Jahren konstant blieben. Die Lesestörung beschränkte sich auf Wörter, und der Patient war lediglich fähig, Buchstabe um Buchstabe zu lesen, so daß das Gesamtwort lediglich rekonstruiert wurde. Bisher beruht die Standardklärung der Alexie ohne Agraphie auf der Diskonnectionstheorie und der vorgelegte Fall verbaler Alexie kann innerhalb dieser Kategorie gedeutet werden. Immerhin wirft

dieser Fall eine Reihe von offenen Fragen auf, welche eine flexiblere Interpretation nahelegen. Neurolinguistische Studien haben den einmaligen Charakter der Alexie ohne Agraphie als klinische Entität in Frage gestellt und sprechen – im Gegensatz zu der Diskonnectionshypothese – zugunsten der Annahme, daß unterschiedliche Arten von Alexie auch eigenständige Syndrome darstellen, jedes davon mit seinen eigenen klinischen Eigenheiten und pathophysiologischen Grundlagen. In diesem Zusammenhang scheinen die Leseeigentümlichkeiten im vorliegenden Fall kompatibel zu sein mit einem Defizit des visuellen Wort-Formsystemes, wie es von Warrington und Shallice postuliert wurde: das ist ein relativ frühes Stadium des Leseprozesses durch welches eine Wortform oder äquivalente Einheit erreicht wird. Dieses System könnte im Bereiche der rechten Hemisphäre lokalisiert sein, wofür ein Argument im vorliegenden Fall, wie auch in anderen Fällen von verbaler Alexie sprechen würde: hier war eine Läsion der linken Hemisphäre erzeugt worden, ohne daß irgend eine selektive Erhaltung der Fähigkeit zum Lesen konkreter Worte nachweisbar gewesen wäre.

Introduction

Alexia without agraphia is a well-defined syndrome in which a literate person loses the ability to read but retains the capacity to write both to dictation and spontaneously [7]. Autopsies of cases of alexia without agraphia have demonstrated the universal presence of lesions of the left occipital lobe, often including the splenium of the corpus callosum [9]. In the context of the widely accepted disconnection hypothesis revived by Geschwind [17], the importance of this double (occipital and callosal) lesion in the genesis of the syndrome has been stressed. The lesion would disconnect the left angular gyrus from its bilateral visual input impeding verbalization of the graphic symbols. In alexia without agraphia, however, there are aspects that remain difficult to explain in terms of callosal disconnection. A major obstacle in accepting this theory is that while the patients are unable to name letters or words, they can name objects, number or colours [20, 23]. Further objections have arisen from demonstration of different varieties of the reading disorder (literal, verbal, sentence, and global alexia) within the main syndrome. Neurolinguistic analyses of alexia without agraphia would seem to suggest that these varieties do not represent different degrees of severity of the same basic disorder, but rather that they represent structurally different forms reflecting significant variations in the site of causative pathology [15, 20].

This report describes the case of a right-handed man who developed a persistent form of alexia without agraphia, resulting from localized haemorrhagic damage to the ventrolateral aspect of the left occipito-temporal junction, with sparing of the corpus callosum. In this case, the reading of words was seriously impaired, while the reading of individual letters was preserved. Spelling was normal and words were deciphered letter-by-letter. This case provides further evidence that the lesions resulting in verbal alexia are localized in the left temporo-occipital region and reinforces the importance of the ventral inputs from the visual cortex in the process of reading.

Case report

A 47-year-old, right-handed man, with a grade school certificate, was admitted to the Neurological Clinic on 28 December 1978 complaining of not being able to read any longer. He had been in excellent health until the day prior to admission, when he experienced the sudden onset of a throbbing headache during lunch. In the ensuing hours the headache improved and he went out to buy a packet of cigarettes. While he was going to the tobacconist's, he wanted to read an advertising sign but was not able to do so. Then, he went into a bookshop, but every attempt to read failed. Therefore, he consulted his family doctor, who referred him to our clinic.

On admission, he was alert and oriented to person, place and time. The neurological examination showed no abnormalities, except for right homonymous upper quadrantanopsia. Visual acuity was normal. The patient was given a formal Language Examination Test (Association for the Development of Neuropsychological Research, Milan, 1974). He showed a specific impairment in word reading, but his ability to read individual letters was preserved, as was his ability to spell (a more detailed reading examination is reported below). His writing capacity was unimpaired. Handwriting was fluid and legible without elisions or substitutions and the patient had no difficulty in writing sentences from dictation, although a few minutes later he could not read without errors what he had written before. He was able to repeat fluently words spelled out by the examiner and to decipher, when blindfolded, single letters and common four-to five-letter words assembled with plastic letters, tracing each of them by both the right or the left index finger. His spontaneous speech was fluent, prosodic, articulate, without any kind of errors. Repetition of words, non-words, and sentences was excellent and the verbal comprehension of even complex commands was immediate. He had no difficulty in naming colours, objects and pictures of objects presented from an unusual view. Number reading was normal and mathematical basic operations were performed verbally without difficulty. Finger recognition as well as right-left discrimination were normal. There was no kind of apraxia. On discrimination of unfamiliar faces he scored 49 out of 51. On the Token Test he scored 32 out of 36. On the Wechsler Adult Intelligence Scale (WAIS), the patient obtained a verbal IQ of 100 and a non-verbal IQ of 82, the latter being affected mainly by slowness in time-dependent tasks; the full-scale IQ was 92. On the picture completion and picture arrangement subtests of the WAIS (the stimuli are complex visual scenes) he scored 9 and 7, respectively. Poppelreuter's figures were recognized within the normal average range and the patient scored normally on a test of discrimination of embedded figures. On the Wechsler Memory Scale (WMS) he scored 82. Digit span was 6 forward and 4 backward in both the WAIS and the WMS.

To clarify further the patient's selective impairment of word reading, an extensive reading examination was devised. In different sessions, the patient was given a large sample of controlled stimuli to read. The considered dimensions of the stimuli were taken from Cornoldi [12], Roncato [27], and Bartolini et al. [5]. All the stimuli (i.e. words and non-words) printed on a white sheet in standard lower-case letters (pica, 5 mm high) were presented one at a time, free field, and without time pressure. On occasions words and non-words were presented in blocks, otherwise randomly intermixed. A set of stimuli was also presented in CaSe AlTernAtiOn (i.e. lower and upper case intermixed) and a set of script-like stimuli (Letraset Juliet, 7.6 mm). All the stimuli were arranged for the following different tasks: (1) *letter reading*: reading aloud (or pointing at) a marked letter confused in an array of different letters (e.g. a n i p f); (2) *word reading*: reading aloud concrete-abstract, high-low frequency words of 5–11 letters (total stimuli = 280); (3) *non-word reading*: reading aloud non-words of different length, all easily pronounceable (total stimuli = 120); (4) *"part of speech" reading*: reading aloud nouns, adjectives, verbs, and functors, all rated for frequency (total stimuli = 80 + 60 + 60 + 100); (5) *lexical decision*: when given a word-non-word pair, pointing at the word or non-word (total stimuli = 60); (6) *comprehension*: matching words to corresponding pictures (48 words plus 48 pictures); (7) *semantic correlation*: matching words semantically, related in a multiple-choice task (30 words semantically related to 30, plus 120 distractors).

To summarize briefly the results of the reading examination, the patient made no errors on the letter reading, lexical decision, comprehension, and semantic correlation tasks. He always knew which was a word and which not. His ability to match words with pictures was perfect as well as his appreciation of the semantic relationship. On the other hand, the patient's word reading was very laborious, marked by hesitations and abnormal slowness. He tended to read silently in the correct order single letters of a given word, to pronounce the letter sequence with frequent pauses, and then to pronounce the whole word as a final unit. As shown in Table 1, a clear effect of the word length was present (longer words taking more time to be pronounced than shorter words) and the reading of non-words took much more time than the reading of words. The reading of stimuli presented with case alternation took the same amount of time as reading of lower-case stimuli. A slight difference was found only for script-like stimuli, which took a little more time to read than printed words. The patient, who had been reported to be a normal reader before the accident, also made a number of errors, most often resulting in single phonemic substitutions (e.g. MARE → MORE; FORBICE → FERBICE; SAPONE → SAPENE; ORSO → ORZO; ALLEANZA → ALLEONTA). Apart from his paralexia, he very rarely made other types of errors that seemed to be visual in nature (e.g. QUANTITA' → VANITA'; SEZIONE → SELEZIONE; DEVIAZIONE → DELUSIONE). The types and distribution of the errors according to the syntactic classes of the stimuli are shown in Table 2. As can be seen, the errors (66 errors out of 620 stimuli) were randomly distributed across the different classes of stimuli. The patient was aware of all the mistakes and able to correct 65% of them immediately. No concrete-abstract or high-low frequency effect was present. No "part of speech" effect was detected. No relationship was observed between errors and the imagery value or meaningfulness of the stimuli. The errors rate in reading non-words was not different from the errors rate in reading words, the only difference in reading words and non-words being the different

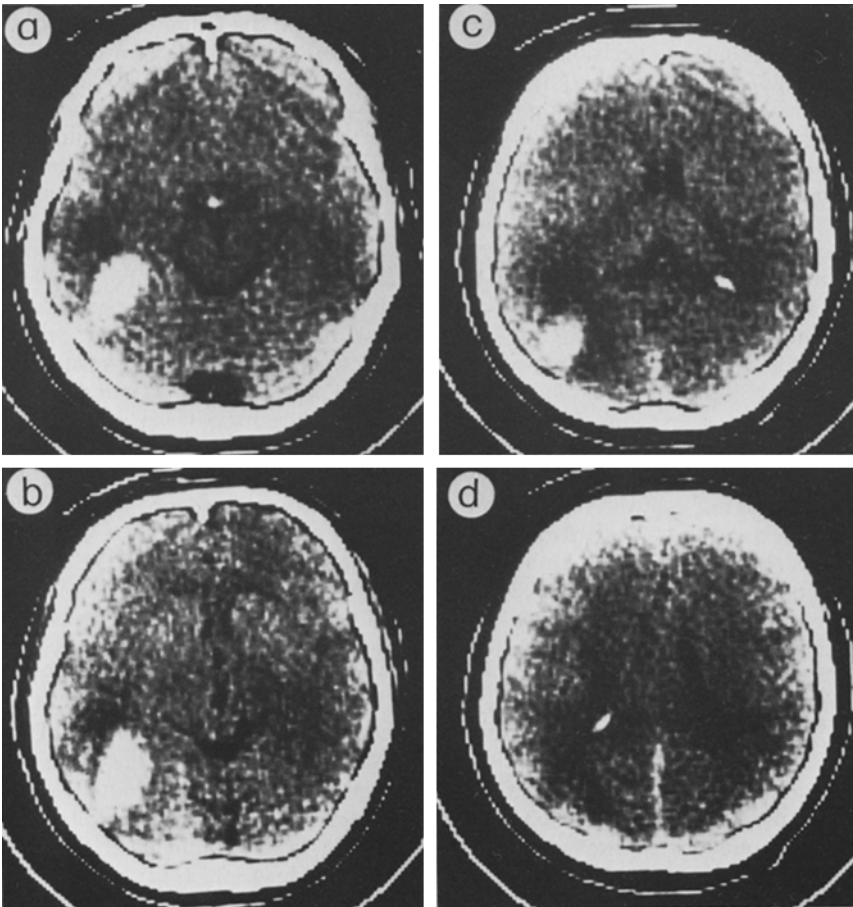
Table 1. Time (in seconds) taken to read a complete list of words and a list of non-words of different length (number of letters). Half of the words were high-frequency stimuli and half low-frequency stimuli

Number of stimuli (words and non-words)	10	20	20	20	10
Number of letters for each stimulus	4	5	6	7	8
Time to read the list of words (s)	189	279	470	580	610
Time to read the list of non-words (s)	248	380	710	930	—

^a 10 words and 10 non-words

Table 2. Type and distribution of errors for different syntactic classes and for non-words. In *brackets*: errors immediately corrected. Total errors out of 500 meaningful stimuli = 48 (9.6%), of which 34 (70.8%) were immediately corrected. Total errors out of 120 meaningless stimuli = 18 (15%), of which 9 (50%) were immediately corrected. Total errors out of 620 stimuli = 66 (10.6%), of which 43 (65.2%) were immediately corrected

Stimulus	Number and type of errors			
	One-letter substitution	Two-letter substitution	Other	Total
Words (280)	16 (16)	7 (5)	3 (1)	26 (22)
Non-words (120)	8 (4)	6 (4)	4 (1)	18 (9)
Adjectives (60)	4 (4)	2 (1)	—	6 (5)
Verbs (60)	2 (1)	2 (—)	3 (1)	7 (2)
Functors (100)	3 (1)	—	6 (4)	9 (5)

**Fig. 1a-d.** Four consecutive slices from inferior (a) to superior (d) from the pre-operative non-enhanced CT scan. The three slices of the low ventricular series (a-c) show a high-density lesion (in the range of blood) in the left inferior temporo-occipital region. In the highest slice of the low ventricular series (c) and in the lowest slice of the high ventricular series (d), the splenium of the corpus callosum appears to be unaffected

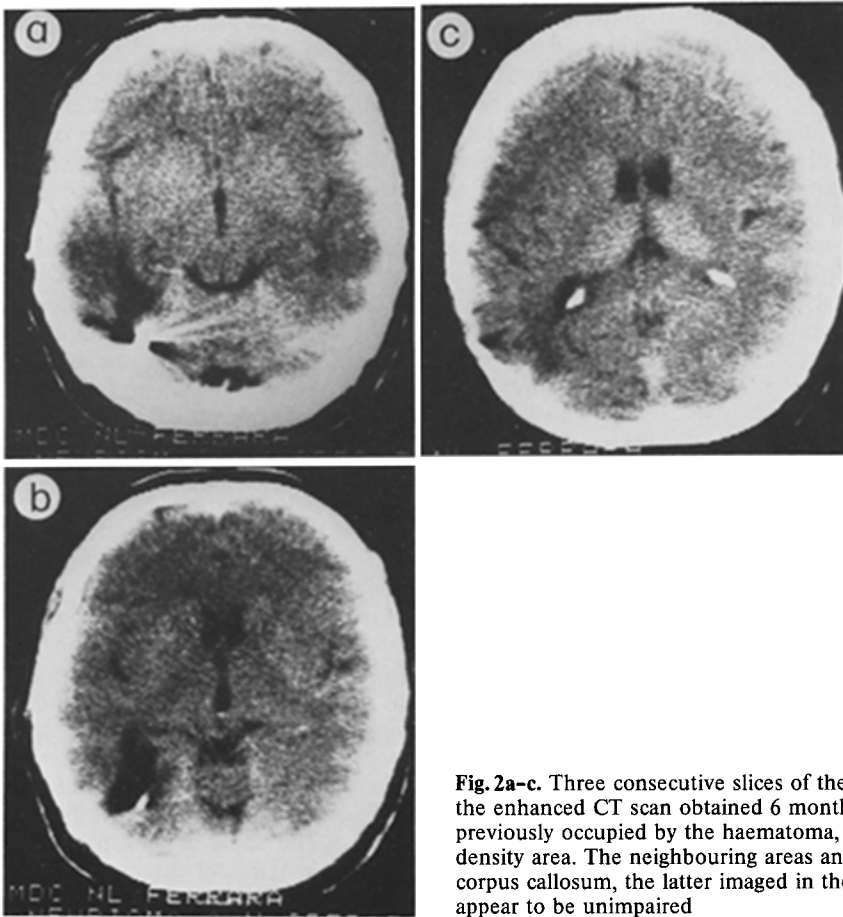


Fig. 2a-c. Three consecutive slices of the low ventricular series from the enhanced CT scan obtained 6 months after surgery. In the region previously occupied by the haematoma, there is evidence of a low-density area. The neighbouring areas and the splenium of the corpus callosum, the latter imaged in the highest slice (c), appear to be unimpaired

lengths of time needed to read them (much longer for meaningless stimuli). The patient was only able to read the shortest sentences owing to his often being depressed as a result of fatigue to read, the continuous stream of attention and the effort needed to retain what he had been reading.

The findings from routine laboratory tests were normal. A non-enhanced CT scan was indicative of a haemorrhagic intracerebral lesion, surrounded by an oedematous area, in the region of the left occipitotemporal junction (Fig. 1). According to the "sequential CT anatomy" data proposed by Gado et al. [16], the lesion was shown in the three slices of the low ventricular series (Fig. 1a, b, c). The lesion damaged the inferior aspect of the left temporal lobe in the part contiguous to the lateral border of the cerebellum (Fig. 1a) and extended into the occipitotemporal white matter reaching, on the lateral aspect of the hemisphere, the posterior part of the inferior temporal gyrus (Fig. 1b) and the contiguous part of the lateral occipital gyrus (Fig. 1b, c). The lesion was surrounded by an oedematous area, with evidence of mass-effect, resulting in obliteration of the homolateral occipital horn. In the highest slice of the low ventricular series (Fig. 1c) and in the lowest slice of the high ventricular series (Fig. 1d), the latter showing no evidence of the lesion, the splenium of the corpus callosum appeared to be unaffected. Intravenous contrast did not produce any modification.

Percutaneous arteriography of the left carotid and retrograde arteriography of the left brachial artery showed filling of thin opacified angiomatous vessels and of a wide arteriovenous shunt in the left inferior temporo-occipital region. The arteriovenous malformation was supplied from vessels arising from

both the inferior temporal branch of the cerebri posterior artery and the posterior temporal artery of the Sylvian system.

Surgical treatment was then decided upon, and 7 days after admission an osteoplastic craniotomy was carried out in the left parieto-temporo-occipital region. A mild expansion of the left posterior inferotemporal region extending to the bordering occipital region was noted. After cortical incision, a haematoma cavity appeared that extended deep into the subcortical white matter. The evacuation of the haematoma provided the exposure of a small arteriovenous malformation in the inferior wall of the haematoma. The feeder of the malformation was clipped and the malformation was totally excised.

During the initial postoperative period, the patient showed only a mild increase of his visual defect. Verbal alexia remained unchanged and no additional neurological sign was detected. Subsequently, the visual defect contracted, and 2 weeks after surgery it had returned to its preoperative level. The patient was discharged on 3 February 1979.

In the course of the following 4 years the patient's visits to the neurological clinic were frequent and regular. On these occasions, no significant change was observed in either the verbal alexia or the right upper quadrantanopsia.

Six months after leaving hospital, an enhanced CT scan was carried out, which demonstrated a low density area in the region previously occupied by the haematoma. As in the preoperative CT scan, the lesion was shown in the three slices of the low ventricular series (Fig. 2), but was especially evident in the lowest (Fig. 2a) and intermediate (Fig. 2b) slices. Thus, the lesion was clearly localized in the region corresponding to the left occipitotemporal junction. The residual postoperative

lesion appeared to be well defined, and it was not surrounded by oedema. There was no evidence of pathology in the neighbouring areas, particularly in the splenium of the corpus callosum.

The most recent neuropsychological examination, in October 1982, showed no significant modification of the alexia; he had even developed a useful strategy for reading based on spelling. When the patient was given a written polysyllabic word, he could only read it after a pause. He repeated each letter to himself, then joined them into a complete word. His capacity to read bisyllabic words was consolidated, as was the capacity to read and to understand very short phrases.

Discussion

The case reported in this study clearly corresponds to those previously described under the terms “verbal alexia” [15, 20], “verbal alexia-simultanagnosia” [24, 26, 34], and “word-form dyslexia” [33]. The patient was unable to read or grasp words globally, but his ability to read or identify single letters was preserved. He was able to read only letter-by-letter, so that the whole word was reconstructed from the auditory names of the letters, presumably relying on his intact spelling skills. Reading was very slow and laborious and attempts to read more quickly were vitiated by paralexical errors, most often resulting in single phonemic substitutions. The time taken to read words was clearly related to their length, and reading script was slightly more laborious than reading print. His ability to name visually presented objects, numbers or colours was intact and the memory span did not account for any memory-deficit-sustaining errors in reading. On CT scan, the patient had a unilateral left-sided lesion in the inferior temporo-occipital region, leaving the splenium and the primary visual cortex intact. The optic radiations were partly disrupted, as suggested by the right superior quadrantanopsia.

So far, the disconnection explanation is the standard explanation of alexia without agraphia [9] and many view verbal alexia as being within this overall category [8, 19, 31]. In the context of the disconnection hypothesis, the different forms of alexia (literal, verbal, and global alexia) that are traditionally described are interpreted as points in a continuum, depending on variations in the intensity of the same basic disorder [8]. In all cases, the underlying pathology should disconnect the left angular gyrus from its bilateral visual input, impeding verbalization of graphic symbols. This hypothesis, however, meets with a number of problems that have not yet been understood satisfactorily. A persisting problem is how to explain the fact that while the patients are unable to name words, they can name objects, numbers or colours [3, 20, 23]. In Geschwind's opinion [17], object naming and number reading might be preserved because visually presented objects and numbers arouse tactile associative activity in the brain, leading to excitation of anterior areas in the right hemisphere, which is then transmitted to the left angular gyrus through callosal fibres anterior to the splenium. This is regarded as an ad hoc explanation [20], which is difficult to reconcile with recent observations on the nature of the callosal code [10, 23]. On the other hand, this explanation does not account for the preserved ability to name colours in several cases of alexia without agraphia, since colours, like words, have verbal associations but, unlike objects, no tactile ones [18]. Some authors [2, 13, 19] have related this occasional preservation of colour naming in alexia without agraphia to the preservation of the dorsal aspect

of the splenium; as a consequence, the view has emerged that while colour naming is served by both the dorsal and ventral outflow from the visual areas to the left angular gyrus, reading is served by the ventral outflow only [2, 19]. This latter view makes the anatomical location observed in our case of verbal alexia without accompanying colour naming defect fully compatible with the disconnection hypothesis. In our case, as in other published cases of verbal alexia [1, 20–22, 24, 30, 31], the reading disorder resulted from localized damage to the left inferior temporo-occipital region. Although the splenium was thought to be intact, the lesion was in the precise location to disrupt both the inferior association tracts from the left visual association areas to the left angular gyrus and the inferior callosal fibres coming from the right visual areas as they pass from the splenium to the dominant hemisphere, producing the same effect of the classical splenio-occipital lesion. However, even though this explanation appears plausible from an anatomical point of view, there remains the problem of why the reading failure should be restricted to word identification only. Within the disconnection hypothesis [8], letters might be spared because they would represent less complex visual stimuli and, therefore, would be handled better than words by the damaged transmission pathways. This assumption has been questioned by Warrington and Rabin [32], who showed that short-term storage of letter strings in patients with unilateral cerebral lesions imposes a greater information load than a word of the same length. More recently, describing two personal cases of verbal alexia caused by unilateral left-sided lesions, Warrington and Shallice [33] concluded that the dissociation between letter span and word reading makes an explanation of spelling alexia in terms of the classical disconnection hypothesis most implausible.

Throughout the history of studies on alexia, arguments have been provided that verbal alexia is a component element of the syndrome of simultanagnosia, i.e. the failure of the capacity to comprehend the “wholes” though the capacity to name or identify the single parts is preserved [24, 26, 34]. In our case of verbal alexia no formal test of simultanagnosia was performed, but the results on both the picture completion and picture arrangement subtests of the WAIS, together with the normal performance on Poppelreuter's figures and on the embedded figures indicate that complex picture interpretation was relatively unaffected. A similar dissociation between letter-by-letter reading and piecemeal perception of complex scenes has recently been observed by Warrington and Shallice [33], who concluded that the status of simultanagnosia as a syndrome must be questioned.

During the last two decades, new impetus for research on alexia has been provided by application of neurolinguistic models. In general, the neurolinguistic studies support the notion that reading is a multistage process and that alexia without agraphia, like alexia with agraphia and aphasic alexia, may be dissociated according to linguistic characteristics into several distinctive syndromes, each with its own clinical features and pathophysiological basis [4, 11, 15, 20, 25, 28, 29]. Within this approach, the idea has emerged that reading involves at least two processes, which must be distinguished at the perceptuovisual level as well as at the linguistic level [6]. The first process (*phonological reading process*) would be analytical at the visual level and phonological at the linguistic level, while the second process (*non-phonological or lexical reading process*) would be global at the visual level (the different letters of a word are grasped simultaneously and globally as a unit) and lexical or semantic at the linguistic level [6].

In our patient, words were read analytically, letter-by-letter, and non-words, which should be read by means of the phonological reading process only [14], were read almost as efficiently as words. Semantic errors were not observed and variables such as frequency, concreteness and "parts of speech" did not influence the reading performance. This would suggest that his phonological processing was intact and that he was suffering from a disturbance of the non-phonological reading process, presumably at a level prior to the semantic stage. In other words, the properties of the reading disorder in our case seem compatible with a deficit of the visual word-form system recently postulated by Warrington and Shallice [33], that is the stage between early visual processing and semantic processing, through which a visual word-form is attained. This system might be lateralized to the left hemisphere [33], as suggested by the absence of any differential preservation of the reading of concrete words and by the fact that many patients with letter-by-letter reading had sustained damage to the left hemisphere, as did the patient described in this study.

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