

## Chapter 2

# The Role of Cognitive Neuropsychology in Clinical Settings: The Example of a Single Case of Deep Dyslexia

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### The Role of the Neuropsychologist

In this chapter, we first define the role of a cognitive neuropsychologist within the diagnostic team,<sup>1</sup> and then exemplify it by means of a case report describing the cognitive performances of a stroke patient presenting with deep dyslexia. The purpose of this chapter is to illustrate the overhauling of mere clinical labels to reach the objective of identifying the spared and impaired cognitive processes as depicted by the relevant theoretical models of mental functions. Detecting the range of spared cognitive processes is not simply a matter of completeness. Rather, it is an integral aspect of the neuropsychological diagnostic procedure. Once a deficit has been ascertained by means of reliable and standardized instruments, a hypothesis is usually formulated to account for it. To consolidate such diagnostic hypothesis requires additional supporting evidence, but a solid diagnostic confirmation comes from attempts to falsify the postulated hypothesis.

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<sup>1</sup> The introductory text of this chapter capitalises on a previous publication by the same authors: Cubelli and Della (2011).

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The etymological meaning of term diagnosis is “to know thoroughly” (by scrutinising signs and symptoms). Diagnosing implies two consequent yet independent processes: to identify a disease and to find its cause(s). Suppose that a person with abrupt language problems seeks help from a general practitioner who acknowledges the deficits and sends her for neurological consultation. The neurologist, informed by a series of clinical and instrumental exams, confirms the presence of a deviant behaviour and formulates a possible diagnosis identifying the disease, for instance minor left hemisphere ischemia. In parallel, the language impairment needs to be identified, quantified and interpreted. The outcome of off-the-shelf psychometric assessments will assist in determining that this patient’s language difficulties could be labelled as Broca’s aphasia, which is a shortcut description of her impairment. The aim of a skilled neuropsychologist though is to investigate the cognitive causes of this impairment, which can be multifarious (Mohr et al. 1978; Caramazza 1984). Limiting the examination to offering a tag would only attain the scope of corroborating the problem as lamented by the patient and her carers, perhaps by labelling it more eloquently, like a doctor diagnosing a knee pain as *gonalgia*.

In sum, cognitive neuropsychologists are called to investigate the impairments of cognitive processes following brain damage and to account for the observed patterns of spared and impaired abilities “in terms of damage to one or more components of a theory or model of normal cognition” (Ellis and Young 1995, p. 4). In turn, this refined diagnosis consents to take an informed decision on whether or not to initiate a cognitive treatment and, if so, to make precise the targets and the working hypotheses of rehabilitation programmes.

Neuropsychologists use relatively simple tasks, such as reading aloud, drawing figures, recognising objects, or memorising lists of words, and are equipped with pre-packed test batteries. The apparent simplicity of the neuropsychologist’s trademark instruments is deceptive. The core competence of a neuropsychologist is not solely to administer the tests (which could be presented by different professionals or by trainees), but to plan the individual assessment, to refine the testing programme, to decode the findings, and to unravel the observed pattern of performance. Central to their remit is the *interpretation* of the outcome from such tests, based on both accuracy scores and the qualitative analysis of errors. Like a radiologist who could carry out a scan, but whose main chore is to interpret it, the neuropsychologist is asked to derive hypotheses on the patient’s cognitive functioning. Hence, the diagnostic process should not be merely applying gross clinical labels (i.e. Broca’s aphasia, dysexecutive syndrome, unilateral neglect and episodic amnesia), but to identify the damaged component(s) of the cognitive processes in individual patients. This approach would of course require considerable time per patient asking for a different organization of the services offered. It would also entail a greater cultural and political appreciation of the relevance of the professional role of the neuropsychologists in diagnosing and managing individual issues posed by patients.

## *The Neuropsychological Interpretation*

A single error is per se opaque. The ambiguity of single errors needs to be disentangled considering the overall pattern of spared and impaired abilities and their matching onto the relevant cognitive model.

It is paramount to distinguish between error classification and their interpretation. Take a patient who reads the word “beep” as “peep”; this error could be classed as letter substitution, but it could be interpreted by means of six different accounts. The error could be (i) perceptual ( $b \rightarrow p$ ), revealing a problem in coding the spatial orientation of the letter shape, which will also involve non-orthographic stimuli; (ii) orthographic ( $\langle B \rangle \rightarrow \langle P \rangle$ ), due to a deficit in processing the letter identity, specific to reading tasks; (iii) lexical semantic (beep  $\rightarrow$  peep), because the stimulus and response pair include two semantically similar words; (iv) phonological ( $/b/ \rightarrow /p/$ ), involving one distinctive feature, with a voiced stop consonant replaced by its unvoiced cognate, due to a phonetic deficit which would be apparent also in spontaneous speech; (v) attentional (beep  $\rightarrow$  -eep), due to a defective coding of the beginning letter, as in neglect dyslexia; (vi) contextual (xxxP  $\rightarrow$  PxxP), a sequential error called “retrograde assimilation or anticipation” (Lecours and Lhermitte 1979), due to a deficit in planning the correct phonological sequence in short-term memory. The ambiguity of single errors needs to be disentangled considering the overall pattern of spared and impaired abilities and their matching onto the relevant cognitive model.

The labelling of an error, like “letter substitution,” derives from agreed mutually exclusive categories, that is, a given response could be listed under one, and one only, error category. However, this kind of errors could be interpreted by means of several different accounts. Similarly, errors which can be classed differently may derive from the same underlying problem. Consider, for instance, phonemic paraphasias (e.g. table  $\rightarrow$  pable) and formal paraphasias (e.g. table  $\rightarrow$  cable), whereby the response is a nonword and a word, respectively. In most cases, these two types of error reflect the same output disorder (see Blanken 1990).

Classing an error does not imply its interpretation, which instead ought to be vetted against the full assessment and nested within verified cognitive models. A naming error, e.g. tiger  $\rightarrow$  lion, classed as semantic paraphasia, does not necessarily imply the impairment of semantic knowledge, rather it could be traced to a pre-semantic deficit, as in associative visual agnosia, or it could result from a post-semantic deficit, as in anomic aphasia (Caramazza and Hillis 1990a). The way to disentangle the origin of a given error is to both compare performance in different tasks and to consider the frequency of other error types. In case of semantic paraphasias in picture naming tasks, the deficit would be pre-semantic if the naming performance was within the normal limits with stimuli presented in other sensory modalities (haptic or verbal) and other errors were mainly characterised by visual paraphasias (i.e. the name of a visually similar objects; e.g. apple  $\rightarrow$  ball). In contrast, the deficit would be post-semantic if the semantic errors were present in

all naming tasks independently from the sensory modality of stimulus presentation and the most frequent errors were circumlocutions (e.g. camel → the animal with humps...in Egypt). Finally, the deficit could be semantic in nature. In this case, semantic errors should be coupled with “don’t know” errors and observed in all tasks requiring the access to semantic knowledge, including nonverbal tests, like drawing from memory. Only transcoding tasks, like repeating, reading or writing nonwords, should be errorless.

## ***Psychometrics Is Not All***

Good tests are furnished with norms relative to validated cut-off scores derived from a reference sample, which allow the identification of performances that deviate more than 2 standard deviations from psychometric values like the median, or fall below the 5th percentile. These arbitrary criteria can give rise to false positives or false negatives, which could be avoided by acknowledging that a poor performance does not necessarily equate to a pathological one.

To assess a given cognitive function one single test cannot suffice, as the test–function correspondence is weak. The Token Test (De Renzi and Faglioni 1978) is recognised as a good test to assess language comprehension, yet it includes only a limited number of lexical units (names of shapes, sizes and colours). It does not assess linguistic processes like inferential knowledge (“I heard a dog” conveys the information that an unknown and unseen dog is barking far from me) or the assignment of thematic roles (the role of agent is assigned to nouns occupying pre-verbal and post-verbal positions in active and passive sentences, respectively). Moreover, there are several different ways to fail the Token Test. A patient may be inaccurate, not because of aphasia, but because of colour agnosia or working memory problems in binding shapes to colours, or deficits in keeping track of the word sequence.

Moreover, the validity of a test should not be taken for granted. A test might not assess exactly what it was devised for, for instance because of faulty selection of the stimuli. The Judgement of Line Orientation Test (Benton et al. 1978), widely assumed to detect selective visuospatial deficits in right-hemisphere-damaged patients, is biased by an uneven distribution of the stimulus lines, which are easier to discriminate in the left space thus favouring left-brain-damaged patients who tend to be more accurate in processing ipsilateral information (Treccani et al. 2005).

## ***Structure of the Neuropsychological Assessment***

A sound neuropsychological assessment should entail separate but intertwined steps. After an interview aimed at gathering a targeted personal and clinical history and a screening phase, whereby a comprehensive battery of brief tests (Brazzelli et al. 1994; Dudas et al. 2005) is given, a full-blown neuropsychological examination is delivered based upon the information gleaned with the preliminary steps. Here, the

neuropsychologist’s tools are tests or batteries geared at investigating the presence and the severity of specific disorders (Wilson et al. 2008; Huber et al. 1984), by means of which one could attribute clinical labels. The diagnostic process should not stop at this stage, as clinical labels cover a wide range of cognitive disturbances lumped together under a syndromic umbrella. To identify the precise locus of cognitive impairment is the objective of the latter step, in which the neuropsychologist should use experimental tests, culled from the literature, or even devised ad hoc.

Case Report

BR (fictitious initials), a right-handed Italian craftsman with 5 years of schooling, was 49 years old when he suffered a left middle cerebral artery stroke. A series of neuroimaging investigations disclosed a large fronto-temporo-parietal ischemia. At admission, he presented with right paresis and non-fluent aphasia.

Eight months after his stroke, he underwent a comprehensive neuropsychological assessment (see Table 2.1) which demonstrated oral and ideomotor limb apraxia (Spinnler and Tognoni 1987) but no signs of visuospatial neglect, tested by means of tasks requiring cancellation of segments (Albert 1973) or target objects (Gauthier et al. 1989; Wilson et al. 1987), drawing geometrical figures (Spinnler and Tognoni 1987) and line bisecting (Halligan and Marshall 1989). He obtained the age and education adjusted score 20/36 on the Raven’s Coloured Progressive Matrices, indicating a performance within the normal range (Basso et al. 1987). The output of the Italian version of the AAT (Luzzatti et al. 1991) confirmed the diagnosis of Broca’s aphasia. His language abilities were further investigated by means of the BADA (a battery for the assessment of aphasic disorders, Miceli et al. 1994; (see Table 2.2) In particular, tasks assessing language auditory input, phonemic discrimination, lexical decision and word–picture matching tasks were at, or near, ceiling. His language was non-fluent, his performance in the picture-naming task was good but characterised by some semantic errors (e.g. microfono [microphone] → voce [voice]) or agrammatic circumlocutions (e.g. serratura [lock] → buco [hole] ... chiave [key]). His reading abilities were poor and characterised by semantic

**Table 2.1** BR’s neuropsychological assessment. The asterisk indicates scores below age and education adjusted cut-off point corresponding to the inferential lower 5th percentile of the population

| Test                                  | Range | Cut-off | Raw score | Adjusted score |
|---------------------------------------|-------|---------|-----------|----------------|
| Token Test                            | 0–36  | 29      | 13        | 14*            |
| Raven’s Coloured Progressive matrices | 0–36  | 18      | 17        | 20             |
| Copying of Geometrical Figures        | 0–14  | 7.75    | 10        | 9.5            |
| Imitation of oral movements           | 0–20  | 16      | 1         | 0.75*          |
| Imitation of limb movements           | 0–20  | 16      | 15        | 15.75*         |
| Corsi spatial span                    | 0–10  | 3.5     | 2         | 1.5*           |

**Table 2.2** BR’s performance on BADA, a battery for the assessment of aphasic disorders

| Subtests                    | Correct responses (%) |
|-----------------------------|-----------------------|
| Phonological discrimination | 60/60 (100)           |
| <i>Oral repetition</i>      |                       |
| Words                       | 17/45 (38)            |
| Nonwords                    | 19/36 (53)            |
| Auditory lexical decision   | 76/80 (95)            |
| Visual lexical decision     | 37/80 (46)            |
| Auditory word comprehension | 38/40 (95)            |
| Visual word comprehension   | 34/40 (85)            |
| <i>Oral naming</i>          |                       |
| Objects                     | 25/30 (83)            |
| Actions                     | 11/28 (39)            |
| <i>Reading</i>              |                       |
| Words                       | 16/92 (17)            |
| Nonwords                    | 0/45 (0)              |

paralexias (e.g. mandorla [almond] → fiore [flower]) which are pathognomonic of deep dyslexia (Coltheart et al. 1980).

Deep dyslexia is a multi-componential syndrome resulting from heterogeneous functional deficits. Accordingly, we have further assessed BR to make precise the cognitive loci of his reading impairment. In a series of testing sessions, he was requested to read aloud a total of 1265 words and 192 nonwords. BR was unable to read nonwords and had serious difficulties in reading words (see Table 2.3).

Detailed scrutiny of subgroups of items from the list of words allowed us to further analyse the effects of relevant linguistic variables. BR read better concrete (25/92, 27%) than abstract (1/92, 1%) words and read better nouns (10/70, 14%) than verbs and grammatical functors (0/140). However, his superiority in reading high-frequency words (21/138, 15%) versus low-frequency words (13/138, 9%) was not significant (chi-square=0.14, ns). The presence of the lexical effect and semantic errors together with the observation of the concreteness effect and the grammatical class effect are indicative of the diagnosis of deep dyslexia (Shallice 1988, Chap. 5; Coltheart et al. 1980). The lack of a significant frequency effect is not surprising as this effect has not been consistently observed in cases of deep dyslexia, since its first description (case GR, Marshall and Newcombe 1966).

Reading errors were classified according to the categories identified by Coltheart et al. (1980, Table 6). These included semantic errors, when stimulus and response share semantic properties but differ orthographically, and visual errors which define

**Table 2.3** BR’s overall performance on word and nonword reading

|                   | Words ( <i>n</i> =1265) | Nonwords ( <i>n</i> =192) |
|-------------------|-------------------------|---------------------------|
| Correct responses | 164 (13 %)              | 0                         |
| Omissions         | 177 (14 %)              | 57 (29.7%)                |
| Errors            | 924 (73 %)              | 135 (70.3 %)              |

**Table 2.4** Paralexias produced by BR subdivided according to error categories

| Error type             | <i>n</i> (%) | Examples                                                                                                                                                    |
|------------------------|--------------|-------------------------------------------------------------------------------------------------------------------------------------------------------------|
| Semantic               | 55 (5.9)     | finestra [ <i>window</i> ]; → porta [ <i>door</i> ]; ieri [ <i>yesterday</i> ] → domani [ <i>tomorrow</i> ]                                                 |
| Visual                 | 481 (52.1)   | penna [ <i>pen</i> ] → pane [ <i>bread</i> ]; forma [ <i>shape</i> ] → forbici [ <i>scissors</i> ]                                                          |
| Visual and/or semantic | 35 (3.8)     | satellite [ <i>satellite</i> ] → stella [ <i>star</i> ]; corvo [ <i>crow</i> ] → cornacchia [ <i>carrion crow</i> ]                                         |
| Visual-then-semantic   | 63 (6.8)     | cabina → [ <i>hut</i> ] → fucile [ <i>gun</i> ] via carabina [ <i>rifle</i> ]; domanda [ <i>question</i> ] → donna [ <i>woman</i> ] via uomo [ <i>man</i> ] |
| Morphological          | 77 (8.3)     | dente [ <i>tooth</i> ] → dentista [ <i>dentist</i> ]; posto [ <i>place</i> ] → posti [ <i>places</i> ]                                                      |
| Unclassified           | 213 (23.1)   | senza [ <i>without</i> ] → sacchi [ <i>sacks</i> ]; genere [ <i>gender</i> ] → gelato [ <i>ice cream</i> ]                                                  |

responses containing at least 50 % of the stimulus letters without sharing semantic relation with it. When responses were both visually and semantically related to the stimulus, the errors were classed as visual and/or semantic. Following the examples (sympathy → orchestra assuming the mediation of symphony) given by Marshall and Newcombe (1966), errors were classed as visual-then-semantic when a presumed, not overt, visual error preceded a semantic conversion. Reading errors were morphological when the response was characterised by a substitution of inflexional (e.g. card → cards) or derivational (e.g. teach → teacher) suffixes.

When the patient uttered more than one response for a given stimulus, the last one was considered for the error analysis. Table 2.4 details the types of word-reading errors committed by BR. The bulk of his paralexias was visual errors, corresponding to what Shallice (1988) labels as “input type,” whereby the deficit involves the pre-semantic processing phase of the stimulus. With nonwords 60 % of all errors were visual; the remaining nonword paralexias included 5.2 % of visual-then-semantic and 34.8 % of unclassified errors. He never produced a nonword in response to either word or nonword stimuli; that is, he either omitted to respond or produced a wrong word.

The cognitive profile of BR was compatible with a clinical diagnosis of deep dyslexia. Within the dual-route model of reading (Coltheart et al. 2001), BR’s deficits affected both the lexical and the sublexical route (nonword reading was at floor, word reading was also poor and characterised by several different error types). It was necessary though to interpret the functional loci of BR’s errors, in particular his visual errors which are paramount.

## *Interim Formulation*

BR’s pattern of performance revealed a severe symptom complex due to multiple functional disorders. First of all, the sublexical procedure converting orthography



to phonology appeared to be completely damaged and unavailable. BR was totally unable to read nonwords aloud; no correct responses were given. He produced only omissions (“don’t know” responses) and substitutions with visually similar words; he never produced nonwords as responses.

As reading of known words was also affected, the lexical-semantic route must have been impaired. Most erroneous responses were substitutions of the stimuli with semantically and/or visually similar words. The sources of semantic errors could be various. They could result at the level of orthographic input lexicon, but if this was the case, semantic errors should be limited to reading aloud. BR produced semantic errors also in object naming; therefore, these errors should have had their origin at post-orthographic levels. If they were due to an impairment of the semantic system, tasks assessing word comprehension, like word to picture matching tasks, should have also been defective. However, as reported in Table 2.2, his performance on both auditory and written word comprehension tasks was quite normal. Finally, the source of the semantic errors could be located at the level of the phonological output lexicon. This interpretation is suggested by the presence of circumlocutions in object naming tasks, which revealed a deficit in accessing the phonological lexicon, confirmed by the very small number of errors on word comprehension.

The most frequent errors were visual paralexias, i.e., substitutions with visually similar words. Where did they come from? They cannot be attributed to the impaired grapheme-to-phoneme conversion mechanism because the patient never produced nonwords in his responses; therefore, it is highly implausible that this kind of errors could stem from the sublexical level. Visual paralexias can be phonological or orthographic in nature. However, since almost no phonemic paraphasias were observed in BR’s spontaneous speech and naming tasks, these errors have to be considered orthographic and not phonological, and could be located either at the lexical or at the prelexical level.

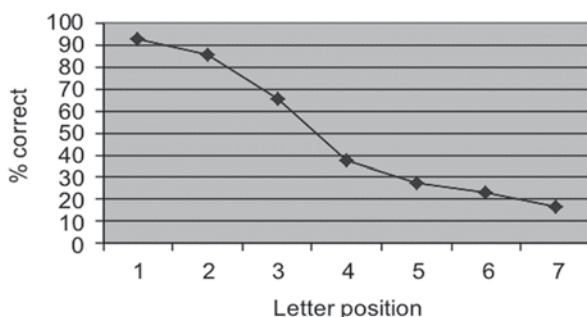
The position of the individual letters involved in errors allows us to disentangle the origin of these errors. Indeed, errors at the lexical level should involve any letters independently of their position within the stimuli; in contrast, prelexical errors should be spatially specified and limited to a given side. As shown by the examples given in Table 2.4, the latter was the case: BR’s errors often involved the endings of the words.

### ***BR’s Reading Errors: A Case of Neglect Dyslexia?***

Following Hillis and Caramazza (1991), a Letter Position Effect was calculated considering a subset of 387 word whose length ranged from 4 to 7 letters (four letters  $n=59$ , five letters  $n=170$ , six letters  $n=99$  and seven letters  $n=59$ ). Figure 2.1 illustrates the outcome showing the percent correct (letter in correct position) within these words. It appears at face value that there is an order effect, whereby the beginnings of the words are better read than their endings. This asymmetrical distribution of errors is compatible with right-sided neglect dyslexia (Vallar et al. 2010).



**Fig. 2.1** Letter position effect in BR's single-word reading. Percent correct (letter in correct position) within 4–7 letter words is shown. An order effect emerges: the beginning of the words is better read than their ending



Indeed, several of BR's reading errors met the defining criteria of neglect paralexias set forth by Ellis et al. (1987): paralexias could be classified as right neglect errors if the target and the response were identical to the left of an identifiable neglect point and there were no letters in common to the right of this point. Neglect paralexias were identified within each error category: visual (zio [uncle] → zingara [gypsy]), visual and/or semantic (canguro [kangaroo] → cane [dog]), visual then semantic (anzi [rather] → vecchi [old] through "anziani" [elderly]), morphological (lupo [wolf] → lupa [she-wolf]) and unclassified (senso [sense] → sedia [chair]). Also errors in reading nonwords could be classified as neglect paralexias: visual (curete → cucina [kitchen]) and visual then semantic (nafò → benzina [petrol] ... no! nafta [naphtha]).

Considering all BR's errors in reading words, taken from all categories with a visual component ( $n=869$ ), a total of 279 errors (32%) could be defined as right neglect paralexias according to Ellis et al. (1987). This proportion is less than that reported in cases of left neglect dyslexia due to right hemisphere damage (e.g. see Table 2.3 in Cubelli et al. 1991) and would not comply with the diagnosis of neglect dyslexia whereby neglect paralexias are the majority of observed errors. According to Ellis et al.'s criteria, none of the letters right to the neglect point should be shared between the stimulus and the response. However, in Italian almost all words end with a vowel, in particular "a, e, o." Therefore, the probability that the two words share the ending vowel simply by chance is sizeable. To overcome this confound, the less popular and looser criteria proposed by Hillis and Caramazza (1990) were adopted. These criteria assume that the diagnosis of neglect paralexia is satisfied when all the following conditions are observed: the response is identical to the target by at least two letters from the left end (beginning), there is at least one unshared letter to the right of the shared ones, and the stimulus and the response should not contain identical clusters of two or more letters to the right of the shared segment. Applying these criteria the proportion of BR's errors classifiable as right neglect paralexias rises to 61% ( $n=529$ ), which constitutes the majority of his errors and is compatible with the diagnosis of neglect dyslexia.

To tackle this hypothesis an ad hoc test was devised: the auditory–visual matching task. The patient was presented with two series of 16 trials each composed of four words characterised by having either the first three letters in common (e.g.

POLIZIA [police], POLENTA [maize flour], POLLINE [pollen] and POLMONE [lung]) or the last three letters in common (e.g. FARMACO [drug], UBRIACO [drunken], SINDACO [major] and ZODIACO [zodiac]). The four words in each trial were visually presented on flash cards one above the other; the task of the patient was to point to the word named by the examiner. BR's correct response in the trials with words sharing the same beginning was 8/16 (50%), whereas his performance with words sharing the same ending was 15/16 (93.75%).

Neglect dyslexia can result from deficits at different levels of representation (Hillis and Caramazza 1995): viewer-centred, stimulus-centred and word-centred. Neglect dyslexia associated with left hemisphere damage is hallmarked by the presence of word-centred neglect (Vallar et al 2010, p. 231) and may occur in the absence of unilateral spatial neglect (e.g. see case RYT by Warrington 1991). Therefore, BR should present with word-centred neglect. Should he present with an effect of spatial orientation, typical of stimulus-centred neglect sensitive to the physical features of the stimuli, the word-centred neglect hypothesis would be falsified. Word-centred representation codifies the abstract orthographic information specifying the identity and the order of the constituent letters independently of their physical appearance; hence, it should not be modulated by the spatial orientation of the stimulus. BR was assessed with a reading task in which the words were presented vertically, with individual letters one below the other. A total of 23 stimuli were used. Adopting Hillis and Caramazza's (1990) criteria, BR produced 17 (74%) right neglect paralexias, for instance he read "mela" [apple] as "mese" [month].<sup>2</sup> This proportion of errors is akin to that observed with horizontal stimuli (i.e. 61%).<sup>2</sup> The lack of spatial orientation effect suggests that BR's neglect paralexias were due to a deficit at the level of word-centred representation, which specifies the ordinal information of the letters within the word independently of their actual physical properties, such as size, font, case, spacing between letters, as well as the spatial orientation of the stimulus.

This interpretation is consistent with the performance of other patients with deep dyslexia reported in the literature. Three of them produced many errors, either visual or visual-then-semantic, that could be classified as neglect paralexias: thin → think; earl → deaf (case GR, Marshall and Newcombe 1966); bow → bowl; coward → Cowes (a place on the Isle of Wight; case KF, Shallice & Warrington 1975); revolt → revolver; charter → map (case DE, Patterson 1980). Siakaluk and Buchanan (2001) first described the case of a patient presenting with both right neglect dyslexia and deep dyslexia. Errors were semantic paralexias (e.g. ill → sick) and neglect paralexias (e.g. shown → short). Also Berndt et al. (Greenwald and Berndt 1999; Petrich et al. 2007; Berndt et al. 2005) described a series of left-brain-damaged patients who showed visual errors affecting the final letter of the stimulus words. As no visual spatial neglect and attentional disorders were detected, they stated that the positional errors were due to a selective disorder in coding orthographic ordinal information rather than being the direct consequence of neglect

<sup>2</sup> With Ellis et al.'s (1987) criteria the responses classified as right neglect paralexias were 35%, again similar to the proportion identified with horizontal stimuli (i.e., 32%).



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