Deep dyslexia in the two languages of an Arabic/French bilingual patient

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Abstract

We present a single case study of an Arabic/French bilingual patient, ZT, who, at the age of 32, suffered a cerebral vascular accident that resulted in a massive infarct in the left perisylvian region. ZT’s reading displays the characteristics of the deep dyslexia syndrome in both languages, that is, production of semantic, visual, and morphological errors, and concreteness effect in reading aloud and impossibility of reading nonwords. In the first part of this paper, using a three-route model of reading, we account for the patient’s performance by positing functional lesions, which affect the non-lexical, the semantic lexical and the non-semantic lexical routes of reading. Phonological priming observed in a cross-language visual lexical decision task indicates that implicit assembled phonological recoding is possible. The above lesions and implicit nonword reading characterize the output form of deep dyslexia. However, error distribution reveals dissociations across languages (e.g. the semantic error rate is higher in French whereas translations are more frequent in the Arabic testing) that cannot be accounted for within a three-route model. In the second part, extensions to Plaut and Shallice’s connectionist model (\textit{Cognitive Neuropsychology}, 10 (5) (1993) 377) are proposed to account for the translinguistic errors observed. ZT’s error distribution is compared to that obtained by Plaut and Shallice after lesions had been applied at different locations through the 40–60 network. The overall syndrome of deep dyslexia found in both languages is explained as resulting from lesions along the direct (O $\rightarrow$ I) and output (S $\rightarrow$ Ip, Ip $\rightarrow$ P) pathways of reading. Lesions along the output pathway mostly affecting S $\rightarrow$ Ip connections in French and Ip $\rightarrow$ P connections in Arabic account for discrepancies in ZT’s error pattern across tasks and languages. This case study demonstrates the superiority of a connectionist approach for
1. Introduction

Early accounts of deep dyslexia (Marshall & Newcombe, 1973) describe it as a symptom-complex characterized by semantic errors in reading, together with the co-occurrence of derivational and visual errors, substitutions of function words, and difficulty in or even impossibility of reading nonwords. Coltheart (1980a) extends to the initial list a set of other co-occurring symptoms, i.e. lexical derivation of phonology from print impaired, abstract words harder to read aloud than concrete words, writing spontaneously or to dictation impaired, auditory-verbal memory impaired, reading of a word dependent on its context, verbs harder to read than adjectives which are harder than nouns, function words harder to read than content words. The list is then reviewed and edited in Coltheart, Patterson, and Marshall (1987) who take out from the list the three last features, thus limiting to eight the number of symptoms claimed to co-occur mandatorily with semantic errors in deep dyslexia.

Initial attempts to account for the deep dyslexia syndrome were proposed within cognitive neuropsychological models. The multiple-impairment hypothesis postulates that several deficits along the lexical route (semantic and non-semantic routes for reading) and the non-lexical route are responsible for the co-occurrence of the types of errors observed in the deep dyslexic patient’s reading performance. More specifically, within the three-route model (Coltheart et al., 1987; Morton & Patterson, 1980; Shallice & Warrington, 1980), semantic and morphological errors in reading aloud, difficulty in reading abstract words and inability to read nonwords aloud are claimed to arise through impairments at four independent lesion sites within the language-processing system: (1) lesion to the assembled pathway that allows the identification of sub-word orthographic segments and their conversion into phonological segments, then into the oral production of a letter string; (2) damage to the addressed pathway which allows reading aloud of words with mediation of the semantic system; (3) impairment of the addressed pathway which allows reading of words without mediation of the semantic system; and (4) damage to the semantics of abstract words.

In subsequent research, however, criticisms have been raised leading to a weakening of the “syndrome-complex” theory. More specifically, even though semantic errors have been found to represent the central symptom of deep dyslexia as claimed initially, the close association and mandatory co-occurrence of the other symptoms have been questioned. For example, Allport and Funnell (1981) reported an absence of part-of-speech effect; Caramazza and Hillis (1990) suggested that some of the symptoms described do not necessarily appear together with semantic errors.

Another functional interpretation of deep dyslexia has been offered by Coltheart
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