Deep Dyslexia Is Right-Hemisphere Reading

Max Coltheart

Department of Psychology, Macquarie University, Sydney, New South Wales 2109
Australia

Two views exist concerning the proper interpretation of the form of acquired dyslexia known as deep dyslexia: (a) that it represents reading by a multiply damaged left hemisphere reading system; (b) that it represents reading which relies extensively on right-hemisphere orthographic and semantic processing. Price, Howard, Patterson, Warburton, Friston, and Frackowiak (1998) have recently reported a brain-imaging study whose results, they claim, “preclude an explanation of deep dyslexia in terms of purely right-hemisphere word processing.” Their claim conflicts with the conclusions of previous published work, which strongly supports the RH hypothesis, work which they do not mention. Furthermore, I argue that their own results also favor the RH hypothesis (even though they claim otherwise); indeed, their results permit the formulation of a much more detailed version of the RH hypothesis than has hitherto been possible. Hence I conclude that the right-hemisphere interpretation of deep dyslexic reading remains the preferred explanation of deep dyslexia. © 2000 Academic Press

Deep dyslexia (Marshall & Newcombe, 1966, 1973; Coltheart, Patterson, & Marshall, 1980) is a form of reading impairment caused by brain damage in which the most prominent—indeed, the defining—symptom is the semantic error in reading aloud; when deep dyslexics are reading single words aloud, with no context and no time pressure, their responses are often semantically but not orthographically or phonologically related to the word they are trying to read. So, for example error might be read as “wrong” or saxophone as “violin” (Price, Howard, Patterson, Warburton, Friston, & Frackowiak, 1998). Other error types also occur during reading aloud, particularly visual errors (sleeve → “sleep”) and morphological errors (paint → “painting”). There are selective problems in reading aloud function words and words with abstract meanings. Nonwords cannot be read aloud at all.

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Address correspondence and reprint requests to Max Coltheart at Department of Psychology, Macquarie University, Sydney, NSW 2109 Australia. E-mail: max@currawong.bhs.mq.edu.au. Fax: +61 2 9850 6059.
Two approaches have been taken to the explanation of this acquired disorder of reading. According to one approach (Morton & Patterson, 1980), which may conveniently be labeled the left-hemisphere (LH) hypothesis, the deep dyslexic is reading aloud by using a version of the normal left-hemisphere reading system which has been multiply damaged. Multiple loci of damage are postulated because of the multiplicity of symptoms in deep dyslexia. No model of the normal reading system contains any component which, when damaged, would cause the reading behavior of that system to exhibit all of the symptoms of deep dyslexia.

According to the other approach (Coltheart, 1980; Saffran, Bogyo, Schwartz, & Marin, 1980), which may conveniently be labeled the right-hemisphere (RH) hypothesis, reading aloud in deep dyslexia involves use of right-hemisphere reading mechanisms.

These two hypotheses differ so grossly in what they claim about the anatomical basis of reading in deep dyslexia that it should be easy to adjudicate between the two hypotheses simply by imaging the brains of deep dyslexics as they read aloud. Two recent papers (Weekes, Coltheart, & Gordon, 1997; Price et al., 1998) report brain imaging studies of deep dyslexic reading with precisely this aim in mind.

The last sentence of the Abstract of Weekes et al. (1997) reads: “These results support the right-hemisphere hypothesis of deep dyslexia.” The last sentence of the Abstract of Price et al. (1998) reads: “These results preclude an explanation of deep dyslexia in terms of purely right-hemisphere processing.”

What could account for the fact that two effectively identical investigations have reached completely opposed conclusions?

WHAT IS THE RH HYPOTHESIS?

Price et al. (1998) refer to this hypothesis at various points in their paper thus: “an explanation of deep dyslexia in terms of purely right-hemisphere processing” (p. 315); “the second hypothesis is that the cluster of deficits associated with deep dyslexia . . . might be the hallmarks of exclusive reliance on a right-hemisphere reading system” (p. 304; “[deep dyslexic reading] is not a simple reflection of a right-hemisphere reading system” (p. 312); “the hypothesis that the reading of deep dyslexics relies exclusively on a right-hemisphere reading system” (p. 312).

But this is wrong. No one has ever claimed that the reading of deep dyslexics relies exclusively on a right-hemisphere reading system.

All parties agree that reading aloud by deep dyslexics is accomplished via a sequence of three processing stages: orthographic processing, then semantic processing, then phonological processing. As long as any one of these three stages can no longer be carried out by the left hemisphere, then some kind of right-hemisphere reading mechanism will be required if reading
aloud is to be achieved. Thus for the RH hypothesis to hold, at least one of these three stages must depend upon the right hemisphere. But, as I have said, the claim that all three of the stages are carried out by the right hemisphere has never been made, so it is not correct to characterize the RH hypothesis as claiming that deep dyslexic reading relies exclusively on a right-hemisphere reading system.

This point is obvious if one consults the original papers on the RH hypothesis. Firstly, the originators of the RH hypothesis, Coltheart (1980) and Saffran et al. (1980), explicitly stated that the RH hypothesis of deep dyslexia is not committed to any view as to which hemisphere is responsible for the phonological production component of reading aloud (e.g. “the laterality of the output mechanism is not, on this view, a critical feature of a right hemisphere model for deep dyslexia”; Saffran et al., p 401). Such statements are of course inconsistent with the claim that in deep dyslexia reading aloud relies exclusively on the right hemisphere.

Second, consider the following quotations:

I begin, therefore, with the assumption that when a deep dyslexic pronounces print, his speech is produced by the left hemisphere. (Coltheart, 1980, p. 351)

The particular version of the right-hemisphere hypothesis which is advanced here is that what is lost in deep dyslexia is access from print to the left-hemisphere lexicon, i.e., orthographic access to this lexicon. When the deep dyslexic is asked to read a word aloud, then this problem, put very crudely, is this: orthographic input to a right-hemisphere linguistic system is possible, but speech output is not; speech output from a left hemisphere linguistic system is possible, but orthographic input is not. If so, the task of reading aloud will require (a) orthographic input to a right-hemisphere reading system followed by (b) transfer from the right hemisphere to the left of information which can be used (c) to select from amongst the phonological forms of word stored in the left hemisphere that form which corresponds with the word being looked at.” (Coltheart, 1980, p. 352)

The argument, then, is that when a deep dyslexic reads aloud, he does so by firstly accessing (correctly or incorrectly) an entry in a right-hemisphere lexicon. From this entry, he retrieves a semantic representation and transmits this representation to the left hemisphere, where it is used to access an entry in the left-hemisphere lexicon. Once a left-hemisphere entry is identified, phonological information is retrieved and, finally, articulated. (Coltheart, 1980, p. 353)

The specific version of the RH hypothesis articulated here quite clearly asserts that in deep dyslexic reading one component of the system used for reading aloud, the phonological output lexicon, is a left-hemisphere process. It is therefore a mistake to characterize the RH hypothesis as one according to which “reading of deep dyslexics relies exclusively on a right-hemisphere reading system,” and it is difficult to see what led Price et al. (1998) to believe that anyone held such a hypothesis.

In any case, Price et al. (1998) are inconsistent in their exposition of the RH hypothesis, since at one point in their paper they actually state it correctly: “If deep dyslexics translate print to meaning in the right hemisphere and then output speech in the left hemisphere . . .” (p. 306).
The upshot of this is that no imaging data that have to do with the anatomical locus of phonological output aspects of reading aloud are relevant to adjudication between the LH and RH hypotheses, since the RH hypothesis is not committed to any view about this locus. What the RH hypothesis is completely committed to is a view about the locus of orthographic and semantic aspects of reading aloud. The RH hypothesis asserts that in deep dyslectic reading aloud, the RH is responsible for orthographic processing, and it also carries out semantic processing.

SUBTRACTION METHODS AND BRAIN IMAGING.

As Price et al. note (p. 305) “Reading aloud involves a large number of cognitive processes including—at a minimum—visual and orthographic analysis, phonological retrieval, articulation, and hearing the sound of the spoken word.” As we have just seen, some of these processes are relevant to adjudication between the LH and RH hypotheses, and some are not. It is possible, by using properly designed experimental tasks and subtractive methodology, to identify specific brain regions associated with specific processing components of the reading aloud task, and in other work by this group this has been attempted (e.g., Howard, Patterson, Wise, Brown, Friston, Weiller, & Frackowiak, 1992). Given that what is critical is localizing the orthographic and semantic components of reading aloud, it is unfortunate that this approach was not adopted by Price et al. All they compared was a reading aloud condition with a rest condition. That makes it extremely difficult to link particular activated regions to particular subcomponents of the reading aloud process. In contrast, in the imaging study of deep dyslexia reported by Weekes et al. (1997) and described below, multiple tasks and subtractive methodology were used.

PREVIOUS RESEARCH RELEVANT TO THE RH HYPOTHESIS

According to Price et al. (p. 305), “previous attempts to adjudicate between these two hypotheses have been inconclusive.” Unfortunately, they neither discuss nor even refer to any such previous attempts. In the past decade there have been three papers published which are directly relevant to such adjudication: Patterson, Vargha-Khadem, and Polkey (1989), Michel, Henaff, and Intrilligator (1996), and Weekes et al. (1997)—and it simply isn’t true that these have been inconclusive.

Patterson et al. (1989) studied the reading of NI, a girl who had developed symptoms of brain pathology at the age of 13 and had a total left hemispherectomy at the age of 15. Data from their study is summarized in Table I along with data from the deep dyslexics CJ and JG studied by Price et al. It is clear that NI’s reading is very similar to the reading of the deep dyslexics CJ and JG. I concur with the conclusion reached by Patterson et al.: “The empirical conclusion is clear: adult deep dyslexics, who may be reading with
The right hemisphere, and N.I., who must be reading with the right hemisphere, are strikingly similar” (p. 56).

Michel et al. (1996) report the case of AC, a 23-year-old college student who suffered a lesion of the posterior part of the corpus callosum. His reading was tested by presenting words and nonwords to the left or right visual hemifields. With right-hemifield presentation no abnormalities in his reading were detected. Table 1 reports data from left-hemifield (right-hemisphere) presentation. Clearly, AC’s left-hemifield reading is highly similar to the reading of the deep dyslexics CJ and JG. I concur with the conclusion reached by Michel et al. (p. 788): ‘‘When AC reads in his LVF he probably utilizes both hemispheres, with the LH being constrained to use uncommon high-level information, both imagistic and semantic, produced by initial processing within the RH.’’ That interpretation by Michel et al. is of course identical to the formulation of the RH hypothesis by Coltheart (1980, p. 353), quoted above.

Weekes et al. (1997) investigated a deep dyslexic, LH, a surface dyslexic, NW (see Weekes & Coltheart, 1992, for more information on NW), and two normal controls. Regional cerebral blood flow (rCBF) was measured using Xenon-133 while these subjects were performing various types of reading tasks designed to permit subtractive analyses.

Brain regions specifically associated with the visual word recognition component of the reading-aloud task were identified by subtracting bloodflow data collected when the task was viewing a row of false font stimuli and responding ‘‘Yes’’ to the onset of each stimulus from bloodflow data collected when the stimulus was a row of letters, either a word or a nonword, to which the subject had to make a vocal lexical decision. For the deep dyslexic LH, but not for the other three subjects, rCBF was greater in the RH than the LH after this subtraction.

Brain regions specifically associated with the spoken word production component of the reading-aloud task were identified by subtracting bloodflow data collected when the task was lexical decision from bloodflow data
collected when the task was reading aloud. The assumption here was that reading aloud a different word on every trial would place greater demands on the phonological retrieval system than merely saying ‘‘Yes’’ or ‘‘No’’ on each trial. All four subjects showed more LH than RH activation here, though the difference was not significant for NW and only marginally significant for one of the control subjects. It was, however, highly significant for the deep dyslexic LH. I agree with the conclusion reached by Weekes et al. (p. 1152): ‘‘When L.H. is reading aloud, she recognizes the word with her right hemisphere; however, she then produces the word from her left hemisphere.’’

These are the three studies carried out in the past decade that are directly relevant to adjudication between the LH and RH hypotheses. Had Price et al. mentioned these studies, it is difficult to see how they would have been able to defend their claim that attempts at such adjudication so far ‘‘have been inconclusive.’’ On the contrary: it is clear that all three studies strongly support the RH hypothesis.

**THE RESULTS OF PRICE ET AL.**

First consider the three specific predictions set out by Price et al. (p. 306) and the relationship between these predictions and their results:

1. If deep dyslexic reading relies on a reduced left-hemisphere system . . . CJ and JG should show activation of left-hemisphere regions associated with semantic processing (extrasylluvian temporal cortices) and phonological retrieval (left posterior inferior temporal cortex and left frontal operculum) and a degree of right-hemisphere activation that is within the range of normal variability.

The results contradict the prediction from the LH hypothesis that in CJ and JG there would be ‘‘a degree of right-hemisphere activation that is within the range of normal variability.’’ Both of the deep dyslexics showed significantly more activation than the normal subjects in several right-hemisphere sites: two right inferior occipital sites (36, −78, −16 and 32, −86, 4), right frontal operculum, and two right cerebellum/parahippocampal sites (24−50, −20 and 24, −40, −20). The significance of some of these sites is discussed further below.

2. If the right hemisphere contributes more to reading in deep dyslexics than in normal subjects, we would expect left-hemisphere reading-dependent activity and additional right-hemisphere activity relative to normals.

These predictions from the RH hypothesis were borne out exactly by the data.

3. If deep dyslexics translate print to meaning in the right hemisphere and then output speech in the left hemisphere, we would expect to see activation in right- (but not left-) hemisphere regions associated with semantics as well as activation of left-hemisphere regions associated with speech output.
There are two predictions here, and both are problematic. The first is problematic because Price et al. do not specify what right-hemisphere regions they believe to be associated with semantics; furthermore, if there are such regions, they would be activated in normals as well as deep dyslexics. But in any case I argue below that this prediction from the RH hypothesis was actually confirmed in the data of Price et al. The second is irrelevant because, as explained above, the RH hypothesis is not committed to the view that output in deep dyslexic reading aloud is from the LH.

Thus prediction 1 (from the LH hypothesis) was disconfirmed, prediction 2 (from the RH hypothesis) was confirmed, and the relevant part of prediction 3 (from the RH hypothesis) was also confirmed. Hence Price et al.’s results favor the RH hypothesis, rather than, as they claimed, precluding it.

THE SIGNIFICANCE OF RIGHT-HEMISPHERE SITES OF ACTIVATION IN DEEP DYSLEXIA

Price et al. adopt the view that, if there are sites in the RH that carry out specific linguistic processing tasks, these sites are likely to be homologous to the LH sites that carry out these tasks. I will also adopt this view.

Hence a natural way to explore the issue of where in their right hemispheres CJ and JG are performing the orthographic, semantic, and phonological processing needed for reading aloud is to consider what LH sites are responsible for such processing when intact subjects are reading aloud and then to study what is going on in the RH homologous sites with CJ and JG.

Orthographic Processing

In their comprehensive review of neuroimaging studies of word reading, Fiez and Petersen (1998, p. 917) point out that a number of left hemisphere regions have been associated with orthographic processing in imaging studies of reading aloud by intact subjects: “left occipital and occipitotemporal regions are involved in visual analysis specific to word-like stimuli.” One such region is the inferior occipital cortex. Rumsey, Horwitz, Donohue, Nace, Maisog, and Andreason (1997) found that an orthographic lexical decision task (distinguishing real words such as DEEP from pseudohomophones such as DEAP) produced activation at an inferior occipital site with coordinates (−36, −68, −16). Madden, Turkington, Coleman, Provenzale, Degrado, and Hoffman (1996), also using visual lexical decision, also observed inferior occipital activation, at (−26, −88, −24). Puce, Allison, Asgari, Gore, and McCarthy (1996) also observed inferior occipital activation, at (−40, −66, −17) and (−37, −71, −22), when subjects were viewing letter strings.

These studies agree rather well as far as the specific coordinates of the activated regions are concerned. It is clear, then, that there is a region of left inferior occipital cortex that is implicated in orthographic processing during
reading. A rough indication of the coordinates of the center of this region might be obtained by averaging the four sets of coordinate values: this gives $(-35, -74, -20)$.

Hence it is of interest to consider what was occurring in the right hemisphere homologue of this region—in and around $(35, -74, -20)$—when CJ and JG were reading aloud. Price et al. report that there were two right occipital sites at which both CJ and JG showed significantly more activation than all of the control subjects. These two sites were $(36, -78, -16)$ and $(32, -86, -4)$. The coordinates of the first of these sites are extraordinarily similar to those predicted from the averaging process described above, and the second site is of course very close to the first. In other words, the right inferior occipital region activated when CJ and JG were reading is exactly homologous to a LH region activated when intact subjects are reading. This provides very strong evidence that when CJ and JG are reading, they are using their right hemispheres for orthographic processing, as required by the RH hypothesis.

**Semantic Processing**

In their review, Fiez and Petersen (1988, p. 917) say “Regions near the border between superior and middle temporal gyrus (BA 22/21) are involved in semantic analysis.” Pugh, Shaywitz, Shaywitz, Constable, Skudlarski, Fulbright, Bronen, Shankweiler, Katz, Fletcher, and Gore (1996) concluded that both left BA22 and left BA21 are specialized for semantic processing. This directs our attention to BA22 and BA21 of the right hemispheres of CJ and JG. For JG, there was a region in right BA22 at $(48, -52, 16)$ which showed significantly greater activation than in any of the control subjects. For CJ, there was a region in right BA21 at $(58, -18, -12)$ which showed significantly greater activation than in any of the control subjects. There were other regions in BA22 for which both the deep dyslexics and the normals showed significant activation. Although the RH temporal regions activated in the two deep dyslexics are not identical, these are regions homologous to LH regions associated with semantics. Consider also that a left temporal region at $(58, -30, 0)$ was identified with semantic processing by Vandenberghhe, Price, Wise, Josephs, and Frackowiak (1996). The right temporal point homologous to this would be $(58, -30, 0)$. That point is close to, and approximately half way between, $(58, -18, -12)$ (activated in CJ) and $(48, -52, 16)$ (activated in JG). These results thus provide very strong evidence that when CJ and JG are reading aloud, they are using their right hemispheres for semantic processing, as required by the RH hypothesis.

Both deep dyslexics also showed activation of some left temporal regions which Price et al. associate with semantic processing. The RH hypothesis makes no predictions as to whether there will be LH semantic activation when deep dyslexics are reading aloud, but the occurrence of such activation
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is easily interpreted as a consequence of communication from RH semantic regions to LH semantic regions. If there is any contribution from a LH phonological output system when deep dyslexics are reading aloud, then information must be transmitted from a RH semantic system to a LH phonological output system, and a simple way for this to occur is RH semantics to LH semantics to LH phonological output system.

**Phonological Output Processing**

The LH region generally responsible for phonological output is the left frontal operculum (Alexander, Naeser, & Palumbo, 1990; Hajek, Valavanis, Yonekawa, Schiess, Buck, & Wieser, 1998; Rosen, Fiez, Hanlon, Dromerick, Linenweber, Petersen, Raichle, & Corbetta, 1998). As discussed above, the RH hypothesis leaves open the question of the laterality of phonological output mechanisms for reading aloud in deep dyslexia. JG showed more activation of the left BA47 than CJ or the controls and more activation of some regions in right BA44/46 than CJ or the controls. CJ showed more activation of the left BA44 (Broca’s area) than JG or the controls. Both CJ and JG showed significantly more activation of the right frontal operculum than any of the controls. This suggested to Price et al. that the two deep dyslexics may have differed with respect to the system used for phonological output during reading aloud, with JG using the RH for this purpose much more than CJ (cf. Saffran et al., 1980, p. 401: ‘‘[in deep dyslexia] the output system may reside in the left hemisphere . . . or, in some cases, the right hemisphere itself may have the capacity to produce speech. Perhaps patients differ in this respect.’’)

These possible individual differences are consistent with other work on the frontal operculi. Hajek et al. (1998) studied four epileptic patients with lesions in or near classical Broca’s area. The left frontal operculum was temporarily inactivated by selective amobarbital administration. For three patients, there was no detectable temporary language impairment here; thereupon, the lesions in or adjacent to the left frontal operculum were completely resected. No postoperative language impairment was detected. In the other patient, there was transient language disturbance after the amobarbital administration; in this patient, a tumor of the left frontal operculum was restrictively resected. This caused a global aphasia, from which the patient recovered. Recovery from aphasia associated with left opercular damage has also been studied by Rosen et al. (1998). The task given to these aphasic patients and to controls was oral completion of visually presented word stems. In controls, this task activated the left frontal operculum. In the three aphasic patients, the task activated the right frontal operculum. Taken together, these results indicate that in some, but not all, patients with damage to the left frontal operculum, phonological output comes to be mediated by the right frontal operculum.
CONCLUSIONS

The results of Patterson et al. (1989), Michel et al. (1996), and Weekes et al. (1997) provide strong support for the RH hypothesis of deep dyslexia. So do the results of Price et al. (1998) (even though these authors claim otherwise), since the patterns of activations seen in their deep dyslexic subjects confirm their predictions from the RH hypothesis and disconfirm their predictions from the LH hypothesis.

Indeed, their results allow a major advance to be made as far as the RH hypothesis for deep dyslexia is concerned, because it is now possible on the basis of these results to propose a much more specific version of that hypothesis, to wit: When deep dyslexics read aloud, orthographic processing such as letter and word recognition occurs in a right inferior occipital region at approximately (−35, −74, −20). This orthographic processing leads to the activation of semantic information in right temporal lobe at BA 21/22. That semantic activation in turn leads (perhaps via LH semantic activation) to activation of phonological output systems in the frontal operculum. Patients appear to differ from each other with respect to the roles of left and right frontal operculi here. Some may use just a LH output system (for example, LH (Weekes et al., 1997)); some may have bilateral phonological output processing (e.g., CJ); and some may rely to a large degree on a RH output system (e.g., JG). No doubt this depends upon the degree of damage to left frontal operculum in individual deep dyslexics.

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