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A LITERATURE REVIEW AND NEW DATA SUPPORTING AN INTERACTIVE ACCOUNT OF LETTER-BY-LETTER READING

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We present a theoretical account of letter-by-letter reading (LBL) that reconciles discrepant findings associated with this form of acquired dyslexia. We claim that LBL reading is caused by a deficit that affects the normal activation of the orthographic representation of the stimulus. In spite of this lower-level deficit, the degraded orthographic information may be processed further, and lexical, semantic, and higher-order orthographic information may still influence the reading patterns of these patients. In support of our position, we present a review of 57 published cases of LBL reading in which we demonstrate that a peripheral deficit was evident in almost all of the patients and that, simultaneously, strong effects of lexical/ semantic variables were observed on reading performance. We then go on to report findings from an empirical analysis of seven LBL readers in whom we document the joint effects of lexical variables (word frequency and imageability) and word length on naming latency. We argue that the reading performance of these patients reflects the residual functioning of the same interactive system that supported normal reading premorbidly.

INTRODUCTION

Letter-by-letter (LBL) reading is the term used to define the reading pattern of premorbidly literate patients who, following brain damage acquired in adulthood, take an abnormally long time to read even single words. This reading deficit is typically associated with a lesion in the posterior portion of the dominant hemisphere and sometimes, but not always, accom-

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panied by a lesion of white matter tracts such as the splenium of the corpus callosum or forceps major (Damasio & Damasio, 1983). The reading performance of such patients is characterised by a "word length effect", a significant increase in naming latency as a function of the number of letters in a string. Times in the order of 1–3 seconds per additional letter in a string have been measured for some LBL readers, although there is considerable variability in reading speed across patients (Hanley & Kay, 1996). When the reading deficit manifests in the absence of other reading, writing, or spelling deficits, it is referred to as "pure alexia". When other written language deficits do accompany the LBL reading, they usually consist of surface dyslexia (Bowers, Bub, & Arguin, 1996; Friedman & Hadley, 1992; Patterson & Kay, 1982) or surface dysgraphia (Behrmann & McLeod, 1995; Rapp & Caramazza, 1991). Although less frequent, there are also reports of at least one case of deep dyslexia (Buxbaum & Coslett, 1996) and one case of phonological dyslexia (Friedman et al., 1993; Nitzberg-Lott, Friedman, & Linebaugh, 1994) accompanying LBL reading.

Two critical empirical findings concerning LBL reading pose difficult challenges for theories of this disorder. One finding is that these patients are impaired at letter processing. A second important finding is that some of these patients have available to them lexical and semantic information about the stimulus, as evidenced in their above-chance performance on lexical decision and semantic categorisation tasks. This latter finding suggests that the visual stimulus has been processed to a sufficient extent to produce such higher-order or later effects on performance. Two classes of theory have emerged, each of which emphasises one of these two paradoxical findings. One class argues that the deficit occurs early in processing, prior to the activation of an orthographic representation, and the early visual deficit observed in LBL readers is consistent with this view. We will refer to such views as *peripheral*, consistent with the Shallice and Warrington distinction in which impairments that adversely affect the attainment of the visual word-form are considered to be peripheral (Shallice, 1988; Shallice & Warrington, 1980). By contrast, if the impairment is at a later stage, the dyslexia is classified as *central*. With respect to LBL reading, central views maintain that the deficit occurs after the activation of a well-specified orthographic description and thus lexical and semantic information can still be accessed.

What is probably evident, even from this brief description, is that the peripheral and central accounts of LBL readings are difficult to reconcile. Peripheral views cannot readily account for the lexical/ semantic findings and the central views do not explain the impaired early visual processing in these patients. In the present paper, we will argue that both sets of empirical findings can be accommodated within a single, interactive reading system to which both hemispheres contribute. We first present in detail the findings for a low-level deficit in LBL readers and then we consider the details of the lexical and semantic findings. Thereafter, we examine not only the peripheral and central accounts but also views that incorporate some aspects of both. Finally, we present our account and, to substantiate it, we review most of the published cases of LBL reading and present new empirical data from seven LBL readers.

KEY EMPIRICAL FINDINGS

Impairment in Early Visual Processing

It is now well established that patients with LBL reading do not activate orthographic representations adequately; for example, many patients are adversely influenced by alterations of the surface characteristics of the stimulus (for example, poorer reading of script than print) and many make mostly visual errors in reading (for example, JAY → "joy"). There are, however, several different explanations for the impairment in activating orthographic representations. Some studies have claimed, for example, that these patients suffer from a general perceptual deficit that impairs all forms of visual processing (Behrmann, Nelson & Sekuler, 1998; Farah, 1991; Farah & Wallace, 1991; Friedman & Alexander, 1984; Sekuler & Behrmann, 1996), although letter and word recognition might perhaps be especially vulnerable (Farah, 1997). Others have claimed that the impairment is specific to orthography and impairs the identification of letters per se (Arguin & Bub, 1993; Karanth, 1985; Kay & Hanley, 1991; Reuter-Lorenz & Brunn, 1990), or affects the rapid processing of multiple forms in parallel (Kinsbourne & Warrington, 1962; Levine & Calvanio, 1978; Patterson & Kay, 1982; Schacter, Rapcsak, Rubens, Tharan, & Laguna, 1990; see also Miozzo & Caramazza, this issue). Still others have maintained that LBL reading arises from insufficient attentional resources, thus forcing a serial, left–right strategy (Rapp & Caramazza, 1991), or from a problem in capacity or in switching visual attention (Buxbaum & Coslett, 1996; Price & Humphreys, 1992). In a recent paper, one of us (Behrmann & Shallice, 1995) argued that the core deficit is one of letter processing and that the time to activate the representation for even a single letter is slow. Importantly, we argued that the processing deficit is not spatially determined, i.e. processing does not proceed from left to right of the array; rather, it has to do with serial order such that letters appearing later in a string (even when all letters are presented at fixation in an RSVP paradigm) are disadvantaged relative to letters appearing earlier in the string.

It is important to note that many of these peripheral views are not necessarily mutually exclusive. For example, although the critical deficit may be one of disordered perceptual processing, an obvious consequence of such a deficit is an impairment in rapid and accurate letter processing and identification (Behrmann & Shallice,1995; Sekuler & Behrmann, 1996). What is central to all these peripheral views (labelled under the heading orthographic access view, [Bowers et al., 1996]), however, is that the fundamental impairment is one of visual processing, arising relatively early and preventing the derivation of an adequate orthographic representation.

Numerous investigators have commented that problems in letter-processing tasks are so common in LBL reading that impaired letter identification is likely to underlie their reading deficit (Coltheart, 1981; Patterson & Kay, 1982). In support of this, Patterson & Kay showed that all four of their patients made letter identification errors, albeit to varying degrees. Patient CH, for example, identified correctly only 16/ 26 upper-case and 10/ 26 lower-case letters and chose the odd letter out (for example, f F E) correctly on only 75% of the trials. Patient TP, on the other hand, identified 25/ 26 lower-case letters and made no errors on cross-case matching (e.g. D d). Interestingly, the types of errors made by all the patients reflected the visual similarity between the target and the error, suggesting that visual feature overlap is a major factor in letter misidentification (see also Perri, Bartolomeo, & Silveri, 1996). Consistent with the findings of letter misidentification, Behrmann and Shallice (1995) maintained that there is no convincing evidence of normal letter processing in any LBL reader. They then posed the challenge that, unless the hypothesis of impaired letter processing was found wanting, this should constitute the default explanation for the reading impairment in patients with LBL reading.

Lexical and Semantic Effects on Reading

Give this evidence, it is particularly puzzling that some LBL readers, even if they cannot explicitly identify the stimulus, can nonetheless demonstrate some lexical and semantic information about it. This result was documented in several fairly early reports of LBL patients (Albert, Yamadori, Gardner, & Howes, 1973; Caplan & Hedley-Whyte, 1974; Kreindler & Ionescu, 1961); even though the patients in these studies could not identify a written word overtly, they were still able to match this target with a word spoken by the investigator, or with a visually presented object. These initial observations of implicit or covert reading abilities of LBL readers in the absence of explicit identification have been upheld in a number of more recent studies. For example, Shallice and Saffran (1986) reported that their patient, ML, was above chance at performing lexical decision and semantic categorisation tasks with stimuli presented too briefly to permit overt identification. Several other studies have similarly shown that their patients can perform lexical decision (Bub & Arguin, 1995) as well as semantic classification of words (for example, living vs. nonliving) at exposure durations too brief for the patients to have identified the target items explicitly (for example, Bub & Arguin, 1995; Howard, 1991). In the largest series, Coslett and colleagues (Coslett & Saffran, 1989a, 1994; Coslett, Saffran, Greenbaum, & Schwartz, 1993) have described five patients who performed well above chance on lexical decision and semantic categorisation tasks with the very same stimuli they could not identify explicitly (see also Saffran & Coslett, this volume). Coslett and colleagues also described two additional patients who fit the definition of optic aphasia and who were completely unable to name letters (or any other stimuli from visual presentation). Even in the absence of letter naming, these two patients were fairly successful at lexical decision and binary categorisation tasks (Coslett & Saffran, 1989b, 1992).

THEORIES OF LETTER-BY-LETTER READING

The two empirical findings of an early deficit and the influence of later lexical and semantic properties of the stimulus on performance are now both well documented in the domain of LBL reading. As mentioned previously, there are two main classes of explanation of LBL reading which differ in the extent to which they emphasise one or the other of these two findings. The peripheral view argues that the deficit occurs early in processing, consistent with the early visual deficit observed in these readers. Proponents of this view have focused on the letter-processing deficit and its underlying mechanism without paying much attention to the later, lexical and semantic effects. The central view argues that the deficit occurs only after (or at least does not prevent) the activation of a well-specified orthographic description and, thus, lexical and semantic information can still be assessed. Within the central view there are two different accounts. One account, although perhaps the less favoured at present, is that the patients have an intact reading system and can activate lexical and semantic knowledge normally, but that the output of this intact system is disconnected from consciousness (Schacter, McAndrews, & Moscovitch, 1988; Young & Haan, 1990). Thus, although subjects can process the information, and the results may be revealed through implicit tasks that do not require explicit processing, such as semantic categorisation or priming, the contents of this system are not available for overt report. This view has probably fallen out of favour for a number of reasons, including the fact that it cannot account for the existence of the early visual processing impairment and that there is no explanation for the hallmark feature of this problem, the letter-by-letter reading itself or the increase in naming latency with word length.

A second central view argues for a visual-verbal disconnection, i.e., that the visual areas involved in reading are anatomically and/ or functionally disconnected from the more semantic/ conceptual areas. The best example of this view is from Déjerine who, in his famous 1891/ 1892 case studies (for overview, see Bub, Arguin, & Lecours, 1993), interpreted the LBL syndrome as a disconnection of the visual verbal input from "the visual memory centre for words", which is located in the left angular gyrus (Geschwind, 1965; Greenblatt, 1973; Speedie, Rothi, & Heilman, 1982). Bowers et al. (1996a; also Arguin, Bub, & Bowers, this issue) have adopted a similar perspective and maintained that the disconnection arises only after orthographic word representations have been activated; thus the disconnection is between orthographic representations (logogens) on the one hand and phonological codes on the other. This disconnection delays (or precludes) access to the phonological code for naming, while leaving lexical decision, semantic categorisation, and the word superiority effect intact. In support of their disconnection account, they showed that the reading performance of a LBL reader, IH, was facilitated by orthographically related primes (e.g. GATE-gate) but, unlike normal readers, not by homophonically related primes (e.g. gait-gate) (Arguin, Bub, & Bowers, this issue). IH also showed no effect of phonemic neighbourhood size on word recognition performance. Given that IH shows no evidence for covert phonological activation, the functional site of the lesion, according to this account, lies between orthographic and phonological processing (but see Montant, Behrmann, & Nazir, 1998, for demonstration of phonological priming in an LBL reader).

In summary, both the peripheral and central explanations of LBL reading do well at accounting for the subset of empirical findings on which they focus. The limitations of most of these accounts, however, is that they do not simultaneously accommodate both the peripheral and central aspects of LBL reading. One exception to this is the view of Coslett and Saffran, which takes into account both of these aspects (Buxbaum & Coslett, 1996; Coslett & Saffran, 1994; Saffran & Coslett, this issue). These authors have suggested that there is a deficit (most likely peripheral and early) in the normal reading system in these patients and that the sequential output pattern typically associated with LBL reading is mediated by the left hemisphere. The covert reading and later lexical/ semantic effects do not depend on the inaccessible (or degraded) word form generated by the left hemisphere. Rather, a separate reading mechanism, subserved by the intact right hemisphere, is responsible for the findings of preserved lexical decision and semantic categorisation. On this *right-hemisphere* interpretation, the lesion in pure alexia prevents visual information from both cortices from accessing lexical and semantic representations in the left hemisphere, but this information can still support the (albeit limited) reading capabilities of the right hemisphere. Consistent with the right-hemisphere view, Coslett et al. (1993) showed that, as the LBL patients became able to engage in covert reading, explicit recognition performance was influenced by word imageability and grammatical class, properties often associated with the reading skills of the right hemisphere (Coltheart, 1980, 1983).

A particularly appealing aspect of this right-hemisphere view is that the later lexical and semantic effects observed in LBL readers are similar to those generated by the right hemisphere of commissurotomy patients as well as by left-hemispherectomy patients (Coltheart, 1983). In fact, a similar explanation has been proposed by some to account for the pattern of reading in patients with deep dyslexia (Coltheart, 1980, 1983; Saffran, Bogyo, Schwartz, & Marin, 1980).

Although the right-hemisphere account of LBL reading takes into account both its peripheral and central aspects, it makes the very specific assumption that these different aspects reflect entirely distinct modes of reading and are mediated by different hemispheres. This assumption would seem to imply that the normal modes of operation of the two hemispheres are altered in LBL reading, with the right hemisphere playing an increased role relative to its normal contribution. However, the exact relationship between the roles of the left and right hemisphere in LBL reading and their roles in normal reading has never been made explicit on the right-hemisphere account. By contrast, we present an alternative theory in which both the early and late effects in LBL reading arise as a result of a peripheral impairment to the normal word reading system that is supported by both hemispheres.

AN INTERACTIVE ACCOUNT OF LETTER-BY-LETTER READING

One possible reaction to the controversy between the peripheral and central accounts of LBL reading is to argue that reconciliation is unnecessary and that a host of different underlying deficits may give rise to this problem. Moreover, individual differences could also arise from the different compensatory strategies adopted by the patients (Price & Humphreys, 1992, 1995). On this view, no uniform account of letter-by-letter reading exists and none is needed. In this paper, however, we argue that a reconciliation between these various positions is both desirable and possible and that LBL may be accounted for by a single, unifying view of the normal word reading system.

Our account takes as its starting point the framework of the Interactive Activation Model of letter and word perception (hereafter IAM; McClelland & Rumelhart, 1981; Rumelhart & McClelland, 1982). The model contains three levels of processing units– letter features, letters, and words– such that elements and their components (e.g. the word MAKE and the letter M in the first position) mutually support each other whereas inconsistent alternatives (e.g. the words MAKE and TAKE) mutually inhibit each other. The critical properties of the model for our purposes are that processing is *cascaded* and *interactive*. Cascaded processing means that partial results at each level, in the form of intermediate levels of activation, propagate to other levels immediately and continuously, rather than waiting until processing at lower levels is complete. Interactive processing means that activation not only propagates from lower to higher levels, but that the activation at higher levels feeds back to lower levels to provide additional support for those lower-level elements that are consistent with the higher-level activation. Thus, cascaded, interactive processing causes early letter activation to feed forward and partially engage word representations, which in turn feed back to the letter level to influence subsequent processing.

We should point out that we are adopting the IAM as a framework for explaining LBL reading not out of any commitment to the localist word representations it contains, but because it provides a clear instantiation of the principles of cascaded, interactive processing. The same principles apply within distributed accounts of lexical processing (e.g. Plaut, McClelland, Seidenberg, & Patterson, 1996; Seidenberg & McClelland, 1989; Van Orden, Pennington, & Stone, 1990) and, as we explicate below, essentially the same account of LBL reading holds within such models.

We claim that the fundamental impairment in LBL reading, following an occipital lesion, is a general perceptual deficit that degrades the quality of visual input. In the IAM, this deficit can be conceptualised as damage to the letter feature level or between this level and the letter level. The impact of this perceptual impairment on word recognition is that it permits only weak or partial parallel activation of the letters in a word. This weak activation does not suffice for explicit identification of the word (i.e. no word unit achieves a sufficiently high level of activation to exceed the response threshold) and the system must resort to sequential processing to enhance the activation of individual letters. Critically, this type of sequential processing is not an abnormal strategy only employed following brain damage, but is the manifestation of the normal reading strategy of making additional fixations when encountering difficulty in reading text (Just & Carpenter, 1987; Rayner & Pollatsek, 1987). For example, normal subjects fixate more frequently in a long word than in a short word in order to enhance the quality of the stimulus (O'Regan & Levy-Schoen, 1989). LBL readers also fixate more frequently; in fact, given the very poor quality of the visual input, they fixate almost every letter (Behrmann, Barton, & Black, 1998), giving rise to the hallmark word length effect. Presumably these fixations aid performance by permitting the increased spatial resolution of the fovea to be applied to multiple locations within the word. In fact, even in the absence of overt saccades, a word length effect would be expected, given that LBL readers can improve perceptual processing by rescaling covert attention from the entire word to apply successively to letters within the word[1].

Even though the word-level activation produced by the initial, weak, parallel letter activation is insufficient to support explicit identification, it nonetheless would be expected to activate the correct word to a greater extent than its competitors, and to produce more activation overall than would be produced by a nonword (see McClelland & Rumelhart, 1981). Thus, assuming this lexical activation propagates further into a semantic system (not implemented in the IAM, but see McClelland, 1987), during the course of the sequential processing, activation from individual letters propagates into the system and adds to the cumulative activation at the word level. Concurrently, this word-level activation feeds back to the letter level to facilitate subsequent recognition of the word's letters. The strength of this top-down support is a function of the degree of partial world-level activation. We assume that, in a more general system including semantics, the degree of higher-level activation would scale not only with frequency (as in the IAM) but also with imageability, such that words of higher frequency or imageability would be more active, and hence provide stronger top-down support for letter activations, than would words of lower frequency or imageability. Consequently, the system would

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[1] Although the IAM does not incorporate mechanisms for overt or covert shifts of attention, the model could be extended to include such a mechanism (see, e.g. Hinton, 1990), and other word reading models that are consistent with cascaded, interactive processing do contain such mechanisms (e.g. Plaut, McClelland, & Seidenberg, 1995)

converge more quickly on responses for high-imageability and high-frequency items compared with low-frequency and low-imageability items.

A further determinant of the degree of the strength of top-down support is the time over which higher-level activation can accumulate, and it is this time factor that gives rise to an interaction between these lexical variables and word length. Given that longer words take longer to process in LBL reading, their higher-level representations have longer to integrate bottom-up support and, therefore, produce stronger top-down effects on performance. Thus, there is more opportunity for frequency and imageability to influence the recognition of seven-letter words compared with three-letter words. Indeed, even in normal subjects, there is a significant interaction of word length and frequency; Weekes (1997) found that, whereas, there was no effect of word length on the naming latency of high-frequency words, there was a small effect for low-frequency words and a more marked effect for nonwords.

The essence of our account, then, is that LBL readers make use of the same cascaded, interactive system as normal readers but are prompted to resort to sequential processing more often (manifest either as multiple eye movements or shift of covert attention) to compensate for the degradation in visual input following the left occipital lesion. Nonetheless, the weak, parallel activation from this input propagates to higher levels of the system to engage lexical/ semantic representations partially, and these representations provide top-down support that facilitates subsequent lower-level processing. This account provides a unified explanation of both the early visual and later lexical and semantic findings in LBL reading.

In fact, an interactive theory of this general form has already been proposed to account for another type of acquired peripheral dyslexia, neglect dyslexia (Behrmann, Moscovitch, Black, & Mozer, 1990; Mozer & Behrmann, 1990). According to this theory, neglect dyslexia arises from a low-level deficit in spatial attention that affects the bottom-up processing of one-side (typically the left) of a visual stimulus. Given that reduced attention does not completely filter out information, but only lowers the likelihood that it is propagated into the system, the unattended information on the neglected side of the stimulus may still engage higher-level processes, albeit to a lesser extent than the non-neglected information. Provided that the attentional deficit is not too severe, stimulus information from the neglected side may be processed sufficiently to engage higher-level (lexical / semantic) representations. Thus, factors such as lexical status and morphological composition can still influence performance, leading to, for example, better reading of words compared with nonwords and better reading of compound words compared with two single, unrelated words (Behrmann et al., 1990; Brunn & Farah, 1991; Làdavas, Umiltà, & Mapelli, 1997; Sieroff, Pollatsek, & Posner, 1988). Thus, as in LBL reading, both "early" effects (e.g. influence of stimulus size, horizontal position, and word length) and "late" effects (e.g. influences of lexical status and morphology) may be observed in the same patient.

To this point, we have cast our views in terms of the IAM, in which the critical lexical effects are mediated by word-specific (localist) processing units. However, as alluded to earlier, this choice is to a large extent merely expository. The same lexical effects can be recast within models of lexical processing employing distributed representations (e.g. Seidenberg & McClelland, 1989; Plaut et al., 1996; Van Orden et al., 1990). In a distributed representation, words are encoded by distinct but overlapping patterns of activity, such that each word is represented by the activity of many units and each unit participates in representing many words. Within a distributed system, lexical knowledge– the fact that certain patterns of activity but not others correspond to familiar stimuli– is reflected not in the structure of the system but in the dynamics of how the units interact. Specifically, lexical knowledge in the form of learned weights on the connections between units causes the activity pattern corresponding to each word to form an *attractor*. What this means is that, when the system is in an unfamiliar pattern of activity– one that does not correspond to a known word– interactions among units alter this pattern so that it moves towards and ultimately settles to the nearest (most similar) familiar attractor pattern. A critical property of attractor dynamics for our purposes is that they can be partial; an activity pattern that is sufficiently far from the nearest attractor may still be pulled towards it (thereby reflecting its lexical properties to a degree) but may not settle it completely (which would correspond to explicit recognition). Thus, the partial activation and competition among word units in the localist IAM corresponds to the partial movement of activity patterns towards word attractors within distributed systems. Yet both types of systems employ cascaded, interactive processing such that partially degraded orthographic input is propagated throughout the system to engage these "lexical" effects to varying degrees. Thus, we claim that our account of LBL within the framework of the IAM also holds for distributed connectionist models of lexical processing.

In fact, many of the effects that we ascribe to top-down activation from word units in the IAM have been demonstrated in existing simulations of distributed attractor networks. For example, Hinton and Shallice (1991) demonstrated relative sparing of categorisation performance with poor explicit identification following lower-level damage to an attractor network that was trained to map orthography to semantics. Plaut and Shallice (1993) replicated this finding in an attractor network that mapped orthography to phonology via semantics, and also showed that, for the words the network failed to read correctly following damage near orthography, it was nonetheless well above chance ($d' = 1.80$)[2] at lexical decision for these words. Plaut and Shallice also demonstrated in a similar network that high-imageability words develop stronger semantic

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[2] This d' value is reported incorrectly as 1.36 by Plaut and Shallice (1993, p. 445).

attractors than low-imageability words– as reflected by their relative robustness to damage– if imageability is instantiated in terms of the relative richness of a word's semantic representations (i.e. how many semantic features are accessed consistently across contexts; also see Plaut, 1995). The stronger semantic attractors for high-imageability words would naturally lead to stronger top-down effects on orthographic representations, although this was not tested directly. And finally, in conjunction with the interactive theory of neglect dyslexia mentioned earlier, Mozer and Behrmann (1990, 1992) simulated the co-occurrence of many of the lower-level perceptual effects and higher-level lexical/ morphological effects observed in neglect dyslexia by damaging input to the attentional mechanism within MORSEL (Mozer, 1991), a network model that implements attractors for words using a "pull-out" network over letter-cluster units.

In contrast with the right-hemisphere account of LBL reading (Buxbaum & Coslett, 1996; Coslett & Saffran, 1994; Saffran & Coslett, this issue), our view does not specifically implicate a particular hemisphere as the locus of the lexical and semantic effects. We have argued that the simultaneous presence of visual and lexical/ semantic findings arise from a single interactive system, involving both hemispheres, and that there is no compelling reason to invoke the separate right hemisphere as the sole (or even primary) mediator of the postlexical effects. Importantly, this view entails that, in LBL readers, the reading system per se is unchanged from its premorbid state; reading behaviour arises as a consequence of degraded input to the same cascaded, interactive systems that supported normal reading premorbidly. Although we do not question the fact that the right hemisphere has some language and reading abilities (Beeman & Chiarello, 1997; Michel, Henaff, & Intriligator, 1996; Vargha-Kadem et al., 1997), our claim is that the lexical and semantic findings in LBL readers do not arise solely from the more primitive right-hemisphere reading system. Rather, these higher-level effects emerge from the residual workings of the interactive reading system that involves both hemispheres, governed by the same computational principles.

What type of evidence would support our interactive account of LBL reading? First, all patients should have an early deficit of some form that prevents the rapid and reliable activation of orthographic information. Second, variables considered to be diagnostic of later lexical processes should also influence the reading performance of these patients. In support of our position, in this paper we present a review of the existing literature as well as new empirical data obtained from seven LBL readers. As the findings will indicate, both the literature review and the empirical data are consistent with our position, showing that early visual deficits are identifiable in almost every patient (and not demonstrably absent in the remaining cases) and that, when they have been investigated carefully, strong effects of lexical variables are also observed. That a partial low-level deficit can permit partial higher-level activation (supporting lexical decision and categorisation performance) that further influences lower-level processing (producing frequency and imageability effects on reading latencies) is, on our view, a direct consequence of the cascaded, interactive nature of the normal reading system.

REVIEW OF PUBLISHED CASES OF LETTER-BY-LETTER READING

This review of the published studies of 57 LBL readers was undertaken both to document the existence of early deficits and to examine the extent to which there is higher-level processing in these patients. Although this review is intended to be as comprehensive as possible (see Table 1), we have deliberately excluded several papers. Many of the excluded papers have a different focus and do not provide sufficient detail for our analysis; for example, some papers focus on the anatomical aspects of the case rather than on the reading performance per se (Ajax, 1967; Damasio & Damasio, 1983; Greenblatt, 1973), others describe aspects of the patient's performance such as the intact reading of stenography (Regard, Landis, & Hess, 1985), an associated deficit in music reading (Horikoshi et al., 1997), or associated colour deficits (Freedman & Costa, 1992), which are unrelated to the issue at hand, and yet other papers report a rehabilitation procedure for the patient without including much detail on the patients' pre-therapy reading performance (Kashiwagi & Kashiwagi, 1989; Tuomainen & Laine, 1991). There are also a small number of papers that are not included simply because the description of the patient's reading is unclear or insufficiently detailed for our purposes (Caplan & Hedley-White, 1974; Kreindler & Ionescu, 1961), although we have generally made reference to these papers elsewhere in the text. We are also aware of posters of LBL readers presented at conferences and have not included those (with the exception of the Vigliocco, Semenza, & Neglia, 1992, because the description of the patients is sufficiently detailed). We have also come across a few papers published in other languages (El Alaoui-Faris et al., 1994), but have restricted our analysis to published English papers. Finally, to make the table as comprehensive as possible, we have included our own patients at the end. To do so, we have combined the findings from the empirical analyses we describe in the latter part of this paper with previous descriptions of these patients' reading performance.

Procedure

For this analysis, we reviewed the published papers to determine whether there was any evidence for a peripheral deficit in the patient. If the authors of a paper classified the patient's deficit as occurring early (peripherally or prelexically), this was counted as positive evidence. We also took as positive evidence a reported perceptual difficulty in single-letter processing or identification or a more general perceptual deficit in which other visuoperceptual abilities are impaired, even if the authors did not classify the patient in the peripheral group per se. We also determined whether there was evidence for lexical and semantic effects in these same LBL readers. This consisted of reviewing the reported papers and determining whether there was positive evidence for: (1) covert reading under brief exposure (either in lexical decision or semantic categorisation tasks), (2) a word superiority effect, (3) effects of frequency, imageability, regularity, and part-of-speech on naming words, and (4) effects of frequency and imageability on lexical decision. With regard to the word superiority effect, we simply noted the better report of items in words over nonwords in any experimental paradigm (not solely the standard Wheeler-Reicher type task) and did not take special note of a pseudoword effect (better report of items in legal pseudowords over illegal nonwords).

In analysing the effects of lexical variables such as frequency, imageability etc., we considered their impact on naming and on lexical decision under both brief and prolonged exposure duration. We have only documented whether these variables significantly influence the patient's reading performance (i.e. as a main effect) in either reaction time or accuracy. Very few studies present the interaction between these different variables and word length (see Doctor, Sartori, & Saling, 1990, for an exception) so we do not consider the interactions in our tabulation, although we consider it to be crucial for our interactive view. Finally, some studies (for example, Buxbaum & Coslett, 1996; Coslett & Monsul, 1994; Coslett & Saffran, 1989a; Coslett et al., 1993; Doctor et al., 1990; Friedman et al., 1993; Shallice & Saffran, 1986) assess the reading of other higher-order lexical, orthographic, or semantic variables, for example a comparison of performance on suffixed/ pseudosuffixed words. Although these other variables are relevant to the issue under investigation, reports of this were too few to warrant a separate category.

Because this analysis is retrospective, there are obviously a number of problems. In many cases, the critical variables are not tested (or perhaps tested but not reported or analysed statistically). Even when they are tested systematically, the dependent variable is often a measure of accuracy rather than reaction time. Yet the effects in LBL reading are typically more robust (and perhaps only evident) in reaction time, given the high degree of accuracy in many cases. We have not differentiated between the various dependent measures and simply note the presence of the main effect using either metric.

Results

Evidence for a Peripheral Deficit

Many researchers have previously observed that LBL readers invariably perform poorly on tests of letter recognition, although, to date, there has been no substantiation of this claim. Almost all the patients we reviewed had some noticeable problem with letter processing, as is evident from the first column of Table 1. Indeed, 50 of the 57 patients have a positive check mark in the first column. Although some of these patients were highly accurate on naming single letters for an unlimited exposure, a finding which might suggest no obvious impairment, the time to name the individual letters is often abnormally long. For example, DR (Doctor et al., 1990) correctly identified 24/ 26 lower-case and 25/ 26 upper-case letters, but

Table 1. Review of Published Cases of Letter-by-letter Reading

		Brief Exp					*Naming*			*LD*	
Patient	*Study*	*PL*	*LD*	*SC*	*WSE*	*Fr*	*Im*	*Rg*	*PS*	*Fr*	*Im*
DM	Arguin & Bub (1993, 1994a,b)	✓	✓	✓	✓	✓	–	–	–	✓	–
	Bub & Arguin (1995)										
EL	Behrmann et al. (1998)	✓	×	×	–	✓	✓	–	–	–	–
	Montant et al. (1998)										
IH	Bowers, Arguin, & Bub (1996)	✓	–	–	✓	✓	–	✓	–	–	–
	Bowers, Bub, & Arguin (1996)										
	Arguin et al. (this issue)										
JV	Bub, Black, & Howell (1989)	✓	–	–	✓	×	–	–	–	–	–
n/ a	Buxbaum & Coslett, (1996)	✓	–	–	–	×	✓	×	✓	–	–
1	Coslett & Saffran (1989a)	✓	✓	✓	–	+	✓	–	✓	✓	×
2	Coslett & Saffran (1989a)	✓	✓	✓	–	–	–	–	+	✓	×
3	Coslett & Saffran (1989a)	✓	✓	✓	–	✓	✓	–	✓	✓	×
4	Coslett & Saffran (1989a)	✓	✓	✓	–	–	–	–	–	✓	–
JWC	Coslett et al. (1993)	✓	✓	✓	–	–	–	–	–	✓	✓
DR	Doctor et al. (1990)	✓	✓	✓	–	–	✓	–	–	–	–
RE	Feinberg et al. (1995)	✓	✓	✓	–	×	×	–	–	✓	✓
n/ a	Friedman & Alexander (1984)	✓	–	×	–	–	–	–	×	–	–
BL	Friedman & Hadley (1992)	?	–	–	✓	✓	×	✓	×	–	–
TL	Friedman et al. (1992)	✓	–	–	–	–	–	–	✓	–	–
n/ a	Grossi et al. (1984)	✓	–	+	–	–	–	–	–	–	–
DC*	Hanley & Kay (1996)	✓	–	–	✓	–	–	–	–	–	–
n/ a	Henderson et al. (1995)	✓	–	–	–	–	–	×	–	–	–
PM	Howard (1991)	✓	×	×	–	–	×	×	–	–	–
KW	Howard (1991)	✓	×	✓	+	✓	✓	×	–	–	–
PD	Kay & Hanley (1991)	✓	–	–	×	✓	✓	–	–	–	–
	Hanley & Kay (1992, 1996)										
L	Kinsbourne & Warrington (1962)	✓	–	–	–	+	–	–	–	–	–
P	Kinsbourne & Warrington (1962)	✓	–	–	–	+	–	–	–	–	–
S	Kinsbourne & Warrington (1962)	✓	–	–	–	+	–	–	–	–	–
C	Kinsbourne & Warrington (1962)	✓	–	–	–	+	–	–	–	–	–
n/ a	Landis et al. (1980)	×	–	+	–	–	–	✓	✓	–	–
ON	Lazar & Scarisbrick (1993)	✓	–	–	–	–	–	–	–	–	–
CC	Levine & Calvanio (1978)	✓	–	–	✓	–	–	–	–	–	–

RT	Levine & Calvanio (1978)	✓	–	–	✓	–	–	–	–	–	–
GC	Levine & Calvanio (1978)	✓	–	–	×	–	–	–	–	–	–
CP#	Montant, Nazir, & Poncet (this issue)	✓	–	–	–	–	–	–	–	–	–
MW	Patterson & Kay (1982)	✓	×	×	–	–	–	–	–	–	–
CH	Patterson & Kay (1982)	✓	×	–	–	–	–	–	–	–	–
TP	Patterson & Kay (1982)	✓	×	×	–	–	–	–	–	–	–
KC	Patterson & Kay (1982)	✓	–	×	–	–	–	✓	–	–	–
SP	Perri et al. (1996)	✓	–	–	–	✓	–	✓	–	–	–
EW	Price & Humphreys (1992)	✓	×	×	–	✓	–	+	–	–	–
HT	Price & Humphreys (1992)	✓	×	×	–	+	–	×	–	–	–
SA	Price & Humprheys (1995)	✓	×	–	–	–	–	–	–	–	–
WH*	Price & Humphreys (1995)	✓	×	–	–	–	–	–	–	–	–
HR*	Rapp & Caramazza (1991)	✓	–	×	–	✓	×	–	–	–	–
PT	Rapcsak et al. (1990)	?	–	–	–	–	–	×	×	–	–
	Schacter et al. (1990)			–	–	–	–	–	–	–	–
WL	Reuter-Lorenz & Brunn (1990)	✓	–	–	✓	–	–	×	–	–	–
ML	Shallice & Saffran (1986)	✓	✓	✓	–	–	–	–	–	✓	–
n/ a	Stachowiak & Poeck (1976)	✓	–	–	–	–	–	–	–	–	–
BY	Staller et al. (1978)	✓	–	–	–	–	–	–	–	✓	✓
BD	Vigliocco et al. (1992)	?	×	–	✓	–	–	–	–	–	–
RG	Vigliocco et al. (1992)	?	×	–	✓	–	–	–	–	–	–
RAV	Warrington & Shallice (1980)	?	–	×	×	–	×	–	–	–	–
JDC	Warrington & Shallice (1980)	?	–	–	–	–	–	–	–	–	–
Our Patients											
DS	Behrmann, Black & Bub (1990) Behrmann & Shallice (1995) Sekuler & Behrmann (1996)	✓	–	–	✓	✓	✓	–	–	–	–
PC		✓	–	–	–	✓	×	–	–	–	–
MW	Sekuler & Behrmann (1996)	✓	–	–	–	✓	✓	–	–	–	–
DK		✓	+	+	–	✓	✓	–	–	–	–
MA	Sekuler & Behrmann (1996)	✓	–	–	–	✓	✓	–	–	–	–
IS	Behrmann & McLeod (1995)	✓	–	–	✓	✓	×	–	–	–	–
TU	Farah & Wallace (1991) Sekuler & Behrmann (1996)	✓	–	–	–	✓	✓	×	–	–	–

PL = Prelexical; LD = Lexical Decision; SC = Semantic Category; WSE = Word Superiority Effect; Fr = Frequency; Im = Imageability; Rg = Regularity; PS = Part of Speech; ✓ = evidence for; × = evidence against; + = some evidence for; ? = unclear; – = not tested; * = left-handed; # = ambidextrous.

the time to do so was 2.03 sec and 1.75 sec, respectively. Patients PD and DC (Hanley and Kay, 1996; Kay & Hanley, 1991) also made a relatively small number of letter misidentifications (26/ 26 and 25/ 26 lower-case letters, respectively) but their speed of processing was dramatically slowed relative to a control subject. This slowing was particularly evident when the two patients performed a letter-matching task and made same/ different judgements on two letters (e.g. aR) presented simultaneously or sequentially (500msec SOA). The control subject showed a slight reaction time advantage for the sequential over the simultaneous condition. PD's reaction times were similar to the control in the sequential condition but he took roughly 400msec longer when the letters were presented simultaneously. DC was even slower than PD on the simultaneous condition but, in addition, was far slower than PD or the control subject on the sequential condition (see Hanley & Kay, 1996, Fig. 4). These findings suggest that neither of these patients process letters normally.

There remain seven contentious cases for whom a letter processing deficit is not clearly apparent. We consider each in turn. Patient BL scored 25/ 26 correct on upper-case naming but we have no indication of the time required to do so (Friedman & Hadley, 1992). There is also no report of other letter processing tests nor tests of general visual processing for BL. We do know, however, that BL showed a very steep serial position curve in reporting letters from four-item strings under tachistoscopic presentation. Whereas performance was reasonably accurate for items in position 1 (almost at 100% on word stimuli), performance dropped below 50% for items in position 4 of words (and close to floor for items in position 4 of nonwords). A similar account of preserved accuracy but abnormal speed of processing may account for the patient reported by Landis et al. (1980). Although this young man accurately identified single letters, this was done so hesitantly, especially when the letters were intermixed with numbers (see similar observation by Polk and Farah, reported in Farah, 1997). Both BD and GR (Vigliocco et al., 1992) performed similarly to normal control subjects on a letter-matching task under simultaneous and sequential conditions, suggesting that speed is normal for them. We do not, however, know what their accuracy was on this task. When presented with single letters for 2 sec on a separate task, a duration that is extremely long for normal subjects, BD identified 88% correctly and GR 90%. The errors produced were all visual confusions with the target (for example, d/ b, v/ u). It appears then that even these two subjects might not have normal letter processing and that the apparently normal reaction times may be due to a speed-accuracy trade-off.

Both RAV and JDC (Warrington & Shallice, 1980) appear to identify single letters particularly well, even when separated by either alphanumeric characters (Expt. 1) or when flanked by distractors (Expt. 2). Nonetheless, both patients are more affected than normal readers by items presented in script compared with print. Moreover, RAV appear to show an interaction in reaction times between script/ print and word length. For three-letter words, the difference between script/ print is 2.4 sec, whereas the difference increases to 5 sec for seven-letter words. Both the differences between script and print and the interaction with word length have been taken to be strong indicators of an early visual processing deficit (Farah & Wallace, 1991). Lastly, PT (Rapcsak, Rubens, & Laguna, 1990; Schacter et al., 1990) performed well on tasks of letter discrimination (letters vs. nonletters or mirror-reversed letters; 100%), cross-case matching (95%), and pointing to letters (100%). Although accuracy was low for letter naming, with a score of only 15/ 26 for upper-case and 15/ 26 for lower-case letters, this might possibly be a result of an anomia rather than of a letter recognition deficit per se. Once again, however, we have no indication that PT performs these simple letter tasks at normal speeds. Whether her letter processing is indeed normal, therefore, remains unanswered at present.

The findings from the review thus far suggests that there is no single subject for whom letter recognition is definitively normal. As is evident, patients may be impaired in speed, even if not in accuracy, and sometimes the converse is true. Additionally, as noted by Patterson and Kay (1982), even high accuracy might not be a satisfactory indicator of letter processing skill; an accuracy score of 85% in letter naming may not seem too serious an impairment but this might become very debilitating in a more taxing task when the subject is required to represent letters for the purposes of word recognition.

An important question that remains, then, is what are the consequences of this peripheral deficit for reading. We have assumed that the letter processing deficit plays a causal role in LBL reading and that, because the weakened activation is insufficient, subjects make both overt and covert gaze shifts to enhance the letter activation. A correlation between reading speed and the accuracy of single-letter identification, suggestive of a strong relationship between the two, was described by Shallice (1988) in a small group of eight LBL readers. This correlation, however, does not seem to be perfectly upheld, as a recent study described two patients with fairly similar letter recognition patterns but with very different performance in word recognition (Hanley & Kay, 1996). The exact relationship between poor letter and word recognition processes is beyond the scope of this paper. Also beyond the scope of this paper is whether the poor letter processing derives from an even more fundamental perceptual problem, as we (amongst others) have argued elsewhere (Behrmann et al., 1998; Sekuler & Behrmann, 1996; see also Chialant & Caramazza, this issue), or whether it is restricted to alphanumeric stimuli. For the present purposes, we are only interested in establishing that, across the population of LBL readers, there is strong evidence for a peripheral deficit that adversely affects letter processing. We now turn to the question of whether variables associated with later stages of word recognition are also observed in this population.

Evidence for Lexical and Semantic Effects

The major problem we encountered in this review is that very few studies systematically document the higher-level effects in the reading behaviour of LBL readers (especially in cases who are a priori diagnosed as having a peripheral impairment). Because the interest has traditionally been on the effect of word length, unconfounded by other variables, stimuli are standardly matched on frequency and imageability, for example, and length alone is manipulated. In those few cases where these other variables are tested and reported, performance is measured in terms of accuracy rather than the more sensitive measures of reaction time and the statistical analyses of both the main effects and their interaction are often omitted. Nevertheless, we have reviewed the literature and present the findings in Table 1. We discuss covert or tacit reading, the word superiority effect, and the influence of lexical variables separately.

Positive evidence for preserved implicit reading under brief exposure is found in only a small number of cases. Aside from the studies by Coslett and Saffran and colleagues, who document covert effects in five LBL readers, definitive covert processing is reported only in patients ML (Shallice & Saffran, 1986), KW (Howard, 1991) (in semantic but not lexical decision tasks), DR (Doctor et al., 1990), and RE (Feinberg, Duckes-Berke, Miner, & Roane, 1995). A positive trend in this direction is also noted in Grossi, Fragassi, Orsini, Falco, & Sepe (1984) and in Landis, Regard, and Serrat (1980), and we have some preliminary evidence of relatively good semantic categorisation under brief exposure in one of our own patients, DK. By contrast, the absence of covert abilities has been documented in several studies (Howard, 1991; Patterson & Kay, 1982; Price & Humphreys, 1992, 1995; Vigliocco et al., 1992; Warrington & Shallice, 1980) and in some of our own studies (Behrmann, Black, & Bub, 1990; Behrmann & McLeod, 1995; Behrmann & Shallice, 1995). Why there are such marked individual differences is taken up further in the General Discussion.

With respect to the word superiority effect, as with the covert reading, only a small subset of the population has been tested and so strong claims about the effect of orthographic context should be made cautiously. Of the 16 reported instances, 12 patients show a word superiority effect, one shows a trend in that direction, and the remaining 3 are not influenced by orthographic context. One of these 16 patients is DS who, in our initial testing in the first year post-stroke, did not show a word superiority effect (Behrmann et al., 1990) on a Wheeler-Reicher type task. In subsequent testing, roughly 5 years post-stroke, DS did show a word superiority effect on one task, reporting the beginning and end letters more accurately from words than from nonwords. DS, however, did not show an advantage for words in a second task that required a decision on whether two strings (both words or both nonwords) placed one above the other for an unlimited duration were the same or different (Behrmann & Shallice, 1995).

A suggestion for why the word superiority effect is only observed in some but not other cases has been made by Farah and Wallace (1992; see also Farah, 1997). They observed that at least two of the patients who showed the word superiority effect had a letter recognition profile that was similar to normal subjects (referred to as an "ends-in" profile). At least two of the cases who did not show a word superiority effect showed a gradient of letter recognition, with best performance on the initial letter and decreasing accuracy across serial position. They attribute the presence of a word superiority effect to the presence vs. absence of the letter-by-letter gradient. In fact, even within a single subject, one can see this at work: Bub, Black, and Howell (1989) found that their patient showed a word superiority effect in only one experiment and it was in this single experiment that the patient did not show a left–right gradient in accuracy across letter positions. The suggestion that strategy determines the presence of a word superiority effect may well explain the variance in the data on this effect in LBL readers. In fact, even in normal subjects, focusing sequentially on individual letters affects top-down effects; specifically, the word superiority effect is reduced when normal subjects attempt to read letters in particular positions rather than distributing their attention across the entire stimulus array (Johnston & McClelland, 1980). Unfortunately, the patients who show covert reading are generally not tested for a word superiority effect and vice versa, making it difficult to reach conclusions about the relationship between implicit reading and higher-order orthographic processing.

Of the 26 subjects tested for effects of frequency on word naming, 17 are influenced by frequency either in reaction time or in accuracy, including all 7 of our patients. A further six subjects show some effect of frequency (statistics are not always available) and only three subjects are not obviously influenced by frequency. One of the 3 subjects who does not show a frequency effect is patient RE (Feinberg et al., 1995), whose performance is so poor that he is unable to read even a single word aloud. The absence of a frequency effect then might simply result from a floor effect in this patient.

Of the 19 subjects tested for imageability in naming, again in reaction time or in accuracy, 12 show a positive result (5 of our 7 patients). Again, one of the subjects who does not is patient RE, and this may again result from a floor effect. Five out of 14 subjects show an effect of regularity on naming and 5 out of 9 subjects show an effect of part-of-speech on their performance. Few subjects are tested on these lexical variables in lexical decision, as is evident in the final two columns of Table 1. All eight patients for whom frequency data are available for lexical decision are affected by frequency. There are imageability data for only five patients in lexical decision, and two of these show a positive finding.

Taken together, the review of the later lexical and semantic effects is not strongly conclusive. Too few patients are tested on the higher-order variables and when they are, they are usually not tested on all of the different tasks or different variables, making comparisons difficult. One finding worth noting, however, is that across the population of 57 subjects, there are only 13 subjects who do not show any lexical or semantic effects on any of the measures. A further four patients have not been tested on any of the measures and so the presence of the later effects for them remains indeterminate (Lazar & Scarisbrick, 1973; Montant, Nazir, & Poncet, this issue; Stachowiak & Poeck, 1976; Warrington & Shallice, 1980; patient JDC). A central finding of the review, then, is that the great majority of patients show some form of lexical/ semantic effects on reading performance.

Summary of Review

The evidence for a peripheral deficit is strongly supported by the review and there is no convincing counter-evidence. In those few cases where accuracy or even reaction times appear to be within normal limits, it is often the case that this could result from a speed–accuracy trade-off and poorer performance is generally seen on the complementary metric. These results substantiate the initial part of our account and endorse the previous observation of a deficit in letter processing that is common to all LBL readers. Unfortunately, strong conclusions about later lexical and semantic effects are not as obvious from this review, as there are too many empty cells in the database. An important finding, though, is that these later effects are not restricted to a small subgroup of the population and there is at least partial evidence for them in more than two-thirds of the patients.

EMPIRICAL DATA FROM LETTER-BY-LETTER READERS

Given the paucity of data on the lexical and semantic effects in LBL patients, we have undertaken a retrospective analysis of data collected from seven who have participated in our studies over the last decade. In this analysis, we specifically explore whether both a peripheral visual processing deficit and the effect of lexical and semantic variables are observable across this group. All of these patients are highly accurate in single-letter identification under unlimited exposure duration although, as pointed out previously, this in no way indicates that letter recognition is normal and, in some instances, we know that it is not (for example, patient DS; see Behrmann & Shallice, 1995). It is also the case, though, that these patients are much more impaired than their control counterparts in identifying two or three random letters presented under brief exposure (for example, patient DS, see Behrmann, Black, & Bub, 1990; patient IS, Behrmann & McLeod, 1995).

Our view of LBL reading predicts that, even in those patients with a peripheral deficit, one would still see effects of word frequency and imageability on performance. On this account, we therefore predict significant main effects of word frequency and imageability that will reflect the strength of the top-down contribution. We also predict that as word length increases, the difference between high- and low-frequency and the difference between high- and low-imageability items will increase, given that for longer words there is more opportunity for top-down effects to influence performance.

Subjects

Seven LBL readers are included in this analysis. Five of them have been described in detail in other publications (see final seven rows of Table 1 for citations), whereas the remaining two, PC and DK, have only been tested fairly recently. The subjects will only be described briefly in this paper and the reader is referred to the more detailed publications for further information. All subjects are right-handed and native speakers of English. Biographical information and anatomical details of their lesions as well as some reading data (which will become relevant later) are provided in Table 2.

Evidence for an Early, Peripheral Deficit

As mentioned previously, all of these patients do reasonably well, although not perfectly, on single-letter identification under unlimited exposure duration, although performance is markedly poorer than normal subjects when brief exposure is used. For example, patient DK performed perfectly on single-letter identification when the letters were presented for an unlimited duration. He made 30% errors when the letters were presented for 250msec (even without a mask) and 40 errors at 100msec exposure duration. Similar patterns are seen in other patients. Those patients who perform well on single-letter identification even at brief exposure duration may then do poorly when two or three letters are present; patient IS, for example, did well with single letters even at the presentation limits of the computer (17msec; no masking) but performance was poor on two- and three-item arrays even when exposure duration was more than tripled (Kinsbourne & Warrington, 1962; Levine & Calvinio, 1978). Relative to normal subjects, who can identify letters at 10msec with masking (Sperling & Melcher, 1978), these patients are much slower at letter identification.

Four of the seven subjects participated in the study by Sekuler and Behrmann (1996), which documented the ability of LBL patients to process visual stimuli that are not alphanumeric. In that study, patients completed three different experiments: they performed perceptual fluency judgements under time pressure, made same/ different judgements to contour features that appeared on objects, and searched for a target in visual displays that increased in the number of distractor items. In all three experiments, these patients performed significantly more poorly than their control counterparts. These findings suggest that all four of these patients suffered from a general perceptual deficit, which extended beyond their ability to deal with orthographic material. It is this more fundamental visual perceptual problem, we claim, that gives rise to their LBL pattern in the first instance.

Five of these same seven subjects (excluding PC and TU) have also participated in a recent study examining their accuracy and reaction time to name black-and-white line drawings of high and low visual complexity, taken from the Snodgrass and Vanderwart (1980) set of pictures (Behrmann et al., 1998). Whereas age-

Table 2. Biographical and Lesion Details of the Seven Letter-by-letter Readers

Patient	Age[a]	Etiology	CT Scan Results	Other Relevant Behaviours	Freq[b]	Image[c]
DS	37	Posterior cerebral artery occlusion; migrainous	L occipital infarction	upper quadrantanopia	397**	72**
PC	63	Resected meningioma	L occipitotemporal (area 19/ 37 boundary)	R homonymous hemianopia	1651*	3039**
MW	67	Infarction	L occipital lobe infarction	R homonymous hemianopia; ensuing depression	570*	292**
DK	65	Posterior cerebral artery infarction & mass effect	L occipital lobe infarction	R homonymous hemianopia	588**	443
MS	37	Closed head injury	No focal CT lesion; bilateral frontal slowing EEG	R homonymous hemianopia; surface dysgraphia	1552**	1629*
IS	46	Posterior cerebral artery infarction	L occipital-temporal region including hippocampus, fusiform and lingual gyri	R upper quadrantanopia; mild memory deficit; surface dysgraphia	540**	99*
TU	56	Resected arteriovenous malformation	L occipital haemorrhage; additional L temporal damage	R homonymous hemianopia; R hemiparesis; marked anomia	968**	388

[a] Age refers to the age at which the initial testing took place. Some patients have participated in subsequent follow-up studies and the age of testing is then obviously different.

[b] Frequency score in msec.

[c] Imageability score in msec.

*$P < .05$; $P < .01$.

matched normal control subjects showed a 152msec reaction time advantage for low-complexity items over high-complexity items, the LBL readers (with the possible exception of patient MW) showed a disproportionate increase in reaction times to name the high-complexity items. Averaged across the patients, low-complexity items were named 530msec faster than their high-complexity counterparts. Taken together, these data suggest that these patients have a deficit that affects their processing of several classes of visual stimuli and that, under rigorous testing conditions, this more widespread perceptual deficit may be uncovered.

Based on these findings, we can definitively conclude that there is an early deficit in six of the seven LBL readers. We do not have sufficient information about PC, but the limited data obtained from her single-letter processing suggest that she too may be impaired in early stages of processing prior to activating an orthographic representation. Whereas she is able to identify accurately all three letters from a random letter string (e.g. YFS) presented for an unlimited exposure duration, she only identifies about 70% of the letters when the exposure duration is around 1sec, suggesting that her letter identification threshold is probably abnormally high. Unfortunately, PC died before we were able to obtain any further data.

Evidence for Lexical and Semantic Influences on Word Recognition

There are a number of different lexical variables that might potentially serve as markers of higher-order processing in a naming latency task, including effects of word frequency, differences between word and nonword reading (word superiority), part-of-speech effects, and an effect of imageability or concreteness (Coltheart, 1980; Warrington & Shallice, 1980). We have chosen to use word frequency and imageability, both of which are assumed to be reliable indicators of access to a preserved word form system (Kay & Hanley, 1991), and for which there is likely to be sufficient data for analysis. These two variables have also been studied extensively in normal readers and there are several models detailing their specific effects in word recognition. A recent analysis of the effects of frequency, for example, shows that word frequency is a critical determinant of the speed of lexical access (what Monsell et al. referred to as “lexical identification”) (Monsell, Doyle, & Haggard, 1989). Frequency effects can be explained, depending on whether one's model is localist or distributed, as arising from higher resting levels of activation, lower thresholds, or increased bias on the weights for high-frequency compared with low-frequency words (McClelland & Rumelhart, 1981; Morton, 1979; Plaut et al., 1996; Seidenberg & McClelland, 1989). Thus, the speed by which a word is recognised is determined by the time to reach threshold or for the network to settle on an interpretation of the stimulus; the time will thus be less for high-frequency than for low-frequency items.

Imageability effects have not received as much press as frequency effects, although there is little doubt that the influence of imageability on performance arises at later stages and is indicative of semantic processing. Strain et al. (Strain, Patterson, & Seidenberg, 1995), for example, showed that normal readers are slower and make more errors on low-imageability than high-imageability words but only for low-frequency exception words. This three-way interaction between imageability, frequency, and regularity is explained with reference to a system in which the time to translate orthography to phonology varies. When this translation process is slow or noisy, as is the case for low-frequency exceptions, words with rich semantic representations (i.e. high-imageability words) are most likely to benefit from this interaction. This interpretation of how later effects (semantic representations in this case) interact with somewhat earlier processes (translating orthography to phonology in this case) is consistent with the interactive account of LBL reading that we have proposed.

Stimuli and Procedure

To examine the effects of frequency and imageability on the reading behaviour of LBL readers, we analysed naming latency data for single words collected from these seven patients. At some point during their testing, these patients read aloud lists of 60 words presented in a variety of fonts and sizes containing 20 items each of 3-, 5-, and 7-letter words. These lists and forms of presentation are used standardly in our laboratory to measure the naming latency of LBL readers and patients typically read three of four such lists during their preliminary testing. We have included as many word lists as possible for each subject apart from those lists in which the words were presented in script or cursive font. Frequency and imageability are orthogonally crossed with word length in each list. The cut-off for frequency was 20 per million, with items below that classified as low in frequency and items above that classified as high in frequency (Kučera & Francis, 1967). Imageability is standardly hand-coded but, for the purpose of this analysis, word imageability was taken from the MRC Database; items which did not have an imageability rating in this listing were excluded. Again, imageability was converted to a categorical variable with items exceeding 525 being classified as high in imageability and those below this cut-off as being low in imageability.

In all cases, the words were presented on a computer screen to the left of central fixation to circumvent the right visual field defect that was present in all patients. The words were right-justified so that the final letter of each word appeared in the character space just to the left of fixation. The standard procedure was as follows: a central fixation point appeared for 500msec. After a 1 sec delay, the word appeared and remained on the screen for an unlimited duration until a response was made. The visual angle subtended by stimuli of three, five- and seven-characters in length were 1.5°, 2.4° and 3.6° respectively. Words were presented in black font against a white background. Both reaction time (via a voice-relay-key) and accuracy (recorded by the examiner) was measured. In all patients, except in the case of DK, presentation and timing was controlled by Psychlab (Bub & Gum, 1988) and a Macintosh Classic or IIci was used, whereas for DK, presentation was controlled by PsyScope (Cohen, MacWhinney, Flatt, & Provost, 1993) on a Powerbook 540C.

Results

Reading responses to a total of 24 word lists (*N* = 1440, cumulative across subjects) were collected from the subjects. A number of trials, however, were excluded for a variety of reasons: imageability ratings were unavailable for 315 trials, patients made errors on 168 trials, the microphone was not triggered or the subject coughed on a further 50 trials and 2 trials were extreme outliers from the subjects' reaction time distribution (more than four SDs from the mean). A repeated-measures ANOVA with word length crossed with word frequency and word imageability was performed on the remaining 905 trials. Subject was included as a further between-subject variable so that profiles for the individual subjects could be drawn up. We note that the amount of data per subject is not equivalent (given that different subjects read differing numbers of lists and error rates differed) and so the estimates of the effects are better for some subjects than for others; subject PC has the least data and her performance is the most variable (see all Figs). As mentioned earlier, unfortunately PC died before we were able to collect further data. The number of trials per subject is included in both Figs 2 and 3.

The reaction time (RT) to name a word as a function of string length is plotted for each subject individually in Fig. 1. Along with each line is the value of the slope calculated for that subject in a linear regression analysis, with RT set against word length. As is evident from this figure, subjects differ fairly dramatically in their base reaction time [$F(6,821) = 118.7$, $P < .0001$]. Also evident from this figure is the significant main effect of word length [$F(2,821) = 167.3$, $P < .0001$], with a mean across all subjects of 1442, 2465, and 3520msec for 3-, 5-, and 7-letter words, respectively, indicating that roughly an extra 500msec is required to process each additional letter.

Although every subject shows the hallmark monotonic relationship between reaction time and word length, subjects differ in the slope or increase in RT per-each additional letter [$F(12,821) = 18.3$, $P < .0001$]. At the highest extreme is MA, who shows an increase of 1409.3msec per letter. At the bottom extreme, the mildest LBL subject, DS, who had a stroke roughly 10 years ago (Behrmann, Black, et al. 1990), shows an increase of 97msec per letter. Although this latter slope is rather flat in comparison with the population of LBL readers, it must be noted that this increase is roughly three times greater than the maximum of 30msec per letter needed by normal subjects in naming words presented to the left visual field (Henderson, 1982; Young & Ellis, 1985). Indeed, some studies have demonstrated that normal subjects show no effect at all of word length on naming latency for words (Schiepers, 1980; Weekes, 1997). Any reliable increase in latency as a function of string length might, therefore, be considered abnormal. On the same word lists as those read by our LBL readers, normal subjects, who served as control

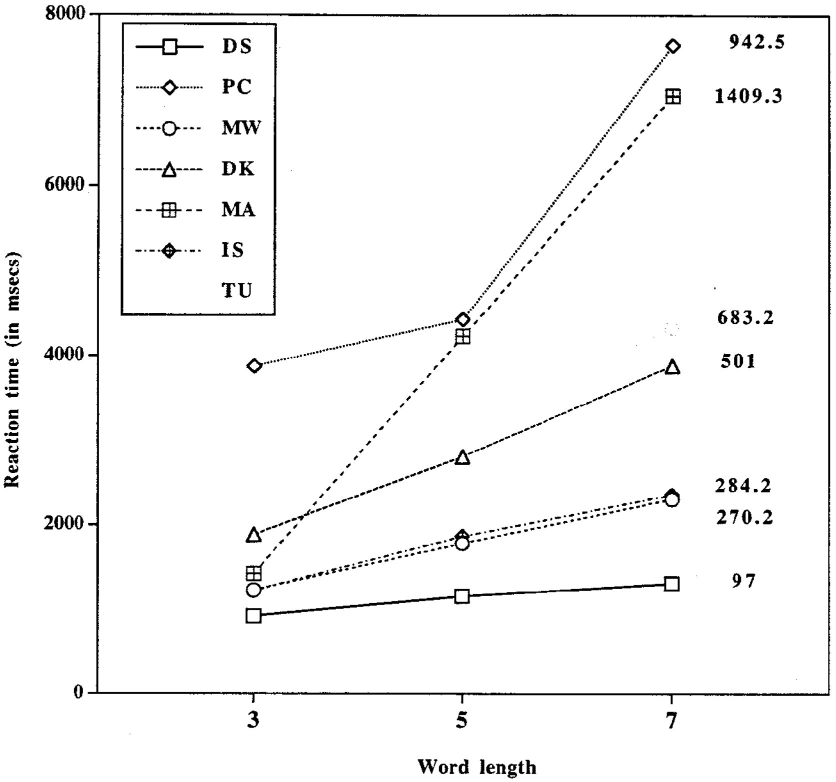

Fig. 1. Mean reaction times for the seven letter-by-letter readers as a function of word length and the slope of the regression function.

subjects for some of the patients (some of whom are elderly), typically showed very flat slopes; control subject BR (Behrmann & McLeod, 1995), for example, showed an increase in RT of 15.3msec for each additional letter in a word, whereas control subjects AS and JD, averaged, show a slope of 9.4msec (Behrmann et al., 1998). Relative to these values, all of our subjects are markedly abnormal, including the mildest subject, DS.

Importantly for the present purposes is that, across these seven subjects, word frequency significantly affected reaction time [*F*(1,821) = 41.8, $P < .0001$]; high-frequency words ($N = 566$) were named, on average, 884msec faster than low-frequency ($N = 339$) words. The differences between the mean RTs for high- and low-frequency words for each patient are included in Table 2. A one-way ANOVA performed separately for each subject, comparing RTs for high- and low-frequency words, yielded significant values for every subject. The effect of frequency did not vary as a function of word length, collapsed across subjects [*F*(2,821) = 2.06, $P < .13$], and the frequency effects remained relatively constant across subjects [$F(6,821) = 1.17$, $P < .32$]. However, the three-way interaction of frequency by word length by subject is significant. This is seen in Fig. 2 which shows the mean reaction times for the individual patients for high- and low-frequency words as a function of word length. It is important to note that the y-axis on this figure differs across subjects. As is evident from this figure, the difference between high- and low-frequency words increased as a function of word length, although this held to a varying degree across subjects [$F(12,821) = 1.76$, $P < .05$]. The interaction is particularly evident in patients DS, MW, DK, and IS and, to a lesser degree, in MA. It is only in patient PC (for whom the least data are available) that this function does not follow the predicted pattern. Interestingly, there is a good correlation across the subjects between the severity of the LBL reading (defined by the slope of the regression curve on the naming latency data in Fig. 1) and

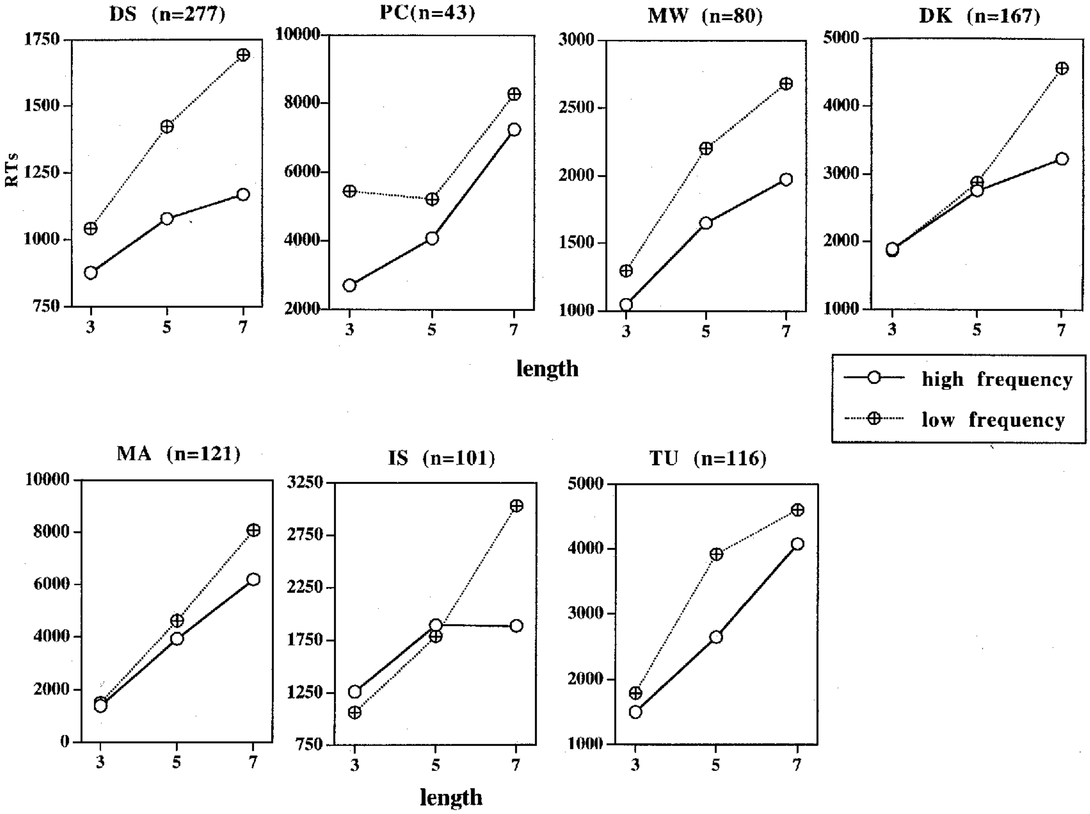

Fig. 2. Reaction times for the seven letter-by-letter readers, plotted individually, as a function of word length for high- and low-frequency words.

the effect of frequency on performance (defined as the msec difference between naming high- and low-frequency words ($r^2 = .85$, $P < .005$), indicating a close relationship between severity and frequency, as would be predicted by our account.

Word imageability also significantly influenced naming [$F(1,821) = 52.3$, $P < .0001$]; high-imageability words ($N = 455$) were reported 696msec faster than low-imageability words ($N = 450$) on average, although the extent of this difference varied across subjects [$F(1,821) = 10.6$, $P < .0001$]. The differences in mean RT for high- and low-imageability words for each subject are presented in Table 2 along with the significance values obtained from a one-way ANOVA performed separately for these data. Five of the seven patients showed a significant difference between high- and low-imageability stimuli. The advantage for high- over low-imageability words was, however, influenced by word length [$F(2,821) = 20.7$, $P < .0001$]; the difference between high- and low-imageability words is 169msec for 3-letter words compared with an 852msec difference for 7-letter words. Again, this difference as a function of word length varies across subjects. This is evident in Fig. 3 in which the mean reaction times for the individual patients with high- and low-imageability words are plotted as a function of word length. Although there is a general trend in all subjects (except for subject DS) for the difference between high- and low-imageability words to become increasingly magnified as word length increases [$F(12,821) = 7.6$, $P < .0001$], these data are more variable than the frequency data and the pattern across subjects is less clear. The expected interaction, however, is clearly evident for patients PC, MW, DK, MA, and TU and less clear in DS and IS (although the increase in 5-letter words is perhaps a bit more evident for IS). It is also worth noting that PC's data are particularly variable; PC has a total of only 43 data points, 5 of which are 7-letters long and low-imageability, and thus the very long mean RT of 13049msec for this last cell should be interpreted cautiously. There was a positive correlation between the severity of the LBL deficit (again, using the value of the regression slope in naming latency) and the imageability effect (defined as the msec difference between naming high- and low-imageability words) ($r^2 = .53$, $P < .06$), again indicating a relationship between severity and imageability, although this correlation was not as strong as was the correlation with frequency.

Summary of Empirical Data

The findings from the empirical study are fairly straightforward. In a group of LBL patients, all of whom show the characteristic effect of word length on reaction time, we see a difference in performance between high- and low-frequency items in all seven patients and a difference between high- and low-imageability words in five of them. Moreover, both frequency and imageability interact with the length of the string such that the frequency effect and the imageability effect are more marked for long than for short words. The exact discrepancy between the high and low items as a function of word length is evident to

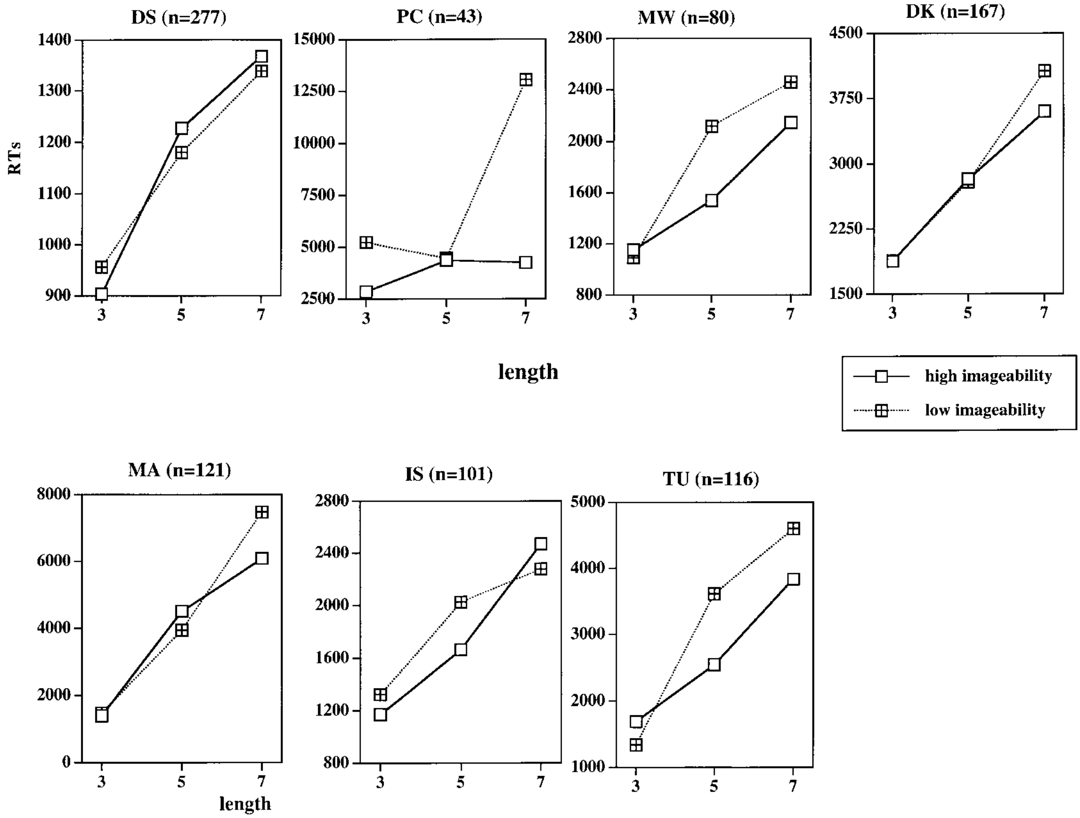

Fig. 3. Reaction times for the seven letter-by-letter readers, plotted indivdually, as a function of word length for high- and low-imageability words.

a greater degree in some patients than in others and this interaction is more pronounced in the frequency than in the imageability analysis.

DISCUSSION

Letter-by-letter reading is a form of acquired dyslexia in which patients show a monotonic relationship between word length and reaction time, as reflected in both naming and lexical decision tasks. There are two major findings in the LBL literature: The first is that patients have a deficit at a peripheral stage of processing. Evidence supporting a peripheral impairment comes from the finding that many, if not all, patients are unable to represent orthographic input normally and they show an impairment in letter processing. The second finding is that the reading performance of many patients is influenced by lexical and semantic variables, suggesting that the deficit occurs only after an adequate orthographic representation is activated. Support for a central impairment comes from the finding that many LBL readers appear to have processed the visual stimulus sufficiently to have derived semantic, lexical, and higher-order orthographic information from it, as indicated by their ability to perform semantic categorisation tasks or to make lexical decisions about stimuli that they have not identified explicitly.

In this paper, we put forward an account of LBL reading that reconciles these disparate findings. We argue that there is a fundamental peripheral deficit in LBL that adversely affects parallel letter processing. Despite this lower-level deficit, however, some information is still propagated to higher-level (lexical and semantic) representations. In an attempt to enhance orthographic activations, subjects employ the normal reading strategy of making additional fixations (or covert attention shifts) when encountering difficulties in text. This sequential letter processing adds further to the activation of lexical/ semantic representations. Due to the interactive nature of processing, these higher-level representations feed back to provide support for subsequent orthographic processing. The strength of this support depends on the degree of higher-level activation, which we assume scales with word frequency and imageability. Because patients take longer to process words with more letters, the lexical/ semantic activation has a longer time in which to accumulate and influence the degraded letter activations. The main point is that, in an interactive system, impoverished input can still activate lexical and semantic information and, moreover, that the strength of this activation over time is related to the length of the word. Thus, covert or tacit reading in the form of semantic categorisation and lexical decision is possible despite poor stimulus identification; lexical variables such as frequency and imageability may still influence word recognition, and lexical/ semantic representations may feed back onto letter representations and produce a word superiority effect. Thus, just as higher-level representations can compensate for or mitigate against the spatially degraded input in neglect dyslexia (Mozer & Behrmann, 1990), so too can such representations feed back and support the weakened orthographic activation in LBL reading.

In support of this theoretical position, we cite evidence from a review of published reports of 57 patients with LBL reading. There was positive evidence in almost every case for the presence of a peripheral deficit and, even in those few cases for which no definitive evidence existed, there was no compelling counter-evidence. The findings from this review regarding the higher-level lexical and semantic effects were somewhat less conclusive but this was largely because of the dearth of data. Relatively few patients have been tested systematically for the influence of different variables on performance and, even when there was some suggestive evidence in a case report, the effects were not usually evaluated across a sufficient range of measures to provide a full description of the patient's performance. Perhaps the most relevant finding from this review, however, was that more than two thirds of the patients showed some evidence of higher-level effects, whether in implicit reading under brief exposure, in the presence of a word superiority effect, or in the influence of lexical variables on reading performance. The key point, then, is that in many of these patients with peripheral deficits, there is also a strong suggestion of some kind of lexical and semantic processing.

To substantiate our position further, we carried out a retrospective empirical analysis of the reading performance of seven LBL readers we have tested over the last decade. We have previously determined that these patients all suffer from a peripheral impairment of some form. In the current analysis, we demonstrated a significant main effect of word frequency and word imageability on reaction time in almost all cases and, in most cases, these effects interacted significantly with word length (although to varying degrees and with more variance in imageability than in frequency). We suggest that this interaction is specifically predicted by an interactive account. In particular, we argue that, because of the additional time required by LBL readers for longer words, there is more time in which higher-level activation can accumulate and affect reading performance.

Our review of the literature has turned up a number of other cases in whom an interaction between a lexical variable and word length has also been demonstrated. For example, Bowers, Bub, & Arguin (1996) documented the effect of word frequency on the accuracy and reaction time of patient IH's reading responses. Although a statistical analysis was not provided, high-frequency words were read better and faster overall than were low-frequency words. Moreover, the discrepancy between the two types of items was greater for longer than shorter words: whereas there was no accuracy difference for high- and low-frequency four-letter words, there was a difference for longer words. Interactions of word length and imageability have also been reported in a few cases. Patient DR (Doctor et al., 1990), for example, took longer to read low- than high-imageability printed words, particularly as word length increased. A similar trend, albeit nonsignificant, was also observed in this patient for handwritten words. Finally, JH (Buxbaum & Coslett, 1996) showed a dramatic increase in reaction time (and decrease in accuracy) as word length increased, but this was especially true for low-imageability words. Thus, the difference in accuracy between high- and low-imageability words was approximately 10% but increased to approximately 45% for eight-letter words (see their Fig. 7).

In addition to showing the predicted interaction between word length and imageability, patient JH (Buxbaum & Coslett, 1996) provides additional support for our account. JH was clearly impaired at lower-level processing: He was unable both to represent information on the right of a display and to distribute attention with sufficient resolution for the purposes of letter identification. Despite this impaired peripheral processing, however, JH's reading performance showed a part-of-speech effect (nouns were read better than functors), an effect of lexical status (words were read better than nonwords), and an effect of morphological composition (pseudosuffixed words were read better than suffixed words). The two major findings, a peripheral deficit concurrent with lexical influences on reading, and an interaction between word length and lexical variables, provide clear support for our unitary view of letter-by-letter reading.

There are a number of suggestions in the literature that bear some resemblance to, or even foreshadow, the view we have proposed here. More than a decade ago, Shallice and Saffran (1986), in their explanation of patient ML's tacit or covert reading abilities, suggested that weak input from an impaired word-form system might still allow sufficient activation of a semantic representation that would suffice for a task like semantic categorisation. They argued, however, that inhibition might be inadequate in the system. Because it is necessary to converge on a single correct output for stimulus identification, the alternatives must be inhibited and patient ML was unable to do so. Thus, weakened but sufficient activation in combination with inadequate inhibition might give rise to above-chance semantic categorisation in the absence of stimulus identification (see Friedman et al., 1993, for a similar account and Hinton & Shallice, 1991, for an interpretation of ML's data that is consistent with this). Along similar lines, Bub et al. (1989) have suggested that, if activation is sufficient, even if not of normal strength, lexical decision and semantic priming may still be possible. Similar ideas about weakened activation and/ or inhibition have also been suggested to explain phenomena of tacit recognition in other neuropsychological disorders, such as prosopagnosia or implicit/ explicit memory differences (Farah, 1994), and even in normal subjects. Monsell et al. (1989), for example, have postulated a mechanism similar to ours to account for the finding that normal subjects can perform lexical decision well without being able to identify the stimulus. They claimed that a subthreshold signal from the target relative to the activity of neighbouring items may enable a legitimate word to be distinguished from nonwords but may not suffice for identification. If the subjects monitor the activation in the system, “the familiarity assessment process”, then high-frequency words which have stronger representation and are more familiar can bias the subject towards a positive response before unique identification has occurred (see Plaut & Shallice, 1993, for connectionist simulations consistent with this idea).

MODELS OF LETTER-BY-LETTER READING

There are no existing models of reading that fully instantiate our theory of the mechanism underlying the performance of LBL readers. However, there are a number of models which, in their behaviour under damage, exhibit many of the critical properties on which our account is based. As mentioned in the Introduction, the fact that a partial peripheral impairment may give rise to both lower- and higher-level effects has already been demonstrated with respect to lexical influences on reading performance in neglect dyslexia (Mozer & Behrmann, 1990), in the preservation of semantic categorisation and lexical decision with impaired overt recognition, and in the occurrence of semantic errors following visual damage in deep dyslexia (Hinton & Shallice, 1991; Plaut & Shallice, 1993). In the specific context of LBL reading, support for co-occurrence of lower- and higher-level effects following peripheral damage has recently been provided by Mayall and Humphreys (Mayall & Humphreys, 1996) who implemented a connectionist model whose architecture incorporated some of the properties of the IAM. In a single architecture, reflecting the normal word processing system, Mayall and Humphreys were able to reproduce many of the covert abilities of LBL patients when they lesioned the network. They tested both early lesions (near the input layer) and more central lesions (to hidden units). Of primary concern to us is the pattern of results obtained with the former, peripheral type of disturbance. When a percentage of the input units were disconnected from the hidden units, letter naming, although poor (35% correct) was significantly better than word naming (9% correct). The errors produced were mostly visual rather than semantic. In addition, a greater number of correct semantic categorisation and lexical decision responses were observed compared with naming responses. Finally, effects of word frequency and imageability were noted in naming, categorisation, and lexical decision. This ensemble of findings is precisely what one would expect with a cascaded, interactive model.

A significant limitation of Mayall and Humpreys' (1996) simulation, however, is that it does not address the hallmark characteristic of LBL readers– namely, their letter-by-letter reading. More recently, Plaut (1998) has demonstrated properties of LBL reading following peripheral damage to the "refixation" model first described by Plaut et al. (1995). The model generates a sequence of phonemes as output in response to orthographic input presented over position-specific letter units. A critical aspect of the model is that, if it encounters difficulty in generating a particular phoneme in the course of pronouncing a word, it can refixate the input (using an internally generated attentional signal) to bring the corresponding peripheral orthographic segment to "fixation", where performance is better. Early on in training, the model fixated virtually every grapheme but, by the end of training, it read correctly 99.3% of the 2998 monosyllabic words on which it was trained, producing an average of only 1.3 fixations per word. When letter activations were corrupted by noise, correct performance dropped to 90.1% correct. Using a median split on frequency, accuracy was greater on high- versus low-frequency words (92.1% vs. 88.6%, respectively) and short versus long words (e.g. 92.0% for 4-letter words vs. 85.2% for 6-letter words). Critically, among words pronounced correctly, the average number of fixations per word increased from 1.3 to 1.93. Moreover, the number of fixations– a loose analogue to naming latency– was strongly influenced by the length of the word. For example, the model made an average of 1.76 fixations for 4-letter words but 2.92 fixations for 6-letters words. The model also made fewer fixations to high- versus low-frequency words (means 1.86 vs. 2.00, respectively). Finally, and most importantly for the current account of LBL reading, there was a clear interaction of frequency and length: The difference in the number of fixations for high- versus low-frequency words was 0.04 for 4-letter words but 0.20 for 6-letter words. Thus, under peripheral damage, the model exhibited the hallmark word-length effect characteristic of LBL reading, combined with the appropriate higher-level effects: A word frequency effect that was greater for long compared with short words. However, the model contains no semantic representations and, thus, is unable to account for the effects of imageability on LBL reading, nor the relatively preserved lexical decision and semantic categorisation performance of these patients.

ADDITIONAL ISSUES

There are three outstanding issues that require discussion in the context of our theory of LBL reading. The first concerns effects of stimulus degradation in normal readers, the second concerns the heterogeneity in the performance of LBL readers, and the last concerns the substrate mediating the lexical and semantic effects. We take up these issues in turn.

Effects of Stimulus Degradation in Normal Readers

A straightforward prediction that one might make from our account is that normal subjects should reveal a word length effect when tested under conditions of stimulus degradation (and that this should interact with frequency and imageability). Exactly what the parameters of this degradation are remains unclear and needs to be determined. There are, however, a few studies that have examined this issue in normal subjects. An early study by Terry, Samuels and LaBerge (1976; Expt 1) had normal subjects press a response button when the presented item was an animal word (two thirds of all words were animal words and were the highest-frequency animal words). The words varied in length from three to eight letters and the letters could be degraded or not. Unfortunately, conclusions from this study are tenuous for our purposes. First, it appears that the degradation had no effect on accuracy, as evident in the error data (op. cit. p. 580). Given that this is a potential ceiling effect, it is not surprising that there is no interaction between degradation and word length even in reaction times. Second, the results for the longer words (seven and eight letters) could not be calculated as there were too few observations in these cells. Finally, because the task is more like a decision task than a naming latency task, the effect of word length, if it exists, is likely to be smaller than is the case in standard naming latency tasks (see Henderson, 1982, for review of relevant findings).

In a more recent study, Snodgrass and Mintzer (1993), have examined the effect of stimulus degradation on word length in the context of studying how orthographic neighbourhood and word frequency influence the speed of word recognition. They presented degraded word stimuli of varying lengths to normal readers and gradually improved the quality of the stimulus until it could be identified. The threshold of time to identification did not vary as a function of word length nor did it vary as a function of neighbourhood or frequency, as one might have expected (see Snodgrass & Mintzer, 1993; Table1, p. 251). The absence of a correlation between the threshold of identification and any of these other variables, as acknowledged by the authors, might be a function of the fact that frequency and neighbourhood size were not particularly carefully controlled. In light of this, the authors go on to a second experiment in which these variables are controlled and the expected results obtained. Unfortunately, length is not controlled in this second experiment and so conclusions about the effect of degradation of word length in normal readers remains to be determined.

A final experiment, perhaps the most closely related to the focus of this paper, is that of Farah and Wallace (1991) who plotted naming latency as a function of word length for stimuli that were either masked (as a proxy for stimulus degradation) or not. Whereas latency differed significantly as a function of word length and also of visual quality, there was no interaction between them. On the surface, this seems to be a challenge to our account. Important to note, however, is that Farah and Wallace (1991) instructed their subjects to read letter-by-letter as they were particularly interested in the visual impairment hypothesis of pure alexia. Unfortunately, for our sake, this does not constitute a relevant test of our account, given that strategic effects can significantly alter the nature of the reading processing (see following section on individual differences) and also that under these conditions, subjects are unlikely to make an initial attempt at parallel identification, as we propose is the case for LBL readers. Taken together, the findings from these three studies fail to show the expected interaction of word length and stimulus degradation. However, none of these studies appears to be a good test of the hypothesis given the particular methodological choices the experimenters have made. Our prediction still holds then: If one can simulate the nature of the peripheral impairment in LBL readers in normal subjects, then interactions with word length as well as the other lexical variables should be observed. Preliminary data are encouraging in that an interaction between word length with frequency and with imageability is more evident with increased stimulus degradation (Nelson, Behrmann, & Plaut, 1998).

Individual Differences in Letter-by-letter Reading

A particularly striking finding that seems to challenge to our account concerns the range of individual differences observed in the LBL readers. As is evident from Table 1, whereas some patients show striking lexical and semantic effects, others do not. There are several possible explanations for this variability. One explanation might be that this simply reflects limitations in the available data. Patients are not routinely and systematically tested and so the sparse data may be exaggerating the differences between patients. Although it is true that additional data are sorely needed, this is unlikely to be the sole explanation. It is also unlikely that individual differences in literacy or reading skill can fully explain the range of variability in the LBL reading profiles (Hanley & Kay, 1996).

Perhaps a more persuasive explanation for differences among LBL readers concerns the severity of the deficit. In Mozer and Behrmann's (1990) interactive theory of neglect dyslexia, the difference between the two patients is explained by the severity of the early attentional deficit. Thus, Mozer and Behrmann suggested that, in the case of the more severe neglect patient, the orthographic input was far too degraded to activate the corresponding higher-order lexical and semantic representations. In the case of the more mild neglect patient, there was sufficient bottom-up activation of higher-order representations, and effects of lexical status and morphological composition were observed. We suggest that differences in severity of impairments may also underlie the individual differences observed among LBL readers. For example, Behrmann, Black, et al. (1990) showed that, in the early stages post-stroke, DS did not show a word superiority effect in her reading. When she was retested several years later, her reading had improved (as measured in the slope and intercept of the latency-per-letter function), although she was still an LBL reader. Importantly, at this later stage, she now showed a marked difference in reading of words versus nonwords. Consistent with this, Coslett and Saffran (1989a) found that the postlexical effects in their patients became more apparent after partial recovery had taken place. For example, patient JG gradually regained the ability to perform tacit reading over a 3-month period and showed imageability and part-of-speech effects in reading (Coslett & Monsul, 1994).

These findings suggest that there may be an inverted-U function relating the severity of impairment to the strength of higher-level effects observed in LBL readers. When the visual input is well processed, top-down support is largely unnecessary, and when the deficit is too severe, higher-order representations are not strongly engaged. It is only in the middle range, when the orthographic input is still sufficiently intact, that the later lexical effects become apparent. At this point, then, when there is sufficient activation, the exact magnitude of the higher-order effects depends on the severity of the deficit. The slower the reader (or the more marked the word length effect), the longer the time for the top-down processes to play a role. This is well illustrated in our group of seven patients in whom there is a correlation between the severity of LBL reading (defined by the slope of the regression curve on the naming latency data; Fig. 1) and the frequency effect (difference in ms between high- and low-frequency words). There is also a positive correlation between slope values and the imageability effect, although this correlation is more modest than the frequency effects. Thus, the more severely affected the LBL reader (within the range permitting higher-level activation), the greater the effect of the lexical variables.

The claim that severity, defined this way, might determine the extent of the lexical/ semantic effects, is interesting but is likely to be too simple. The slope of the reading function, in our account, is determined by the severity of the lower-level deficit, but we know that the correlation between the degree of peripheral impairment and reading performance is not perfect. Hanley and Kay (1996) have described fairly large differences in the reading performance of two LBL patients with roughly equivalent letter identification performance. Their interpretation of the discrepancy between the patients echoes that of Patterson and Kay (1982), who invoked a second lesion (in addition to the peripheral one) to account for the differences between patients. Specifically, they proposed that an additional impairment at the level of the word form system is present in some but not other LBL readers.

Some authors have suggested that yet another potential source of individual differences is parametric variation in the strength of overall inhibition within the lexical system. As mentioned previously, according to Shallice and Saffran (1986) it is the strength of inhibition that is critical for the system to converge on a single response for identification. Sufficient (even if not normal) activation, on the other hand, might be adequate for semantic categorisation. One might imagine that differing degrees of inhibition and activation might lead to different patterns of performance, only some of which produce results consistent with higher-level effects on performance. Arguin and Bub (1993), in their demonstration of letter priming in LBL reading in a connectionist model, have made a similar point. They argued that the crucial parameters are the amounts of inhibition and activation and the balance between them. It is this balance that may vary from one patient to another. Differences in response threshold and confidence in responding might also bring about differences in the overall pattern of reading performance across patients.

A final but important difference concerns the strategies individual subjects might employ in compensating for their peripheral impairment. It is now well recognised that particular strategies can diminish and possibly even eliminate lexical/ semantic effects (also, Howard, 1991). Farah and Wallace (1991) pointed this out in relation to the presence/ absence of the word superiority effect and the type of serial letter strategy used, whether "ends-in" or left-to-right. But perhaps the best illustration of the importance of considering strategy effects comes from patient JWC (Coslett et al., 1993), who appeared to be able to use two distinct strategies, only one of which produced covert reading. When JWC was instructed to name words, he employed a laborious, serial LBL strategy that eventually resulted in explicit word identification. In contrast, when instructed to make lexical decisions on semantic categorisations on briefly presented stimuli, he adopted a "whole-word" strategy that was fast and less effortful but which failed to provide explicit word identification. Based on these findings, Coslett and colleagues argued that differences in task demands or instructions may be a critical factor that affects the emergence of the higher-level effects and, presumably, in their account, the involvement of the right hemisphere in reading. They recommended that patients be discouraged from employing the slow and inefficient left-hemisphere LBL procedure during rehabilitation and should instead be prompted to adopt the more parallel whole-word strategy (but see Chialant & Caramazza, this issue.

The Locus of the Lexical/Semantic Effects

The last remaining topic for discussion concerns the debate between the right-hemisphere view and our own. The former, two-system account argues that the LBL reading and the word length effect are a consequence of the processing of the impaired left hemisphere, whereas covert reading and later lexical/ semantic effects are mediated by the right hemisphere (Coslett & Saffran, 1993; Saffran & Coslett, this issue). We have maintained instead that the empirical data and profiles of performance associated with LBL readers do not require such a dichotomy. Rather, we have argued that the full range of effects in LBL reading can be explained in terms of properties of the normal interactive lexical processing system located in both hemispheres. Although the right hemisphere undoubtedly contributes to performance, as does the left hemisphere, the observed LBL pattern is a reflection of the combined processing of both hemispheres, both for the sequential reading pattern and the higher-level effects. After damage to the left hemisphere, we would argue, the dynamic cooperation between the hemispheres and their relative contribution to the output continues as was the case premorbidly. Importantly, on our account, the two hemispheres are governed by the same computational and interactive principles and the final manifestation of LBL reading is the result of the dynamic processing within and between both hemispheres.

What data, then, might compel us to abandon this view and invoke a stronger right-hemisphere account, or a greater division of labour between the hemispheres? One source of evidence often cited in support of the right-hemisphere account is the apparent similarity in the pattern of performance in LBL patients and in the right hemisphere of commissurotomy and left-hemispherectomy patients. These last two groups of patients can make semantic judgements but are poor at processing low-imageability words, grammatical morphemes, and functors– precisely the pattern reported for LBL readers (Coslett & Saffran, 1994). The qualitative similarity between the patient groups suggests that the LBL readers might well be accessing a limited-capability right-hemisphere reading system. There are, however, some important differences between these various populations both qualitatively and quantitatively (Baynes, 1990). For one, unlike deep dyslexic patients, who are thought to be reading primarily with the right hemisphere given the large extent of their left-hemisphere damage, LBL readers almost never make semantic paralexias. A possible response to this discrepancy is to claim that LBL readers are able to access the phonology for the initial letter via the left hemisphere and this prevents the production of semantic errors (a similar view is cited by Marshall & Newcombe, 1973, in the case of deep dyslexic patients who make fewer semantic paralexias over time). Even if this were the case, this would amount to the same claim as we are making: Both hemispheres contribute to the final output. Setting the semantic errors aside, however, if it were the case that LBL readers were engaging the right hemisphere to such an extent, we might expect that they would read words as well as patients with deep dyslexia. Some deep dyslexia patients read concrete words extremely well, and so the expectation is that this should also be the case for LBL readers. This, however, is not the case.

Perhaps the evidence to distinguish between these accounts will only come from physiological studies that directly investigate the contribution of the two hemispheres to reading in letter-by-letter patients. One such study is that by Coslett and Monsul (1994), which used transcranial magnetic stimulation (TMS) applied separately to the left and right hemisphere of their LBL patient, JG. Interestingly, JG's reading was significantly disrupted by TMS to the right (from 71% to 21%, and showed an interaction with frequency), but not to the left hemisphere (68% to 61%). This finding is strongly indicative of the contribution of the right hemisphere to reading. Interestingly, JG's reading was not totally disrupted by right-hemisphere TMS; this might indicate that either the TMS only partially disrupted the right-hemisphere function or it totally disrupted right-hemisphere function and the residual reading was mediated by the left hemisphere. Although this finding does seem, on the face of it, to suggest strongly that it is the right-hemisphere that is mediating reading, it should be pointed out that JG is a somewhat anomalous LBL patient. He exhibited pure alexia after suffering small, exclusively subcortical lesions to the splenium of the corpus callosum and in the region of the left lateral geniculate nucleus (he also showed a dilatation of the left occipital lobe on subsequent CT scans, perhaps reflecting degeneration secondary to the lateral geniculate damage). The subcortical nature of the damage is different from that in the overwhelming majority of LBL patients, whose lesions are cortical (Black & Behrmann, 1994). Thus, although the findings from JG are indeed provocative, we do not think that they are sufficiently firm on their own just yet to motivate a strong right-hemisphere account.

Our conclusion, then, is that there are no data that compel us to accept the view that the right hemisphere solely or even primarily subserves the lexical and semantic effects in LBL reading. Given this, we maintain that the normal premorbid reading system, degraded by brain damage, continues to function, and, because of its interactive nature, gives rise to the expected patterns of lower- and higher-level effects. Perhaps the final adjudication will come from neuroimaging studies of LBL readers in which brain activation of each hemisphere is calculated separately and correlated with the lexical/ semantic effects.

CONCLUSION

We have presented a unitary account of LBL reading that, we have argued, reconciles previously discrepant findings. We have claimed that a deficit in letter processing (perhaps attributable to an even more fundamental perceptual impairment) is common to all LBL readers, and that this deficit prevents the normal activation of orthographic representations. It is the severity of this impairment that determines the extent to which top-down activation from higher-order lexical and semantic representations can feed back and mitigate the lower-level deficit. When the deficit is too severe, little top-down influence will be observed. Once the activation is sufficient, however, the more degraded the input representation and the longer the time required for processing, the more time is available for top-down support to accumulate. We have suggested that, in the context of a cascaded, interactive system, it is possible to observe lexical and semantic effects simultaneously with a peripheral lesion that affects lower-level processing. We have also proposed that the observed reading pattern, including the sequential LBL reading itself, arises from the residual function of the normal reading system that probably involves both the left and right hemispheres and that there is no reason to invoke the right-hemisphere reading system as the primary mediator of the lexical and semantic effects.

REFERENCES

Ajax, E.T. (1967). Dyslexia without agraphia. *Archives of Neurology, 17*, 645–652.

Albert, M.L., Yamadori, A., Gardner, H., & Howes, D. (1973). Comprehension in alexia. *Brain, 96*, 317–328.

Arguin, M., & Bub, D. (1993). Single character processing in a case of pure alexia. *Neuropsychologia, 31(5)*, 435–458.

Arguin, M.E., & Bub, D. (1994a). Functional mechanisms in pure alexia: Evidence from letter processing. In M.J. Farah and G. Ratcliff (Eds.), *The neuropsychology of high-level vision* (pp. 149–171). Hillsdale, NJ: Lawrence Erlbaum Associates Inc.

Arguin, M.E., & Bub, D. (1994b). Pure alexia: Attempted rehabilitation and its implications for the interpretation of the deficit. *Brain and Language, 47*, 233–268.

Arguin, M.E., Bub, D., & Bowers, J.S. (this issue). Extent and limits of covert lexical activation in letter-by-letter reading. *Cognitive Neuropsychology, 15*, (1/ 2).

Baynes, K. (1990). Language and reading in the right hemisphere: Highways or biways or the brain. *Journal of Cognitive Neuroscience, 2*, 159–179.

Beeman, M., & Chiarello, C. (1997). *Right-hemisphere language comprehension: Perspectives from cognitive neuroscience*. Hillsdale, NJ: Lawrence Erlbaum Associates Inc.

Behrmann, M., Barton, J.J., & Black, S.E. (1998). *Eye movements reveal the sequential processing in letter-by-letter reading*. Manuscript in preparation.

Behrmann, M., Black, S.E., & Bub, D. (1990). The evolution of pure alexia: A longitudinal study of recovery. *Brain and Language, 39*, 405–427.

Behrmann, M., & McLeod, J (1995). Rehabilitation for pure alexia: Efficacy of therapy and implications for models of normal word recognition. *Neuropsychological Rehabilitation, 5*, 149–180.

Behrmann, M., Moscovitch, M., Black, S.E., & Mozer, M. (1990). Perceptual and conceptual factors in neglect dyslexia: Two contrasting case studies. *Brain 113(4)*, 1163–1183.

Behrmann, M., Nelson, J., & Sekuler, E. (1998). *Visual complexity in letter-by-letter reading: "Pure" alexia is not so pure*. Neuropsychologia, in press.

Behrmann, M., & Shallice, T. (1995). Pure alexia: An orthographic not spatial disorder. *Cognitive Neuropsychology, 12(4)*, 409–454.

Black, S.E., & Behrmann, M. (1994). Localization in alexia. In A. Kertesz (Ed.), *Localization and neuroimaging in neuropsychology* (pp. 331–376). San Diego, CA: Academic Press.

Bowers, J.S., Arguin, M., & Bub, D.N. (1996). Fast and specific access to orthographic knowledge in a case of letter-by-letter reading. *Cognitive Neuropsychology, 13(4)*, 525–567.

Bowers, J.S., Bub, D., & Arguin, M. (1966). A characterization of the word superiority effect in pure alexia. *Cognitive Neuropsychology, 13*, 415–441.

Brunn, J.L., & Farah, M. (1991). The relation between spatial attention and reading: Evidence from the neglect syndrome. *Cognitive Neuropsychology, 8(1)*, 59–75.

Bub, D., & Arguin, M. (1995). Visual word activation in pure alexia. *Brain and Language, 49*, 77–103.

Bub, D.N., Arguin, M., & Lecours, A.R. (1993). Jules Déjerine and his interpretation of pure alexia. *Brain and Language, 45*, 531–559.

Bub, D., Black, S.E., & Howell, J. (1989). Word recognition and orthographic context effects in a letter-by-letter reader. *Brain and Language, 36*, 357–376.

Bub, D., & Gum, T. (1988). *Psychlab experimental software.* Montreal: Montreal Neurological Institute.

Buxbaum, L., & Coslett, H.B. (1996). Deep dyslexic phenomenon in a letter-by-letter reader. *Brain and Language, 54*, 136–167.

Caplan, L.R., & Hedley-Whyte, T. (1974). Cuing and memory dysfunction in alexia without agraphia. *Brain, 97*, 251–262.

Chialant, D. & Caramazza, A. (this issue). Perceptual and lexical factors in a case of letter-by-letter reading. *Cognitive Neuropsychology, 15*(1/ 2).

Cohen, J.D., MacWhinney, B., Flatt, M., & Provost, J. (1993). PsyScope: A new graphic environment for designing psychology experiments. *Behavioural Research Methods, Instruments and Computers, 25*, 257–271.

Coltheart, M. (1980). Deep dyslexia: A right hemisphere hypothesis. In M. Coltheart, K.E. Patterson, & J.C. Marshall (Eds.), *Deep dyslexia* (pp. 326–380). London: Routledge.

Coltheart, M. (1981). Disorders of reading and their implications for models of normal reading. *Visible Language, 15*, 245–286.

Coltheart, M. (1983). The right hemisphere and disorders of reading. In A.W. Young (Ed.), *Functions of the right cerebral hemisphere*, London: Academic Press.

Coslett, H.B., & Monsul, N. (1994). Reading with the right hemisphere: Evidence from transcranial magnetic stimulation. *Brain and Language, 46*, 198–211.

Coslett, H.B., & Saffran, E.M. (1989a). Evidence for preserved reading in "pure" alexia. *Brain, 112*, 327–359.

Coslett, H.B., & Saffran, E.M. (1989b). Preserved object recognition and reading comprehension in optic aphasia. *Brain, 112*, 1091–1110

Coslett, H.B. & Saffran, E.M. (1992). Optic aphasia and the right hemisphere: A replication and extension, *Brain and Language, 43*, 148–161.

Coslett, H.B., & Saffran, E.M. (1994). Mechanisms of implicit reading in alexia. In M.J. Farah & G. Ratcliff (Eds.), *The neuropsychology of high-level vision* (pp. 299–330). Hillsdale, NJ: Lawrence Erlbaum Associates Inc.

Coslett, H.B., Saffran, E.M., Greenbaum, S., & Schwartz, H. (1993). Reading in pure alexia. *Brain, 116*, 327–359.

Damasia, A., & Damasio, H. (1983). The anatomic basis of pure alexia. *Neurology, 33*, 1573–1583.

Doctor, E.A., Sartori, G., & Saling, M.M. (1990). A letter-by-letter reader who could not read nonwords. *Cortex, 26*, 247–262.

El Alaoui-Faris, M., Bienbelaid, F., Alaoui, C., Tahiri, L., Jiddane, M., Amarti, A., & Chkili, T. (1994). Alexie sans agraphie en langue arabie etude neurolinguistique et IRM. *Revue Neurologique (Paris), 150*, 771–775.

Farah, M.J. (1991). Patterns of co-occurrence among the associative agnosias: Implications for visual object recognition. *Cognitive Neuropsychology, 8(1)*, 1–19.

Farah, M.J. (1994). Visual perception and visual awareness after brain damage: A tutorial review. In C. Umiltà & M. Moscovitch (Eds.), *Attention and performance XV: Conscious and nonconscious* *information processing* (pp. 37–76). Cambridge, MA: Bradford Books, MIT Press.

Farah, M.J. (1997). Are there orthography-specific brain regions? Neuropsychological and computational investigations. In R.M. Klein & P.A. McMullen (Eds.), *Converging methods for understanding reading and dyslexia.* Cambridge, MA: MIT Press.

Farah, M.J., & Wallace, M. (1991). Pure alexia as a visual impairment: A reconsideration. *Cognitive Neuropsychology, 8(3/4)*, 313–334.

Feinberg, T.E., Dyckes-Berke, D., Miner, C.R., & Roane, D.M. (1995). Knowledge, implicit knowledge and metaknowledge in visual agnosia and pure alexia. *Brain, 118*, 789–800.

Freedman, L., & Costa, L. (1982). Pure alexia and right hemiachromatopsia in posterior dementia. *Journal of Neurology, Neurosurgery and Psychiatry, 55(6)*, 500–502.

Friedman, R.B., & Alexander, M.P. (1984). Pictures, images, and pure alexia: A case study. *Cognitive Neuropsychology, 1(1)*, 9–23.

Friedman, R.B., Beeman, M., Lott, S.N., Link, K., Grafman, J., & Robinson, S. (1993). Modality-specific phonological alexia. *Cognitive Neuropsychology, 10*, 549–568.

Friedman, R.B., & Hadley, J.A. (1982). Letter-by-letter surface alexia. *Cognitive Neuropsychology, 9*, 1–23.

Geschwind, N. (1965). Disconnection syndromes in animals and man. *Brain, 88*, 237–294.

Greenblatt, S.H. (1973). Alexia without agraphia. *Brain, 96*, 307–316.

Grossi, D., Fragassi, N.A., Orsini, A., Falco, F.A.D., & Sepe, O. (1984). Residual reading capability in a patient with alexia without agraphia. *Brain and Language, 23*, 337–348.

Hanley, J.R., & Kay, J. (1992). Does letter-by-letter reading involve the spelling system? *Neuropsychologia, 30*, 237–256.

Hanley, J.R., & Kay, J. (1996). Reading speed in pure alexia. *Neuropsychologia, 34(12)*, 1165–1174.

Henderson, L. (1982). *Orthography and word recognition in reading.* London: Academic Press.

Henderson, V., Friedman, R., Teng, E., & Weiner, J. (1985). Left hemisphere pathways in reading: Inferences from pure alexia without hemianopia. *Neurology, 35*, 962–968.

Hinton, G.E. (1990). Mapping part-whole hierarchies in connectionist networks. *Artificial Intelligence, 46*, 47–75.

Hinton, G.E., & Shallice, T. (1991). Lesioning an attractor network: Investigations of acquired dyslexia. *Psychological Review, 98(1)*, 74–95.

Horikoshi, T., Asari, Y., Watanabe, A., Nagaseki, Y., Nukui, H., Sasaki, H., & Komiya, K. (1997). Music alexia in a patient with mild pure alexia: Disturbed meaningful perception of nonverbal figures. *Cortex, 33*, 187–194.

Howard, D. (1991). Letter-by-letter readers: Evidence for parallel processing. In D. Besner and G.W. Humphreys (Eds.), *Basic processes in reading: Visual word recognition* (pp. 34–76). Hillsdale, NJ: Lawrence Erlbaum Associates Inc.

Johnston, J.C., & McClelland, J.L. (1980). Experimental tests of a hierarchical model of word identification. *Journal of Verbal Learning and Verbal Behavior, 19*, 503–524.

Just, M.A., & Carpenter, P. (1987). *The psychology of reading and language comprehension.* Boston, MA: Allyn & Bacon.

Karanth, P. (1985). Dyslexia in a Dravidian language. In K.E. Patterson, J.C. Marshall, & M. Coltheart (Eds.), *Surface dyslexia* (pp. 251–260). Hove, UK: Lawrence Erlbaum Associates Ltd.

Kashiwagi, T., & Kashiwagi, A. (1989). Recovery process of a Japanese alexic without agraphia. *Aphasiology, 3*, 75–91.

Kay, J., & Hanley, R. (1991). Simultaneous form perception and serial letter recognition in a case of letter-by-letter reading. *Cognitive Neuropsychology, 8(3/4)*, 249–273.

Kinsbourne, M. & Warrington, E.K. (1962). A disorder of simultaneous form perception. *Brain, 85*, 461–486.

Kreindler, A., & Ionescu, Y. (1961). A case of “pure” word blindness. *Journal of Neurology, Neurosurgery and Psychiatry, 24*, 275–280.

Kučera, F., & Francis, W.N. (1967). *Computational analysis of present-day American English.* Providence, RI: Brown University Press.

LaBerge, D. (1983). Spatial extent of attention and letters and words. *Journal of Experimental Psychol-*

ogy: Human Perception and Performance, 9, 371–379.

Làdavas, E., Umiltà, C., & Mapelli, D. (1997). Lexical and semantic processing in the absence of word reading: Evidence from neglect dyslexia. *Neuropsychologia, 35(8)*, 1075–1085.

Landis, T., Regard, M., & Serrat, A. (1980). Iconic reading in a case of alexia without agraphic caused by a brain tumour. A tachistoscopic study. *Brain and Language, 11*, 45–53.

Lazar, R.M., & Scarisbrick, D. (1973). Alexia without agraphia: A functional assessment of behaviour in focal neurologic disease. *The Psychological Record, 43*, 639–650.

Levine, D.M., & Calvanio, R.A. (1978). A study of the visual defect in verbal alexia-simultanagnosia. *Brain, 101*, 65–81.

Marshall, J.C., & Newcombe, F. (1973). Patterns of paralexia: A psycholinguistic approach. *Journal of Psycholinguistic Research, 2*, 175–199.

Mayall, K., & Humpreys, G.W. (1996). A connectionist model of alexia: Covert recognition and case mixing effects: *British Journal of Psychology, 87*, 355–402.

McClelland, J.L. (1979). On the time relations of mental processes: An examination of systems of processes in cascade. *Psychological Review, 86*, 287–300.

McClelland, J.L. (1987). The case for interactionism in language processing. In M. Coltheart (Ed.), *Attention and Performance XII* (pp. 3–36). Hove, UK: Lawrence Erlbaum Associates Ltd.

McClelland, J.L., & Rumelhart, D.E. (1981). An interactive activation model of context effects in letter perception: Part 1. An account of basic findings. *Psychological Review, 88(5)*, 375–407.

Michel, F., Henaff, M.A., & Intriligator, J. (1996). Two different readers in the same brain after a posterior callosal lesion. *NeuroReport, 7*, 768–788.

Miozzo, M., & Caramazza, A. (this issue). Varieties of pure alexia: The case of failure to access graphemic representations. *Cognitive Neuropsychology, 15*(1/ 2).

Monsell, S., Doyle, M.C., & Haggard, P.N. (1989). Effects of frequency on visual word recognition tasks: Where are they? *Journal of Experimental Psychology: General, 118*(1), 43–71.

Montant, M., Behrmann, M., & Nazir, T. (1998). *Lexical activation in pure alexia.* Manuscript in preparation.

Montant, M., Nazir, T.A., & Poncet, M. (this issue). Pure alexia and the viewing position effect in printed words. *Cognitive Neuropsychology, 15*(1/ 2).

Morton, J. (1969). The interaction of information in word recognition. *Psychological Review, 76*, 165–178.

Morton, J. (1979). Word recognition. In J. Morton & J.C. Marshall (Eds.), *Psycholinguistic Series 2*. London: Elek.

Mozer, M.C.. (1991). *The perception of multiple objects: A connectionist approach*. Cambridge, MA: MIT Press.

Mozer, M.C., & Behrmann, M. (1990). On the interaction of selective attention and lexical knowledge: A connectionist account of neglect dyslexia. *Journal of Cognitive Neuroscience, 2*(2), 96–123.

Mozer, M.C., & Behrmann, M. (1992). Reading with attentional impairments: A brain-damaged model of neglect and attentional dyslexia. In R. Reilly & N.E. Sharkey (Eds.), *Connectionist approaches to natural language processing* (pp. 409–460). Hove, UK: Lawrence Erlbaum.

Nelson, J., Behrmann, M., & Plaut, D.C. (1998). *The interaction of stimulus degradation and word length with frequency and with imageability*. Manuscript in preparation.

Nitzberg-Lott, S., Friedman, R.B., & Linebaugh, C.W. (1994). Rationale and efficacy of a tactile-kinaesthetic treatment for alexia. *Aphasiology, 8*, 181 195.

O'Reagan, K., & Levy-Schoen, A. (1989). Eye-movement strategy and tactics in word recognition and reading. In K. Rayner & A. Pollatsek (Eds.), *The psychology of reading* (pp. 361–383). Englewood Cliffs, NJ: Prentice Hall.

Patterson, K.E., & Kay, J. (1982). Letter-by-letter reading: Psychological descriptions of a neurological syndrome. *Quarterly Journal of Experimental Psychology, 34A*, 411–441.

Perri, R., Bartolomeo, P., & Silveri, M.C. (1996). Letter dyslexia in a letter-by-letter reader. *Brain and Language, 53*, 390–407.

Plaut, D.C. (1995). Double dissociation without modularity: Evidence from connectionist neuropsychology. *Journal of Clinical and Experimental Neuropsychology, 17*(2), 291–321.

Plaut, D.C. (1998). Connectionist modelling of normal and impaired word reading. Manuscript in preparation.

Plaut, D.C., McClelland, J.L., Seidenberg, M.S. (1995). Reading exception words and pseudowords: Are two routes really necessary? In J.P. Levy, D. Bairaktaris, J.A. Bullinaria, & P. Cairns (Eds.), *Connectionist models of memory and language* (pp. 145–159). London: UCL Press.

Plaut, D.C., McClelland, J.L., Seidenberg, M., & Patterson, K.E. (1996). Understanding normal and impaired word reading: Computational principles in quasi-regular domains. *Psychological Review, 103*, 56 115.

Plaut, D.C., & Shallice, T. (1993). Deep dyslexia: A case study of connectionist neuropsychology. *Cognitive Neuropsychology, 10(5)*, 377–500.

Price, C.J., & Humphreys, G.W. (1992). Letter-by-letter reading? Functional deficits and compensatory strategies. *Cognitive Neuropsychology, 9(5)*, 427–457.

Price, C.J., & Humphreys, G.W. (1995). Contrasting effects of letter-spacing in alexia: Further evidence that different strategies generate word length effects in reading. *Quarterly Journal of Experimental Psychology, 48A*, 573–597.

Rapcsak, S.Z., Rubens, A.B., & Laguna, J.F. (1990). From letters to words: Procedures for word recognition in letter-by-letter reading. *Brain and Language, 38*, 504–514.

Rapp, B.C., & Caramazza, A. (1991). Spatially determined deficits in letter and word processing. *Cognitive Neuropsychology, 8(3/4)*, 275–312

Rayner, K., & Pollatsek, A. (1987). Eye movements in reading: A tutorial review. In M. Coltheart (Ed.), *Attention and performance XII: The psychology of reading* (pp. 327–362). Hove, UK: Lawrence Erlbaum Associates Ltd.

Regard, M., Landis, T., & Hess, K. (1985). Preserved stenography reading in a patient with pure alexia. *Archives of Neurology, 42*, 400–402.

Reuter-Lorenz, P., & Brunn, J. (1990). A prelexical basis for letter-by-letter reading: A case study. *Cognitive Neuropsychology, 7*, 1–20.

Rumelheart, D.E., & McClelland, J.L. (1982). An interactive activation model of context effects in letter perception: Part 2. The contextual enhancement effect and some tests and extensions of the model. *Psychological Review, 89*, 60–94.

Saffran, E.M., Bogyo, L.C., Schwartz, M.F., & Marin, O.S.M. (1980). Does deep dyslexia reflect right hemisphere reading? In M. Coltheart, K.E. Patterson, & J.C. Marshall (Eds.), *Deep dyslexia* (pp. 381–406). London: Routledge.

Saffran, E.M., & Coslett, H.B. (this issue). Implicit vs. letter-by-letter reading in pure alexia: A tale of two systems. *Cognitive Neuropsychology, 15*(1/ 2).

Schacter, D., McAndrews, M.P., & Moscovitch, M. (1988). Access to consciousness: Dissociations between explicit knowledge in neuropsychological syndromes: In L. Weiskrantz (Ed.), *Thought without language.* Oxford: Oxford University Press.

Schacter, D., Rapcsak, S.Z., Rubens, A.B., Tharan, M., & Laguna, J. (1990). Priming effects in a letter-by-letter reader depend upon access to the word form system. *Neuropsychologia, 28(10)*, 1079–1094.

Schiepers, C. (1980). Response latency and accuracy in visual word recognition. *Perception and Psychophysics, 27*, 71–81.

Seidenberg, M., & McClelland, J.L. (1989). A distributed, developmental model of word recognition and naming. *Psychological Review, 96*, 523–568.

Sekuler, E., & Behrmann, M. (1996). Perceptual cues in pure alexia. *Cognitive Neuropsychology, 13(7)*, 941–974.

Shallice, T. (1988). *From neuropsychology to mental structure.* Cambridge: Cambridge University Press.

Shallice, T., & Saffran, E. (1986). Lexical processing in the absence of explicit word identification:

Evidence from a letter-by-letter reader. *Cognitive Neuropsychology, 3(4)*, 429–458.

Shallice, T., & Warrington, E.K. (1980). Single and multiple component central dyslexic syndromes. In M. Coltheart, K.E. Patterson, & J.C. Marshall (Eds.), *Deep dyslexia*. London: Routledge.

Sieroff, E., Pollatsek, A., & Posner, M.I. (1988). Recognition of visual letter strings following injury to the posterior visual spatial attention system. *Cognitive Neuropsychology, 5(4)*, 427–449.

Snodgrass, J.G., & Mintzer, M. (1993). Neighbourhood effects in visual word recognition: Facilitatory or inhibitory? *Memory and Cognition, 21(2)*, 247–266.

Snodgrass, J.G., & Vanderwart, M.A. (1980). A standardised set of 260 pictures: Norms for name agreement, image agreement, familiarity and visual complexity. *Journal of Experimental Psychology: Learning, Memory and Cognition, 6*, 174–215.

Speedie, L., Rothi, L.J., & Heilman, K.M. (1982). Spelling dyslexia: A form of cross-cuing. *Brain and Language, 15*, 340–352.

Sperling, G., & Melcher, M.J. (1978). Visual search, visual attention and the attention operating characteristic. In J. Requin (Ed.), *Attention and performance*, Vol. 7. Hillsdale, NJ: Erlbaum.

Stachowiak, F.L., & Poeck, K. (1976). Functional disconnection in pure alexia and color naming deficit demonstrated by facilitation methods. *Brain and Language, 3*, 135–143.

Staller, L., Buchanan, D., Singer, M., Lappin, J., & Webb, W. (1978). Alexia without agraphia: An experimental case study. *Brain and Language, 5*, 378–387.

Sternberg, S. (1969). The discovery of processing stages: Extensions of the Donders method. *Acta Psychologia, 30*, 276–315.

Strain, E., Patterson, K., & Seidenberg, M.S. (1995). Semantic effects in single-word naming. *Journal of Experimental Psychology: Learning, Memory & Cognition, 21(5)*, 1140–1154.

Terry, P., Samuels, S.J., & LaBerge, D. (1976). The effects of letter degradation and letter spacing on word recognition. *Journal of Verbal Learning and Verbal Behavior, 15*, 577–585.

Townsend, J.T. (1990). Serial vs. parallel processing. *Psychological Science, 1*, 46–54.

Tuomainen, J., & Laine, M. (1991). Multiple oral re-reading technique in rehabilitation of pure alexia. *Aphasiology, 5*, 401–409.

Van Orden, G.C., Pennington, B.F., & Stone, G.O. (1990). Word identification in reading and the promise of subsymbolic psycholinguistics. *Psycholinguistic Review, 97(4)*, 488–522.

Vargha-Kadem, F., Carr, L.J., Isaacs, E., Brett, F., Adams, C., & Mishkin, M. (1997). Onset of speech after left hemispherectomy in a 9-year-old boy. *Brain, 120*, 159–182.

Vigliocco, G., Semenza, C., & Neglia, M. (1992). Different deficits and different strategies in letter-by-letter reading. Paper presented at the Academy of Aphasia, Toronto.

Warrington, E.K., & Shallice, T. (1980). Word-form dyslexia. *Brain, 103*, 99–112.

Weekes, B.S. (1997). Differential effects of number of letters on word and nonword naming latency. *Quarterly Journal of Experimental Psychology, 50A(2)*, 439–456.

Young, A.W., & Ellis, A.W. (1985). Different methods of lexical access for words presented in the left and right visual hemifields. *Brain and Language, 24*, 326–358.

Young, A.W., & De Haan, E. (1990). Impairments of visual awareness. *Mind and Language, 5*, 29–48.