PATIENT-BASED APPROACHES
TO COGNITIVE NEUROSCIENCE
Second Edition
edited by
Martha J. Farah
Todd E. Feinberg
2006 MIT

Chapter 20

ACQUIRED DISORDERS OF READING

H. Branch Coslett

The study of acquired dyslexia or disorders of reading dates at least to the contributions of Déjerine, who, in 1891 and 1892, described two patients with quite different patterns of reading impairment. Déjerine's first patient[1] developed an impairment in reading and writing subsequent to an infarction involving the left parietal lobe. Déjerine termed this disorder "alexia with agraphia" and attributed the disturbance to a disruption of the "optical image for words," which he thought to be supported by the left angular gyrus. In an account that in some respects presages contemporary psychological accounts, Déjerine concluded that reading and writing required the activation of these "optical images" and that the loss of the images resulted in an inability to recognize or write familiar words.

Déjerine's second patient[2] was quite different. This patient was unable to read aloud or for comprehension but could write, a disorder that Déjerine designated "alexia without agraphia" (also known as agnosic alexia and pure alexia). The patient had a right homonymous hemianopia from a left occipital lesion, which included the fibers carrying visual information from the right to the left hemisphere. Déjerine explained alexia without agraphia in terms of a "disconnection" between visual information confined to the right hemisphere and the left angular gyrus, which he assumed to be critical for the recognition of words.

After the seminal contributions of Déjerine, the study of acquired dyslexia languished for decades, during which the relatively few investigations that were reported focused primarily on the anatomic underpinnings of the disorders. The study of acquired dyslexia was revitalized, however, by the elegant and detailed investigation by Marshall and Newcombe,[3] demonstrating that by virtue of a careful investigation of the pattern of reading deficits exhibited by dyslexic subjects, distinctly different and reproducible types of reading deficits could be elucidated. These investigators described a patient (GR) who read approximately 50 percent of concrete nouns but was severely impaired in the reading of abstract nouns and all other parts of speech. The most striking aspect of GR's performance, however, was his tendency to produce errors that appeared to be semantically related to the target word (e.g., *speak* read as "talk"). Marshall and Newcombe[3] designated this disorder "deep dyslexia." These investigators also described two patients whose primary deficit appeared to be an inability to derive the pronunciation of irregularly spelled words, such as "yacht." This disorder was designated "surface dyslexia."

On the basis of these data, Marshall and Newcombe[3] concluded that the meaning of written words could be accessed by two separate and distinct procedures. The first was a lexical (whole-word) procedure whereby familiar words activated the appropriate stored representation (or visual word form), which, in turn, activated meaning; reading in deep dyslexia was assumed to involve this procedure, labeled A in Fig. 20-1.

The second procedure was assumed to be a phonologically based process in which "grapheme-to-phoneme" (hereafter termed "print-to-sound") correspondences were employed to derive the appropriate phonology (that is, "sound out" the word); the reading of surface dyslexics was assumed to be mediated by this nonlexical procedure, labeled B in Fig. 20-1. Although a number of Marshall and Newcombe's specific hypotheses have been criticized, their argument that reading may be mediated by two distinct procedures has received considerable empirical support. Indeed, although it has occasionally been questioned,[4,5] the dual-route model of reading has provided the conceptual framework that has motivated most subsequent studies of acquired dyslexias and animates the present discussion.

In this chapter we briefly summarize the clinical features and conceptual basis of the major types of acquired dyslexia. Additionally, the possible role of

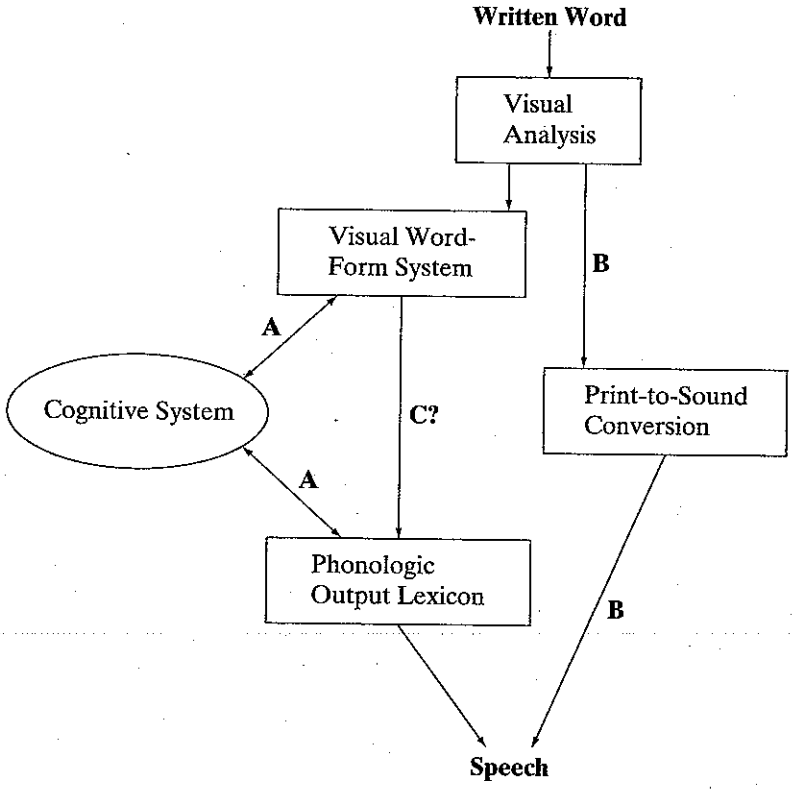

Figure 20-1
A diagram of an information-processing model of reading incorporating three procedures for oral reading.

the right hemisphere in reading is briefly discussed. Finally, recent efforts to develop computational models of normal reading and acquired dyslexia are briefly described.

PERIPHERAL DYSLEXIAS

A useful starting point in the discussion of the dyslexias is the distinction offered by Shallice and Warrington[6] between "peripheral" and "central" dyslexias. The former are conditions characterized by a deficit in the processing of visual aspects of the stimulus that interferes with matching the familiar word to its stored orthographic representation or "visual word form."[6] Central dyslexias, in contrast, are attributable to an impairment of "deeper" or "higher" reading mechanisms by means of which visual word forms gain access to meaning or speech production mechanisms. The major types of peripheral dyslexia are briefly described below.

Alexia without Agraphia (Pure Alexia)

The classic syndrome of alexia without agraphia or pure alexia is perhaps the prototypical peripheral dyslexia. As noted above, the traditional account[2,7] of this disorder attributes the syndrome to a "disconnection" of visual information, which is restricted to the right hemisphere, from the left-hemispheric word-recognition system.

Though these patients do not appear to be able to read in the sense of fast, automatic word recognition, many are able to use a compensatory strategy that involves naming the letters of the word in serial fashion; they read, in effect, letter by letter. Using the slow and inefficient letter-by-letter procedure, pure alexics typically exhibit significant effects of word length, requiring more time to read long as compared to short words. In contrast to the central dyslexias, performance is typically not influenced by linguistic factors such as parts of speech (e.g., noun versus functor), the extent to

which the referent of the word is concrete (e.g., *table*) or abstract (e.g., *destiny*), or whether the word is orthographically regular (that is, can be "sounded out").

A number of alternative accounts of the processing deficit in pure alexia have been proposed. Thus, some investigators have proposed that the impairment is attributable to a limitation in the transmission of letter identity information to the visual word system,[8] an inability to directly encode visual letters as abstract orthographic types,[9,10] or an inability to encode multiple visual shapes of any sort in rapid succession.[11,12] Other investigators have argued that the disorder is attributable to a disruption of the visual word-form system itself.[13,14]

Although most reports of pure alexia have emphasized the profound nature of the reading deficit, often stating that patients were utterly incapable of reading without recourse to a letter-by-letter strategy,[7,8] a number of investigators have reported data demonstrating that at least some pure alexic patients are able to comprehend words that they are unable to explicitly identify.[15–17] This capacity has been attributed by some investigators (e.g., Ref. 17) to the operation of a reading procedure based in the right hemisphere.

The anatomic basis of pure alexia has been extensively investigated. Although on rare occasions associated with lesions that "undercut" or disconnect the posterior perisylvian cortex on the left,[18] the disorder is typically associated with a lesion in the posterior portion of the dominant hemisphere, which compromises visual pathways in the dominant hemisphere and disrupts white matter tracts (such as the splenium of the corpus callosum or forceps major) critical for the interhemispheric transmission of visual information.[19,20]

Neglect Dyslexia

Neglect dyslexia, which is most commonly encountered in patients with left-sided neglect, is characterized by a failure to explicitly identify the initial portion of a letter string. Interestingly, the performance of patients with neglect dyslexia is often influenced by the nature of the letter string; thus, patients with this disorder may fail to report the initial letters in nonwords (e.g., the "ti-" in a nonword such as "tiggle") but read real words (e.g., "giggle") correctly (Ref. 21; see also Refs. 22 and 23). The fact that performance is affected by the lexical status of the stimulus has been taken to suggest that neglect dyslexia is not attributable to a failure to register letter information but reflects an attentional impairment at a higher level of representation (see also Chap. 7).

Although neglect dyslexia is generally seen in the context of the neglect syndrome (see Chaps. 14 and 15), it has occasionally been observed in isolation or even in the context of neglect of the opposite side of space.[24]

Attentional Dyslexia

Perhaps the least studied of the acquired dyslexias, attentional dyslexia is characterized by the relative preservation of single-word reading in the context of a gross disruption of reading when words are presented in text or in the presence of other words or letters.[25–28] Patients with this disorder may also exhibit difficulties identifying letters within words, even though the words themselves are read correctly,[25] and be impaired in identifying words flanked by extraneous letters (e.g., "lboat"). We[28] have recently investigated a patient with attentional dyslexia secondary to autopsy-proven Alzheimer disease who produced frequent "blend" errors in which letters from one word of a two-word display intruded into the other word (e.g., "take lime" read as "tame"). Although several accounts for this disorder have been proposed, the disorder has been attributed by several investigators to an impairment in visual attention or a loss of location information. As visual attention may be critical to mapping the location of visually presented objects, these accounts are not clearly distinguishable.

CENTRAL DYSLEXIAS

In this section we briefly describe the clinical features and conceptual basis of the major types of central dyslexia including "deep," "phonologic," and "surface" dyslexia. Additionally, the phenomenon of "reading without meaning" is discussed.

Deep Dyslexia

Deep dyslexia, the most extensively investigated central dyslexia (see, for example, Coltheart and

colleagues[29]) is in many respects the most compelling. The allure of deep dyslexia is due in large part to the intrinsically interesting hallmark of the syndrome, *semantic errors*. When shown the word *castle*, a deep dyslexic may respond "knight"; similarly, these interesting patients may read *bird* as "canary." At least for some deep dyslexics, it is clear that these errors are not circumlocutions and that the patients are not even aware that they have erred.

While semantic errors are typically regarded as essential for the diagnosis of deep dyslexia, the frequency with which deep dyslexics produce them is quite variable; for some patients, semantic errors may represent the most frequent error type, whereas for others they constitute a small proportion of reading errors. These patients also produce a variety of other types of reading errors, including "visual" errors in which the response bears a clear visual similarity to the target (e.g., *skate* read as "scale") and "morphologic" errors, in which a prefix or suffix is added, deleted, or substituted (e.g., *scolded* read as "scolds"; *governor* read as "government").

Additional hallmarks of the syndrome include a greater success in reading words of high as compared to low imageability. Thus, words such as *table, chair, ceiling,* and *buttercup,* the referents of which are concrete or imageable, are read more successfully by deep dyslexics than words such as *fate, destiny, wish,* and *universal,* the referents of which are abstract.

Also characteristic of the syndrome is part-of-speech effect, such that nouns are read more reliably than modifiers (adjectives and adverbs), which are, in turn, read more accurately than verbs. Deep dyslexics manifest particular difficulty in the reading of functors (a class of words that includes pronouns, prepositions, conjunctions, and interrogatives such as *that, which, they, because, under,* etc.). The striking nature of the part-of-speech effect is illustrated by the patient reported by Saffran and Marin[30] who correctly read the word *chrysanthemum* but was unable to read the *the*! Many errors to functors involve the substitution of a different functor (*that* read as *which*) rather than the production of words of a different class, such as nouns or verbs.

As functors are, in general, less imageable than nouns, verbs, or adjectives, some investigators have claimed that the apparent effect of part of speech is in reality a manifestation of the pervasive imageability effect described above.[31] We have reported a patient, however, whose performance suggests that the part-of-speech effect is not simply a reflection of a more general deficit in the processing of low-imageability words, as the difference remained after functors and content words were matched for imageability.

Finally, all deep dyslexics exhibit a substantial impairment in the reading of nonwords; when confronted with letter strings such as *flig* or *churt,* deep dyslexics are typically unable to employ print-to-sound correspondences to derive phonology; nonwords frequently elicit "lexicalization" errors (e.g., *flig* read as "flag"), perhaps reflecting a reliance on lexical reading in the absence of access to reliable print-to-sound correspondences.

How can deep dyslexia be accommodated by the model of reading depicted in Fig. 20-1? Several alternative explanations have been proposed. Most investigators agree that multiple processing deficits must be hypothesized to account for the full range of symptoms found in deep dyslexia. First, the strikingly impaired performance in reading nonwords and other tasks assessing phonologic function suggests that the print-to-sound conversion procedure is disrupted. Second, the presence of semantic errors and the effects of imageability (a variable usually thought to influence processing at the level of semantics) have been interpreted by many investigators as evidence that these patients also suffer from a semantic impairment; it should be noted in this context, however, that some deep dyslexic patients perform well on tests of comprehension with words they are unable to read aloud. Semantic errors in these patients have been attributed to a deficit in or access to representations in the output phonologic lexicon (Ref. 33; see also Ref. 6). Last, the production of visual errors has been interpreted by some to suggest that these patients suffer from an impairment in the visual word-form system. Other investigators (e.g., Coltheart,[34] Saffran and coworkers[35]) have argued that deep dyslexics' reading is mediated by a system not normally used in reading—that is, the right hemisphere. We will return to the issue of reading with the right hemisphere below.

Although deep dyslexia has occasionally been associated with posterior lesions, this disorder is typically encountered in association with large perisylvian

lesions extending into the frontal lobe. As might be expected given the lesion data, deep dyslexia is usually associated with global or Broca's aphasia but may rarely be encountered in patients with fluent aphasia.

Phonological Dyslexia: Reading without Print-to-Sound Conversion

First described in 1979 by Derouesne and Beauvois,[36] phonological dyslexia is, perhaps, the "purest" of the central dyslexias in that the syndrome appears to be attributable to a selective deficit at some stage in the procedure mediating the translation from print to sound. Thus, although in many respects less arresting than deep dyslexia, phonological dyslexia is of considerable theoretical import. It is of interest to note that the existence of this syndrome was *predicted* by dual-route accounts of reading similar to that proposed by Marshall and Newcombe[3] and subsequently identified when dyslexic patients were assessed with theoretically motivated tasks. It has since become the subject of intensive study by cognitive neuropsychologists interested in the organization of reading in the brain.[37]

Phonological dyslexia is a relatively mild disorder in which reading of real words may be only slightly impaired. Many patients with this disorder, for example, correctly read 85 to 95 percent of real words (e.g., Refs. 32, 36, 38). Some patients with this disorder read all different types of words with equal facility,[38–40] whereas other patients are relatively impaired in the reading of functors.[41,42] Unlike patients with surface dyslexia, described below, the regularity of print-to-sound correspondences is not relevant to the performance of phonological dyslexics; thus, these patients typically pronounce orthographically irregular words such as *colonel* and words with standard print-to-sound correspondences such as *administer* with equal facility. Most errors in response to real words appear to have a visual basis, often involving the substitution of visually similar real words (e.g., *topple* read as "table").

The striking and theoretically relevant aspect of the performance of phonological dyslexics is a substantial impairment in the oral reading of nonword letter strings. A number of investigators have described patients with this disorder, for example, who read more than 90 percent of real words of all types yet correctly pronounce only about 10 percent of nonwords.[32,36] Most errors in nonword reading involve the substitution of a visually similar real word (e.g., *phope* read as "phone") or the incorrect application of print-to-sound correspondences (e.g., *stime* read as "stim," rhyming with "him").

Within the context of the reading model depicted in Fig. 20-1, the account for this disorder is relatively straightforward. The patients' good performance with real words suggests that the processes involved in normal "lexical" reading—that is, visual analysis, the visual word-form system, semantics, and the phonological output lexicon—are at least relatively preserved. The impairment in nonword reading suggests that the print-to-sound translation procedure is disrupted.

A final point of interest is that a number of phonological dyslexics exhibit substantial deficits in processing morphologically complex words—that is, words with prefixes and suffixes.[38,42] The explanation for this association is not clear.

Phonological dyslexia has been observed in association with lesions in a number of sites in the dominant perisylvian cortex and, on occasion, with lesions of the right hemisphere (e.g., Ref. 42). Damage to the superior temporal lobe and angular and supramarginal gyri in particular is found in most but not all patients with this disorder. Although quantitative data are lacking, the lesions associated with phonological dyslexia appear to be smaller on average than those associated with deep dyslexia.

Just as there is variability with respect to the lesion site associated with phonological dyslexia, there is variability with respect to the type and severity of aphasia observed in these patients. A phonological dyslexic reported by Derouesne and Beauvois,[36] for example, did not exhibit a significant aphasia, whereas Funnell's patient W.B.[38] appears to have had a severe nonfluent aphasia.

Surface Dyslexia

Surface dyslexia is a disorder characterized by the inability to read words with "irregular" or exceptional print-to-sound correspondences. Patients with surface dyslexia are thus unable to read aloud words such as *colonel, yacht, island, have,* and *borough,* the pronunciation of which cannot be derived by phonological or "sounding out" strategies. In contrast, these patients

read words containing regular correspondences (e.g., *state, hand, mint, abdominal*) as well as nonwords (e.g., *blape*) quite well.

As noted above, normal subjects may read familiar words by matching the letter string to a stored representation of the word and retrieving the pronunciation by means of a mechanism linked to semantics (or, as discussed below, by means of a nonsemantic "direct" route). As this procedure involves the activation of stored representations, the pronunciation of the word is not computed by rules but is retrieved; consequently, the regularity of print-to-sound correspondences would not be expected to play a major role in performance.

In the context of a dual-route model of reading, the sensitivity to the regularity of the print-to-sound correspondences provides prima facie evidence that the impairment in surface dyslexia is in the mechanism(s) mediating lexical reading. Similarly, the preserved ability to read regular words and nonwords provides compelling support for the claim that the procedures by which pronunciations are computed by the application of print-to-sound correspondences are at least relatively preserved.

Noting that there is substantial variability in the performance of surface dyslexics with respect to leading latencies as well as accuracy, Shallice and McCarthy[43] suggested that the syndrome of surface dyslexia be fractionated. Type 1 surface dyslexia, they suggested, is characterized by effortless and accurate reading of nonwords and regular words with poor performance with irregular words only. Type 2 surface dyslexia, in contrast, is characterized by slow, effortful reading; although these patients read irregular words less well than regular words and nonwords, they make errors with all types of stimuli. More recently, Shallice[44] suggested that at least for patients with type 2 surface dyslexia, the syndrome may reflect an attempt to compensate for damage to early stages of the reading process.

Other investigators have suggested that the syndrome may be fractionated even more. Thus, for example, surface dyslexia may be associated with disruption of the visual word-form system,[3] with a disruption of semantics (in conjunction with deficit in the "direct" route),[45,46] or with a lesion involving the phonological output lexicon.[47] Indeed, Coltheart and Funnell[48] proposed that within the context of a multiroute model of reading, surface dyslexia might be associated with as many as seven distinct types of impairment.

Finally, if as suggested above, patients with surface dyslexia are unable to access semantics by means of a direct lexical procedure, one might ask how these patients derive word meaning. At least for some surface dyslexics, access to a word's meaning appears to occur only after the phonological form of the word has been derived. Thus, when presented the word *listen,* a patient described by Marshall and Newcombe[3] responded "Liston" and added "that's the boxer."

The anatomic correlate of surface dyslexia has not been well established. Indeed, in recent years the syndrome has been reported most frequently in the context of dementia.[46,49–54] Accordingly, surface dyslexia in demented patients is sometimes termed "semantic dyslexia." Many of these patients have exhibited brain atrophy most prominent in the temporal lobes (e.g., Refs. 50 and 53).

Reading without Meaning

In 1979, Schwartz and coworkers[45] reported a patient (WLP) who exhibited a profound loss of semantics in the context of dementia. Her performance was of particular interest because, unlike patients with surface dyslexia, she correctly read aloud both regular and irregular words that she was unable to comprehend. Thus, for example, when asked to sort written words into their appropriate semantic categories, she correctly classified only 7 of 20 animal names; critically, WLP correctly read aloud 18 of these animal names, including such orthographically ambiguous or irregular words as *hyena* and *leopard.* The same basic phenomenon—that is, the ability to read aloud regular and irregular words that the patient does not understand—has subsequently been reported by a number of investigators (see Refs. 55 and 56).

The pattern of performance exhibited by WLP and similar patients is of considerable theoretical interest. Recall that to this point, two procedures have been described by which written words may be pronounced. The first (labeled A in Fig. 20-1) involves the activation of an entry in the visual word-form system, access to semantic information, and ultimately activation of

an entry in the phonological output lexicon. The second (B in Fig. 20-1) involves the nonlexical print-to-sound translation process. Reading without semantics is of interest precisely because it cannot readily be accommodated by such an account. The fact that these patients do not comprehend the words they correctly pronounce indicates that their oral reading is not mediated by the semantically based reading procedure. Additionally, the fact that these patients can read irregular words suggests that they are not relying on a sublexical print-to-sound conversion procedure.

How, then, do these patients read aloud? Several explanations have been proposed. One response was to suggest that oral reading may be mediated by a third mechanism or route (e.g., Ref. 57). This mechanism was assumed to be lexically based, involving the activation of an entry in the visual word-form system and the "direct" activation of an entry in the phonological output lexicon (C in Fig. 20-1); note that this procedure differs from the lexical procedure described above in that there is no intervening activation of semantic information. Based on the analysis of a phonological dyslexic's performance across a variety of reading, writing, and repetition tasks, we[32] have reported data providing additional support for the existence of a lexical but nonsemantic reading procedure. An alternative hypothesis was proposed by Shallice and colleagues (Refs. 44 and 46; see also Ref. 58). These investigators attempted to explain reading without semantics within the context of a dual-route model by proposing that the phonological reading procedure employs not only grapheme-to-phoneme correspondences but also correspondences based on larger units including syllables and even morphemes. Thus, on this account, WLP and similar patients are assumed to compute the pronunciation of irregular words they cannot understand by relying on the multiple levels of print-to-sound correspondences available in the phonological system. Finally, Hillis and Caramazza[59] have suggested that the apparent ability to read without meaning is attributable to the fact that, while the patient is impaired, the semantic and phonological reading procedures provide partial information that constrains the subject's responses. Thus, on this account, neither the semantic nor phonological procedure is assumed to be capable of generating the correct response, but the combination of partial phonological and incomplete semantic information is often sufficient to identify the stimulus.

READING AND THE RIGHT HEMISPHERE

One important and controversial issue regarding reading concerns the putative reading capacity of the right hemisphere. For many years investigators argued that the right hemisphere was "word blind."[2,6,7] In recent years, however, several lines of evidence have suggested that the right hemisphere may possess the capacity to read. One seemingly incontrovertible line of evidence comes from the performance of a patient who underwent a left hemispherectomy at age 15 for treatment of seizures caused by Rasmussen's encephalitis;[60] after the hemispherectomy, the patient was able to read approximately 30 percent of single words and exhibited an effect of part of speech; she was also utterly unable to use a print-to-sound conversion process. Thus, in many respects this patient's performance was similar to that of a person with deep dyslexia, a pattern of reading impairment that has been hypothesized to reflect the performance of the right hemisphere.[34,35]

The performance of some split-brain patients is also consistent with the claim that the right hemisphere is literate. These patients may, for example, be able to match printed words presented to the right hemisphere with an appropriate object.[61,62] Interestingly, the patients are apparently unable to derive sound from the words presented to the right hemisphere; thus, they are unable to determine if a word presented to the right hemisphere rhymes with an auditorially presented word.

Another line of evidence supporting the claim that the right hemisphere is literate comes from evaluation of the reading of patients with pure alexia and optic aphasia.[17,63] We reported data, for example, from four patients with pure alexia who performed well above chance on a number of lexical decision and semantic categorization tasks with briefly presented words that they could not explicitly identify. Three of the patients who regained the ability to identify rapidly presented words explicitly exhibited a pattern of performance consistent with the right-hemisphere reading hypothesis. These patients read nouns better than functors and

words of high (e.g., *chair*) better than words of low (e.g., *destiny*) imageability. Additionally, both patients for whom data were available demonstrated a deficit in the reading of suffixed (e.g., *flowed*) as opposed to pseudo-suffixed (e.g., *flower*) words. These data are consistent with a version of the right-hemisphere reading hypothesis postulating that the right-hemisphere lexical-semantic system primarily represents high imageability nouns. On this account, functors, affixed words, and low imageability words are not adequately represented in the right hemisphere.

Finally, we reported data from an investigation with a patient with pure alexia in which transcranial magnetic stimulation (TMS) was employed to directly test the hypothesis that the right hemisphere mediates the reading of at least some patients with acquired dyslexia.[64] We reasoned that if the right hemisphere provides the neural substrate for reading, the transient, localized disruption of cortical processing caused by TMS of the right hemisphere would interfere with reading. An extensively investigated patient with pure alexia who exhibited the reading pattern described above was asked to read aloud briefly presented words, half of which were presented in association with TMS. Consistent with the hypothesis that his reading was mediated by the right hemisphere, stimulation of the right hemisphere interfered with oral reading, whereas left-hemisphere stimulation had no significant effect.

Although a consensus has not yet been achieved, there is mounting evidence that, at least for some people, the right hemisphere is not word-blind but may support the reading of some types of words. The full extent of this reading capacity and whether it is relevant to normal reading, however, remain unclear.

COMPUTATIONAL MODELS OF THE DYSLEXIAS

To this point, the discussion of acquired reading disorders has been motivated by a widely though not universally (see Refs. 4 and 5) accepted multiroute information processing model of reading. In recent years, however, computer-implemented parallel distributed processing (PDP) models of cognitive processing have made important contributions in many domains of cognitive science, including reading (see Chap. 7). These models, which differ from traditional information processing models in that they offer (and in fact require) greater specification of the manner in which information is represented and processed, have called into question the necessity of hypothesizing two routes to account for the syndromes reviewed here. Although a detailed discussion of these models is beyond the scope of this chapter, several PDP accounts of reading are briefly summarized below.

Seidenberg and McClelland[65] have reported a PDP model of single-word reading in which the procedure for computing pronunciation directly from orthography (that is, without semantic mediation) is assumed to be mediated by a single network in which orthographic patterns are linked to phonological representations by means of an intermediate "hidden layer."[65] In contrast to the information processing accounts described above, this model does not postulate a discrete "lexical" or word-representation procedure or distinct lexical and sublexical procedures for the computation of phonology. Of particular relevance in the present context is the fact that investigators have attempted to simulate the performance of dyslexic patients by modifying or "lesioning" this PDP model. Patterson and colleagues,[66] for example, have attempted to model the performance of surface dyslexics by eliminating a proportion of the connections or units at different "lesion" sites. Although the simulations do not appear to capture all of the characteristic features of the performance of surface dyslexics, the lesioned models generate data that are in many interesting and important respects similar to those of patients. More recently, Plaut and Shallice[67] have reported a series of simulations of different PDP architectures in an attempt to model the performance of patients with deep dyslexia.

Finally, Seidenberg and Joanisse have recently extended their computational approach to reading to an issue of considerable theoretical importance: the reading of prefixed and suffixed (that is, "multimorphemic") words.[68] On the basis of empiric studies with normals as well as data from a computational model, Gonnerman et al.[69,70] argue that morphologic structure is an "emergent, interlevel representation that mediates computations between form and meaning" rather than an explicit level of representation.

An alternative computational account of reading has been developed by several investigators. Reggia and coworkers[71] developed a model that incorporates both lexical and nonlexical procedures for the computation of phonology. This model, which employs a competitive distribution of activation to govern interaction between competing concepts, simulates many aspects of normal reading performance. In a series of elegant investigations, Coltheart and colleagues[72,73] have described a computationally instantiated version of dual-route theory similar to that presented in Fig. 20-1, the "dual-route cascaded" model. This account incorporates a "lexical" route (similar to C in Fig. 20.1) as well as a "nonlexical" route by which the pronunciation of graphemes is computed on the basis of position-specific correspondence rules. Like the PDP models described above, the dual-route cascaded model accommodates a wide range of findings from the literature on normal reading. And as with the PDP models, "lesioning" the dual-route cascaded model produces disorders that are, at least in many respects, similar to acquired dyslexias described earlier in this chapter.[74]

A full discussion of the relative merits of these models as well as other approaches to the understanding of reading and acquired dyslexia (e.g., Ref. 75) is beyond the scope of this chapter. It would appear likely, however, that investigations of acquired dyslexia will help to adjudicate between competing accounts of reading and that these models will continue to offer critical insights into the interpretation of data from brain-injured subjects.

REFERENCES

1. Déjerine J: Sur un cas de cécité verbale avec agraphie, suivi d'autopsie. *C R Séances Soc Biol* 3:197–201, 1891.
2. Déjerine J: Contribution à l'étude anatomo-pathologique et clinique des différentes variétés de cécité verbale. *C R Séances Soc Biol* 4:61–90, 1892.
3. Marshall JC, Newcombe F: Patterns of paralexia: A psycholinguistic approach. *J Psycholinguist Res* 2:175–199, 1973.
4. Marcel AJ: Surface dyslexia and beginning reading: A revised hypothesis of the pronunciation of print and its impairments, in Coltheart M, Patterson KE, Marshall JC (eds): *Deep Dyslexia.* London: Routledge, 1980.
5. Van Orden GC, Pennington BF, Stone GO: Word identification in reading and the promise of subsymbolic psycholinguistics. *Psychol Rev* 97:488–522, 1990.
6. Shallice T, Warrington EK: Single and multiple component central dyslexic syndromes, in Coltheart M, Patterson K, Marshall JC (eds): *Deep Dyslexia.* London: Routledge, 1980.
7. Geschwind N: Disconnection syndromes in animals and man. *Brain* 88:237–294, 585–644, 1965.
8. Patterson K, Kay J: Letter-by-letter reading: Psychological descriptions of a neurological syndrome. *Q J Exp Psychol* 34A:411–441, 1982.
9. Arguin M, Bub DN: Pure alexia: Attempted rehabilitation and its implications for interpretation of the deficit. *Brain Lang* 47:233–268, 1994.
10. Arguin M, Bub DN: Single-character processing in a case of pure alexia. *Neuropsychologia* 31:435–458, 1993.
11. Kinsbourne M, Warrington EK: A disorder of simultaneous form perception. *Brain* 85:461–486, 1962.
12. Farah MJ, Wallace MA: Pure alexia as a visual impairment: A reconsideration. *Cognit Neuropsychol* 8:313–334, 1991.
13. Warrington EK, Shallice T: Word-form dyslexia. *Brain* 103:99–112, 1980.
14. Warrington EK, Langdon D: Spelling dyslexia: A deficit of the visual word-form. *J Neurol Neurosurg Psychiatry* 57:211–216, 1994.
15. Landis T, Regard M, Serrat A: Iconic reading in a case of alexia without agraphia caused by a brain tumor: A tachistoscopic study. *Brain Lang* 11:45–53, 1980.
16. Shallice T, Saffran EM: Lexical processing in the absence of explicit word identification: Evidence from a letter-by-letter reader. *Cognit Neuropsychol* 3:429–458, 1986.
17. Coslett HB, Saffran EM: Evidence for preserved reading in pure alexia. *Brain* 112:327–329, 1989.
18. Greenblatt SH: Subangular alexia without agraphia or hemianopia. *Brain Lang* 3:229–245, 1976.
19. Binder JR, Mohr JP: The topography of callosal reading pathways: A case-control analysis. *Brain* 115:1807–1826, 1992.
20. Damasio AR, Damasio H: The anatomic basis of pure alexia. *Neurology* 33:1573–1583, 1983.
21. Sieroff E, Pollatsek A, Posner MI: Recognition of visual letter strings following injury to the posterior visual spatial attention system. *Cognit Neuropsychol* 5:427–449, 1988.
22. Behrman M, Moscovitch M, Black SE, Mozer M: Perceptual and conceptual mechanisms in neglect dyslexia. *Brain* 113:1163–1183, 1990.

23. Berti A, Frassinetti F, Umilta C: Nonconscious reading? Evidence from neglect dyslexia. *Cortex* 30:181–197, 1994.
24. Costello AD, Warrington EK: The dissociation of visual neglect and neglect dyslexia. *J Neurol Neurosurg Psychiatry* 50:110–116, 1987.
25. Shallice T, Warrington EK: The possible role of selective attention in acquired dyslexia, *Neuropsychologia* 15:31–41, 1977.
26. Price CJ, Humphreys GW: Attentional dyslexia: The effects of co-occurring deficits. *Cognit Neuropsychol* 6:569-592, 1993.
27. Warrington EK, Cipolotti L, McNeil J: Attentional dyslexia: A single case study. *Neuropsychologia* 31:871–886, 1993.
28. Saffran EM, Coslett HB: "Attentional dyslexia" in Alzheimer's disease: A case study. *Cognit Neuropsychol.* In press.
29. Coltheart M, Patterson K, Marshall JC (eds): *Deep Dyslexia*. London: Routledge, 1980.
30. Saffran EM, Marin OSM: Reading without phonology: Evidence from aphasia. *Q J Exp Psychol* 29:515–525, 1977.
31. Allport DA, Funnell E: Components of the mental lexicon. *Phil Trans R Soc Lond B* 295:397–410, 1981.
32. Coslett HB: Read but not write "idea": Evidence for a third reading mechanism. *Brain Lang* 40:425–443, 1991.
33. Caramazza A, Hillis AE: Where do semantic errors come from? *Cortex* 26:95–122, 1990.
34. Coltheart M: Deep dyslexia: A right hemisphere hypothesis, in Coltheart M, Patterson K, Marshall JC (eds): *Deep Dyslexia*. London: Routledge, 1980.
35. Saffran EM, Bogyo LC, Schwartz MF, Marin OSM: Does deep dyslexia reflect right-hemisphere reading? in Coltheart M, Patterson K, Marshall JC (eds): *Deep Dyslexia*. London: Routledge, 1980.
36. Derouesne J, Beauvois M-F: Phonological processing in reading: Data from alexia. *J Neurol Neurosurg Psychiatry* 42:1125–1132, 1979.
37. Coltheart M (ed): *Special Issue on Phonological Dyslexia, Cognit Neuropsychol,* 13, 1996.
38. Funnell E: Phonological processes in reading: New evidence from acquired dyslexia. *Br J Psychol* 74:159–180, 1983.
39. Bub D, Black SE, Howell J, Kertesz A: Speech output processes and reading, in Coltheart M, Sartori G, Job R (eds): *Cognitive Neuropsychology of Language*. Hillsdale, NJ: Erlbaum, 1987.
40. Friedman RB, Kohn SE: Impaired activation of the phonological lexicon: Effects upon oral reading. *Brain Lang* 38:278–297, 1990.
41. Glosser G, Friedman RB: The continuum of deep/ phonological dyslexia. *Cortex* 26:343–359, 1990.
42. Patterson KE: The relation between reading and psychological coding: Further neuropsychological observations, in AW Ellis (ed): *Normality and Pathology in Cognitive Functions*. London: Academic Press, 1982.
43. Shallice T, McCarthy R: Phonological reading: From patterns of impairment to possible procedures, in Patterson KE, Coltheart M, Marshall JC (eds): *Surface Dyslexia*. London: Erlbaum, 1985.
44. Shallice T: *From Neuropsychology to Mental Structure*. Cambridge, England: Cambridge University Press, 1987.
45. Schwartz MF, Saffran EM, Marin OSM: Dissociation of language function in dementia: A case study. *Brain Lang* 7:277–306, 1979.
46. Shallice T, Warrington EK, McCarthy R: Reading without semantics. *Q J Exp Psychol* 35A:111–138, 1983.
47. Howard D, Franklin S: Three ways for understanding written words, and their use in two contrasting cases of surface dyslexia (together with an odd routine for making "orthographic" errors in oral word production), in Allport A, Mackay D, Prinz W, Scheerer E (eds): *Language Perception and Production*. New York: Academic Press, 1987.
48. Coltheart M, Funnell E: Reading writing: One lexicon or two? in Allport DA, MacKay DG, Printz W, Scheerer E (eds): *Language Perception and Production: Shared Mechanisms in Listening, Speaking, Reading and Writing*. London: Academic Press, 1987.
49. Warrington EK: The selective impairment of semantic memory. *Q J Exp Psychol* 27:635–657, 1975.
50. Hodges JR, Patterson K, Oxbury S, Funnell E: Semantic dementia: Progressive fluent aphasia with temporal lobe atrophy. *Brain* 115:1783–1806, 1992.
51. Patterson K, Hodges J: Deterioration of word meaning: Implications for reading. *Neuropsychologia* 30:1025–1040, 1992.
52. Graham KS, Hodges JR, Patterson K: The relationship between comprehension and oral reading in progressive fluent aphasia. *Neuropsychologia* 32:299–316, 1994.
53. Breedin SD, Saffran EM, Coslett HB: Reversal of the concreteness effect in a patient with semantic dementia. *Cognit Neuropsychol* 11:617–660, 1994.
54. Cipolotti L, Warrington EK: Semantic memory and reading abilities: A case report. *J Int Neuropsychol Soc* 1:104–110, 1994.
55. Friedman RB, Ferguson S, Robinson S, Sunderland T: Dissociation of mechanisms of reading in Alzheimer's disease. *Brain Lang* 43:400-413, 1992.

56. Raymer AM, Berndt RS: Models of word reading: Evidence from Alzheimer's disease. *Brain Lang* 47:479–482, 1994.
57. Morton J, Patterson KE: A new attempt at an interpretation, or an attempt at a new interpretation, in Coltheart M, Patterson K, Marshall JC (eds): *Deep Dyslexia*. London: Routledge, 1980.
58. McCarthy RA, Warrington EK: Phonological reading: Phenomena and paradoxes. *Cortex* 22:359–380, 1986.
59. Hillis AE, Caramazza A: Mechanisms for accessing lexical representations for output: Evidence from a category-specific semantic deficit. *Brain Lang* 40:106–144, 1991.
60. Patterson K, Vargha-Khadem F, Polkey CF: Reading with one hemisphere. *Brain* 112:39–63, 1989.
61. Zaidel E: Lexical organization in the right hemisphere, in Buser P, Rougeul-Buser A(eds): *Cerebral Correlates of Conscious Experience*. Amsterdam: Elsevier, 1978.
62. Zaidel E, Peters AM: Phonological encoding and ideographic reading by the disconnected right hemisphere: Two case studies. *Brain Lang* 14:205–234, 1981.
63. Coslett HB, Saffran EM: Preserved object recognition and reading comprehension in optic aphasia. *Brain* 12:1091–1110, 1989.
64. Coslett HB, Monsul N: Reading and the right hemisphere: Evidence from transcranial magnetic stimulation. *Brain Lang* 46:198–211, 1994.
65. Seidenberg MS, McClelland JL: A distributed, developmental model of word recognition and naming. *Psychol Rev* 96:522–568, 1989.
66. Patterson KE, Seidenberg MS, McClelland JL: Connections and disconnections: Acquired dyslexia in a computational model of reading processes, in Morris RGM (ed): *Parallel Distributed Processing: Implications for Psychology and Neurobiology*. Oxford, England: Oxford University Press, 1989.
67. Plaut D, Shallice T: Deep dyslexia: A case study of connectionist neuropsychology. *Cognit Neuropsychol* 10:377–500, 1993.
68. Joanisse MF, Seidenberg MS: Impairments in verb morphology after brain injury: A connectionist model. *Proc Natl Acad Sci U S A* 96:7592–7597, 1999.
69. Plaut DC, Gonnerman LM: Are non-semantic morphological effects incompatible with a distributed connectionist approach to lexical processing? *Lang Cogn Proc* 15:445–485, 2000.
70. Gonnerman M, Devlin JT, Andersen ES, Seidenberg MS: Derivational morphology as an emergent interlevel representation (personal communication, 2002).
71. Reggia J, Marsland P, Berndt R: Competitive dynamics in a dual-route connectionist model of print-to-sound transformation. *Complex Systems* 2:509–547, 1988.
72. Rastle K, Coltheart, M: Serial and strategic effects in reading aloud. *J Exp Psychol Hum Percept Perform* 25:482–503, 1999.
73. Coltheart M, Rastle, K, Perry C, et al: DRC: A dual route cascaded model of visual word recognition and reading aloud. *Psychol Rev* 108:204–256, 2001.
74. Coltheart M, Langdon R, Haller M: Simulations of acquired dyslexias by the DRC model, a computational model of visual word recognition and reading aloud, in Proceedings of the 1995 Workshop on Neural Modeling of Cognitive and Brain Disorders, College Park, MD: University of Maryland, June 8–10, 1995.
75. Van Orden GC, Jansen op de Haar MA, Bosman AM: Complex dynamic systems also predict dissociations but they do not reduce to autonomous components. *Cognit Neuropsychol* 14:131–165, 1997.