CHAPTER FOUR

The Acquired Dyslexias

The brain is the organ of the mind. It is also an organ of the body and, as such, is susceptible to injury and illness. The most common cause of injury to the brain is a stroke; that is, a disruption of the blood supply to a part of the brain which may be caused by the bursting of an artery (haemorrhage) or the blockage of an artery by a blood clot. Working in France in the 1860s, Paul Broca showed that, in right-handed people, language problems tend to occur after damage to the left rather than the right half of the brain. It is the left half, or hemisphere, of the brain which in most people is primarily responsible for language abilities, including the skills of reading and writing.

Disorders affecting the comprehension or production of spoken language which occur as a consequence of brain injury are known as aphasias. There are many different types of aphasia, depending upon just what aspects of speech processing have been impaired (Ellis & Young, 1988; McCarthy & Warrington, 1990). Aphasic patients often experience reading difficulties which are part and parcel of their more general language impairments. On other occasions, reading problems are the predominant symptom, or cannot be explained solely by reference to the broader aphasia. In either case, we would talk about the patient suffering from an “acquired dyslexia”. Acquired dyslexia was studied in the late nineteenth century by neurologists such as Carl Wernicke, but most of the research on acquired dyslexias has been done since the mid-1970s. Cognitive psychology, which is the study of normal mental processes such as perception, memory, language and reading, came together at that point with neuropsychology, which is the study of the human brain and its functions, to create cognitive neuropsychology, the study of disorders of cognitive function that arise as a consequence of brain injury. When cognitive neuropsychologists investigate acquired dyslexia, their approach is
not so much to ask which part of the brain is damaged in which form of reading disorder, but to ask which part of the normal reading process have been damaged or lost. That is, they seek to explain different patterns of reading breakdown by reference to models of the normal, skilled reading process such as that presented in Fig. 3.1.

Such cognitive models incorporate a number of different processes ranging from letter identification and visual word recognition, to semantic comprehension and phonological appreciation of the sound-forms of words. If brain injury can affect each of those aspects of reading, damaging certain aspects in one patient and other aspects in another, then we should expect to see a variety of qualitatively different forms of acquired dyslexia, which we do. We might also expect to be able to explain each form of acquired dyslexia by giving an account of which aspects of normal skilled reading have been impaired (and how), and which remain more or less intact. That, broadly speaking, is the goal of the cognitive neuropsychological study of acquired dyslexia.

But cognitive neuropsychologists also want to be able to use data from patients to test and develop theories of normal skilled reading. They want data from patients to be regarded as being on a par with data from laboratory experiments with normal subjects. For example, a cognitive neuropsychologist might assert that it would not be possible to observe a particular pattern of acquired reading disorder if the normal reading system were organised in one way, but could be accounted for if the normal system were organised in another, different way. Examples of this mode of argument will be presented in this chapter. Virtually all of the work done on acquired dyslexia has concentrated on patients' ability to read single words, so it is models of word recognition rather than theories of sentence or text processing that have been considered relevant, and that studies of acquired dyslexia have been directed towards.

Shallice and Warrington (1980) introduced a useful distinction between peripheral and central acquired dyslexias that we shall follow here. In terms of the model presented in Fig. 3.1, peripheral dyslexias are disorders in which the visual analysis system is damaged, resulting in a range of conditions in which the perception of letters in words is impaired. The central dyslexias are a collection of disorders in which processes beyond the visual analysis system are damaged, resulting in difficulties affecting the comprehension
and/or pronunciation of written words.

PERIPHERAL DYSLEXIAS

Neglect Dyslexia

Ellis, Flude, and Young (1987) reported the case of patient V.B. who, unlike most acquired dyslexics, was not aphasic—her capacity to speak and to understand the speech of others was unimpaired by the stroke she had experienced. This was because V.B.’s stroke had affected the right side of her brain rather than the left side which, as we have seen, is for most people the side which controls language use. Nevertheless, V.B. had problems with reading. When she was trying to read passages of text she would often make no attempt to read the first few words of each line. When shown single words, she made errors affecting the first letter or two. For example, she misread NUN as “run”, YELLOW as “pillow”, CLOVE as “love” and HADDOCK as “paddock”. If V.B. was asked to read a word and also explain what it meant, the definition she gave always matched the error she made. Thus, she misread RICE as “price” and defined it as “how much for a paper or something in a shop”, misread LIQUID as “squid” and said it was “a kind of sea creature”, and misread CLOVER as “lover”, adding, “partner, or someone you have an affair with; a sweetheart”.

V.B.’s problem was definitely visual in nature. Her identification of words spelled aloud to her was excellent, and if a passage of text was rotated clockwise through 90° so that the lines ran from top to bottom rather than from left to right, her performance improved considerably. The majority of her errors involved replacing rather than simply deleting the initial letters of words. This was true even when deleting the first letter or two left a real word. Thus she misread FABLE as “table” rather than “table”, and BEAT as “beat” rather than “eat”. That is, even when she got the first letters of a word wrong, she seemed in some way to know that there were letters there.

One account of this form of neglect dyslexia asserts that the problem arises from a failure to attend to the left side of words. Riddoch, Humphreys, Cleeton, and Fery (1991) sought to test this by examining the effect of deliberately drawing a patient’s attention to the left sides of words. They described a patient, J.B., with a neglect dyslexia similar to V.B.’s. Examples of J.B.’s errors are GROSS
misread as “cross” and BOUGH as “slough”. Riddoch et al. tested the attentional theory by placing a hash sign (#) to the left of words and instructing J.B. to locate the hash before attempting to read each word aloud. Performance improved relative to his reading of the same words without hashes, lending support to the attentional theory.

The attentional problem experienced by V.B., J.B. and other patients like them affects their ability to identify the beginning letters of words though they retain some awareness of the presence of letters in those initial positions. Somewhat different varieties of neglect dyslexia requiring some-what different explanations are reviewed by Ellis, Young, and Flude (1993).

Attentional Dyslexia

Given the fact that we have just ascribed neglect dyslexia to a disorder of attention, it is more than a little confusing to find the next variety of acquired dyslexia labelled “attentional dyslexia”. Shallice and Warrington (1977) described two acquired dyslexic patients who were reasonably good at reading words presented singly and who could name single letters almost perfectly. These patients’ problems arose when there were several letters in a row or several words on the page. They then began to make errors. Where words were concerned, the errors they made were very similar to errors that can be observed in normal skilled readers if groups of words are presented very briefly. Under those circumstances, letters may “migrate” from one word to another, so that a subject shown GLOVE and SPADE may report having seen “GLADE” (Allport, 1977; Mozer, 1983). The difference between these normal readers and Shallice and Warrington’s (1977) patients is that the patients made errors even when given unlimited time to read the words. Shallice and Warrington suggest that an attentional process is required to focus upon a letter or word that is being identified and to ensure that one is not flooded with information from elsewhere in the visual world. This attentional process is deficient in attentional dyslexia and is presumably prevented from functioning properly in normal readers when groups of words are displayed for very brief intervals.

Letter-by-letter Reading

Some patients, faced with a written word, will work their way
her illness, she could read aloud both regular and irregular words and could also read nonwords aloud.

As far as one can tell, W.L.P. was not reading via meanings. She could read nonwords, so her sublexical letter-sound conversion procedures were preserved, but she could also read irregular words aloud correctly, implying preservation of whole-word reading via the lexicon. Yet comprehension of written words was very poor. In terms of Fig. 3.1, we would say that W.L.P. had an impaired semantic system but was still able to read words aloud using the connections between the visual input lexicon and the speech output lexicon. Indeed, the existence of patients like W.L.P. might be taken as evidence for the existence of such a non-semantic but lexical reading procedure. W.L.P.'s ability to read nonwords implies that the connections between the visual analysis system and the phoneme level were also preserved.

**Surface Dyslexia**

It is normal in reviews of this kind to present the symptoms of a particular kind of disorder and then discuss the interpretation, but for this and the next variety of acquired dyslexia it is easier to take the reverse approach—to present the interpretation and then work back to the symptoms.

The term “surface dyslexia” was coined by John Marshall and Freda Newcombe in their seminal paper, “Patterns of paralexia: A psycholinguistic approach”, published in 1973. Surface dyslexics show a high reliance on the sublexical procedure in reading aloud; that is, on letter–sound conversion using the route connecting the visual analysis system to the phoneme level. They treat once-familiar words as if they were unfamiliar, breaking them down into their component letters and letter groups, converting each into phonemes and pronouncing the resulting sound sequence. This works reasonably well for regular words because they are by definition the words whose letter–sound relations match the normal correspondences of English. Surface dyslexics are, however, prone to misread irregular words, pronouncing them as if they were regular. Thus ISLAND becomes “izland”, SUGAR becomes “sudger” and BROAD becomes “brode”.

Better reading aloud of regular than irregular words with regularisation errors to irregular words are the hallmarks of surface dyslexia (Patterson, Marshall, & Coltheart, 1985). We shall
laboriously through it, identifying the letters one at a time before trying to say what the word is. Such patients are known as “letter-by-letter readers” (Patterson & Kay, 1982; Warrington & Shallice, 1980). It is important to appreciate that these patients are not reading phonically: they convert letters into their names (Aitch, Vee) not their sounds (“ihn”, “vuh”). Although their word recognition is slow and error-prone, they read irregular words (“Y, A, C, H, T...yacht”) as successfully as regular words (“S, H, I, P... ship”).

Because they identify one letter at a time, the longer a word is, the longer they take to identify it. This is a hallmark of letter-by-letter reading, even in patients who employ the strategy without naming the letters aloud. The speed with which skilled readers can identify familiar words shows only a very small effect of the number of letters in a word, because the visual analysis system of skilled readers can identify the component letters of a word simultaneously, and can transmit those letters “in parallel” to the visual input lexicon. Letter-by-letter readers, in contrast, are reduced to a serial, one-letter-at-a-time identification process (though precisely how they identify words, and what role letter names play in identification, is still unclear).

**CENTRAL DYSEXIAS**

Non-semantic Reading

Schwartz, Marin, and Saffran (1979) and Schwartz, Saffran, and Marin (1980) describe a 62-year-old woman, W.L.P., who was suffering from a progressive dementia, including a generalised loss of memory. At one stage in her illness, W.L.P. was quite unable to match written animal names against their appropriate pictures. When presented with 20 low-frequency animal names such as hyena, leopard and llama, together with some names of body parts and some colour names, she could only sort out 7 of the 20 animal names as referring to animals at all. Nevertheless, she managed to read aloud 18 of the 20 animal names, including the three mentioned above, and made only minor errors on the other two. In general, W.L.P. displayed a remarkable capacity to read words aloud despite showing very little evidence of understanding many of them, and despite being quite unable to use them in her own limited spontaneous speech. Nevertheless, until the final stages of
Phonological Dyslexia

Phonological dyslexia is in many ways the mirror image of surface dyslexia. The sublexical procedure that mediates much of the reading performance of a surface dyslexic is precisely the procedure that is impaired in phonological dyslexia. The result is a mild form of acquired dyslexia, which could easily be missed if one were not on the look out for it, a fact which may explain why it has only been noticed and reported relatively recently.

Phonological dyslexics are no longer able to make effective use of the sublexical reading procedure represented in Fig. 3.1 by the connection between the visual analysis system and the phoneme level. As a result, they are virtually unable to read unfamiliar words or invented nonwords aloud. Familiar words, in contrast, can be read reasonably successfully. Patient W.B. reported by Funnell (1983) read 93 of a set of 100 common nouns correctly but could manage only 2 of 20 simple nonwords. Often the best she could do was to read a nonword as a similar-looking familiar word (e.g. reading COBE as “comb”, PLOON as “spoon” and FUDE as “fudge”). These errors are known as “lexicalisations”. The relative
preservation of word reading suggests (among other things) a preservation of the visual input lexicon. W.B. seemed to be able to make a response to a nonword by allowing its sequence of letters to activate the closest recognition unit in the visual input lexicon.

Phonological dyslexia may seem an obscure form of reading disorder (patients with acquired dyslexia who can read real words quite well), but it is of theoretical interest for at least two reasons. One is the “double dissociation” it forms with surface dyslexia. Phonological dyslexics read on a whole-word basis and are severely impaired at the sort of sublexical processing required to read unfamiliar words and nonwords aloud. Surface dyslexics, in contrast, rely heavily on sublexical processing and have impaired whole-word recognition processes. So phonological dyslexics have reasonably intact whole-word recognition but impaired sublexical procedures, whereas surface dyslexics show the opposite pattern. Cognitive neuropsychologists interpret such “double dissociations” as indicating that whole-word and sublexical reading are mediated by cognitive processes (modules) that are at least to some degree separate within the mind and brain. That is, they interpret the contrast between phonological and surface dyslexia as supporting the separation in Fig. 3.1 of sublexical letter-sound conversion procedures mediated by connections between the visual analysis system and the phoneme level from whole-word recognition mediated by the visual input lexicon.

The second reason why acquired phonological dyslexia is of interest is that many of the children diagnosed as having developmental dyslexia present with symptoms very like those of acquired phonological dyslexia, so much so that it has become quite common for psychologists to talk about “developmental phonological dyslexia”. We shall discuss possible parallels between developmental and acquired dyslexia, and some reasons for caution, in Chapter 8.

**Deep Dyslexia**

Imagine yourself seated at a table in a hospital room. Opposite you is a patient who has suffered a stroke but who is alert and interested. You have brought along a pack of plain cards each of which has a single word written on it. You hold up a card bearing the word APE. “Can you read this word aloud for me?” you ask. “Certainly”, replies the patient, “that’s monkey”. The next card you hold up
bears the word SOUL, which the patient reads as “soup”. He then goes on to read BABY correctly, LOVELY as “loving”, HIS as “in”, FOREST as “trees”, WINDOW correctly, BOAP as “don’t know”, CHANCE as “don’t know”, SYMPATHY as “orchestra”, SIGNAL as “single”; QUIT as “perhaps”, BELIEF as “pray”, WAS as “one of those little words—don’t know”. WHEN as “chick” and BUILDING as “builder”. If this did indeed happen to you, then you would know you were sitting opposite a patient with “deep dyslexia” (Coltheart, Patterson, & Marshall, 1987).

Deep dyslexics find words like BABY, CHURCH or TABLE, which have concrete, imaginable referents, easier to read than abstract words like BELIEF, TRUTH or JUSTICE. Like phonological dyslexics, deep dyslexics also find unfamiliar words and nonwords virtually impossible to read aloud. Deep dyslexics also make several different types of reading error. First and most striking are the “semantic errors” such as misreading APE as “monkey”, FOREST as “trees” or BELIEF as “pray”. Secondly, there are visual errors (e.g. reading SOUL as “soup” or SIGNAL as “single”). A third type of error appears to be a combination of a visual error followed by a semantic error. For example, it is assumed that the patient who misread SYMPATHY as “orchestra” had made a visual error (misreading SYMPATHY as SYMPHONY, and followed that with a semantic error to convert SYMPHONY into “orchestra”). Similarly, the patient who misread WHEN as “chick” is presumed to have committed a visual error (WHEN to HEN), followed by a semantic error (HEN to “chick”).

LOVELY misread as “loving” and BUILDER as “building” are placed in a separate category of “derivation errors”, though we may note that the target and error words are both visually and semantically related in these cases. Finally, HIS misread as “in” or QUIT as “perhaps” are termed “function word substitutions”. It seems surprising that deep dyslexics have such difficulty with what are some of the most common words in the language, but the explanation may lie in the fact that although they are common words, the meanings of function words like IN, QUIT and PERHAPS are abstract and hard to visualise. It may be the abstractness of function words that makes them hard for deep dyslexics. Note, though, that the errors made to function words usually involve substituting another function word, so the patient seems to know at some level what sort of word he or she is being
asked to read.

Although patients with symptoms like those of deep dyslexia have been reported from time to time over the years (Marshall & Newcombe, 1980), the first full descriptions were provided by Marshall and Newcombe in 1966 and again in the same 1973 paper in which surface dyslexia was reported. Marshall and Newcombe adopted the same approach to explaining deep dyslexia as they took to explaining surface dyslexia; that is, trying to explain the disorder in terms of impairment to components of a model of normal reading. The semantic errors and the imageability effect (better reading of concrete than abstract nouns and, perhaps, the problem with function words) suggest two things. One is that deep dyslexics attempt to read via the semantic system; the second is that there is damage in or around that system. At least some deep dyslexics are able to recognize abstract words as words in a lexical decision task even though they cannot read them aloud (Patterson, 1979). This suggests that in these patients the visual input lexicon continues to function, but the direct connections between the visual input lexicon and the speech output lexicon (which, if present, could support non-semantic reading) must be lost.

The almost complete inability of deep dyslexics to read nonwords aloud suggests that they have also lost the capacity for sublexical letter-sound conversion; that is, the connections between the visual analysis system and the phoneme level are gone. This may be a necessary condition for semantic errors to occur in any number, because even a little phonetic skill might prevent a patient from misreading FOREST as “trees” (because the patient would know that the correct pronunciation should start with an “F” sound).

Any explanation of deep dyslexia in terms of damage to the normal reading process will have to postulate several different impairments. The appropriateness of such accounts of deep dyslexia has, however, been challenged by Coltheart (1980; 1983). He argues that in deep dyslexia we are not seeing the effects of loss of some aspects of the normal, left hemisphere reading system. Instead, Coltheart argues that in deep dyslexics most of the left hemisphere reading processes have been completely destroyed and the residual reading abilities are mediated by the patient’s right hemisphere, which is normally considered “non-verbal” but which may play host to certain limited language skills, at least in some people. Some support for this hypothesis has come in the form of a
The report by Patterson, Vargha-Khadem, and Polkey (1989) of the case of a 17-year-old girl, N.I. After an apparently normal childhood, N.I. became severely epileptic with almost continuous seizures and a progressive degeneration of the left cerebral hemisphere, which would almost certainly have proved fatal had the diseased hemisphere not been removed in its entirety. After a period of recovery, N.I. was shown to have retained some reading capacity, but her residual reading abilities showed strong similarities to those of deep dyslexia patients. N.I. was significantly better at reading concrete than abstract nouns, made semantic errors (e.g. misreading ARM as “finger”) and visual errors (e.g. misreading BUSH as “brush”), and was very poor at reading non-words.

The report of N.I.’s deep dyslexic reading after surgical removal of the left hemisphere clearly supports the right hemisphere hypothesis of deep dyslexia, but the rival view that deep dyslexia can, at least sometimes, reflect the residual capabilities of a damaged left hemisphere, still has its adherents. We should note, though, that if the right hemisphere hypothesis proves to be correct, then studying deep dyslexia may not teach us much about normal reading processes. At best, we might learn about the properties of a secondary system whose contribution, if any, to normal reading we would then have to work out.

FURTHER READING


