

Studies in alexia.

Part I – The alexias and models of reading.

Part II – A case study.

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1. Introduction.

*"Twas brillig, and the slithy toves
Did gyre and gimble in the wabe:
All mimsy were the borogroves,
And the mome raths outgrabe."*

Such is the first verse of the poem "Jabberwocky" in Lewis Carroll's *Through the looking-glass*. Read the first time, it makes no sense. Most of the words used do not even exist as such, they were made up. Read out loud, one can hear the sounds and feel the rhythm, and might get a hint of what the author intended the words to mean. The rest is left to the reader's imagination. It says what you want it to say.

Usually when we read, we know the words and do not make any effort to understand them - we might hardly notice what we are doing. But with a text like "Jabberwocky", or when we encounter words we do not immediately understand, we become extremely aware of our "secondary" reading strategies. Sometimes the context in which a word is placed can give us some information about its meaning, so reading the sentence once more might help. We might try sounding the words out, pronouncing them in different ways, or even try to spell them out loud to ourselves, to make sure we got the letters right. If everything else fails, we might ask someone what the word means, or look it up in a dictionary.

What this example makes clear is that reading is an extremely complex cognitive process. What makes us able to read words we know fluently, and approximate the sounds of words we have never encountered before? What cognitive processes are responsible for these abilities, how are they organised, and in which parts of the brain are they localised? What happens when the reading process breaks down? These are some of the questions in the cognitive neuropsychological study of reading. This paper is an attempt to answer some of them.

1.1. Sorting out the nomenclature.

The term *alexia* is derived from Greek, and literally means "without word" or "not word". In the clinic the word refers to an inability to read acquired after some sort of damage to the brain in previously literate individuals.

Some authors use the term *acquired dyslexia* synonymously with alexia, but "dyslexia", especially when it refers to one particular syndrome like "deep dyslexia" can easily be confused with *developmental dyslexia*, and I therefore prefer the term *alexia*.

The alexias have been distinguished along several dimensions both in neurology and neuropsychology. In the neurological literature one has commonly divided the alexias descriptively as *alexia with agraphia* and *alexia without agraphia*, or with reference to their anatomical substrate, hence calling them temporo-parietal and posterior alexias. While there are numerous subgroups of alexia with agraphia, alexia without agraphia is usually considered a single syndrome. A third type of alexia has also been observed, usually in the presence of Broca's aphasia, and is simply referred to as *the third alexia*, or frontal/anterior alexia.

In an early neuropsychological paper on alexia, Shallice & Warrington (1980) suggested a distinction between the *central* and the *peripheral* alexias (Shallice & Warrington, 1980). The peripheral alexias affect visual processing and analysis of the written word, while the central alexias are the result of deficits at later stages in the reading process. An overview of the different central and peripheral alexias can be seen in Table 1, below.

Table 1.

The alexias - cognitive neuropsychological terms.

Central alexias (Alexia with agraphia)	Peripheral alexias:
Phonological alexia	Neglect alexia
Surface alexia	Attentional alexia
Deep alexia	Pure alexia (alexia without agraphia) ¹

¹ Pure alexia is commonly referred to as letter-by-letter (LBL) reading in cognitive neuropsychological papers. Further clarification of these two concepts is found in the chapter on pure alexia.

1.2. Overview.

Understanding cognitive functions is not an easy matter. We cannot observe cognition directly, and must therefore rely on indirect methods for measuring and explaining it. In cognitive neuropsychology, theory building and empirical studies are closely knit together. The basic methods include studying patients who have suffered some form of damage to the brain, describing their cognitive deficits and trying to locate the injury, e.g. by brain scans. With this information, one tries to create a model of how the cognitive area in question might function normally. For example, Marshall & Newcombe (1973) created a model of word reading by analysing the different reading errors they had observed in their patients, and at the same time they constructed a taxonomy for categorising acquired reading problems.

This paper consists of two parts. Part I is a description of the theoretical basis on which the case study in Part II was conducted. At the same time the results of the case study in Part II have influenced the contents of Part I. The focus in the paper is mainly on the contributions of cognitive neuropsychology to the study of reading and acquired disorders of reading. Thus, data from alexic patients and suggested cognitive models will be at the core of the presentation, but some evidence from neurology and connectionist modelling will also be considered.

The main focus in Part I is on models of reading, and how they can account for the patterns of breakdown seen in alexic patients - How does the normal reading system work, and what happens when it is damaged ?

Part I includes a description of the dual-route model of reading and the empirical grounds on which it is founded, mainly the study of the central alexias. Two other models of reading are also considered in relation to the empirical evidence from studies of alexia, the "two-route lexical model" and a connectionist model of word processing.

Pure alexia is the only peripheral alexia discussed in the paper, and it will be given special consideration because of its relation to the case study in Part II. Anatomical considerations as well as the cognitive deficits believed to be responsible for pure alexia will be discussed in some detail, but an exhaustive review is not attempted.

While the number of papers on pure alexia and alexia with agraphia is enormous, the literature on the third alexia is less than sparse. There are no cognitive neuropsychological studies of this disorder on record, and the literature considered mainly comes from the field of neurology. A special case of the third alexia, also related to the case study in Part II, will be presented in some detail (Anderson et al., 1990).

In Part II a unique case of alexia is presented, which poses some interesting questions about the organisation of the reading process. The patient (JM) is impaired in reading and writing letters but not numbers, a pattern of performance only reported once before, in a patient with a lesion in premotor cortex (Anderson, Damasio & Damasio, 1991). The performance of JM is not easily explained within existing models of reading, and in attempting to explain his deficit in reading two main problems will be considered:

An interesting feature of JM's problems in reading and writing is that his oral spelling and naming to oral spelling is preserved, abilities that are usually disturbed in the third alexia, but generally spared in pure alexia. Since JM's writing is spatially distorted, we do not know whether his written spelling is intact. The question is: Can we make any inferences about JM's written spelling on the basis of his oral spelling abilities? In answering this question, I will review other cases in which oral spelling has been preserved and writing compromised, and the explanations considered in these cases.

The second question concerns the clear dissociation between numbers and letters observed in JM. How can cognitive theories account for this dissociation? In answering this question, evidence from cognitive neuropsychology, imaging studies and neural networks will be considered.

Reading is an acquired cognitive function, and might not be organised in the same way in different individuals. Whether or not such functions can be localised in the brain, and can be selectively damaged, are important questions, which will also be considered in Part II.

Part I.

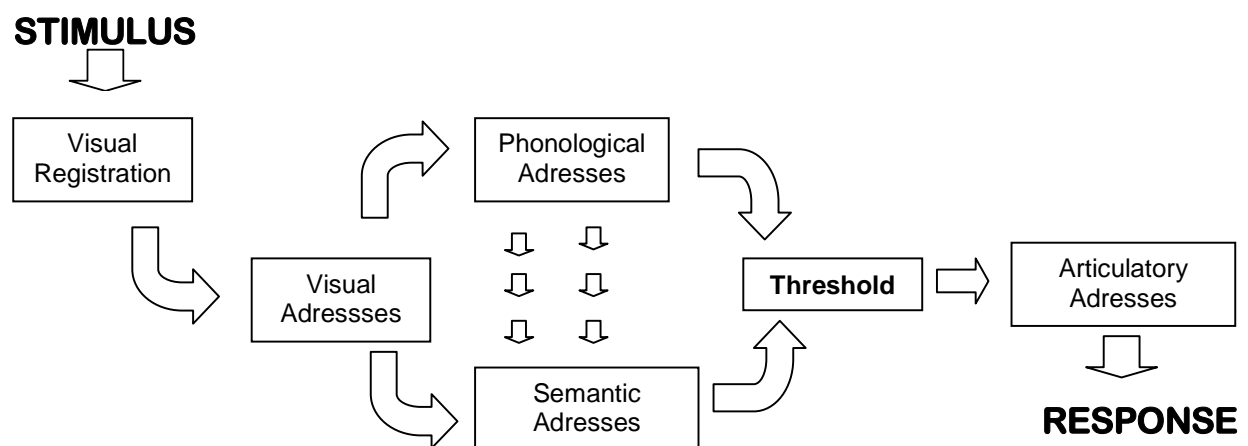
The alexias and models of reading.

2. Patterns of paralexia - the cognitive neuropsychological study of reading.

In their seminal paper, Marshall and Newcombe (1973) set the methodological standard for the new scientific approach - cognitive neuropsychology. They reviewed the literature on (parieto-occipital) alexia and presented six case studies of their own. They observed that: "It is comparatively rare for the dyslexic patient to be totally unable to read; far more common is the situation where the subject reads some words correctly, others not at all, and on yet others makes paralexia errors of various types." (Marshall & Newcombe, 1973; 176). Their idea was to analyse and classify these errors, and to interpret them within a model of the normal reading process. They believed that language skills are so tightly organised in the brain that only some patterns of breakdown will be possible. By analysing the patterns of breakdown, then, one should be able to say something about how the normal reading process is organised.

Marshall & Newcombe (1973) identified three different kinds of alexia by classifying the errors their patients made when reading single words out loud. On the basis of this analysis, they suggested a reading model consisting of two different routes from print to sound, thus the name *dual-route model*. (See Figure 1 below.)

Figure 1. The model of normal reading suggested by Marshall & Newcombe (1973).



One route transforms written words directly into sound, not accessing the meaning of the word, a process called *grapheme-phoneme conversion*, which makes reading of non-familiar words possible.

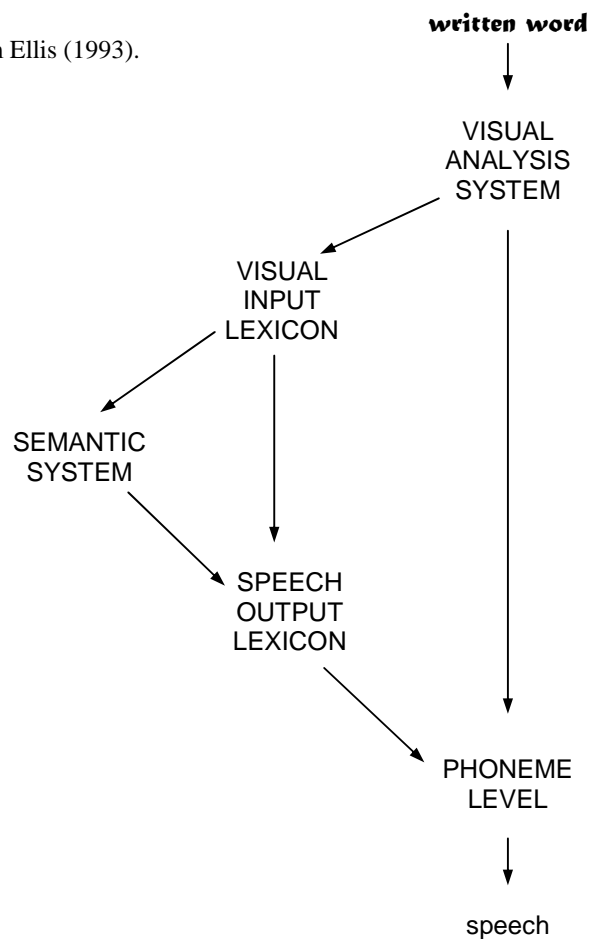
The other route activates a word's meaning, its semantic address, before the word is pronounced. Visual processing of the written word precedes both routes.

2.1. Dual-route model.

There are several versions of the dual-route model of reading (e.g. Coltheart, 1980, Ellis, 1993, Ellis & Young, 1996), but they all include the main features of Marshall & Newcombe's (1973) model. Ellis (1993) presented what he called a "hybrid" of dual-route models (Fig. 2), which I will use as a starting point in describing the normal reading process and the breakdowns believed to cause the different types of alexia.

Figure 2.

A dual-route model of reading. From Ellis (1993).



According to dual-route models, the process of single word reading starts in the *visual analysis system*, which identifies the letters of the word and their position in the string. The system determines which letter is seen, and converts it into what Coltheart (1987) calls "abstract letter identities", a general representation of a letter. These general representations are independent of the type, or font, with which the letters are written, that is, they will be the same for the letter-strings wind, *wind* and WIND.

These representations are then processed further, to the *visual input lexicon*², which contains representations of all familiar written words. The string is then matched to one of the stored representations, and the word is recognised.

Still, the word is not yet understood. Understanding, and connections to other associated words, is achieved when the information reaches the semantic system³. The semantic system contains associations or connections between different concepts, and their different representations (e.g. the taste and image of an apple will be connected), and is usually seen as a network through which activation spreads along the connections or associations between different concepts or "nodes" (Collins & Loftus, 1975).⁴

2.1.1. Reading out loud.

When one is reading silently, these are the only necessary stages of processing. But in the neuropsychological research on alexia, the most common object of study has been reading (single words) aloud, which means that we also need to describe the output - how word representations translate into spoken words. As indicated by the name, dual-route model proposes two different ways in which words can be translated into sound.

First, there is the *semantic route*, which includes all the stages described to this point. A representation of a word in the semantic system holds information about its meaning, but not about

² Coltheart (1987) refers to this as the *Orthographic Input Lexicon*, and it is also sometimes referred to as the *Visual Word Form System* (Warrington & Shallice (1980).

³ Also called the cognitive system (Coltheart, 1987)

⁴ There is some disagreement as to whether there is a unitary semantic system, or if it is built up by several modality- or category-specific subsystems. For our purposes, all we need to know is that a word is understood insofar as it activates an entry in the semantic system, and the issue of "multiple semantics" will therefor not be discussed.

its sound. The phonemes, or sounds, of familiar words are specified in the *speech output lexicon*⁵. This may be straight-forward enough when we are to pronounce short words, consisting of few phonemes, but with longer words, the order of the phonemes also need to be specified. Most models propose a "phonological short term store" for this purpose, a store where the phonemes of words, and maybe even short sentences are being held "waiting" to be articulated. Ellis (1993) calls this the phoneme level, while Coltheart (1987) simply calls it a response buffer.

But what with unfamiliar words, like the ones in the poem *Jabberwocky* ? How do we process words before they become familiar, and have representations in the visual input lexicon, the semantic system and the speech output lexicon ? The experimental studies of this have focused on how we read non-words out loud, and have specified a route connecting the visual analysis system with the phoneme level.⁶ The process involved is usually referred to as *grapheme-phoneme conversion*, indicating that the letters, or graphemes, of a word are directly translated into sound. This route is often referred to as the *sublexical route*.

2.2. Evidence for dual-route model.

Shallice & Warrington (1980) made a distinction between the *central* and the *peripheral* alexias, the central ones arising from problems within elements of the dual-route system, while the peripheral alexias are results of damage in or before the visual input lexicon. The main evidence for a dual-route model of word processing is derived from the central alexias, which also support the model as an explanatory framework. As mentioned in the introduction, the central alexias are surface alexia, phonological alexia and deep alexia.

2.2.1. Surface alexia

The most apparent symptom in surface alexia is that patients read known words as if they were unfamiliar, that is, they rely on the sublexical route. Because they read by translating graphemes directly into phonemes, they are more likely to read regular words correctly, and they often make so-

⁵ Might also be called *Phonological Output Lexicon* (Coltheart, 1987).

⁶ A non-word is a word invented by experimental psychologists, and is nothing more than a letter string having no meaning. Some authors make a distinction between non-words, which are implausible letter combinations, and pseudowords, which are easily translated into sound, and might look like a real word. I will use the terms interchangeably.

called regularisation errors. For example, Marshall & Newcombe's (1973) patient JC read *listen* as *liston*, and *insect* as *insist*, and he attached meaning to the word he pronounced, not the presented word. His reading was also influenced by word frequency, and he was slightly better at reading concrete than abstract words. The existence of surface alexia strengthens the basis for postulating a separate route for grapheme-phoneme conversion, independent of semantic access, so in this way it supports the dual-route model.

What is not clear, though, is where the functional deficit in surface alexia lies. A brief look at the model will reveal that disturbance at several different loci will result in a reliance on the sublexical route. As is probably the case in JC (Marshall & Newcombe, 1973), the deficit could be at the level of the visual input lexicon. JC had good comprehension of spoken words, and only slight word finding difficulties in normal speech, indicating that his semantic system and his speech output lexicon were intact.⁷ Warrington (1975) reported two patients who had greater difficulties reading exception words than regular words. These patients also had problems in other semantic tests (confrontation naming, naming to description, word-picture matching), indicating that true semantic damage was responsible for their reading difficulties. The disparity among surface alexics may be diagnostically inconvenient, but both patterns of breakdown presented can be explained within the dual-route model, and support the notion of a sublexical procedure in reading.

2.2.2. Phonological alexia

According to Ellis and Young (1996), *phonological alexia* was detected because the dual-route model predicts its existence. In the model (Fig. 2), the translation from letters to sounds can be disrupted if the link connecting the visual analysis system and the phoneme level is damaged. This would result in an inability to read unfamiliar words and non-words, which is the core symptom of phonological alexia.

Beauvois & Dérouesné (1979) described the first case of phonological alexia. Their patient (RG) was pretty good at reading nouns and content words, but very impaired in reading non-words out loud, a pattern that also was true for familiar (e.g. his birthdate) vs. unfamiliar number strings. RG could still write both spontaneously and to dictation, although he had a moderate degree of

⁷ This argument assumes a unitary semantic system.

dysorthographia. He was still able to write non-words to dictation and spell them out loud. Beauvois & Dérouesné (1979) showed that his alexia was not "pure", or occipital, because he was quite impaired in identifying the combinations of letters in words and translate them into sounds. On the contrary, when reading non-words, RG would often pronounce a *word* similar to the non-word. The alexia observed also differed from frontal alexia in that the patient was not aphasic, and from deep alexia, because RG did not produce any semantic errors.⁸ Beauvois & Dérouesné's (1979) case was important in two major ways. First it showed that phonological alexia was dissociated from the other alexias. Secondly, but more importantly, it showed that the sublexical route can be selectively damaged, and thereby established the functional independence of the two reading routes.

Apart from the impairment in reading pseudowords, phonological alexics can also be better at reading content words than function words, and their reading can be affected by concreteness and frequency. Friedman (1996) suggested that the alexic symptoms accompanying a selective impairment in reading non-words appear in predictable succession: "we can expect to see morphologic paralexias, (...) a relative deficit in reading functors compared with nouns, a relative deficit in reading verbs compared with nouns, and a relative deficit in reading abstract compared with concrete words - *in that order*." (p.120, Italics original.)

Surface alexia and phonological alexia together make up a *double dissociation*, which is neatly explained by dual-route model - surface alexics can only read via the sublexical route, while (some) phonological alexics can do everything but read by this route.

2.2.3. Reading without meaning - triple route model.

In Figure 2 there is also a third route from print to sound, connecting the visual input lexicon directly to the speech output lexicon. Schwartz et al. (1980) reported a patient (WLP) who suffered from a progressive breakdown in semantic memory, and noted that she was able to read words she could not understand out loud.⁹ Her lack of understanding was evidenced by her comments, reading the word *hyena*, she commented "*hyena....what in the heck is that ?*" (Schwartz et al., 1980; 261). What was surprising about her reading was that she could read exception words like *tortoise* and *leopard*,

⁸ Deep alexia, pure alexia and frontal alexia will all be presented later.

⁹ This might have been a case of what is now known as semantic dementia, but Schwartz et al. (1980) termed it "anomic dementia".

without being able to match the words to the corresponding pictures, or categorise them. She could also read visually similar words with diverging pronunciations correctly (e.g. *ear* and *bear*). In a dual-route model, like the one suggested by Marshall & Newcombe (1973) this pattern of performance is not possible. WLP could read exception words, which cannot be done using the sublexical route, and yet she clearly did not access any semantic information about the words she was reading. This led Schwartz et al. (1980) to propose a third route, linking the visual input lexicon directly to the speech output lexicon. Shallice & Warrington (1980) also argued for the existence of a third route, connecting the representations of familiar written words directly to their pronunciations, bypassing semantics. This "triple-route" model has now come to be widely accepted as a framework for interpreting reading disorders, but is still commonly referred to as the dual-route model, as the main distinction is still between lexical and sublexical processing.

2.3. Deep Alexia

The core manifestation of *deep alexia* is that patients with this syndrome make semantic errors in reading, they substitute synonymous words, e.g. read *city* as *town*. Marshall & Newcombe (1973) also noted three other kinds of error produced by their deep alexic patients: visual errors (e.g. *perform* read *perfume*), derivational errors (e.g. *defend* read *defence*) and a combination of derivational and semantic errors (e.g. *arrive* read *departure*).

Coltheart (1980a) reviewed 21 cases of deep alexia, and noted another kind of error often produced, namely function word substitutions (e.g. *him* read *me*). He also defined two other categories of errors seen in deep alexia, which are both a bit more problematic. First there are the "visual-then-semantic" errors, for which the famous example is *sympathy* read *orchestra* (Marshall & Newcombe, 1973) where one believes that the patient has misidentified the word (visually) as *symphony*, and then made a semantic error. The problem is of course that no one can know for sure if that is really the case.

Even more problematic, given that the classification of these errors is supposed to give insight into the reading process, and where it goes wrong, are the "visual and/or semantic" errors, an example of which is *shirt* read *skirt*. This could equally well be a visual and semantic error, and therefore ends up in this not quite satisfying error category. And even with this extensive set of error classifications or categories, one may still end up with what Coltheart (1980a) calls "unclassifiable errors". On top

of making all these errors in reading words, deep alexics are also unable to read non-words (Marshall & Newcombe, 1973; Coltheart, 1980a).

There is some variability among patients with deep alexia. Some may be able to read some words out loud, and the syntactic category and imageability of the word influence these remaining reading skills. Deep alexics are better at reading concrete, highly imaginable words, and they read content words better than function words (Coltheart, 1980a). Marshall & Newcombe's (1973) patients also had a tendency to produce concrete nouns as responses, regardless of the syntactic category of the stimulus.

Explaining deep alexia is clearly not as straightforward as the other cases of parietal alexia, and there are (at least) two different ways of explaining the syndrome. Some authors (e.g. Shallice & Warrington, 1980; Glosser & Friedman, 1990; Shallice & Plaut, 1993) believe that they observe the residual functions of a normal reading system. In a dual-route model then, both the grapheme-phoneme route, and the connections between the visual input lexicon and the speech output lexicon must be damaged. There also seems to be a semantic problem in deep alexia, which might reflect either problems in accessing the semantic system, or within the system itself. Some deep alexics (Marshall & Newcombe, 1973) make semantic errors in spontaneous speech, and some have problems in other semantic tests, e.g. in word-picture naming tests (Nolan & Caramazza, 1983), indicating true semantic damage.

Another explanation for deep alexia, put forward by Coltheart (1980b), suggests that the entire left-hemisphere reading system is destroyed in these patients, and that their residual word reading skills are the result of right-hemisphere functions. He partly builds this theory on studies of patients having had left hemispherectomies, and partly on studies of pure alexics. Coltheart (1980b) argues that the lesions causing deep alexia are so extensive, that it is unlikely that any reading skills should be left in the damaged hemisphere. Built on observations that the right hemisphere is not able to derive phonology from print, he still believes the actual spoken output to be produced in the left hemisphere.

When deep alexics are presented with a word for reading, it will be identified by a visual input lexicon in the right hemisphere which activates a representation in the semantic system on the right.

Information is then transmitted to the lexicon in the left hemisphere for pronunciation (Coltheart, 1980b).

2.4. An accident of history ?

Recently it has been suggested that deep and phonological alexia are really points on a continuum, and do not represent entirely different patterns of breakdown in the reading process (Glosser & Friedman, 1990; Friedman, 1996). As mentioned earlier, phonological alexia may be accompanied by other symptoms than an inability to read non-words, and according to Friedman (1996) these always appear in a predictable sequence. The only deep alexic symptom never observed in phonological alexia is (by definition) semantic errors, in which case the diagnosis changes to deep alexia.

Friedman (1996) reviewed recent literature on deep alexia and found five cases in which patients remitted to phonological alexia. Two of these cases are described in detail by Glosser & Friedman (1990). In all these cases the pattern of recovery is the exact opposite of that defining the severity of phonological alexia.¹⁰ While the semantic paralexias were always the first to remit and the other symptoms were reduced or remitted completely, the ability to read non-words was the last to be restored. None of the patients reviewed regained their pre-morbid skills for non-word reading. This led Friedman (1996; 127) to suggest that “the reason that deep and phonological alexia are so commonly treated as distinct entities may be an accident of history”. Since deep alexia was observed first, semantic paralexias became the defining feature of the syndrome, and since Beauvois & Dérouesné’s (1979) patient did not produce these errors, they had to come up with a new label for his alexia. The continuum of deep and phonological alexia is not easily explained within the dual-route model of reading, where semantic paralexias and the (in)ability to read pseudowords are dependent on entirely different routes. It is not consistent with the right hemisphere account of deep alexia either. If the remaining reading skills of deep alexics is the result of right hemisphere activation, but the reading pattern displayed by phonological alexics is dependent on remaining skills in the left hemisphere, one would not expect deep alexia to evolve into phonological alexia. Another finding challenging the right-hemisphere account of deep alexia is that a patient displaying

¹⁰ As mentioned in the chapter on phonological alexia, Friedman (1996) suggested that the order in which the errors occur is this: Impairment in non-word → morphologic paralexias → relative deficit in reading functors compared with nouns → a relative deficit in reading verbs compared with nouns → a relative deficit in reading abstract compared with concrete words.

many symptoms of deep alexia lost the ability to read after a second, *left-hemisphere* stroke (Roeltgen, 1987).

2.5. Dual-route model - reconsidered.

The dual-route model of reading contains two functionally independent processes for transforming print into sound: The sublexical route translates graphemes into phonemes directly, while lexical and semantic knowledge is accessed via the lexical route. Within this framework, non-word reading does not depend on, and is not influenced by lexical variables, but operates according to abstract rules for spelling to sound correspondence (Coltheart, 1980c). The notion of a sublexical procedure in reading has been at the core of the criticism against dual-route models (e.g. Humphreys & Evett, 1985; Glosser & Friedman, 1990) and attempts have been made at constructing models including only the two lexical routes.¹¹

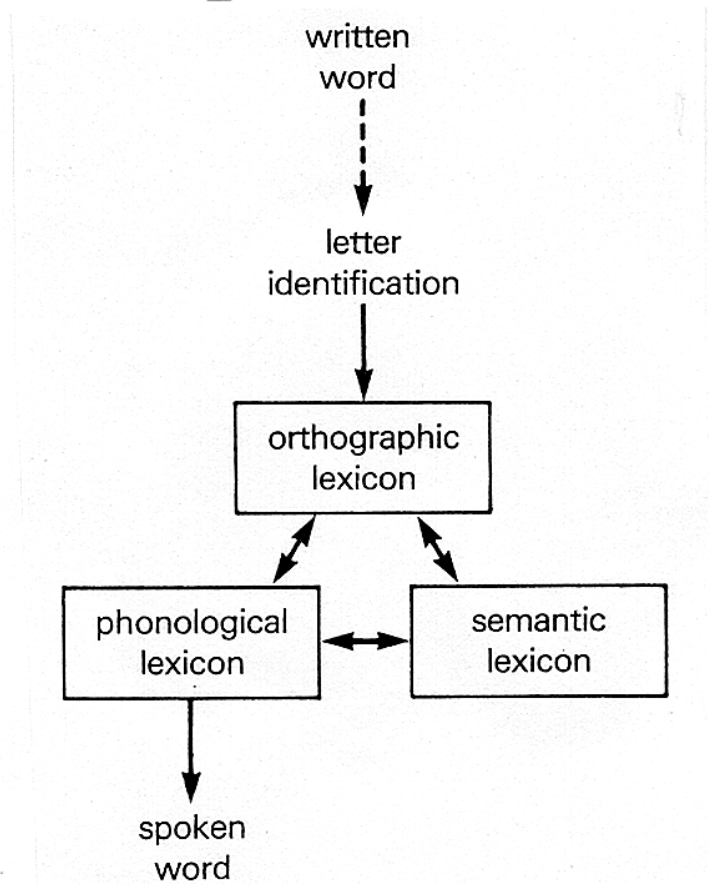
A lexical model was presented by Glosser & Friedman (1990) to account for the continuum of deep- and phonological alexia (see Fig. 3). In this model, all letter strings can be pronounced via the same mechanism, through connections between the orthographic lexicon (which corresponds to the visual input lexicon) and the phonological lexicon (i.e. the speech output lexicon).

When a letter string is perceived, visually similar strings will be activated in the orthographic lexicon, which in turn activates phonological representations. If the string is a known word, the matching string will have the highest level of activation, and will activate the word's pronunciation. This process may operate directly, or via the semantic system. If the string is an unfamiliar combination of letters, words with similar letter combinations will be activated both in the orthographic and in the phonological lexicon. A pronunciation will be derived from all these partially activated words.

Figure 3.

The two-route lexical model of reading

(Glosser & Friedman, 1990).



In the lexical model, the impairment in non-word reading observed in deep- and phonological alexia can be conceptualised as a disturbance of the route connecting the orthographic lexicon to the phonological lexicon. If words similar to the presented non-words are not sufficiently activated in the phonological lexicon, non-words cannot be pronounced. Glosser & Friedman (1990) suggested that this is the mechanism responsible for impairments in non-word reading, and that a mild deficit in accessing the phonological lexicon would be sufficient to produce this. A more severe disturbance in the same route could result in a deficit in reading words with no independent semantic representations, like function words and syntactic morphemes (affixes), since these cannot be read via the semantic route. In this way an inability to read function words will always be accompanied by a deficit in non-word reading, which, according to Glosser & Friedman (1990, Friedman, 1996),

¹¹ This model is referred to as the "Two-route lexical model of reading", which is easily confused with the name "dual-route", and I will therefore refer to it simply as "the lexical model".

is also the case. To account for the semantic errors made by deep alexics, and the effect of concreteness and imageability on their reading performance, Glosser & Friedman (1990) suggest a second, independent deficit in semantic processing. A semantic deficit in itself cannot account for the production of semantic paralexias, since other patients with semantic disturbances (e.g. aphasic patients) do not produce such errors, but when a disturbance in the semantic system (or in accessing it) occurs at the same time as a deficit in phonological processing, semantic paralexias might occur (Glosser & Friedman, 1990).

So: the continuum of deep and phonological alexia might be accounted for in a purely lexical model of word reading. A problem with this model, though, which is also valid for the dual-route model, is that "partial" damage to one or more routes needs to be postulated to account for the different alexias.

2.6. Connectionist models.

The dual-route model of reading, as well as the lexical model, rely on box-and-arrow schemas, indicating independent functions and processes, to conceptualise the normal reading process. A completely different way of viewing cognitive processing comes from connectionist modelling, where knowledge is seen as *distributed* between nodes or units, and *patterns* of activation in the model make up the representations. Usually, such models are implemented in neural networks, and simulations of the process in question are run. One of the clear advantages of creating these networks, is that all computational assumptions and predictions must be made explicit to make the model work in practice. The dual-route model, on the other hand, is not so explicit regarding which computational processes are involved.

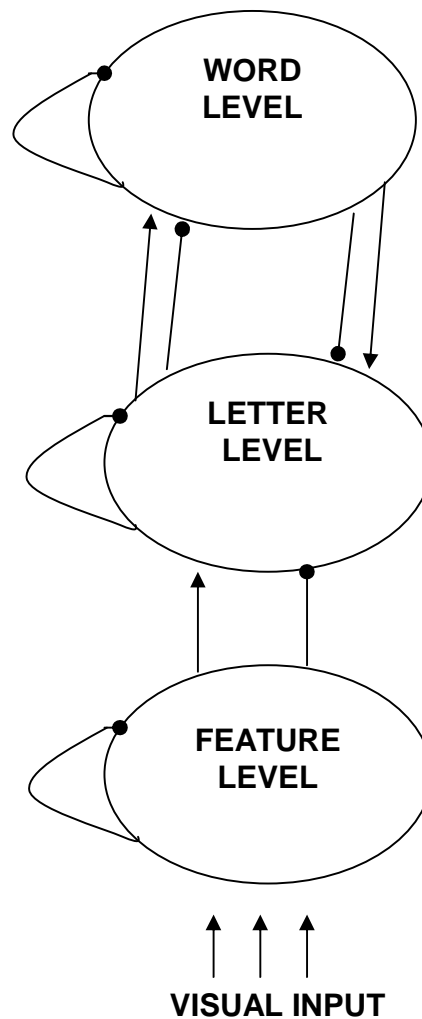
Shallice & Plaut (1993) created a connectionist network that mapped orthography to sound via semantics. By lesioning this network, they were able to simulate a wide range of symptoms of deep alexia, and their findings suggested that the remaining reading skills in deep alexia are not products of the right hemisphere, but the output of a degraded normal reading system. Their most important point, though, might be that: "The approach can be viewed as an elaboration of, rather than an alternative to, more traditional box and arrow theorising within cognitive neuropsychology." (p.383).

2.6.1. A model of word recognition and reading.

McClelland & Rumelhart (1981) created a connectionist model of the reading process, the *interactive activation model (IAM)*, specifying three levels in word perception, the feature level, the letter level and the word level. In this model perceptual processing is seen as an interactive parallel process, where bottom-up (data-driven) and top-down (conceptually driven) processing between the different levels take place at the same time. In the model several letters can also be identified simultaneously, that is, in parallel. (The model can be seen in figure 4.) According to the IAM, when a word is perceived, the different features of the letters are first detected (e.g. the bottom horizontal line in the letter *E*), at the feature level. The nodes representing letters with such a feature are then activated on the letter level (e.g. *E* and *L*), and words containing these letters will be activated on the word level.

Figure 4.

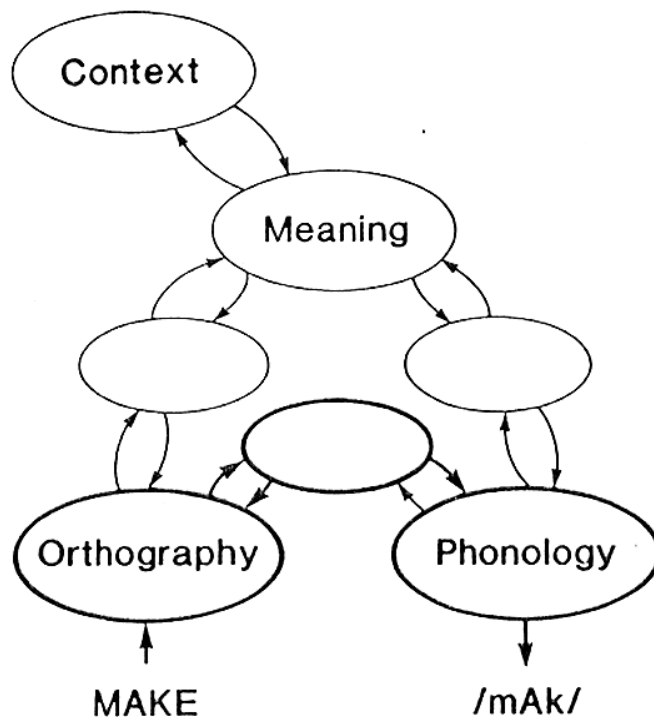
The IAM model of words recognition (McClelland & Rumelhart, 1981), with excitatory and inhibitory connections between levels.



Nodes representing words and letters without this feature will be inhibited. The activation on the letter level is passed on before all the letters are identified, in this case activating or "warming up" all words with an *E* in them. The letter level also encodes letter position, so that all words with *E* in the first position are activated, and all others inhibited. In the model several letters can also be identified simultaneously, that is, in parallel.. As indicated by the name, the activation in this model is interactive, and information is processed both ways: the word level feeds back to the letter level, which feeds back to the feature level inhibiting nodes not fitting the pattern (e.g. nodes for letters never seen in words starting with *E*).

The IAM can recognise words, but it cannot understand them or pronounce them. Seidenberg & McClelland (1989) expanded the model to include semantics and phonology, and implemented the link between orthography and phonology in a computer simulation. As can be seen in figure 5, the model looks a bit like the lexical model suggested by Glosser & Friedman (1990).

Figure 5. Seidenberg & McClelland's (1989) lexical model of reading. The **bold** lines indicate the processes modelled in their computer simulation.



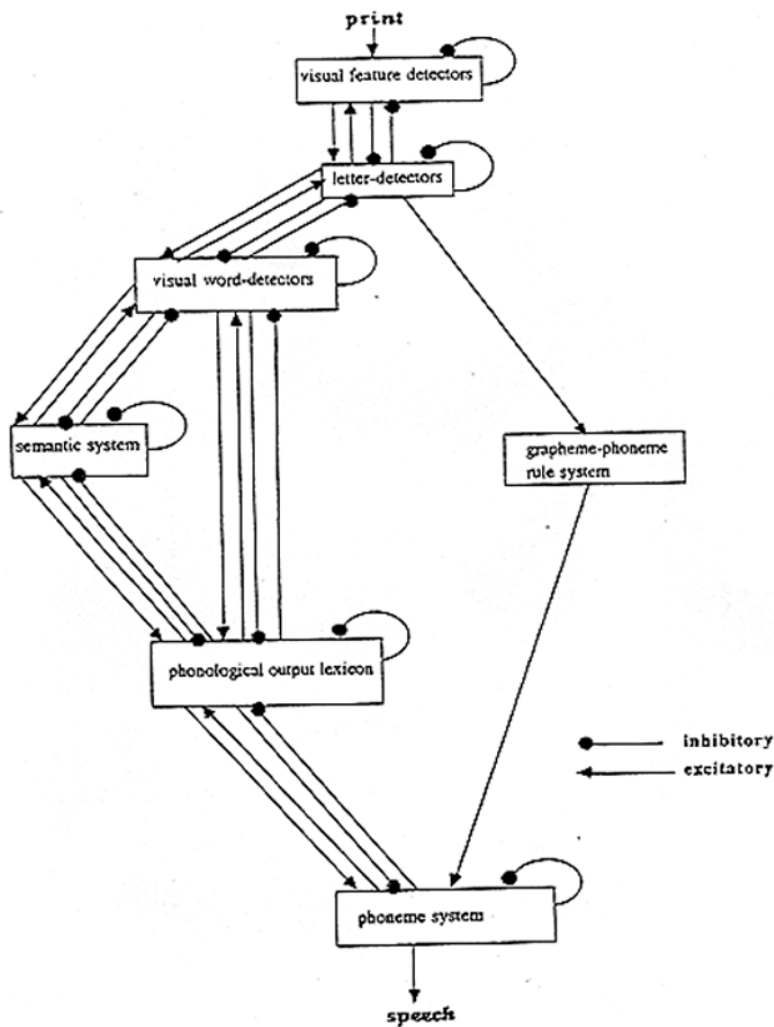
What Seidenberg & McClelland (1989) tried to prove, was that, only the lexical route from print to sound is necessary to account for the different patterns of alexia, and that their lexical model would be able to pronounce regular words, irregular words and non-words, relying on the same computational processes.

They trained the network on 2897 words, and then tested its reading performance on different classes of words. The network performed well on reading words, both regular and irregular, while its performance on non-words was at 50-65% correct. This led Seidenberg and McClelland (1989; 564) to conclude that their model "offers an alternative that dispenses with this two-route view in favour of a single system that also seems to do a better job of accounting for the behavioural data." This is not quite the case, though. On the non-word lists that the network was tested, normal readers usually get about 90% correct, while the network only scored 55% average, which is lower than most surface alexics (Besner et al., 1990, cited in Coltheart et al., 1993). This makes the model unable to account for surface and phonological alexia, since non-word reading is an important parameter in diagnosing both syndromes. In other words, the model does not account for the behavioural data of neither normal or impaired readers concerning non-word-reading. In the model, non-word reading is performed by analogy to real words, and since the amount of words the network was trained on was very limited, this might account for its poor non-word reading (Seidenberg & McClelland, 1990).

2.7. Dual-route - cascaded.

One of the assumptions underlying IAM, is that it is cascaded and interactive, and Coltheart et al. (1993) incorporated these computational principles in the dual-route model, creating the *Dual-Route Cascaded* (DRC) model of reading (fig 6). All the main features of dual-route model are maintained, while the principles of the IAM are responsible for letter- and word- recognition. The main alteration from the classical dual-route model, is that the activation between levels of lexical processing is interactive and cascaded.

Figure 6. The dual-route cascaded model suggested by Coltheart et al. (1993). Note that all the main features of the standard model (fig. 2) are maintained. The important new aspect in the model is the interactive activation (excitatory and inhibitory) within and between levels.



Coltheart et al. (1993) also implemented this model in a neural network and trained it on the words used by Seidenberg & McClelland (1989). The network was then tested on the same lists of non-words on which the Seidenberg/McClelland network scored only 50-65% correct. The DRC read 98% of these non-words correctly, indicating that the failure of the Seidenberg/McClelland network to read these words was not due to the size of the training corpus as Seidenberg & McClelland (1990) had suggested. Coltheart et al. (1993) concluded that separate lexical and sub-lexical routes are important features in the normal reading system and must therefore be included in models of reading.

3. Pure Alexia.

Dejerine (1892, translated by Rosenfield, 1988) was the first to describe alexia without agraphia. His patient, Monsieur C, presented with a complete inability to read words and musical notation, and he could hardly recognise any letters. In trying to identify letters, Monsieur C would copy their shape into the palm of his hand, and thereby "tactilely" identify them, and with some difficulties he could copy the letters - line by line - onto paper. Dejerine noted that his patient was better at identifying numbers than letters, and could perform fairly complex arithmetic operations, like adding two 8-digit numbers¹² (Bub et al., 1993). Landolt, the ophthalmologist whom Monsieur C first contacted, observed that he did not read multidigit numbers as wholes, but spelled them out ("1-1-2 makes 112").

Monsieur C could still write, and although his handwriting might have been a bit clumsier than it was pre-morbidly, he could write whole paragraphs to dictation or from memory. A few minutes later he could not read what he had written. Monsieur C had a visual field defect on the right side, although it was not a complete hemianopia. Dejerine (1892, in Rosenfield, 1988) noted that: "He was, in reality, more hemiachromatopsic¹³ than literally hemianopsic." Landolt also noted that: "The (right) hemianopia is not absolute, (...) the objects appear more obscure and less clear than in the other half; furthermore we must add that the extreme limits of the visual fields are normal." (Landolt, cited in Damasio & Damasio, 1986; 162).¹⁴

Four years after his first injury Monsieur C lost the ability to write, and could no longer speak, but he could still understand spoken language, and make himself understood by gestures and mimicking.

3.1. The anatomy of pure alexia.

Dejerine (1892, in Rosenfield, 1988) found two different lesions in Monsieur C's brain at autopsy (see fig. 7). The more recent lesion had destroyed the angular gyrus, which Dejerine believed contained "the visual centre for words", and this could explain Monsieur C's loss of the ability to write ten days before his death. The older lesion involved the mesial occipital cortex and the

¹² Note that to perform this operation, Monsieur C did not have to identify more than one digit at the time.

¹³ Hemiachromatopsia: Loss of colour vision in one half of the visual field.

splenium of the corpus callosum. Dejerine believed that this lesion prevented visual information about words and letters from reaching the visual centre for words in the angular gyrus, thereby leaving the patient unable to read, but since this centre itself was intact, the patient could still write. Although often cited differently, Dejerine did not believe that the lesion of the splenium was of any importance in Monsieur C's alexia, but that the lesion in the white matter of the left occipital lobe in itself was enough to prevent visual information from reaching the angular gyrus (Damasio & Damasio, 1983).

Figure 7.

(From Bub et al., 1993) The old lesion, marked X, acted to disconnect the right and left occipital lobes from the visual centre for words (Pc), which Dejerine suggested was causing pure alexia. The area Pc was destroyed by Monsieur C's second insult, leaving him unable to write as well as read.



In 1965, Norman Geschwind reintroduced Dejerine's original observations in his overview of what he termed "disconnexion syndromes" and speculated that the visual centre for words in the angular gyrus is a "region which turns written language into spoken language, and vice versa." He suggested that this centre also stored the memory of "the rules of translation" between script and sound. Geschwind, as opposed to Dejerine, believed that the splenial lesion was important in causing pure

¹⁴ This fact has been more or less ignored by recent authors referring to Dejerine, causing quite a bit of confusion.

alexia, that this lesion acted "to disconnect the right visual region from the angular gyrus". Because of the hemianopia, the visual areas on the left will not receive any visual information, and the angular gyrus is left without any input. Because the visual word centre itself is intact, patients can still write.

There are some problems with Geschwind's (1965) interpretation of Dejerine's case though, which also has consequences for his theory of pure alexia. First of all, Geschwind failed to notice Dejerine's note that Monsieur C did in fact not have a total hemianopia preventing visual information from reaching the left hemisphere. Secondly, Geschwind overlooked the fact that Monsieur C's number reading was not at all normal, he wrote: "(...) in Dejerine's case number reading was perfect." (Geschwind, 1965; 281). Still, Geschwind's paper was important, in that he reintroduced Dejerine's observations and captured the attention of neurologists and neuropsychologists, who had ignored the syndrome of pure alexia for a long time.

Greenblatt (1973) presented a case of pure alexia without hemianopia or colour anomia. The patient had a tumour affecting the inferomedial white matter in the left occipital lobe and the splenium of the corpus callosum. Intuitively one should think that this would pose a problem for the disconnection theory (Geschwind, 1965), where the hemianopia is believed to prevent visual information from reaching the left angular gyrus. But Greenblatt (1973) believed that the lesions in this case disconnected the left angular gyrus from "its bilateral visual input", and was therefore an example of a disconnection functionally similar to the one described by Geschwind. It is worth noting that this patient's reading of numbers and her written arithmetic were reported to be intact.¹⁵

Damasio & Damasio (1983) reviewed four major case studies on pure alexia, and presented 16 patients of their own, with the intention of clarifying the lesions in the different patients described. All the patients had some form of a visual field defect, either a complete right hemianopia or an upper right quadrantanopia with hemiachromatopia in the lower right quadrant. The behavioural findings included colour anomia in seven patients, two had verbal memory defects, six displayed optic ataxia, and two had prosopagnosia. Thirteen of the patients had damage involving the mesial

¹⁵ This patient was not tested very thoroughly, she died six days after being admitted to hospital. It is also worth noting that the pt. could not co-operate in a visual field testing, and that: "at times she seemed to cock her head slightly to the right, as if to place her visual target in her left field." (Greenblatt, 1973, p.308)

occipital region, while seven had occipito-temporal lesions. The lateral occipital region was involved in five patients. Surprisingly, lesions in the splenium itself were found in only two patients, but eleven had damage to the forceps major, and thirteen in the white matter of the paraventricular region.

Damasio & Damasio (1983) concluded that lesions associated with pure alexia include three major types, with different accompanying symptoms. (See table. 1)

Table 1. Lesion sites and visual field defects associated with pure alexia, and accompanying cognitive deficits. Based on Damasio & Damasio (1983).

	Type 1	Type 2	Type 3a	3b
Lesion site	-Mesial occipital cortex -White matter of the occipito-temporal junction. (Including paraventricular area.) -Left half of splenium and forceps major.	-Optic radiations or calcarine region - Paraventricular matter (damaging interhemispheric pathways within left hemisphere).	-Paraventricular white matter -Inferior visual association cortex - Inferior part of optic radiation	-Occipito-temporal, involving paraventricular white matter. - May involve mesial and inferior visual association cortex. - Splenium may be affected.
Visual field	Right hemianopia	Right hemianopia	Upper right quadrantanopia. Achromatopia in lower quadrant.	Full visual fields, with or without hemiacromatopia.
Associated cognitive impairments	-Colour anomia -May have verbal amnesia	No colour anomia or verbal amnesia.	No colour anomia or verbal amnesia.	No colour anomia.

Damasio & Damasio (1983) agree with Dejerine that a lesion of the splenium proper is not necessary in causing pure alexia, but stress the fact that the white matter connecting the visual areas of the two hemispheres must be damaged.

3.2. The cognitive neuropsychology of pure alexia.

The anatomical substrate being reasonably clear, one should think that there would be a fair degree of consensus about what cognitive impairments cause pure alexia. But, the area of research being rediscovered not that long ago, major controversies are still not resolved. This may in part be because - while neurologists have focused on the more general aspects of the syndrome, and "explaining" it in terms of its anatomy - neuropsychologists have been trying to pinpoint the cognitive impairments causing pure alexia, and how these can be explained in a (theoretical) model of reading. Another reason for this controversy is that both the lesions involved and the symptoms

observed differ between patients. This has led some authors (e.g. Price & Humphreys, 1992; Chialant & Caramazza, 1998) to suggest that different functional deficits may lead patients to adopt a letter-by-letter strategy in reading. Others believe that there are different types of pure alexia, resulting from different or co-occurring lesions (Patterson & Kay, 1982; Hanley & Kay, 1996). Like Arguin, Bub & Bowers (1998) and Behrmann Plaut & Nelson (1998) I believe that *one* explanation for the similarities and the differences between the studied cases of pure alexia can be found, and that will be the starting point in the following analysis.

The core symptom in pure alexia, apart from preserved ability to write, is *letter-by-letter reading* (LBL). The patient literally spells out the words before naming them. Sometimes patients spell the word out loud, but LBL-reading can also be inferred from a linear relationship between the number of letters in a word and the time taken to read it. Some authors use the term LBL-reading synonymously with pure alexia, but as Price and Humphreys (1992, 1996) point out, it is important to keep the two apart. Pure alexia is the name of the syndrome, LBL-reading a symptom or a compensatory strategy adopted.¹⁶

LBL-reading has been observed in surface alexia (Friedman & Hadley, 1992), with surface agraphia, (Rapp & Caramazza, 1991), and deep alexic phenomena have been observed in one LBL-reader (Buxbaum & Coslett, 1996). In other words, LBL can be associated with different functional deficits. Some authors (e.g. Kay & Hanley, 1991; Bowers et al., 1996, 1998) have used letter-by-letter reading as the sole criterion for selecting cases and have included patients with agraphia or other cognitive deficits. This makes a distinction between the symptom or strategy of reading letter-by-letter, and the syndrome to which it is attached, even more important. Some of the confusion could perhaps have been avoided if the use of concepts and the recruiting of patients were a bit more stringent. If both the absence of agraphia and other cognitive deficits *and* word-length effect in reading were used as criteria for selecting patients for studies of pure alexia, it might help to clarify matters.

Behrmann & Shallice (1995) defined what they called *prototypical pure alexia*, and using the pureness of the deficit might help clearing up some of the chaos. I will therefore try to consider only

¹⁶ I will use the terms pure alexia and alexia without agraphia interchangeably

the cases of really pure alexia, that is, without agraphia or other major disturbances in language or visual perception. Patients with verbal amnesia and/or colour anomia will be defined as pure alexics, based on Damasio & Damasio's (1983) classification.

The different theories of pure alexia can roughly be divided into two groups, those favouring a *central* explanation, and those presenting more *peripheral* views, the peripheral ones being the most favoured recently. The traditional disconnection theory is an example of a central explanation, claiming that the problem arises after visual processing. Within the peripheral accounts, there is some disparity. Some believe that the deficit is specific to orthographic material (Reuter-Lorenz & Brunn, 1990; Kay & Hanley, 1991; Arguin & Bub, 1993), while others (Friedman & Alexander, 1984; Farah, 1990, 1994; Behrmann, Nelson & Sekuler, 1998) argue that a more general visuo-perceptual deficit causes patients to read letter by letter.¹⁷

3.2.1. A historical overview.

In modern neuropsychology, the first cases of LBL-reading were described by Kinsbourne and Warrington in 1962. They observed what they called "spelling dyslexia" in four patients who were not impaired in identifying singly presented letters or numbers. These patients had difficulties perceiving simultaneously presented objects or pictures, as well as letters, and Kinsbourne and Warrington (1962) interpreted their reading problem as resulting from their simultanagnosia.

As mentioned in the chapter on localisation, Geschwind (1965) reintroduced Dejerine's original contribution, and reminded the world of neurologists and neuropsychologists that Monsieur C did not have any impairments in object perception and naming, even with complex objects. Instead he argued that pure alexia was in fact pure, and agreed with Dejerine that the angular gyrus contains a visual memory centre for words, which could explain Monsieur C's ability to identify letters tactilely as well as his preserved writing skills. Geschwind (1965) theorised that this "visual word memory" contained visual-auditory associations, which could explain why patients with pure alexia retain the ability to spell and to name words to oral spelling, while patients with alexia with agraphia do not.

¹⁷ I am purposely using the term LBL reading here, since some of these authors do not believe that pure alexia is pure, and include patients with other visuo-perceptual deficits in their studies.

Even though visual information cannot reach the angular gyrus for identification, the "word images" are still accessible for oral output, allowing words to be spelled out loud. The output channels are also preserved, enabling pure alexics to spell words orally and in writing.

In 1978 Levine & Calvanio, referring back to Kinsbourne & Warrington (1962) presented three patients with alexia and simultanagnosia, of which one had agraphia. Their patients did not have problems in single letter identification tasks, or in identifying one of three simultaneously presented letters, when they knew where in the string the target letter would be.¹⁸ Inspired by Warrington & Rabin's (1971) finding, that patients with left, posterior lesions were better at reporting letters from letter-strings approximating words, than from nonsense strings, Levine & Calvanio (1978) tested their patients' ability to report letters from words and non-words. They found that more letters embedded in words than in random letter strings were reported by their patients, but only when they knew in advance that a real word would be displayed. In a task where the subjects were told that they would only see non-words, one patient did not show this "word superiority effect" on letter reporting.

Levine & Calvanio (1978) also noted that none of their patients were impaired in face recognition or identification, which to them indicated that "(...) tasks requiring ID of a single, though complex, visual pattern appear to have a different neuropsychological basis from tasks requiring the ID of a *compound* array consisting of multiple identifiable components, where misidentification of any component will usually impair identification of the array." (p.78, (Italics original)). They concluded that verbal alexia - simultanagnosia is a disturbance in the perceptual analysis of compound arrays of stimuli.

Warrington & Shallice (1980) challenged this view of pure alexia. They presented two patients whose main symptom was pure alexia (in their terms, spelling dyslexia), but who did not have problems in perception and identification of complex pictures. They concluded that pure alexia was dissociated from simultanagnosia, and that lower level perceptual problems were not responsible for their patients' inability to read. Warrington & Shallice (1980) suggested that the cause of pure alexia lies at the level of the visual word form system, which they defined as the system "which parses

¹⁸ One of their patients, RT, was impaired in letter naming, and was allowed to draw a letter as a response in all tests.

(multiply and in parallel) letter strings into ordered familiar units and categorises these units visually." (p.109).

Warrington & Shallice (1980) also tried to encourage whole word reading in their patient, by presenting them with script and by presenting words tachistoscopically, and showed that their patients were unable to read in this manner. One patient was also asked to match the words he failed to read to one of four pictures, but did not perform above chance. This patient was also tested on sets of concrete and abstract high frequency words for reading, but no significant difference between sets was found.

To Warrington & Shallice (1980) this indicated that this patients was not able to read via the right-hemisphere, a mechanism suggested to account for the preserved reading in deep alexia, and they found it unlikely that the right hemisphere has much capacity for comprehending written words.

Patterson & Kay (1982) reviewed the neurological and neuropsychological observations and explanations of pure alexia, and stressed the need for cognitive explanations of the syndrome. Following the methods of Marshall & Newcombe (1973), they studied four patients who read letter-by-letter, evaluated their abilities to comprehend written words and analysed their reading errors. They observed three types of errors in their patients: letter misidentification (men; h-e-n; hen), misnaming a word when letter identification was correct (head; h-e-a-d; heed), and "other" errors. Patterson & Kay (1982) concluded that there are two distinct varieties of letter-by-letter reading. Two of their patients made many errors because they misnamed or misidentified letters, and because they would often read the first few letters of a word, and then guess. If these patients got the letters right, they pronounced the word correctly, and Patterson & Kay (1982) referred to them as "classical" or "pure" letter-by-letter readers. The other two patients would often misname a word, even though they spelled the letters out right, and they also made regularisation errors, which is a defining feature of surfaces alexia.

Patterson & Kay (1982) also addressed the issue of whether or not pure alexias can access information about words they cannot read, that is, if the letter-by-letter strategy is only adopted in order to name words out loud, not to understand them. At the same time they wanted to test the hypothesis of reading in the right hemisphere, suggested as the basis for the remaining reading skills in deep alexics (Coltheart, 1980b). In their experiments, Patterson & Kay (1982) failed to find any evidence of preserved comprehension of words not read letter-by-letter by their patients.

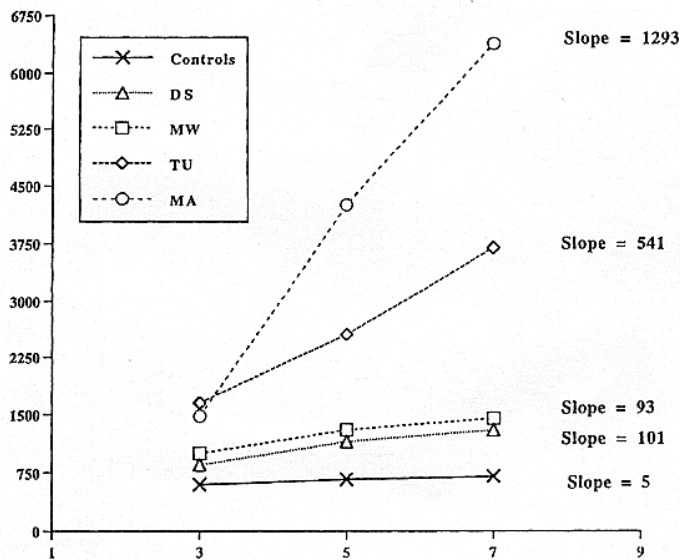
Friedman & Alexander (1984) challenged the traditional disconnection view of pure alexia as it was formulated by Geschwind (1965), namely that the left occipital lesion prevents visual stimuli to enter the left hemisphere, and the lesion in the splenium prevents visual information from the right hemisphere from reaching the language centres. They questioned this theory partly on logical-deductive grounds: "If all visual stimuli are prevented from reaching the language centres, then why is only reading impaired?" (Friedman & Alexander, 1984;10). They presented a patient with pure alexia, and showed that his performance in naming tests appeared normal in traditional paper and pencil tests, but that his naming latency increased with object complexity when pictures were presented tachistoscopically. They concluded that their patient's reading problem was a behavioural manifestation of a general problem in the automatic identification of visual stimuli, not specific to orthographic material.

3.3. Key questions in pure alexia.

As mentioned earlier, the defining symptoms of pure alexia is the absence of agraphia and the so called "word length effect": The time taken to read a word increases with the number of letters, because the patients have to spell out (or silently read) each letter in the word. Still, there is considerable variation in reading speed among patients with pure alexia.

Figure 8.

From Sekuler & Behrmann (1996). Reaction times for 4 LBL-readers and controls, illustrating the linear relationship between string length and reaction times (in msec). The figure also clearly illustrates the variability in reading speed between patients.



Some use more than one second per letter, some apparently read normally, but with computerised testing it can be revealed that their naming latency increases with word length, and there are patients like Monsieur C who cannot even identify single letters. (See figure 8.)

The major question, then, is: how can we explain this variability, and even more important, what causes the word length effect in the first place ?

3.3.1. Object perception in pure alexia.

A central issue in the study of pure alexia is the question of whether it is really the most striking feature of a more basic impairment in object perception and recognition, or if it is indeed a pure deficit affecting the processing of alphanumeric material, or even alphabetic characters only.

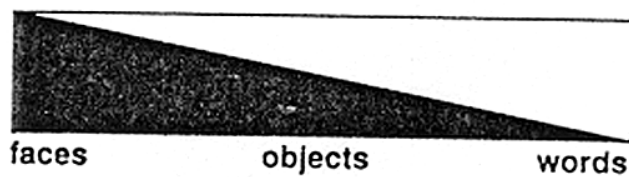
Farah, (1990, 1994) created a model within which alexia, object agnosia and prosopagnosia are seen in a continuum. As mentioned earlier, Levine & Calvanio (1978) observed that their patients were not impaired in recognition or identification of faces, and suggested that different perceptual mechanisms were responsible for reading and face identification. Farah's (1990,1994) model is built on similar observations. She suggested that in perception we make use of two types of structural descriptions¹⁹, which are of variable importance for identifying faces, objects and words (See fig.9

¹⁹ Farah (1990; 127) explains the term "structural description" as "referring to visual representations of shape that are composed of parts and the spatial relations between the parts."

below). One type involves decomposing the stimuli into many smaller and simpler parts, while the second type is relying on the description of few, but complex parts of an item.

Figure 9.

From Farah (1994). A graphical representation of the roles of the two suggested structural description systems in face- object- and word perception.



Face perception will mostly rely on the structural description of few, but complex parts - faces are perceived in a "holistic" manner. Word perception and reading, on the other hand, are dependent on decomposing the word into its constituent letters, and maybe even letter parts. Object perception lies between the two, and is partly dependent on both types of structural description. In essence, this model of perception implies that agnosia for objects will occur with either prosopagnosia or pure alexia. The latter two will never occur together, unless the patient also has agnosia for objects. The rationale for suggesting such shared mechanisms for face-, object- and word perception, is that it leaves it unnecessary to infer separate neural mechanisms for reading, which is a phylogenetically young ability, and, according to Farah (1990, 1994) is therefore less likely to rely on a separate "new" system.²⁰

When Farah (1990) suggested this model, cases to oppose the idea had not yet been observed, but Ruminati et al.(1994; Ruminati & Humphreys, 1997) reported a patient with agnosia for objects without alexia or prosopagnosia which cannot be explained within Farah's model. This patient had problems in accessing semantics from vision, making visual naming errors, and was impaired in object decision, which according to Ruminati and Humphreys (1994) is an indication of problems in creating a structural description. The patient did not have problems in reading or in naming or

²⁰ This issue will be more thoroughly discussed in Part II.

remembering faces. Defending her model, Farah (1997a; 1997b) argued that problems in accessing semantics from vision is not a perceptual deficit, that the patient described might even have problems within the semantic system, and that her model would still be valid for patients with actual perceptual problems. More threatening to Farah's model is a case recently presented by De Renzi & Di Pellegrino (1998). They reported a patient (Anna) with prosopagnosia and alexia, with intact recognition of object drawings. Anna was able to identify overlapping figures and performed well in Street completion test, both considered perceptually demanding tests. A similar case has also been reported by Buxbaum, Glosser & Coslett (1999). Their patient (WB) was unable to recognise faces and words, but performed relatively well on object recognition tests. This pattern of deficits is not possible within Farah's model, at least not without some (major) modifications to the framework of interpretation.²¹

So let us accept for now that word reading can be selectively damaged, object perception and naming being intact, and turn to the other main findings in the study of pure alexia.

In an extensive literature review on the subject, Behrmann, Plaut & Nelson (1998) pinpoint two major empirical findings that needs to be explained by a theory of pure alexia, and which have posed problems for several theories: "One finding is that these patients [LBL-readers] 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 by their above chance performance on lexical decision and semantic categorisation tasks." (p. 8).

3.3.2. Impaired letter processing

Behrmann & Shallice (1995) suggested that impaired letter processing should constitute the default explanation for pure alexia, since normal letter identification has not been proven in any LBL-readers²². In an extensive review of LBL-cases Behrmann, Plaut & Nelson (1998) found evidence for impaired letter processing in 50 of the 57 cases reviewed, and no convincing contra-evidence in the remaining cases. Still the question of what causes this deficit remains to be answered. Even though the core deficit does not seem to affect object processing, at least not in the way Farah (1990;

²¹ Even if Farah should be defeated, and her model disproved, she still deserves credit for making her model so explicit that it can be empirically falsified, an attribute unfortunately not characterising all cognitive theories.

²² Whether or not they are pure alexics.

1994) suggested, there are still multiple levels at which the problem could arise. As pointed out by Behrmann & Shallice (1995) impaired letter activation *can* result from a basic impairment in perceptual processing. It could also reflect a problem in feature encoding or feature integration, leading to incomplete or degraded structural descriptions of letters, or a problem in matching these descriptions to stored letter representations.

Arguin & Bub (1993) tested these possibilities in a pure alexic patient (DM), and found that his performance on tests of feature encoding and feature integration was normal. They concluded that the deficit (at least in this case) was in matching the structural descriptions to stored letter representations, that is, intact perceptual information failed to activate representations on the letter level. In an interactive system, like the IAM, this would make the information from the letter level insufficient to activate the correct word-representation, because too many alternative words will be activated simultaneously. Arguin & Bub (1993, 1994) suggested that the LBL-strategy is adopted because the neighbourhood-size is greater for words than for letters.

The concept of “neighbourhood size” refers to the number of confusable or very similar words or exemplars. This will be limited for letters, since the number of structurally similar letters is rather small. For words, this neighbourhood density can be much greater, and will lead to competing activation between two or more words. The rationale for adopting a letter-by-letter strategy is that there is a greater chance for identifying single letters correctly, than for whole words (Arguin & Bub, 1993, 1994).

Behrmann & Shallice (1995) reported a patient (DS), in whom slowed letter identification led to a word length-effect in reading, and suggested that this might be due to a more general deficit in perceptual processing. Sekuler & Behrmann (1996) tested this hypothesis in their study of DS and four other patients who read LBL, and found evidence for impaired visual processing in all of them.²³ Compared to controls, the patients showed deficits in three tests of perceptual fluency, one with letters, one with numbers and one using different shapes as stimuli. Their patients were also impaired in a test requiring the processing of parts of objects. These objects varied in configural goodness (how much they looked like a unity), and relative to controls, the patients showed a greater

²³ One of these patients had surface agraphia, one was suffering from ensuing depression, and one had anomia for fruit and vegetables. They were all slowed in naming visually presented pictures.

increase in response time for the poor configuration-objects. Sekuler & Behrmann (1996) concluded that letter-by-letter reading is caused by a general deficit in the visual processing of "perceptually demanding stimuli". They did not find any evidence that the number of parts of an object affected the time taken to process it, which would be predicted by Farah's (1990, 1994) model.

Sekuler & Behrmann (1996) suggest that in reading, there are no external cues (like configural goodness or symmetry) to rely on, and that this is the reason why the major deficit observed in these patients is LBL-reading.

3.3.3. Covert reading in pure alexia.

Even though pure alexics are unable to read out loud without resorting to the LBL-strategy, they can sometimes make accurate judgements about words they are not able to read letter-by-letter. The first report of this kind of *implicit reading* or *covert lexical activation*, was made by Shallice & Saffran (1986). They demonstrated that ML, an LBL-reader, was able to make semantic and lexical decisions about briefly presented words with an accuracy level above chance, even though the exposure durations were too short to allow letter-by-letter reading.²⁴ This finding has later been replicated in other pure alexics, and the response accuracy in these tests has been shown to be independent of word length (Coslett & Saffran, 1989; Bub & Arguin, 1995).

Another source of evidence for covert reading comes from observations that some pure alexics show a word-superiority effect in letter reporting. If serial letter-by-letter processing was the only way these patients could process written material, one should expect them to report as many letters from random letter strings as from words. Some patients have been shown to report more letters from briefly presented words than from random letter strings (Levine & Calvanio, 1978; Reuter-Lorenz & Brunn, 1990), indicating that some information about words presented too briefly to be read LBL is available to them.

Although some studies have failed to demonstrate covert reading in pure alexics (Warrington & Shallice, 1980; Patterson & Kay, 1982; Behrmann, Black & Bub, 1990; Behrmann & Shallice, 1995;

²⁴ This pt. also had optic aphasia and anomia for visually presented objects. Hence an LBL-reader, not (necessarily) a pure alexic.

Price & Humphreys, 1992, 1995), a theory of pure alexia should be able to explain both the fact that it is sometimes observed, and that sometimes it is not.

There are two major hypotheses concerned with what causes covert lexical activation in pure alexia. Some authors (Arguin et al., 1998; Behrmann, Plaut & Nelson, 1998; Shallice & Saffran, 1986) believe that implicit reading reflects residual operations in a damaged normal reading system. Arguin et al. (1998) interpret pure alexia within the IAM and argue that pure alexics "have access to a lexical access procedure that operates (...) rapidly and in parallel, but which cannot reliably support explicit word recognition on its own". This argument is partly built on Howard's (1991) observation that his pure alexic subjects made "fast responses" to some words, indicating that they are able to process letters in parallel, and that they only resort to serial letter-by-letter processing when parallel processing fails.²⁵ The activation this parallel processing creates may be enough to make semantic or lexical judgements, but not to support overt reading. Arguin et al. (1998, Arguin & Bub, 1993) believe that this is partly because the neighbourhood-size (the amount of very similar items) is greater for words than for letters. On the issue of why implicit reading is only evident in some patients, Bub & Arguin (1995) argue that the extent to which processes of activation and inhibition on the word and letter level are impaired may vary between subjects, allowing lexical/semantic judgements to be based on information processed in parallel in some but not in others.²⁶

Another explanation suggested to account for the performance of pure alexics in tests of covert reading is that it reflects activity in a separate system, not part of the normal reading system.²⁷ Coslett and colleagues (Coslett & Saffran, 1989; Coslett & Saffran, 1994; Coslett et al., 1993; Saffran & Coslett, 1998) argue that letter-by-letter reading is a compensatory strategy adopted because the damaged left hemisphere can no longer process written material, and therefore words and letters are

²⁵ Absolute RT's are rarely reported in the literature, and it is therefore not clear if Howard's (1991) subjects were special, or if *fast responses* can be demonstrated in other pure alexics.

²⁶ Again we run into problems of definition. Although covert reading has been documented in some pure alexics (Shallice & Saffran, 1986, Bub & Arguin, 1995, Coslett & Saffran, 1989), some of the cases described do not belong in this group. For instance Arguin, Bub & Bowers (1998, Bowers, Arguin & Bub, 1996; Bowers, Bub & Arguin, 1996) base their conclusions mainly on their studies of patient IH who, besides using a letter-by-letter strategy in reading, shows no signs of pure alexia. On the contrary he has surface agraphia, shows signs of surface alexia in reading and is anomic. The exact site of the lesion in IH is not clear either, which suggests to me he should be "withheld as evidence", since the only contribution of this case is to confuse matters further. (Note that the same patient is referred to as a pure alexic in one paper (Bowers, Arguin & Bub 1996) and a LBL-surface alexic in the other (Bowers, Bub & Arguin, 1996).)

²⁷ The authors claim that this system does not contribute to normal reading, but this is not a necessary assumption.

perceptually processed in the right hemisphere. This information must then be transferred to the left to be articulated. Because the right hemisphere has only limited word-reading abilities, the constituent letters must be identified serially, and are then transferred to the language areas on the left. According to this view, implicit reading reflects activation in the visual input lexicon and the semantic system in the right hemisphere, which is "biased toward high-frequency, concrete words" (Saffran & Coslett, 1998), and more importantly *does not have access to phonology*.

On the question of why covert reading is only observed in some pure alexics, Coslett et al. (1993) suggest that the strategy adopted by patients may be important.²⁸ We know, from blindsight patients amongst others, that patients may be reluctant to engage in implicit tasks. Still, Coslett et al. (1993; 35) "acknowledge that at least some of the apparent inconsistency between pure alexics may be attributed to individual differences in patients' processing abilities."

3.4. An attempt at integration.

Behrmann, Plaut & Nelson (1998) attempted to integrate these views on pure alexia. They tried to accommodate the experimental findings of both impaired letter processing and covert reading, claiming that other theories focus only on subsets of the empirical findings. They suggest that letter-by-letter reading is the result of the remaining capacity in the normal reading system, which is bilaterally distributed, cascaded and interactive. In a cascaded system, partial information about a stimulus can be passed on to higher levels in the system, and since the system is also interactive, activation on the superior level will feed back to preceding levels. Behrmann, Plaut & Nelson (1998) use the Interactive Activation Model (McClelland & Rumelhart, 1981) as a starting point in their analysis, and suggest that the deficit in pure alexia (they call it LBL-reading) lies either at the feature level, or between the feature level and the letter level. This results in weak activation on the letter level, not sufficient for explicit word identification, and letters must be processed sequentially (letter-by-letter) to enhance activation and allow the word to be identified. The initial weak information on the letter-level may be passed on in the system though, partially activating representations in the lexicon and the semantic system. The IAM does not include connections to the semantic system, but Behrmann, Plaut & Nelson (1998) assume that the activation of semantic and lexical knowledge will operate in the same manner as the rest of the system. Partial word information will be passed on to

²⁸ Coslett et al. (1993) suggest that instructions like: "Try to derive a feeling for the word, do not read it letter-by-letter. Do not try to name the words. " might encourage implicit reading. They also encourage their patients to guess in these tasks.

the lexical or semantic level, and this information might be sufficient to allow the patient to make lexical or semantic judgements about stimuli they cannot explicitly identify. The variability between patients on tests of covert reading can then be explained by individual differences in the amount of information or activation being passed on in the system.

According to Behrmann, Plaut & Nelson (1998), the reading system is bilaterally distributed, and letter-by-letter reading reflects the combined processing of both hemispheres, which both make use of cascaded, interactive processing. This is also valid for covert reading, which is assumed to be the result of dynamic processing within and between both hemispheres, operating on degraded input. An example of how this bilaterally distributed system could work is also provided by Behrmann, Plaut & Nelson (1998). Unlike deep alexics, who some believe are reading via right hemisphere systems (e.g. Coltheart, 1980b), pure alexics almost never make semantic errors in reading. This could be because, even with short exposure durations, phonology for the first letter in the word can be accessed via the left hemisphere system, preventing activation of words not starting with that letter. In this way, both hemispheres contribute to the final output.²⁹

3.5. Pure alexia - concluding comments.

Even though the lesions causing pure alexia have been described in detail and the cognitive deficits studied intensively, no unitary account of the syndrome has yet been reached. This may in part be because, while neurologists have identified the neural substrate for pure alexia, cognitive neuropsychologists have attempted to pinpoint the cognitive deficit causing letter-by-letter reading. They are simply asking different questions. Neurologists ask: Why is reading impaired and writing spared? In cognitive neuropsychology the main question has been: What causes patients to read letter-by-letter, and how can this be explained in a model of reading? It is no easy matter to superimpose cognitive models on brain structures, and I will not attempt to do it here. Still, there seems to be a need for integrating the different approaches. The cognitive neuropsychological study of LBL-reading has generated an enormous knowledge base, and it might soon be time to try to put the pieces together. So far, the IAM framework seems to be able to account for all the findings concerning pure alexia, and Behrmann, Plaut & Nelson's (1998) interpretation of both the deficit in

²⁹ The authors do not postulate this as the mechanism preventing pure alexics from making semantic errors in reading, but use it merely as an illustration of how the two hemispheres could interact in reading.

letter processing and covert reading seems to be the most convincing. The only difference from Arguin & Bub's (1993, 1994) explanation seems to be the postulation of right-hemisphere contribution in normal reading, for which there is no compelling evidence yet. In conclusion, the major empirical findings in the study of pure alexia can be explained within an interactive model of word recognition and semantic access, but how these processes relate to other cognitive functions like vision and language still needs to be clarified.

4. The third alexia.

As we have seen, Dejerine has been credited with having defined alexia with agraphia and pure alexia, but he also described a third variety of alexia, occurring in patients with Broca's aphasia. Among seven patients with Broca's aphasia, Dejerine & Mirallié (1895, in Henderson, 1984) found different degrees of alexia: "Whereas most recognised isolated words, especially common words, none understood an entire sentence. One patient recognised neither letters nor words." (p. 431). One patient was able to read words, but totally unable to identify their constituent letters. This led Dejerine (cited in Henderson, 1984; 431) to propose that "many Broca's aphasics identify words holistically according to the overall gestalt independent of the individual letters."

4.1. Alexia in Broca's aphasia.

Benson, Brown & Tomlinson (1971) reviewed the "varieties of alexia", and presented three patients with anterior alexia in the presence of Broca's aphasia. They noted that letter anomia is an important feature of anterior alexia, and that patients with this syndrome usually read words "by gestalt" or not at all. They showed that their patients did not have problems in matching, sorting or pointing to letters, indicating that the problem was in name retrieval, not in perception. Benson et al. (1971) also presented three cases of pure alexia, and noted that these patients read letters better than words, while the aphasic patients read words better than letters.

In 1977 Benson reviewed 61 cases with Broca's aphasia, in search of the third alexia. Generally, Benson (1977) emphasised that in the evaluation of alexia in aphasia, one must consider two matters: Firstly, one cannot infer alexia from an inability to read out loud in aphasic patients. If a patient is able to comprehend written material, he should not be considered alexic. Secondly, one must be sure that the patient could in fact read prior to the brain insult.

Of the 61 cases with relatively pure Broca's aphasia reviewed by Benson (1977), 51 had some degree of alexia. 17 of these were considered as being mildly alexic, but still "almost invariably" denied being able to read. When appropriately tested, it turned out that these (17) patients could carry out simple written commands, and were able to comprehend parts of written paragraphs, although they made some errors. The remaining 34 patients were truly unable to read. Benson's (1977) aim was to establish the third alexia as a clinical entity - to show that it could be dissociated from pure alexia and central alexia(s). He concluded that the symptoms of, and the cerebral substrate for the third alexia differs from the other alexias, and that the deficit is severe enough "to deserve the term of alexia" (p.331).

Kirshner & Webb (1982) studied four aphasic patients, of whom two had global aphasia and two Broca's aphasia. They found that although these patients were completely unable to identify or name single letters they were able to read some words, mainly short, concrete, highly imaginable high frequency ones. None of them were able to read simple, easily pronounceable pseudoword trigrams (e.g. bab) - they were not able to transform graphemes into phonemes. Kirshner & Webb (1982) suggested that while "visual word form" perception is a function of the left occipital lobe (...) letter naming and grapheme to phoneme conversion seems to be a function of the left anterior hemisphere.

In sum, the symptoms of the third alexia include anomia for letters, but most patients understand some written material - they can read some single words holistically. Severe agraphia also accompanies frontal alexia: copying is usually poor, partly because patients usually have right hemiparesis, and letters are often omitted. Patients are also impaired in spelling out loud and in comprehension of spelled words (Benson & Ardila, 1996). To this date, no cognitive neuropsychological studies of the third alexia have been conducted and no cognitive explanation has been suggested for this breakdown in the reading process. Kirshner & Webb (1982) suggested that

grapheme-phoneme conversion might be a function of the anterior left hemisphere, a position that is strengthened by the cases presented in the next sections.

4.2. Impaired numbers but not letters - The third alexia in pure form ?

Anderson, Damasio & Damasio (1990) presented a unique case of anterior alexia. Their patient, a 58-year old, right handed woman, developed alexia and agraphia following a surgical lesion in the left premotor cortex, in an area usually referred to as *Exner's area*. This area is localised in Brodmann's area 6, in front of the motor area for the hand.

Their patient was not aphasic, and was otherwise neurologically and cognitively intact.

She had severe problems with word reading. Out of 400 presented words, she was only able to read 83 out loud, (mainly high imageability, concrete words,) identifying them immediately in a "gestalt" manner. She did not comprehend any of the words she failed to read, and she was completely unable to read pseudowords.

In a letter identification task, she correctly identified 54/112 large, typewritten letters, and her errors were mainly visual (e.g. S-Z, B-D), still she was able to describe the shape of letters from memory. She scored 8/12 in a letter matching task. In naming to oral spelling, she scored 7/11 correct, but she was able to spell presented words out loud.

In contrast to her inability to read letters and words, she was perfectly able to read numbers ranging from one to seven digits correctly, and she could also identify single digits within complex numbers.

Her writing of letters and words was slow and laborious, and she was not able to write one single word in a legible form, either to dictation or from memory: "Graphemes were poorly formed, and often not formed at all" (Anderson et al. 1990; 757). When she did get the letters right, she wrote them on top of each other, leaving the words virtually unreadable. This writing pattern included her own name. She was also unable to copy letters, but this improved a bit between test-sessions. On the other hand, she could write single- and multidigit numbers correctly to dictation, and she was able to perform written calculations with perfect ease. She could also copy written numbers correctly.

Her impairment in letter identification, along with some whole word-reading and, of course, the site of her lesion, would surely classify her as suffering from anterior alexia, possibly the first case of "the third alexia in pure form" described.

4.3. The role of Exner's area in reading.

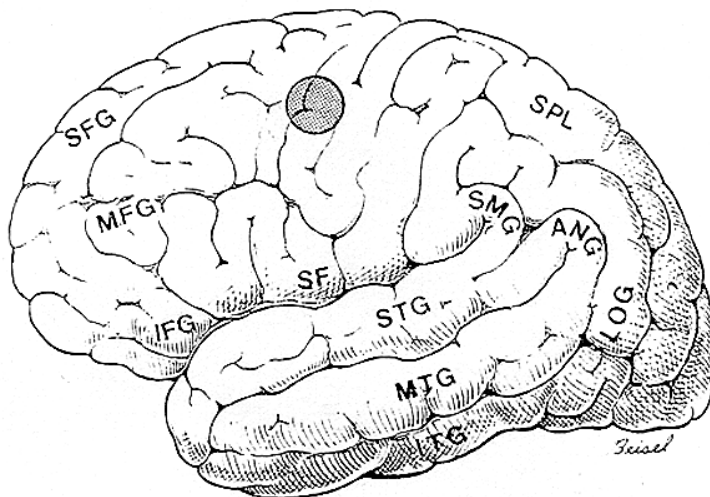
Exner (1881) has been credited with suggesting the existence of a cerebral centre for writing, located at the foot of the second frontal gyrus: the area of premotor cortex lying directly in front of the primary motor area for the hand. (See fig 10.)

Nielsen (1946) also believed that this area was essential in writing, and suggested that it is "especially trained from childhood through the formation of engrams to function as a writing center" (p. 41). Nielsen (1946) also noted that this was not an isolated centre. Associations to the angular gyrus - to the mental images of letters - were necessary to constitute a writing mechanism.

Figure 10.

Lateral view of the left hemisphere. The grey circle marks the approximate position of Exner's area.

From Roeltgen (1993).



Andersen et al. (1990) attempted to explain why a lesion in the "writing area" should also affect reading, and suggested that the visual shape of letters, their sounds, and the motor patterns needed to write them become associated through learning. The "neural network" for letter knowledge will

thereby contain both sensory and motor representations, and Exner's area might play a role in coactivating these representations.

Ritaccio et al. (1992) studied a patient with reading epilepsy, in whom seizures were provoked by reading only when he was vocalising or subvocalising. Speaking normally did not provoke seizures, and neither did reading when subvocalisation was prevented, leading the authors to suggest that grapheme-phoneme conversion was the main precipitant of the attacks. The patient had a lesion in Exner's area, leading Ritaccio et al. (1992) to suggest that Exner's area might be important in grapheme-phoneme conversion. This fits neatly with Benson et al.'s (1971) finding that their patients with the third alexia were impaired in letter naming, but not in matching or sorting letters. The fact that Anderson et al.'s (1990) patient was impaired in non-word reading also supports this suggestion, but their patient was impaired in reading all words, except some high-frequency, concrete words, suggesting a deficit affecting more than just grapheme-phoneme conversion.

Drawing any conclusions about the role of Exner's area in reading is difficult, considering the small number of patients with circumscribed lesions in this area reported in the literature. Anderson et al. (1990) reviewed their records, which contain more than 1200 cases, but found only four with damage limited to the superior part of the left premotor area. Three of these patients had agraphia, and two had alexia in the acute phase. Most other patients with lesions involving this area have more extensive damage, which makes pinpointing the neural substrate responsible for the functional deficits observed nearly impossible.

No cognitive explanations for the third alexia, with or without aphasia, have yet been suggested. The findings in the neurological literature indicate, though, that Exner's area might be important not only in grapheme-phoneme conversion. Anderson et al.'s (1990) patient failed to read most of the words she was presented with. She not able to read non-words, leading the authors to compare her deficit to phonological alexia. But if the sublexical route was selectively damaged, she should be able to read all real words aloud. The fact that she was not indicates that her lexical route is also damaged, and since she is severely impaired in both naming letters and in letter matching tasks, there also seems to be a problem at the letter level. How these mechanisms would fit into a model of reading, I dare not speculate.

5. Reading and the alexias - concluding comments.

*"Twas brillig, and the slithy toves
Did gyre and gimble in the wabe:
All mimsy were the borogroves,
And the mome raths outgrabe."*

We have now come a little closer to understanding the mechanisms at work in reading a text like *Jabberwocky*. According to dual-route model, we are able to read these nonsense words using a process called grapheme-phoneme conversion: we translate the printed words directly into sounds. Real words on the other hand, are read via the lexical route, allowing us to remember their pronunciations and access their meanings. While surface alexics might be able to read this poem, neither deep nor phonological alexics would, since their ability to convert print to sound is compromised. Pure alexics might succeed at reading the poem, with very long reaction times, while patients suffering from the third alexia would not at all be able to decipher this text into sound.

Critics of the dual-route model of reading have suggested that the normal reading process and the different alexias might be accounted for within a distributed model. Still, Seidenberg & McClelland's (1989) computational model would not have been able to read *Jabberwocky*, since it does not read non-words very well. A lexical model that can read non-words as well as regular and irregular words might be designed, but so far the dual-route model seems to do the best job in accounting for the alexias. The cascaded, interactive aspect, added to the dual-route model by Coltheart et al. (1993), seems to make it even more powerful as an explanatory framework. Coltheart et al. (1993) also included the principles of the IAM (McClelland & Rumelhart, 1981) in the dual-route cascaded model, suggesting that pure alexia might also be interpreted within this framework.

The dual-route cascaded model is probably closest to being a unitary account of the alexias, but a lot of questions are still to be answered. The role of the right hemisphere in normal reading, if it has any, is yet to be clarified. To clarify what role the "writing centre" might have in reading, more studies and cognitive explanations are desperately needed.

Science is cumulative, and a scientific approach based on single case studies needs to accumulate many studies before being able to generalise the results. The case presented in the next part might be a contribution to the rapidly growing database of the cognitive neuropsychology of reading.

Part II:
A case study.

1. Introduction.

A number of different patterns of breakdown in the reading process subsequent to brain damage have been reported, often as single case studies. In this section a peculiar case of alexia will be presented. The patient (JM) presented with a highly specific alexia affecting only letters and not numbers. This pattern of performance has only been reported once before (Anderson et al., 1990), the opposite has also been observed (Cipolotti, 1995). JM is also impaired in writing letters, but shows no deficit in spelling, and his other cognitive abilities seem intact. Two main topics will be discussed in relation to the case study: the dissociation of writing and spelling in alexia with agraphia and the dissociation of number and letter reading. Both of these lead to the question of whether learned abilities can be studied in the same way as other, more basic cognitive functions, a question that will also be considered.

2. Case report. ³⁰

The subject.

JM is an 18 year old man, who presented with concussion after being in a car accident in April 1999. He was discharged from hospital after 24 hours, no post-trauma symptoms were noted. When he got home he realised that he could no longer read. JM was then examined by an ophthalmologist, who found that JM had severe problems with letter-identification, but could still read numbers. His writing was observed to be clumsily formed, but basically intact. On June 8th, 1999, JM went totally blind for about ten minutes, followed by sickness and headache. He was admitted to hospital, where binasal, peripheral visual field defects were found. Six months later his visual fields were found to be intact. No other neurological symptoms were noted. JM is fully right handed (+100 at the Edinburgh handedness inventory). Before the accident, he was a student at a commercial college and his grades were on an average level.

JM has not been in school since the accident. In October 1999, he got a part time job in a warehouse. He has been seeing a language therapist regularly since September 1999, and has an extensive training programme on which he practises at home. Since the summer of 1999 he has had daily headaches, and he tires very easily.

JM has a monozygotic twin brother (KM) who has been tested as a control in some of the tests.

Brain-imaging:

CT- scan 10.06.99 showed no abnormalities.

³⁰ This study was conducted by the author in close co-operation with Anders Gade, Hanne Udesen and Christian Gerlach.

MR-scan 16.06.99 showed no abnormalities. A hyperintensity was noted on MR-angiography in the mid portion of the basilar artery, but this might not be abnormal.

SPECT scan 17.11.99 showed no certain abnormalities. There might be a small flow defect in the parieto-occipital area of the right hemisphere, which may include posterior temporal regions, but since this is at the limit of the resolution, this is an uncertain finding.

MR-scan, 17.11.99 showed no abnormalities.

Summary: None of the various brain-scannings performed show any certain abnormalities.

Neuropsychological evaluation.

JM was mainly tested from 29.06.99-20.07.99. He has also been tested on two later occasions, at which point he was in a language training programme, which may have interfered with the test-results. Results obtained after 20.07.99 will be marked in the following.

General abilities:

Neuropsychological assessment of JM revealed a verbal IQ below normal, and a performance IQ in the upper normal range. We also tested JM's twin brother (KM) with a set of tests of general abilities, and a comparison between the two reveals an almost identical pattern. This indicates that JM's low scores on verbal subtests reflect his pre-morbid abilities, and are not a result of his injury.

(Note: KM was tested 23.11.99)

Table 1. Test results in standardised tests:

JM and KM (twin brother).

WAIS-subtests (raw scores):

	JM	KM
Vocabulary	30/80	30/80
Information	14/29	14/29
Similarities	11/26	11/26
Picture arrangement	30/36	28/36
Digit symbol	36	-
Incomplete pictures	16/21	20/21

Raven

Advanced Progressive Matrices, Set I.

	JM	KM
Scoring first answer	4/12	5/12
Self correcting	3/12	2/12
Sum correct	7/12	7/12

RH - Basic battery:

	JM	KM
Digit span forwards	8	5
Digit span backwards	8	6
Sentence repetition	16/22	16/22
Mental Arithmetic	18/20	12/20
Block design	12/12 Mean time: 12,7"	12/12 Mean: 12,1"
Trails A	26"	-

BORB: *

Picture naming (low frequency items)	14/15
Unusual views - recognition	13/15
Drawing from memory	9/9

VOSP: *

Shape detection (screening)	10/10
Dot counting	10/10

BDAE: *

Naming, all items.	48/60
Cookie theft picture	Described in fluent language, including all details.

Rey's:

Complex figure-copy	35/36
Complex figure-retention	32/36
Verbal Memory test	Retention OK, except for letters

Face perception and recognition:

Famous Faces, recognition	15/20
Famous Faces, naming	14/15
Rivermead face-recognition	10/10

Others:

* BORB: Birmingham Object Recognition Battery.

* VOSP: The Visual Object and Space Perception Battery.

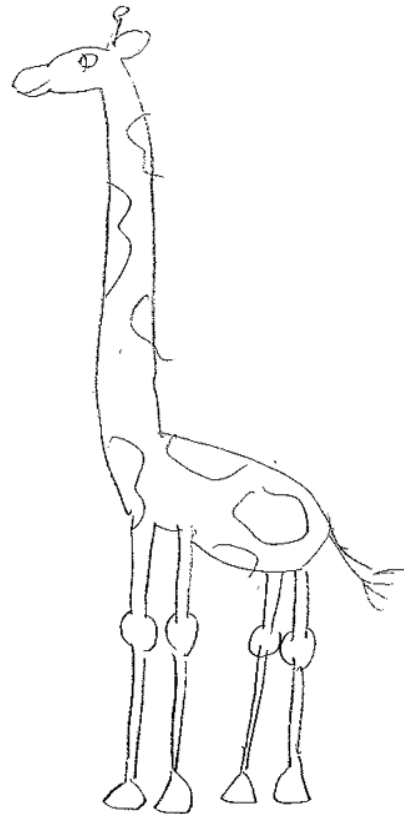
* BDAE: Boston Diagnostic Aphasia Examination.

Street	19/20
Poppelreuter	15/15 in 35 sec's.
Colour naming	10/10
Colour recognition	10/10
Stroop	47/50
Copy of house	3/3
MMSE, copy of figure	1/1

Visuoperceptual and language abilities:

The results on the visuoperceptual tests show that JM does not have difficulties in naming or recognising objects, faces or colours. He shows no problems with integrating fragmented pictures (Street), separating simultaneously presented pictures (Poppelreuter), or mentally transforming images (BORB: Unusual views). His visuo-constructional abilities are also intact, as evidenced by correct copying of figures and perfect drawings from memory. (See Figure 1a) and b.)

Figure 1. a) JM's copy of a house (left). **b)** JM's drawing of a giraffe (from memory).



Language:

JM's speech was fluent and prosodic, although he generally spoke in a low voice and sometimes mumbled. He had no word-finding difficulties in conversational speech, and performance was normal on tests of confrontation-naming (BDAE and BORB). Sentence repetition and his score in the vocabulary subtest of the WAIS were below normal, but at the same level as KM.

Reading.

Preliminary observations.

When presented with short (3-5 letters), high frequency words, and asked to read them, JM spontaneously covered up all letters but one, and tried to identify them one by one. He volunteered that he had begun this strategy when he first realised he could not read. He frequently misidentified letters, and often came up with several alternatives or left letters out if he could not identify them. For instance, when presented with the word *ankel* he read *a - h* or *n - don't know - e - l*, and then tried to guess what the word was. (In this particular case, he failed.)

Experimental investigations:

Single letter identification:

Stimulus material: The letters presented were written in 48 point Times New Roman, and were presented one by one.

Reaction times were measured using a hand-held stop watch.

During the first test-session JM was able to identify 13/29 upper case letters correctly, with an average reaction time of 6,3 seconds, and 18/29 lower case letters with a mean time of 10,7 sec's.

On a retest one month later, using the same material, JM was able to correctly identify 12/29 upper case letters, the mean time being 8,8 seconds.

JM is clearly impaired in letter identification when letters are presented visually. To investigate whether his performance would improve with multimodal stimulation, we presented him with large wooden letters (approximately 10 cm tall).

When allowed to feel their shape, as well as look at them JM correctly identified 8/10, with a mean time of 3,9 seconds.

Inspired by Monsieur C's superior ability to identify letters by touch when they were drawn in his hand, we also tested JM's ability to identify letters this way. When the experimenter outlined letters in JM's palm, he correctly identified 4/7, claiming he did not find this any easier than identifying them by sight.

Imagery.

To investigate JM's ability to imagine letters and their shapes, we asked him to decide whether a named letter had only straight lines or included curved lines. JM scored 8/8 in this test.

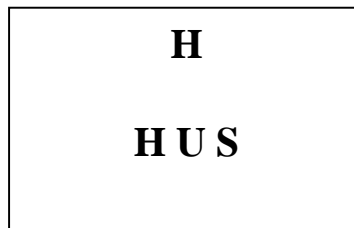
In a letter decision task with letters and non-letters, JM scored 9/12, classifying three letters as non-letters.

We constructed a letter decision task consisting of normal letters, mirror reversed letters and letters turned upside down. JM could not classify any of the letters when they were presented simultaneously. When allowed to cover them up, and identify them feature-by-feature, he scored 8/10, classifying two letters presented upside down as real letters.³¹

Letter matching:

We presented JM with a letter matching task, with both same case and cross case matching. In half the trials, the target letter was placed within a word, in the other half it was part of a random letter string. There were either two or four distractors. (An example can be seen in figure 2.)

Figure 2. Letter matching. JM was asked to find the letter corresponding to the target presented on top.



As in reading, JM covered up all letters but one, and identified them serially. The results are found in table 2.

Table 2.

Number of letters	Word/non-word	Same /-crosscase	Score
3 letters	word	same case	2/3
3 letters	word	cross case	2/3
5 letters	word	same case	3/3
5 letters	word	cross case	2/3
5 letters	non-word	same case	3/3
5 letters	non-word	cross case	2/3

Seeing that JM was impaired in this task, we presented him with a simpler matching task.

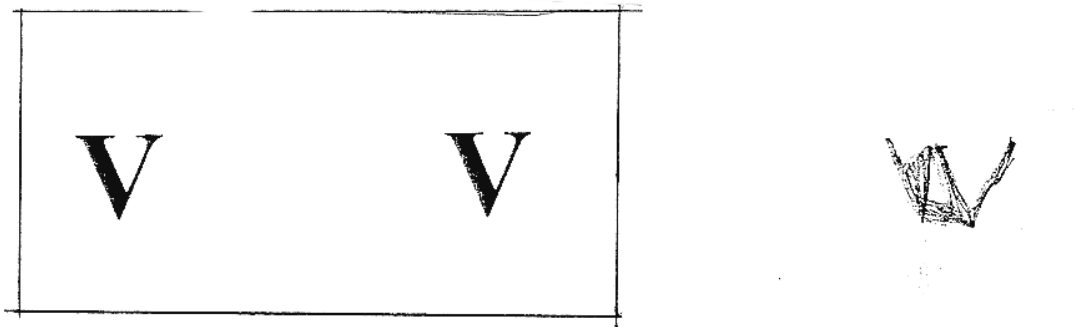
We presented JM with two 24 point, upper case letters typed side by side, and asked him if they were the same or different.

Without covering up one letter, and identifying them one by one, JM could not answer this question. Using this strategy he correctly matched 1/5 letter pairs.

JM also made a drawing of what he saw when he was looking at the letter pairs, which indicates that he cannot separate the letters from each other. The stimulus card and his drawing are shown in figure 3.

³¹ This test was administered 23.11.99, and the feature-by-feature identification strategy used by JM in this case was something he had learned from his language therapist. Although he had used a similar strategy before, it had not been on a feature level, but on a letter level.

Figure 3. A stimulus card from the simple letter matching task (left), and JM's drawing of what he is seeing when looking at it (right).



Word reading:

When attempting to read words, JM covers up all letters but one with his fingers, thus serially identifying the letters, and sounds them out loud. He frequently misidentifies letters and ends up with letter-strings having no meaning. When this happens, he guesses, and sometimes ends up with the right word, still not really reading it.

Stimuli: All words presented for reading were written with capital letters in 36 point Times New Roman, on separate cards. Word frequency measures were found in Bergenholtz (1992), and concreteness ratings are based on Paivio et al. (1968).

During the first test-session, JM was able to read 2/5 three-letter low frequency (less than 20 per million), concrete words correctly, with RT's at 12 and 15 seconds. He failed reading any five letter words correctly. Misidentification of letters accounted for all his errors, and he always substituted a visually similar letter, e.g. *ENG* was read *FNC*.

On a later occasion³², JM was tested on a list of high-frequency, concrete words. He correctly read 5/5 three-letter words with an average time of 26 seconds, and 4/5 five-letter words at an average time of 38 seconds. He also read 5/5 three-letter non-words correctly. The non-words were constructed from changing one letter in words from the high-frequency, concrete word list. Curiously, his average time for reading non-words was 16 seconds, which is faster than his reading of words.

Whole word reading:

³² 23.11.99

We presented JM with short "symbolic" words like *TV* and *WC*, to see whether or not he was capable of reading such words in a "gestalt manner". When prevented from covering up letters he could not read these words, and could not guess what they were. He could not read his own name in a glance.

We also attempted to test JM's whole word reading skill using a computerised lexical decision test, in which words and non-words were presented too briefly to allow letter-by-letter reading. JM was instructed to attend to the whole word, and indicate whether it was a real word or a non-word. He was encouraged to guess if he was unsure. JM gave up after very few trials. He claimed that the test did not make any sense to him, and he had no idea whether real words or non-words were presented, as he could not read them. The test was then discontinued.

Writing.

When writing to dictation or from memory, JM superimposes letters on each other, making the words virtually unreadable (see figure 4). He does this even when writing his own name and address.

Figure 4. JM's attempt to write:

a) his own name

[deleted]

b) the words *hus*, *bil* and *kat*.



When presented with a grid system, JM was able to place letters within the squares, and did not superimpose. He wrote 14/14 letters correctly to dictation, and could write his name and address.

When asked to write the alphabet from memory, JM could not remember the shape of two letters (*g* & *e*). All other letters were legible.

When instructed to move his hand between letters, he was able to write a simple sentence without superimposing letters, on lined paper.

Copying:

When instructed to copy letters, JM first tried to identify them, and then write the letter. Using this strategy, he correctly copied (or wrote) 10/14 letters. He only abandoned this strategy, when he could not identify the presented letter, and

then tried to draw what he saw, the result being either the wrong letter, or (in one instance) an illegible shape. Three of the errors in this test are based on misidentification of the stimulus ($B \rightarrow R, Y \rightarrow V, a \rightarrow o$).

Copying Greek letters:

As we have already seen, JM was not impaired in copying figures, not even Rey's complex figure, but at the same time, he was impaired in copying letters. In letter copying, he first tried to identify the letter, and then write it. To test the specificity of his deficit, and to see what he would do when he could not identify the stimulus and then write it, we asked him to copy ten Greek letters. JM did not recognise any of the letters presented (including π), and only 1/10 letters was copied correctly, the letter "ε", which (according to JM) looks like a mirror-reversed "3".

Oral spelling:

10/10 high frequency words (4-8 letters) were spelled correctly. JM also spelled 3/3 seven-letter abstract words, and 5/5 nine-letter abstract words correctly.

Naming to oral spelling:

When the examiner spelled words out loud, JM named 30/30 correctly. The list consisted of 3-5 letter, high frequency nouns, both concrete and abstract.

Number reading:

Identification:

Stimuli: All numbers were written in 48 point Times New Roman.

JM read 10/10 singly presented numerals correctly, with RT's below 1 second.

He read 15/15 multidigit numbers (3-5-7 digits) correctly, with RT's at or below 1 second.

Accessing semantics from numbers:

We constructed a set of well known numerals, consisting of famous historical dates, JM's birthdate and his postal code, to assess whether he could access semantics from numbers. JM scored 4/7 in this test. We also tested KM on the same numbers, and he obtained the same score, answering correctly on the same items as JM.

Number writing:

JM is able to write single- and multidigit numbers to dictation, not superimposing as he does with letters.

He wrote 5/5 single, and 21/21 multidigit (2-7 digits) correctly.

JM is also able to perform written calculations. (See fig 5.)

Figure 5. Example of JM's number writing and written arithmetic.

$$\begin{array}{r} 23 \times 12 \\ \hline 36 \\ 240 \\ \hline 276 \end{array}$$

$$\begin{array}{r} \\ 809 \times 47 \\ \hline 423 \\ 000 \\ 37600 \\ \hline 38023 \end{array}$$

$$\begin{array}{r} 434 : 7 = \underline{\underline{62}} \\ 42 \downarrow \\ 14 \\ 14 \\ \hline 0 \end{array}$$

$$\begin{array}{r} 621 : 9 = \underline{\underline{69}} \\ 54 \downarrow \\ 81 \\ 81 \\ \hline 0 \end{array}$$

Summary.

JM shows a selective impairment in reading and writing, other cognitive functions not being affected.

Neuropsychological evaluation of JM revealed a verbal IQ below normal, and a performance IQ in the upper normal range. An identical pattern of performance was observed in JM's twin brother, indicating that this discrepancy is habitual, and not a reduction of pre-morbid cognitive abilities. JM's visuo-perceptual and -constructional abilities are also intact, as are his general language abilities.

JM is severely impaired in single letter identification, and because he uses a letter-by-letter strategy in reading, his response-times in reading are extremely long. The time taken to read a word seems to depend as much on which letters the words consist of, as on lexical variables (frequency and concreteness), since JM identifies some letters more rapidly than others. No whole word reading was observed.

In writing, JM superimposes letters on each other, making the words virtually unreadable. We have not observed any spelling errors, but some letters are poorly formed. When presented with a grid, JM is able to separate letters from each other. JM's naming to oral spelling is good, as is his spelling of dictated words.

JM was able to identify written numbers (1-7 digits) quickly and correctly, and he has access to semantic knowledge from numbers. His number writing is flawless, as is his written arithmetic.

There is a striking dissociation between JM's reading and writing of letters and numbers. While he is severely impaired in reading and writing words and letters, his number reading skills and written arithmetic are intact.

2.2. Impaired letters but not numbers.

JM's pattern of performance closely resembles that displayed by Anderson et al.'s (1990) patient.³³ They are both severely impaired in letter recognition, while their other visuo-perceptual abilities, including number reading, are intact. Both patients also show the same deficit in writing, superimposing letters on each other, but can still write numbers and perform written arithmetic.

³³ For convenience, this patient will be referred to as ADD in the following.

It seems that ADD is more impaired in writing than JM, though. Her writing was extremely slow, and in a letter dictation task, only 2/15 of her letters were legible. JM, on the other hand, could write 26/28 letters in the alphabet correctly. ADD was also impaired in naming to oral spelling, but her ability to spell words named by the examiner was intact. JM performed excellently in both these tasks. Another interesting difference between the two patients is that ADD was able to read some words "holistically", an ability we have not been able to demonstrate in JM. On the other hand, ADD was unable to read non-words. JM was able to read non-words, using his letter-by-letter strategy.

But are the differences between these two patients only a matter of strategy ?

Since their patient could read some short, concrete words out loud, but were unable to read non-words, Anderson et al. (1990) compares her deficit to phonological alexia. This does not hold for JM, who, by using a letter-by-letter strategy, is able to read non-words. ADD was severely impaired in letter naming, and would probably not be able to read the way JM attempts to do.

Anderson et al. (1990) seem inclined to classify their patient as agraphia. They do not specify the type of agraphia, but note that "the predominant feature (...) was severely defective grapheme formation"(p.761), and that her ability to write numbers indicated that the repertoire of movements necessary for writing were spared. JM's grapheme formation was not as severely distorted as ADD's, and one might speculate if his deficit in writing was mainly a spatial one.

One major problem in the study of JM, is that no lesions were identified on the various brain scans performed. This might suggest that his symptoms reflect a psychogenic deficit. The similarity between JM and ADD, and the specificity of the symptoms indicate that they are the result of some sort of cerebral injury, even though it cannot be demonstrated.

There are two major aspects of JM's alexia that deserve further discussion. His preserved ability to spell and name words to oral spelling and the absence of whole word reading does not fit with the pattern of performance displayed by ADD or other patients suffering from the third alexia. The clear dissociation of number and letter reading and writing observed in JM and ADD also poses an interesting question about the organisation of the reading process. A problem concerning both these

questions is whether reading, a phylogenetically young ability which is learned via instruction, is organised the same way as other cognitive abilities and can be studied in the same manner. The cognitive neuropsychological study of reading is based on the (often implicit) assumption that the organisation of the processes involved is shared between individuals, regardless of how they learned to read, how often they engage in reading and what kind of material they read. This assumption will be discussed in section 3. A related question, whether acquired abilities can be selectively damaged leaving related cognitive functions intact, will also be addressed.

2.2. Alexia and agraphia with spared spelling and comprehension of spelled words.

One of the interesting aspects of JM's alexia (and possible agraphia) is his preserved ability to spell words out loud and name to oral spelling. In pure alexia, spelling is usually preserved, an ability which patients with this syndrome actively make use of in their (compensating strategy of) letter-by-letter reading. These patients can also write, though, probably because their angular gyrus is intact (Geschwind, 1965; Damasio & Damasio, 1983). In the third alexia (in aphasia), on the other hand, both spelling and naming to oral spelling are impaired (Benson 1979). ADD's spelling was intact, but her comprehension of spelled words was impaired (Anderson et al., 1990). We did not observe any spelling errors in JM's writing, and his writing impairment may be mainly spatial. The general question addressed in this section is whether we can infer that JM's written spelling is intact on the basis of his performance on tests of oral spelling. More specifically, the question is: Has preserved oral spelling been observed with impaired written spelling and alexia ?

Some cases have been described in which intact spelling has been associated with agraphia and alexia, and once again we turn to Dejerine for a description of the first case.

Dejerine & André-Thomas (1904³⁴) described a patient with alexia and agraphia, but with intact spelling and naming to oral spelling. They hypothesised that this pattern of breakdown was caused by two separate lesions, one in the posterior part of the left hemisphere causing pure alexia, and a separate lesion disconnecting motor control of the left hand from the language areas at the left (hemiplegia prevented the patient from writing with his right hand). The first lesion prevented visual information from reaching the angular gyrus while the second disrupted graphic information from

³⁴ Dejerine & André-Thomas (1904) are cited in: Kinsbourne & Rosenfield (1974) and Rothi & Heilman (1981).

reaching the motor areas in the right hemisphere. The left angular gyrus was spared, leaving spelling and naming to spelling intact (Dejerine & André-Thomas, 1904).

The patient later died of a massive haemorrhage of the right hemisphere, and only the first lesion was confirmed at autopsy. The angular gyrus was found intact.

Albert et al. (1973) reported a patient with mild aphasia, alexia and agraphia, whose oral spelling abilities were relatively preserved, compared to his written spelling and reading. A Technetium-scan showed an area of increased uptake in the left occipito-parietal region.

The patient could only read 10-20% of short, common words presented to him, and his identification of singly presented letters was roughly on the same level. On the other hand he successfully read 90% of singly presented numerals correctly. His spelling and naming to oral spelling was far better than his reading and written spelling, but his performance deteriorated with word length and complexity. One aspect of this patients' performance worth noting is that he could access semantic information from words he failed to read. For instance, he made no errors in an "odd man out"-test with written words. He could also readily detect misspellings in words and indicate if a letter string represented a real word or a pseudoword. This led Albert et al. (1973) to conclude that the patient had lost his phonemic reading ability, and they suggested that this was caused by "a functional disconnexion between visual, auditory and motor systems involved in the understanding of written language"(p.328).

Kinsbourne & Rosenfield (1974) reported a patient (CM) with left posterior parieto-temporal ischemia, who presented with mild aphasia and several symptoms associated with Gerstmann's syndrome (finger agnosia, left-right disorientation and impairments in mental arithmetic).

Furthermore, CM had mild constructional apraxia and mild right-sided neglect.

They observed that CM was far better at spelling words orally than in writing (100% vs. 53% correct), and also better at naming words to oral spelling than writing spelled out words (100% vs. 57% correct). They found no difference in number reading or writing (e.g. 56 spelled "five-six" or "fifty-six"). Kinsbourne & Rosenfield (1974; 224) concluded that "CM's ability to implement letter choice using visual shapes rather than sounds was selectively impaired". They suggested that the deficit seemed "to involve a posteriorly-located facility which transforms letter sounds or letter

names into visual representation [on the basis of which] the efferent systems are (...) programmed to write."

They also pointed out that their observations ruled out Dejerine and André Thomas' (1904) hypothesis as a possible explanation, since CM was writing with his right hand.

Mohr (1976) reported a patient with alexia and agraphia, whose ability to spell and name to oral spelling was superior to his reading and writing of the same words. A brain scan of this patient showed an area of high uptake in the left posterior parietal region near the midline.

Rothi & Heilman (1981) presented a patient who, while being alexic and agraphic, showed preserved ability to spell words out loud and name to oral spelling.³⁵ In reading words, their patient would sound out each letter and then produce the word, and he could not be persuaded to (try to) use any other reading strategy. He was not able to decipher whole sentences using this strategy. The patient also spelled out loud when trying to write, but often produced a letter (grapheme) not corresponding to the sound. His copying of words was also impaired, as was his writing with anagram letters, although this was better than his written spelling. Number reading was not tested. The patient was able to perform simple calculations, but it is not clear whether they were presented orally or in writing. The patient was able to spell irregular words out loud, indicating that his visual images of words were intact, but that the patient did not have visual access to it. Rothi & Heilman (1981) suggested that his impairment was located in a "graphemic area", which is "responsible for distinguishing the features of a grapheme and for guiding motor programming in grapheme production" (p.8). To compensate for this deficit, the patient utilised a more general purpose visual system, seeing letters as objects, and then named them out loud. Unfortunately, a CT-scan of this patient showed no focal abnormalities, and EEG only showed mild diffuse slowing, leaving the lesion causing the impairment unknown.

An important point made by Rothi & Heilman (1981) is that the reading strategy adopted after a brain insult causing reading difficulties is likely to be related to strategies used premorbidly. Their argument is that skilled readers may have more than one reading strategy available to them, and

³⁵ The authors claim his letter recognition abilities were intact, still reading a word took him from 4-10 seconds, which is slower than many LBL-readers.

some of these strategies or reading routes might still be available subsequent to brain damage. People who can read, but are not particularly good at it, or have not engaged much in reading, might not have alternative strategies available to them postmorbidity. Put another way: people that do not read much are not likely to have many entries in the visual input lexicon, and must therefore rely on grapheme-phoneme conversion for many words. If this pathway is disrupted by brain damage, reading ability will be more severely affected than for people with an extensive "sight vocabulary". Price & Humphreys (1995) made a similar point, speculating that "patients [might] adopt contrasting reading strategies because they are differentially able to utilise a back-up system" (p.593).

This poses an interesting problem for the cognitive neuropsychological study of reading though. If premorbid skills are likely to affect the strategies adopted by alexics, diagnosing patients and reaching conclusions about the normal reading system on the basis of errors and strategies used postmorbidity will be extremely difficult. Patients might display different symptoms subsequent to brain damage, depending on how they were reading in the first place: Reading might be differently organised in our brains, depending how we learned it, and how (much) we have been reading since. This problem will be discussed more thoroughly in section 3.

It is evident from the cases reviewed in this section, that we cannot conclude that JM's written spelling is preserved based on his oral spelling skills. The explanation for the dissociation between oral and written spelling suggested by Rothi and Heilman (1982) seems to be able to account for JM's deficit in reading and writing. A deficit affecting an area responsible for both perception of letter features and motor programming for grapheme production could explain JM's impairments. Rothi & Heilman (1982) also suggested that their patient relied on a general purpose structural description system for letter identification, and this could also be valid for JM. Interestingly Albert et al's (1973) patient was better at identifying numbers than letters, like JM is, and Kinsbourne & Rosenfield's (1974) patient did not show the difference in writing and spelling of numbers as he did with letters and words.

2.3. The dissociation of number and letter reading.

In cognitive neuropsychology, two functions, processes or systems are said to be independent, if one can be disrupted without affecting the other, and this is often referred to as a simple dissociation.

The evidence becomes more compelling if one can demonstrate a double dissociation, i.e. that one function (e.g. letter reading) is selectively impaired in one patient, while another function (e.g. number reading) is selectively impaired in another patient.

A selective deficit in reading letters compared to numbers is evident both in Anderson et al.'s patient, and in JM, and the reversed pattern has also been described (Cipolotti, 1995), creating a double dissociation. SF, a patient suffering from probable dementia of Alzheimer's type, had a severe impairment in reading Arabic numerals aloud while his ability to read letters and words was spared (Cipolotti, 1995). Since there were no focal lesions in this case, one can only speculate on the neural areas involved.

As we have seen, letter processing is impaired in pure alexia, even though most patients can identify singly presented letters quite well. Number reading has not been studied as intensively as word reading in alexic patients. It is generally assumed that number reading can be spared in pure alexia. For instance, Geschwind (1965) stated that "The reading of numbers is also frequently preserved in these cases - *in Dejerine's case number reading was perfect.*"(my italics). In fact, Monsieur C was not at all perfect at reading numbers, although his number reading was far better than his ability to identify letters. As mentioned in Part I, he read multidigit numbers digit-by-digit (1-1-2 spelling 112), the same way most pure alexics read words.

It is curious, that while peripheral accounts of pure alexia often claim that it is caused by a general perceptual deficit, this is usually based on studies of object processing in pure alexic patients (Kinsbourne & Warrington, 1962; Friedman & Alexander, 1984; Farah, 1990, 1994), even though a comparison between number and letter reading might have been more informative.

Cohen & Dehane (1995) studied the number processing skills of two patients with pure alexia. Their major finding was that accuracy in number reading is task dependent. Their patients were more accurate in reading two digits "digit-by-digit" (e.g. 2-4) than as one number (e.g. 24). Their main conclusion was that number *reading* is impaired in pure alexia, even though it is affected to a lesser degree than letter reading.

They interpreted this as an impairment in making structural descriptions of numbers, or what they call a *visual number form*, which can be compared to Warrington & Shallice's (1980) word form system. Cohen & Dehane (1995) noted that letter identification is usually more impaired than single number reading in pure alexia, indicating that these two functions are not dependent on the same processing system.

3. Dissociations and acquired abilities.

Even though numbers and letters are similar in form, it seems that they are processed differently in the brain. Where might this dissociation occur? Recent publications, mainly in the field of connectionist modelling, have emphasised that one should be careful in suggesting independent systems in the brain on the basis of dissociations, since similar dissociations can be modelled in distributed networks (Plaut, 1995; Nobre & Plunkett, 1997). But if we could find studies indicating that processing of letters and of numbers occur in different brain areas we would have a stronger case for localisation.

3.1. Numbers and letters.

Polk & Farah (1998) refer to an unpublished fMRI study testing whether or not the neural substrates for letter and digit recognition are segregated. They found that "at least in some literate subjects, certain extrastriate areas respond significantly more to letters than digits" (Polk & Farah, 1998; 849). Still, the fact that pure alexics are usually impaired in both letter and number reading, although to a different degree, imply that the neural substrate for perception and reading of letters and numbers is partially shared.

One of the important questions asked by Polk & Farah (1994, 1998) is whether learned abilities can be localised in the brain, and if so, which mechanisms could be responsible for this. Reading numbers and letters are phylogenetically recent abilities, so if different cerebral areas respond significantly more to letters than to numbers, this can not be explained genetically. Letters and numbers are also structurally similar, indicating that in perception they would rely on the same mechanisms. How then, do these spatially segregated areas for letter reading come to be?

Polk & Farah's (1994; 1995; 1998) answer is that neural learning is correlation based (i.e. based on Hebbian learning), so that stimuli that occur together in the environment will create associations between neurones responding to these stimuli. The appearance of letters in our environment is both spatially and temporally correlated (letters are usually presented together in time and space), and according to Polk & Farah (1998; 849) this "could interact with the brain's correlation based learning mechanisms (...) to lead to the segregation of letter recognition".

Polk & Farah (1995) modelled this process in a neural network. Given a set of stimuli consisting of clusters of letters and clusters of numbers, spatially localised areas for both letters and digits developed in the network. But in the real world numbers and letters also occur together, and letters generally occur more frequently than numbers. In another study (unpublished, referred by Polk & Farah, 1998), the network was trained on input sets in which numbers and letters occurred together, and letters occurred more frequently than numbers. The network then self-organised to produce a segregated letter area, but digits were not grouped together. This fits neatly with the findings in the imaging studies, where no segregated digit-area was found (Polk & Farah, 1998).

So, neurones that respond to stimuli that occur together will be tightly connected in the brain, and since letters are usually accompanied by other letters, a special, self-organised, brain area will respond more to letters than to other kinds of stimuli. According to this hypothesis, people that are used to seeing letters and digits together, should have less specialised brain areas for letters and digits than the rest of us. Polk & Farah (1994) conducted a behavioural study of number and letter reading to check this assumption. Their starting point was the so-called "pop-out effect", the fact that targets in an array of visually dissimilar distractors can be detected instantly, without the use of serial search. Letter targets among number distractors will thus be detected more rapidly and efficiently than letters among letters. The subjects in this study were (Canadian) postal workers who were used to sorting mail by codes consisting of both numbers and letters. A comparison group consisted of other (US) postal workers and college students. If the hypothesis of co-occurrence is correct, the mail sorters should show a smaller "pop-out effect" than controls because they were used to seeing letters and numbers together, and this was just the pattern observed. On this basis, Polk & Farah (1994; 649) suggested that "the alphanumeric category effect is modulated by environmental co-occurrence; the effect is not just an artefact of physical differences between letters and digits" and

that "*environmental statistics can influence the architecture of vision, even in adulthood*" (my italics).

This causes some problems for the cognitive neuropsychological study of reading though. If the processing of letters and digits is dependent on environmental factors, then one cannot expect to find the exact same pattern of breakdown in patients with identical lesions. Supposing one of the postal workers from Polk & Farah's (1994) study suffered a cerebral insult, one could not expect his cognitive deficits (in the area of reading) to be identical to those of others not used to processing numbers and letters simultaneously. Rothi & Heilman (1981) also suggested that (compensating) strategies available to alexic patients are dependent on premorbid skills, and how much one has been reading. Even if this does not imply that the reading process is organised differently in each of us, it is problematic for the study of "patterns of paralexia", where reading errors made by alexic patients are used as a basis for creating models of normal reading. These errors may be influenced by the patients' premorbid reading patterns, and might not only reflect the extent of impairment subsequent to brain damage.

3.2. Acquired abilities.

If the neural substrate of our capacity to read is dependent on how, and under which circumstances, we have been exposed to words and numbers, it would presumably also be dependent on how we learned it in the first place. Castro-Caldas et al. (1998) demonstrated that literate and illiterate subjects showed different patterns of brain activation during a pseudoword-repetition task, and that illiterate subjects made more mistakes than the literate subjects did in this task. They concluded that "learning to read and write during childhood influences the functional organisation of the adult human brain" particularly affecting language relevant areas (Castro-Caldas et al., 1998; 1060).

Throughout the history of the literate society, there have been several different methods used for teaching children to read and write, presumably leading to individual patterns of organisation in the brain. People also differ in how much they engage in reading. This could imply that reading can be differently affected in each one of us following brain damage, depending on how and when we learned to read letters and numbers, and how we have been doing it since. The worst-case scenario would then be that it is impossible to say anything useful about the functional organisation of the

reading process, let alone the anatomical substrate for reading, based on evidence from brain-damaged patients or brain-imaging studies. Still, since similar patterns of breakdown in the reading process are consistently found following lesions in the same areas in different brains, there must be some constraints on the way reading can be organised.

Patterson & Ralph (1998) challenged the assumption that there are brain regions dedicated to reading, arguing that "disorders of reading may rarely, if ever, occur in isolation, that is, without impairment to (...) ontogenetically earlier capacities of language or visual processing"(p.235). On the other hand, Polk & Farah (1994) claim that "late experience alters vision", and suggest that environmental factors do influence the functional organisation of the brain, a position also held by Castro-Caldas et al. (1998). At the core of this battle lies the question: can reading be selectively damaged ? Rephrased, the question could be: Is pure alexia really pure ?

Patterson & Ralph (1998) asked just this question, and in answering claim that "there is considerable and mounting evidence that L-by-L reading (...) is anything but pure, and is just one manifestation of a visual-processing deficit " (p.237). And so we are back to square one - who ever claimed that letter-by-letter reading was pure ? Of this "considerable and mounting evidence", Patterson & Ralph only refer to one study by Behrmann, Nelson & Sekuler (1998), showing that their LBL subjects' naming latency for pictures increase with picture complexity, relative to control subjects. It is worth noting, though, that two of the five "pure alexics" in this study also had (surface) agraphia, while one was suffering from ensuing depression.

Still, naming latency also increased with complexity in the two subjects who presumably were pure alexics. But this does not prove that all pure alexics are impaired in object processing, or that brain regions important for reading are necessarily also used for object processing. It is peculiar that while impaired object processing keeps coming up as an explanation for pure alexia and has been studied so extensively in alexic patients, number reading has been studied in so few cases. The structural and perceptual similarity between letters and numbers is obvious, and yet they seem to be affected differently in subjects like JM, ADD and Cipolotti's (1995) patient.

Going back to Polk & Farah's (1995, 1998) assumption that neural learning is correlation based, could environmental statistics account for the findings of impaired object processing in some pure alexics ? Do pictures not often occur in the presence of letters ? Even though objects often appear among other objects, line drawings, the stimuli most frequently used to assess object naming, are usually accompanied by text. Could it be, then, that the pure alexics whose object processing is impaired, are used to reading comics, children's books or furniture catalogues ?

It might be time to take a step back from the debate and ask oneself what the fight is really all about.

Can cognitive functions be dissociated from each other, and if so - what does that tell us ?

First of all, a double dissociation indicates that the two functions in question can operate independently of each other. That does not mean, though, that they cannot both rely on the same more basic functions or that they do not interact in a normal brain. Both letter and number reading depend on vision, so to some extent they rely on the same cognitive functions and processes. Reading must also be tightly interconnected with other language functions, both speech and semantics. What a double dissociation does not necessarily imply, is that the cognitive functions in question rely on completely independent systems (Shallice, 1988, Plaut, 1995). So in our case, we do not need to postulate a number system and a letter system to account for the different pattern of performance on numbers and letters seen in JM, ADD & Cipolotti's (1995) patient. But even if we do not postulate different, independent systems for every dissociation found, the dissociations still need to be explained.

4. Back to Exner.

None of the various brain-scannings of JM have revealed any focal damage. Even though there are some discrepancies, the case most similar is the one reported by Anderson et al. (1990) who had a lesion in the left premotor cortex. Exner (1881) suggested that the cortical area just in front of the motor area for the hand might be important for writing, and Anderson et al.'s (1990) case strongly supports this, but what role could this area have in reading ?

Polk and colleagues (Polk, personal communication) conducted some of their fMRI letter vs. digit studies using a head coil placed so they could see the whole brain. With this experimental set-up they replicated their earlier findings of different occipito-temporal activation for letters and digits,

but did not find any reliable activations anywhere else. There was no difference in activation of Exner's area whether the subjects were looking at letters or numbers. Still, Anderson et al.'s (1990) patient did not have any other known lesions than the one in this area, indicating that it was responsible for her impaired reading of letters, while it left her reading or writing of numbers intact. Kirshner & Webb (1982) suggested that anterior brain regions were important for the associations between print and sound, and Ritaccio et al.'s (1996) findings suggest that Exner's area is important in grapheme-phoneme conversion, both findings predicting that activation in these regions will only be seen when words are read out loud. The subjects in Polk et al.'s experiment were only passively viewing the stimuli, not sounding them out, which could explain the lack of frontal activation.

Even though disturbed grapheme-phoneme conversion can account for an inability to name letters and words, patients like the one described by Anderson et al. (1990) should still be able to identify them, for instance in matching tasks. ADD actually failed 4/12 trials in a cross-case matching task, and she could not access semantic information about words she failed to read out loud. Even if we postulate that Exner's area is important in assembling phonology, regardless of reading route, this cannot account for all the findings.

Anderson et al. (1990) suggested that the visual shape of letters, their sounds, and the motor patterns needed to write them become associated through learning, and that Exner's area might play a role in coactivating these representations. Assuming that the coactivation of these patterns is necessary both in reading and writing, but are different for letters and numbers, this could be an appealing cognitive explanation for the impairments of JM and ADD.

5. Case JM - summary.

As we have seen, JM's pattern of performance is not readily classified as either pure alexia or the third alexia, and with no lesion to give us a hint, it is not at all obvious which group he belongs in. The most similar case reported is Anderson et al.'s (1990) patient. She was able to read some words using a whole-word strategy, while not being able to spell or name words to oral spelling, which, according to Benson (1979; Benson & Ardila 1996) are significant features of the third alexia. It is possible that JM would be able to read some words "holistically" if properly motivated, but when shown short "symbolic" words like *TV* or *WC* and prevented from covering up letters, he claimed he

could not see the words clearly and was unable to make any sense of them. He could not even decide whether or not his own name was written on a card, without resorting to his laborious strategy, let alone make lexical decisions about briefly presented words.

On the other hand, JM was able to spell both regular and irregular words out loud, and name them to oral spelling, abilities that, according to Benson & Ardila (1996) are only preserved in pure alexia. Still, Rothi & Heilman's (1981) patient was also able to spell words, both regular and irregular, even though he was impaired in written spelling (i.e. he was not a pure alexic). JM's pattern of performance could be accounted for by an impairment in the "graphemic area" (Rothi & Heilman, 1982), which is important both the identification of letter features, and in programming the movements necessary for writing them.

JM preserved reading and writing of numbers pose some interesting questions. Polk & Farah's (1994; 1995; 1998) findings indicate that a selective impairment in letter identification is possible after damage to extrastriate cortex, but how this could affect writing is not clear. If Anderson et al.'s (1990) coactivation hypothesis should be correct, the pattern of performance displayed by JM and ADD might come about if these patterns of coactivation are different for letters and numbers.

Studying a patient with no localised lesion is tricky. The cognitive neuropsychological study of reading has not yet come so far that one can necessarily infer the neural substrate by analysing the symptoms displayed. With a case like JM, whose symptoms do not map onto an existing type of alexia, there are no definitive clues as to where his lesion might be.

6. Things are not always what they seem.

At this point in the experimental investigation, JM was regularly seeing a clinical psychologist to help him cope with the trauma of the accident. In one session JM submitted to regressive hypnosis, and the therapist found that, being "brought back" to first grade, *JM could read* several pages in his old 1st grade textbook. Still, he claimed (during hypnosis) that he did not understand what he was

reading, and he could not answer simple questions about the text.³⁶ During the next session the therapist also tested his writing, and found JM could write several simple sentences to dictation.

Needless to say, this took us all by surprise. We had considered the possibility of a functional reaction earlier on, and refuted the idea, mostly based on the fact that his pattern of performance had been observed before (Anderson et al., 1990).

6.1. Hypnosis and neuropsychology.

This observation brings about a lot of questions to be answered. First of all: What is hypnosis ? What influence does hypnosis have on brain activation ? What are we really observing when JM is in trance? Could it be the remaining skills of his damaged reading system, or does it imply (beyond reasonable doubt) that his alexia is psychogenic in nature ?

There are reports of classical cases of "hysterical blindness", functional amnesia and sensory disturbances with no apparent physiological cause, but to my knowledge, alexia, (at least in such a pure form) has never been reported as the result of psychological factors or trauma. More than that, we know nothing whatsoever about other alexics' reading performance under hypnosis.

Does the ability to read a 1st grade reading book, presumably consisting mostly of short, highly imaginable high frequency words which are acquired at an early age and possibly "overlearned"), prove that JM is not truly alexic ?

Improved performance in brain damaged patients during hypnosis is not an entirely unknown phenomenon. McKeever et al. (1981) reported a reduction of tactile anomia in a callosotomy patient (CZ) under hypnosis, both with and without age-regression suggestion. In a normal state CZ was virtually unable to name objects palpated with her left hand. She could sometimes write their names with the same hand, though, indicating some right-hemisphere naming abilities. This led the authors to try hypnosis (with and without age-regression) to try to encourage right-hemisphere

³⁶ Having read the sentence: "Soren and Mette were walking in the wood.", JM could not answer the question "Where were Soren and Mette ?"

language skills.³⁷ Their results showed that age regression was not necessary to improve performance. In fact CZ' naming abilities were better in the "no-regression" condition, but her scores during hypnosis were overall better than her normal scores. Her performance also improved when a hypnotic suggestion that she had difficulties speaking, but that she would be able to name the objects placed in her hand, was made. The authors suggested that this might be due to inhibition of the left hemisphere, enabling the right hemisphere to speak, but did not refute the possibility of information being transferred via the intact anterior commissure to the language areas at the left.³⁸

Imagery training during hypnosis has also been reported to improve naming skills in some Broca's aphasics (Thompson et al., 1986), and the activation of non-verbal, non-dominant hemispheric processes was considered as a possible explanation for this finding.

One of the problems with these studies is that it is not at all clear in which way hypnosis contributes to improving the damaged cognitive skills in these patients, mainly because no one quite knows what hypnosis really is or does. Hypnosis is usually defined descriptively as " a social interaction in which one person (...) acts on suggestions from another person (...) for imaginative experiences involving alterations in cognition and voluntary action" (Kihlstrom, 1997). We know that individuals differ in how susceptible they are to hypnosis, and that there might be differences in hemispheric specificity between high and low hypnotisable subjects (Crawford & Gruzelier, 1992). Reviewing the neuropsychological evidence concerning hypnosis, Crawford & Gruzelier (1992; 261) concluded that "these studies suggest greater right-hemispheric involvement during hypnosis in high hypnotisable subjects", and that hypnosis produce left-hemispheric activation (at least in temporal regions) in low hypnotisable subjects.

Could it be then, that when JM is reading during hypnosis he is utilising a different reading process, for example relying on the same mechanisms responsible for covert reading in pure alexia ?

³⁷ This was built on the assumption that language lateralisation may be "less complete, i.e. more bilateral, during childhood than adulthood" (p.179), and that hypnosis increase the use of right hemisphere processes, which might "minimise left hemisphere interference (guessing)" (p.180).

³⁸ McKeever et al. (1981) reported quite a few problems with hypnosis, one of them being controlling the depth of the trance induced, and how to keep it stable across sessions. In one session, CZ did not seem to be in as deep a trance as "usual", and her performance was not significantly improved. In another session, she probably "fell back" into trance after being awoken, increasing her scores in the "no hypnosis" condition. The conclusions are based on averaging the numbers correct from two successful session, the other data being excluded from analysis.

Supposing he is highly hypnotisable, could an increase in right hemisphere activation account for his ability to read the simple material he was presented with ? In light of McKeever et al.'s (1981) findings, this is not entirely inconceivable, but requires further investigation into what skills JM actually shows under hypnosis. If he can only read short, concrete, highly imaginable words, this could indicate right hemisphere involvement.

Looking at it another way, could JM's reading under hypnosis reflect the whole-word reading skills often observed in the third alexia ? According to Benson (1979; Benson & Ardila, 1996), patients with the third alexia are often reluctant to try to read, and will only try (and succeed) with appropriate motivation. With JM being so reliant on his letter-by-letter strategy and covering up of letters, he never gives himself a chance to read words "holistically". Even though formal testing failed to produce any evidence for whole word reading, we might not have tried hard enough. If this is the case, JM should be impaired in letter naming during hypnosis, as patients with the third alexia are (Benson et al. 1971; Benson, 1977).

The fact that JM claims he does not understand what he is reading under hypnosis also poses some interesting questions. First of all, why does he not understand ? As mentioned, he has only read short sentences consisting of words acquired at an early age, which are mostly concrete and highly imaginable. A dual-route interpretation would suggest that he is reading via the sublexical route or utilising the direct lexical route. Either way he cannot access semantics. So far, JM has only been asked questions about the meaning of whole sentences, which is a more demanding task than understanding single words and might rely on more complex cognitive processes than single word reading.

When reading outside hypnosis, in his letter-by-letter fashion, JM understands the words he is reading, so using this strategy he clearly has access to semantics. Could it be, then, that he is able to read via the sublexical or direct lexical route when not under hypnosis, but has adopted a LBL strategy to access semantics. Could the "link" between the input lexicon and the semantic system be damaged, so that he must rely on serial information processing to activate semantic representations ?

6.2. Further investigations.

Fortunately, we got the chance to check some of these hypotheses by examining JM's reading skills while he was under hypnosis.

A trance was induced in JM, and a regressive suggestion that he was back in his 7th year, his first year of school, was given. He was then given his 1st grade textbook and asked to read (the procedure usually adopted by his therapist). JM read very slowly, and his therapist pointed to the words he should read. In an earlier session, JM had tried to read on his own and had had problems following the lines in the book, which is why his therapist was pointing. JM was able to read this text, albeit very slowly. He seemed to read the text word-by-word, though, without any connections between the words making up the sentences.

Since he was reading the book he had actually used when he learned to read, the text might be "overlearned", and his reading it might reflect some kind of an automatic process. We therefore presented JM with texts from two different 1st grade books, which he had never read before.

Text-reading.

The first text consisted of 95 words, and JM made 9 errors, 6 on words repeated in the text (The errors are listed in appendix A). When JM encountered words he could not read, his therapist encouraged him to spell them. JM spelled all words correctly, and pronunciation-errors were made after spelling the words out loud.

The second text was at a more difficult level, but still from a 1st grade book, and consisted of 137 words. JM made 18 errors, 6 on the same words being repeated (App.A). His errors were either regularisations (*kommer* read *komér*) or morphological errors (adding or ignoring suffixes, e.g. *sengen* read *seng*). Some words were not pronounced at all, even though JM could spell them correctly.

Single word reading

JM was also presented with the list of concrete, high-frequency words used in a previous test-session. He read all three-letter words correctly without spelling them out, average time was 2,4 seconds, measured with a hand held stop watch. JM could not read the five-letter words in a glance, but spontaneously spelled them out loud. He correctly identified one word in 10 seconds.³⁹

JM also read 5/5 three-letter non-words correctly with an average time of 9 seconds. 1/5 was pronounced directly, without spelling (out loud).

³⁹ In this test, the therapist was getting worried that JM had been in trance too long, and rushed things a bit, giving JM only 20 seconds per. word to respond. It is possible that JM would have identified the other words if given more time.

Comparing JM's reading of these words with his performance outside hypnosis, he is obviously better when under hypnosis. (See Table 3 for comparison.)

Table 3. Reaction time measures for JM's reading of words and non-words in a normal condition and during hypnosis.

Stimulus	RT - normal	RT – hypnosis	Difference
3 letter words	26"	2,4"	23,6"
5 letter words*	32"	10"	22"
3 letter non-words	16"	9"	7"

* only one word from this list was read correctly during hypnosis;
RT is for this word only. All other times are averaged.

In a normal condition JM read 4/5 of the 5 letter words correctly

Letter identification:

JM was able to identify single letters during hypnosis. When he encountered words he did not recognise instantly, he sounded out each letter before attempting a pronunciation.

Reading comprehension:

JM's therapist had noted that JM could not answer simple questions about the text he read under hypnosis. We wanted to test JM's comprehension of single words, and presented him with the word-picture matching test from BADE. We were not able to persuade JM to read the words silently, as the test-instructions recommend. He spelled them out loud, and then tried to pronounce them. He only succeeded at reading one word out loud, and matched this to the corresponding picture with a great deal of hesitation.

Writing:

During this session, JM only wrote to copy, from a familiar and one unfamiliar text.

When writing on lined paper, JM's writing is neatly formed (See. fig. 6). He seems to be writing with his normal handwriting. Writing on blank paper, the sentences "fall off" towards the end.

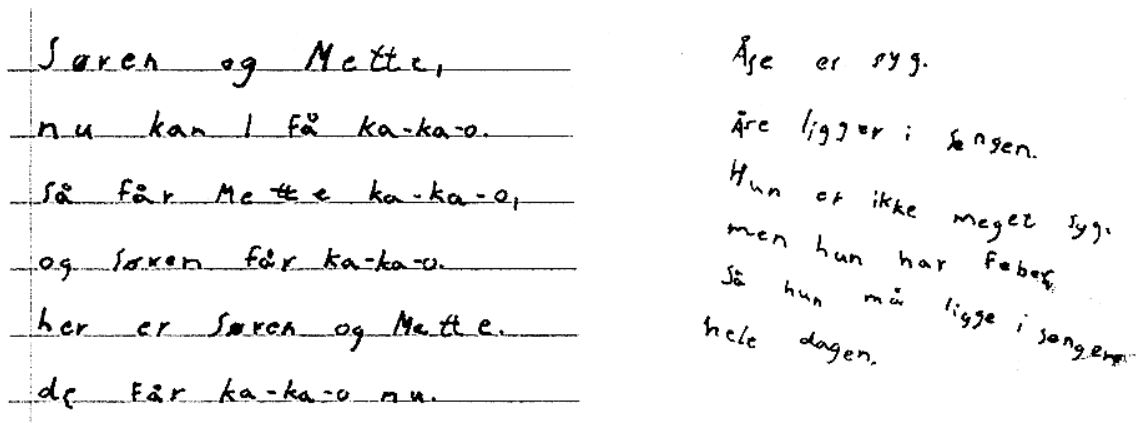
On an earlier occasion (12.01.2000) JM also wrote his name, and a comparison with his writing it outside hypnosis (27.06.99) can be seen below.

[Fig 6. JM's name written during hypnosis. d) Name written outside hypnosis.]

[deleted]

Figure 6.

a) JM's copying from his 1st grade book. b) Copying on blank paper.



Reading without hypnosis:

We presented JM with the 1st grade text after he had been awoken from trance. He looked at it and instantly claimed he could not read it, if not allowed to use his letter-by-letter strategy. When asked to try anyway, he commented that the text was written in lower case letters, which he had great difficulties identifying. He could not be persuaded to read any of the text.⁴⁰

Summary of test-results:

JM was tested while being in hypnotic trance. It was suggested to him that he was back in the 1st grade in school.

In this state, JM could read his own old 1st grade text-book. He pronounced all words correctly without spelling them out loud, but with severely slowed reaction times.

When presented with 1st grade material unfamiliar to him, JM made more errors, most of which were regularisation-errors and morphological errors. When he encountered unfamiliar words, he was encouraged to spell them out loud. Even with this strategy JM failed to pronounce some words. Formal testing of reading comprehension failed.

JM is able to identify single letters when under hypnosis.

When presented with the same material for reading outside hypnosis, JM could not read it.

Comparison of JM's RTs with and without hypnosis on a list of concrete, high-frequency words and non-words reveals that he reads much faster during hypnosis. His reading speed is far from normal, though. He takes an average of 2,4 seconds to read three letter words, and 9 seconds to read the corresponding non-words.

JM can write to copy when under hypnosis, and he is also able to write words and sentences to dictation and from memory. He seems to be using his regular handwriting.

6.3. Case JM - reconsidered.

JM's reading during hypnosis is very different from the pattern we have observed under normal conditions. He is able to read without covering up letters, and he identifies most words without spelling them out loud. He also writes normally. A direct comparison between conditions on a list of concrete, high frequency words and matched non-words, revealed that JM's reaction times were much faster under hypnosis. The question is: Is JM reading in a normal manner during hypnosis ?

JM made quite a few errors when presented with unfamiliar material under hypnosis, indicating that he is not reading normally. His whole-word reading was slow enough to permit letter-by-letter reading. When JM encountered words he could not read, he would spell them out loud, and attempt a pronunciation. In several instances this pronunciation was incorrect. Most of his errors were regularisations, and he also made some morphological errors.

During hypnosis, JM was able to identify single letters, a pattern not consistent with the third alexia, where letter identification is severely impaired and words are read in a gestalt manner or not at all (Benson, 1977; Anderson et al., 1990). He also read some verbs, adjectives and function words correctly. This indicates that JM is not reading via the right hemisphere systems, as this system is believed to know short, concrete words only (Coltheart, 1980b).

JM is also able to write when under hypnosis. Together with reading with very long reaction times, this is the pattern observed in pure alexia. Still, if patients with pure alexia correctly identify all letters in a word, they will also pronounce it correctly. JM on the other hand, mispronounced words after having identified all the constituent letters, in a manner similar to surface alexics.

Regularisation errors are the hallmark feature of surface alexia, and letter-by-letter reading has been observed in this syndrome (Friedman & Hadley, 1992). Could it be, then, that JM is not suffering from neither pure alexia, nor the third alexia, but "letter-by-letter surface alexia" ? Patterson & Kay (1982) reported two patients who read letter-by-letter, and made regularisation errors even when all letters of a words were correctly identified (e.g. *city* - c-i-t-y - *kitty*). They suggested that there are

⁴⁰ Due to the time constraints, we were not able to discuss this further or persuade JM to try to read the text.

actually two types of letter-by-letter reading, Type 1 and Type 2, and that regularisation errors are common in Type 2. These letter-by-letter surface alexics also showed signs of surface agraphia, that is, they also made regularisation errors in writing. One patient was also impaired in single letter identification. The same pattern of performance has been reported in other LBL-readers (Friedman & Hadley, 1992; Hanley & Kay, 1992, 1996).

JM's reading during hypnosis closely resembles that observed in Patterson & Kay's (1982) Type 2 letter-by-letter readers. Unfortunately, his writing has not been studied experimentally under hypnosis, so we do not know if he would also show signs of surface agraphia.

Does JM's reading during hypnosis reflect his remaining reading skills, or are we simply observing the reading pattern of a normal 7-year old ? Regularisation errors are common in children learning to read, and because their sight-vocabulary is yet to be developed, they often resort to spelling words out loud. Marcel (1980) compared children's reading errors to those of surface alexic patients, and found that they were quite similar. He suggested that while children have not developed representations of many words in the input lexicon, surface alexics have lost many of these representations. This leads both groups to attach sound to word fragments, which frequently results in regularisation errors

JM's therapist has also tried hypnosis without regressive suggestions in JM. When presented with a newspaper in this state, he had severe problems in reading the text. Unfortunately we have not been able to formally test JM's performance in this condition, and do not know if his reading errors are similar to the ones he makes when given a regressive suggestion.

Drawing any conclusions about the deficit causing JM's problems in reading on the basis of performance during hypnosis seems premature. Still, there are two main factors of JM's reading during hypnosis indicating that he is not simply making the mistakes of a normal seven-year old. First, he does not seem to understand what he is reading, and is not able to answer simple questions about the text he is reading. Secondly, his handwriting during hypnosis does not seem to be that of a seven-year old, suggesting that some capacities acquired later is influencing his performance even when he is given regressive suggestions. If this is the case, he should be able to read in a normal

manner. Even though we were not able to formally test JM during hypnosis without regression, the observations made by JM's therapist indicate that JM is not reading normally in this condition.

Even though the symptoms seem to differ, JM is impaired in reading when in a normal state and during hypnosis. On this basis, it seems fair to conclude that JM is truly alexic, and that his reading problems do not reflect a psychogenic deficit.

7. Patterns of paralexia.

Even in the light of models of normal reading, and the different alexias reviewed in Part I, it is not quite clear what causes JM's impairment in reading. Normally he identifies the letters of a word one by one before naming it. During hypnosis, his naming latencies are extremely long, a pattern that could be caused by serial identification of letters. We have not demonstrated a linear relationship between the number of letters in a word and the time taken by JM to read it, though, and his slowed responses under hypnosis might be due to a deficit occurring after letter processing. Or, his reading during hypnosis might just reflect his reading capacity as a seven year old.

Even though we were unable to investigate it thoroughly, it seems that JM does not understand what he is reading during hypnosis. He was not able to answer simple questions about a text read during hypnosis, which a normal 7-year old would probably be able to do. In a normal state, he is able to understand a word insofar as he can read it. This might indicate that mechanisms we do not quite understand are at work during hypnosis, enabling JM to read words out loud, but not automatically access semantics. It would have been interesting to see if he would be able to match spoken words to pictures, or if he, when given enough time, would be able to match written words to pictures.

His preserved reading and writing of numbers, as opposed to letters, is very similar to the pattern of impairment observed by Anderson et al. (1990), suggesting that he might suffer from "the third alexia in pure form". His oral spelling is intact, as is his naming to oral spelling, which is not consistent with the impairments observed in the third alexia (Benson, 1977). His number reading skills, on the other hand, are not easily explained in terms of pure alexia, where number reading is usually impaired (Cohen & Dehane, 1995).

8. Concluding comments.

Even though we have studied JM's deficit in reading quite intensively, we have not been able to classify his alexia. All the features of his impairments have been reported before, both his preserved spelling abilities and impaired writing, and the dissociation between numbers and letters in reading and writing. Still, the results of the experimental investigation does not leave us with a coherent picture matching a clinical entity. Even though this case study has not produced many answers, it may bring attention to some important questions to be asked in the cognitive neuropsychological study of reading. More research is needed on number reading in alexia, and on the connections between reading and writing in normal and impaired individuals. Cognitive models of the processes involved also needs to be developed.

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Appendix A.

Text 1. Pia og Peter for 1. klasse. Kund Hermansen. København: G.E.C.Gads Forlag; p1-4.

Text 2. Læsebog for 1. skoleår. København: Gyldendal, 1966; p.30.

Error-corpus:

Word	Frequency, pr. mill	Word-class	Pronunciation	Error-type
Text 1.				
siger (4)	1580	verb	si-gér	regularisation
mine	500	pronoun	min	morphological (ignoring suffix)
biler (2)	100	noun, concrete *	bil-ér	regularisation
leget	<10	verb	-	-
Pias	-	Name, genitive	Pjas	regularisation
Text 2.				
ligger	370	verb	li-gér	regularisation
sengen (2)	130	noun, concrete	seng	morphological (ignoring suffix)
meget	1910	adjective	mee-geet	regularisation
ligge	90	verb	li-gér	regularisation + morphological (adding suffix)
dagen	190	noun	-	-
kommer	970	verb	ko-mér	regularisation
komme	790	verb	ko-mér	regularisation + morphological (adding suffix)
feber	<10	noun	-	-
smitte (2)	<10	verb	-	-
gåde (2)	<10	noun	gå-dé	regularisation
langt	440	adverb	-	-
fortælle	230	verb	-	-
hellere	120	adverb	-	-
skibet	20+	noun, concrete*	ski-bét	regularisation
begynder	140	verb	-	-

Frequency ratings from Bergenholtz, (1992)

* - listed by Paivio et al. (1968)