Chapter 3

Brain-Based and Non-Brain-Based Models of Language

John Morton

We have a number of lessons to learn from history. If we are lucky we can avoid making the same mistakes as thinkers in the past. The purpose of this chapter is to show the relationship between the work of the "diagram makers" of the late nineteenth century and current information-processing theory in cognitive psychology. The lesson to be learned is that the work of the early theorists was ignored because of a confusion of objectives. Specifically, what was confused was the objective of representing the elements of processing in the brain and the objective of establishing the localizability of these elements.

Let me start with a quotation from an otherwise thoughtful (i.e., approving) review of the recent book *Deep Dyslexia* (Coltheart, Patterson, and Marshall, eds.; London: Routledge & Kegan Paul, 1980): "Briefly, with regard to the other chapters: Morton and Patterson's review paper on the logogen model provides a theoretical framework for much of the clinical data. Though I would defer comment on the model to those more knowledgeable in this area, I confess to some antipathy to its general style, the discussion of 'routes' between components which have no known neural correlates." (Brown 1981, p. 389) This comment can be countered by one in which a writer states that his objections to current theorizing are "intimately related to the idea of 'localization,' i.e. of the restriction of nervous functions to anatomically definable areas, which pervade the whole of recent neuropathology." (Freud 1906, p. 1).

I do not intend to discuss the issue of localization. This issue is irrelevant to my concerns, which are to characterize the operations of the normal brain and to describe the effects of brain damage in individual patients in terms of the resulting psychological model. These are not the only tasks that face us, however. There follows the question of how the elements of the model are implemented in the brain. There is also

in 'Biological Perspective in Language'.
in. D. Caplan, A.R. Lecours & A. Smith (Eds)
Cambridge, Mass: MIT Press, pp. 40-64.

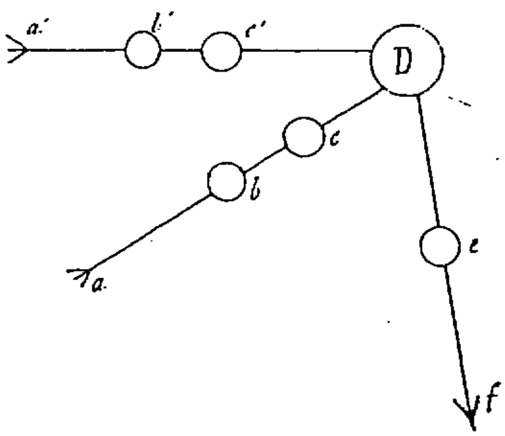
the question of what functions are performed in particular anatomical locations. I believe these are important questions, but that the answers do not affect the form or the nature of the psychological model. The case for such distinctions has been made recently by Caplan (1981) and Marshall (1980), and I will not argue it here in detail. Rather, I will show some relationships between the work of 100 years ago and present work in the hope of illustrating what has been lost through the conceptual confusion.

On Diagrams

The problem for someone with a complex idea is to represent the idea. The form of the representation is the key to communication. My own theories, which focus on the flow of information, have always been represented in the form of diagrams resembling flow charts. I find diagrams an aid to thinking, but I have come to learn that many people cannot work with them. One reason for this may lie in a distinction between visual thinkers and verbal thinkers. Another, deeper reason relates to whether one wishes to discuss behavior or the mechanisms underlying behavior. It is the latter that the diagrams portray, and behavior may be related to diagrams in complex ways.

In the field of neuropsychology, the equivalent dichotomy is between a psychological deficit and a functional deficit (which may be localized; see Caplan 1981). The nineteenth-century diagram makers were often confused between these two because they believed in localization of function. However, I am going to ignore this aspect of their thinking and compare a number of the diagrams as if they were simply information-processing models. Some of the models were arrived at purely *a priori* and then applied by their makers to brain-damaged patients; other models resulted from careful clinical analyses of large numbers of patients. However, in the comparisons that follow I will be concerned only with the topological aspects of the models.

What I will try to do is reduce all the models to a common form. In doing so I will make one simplification and take one liberty. The simplification is to extract those aspects of the models that are concerned with the processing of single words: reading, oral comprehension, repetition, and speech. Writing will be ignored, partly because not all the diagram makers considered it and partly because current theory has less to say about the distinction between speech and writing than about the other distinctions (but see Newcombe and Marshall 1980). The



Baginski - 1871 (from Moutier, 1908, p.34). D - principle center for the construction of ideas. a - ends of auditory nerve. b - center of acoustic perception. c - center of elaboration of thought based on sound (Centrum des Klang-gedachtnisses). a'b'c' - analogous centers for vision. e - center of coordination of movement. f - motor pathways. (Moutier, 1908, adds the note that this was the first "Schema" ever traced.)

Figure 3.1

liberty I will take concerns the nature of the elements in the models. The models share the convention of diagramming "centers" of various kinds, joined together by pathways. In general, the centers are either modality-specific (that is, concerned exclusively with auditory or visual stimuli or with speech) or modality-nonspecific (such as a "center of ideas"). The different theorists ascribed varying amounts and kinds of processing to, say, the auditory center. Such distinctions will be ignored in the interest of making the comparisons among the different models easier to follow.

Many of the versions of the diagrams that follow have been taken from Moutier 1908. A few errors in this source have been spotted; no doubt many more will have been unnoticed.

The Diagram Makers

The earliest model, according to Moutier, was that of Baginski (1871). This is shown in figure 3.1. It makes a distinction between two levels of sensory analysis, "perception" and "elaboration." The model is equivalent to that illustrated in figure 3.2, in which the modality-specific functions have been collapsed into single functions. One implication of this model is that reading can proceed directly from vision. This is in contrast with other nineteenth-century (and twentieth-century) models that require that print be turned into either an acoustic or an articulatory form before it can be understood. This model is also formally equivalent to that of Langdon (1898) (figure 3.3), which distinguishes between writing, speech, and "mimicry" (that is, speech repetition without the

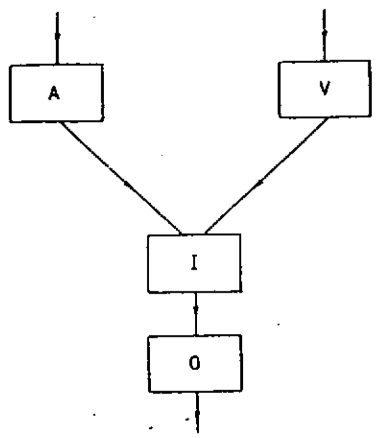


Figure 3.2

A diagram equivalent to those of Baginski (Fig. 3.1), Langdon (Fig. 3.3.) and Moeli (Fig. 3.4). A - auditory specific analysis (for speech). V - visual analysis (for words). I - modality independent analysis ("ideas"), O - speech output processes.

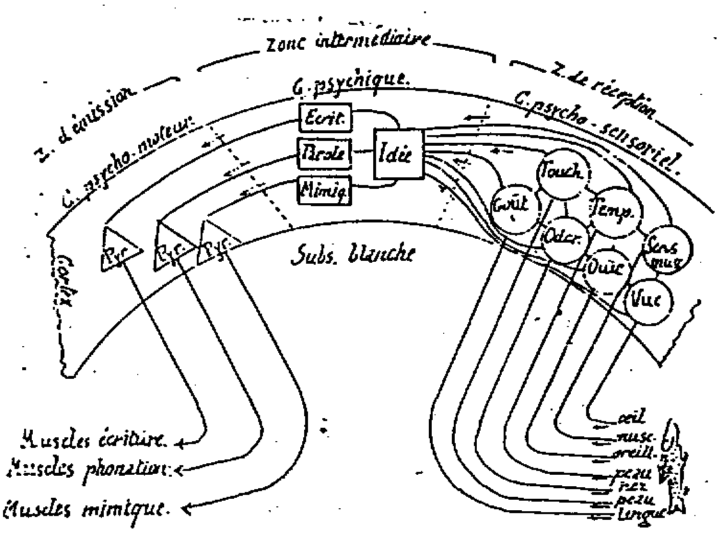


Figure 3.3

Langdon's model (from Moutier, 1908, p.59: who took it from Hughes, 1903).

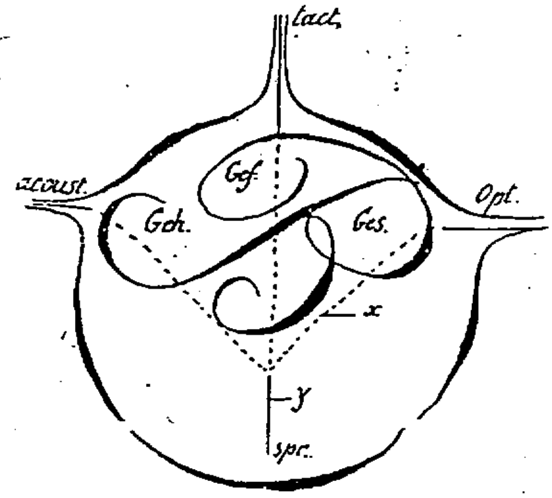


Figure 3.4

Moeli (1891) - from Moutier (1908, p.53). B - ideational center. Spr(Sprache) - elaboration of speech. Acust, tact, oft - sensory pathways. Geh, gef, ges - cerebral centers for hearing, touch and sight. x - shows the localization of a lesion supposed to affect the visual aspects of language. y - a lesion giving global aphasia ('l'aphasie totale').

of sensory distinctions. Despite the anatomical flavor, the diagram conveys the same idea as Baginski's. This is also true of Moeli's (1891) model (figure 3.4), which further indicates the lesions leading to a couple of disconnection syndromes.

A slightly more elaborate model put forward by Ballet (1886) (figure 3.5) allowed for a direct connection between modality-specific input processes and modality-specific output processes. Thus, reading aloud from V (the visual center) to P (the speech center) could go either via the "center of the intellect" or directly via the pathway numbered 6 in the diagram. The same is true of Grasset's (1896) model (figure 3.6), in which the connections among all the modality-specific centers are direction-free.

In essence, Ballet's model is topologically identical to a simplified version of the current logogen model (Morton 1979). In the version of this model shown in figure 3.7, the direct pathways between input and output represent a lexical level. These paths could not be used to read or repeat nonwords. In the full version of the model there are also input-output paths that are nonlexical to allow for the pronunciation of new words, names, or nonwords. It is not clear whether lexical or nonlexical processes were intended by Ballet or Grasset. However, for our present purposes, this distinction is not vital.

A very different concept is represented by Kussmaul's (1877) model (figure 3.8). Here, the auditory center (*B*) is involved in speech (by the route *cbd*) and the visual (reading) center (*B'*) is involved in writing

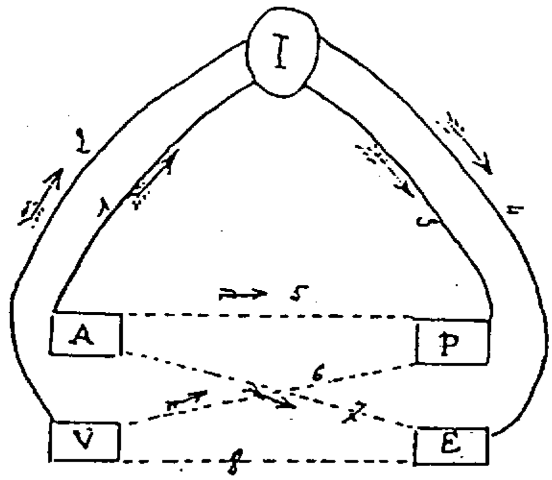


Figure 3.5

Ballet (1886) - from Moutier (1908, p.47). I - center of the intellect. A - auditory center. V - visual center. P - speech. E - writing.

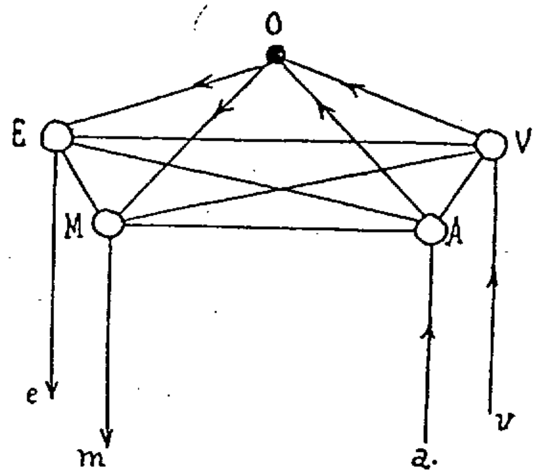


Figure 3.6

Grasset (1896) - from Moutier (1908, p.51). O - ideational center. A - auditory center. V - visual center. M - verbal motor center. E - verbal graphic center.

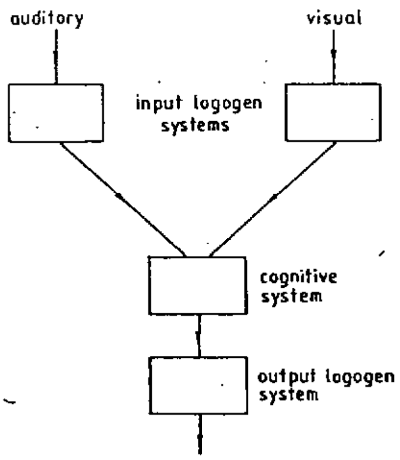


Figure 3.7 A simplified version of the Logogen Model (Morton, 1979).

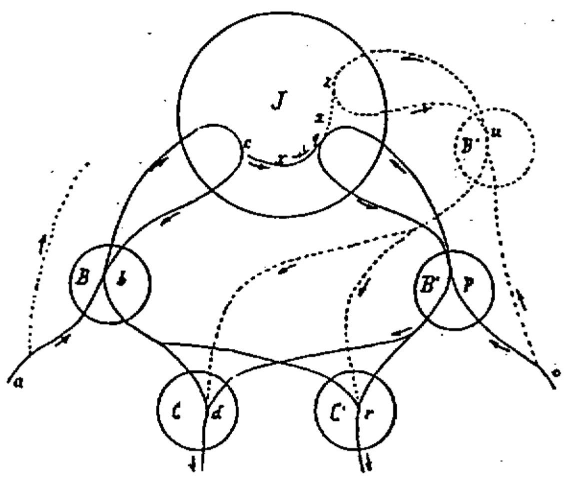
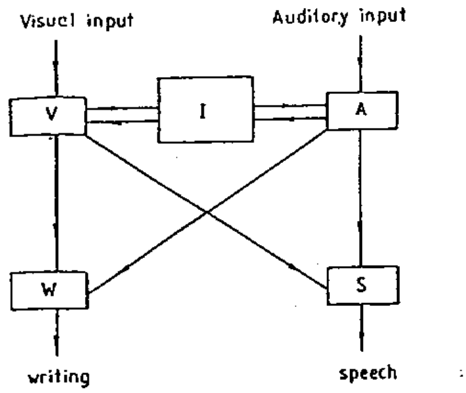


Figure 3.8

Kussmaul (1876) from Moutier (1908, p.41). a - center of ideas. B - auditory center. B' - visual center. B" - center used by the deaf for lipreading and signing. C - speech center. C" - writing center.



The topological equivalent of Fig. 3.8, ignoring centers B" and C". A, V, I and S represent acoustic, visual, ideas and speech centers respectively.

Figure 3.9

(by the pathway *qpr*). The model also allows for repetition without understanding (pathway *abd*) and reading without understanding (pathway *opd*). Kussmaul also indicated that he believed that the deaf operate in essentially the same way as the hearing. In figure 3.9 I have translated Kussmaul's model into the notation used in figure 3.2, leaving out the writing center and the special pathways for the deaf. Suggestions similar to this, in the sense of having input and output functions with shared components, have been put forward recently by Allport and Funnell (1981). Seymour's (1973) model for the processing of words and pictures (figure 3.10) is based on the same principle. As figure 3.11 shows, the same principle is a feature of the earlier versions of the logogen model (Morton 1969). One extra feature of the logogen model was that auditory and visual inputs relevant to language shared the same process. This model gave rise to predictions that were not upheld by data (Clarke and Morton 1983; Morton 1979), and it has been superseded by the model illustrated in figure 3.7.

A variant on the same theme was put forward by Charcot (1883) (figure 3.12). The difference here is that speech is possible without involving the auditory center (IC to CLA), though it is clear that the pathway between CAM and IC is bidirectional. The model thus shares features of the models illustrated in figures 3.2 and 3.9.

The two sets of models discussed above differed as to whether input and output functions were completely separate or shared some components. They shared the feature that the auditory and visual systems were independent of one another. In the three models that follow this is not the case. Rather, the recognition of print depends on mediating

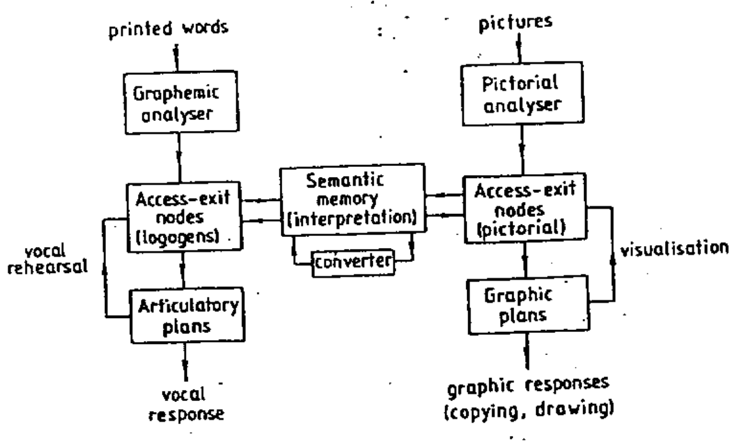


Figure 3.10

Seymour's (1973) model for the processing of verbal and pictorial stimuli.

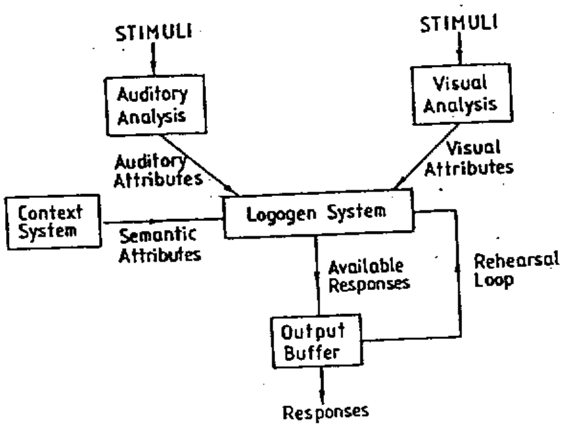
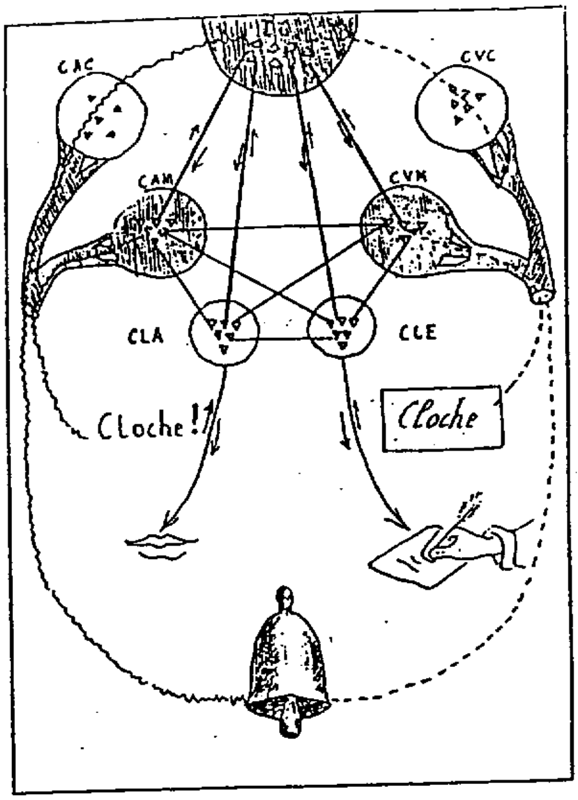


Figure 3.11

The early version of the logogen model (Morton, 1969).
This has been superceded by the version shown in Figure 3.7.



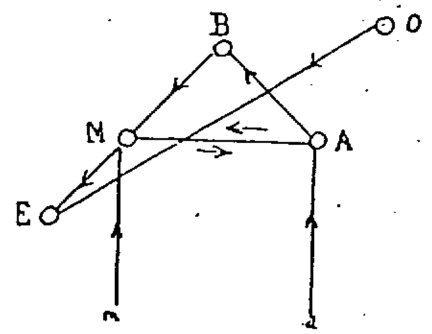
Charcot (1883) from Moutier (1908, p.46).
IC - ideational center.
CAM - auditory center for words. CLA - center for spoken language. CVC - common visual center.
CVM - visual center for words. CLE - writing center. CAC - common auditory center.

Figure 3.12

acoustic processes. The most straightforward and the best-known of these models is that of Lichtheim (1885), illustrated in figure 3.13. Here, print can be recognized only after being recoded into an auditory form via the center A. Elder (1897) and Mills (1898) produced models that included the same idea, as shown in figures 3.14 and 3.15 respectively. Both these authors added a few details on the involvement of the two hemispheres in reception, and Elder considered their involvement in production.

What Went Wrong?

From this selection of diagrams (Moutier includes ten or so more) it is clear that a great deal of effort went into the attempts to formalize a representation of brain processes. It might seem strange that proper contrasts were not made among the models and that proper tests were not made of the various alternatives, but the debate, in general, took



Lichtheim (1885)
a - auditory input. A - auditory center. B - concept center.
O - visual center. E - writing center. M - speech-motory center.
m - speech output.

Figure 3.13

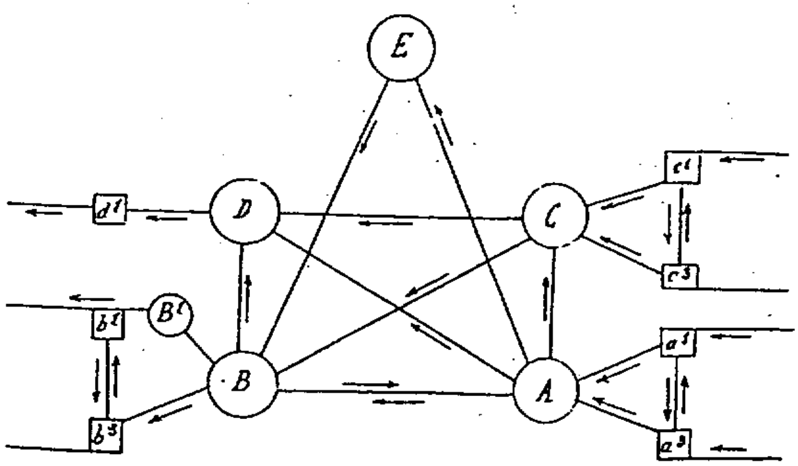


Figure 3.14
Elder (1897-from Moutier (1908, p.51). A - auditory-verbal center. B - psycho-motor center. C - visuo-verbal center. D - writing center. E - ideo-motor center.

other forms. There were exceptions. Lichtheim (1885) specifically discusses how his theory might be falsified and what theoretical resources he had available (for example, postulating multiple lesions). Wernicke (1874) also had clear ideas about the way to proceed:

My conception differs from earlier ones in its consistently maintained anatomical foundation. Previous theories postulated theoretically different centers (a coordination center, a concept center, etc.), but paid no attention to anatomy in doing so, for the reason that the functions of the brain, completely unknown at that time, did not yet justify anatomical conclusions. It is a significantly different approach to undertake a thoroughgoing study of neuroanatomy and, making use of the now almost universally accepted principles of experimental psychology, to transform the anatomical data into psychological form and to construct a theory out of such material. (Wernicke 1874, p. 28; translation from Marshall 1982)

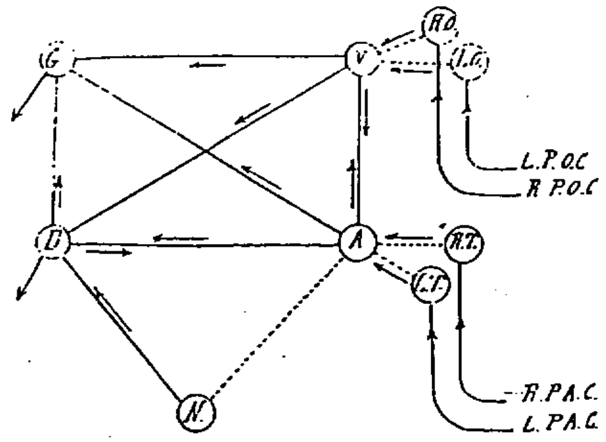


Figure 3.15

Mills (1818) from
Moutier (1908, p.52).
·A·- auditory center.
V - visual center.
B - Broca's area;
G w.·writing center.

However, the purely psychological debate was minimal, and the diagrams foundered for a complex variety of reasons, most of which had to do with the relations supposed to hold between the models and the brain. The data used in the construction of the models were from patients with brain lesions. It was found that, roughly speaking, patients with lesions in the same region of the brain showed the same pattern of symptoms, or syndrome. This proper association of lesion with syndrome gradually changed until lesions were being associated either with "centers" of various kinds or with disconnections between centers. This in turn led to two major objections.

The first objection was that pathways were drawn that had no known anatomical correlates. Freud (1906, pp. 8–9) says: "Lichtheim's scheme . . . postulates new tracts, the knowledge of which is still lacking. If Lichtheim's presentation was based on new anatomical findings any further opposition would be impossible." The contrast Freud was making was with Wernicke's schema, which could be "inscribed into the brain, as the localization of the centers and fiber tracts which it contains has been anatomically verified." (Freud 1906, p. 8) In his final discussion, Freud concludes that "the apparatus of speech . . . presented itself to us as a continuous cortical area in the left hemisphere" extending between the sensory and motor areas (Freud 1906, pp. 102–103). Freud's criticism of Lichtheim, then, was effectively that, although his model was an improvement on Wernicke's, the format required localization if it was to be supported. This seems to do Lichtheim an injustice, as Lichtheim's article indicates that he was trying to separate function from anatomy. Freud also criticized Lichtheim on the grounds that patients did not conform to the predictions of the model (especially in the case of conduction aphasia). However, he failed to produce another model of the same form and failed to produce a different formalism. Head (1926) had similar problems. He comments that Kussmaul's figure (3.8 above) "lacked that definite localization or centers and paths demanded by the popular taste" (p. 64). Head goes on to discuss the "diagram makers" in general: "For every mental act there was a neural element, either identical with it or in exact correspondence. . . . They failed to appreciate that the logical formulae of the intellect do not correspond absolutely to physical events. . . ." (p. 65). The strong anti-localizationist arguments of Freud, Head, and others were sufficient to kill the notion of "centers," and with it the diagrams. Head also had fairly strong views about visual representations of ideas. He says that Kussmaul "unfortunately . . . was seduced into constructing a diagram." Because of this, and because he was interested in the effects of lesions in different parts of the brain, he missed the point that functional models (such as the "logical formulae of the intellect") could be studied independent of the brain.

The second problem with the diagrams lay in the attempt to treat each patient as representing the results of a single lesion. There were created idealized syndromes that were supposed to be related to single lesions, such as Broca's aphasia, Wernicke's aphasia, and conduction aphasia. In fact, these syndromes probably have no single clear exemplars in the whole history of neuropsychology. Attempts to circumvent the problems led to considerable contortions in the use of the models. Lichtheim (1885, p. 465) saw this problem clearly and discussed the need to consider the effects of multiple deficits. Head ignored this sophistication in his attack on Lichtheim, and Freud complained that such freedom "opens the doors to arbitrary explanations."

A third problem was that the psychological concepts were inadequate for the purpose. Some consequences of this are discussed by Arbib, Caplan, and Marshall (1982). It is a pity that there was not more interaction between experimental psychologists and neurologists at the time of the diagram makers. The legitimate rules for diagram making were often broken in the attempt to account in more detail for the symptoms of individual patients. There was no notion of testing normal people in order to get an independent justification for new "centers."

Some of these issues are discussed at greater length in Arbib et al. 1982, Caplan 1981, and Marshall 1980, 1982

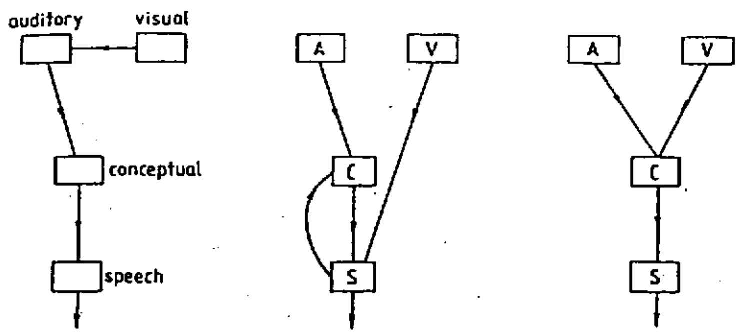


Figure 3.16 Simplified diagrammatic representation of alternative ways of processing visual-verbal inputs.. a) via an auditory code, b) via an articulatory code, c) directly to a conceptual (or semantic) representation. It is assumed that we are concerned with lexical representations and that there is only one such representation for each type of code. (In practice we will expect some combination of all three to be correct.)

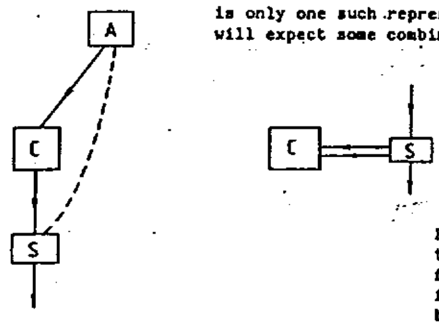


Figure 3.17 Representation of the alternative answers to the question of whether input and output speech functions share some processes. a) with independent input and output processes, b) with shared processes. A, C and S refer to auditory, conceptual and speech processes.

A Hundred Years Later

The major differences among the models illustrated above are easy to summarize.

• When we understand printed or written words, do we have to pass through an acoustic representation (figure 3.16, left) or an articulatory form (figure 3.16, center), or can there be "direct" access from a visual or graphemic representation to a "semantic" or "idea" representation (figure 3.16, right)? These diagrams are the simplest forms of the alternatives. None of the models considered above suggested that visual information has to pass through articulatory information, but such an idea was put forward by Jackson (1868) and others.

• Are there elements in common between the processes of auditory comprehension and speaking (and between reading and writing)? The alternatives here are illustrated in figure 3.17, with the left diagram representing independence and the right diagram representing over-

• What are the natures of the input and output processes in the models? In fact, the answers to the two questions above are contingent on the definitions of the component processes. The main options have to do with whether a lexical representation has been reached. Thus, it is clear in some of the diagrams that, when a direct connection was indicated between input and output, the originator of the model was referring to imitation or copying (in the case of print or script). This could apply equally to linguistic and nonlinguistic stimuli. In other cases the reference was to the processing of words.

One breakthrough in contemporary work that has extended the distinctions among processes has been the careful distinction between words and nonwords. The early clinical work rarely used "nonsense" stimuli, except when patients were asked to read or repeat words from an unfamiliar foreign language. In experimental psychology, until around the early 1970s it was assumed that nonword stimuli could tell us about the way real words were processed (Morton 1979). Now it is clear that distinctions have to be made in talking about the processing of words and nonwords, including the obvious one that nonwords have (by definition) no "semantic" representation. In establishing this distinction we also go some way toward answering the first question, as we will see.

Is There Direct Comprehension of Printed Words?

There are two lines of evidence on this topic, which I will merely sketch. The first line comes from research on normal subjects. The experimental paradigm involves giving subjects a "priming" task (having the subjects make some response to a set of priming words or other stimuli) and then, between 5 and 30 minutes later, testing the effect of the priming task on the identification of these and other words. In the visual modality, the identification task involves presenting words very briefly and the measure is the amount of time the stimuli must be presented for correct identification or the percent correct at a particular exposure duration. With auditory presentation, words are presented in noise and the usual measure is the percent correct at a particular signal-to-noise ratio. It is well established that, when the same stimulus is presented in the two tasks, performance in the identification task is enhanced. The experiments then involve varying the relationship between the stimuli in the two tasks.

By looking at the resulting facilitation, we can draw conclusions about the degree of overlap in the way different stimuli are processed. First, we can examine the level of representation at which these facilitation effects take place. It is clear that it is not a sensory level. Clarke and Morton (1983) showed that when the priming task involved reading either printed words or handwritten words the facilitation of subsequent recognition of printed words was equivalent. With auditory stimuli, Jackson and Morton (1983) looked at the effects of the voice of the speaker. In this experiment the pretraining words were spoken clearly in either a male or a female voice and the test words were in the female voice. The data showed equivalent transfer in the two cases. These data show that the facilitation effect is not sensory. Other data show that it is modality-specific. Clarke and Morton (1983) could only find a small amount of facilitation from initial auditory presentation to subsequent visual recognition, whereas within-modality facilitation is great. Jackson and Morton (1983) found only a little transfer from a visual priming task to auditory recognition. Further, Gipson (in preparation) and Ellis (1982) have reported experiments in which there was significant within-modality transfer but no between-modality transfer at all. In all these cases the priming task involved some semantic processing. Thus, we deduce in answer to the first question above that there are direct routes from both the auditory and visual systems to the comprehension systems such that when one is used there is no necessary involvement of the other.

Further experiments have shown that the facilitation effect is at the level of the morpheme, not that of the word. Murrell and Morton (1974) showed that the morphemic transfer from *SEEN* in the priming task to *SEES* in the recognition task was as great as that from *SEES* to *SEES*. However, there was no transfer at all when the priming word was *SEED*. With auditory stimuli, Kempley and Morton (1982) report similar findings for pairs of words with regular inflections. However, they found no transfer at all between irregularly related pairs of words such as *man/men* and *lost/loses*. Also, they reported no effects of auditory similarity.

These experiments allow us to isolate the facilitation effect at the lexical level. The importance of this is that it helps us to begin to answer the second question, that of the common elements between input and output processes. The crucial test is whether speaking a word affects subsequent auditory recognition. On the basis of the current data we cannot decide between the options in figure 3.18. The first of these

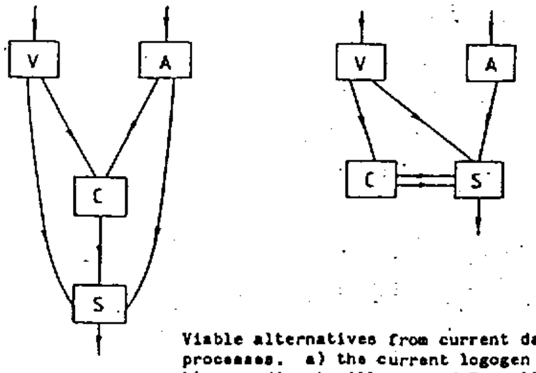


Figure 3.18 Viable alternatives from current data as to the connectedness of various processes. a) the current logogen model (c.f. Figs. 3.7 and 3.1a), b) according to Allport and Funnell (1981). V, A, C and S refer to visual, auditory, conceptual and speech processes.

options represents the current logogen model (as in figure 3.7), whereas the second maintains the idea that there are processes in common between listening and speaking. This latter idea (though not the form of the model) is supported by Allport and Funnell (1981). Until the nature of these processes is specified more fully, it will not be possible to test between the alternatives.

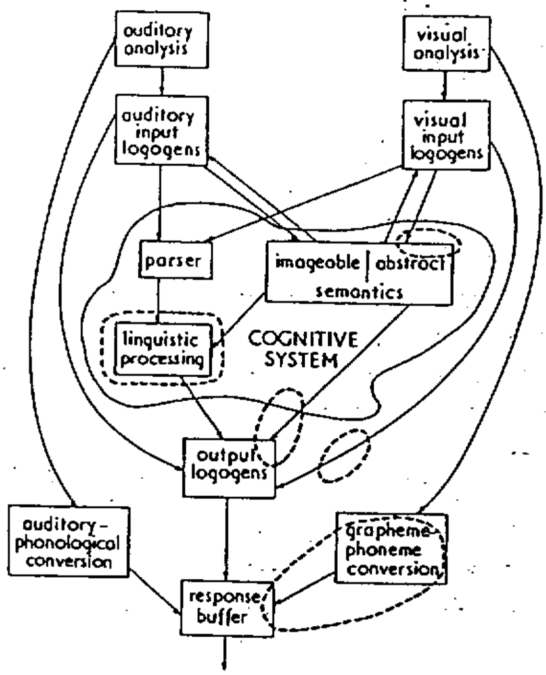
Neuropsychological Evidence

Diagrams of the processes involved in the processing of verbal and pictorial stimuli are currently being used, particularly in Europe, by a number of neuropsychologists and cognitive psychologists interested in the effects of brain damage. (See, for example, Allport and Funnell 1981; Coltheart 1980a, 1980b, 1981; Marcel 1980; Morton 1981; Morton and Patterson 1980a; Marshall and Newcombe 1973; Newcombe and Marshall 1980; Ratcliff and Newcombe 1982; Patterson 1981; and Shallice 1981.) The number of scientists referring to the models or the concepts underlying the models is much greater. These authors share a belief in the utility, if not the necessity, of using single case studies and studying each patient in considerable detail with particular psychological or linguistic theories in mind. The detailed arguments in favor of single case studies are complex, and the dangers are reasonably well known (Shallice 1980). However, the dangers of using group studies, unless the groups are particularly well controlled, are even more serious if one is interested in the nature of normal psychological processes and how they can break down. The crucial distinction is between neurological and psychological defects (Beauvois 1982). It is possible to have a single neurological deficit (such as a reasonably well-localized lesion) with a number of well-defined and psychologically unrelated behavioral deficits. (See, for example, Beauvois, Derouesne, and Saillant 1980.) Notions of "typicality" of types of aphasia and dyslexia seem unimportant when the task of the neuropsychologist is no longer that of identifying the site of a lesion. Thus, it now seems essential to specify whether a patient is agrammatic, or whether receptive, productive or both, before conducting experiments on the processing of language. The mere designation of Broca's aphasia or "nonfluent" or "left anterior lesion" does not enable us to interpret the data. Marshall (1982) and Saffran (1982) have elaborated arguments along these lines. Saffran concludes (p. 333) that "if the classical syndrome categories—or worse, dichotomies such as 'anterior' vs. 'posterior'—continue to be the prevalent units of analysis in aphasia research, the contribution of neuropsychological data to the componential analysis of language will be seriously limited."

Let me say again that if the question is changed the appropriate methodology changes. If the question relates to the functions subsumed by a particular portion of the brain, then of course the location of the lesion is crucial information. If the question relates to recovery patterns, then the classical divisions, or some modification of them, will be vital for helping to characterize the patient groups.

However, the group designations, classical or modern, are of necessity fuzzy. Not all patients in a group share all the symptoms, and patients should thus be selected specifically on the basis of deficits related to the linguistic or psychological variables under investigation. (See, for example, Friederici, Schoenle, and Goodglass 1981 and Caplan, Matthei, and Gigley 1981.) In the same way, we now know that the classical symptoms of the conduction aphasic—problems in repeating single words and memory problems with lists of words or sentences—can be dissociated and so must be distinguished (Shallice and Warrington 1977).

Patients can be validly classified only when variations between patients are less than variations between groups. (See for example, Patterson 1981.) However, even this criterion is subject to restriction by the type of behavior studied. Thus, patient P. W. (Patterson 1978, 1979; Patterson and Marcel 1977; Morton and Patterson 1980a,b) is termed a deep dyslexic, but this designation is only in contrast with phonological dyslexia, letter-by-letter dyslexia, and surface dyslexia (Patterson 1981). That P. W. is agrammatic and nonfluent is irrelevant to his classification as a deep dyslexic, since other deep dyslexics exist who are neither agrammatic nor nonfluent (Coltheart 1980b; David Howard, personal

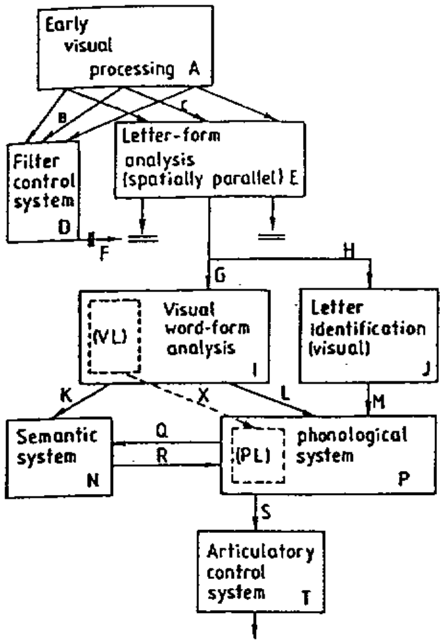


An expanded version of the logogen model, from Morton and Patterson (1980a).
On this diagram are indicated the points of breakdown required to account for the performance of a particular deep dyslexic patient.

Figure 3.19

communication). Morton and Patterson (1980a) have analyzed P. W. in terms of an expanded version of the logogen model. From his reading and comprehension deficits and from his performance on a variety of other tasks we assign five distinct "lesions" on the diagram of the model (figure 3.19); however, it is arguable that only three of these processing deficits lead to the deep dyslexia symptoms. The deficits need not be disconnection deficits in the sense of Geschwind (1965). The lines in the diagram are not intended to represent well-defined fiber tracts. Some of them may correspond to fiber tracts, but that is a separate question.

I do not wish to argue the case for using such psychological models in the study of brain damage, although at least one approach to speech therapy has been based on such models (Hatfield 1982, 1983; Powell 1981). Rather, I will point to the utility of studying patients in the development of the models. In brief, the study of patients has forced changes and complications in the models, on the one hand, and provided evidence in favor of certain constructs on the other hand. To start with, the symptoms of the deep dyslexic patients demonstrate conclusively that written material can be understood without reference to phonological codes. As a second example we can take the patient described



A variant formulation of the processes involved in reading, from Shallice (1981). This version is used to analyze in a single diagram nine types of acquired dyslexia.

Figure 3.20
Shallice's model. VL indicates a visual logogen, PL a phonological logogen. The components indicated by broken lines are relevant only to the approach of Morton and Patterson (1980).

by Schwartz, Saffrin, and Marin (1980) who could read irregular words without apparent comprehension. This patient seems to establish the existence of the route illustrated in figure 3.17 between the visual input logogens and the output logogens, since irregular words cannot be read by the grapheme-phoneme conversion route and the lack of comprehension rules out the route via the cognitive system. However, the varieties of dyslexias currently described mean that certain features of the model in figure 3.17 are inadequate to deal with the data. One elaboration is due to Shallice (1981), whose model is illustrated in figure 3.20. Shallice has identified nine types of dyslexia and shown how the model can accommodate them (table 3.1).

It should be clear that the study of reading is becoming complex and technical. It is not clear that the location in the brain of the lesions that lead to dyslexic symptoms has anything to contribute to the study of the psychological deficits. It is also likely that this approach does not and cannot help to answer the questions being asked by some people who study the brain and behavior. The diagram makers failed

Table 3.1
Acquired dyslexia syndromes.

Syndrome	Representative source[a]	Assumed impairment[b]
Neglect dyslexia	Kinsbourne and Warrington 1962	C or E or G
Attentional dyslexia	Shallice and Warrington 1977	D or F
Word-form dyslexia	Warrington and Shallice 1980	I
Surface dyslexia	Marshall and Newcombe 1973	K or N or R
Phonological alexia	Beauvois and Derouesne 1979	L or P*
Deep (or phonemic) dyslexia	Coltheart et al. 1980	(L or P*) and N
Semantic access dyslexia	Warrington and Shallice 1979	(L or P*) and N
Concrete word dyslexia	Warrington 1981	(L or P*) and N
(Nonsemantic reading	Schwartz et al. 1980	N)

Source: Shallice 1981.
a. Any empirical information about a syndrome is derived from the representative source unless otherwise stated.
b. Letters refer to figure 3.20. Asterisk indicates impairment to part of a subsystem only; in particular, P* does not involve the transformation from R to S, the articulatory output logogen of Morton and Patterson (1980).

because they, and their critics, could not separate out the different questions. The same mistake will not be made again, and brain-based and non-brain-based models of language will coexist. We can only hope that the proponents of the two types will continue to talk to each other.

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Note: the references marked with an asterisk have been taken from Moutier 1908.

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