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Excerpt

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Part I

Introducing Cognitive Neuropsychology

1 From the Diagram-Makers to Cognitive Neuropsychology

1.1 Why Neuropsychology?

For 100 years, it has been well known that the study of the cognitive problems of patients suffering from neurological diseases can produce strikingly counterintuitive observations. From time to time, research workers studying normal function have been strongly influenced by such observations or by the ideas of the neurologists who made them. Bartlett (1932) and Hebb (1949) are two examples. However, in general, neuropsychology has had little impact on the study of normal function.¹

With any knowledge of the history of clinical neuropsychology, it is easy to understand why this neglect occurred. The standard of description of the psychological impairments of patients was low, often being little more than the bald statement of the clinical opinion of the investigator. There was frequently a dramatic contrast between the vagueness of the psychological account of the disorder and the precision with which anatomical investigation of the lesion that had given rise to it was carried out at post-mortem. Also, the field, like psychology itself, could agree on little but the most obvious and basic theories. Typical are the disputes about the existence of the syndrome visual object agnosia, a specific difficulty in the perception of objects when both sensation and the intellect are intact. The syndrome was widely accepted as real in the golden age of the flowering of neuropsychology (1860–1905) (e.g. Lissauer, 1890). Yet its existence was still being denied nearly a century later (e.g. Bay, 1953; Bender & Feldman, 1972).

It is now clear that there are patients whose impairment is appropriately characterised as a visual object agnosia (for reviews, see Rubens, 1979; Ratcliff & Newcombe, 1982; Warrington, 1985; chapter 8). However, other syndromes described in earlier periods have fared less well. In chapters 2 and 4, it will be argued that two syndromes first described in the 1920s and much debated since – the Gerstmann syndrome and simultanagnosia – are not functional entities. More critically, if clinical neurologists with an extensive knowledge of brain-damaged patients frequently considered major ‘syndromes’ described by earlier workers to be artefacts,

1. The term *neuropsychology* will be used to refer to the investigation of the disorders of perception, memory, language, thought, emotion, and action in neurological patients. The word *cognitive*, too, should be interpreted widely to include the higher levels of perception, memory, and the more central aspects of the control of action.

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was it not entirely reasonable for research workers concerned primarily with normal function to ignore the field?

Within the past 15 years, the situation in neuropsychology and its neighbour, neurolinguistics, has changed dramatically. Quantitative descriptions of disorders are now standard. A spate of interesting findings has resulted in theories that seem to be compatible with those developed for the study of normal cognition. In turn, a considerable number of research workers interested primarily in normal function have responded. However, this response has been patchy; within cognitive psychology, some standard textbooks hardly mention neuropsychological findings.²

The reason for this neglect is perhaps that explicitly argued by Henderson (1981) and Crowder (1982a) – that neuropsychological investigations merely follow ones in the normal subject; they do not provide leads. A related possibility is that it arises from the type of empirical assessment produced by Fodor, Bever, and Garrett (1974), which reads a little oddly in the light of the later writings of at least one of the authors – Fodor (1983) – ‘remarkably little has been learned about the psychology of language processes in normals from over a hundred years of aphasia study’ (p. xiv). More probably, it is a result of the argument given by Postman (1975) in a major review article on memory. He dismissed neuropsychological findings, stating merely, ‘The existing data do not impress us as unequivocal, more important extrapolations from pathological deficits to the structure of normal memory are of uncertain validity’ (p. 308). This type of argument was put forward in an elegant fashion much earlier by Gregory (1961):

Suppose we ablated or stimulated various parts of a complex man-made device, say a television receiving set. And suppose we had no prior knowledge of the manner of function of the type of device or machine involved. Could we by these means discover its manner of working? . . . If a component is removed almost anything may happen: a radio set may emit piercing whistles or deep growls, a television set may produce curious patterns, a car engine may back-fire, or blow up or simply stop. . . . In a serial system the various identifiable elements of the output are not separately represented by discrete parts of the system. . . . The removal, or the activation, of a single stage in a series might have almost any effect on the output of a machine, and so presumably also for the brain. . . . The effects of removing or modifying, say, the line scan time-base of a television receiver would be incomprehensible if we did not know the engineering principles involved. Further, it seems unlikely that we should discover the necessary principles from scratch simply by pulling bits out of television sets, or stimulating bits with various voltages and wave forms. (p. 320–322)³

The theoretical problems in drawing inferences from neuropsychological findings will be assessed in detail in chapters 9, 10, and 11. Assume for the present, however, that they can be surmounted, at least in most cases. Neuropsychological

2. Anderson (1980) is an example.
3. Gregory (1961) was not arguing against the use of neuropsychological findings, but for their interpretation in terms of a model of the whole system. Marin, Saffran, and Schwartz (1976), in the early days of cognitive neuropsychology, record meeting essentially this argument in a cruder form: ‘What can you possibly learn about the way a car works (or a vacuum cleaner, or a computer) by pounding it with a sledgehammer’ (p. 868)! To which the authors pointed out that random decomposition results in theoretically useful results – for instance, in high-energy physics!

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evidence has two very beguiling qualities that are best seen by contrast with 'normal' experimental evidence. Empirical phenomena in the corresponding study of normal processes – human experimental psychology – are very slippery things. Many factors affect any experimental procedure. Make a slight change in one aspect – rate of presentation, stimulus material, recall delay, amount of practice, and so on – and the effect disappears or reappears, although according to theory, it should not. Thus even if a phenomenon is narrowly robust, the experimental result provides only a most insecure platform for theoretical inferences. The consequence is that it is easy to take an inessential aspect of the experimental situation as the critical one and enter a theoretical blind alley. So progress remains slow, and the field remains as full as ever of its theoretical Jeremiahs.⁴

Given the task of understanding the organisation of the cognitive system, the first attraction of neuropsychological evidence is that the effects that occur can be both large and counterintuitive. The myriad factors that affect a subject's performance – degree of learning, amount of effort expended, individual differences, characteristics of the stimulus material, and so on – shrink in significance when compared with the magnitude and specificity of the observed deficits. One appears to be receiving a privileged view into the structure of the information-processing system.

The weakness of the empirical methods available in 'normal' human experimental psychology has a second consequence; we have little conception of the extent of the problems that need to be tackled. Empirical investigations in other sciences are often stimulated by the availability of novel techniques. In psychology, they are often triggered by the investigation of novel procedures, a phenomenon that Tulving and Madigan (1970) called the functional autonomy of methods.⁵ Such procedures become fashionable when widely thought to provide answers to important theoretical questions. Ten years and a flood of papers later, the apparently simple original experimental finding is found to be very complex indeed, and its theoretical value is much less clear. So a pessimist could view the history of 'normal' human experimental psychology as a succession of mirages. The end result consists of islands

4. Consider Neisser (1981) referring to memory, but almost certainly he would intend his message to apply more generally: 'The results of a hundred years of the psychological study of memory are somewhat discouraging. We have established firm empirical generalizations, but most of them are so obvious that every ten-year-old knows them anyway. We have made discoveries, but they are only marginally about memory; in many cases we don't know what to do with them, and wear them out with endless experimental variations. We have an intellectually impressive group of theories, but history offers little confidence that they will provide any meaningful insight into natural behavior' (pp. 11–12). His response was to call for the study of memory outside the psychological laboratory, not limited to the artificial products of the investigator's narrow set of concepts. Neuropsychology certainly qualifies! It is ironic that in his summary dismissal of what investigators have learned about memory, Neisser should have failed to mention amnesia and its many dramatic and far from artificial aspects, which challenge clinicians whatever their theoretical predilections.
5. Examples include the Peterson procedure, the recency effect in free recall, the Sternberg procedure, the stimulus suffix effect, the word superiority effect, the use of pattern masking, and Wason's four-card problem.

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of detailed empirical knowledge surrounded by a sea of ignorance, whose size we conceal from ourselves by vague theorising; in any case, its magnitude could hardly be known because we have few independent means of assessing it (see Newell, 1973).

Neuropsychology can help in this second respect, too. Advanced clinical practice contains the distilled 'craft' knowledge of over 100 years of observation of patients by neurologists and neuropsychologists; this understanding is well represented in books such as those by Hécaen and Albert (1978), Lesser (1978), Walsh (1978), De Renzi (1982), Heilman and Valenstein (1985), and Stuss and Benson (1986). This knowledge is rooted in the problems that neurological patients themselves experience. Experimental psychologists may not have investigated drawing or writing, say, because effective techniques for quantification have been difficult to develop for use with normal subjects. However, if a patient loses the skill as a result of disease, it is soon apparent to physicians. So relevant disorders, such as constructional apraxia or agraphia, are well known, and detailed clinical accounts of sub-varieties exist (see Warrington, 1969; Marcie & Hécaen, 1979). As neurological disease affects every part of the brain – if with different incidence rates – the disorders that have been described will probably encompass damage to nearly all the cognitive mechanisms. 'Inverting' the set of disorders that exist might enable us to map the subcomponents of mind. Whether an inversion procedure is conceptually possible and at present practicable is a subject that will be directly addressed later. For two reasons, then, cognitive neuropsychology would be of value for the understanding of normal function if it were demonstrated to be effective.

1.2 Paradigm Shifts in Neuropsychology: The Diagram-Makers and their Critics

In the past, it was to some extent justified for those interested in normal cognition to neglect neuropsychological findings. To understand why the situation has changed, one needs to consider the history of neuropsychology. This has other benefits, too. Without a knowledge of the history of neuropsychology, its terminology is almost impossible to understand. Names like transcortical sensory aphasia, central aphasia, constructional apraxia, Gerstmann syndrome, and lexical agraphia all reflect different conceptual approaches.

There is, though, a more critical reason why the assessment of modern neuropsychology requires some understanding of its history. The evolution of thought about impaired cognitive functioning has a remarkably dialectical quality. Just like experimental psychology, but independently of it, neuropsychology became an embryonic science in the second half of the nineteenth century. Very schematically, its history may be divided into four stages, each dominated by particular schools: the rise of the so-called diagram-makers, with their elaborate models of the mental machinery (1860–1905); the reaction against them (1905–1940); the switch to group studies (1945–1970); and the development of cognitive neuropsychology (since the

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late 1960s). In present-day cognitive neuropsychology, the long-rejected central core of the diagram-makers' approach has been resurrected. Therefore, if cognitive neuropsychology is to be successful, it must obviously guard adequately against the fatal flaws that were present in the approach of 100 years ago! Yet there are signs that this elementary precaution is being neglected.⁶

Broca's claim in 1861 that the seat of language is in the inferior posterior portion of the left frontal lobe is often cited as the event that initiated neuropsychology as a science. In fact, related claims having less anatomical precision had been made a number of times in the preceding 40 years. According to the historian of science Young (1970), the date 1861 is remembered because Broca's demonstration of the localisation of a cognitive function occurred at a time when the scientific community was prepared to treat the idea seriously, events in other sciences having made it an acceptable possibility. Broca's claims certainly provoked a flowering of research. The next 40 years saw a mass of clinical observations and theoretical analyses.

There were two main aspects to the initial claim. The first was that language is a function that can be damaged separately from other cognitive processes. The second was that the function is localisable. The dissociation of language processes from cognitive ones was soon refined much further. In 1869, Bastian differentiated disorders of reading and writing from disorders of speech and to account for the differences used hypothetical anatomical diagrams with processing centres and transmission pathways. In 1874, Wernicke isolated a further form of language disorder, now known as Wernicke's aphasia, and produced a model of a sensory and a motor language centre joined by a transmission pathway. Damage to the motor language centre, he argued, would lead to the form of aphasia that Broca had described; that to the sensory language centre would produce the syndrome that he had discovered. He predicted the existence of a third form of aphasia, conduction aphasia, which should result from damage to the pathway. Ten years later, Lichtheim (1885), having deduced that conduction aphasics should have a specific deficit in repetition, described the first case in which such a difficulty was a central aspect. He then went much further and described four more aphasia syndromes! Two more syndrome labels date from his account: 'transcortical motor aphasia' and 'transcortical sensory aphasia'.

The diagram-makers not only isolated a number of distinct syndromes, but also produced a theoretical framework to explain them. The disorders were predicted from 'interruptions' to different functional components of what were basically processing diagrams. Lichtheim's (1885) theory is especially worth examining. On its appearance, it 'excited universal interest', according to a historian of the period, Moutier (1908). For instance, the influential German neurologist von Monakow,

6. The idea that modern cognitive neuropsychology should learn from the fate of the diagram-makers is derived from Morton (1984). My view of what should be learned is, however, somewhat different from his.

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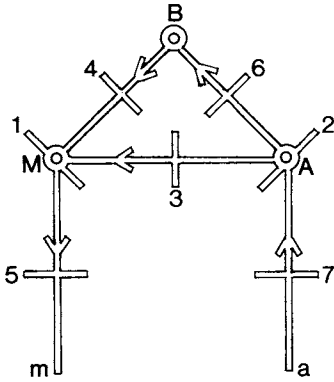


Figure 1.1. The simple version of Lichtheim's (1885) theory. A stands for the centre for 'auditory word-representations'; M, for the centre for 'motor word-representations'; B is where 'concepts are elaborated'; a and m are auditory input and speech motor output, respectively. The numbers 1 to 7, which correspond to 'interruption points', are discussed in the text.

who later broke with the diagram-makers, said that he was 'overwhelmed by the achievement' (quoted in Moutier). Even Head (1926), an arch-enemy of the school, admitted that it had had a profound influence on the clinical theories of the day, although more characteristically, he said that Lichtheim's paper 'reads like a parody of the tendencies of the time' (p. 65). In my view, the paper must be ranked today as one of the finest in the nineteenth century relevant to psychology.

I will consider only the simpler version of the theory in which reading and writing are ignored (Figure 1.1). Lichtheim actually put forward his suggested diagram of the subcomponents of the language system on mainly a priori grounds. His separation of a centre for 'auditory word-representations' from a centre for 'motor word-representations' appears to have been based on Wernicke's (1874) empirical findings. He then argued that children learn to speak by imitation so that a route from one of these centres to the other would be expected to exist. Concepts were then simply assumed to be separately located. From these a priori arguments, he inferred that each of seven 'interruption points', marked in Figure 1.1, should give rise to a different syndrome. Thus for Broca's aphasia, it is at position 1; Wernicke's aphasia, 2; conduction aphasia, 3; transcortical motor aphasia, 4; and transcortical sensory aphasia, 6. Examples were given or a reference cited for each of the claimed varieties of aphasia.

According to Moutier (1908), ten models of this type were produced between 1870 and the beginning of the twentieth century. To the information-processing psychologist of today, some of the diagrams look far more modern than the theories that were appearing in the psychology texts of the time. Consider, for instance, replacing the terms that Lichtheim used most frequently, 'centre for auditory images' and 'centre for motor images', by, say, 'auditory input logogen' and 'artic-

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ulatory (or phonological) output logogen'.⁷ The separation between the two systems is then analogous to that made by Morton (1979a) in later versions of the logogen model. Indeed, Morton (1984) has shown that in the language field, rival models of the period tend to have their modern equivalents. For instance, the models of Kussmaul (1877) and Bastian (1898) contrast with those of Wernicke (1874) and Lichtheim (1885) in having a single centre at the non-semantic level for perceiving and producing speech; an equivalent assumption is the essence of Allport and Funnell's (1981) model, produced 100 years later!

This sort of theoretical development, although most developed for language, was not restricted to it. For instance, with respect to the perception of objects, Lissauer (1890) differentiated between two types of agnosic disorder, in both of which intellect and visual sensation are intact. In apperceptive agnosia, the patient is unable to differentiate between two visually similar items, such as a walking stick and a rolled-up umbrella. In associative agnosia, the patient does not lack the ability to differentiate between visually similar objects, but loses their significance or meaning. Similar developments took place with respect to disorders of reading (Dejerine, 1892) and the organisation of skilled movement (Liepmann, 1900). However, it was over the models of aphasia that the controversy was most fierce and where the most critical defeats occurred.

Despite the modern appearance of the diagram-makers' models to our eyes, their ideas soon come under strong attack, two early critics being Freud (1891) and Bergson (1896). In the first 30 years of the twentieth century, the critics became dominant. The arguments of Marie (1906a) in France, of von Monakow (1910) and Gelb and Goldstein (1920) in Germany, and of Head (1926) in Britain led to general rejection of the approach as pre-scientific. Typical of the contemptuous attitude to their forebears in the field of aphasiology was Head's (1926): 'Incredulous of such scholastic interpretations they [i.e., neurologists in general] lost interest in a problem of so little practical importance. . . . The time was ripe for a ruthless destruction of false gods' (pp. 65–66). It is a historical irony that Wernicke and Lichtheim are now much better remembered than Head.

Hardly surprisingly, given the tone of such criticism, the matter was not just one of intellectual argument. In France, for instance, Dejerine – the principal representative of the diagram-makers' approach – and Marie – the principal critic – never spoke after Marie's challenge, except to enter into gladiatorial public debate. After Marie's victory, the followers of Dejerine were not appointed to the bureaucratically powerful positions within French neurology, even those who were widely

7. One of the most influential information-processing models of language has been Morton's (1969, 1979a) logogen model. The most critical assumption of the model is that a number of interface systems – logogen systems – lie between lower level perceptual (or motor) processes and those systems responsible for semantic and syntactic operations. In the earlier versions of the theory, there was only one such interface system; in the later versions, there are four: phonological input, phonological output, visual input, and visual output for listening, speaking, reading, and writing. Any given logogen system is composed of a set of morphemic units, each of which accumulates information about whether a particular word is present.

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acknowledged as being extremely able. At one stage, the bitterness between the two schools even resulted in a formal challenge to a duel, although over an issue related to another area of neurology (see Lhermitte & Signoret, 1982).

The rejection of the diagram-makers was by no means total. Neurology was too decentralised and international a subject for a basically progressive school of thought to be bureaucratically eliminated. Neurologists like Henschen (1920), Kleist (1934), and Nielsen (1946) continued the diagram-makers' tradition, but until the extensive work of revival by Geschwind (1965), such thinkers were very much in the minority.

The diagram-makers' approach was vulnerable to attack on three main fronts. First, they argued that the functions they postulated could be precisely localised. The evidence on which this was based was very shaky. Thus Marie (1906a), in a famous paper, reanalysed the classic cases of Broca (1861) and argued that in both the critical patients, the lesions had extended far beyond the region that Broca had claimed, so-called Broca's area, in the inferior posterior part of the left frontal lobe. In complementary fashion, patients described as functionally of a Broca type had lesions outside Broca's area (see Moutier, 1908). For the more subtle syndromes described by Lichtheim (1885), the anatomical correspondences claimed for his centres and connections were almost entirely speculative.⁸ The strict reductionist framework in which both the diagram-makers and their critics operated meant that once the anatomical part of their theorising was shaken, the credibility of the psychological side of their position was greatly weakened.

They were also being attacked on quite a different front. It was argued that the psychological concepts they had used were inadequate. For instance, the explanation given by the diagram-makers for Broca's aphasia is that it results from a 'loss of motor images'. In fact, one of the most salient features of the prototypic motor aphasic of the type described by Broca is the inability to construct whole sentences even when individual content words can be uttered. This aspect of Broca's aphasia is now known as 'agrammatism' and is frequently explained as an impairment in syntactic operations. Hughlings Jackson pointed out as early as the 1870s that such difficulties were not at all adequately captured by the concept of 'loss of motor images'. At a more general level, the overall theoretical approach of the diagram-makers must have seemed completely outdated by about 1920, when conceptual frameworks like behaviourism, Gestalt psychology, and mass action were in the ascendance. To adherents of these schools, the approach of the diagram-makers must have seemed medieval.

In accounts of the diagram-makers, it is to these two factors that their defeat is normally attributed (e.g. Luria, 1973). The insubstantial nature of the neuropsychological evidence they produced is probably an equally important factor. If their

8. For modern evidence that is, oddly, compatible with both sides of the argument, see, for example, Kertesz, Lesk, and McCabe (1977), Mohr et al. (1978), and Poeck, De Bleser, and Von Kayserlingk (1984a). Some of the brains that Broca analysed have recently been reanalysed yet again by Signoret, Castaigne, Lhermitte, Abelanet, and Lavorel (1984) using CT-scan procedures. Ironically, their conclusion was that Broca, not Marie, was right!

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empirical accounts of cognitive deficits had been strong, they may well have been able to withstand the theoretical attacks on their position. In fact, their empirical accounts were normally weak. Take the doyen of the approach, Lichtheim. As pointed out earlier, his theoretical system was based on a priori notions of how children learn to speak. He then deduced that certain types of patient should exist, and either described a patient of his own or referred to one in the literature. The process was theory driven, not data driven. Sometimes, as in his empirical account of transcortical sensory aphasia, he produced a fairly impressive case, if one accepts his terse clinical summary on faith. On other occasions, the evidence is flimsy. Thus much of the evidence for his case of transcortical motor aphasia is based on the testimony of the patient's wife. The essential part of his own observations is, 'His vocabulary is copious, but he does not talk much, and speaks in a drawling manner. From time to time he misses a word or construction. . . . He repeats correctly whole sentences, if not too long' (Lichtheim, 1885, pp. 448–449). Anyone who doubted that repetition could be preserved in the absence of spontaneous speech, as required by an interruption at point 4 (see Figure 1.1.), would have little difficulty in disposing of Lichtheim's evidence. Therefore, to give much credence to many of these case descriptions, one has to have personal experience of a virtually identical patient. Yet this is not generally possible. The types of patients whom Lichtheim described are not common. Thus Freud (1891), one of the first major critics, would not accept conduction aphasia as a syndrome. He had never seen such a patient (see Green & Howes, 1977).⁹

By the early twentieth century, even the few neurologists generally favourable to the diagram-makers, such as Henschen (1920) – who made the most thorough survey of their work ever attempted – were forced to admit that the clinical data were frequently insufficient. Opponents, such as Head (1926), were dismissive. He held that even in his own time, 'the methods in general use were too crude to provide satisfactory records' (p. 140). What was required, he said, was 'systematic empirical observation of the crude manifestations of disease' (p. 66). When he undertook this, his overall conclusion was that the more carefully the patient is examined, the less certainly his disorder corresponds to any preconceived category. Indeed, if one tries to press every patient seen in the clinic into one of the diagram-makers' categories, this comment is justified.

Head's criticisms exposed another problem in Lichtheim's approach. One of Lichtheim's most important contributions was to make a methodological distinction between a 'pure case', in which, according to his own model, there was only a single deficit, and a 'mixed case', in which there were multiple deficits. 'Mixed' cases, he argued, were not of theoretical interest. Yet the effects of most lesions will correspond to more than one deficit on his model. Thus it follows that the types of patient whom clinicians see most of the time should be those who have a com-

9. Not all empirical descriptions of this period are so inadequate. Lissauer's (1890) description of associative agnosia is far more detailed. However, in the critical language field, detailed clinical descriptions were not generally available until after 1910 (see De Bleser, 1987).