Cognitive Neuropsychology

A Clinical Introduction

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Preface

The aim of this book is to provide a broad introduction to the core subject matter of clinical cognitive neuropsychology. It is intended both for those coming to this topic for the first time and also for those already in the field interested in an overview of areas which are outside their own specialty. The development of this book had its beginnings in both authors' attempts to communicate "cognitive neuropsychology" to clinical and academic colleagues as well as to students. In this interdisciplinary area of investigation concepts of cognitive psychology are often unfamiliar to those approaching brain–behaviour relationships from the perspective of neurology and physiology. Equally, neurological issues may present difficulties for those with a background in experimental cognitive psychology. We hope that this book will open lines of communication in both directions making the domain of neuropsychology accessible to these audiences and to the wider community of cognitive and neuroscientists.

In order to make the material as widely accessible as possible we have attempted to explain issues in a way that assumes only a limited degree of prior psychological or neurological knowledge. We hope that we have been able to give a reasonably comprehensive overview of this large and complex area of investigation despite our strenuous attempts to avoid the use of undefined specialist jargon and abstruse theoretical discussion. This does not mean that we have avoided "difficult" areas or discussion of controversial theoretical issues. Our primary aim has been to give an overview of areas of debate and to avoid the minutiae of academic arguments and counterarguments.

After a general introduction to the subject matter, the book is divided into chapters, each of which deals with a specific cognitive ability and the analysis of its breakdown in patients with cerebral lesions. Both the choice of topics and their treatment is, of course, necessarily influenced by our research and clinical activities and those of our colleagues, past and present, of the Clinical Psychology Department of the National Hospitals in London. The clinical influence is perhaps most

clearly shown in the organisation of the chapters which directly mirrors the way in which much research has been conducted in direct response to a patient's problems.

First, we give a general introduction to the historical background, followed by a more detailed consideration of the relevant empirical findings. These basic facts are essential for any further analysis of an individual patient's difficulties and provide a necessary starting point for any research. This is followed by a discussion of the neuroanatomical correlates and the issues and debates which these have raised. The third section of each chapter is concerned with theoretical analyses of the relevant complex skills and abilities, drawing both upon the empirical evidence and on the relevant data from neuroanatomy.

We have confined our account entirely to cognitive disorders in neurological patients. The vast literature on animal lesion studies has not been considered; neither has the evidence from studies of neurologically intact subjects. This is not because we consider such work irrelevant for neuropsychology, but rather because any adequate and balanced treatment of this material would have resulted in a volume of encyclopaedic proportions. Whilst clearly realising the limitations of this perspective, it is our belief that a consideration of the evidence from clinical cases provides the central core for an introduction to cognitive neuropsychology.

The writing of this book would not have been possible were it not for the tolerance and good will of our colleagues and students who put up with closed doors and absent supervisors for nearly two years. To them we extend our gratitude. Our thanks are also due to Dr. Marianne Jackson, who provided us with translations of many German texts; to our institutions, the National Hospitals in London and the University of Cambridge, which have supported our research; and to Academic Press for seeing this project through to its fruition. Finally, we are most indebted to the many patient individuals who, despite the personal tragedy of brain damage, have nevertheless cheerfully helped in our research efforts.

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Introduction to Cognitive Neuropsychology

Introduction

Damage to the brain often has tragic consequences for the individual. It can affect those basic skills and abilities which are so necessary for normal everyday life and which are largely taken for granted. The rather hybrid term *cognitive neuropsychology* is applied to the analysis of those handicaps in human cognitive function which result from brain injury. Cognitive neuropsychology is essentially interdisciplinary, drawing both on neurology and on cognitive psychology for insights into the cerebral organisation of cognitive skills and abilities. By cognitive function is meant the ability to use and integrate basic capacities such as perception, language, actions, memory, and thought. The focus of clinical cognitive neuropsychology is on the many different types of highly selective impairments of cognitive function that are observed in individual patients following brain damage. The functional analysis of patients with selective deficits provides a very clear window through which one can observe the organisation and procedures of normal cognition. No account of "how the brain works" would even approach completeness without this level of analysis.

Consideration of cognitive impairments in people with brain damage has a long tradition in clinical medicine. However, as a coherent domain of investigation it has a relatively short history. The description and discussion of cognitive deficits following brain injury dates back to the earliest written records. For example, there is mention of specific language loss in the Edwin Smith papyrus of 3500 B.C., and selective impairments in face recognition and letter recognition were noted by Roman physicians. Few advances were made over the following 2000 years. Despite discovering the orbits of the planets, the

circulation of the blood, and the laws of mechanics, the "seat of the mind" had only moved from the liver to the pineal gland.

By the beginning of the nineteenth century a number of patterns of deficit had been described and were accepted as being due to disease of the brain itself. In the early nineteenth century there were significant advances in medicine, anatomy, and physiology which provided the basis for a more adequate analysis of the sequelae of brain injury. In their first investigations the nineteenth-century researchers placed considerable emphasis on the localisation of damage which gave rise to impaired function. This approach led to a number of insights and arguably led to the development of clinical neurology as an independent specialty. Subsequently the quest for localisation led to a realisation of the complexities of cognitive function. It was recognised that abilities such as language were composed of a number of distinct processing components, each of which could break down independently of the others. This analytic approach to patterns of breakdown forms the basis of much contemporary cognitive neuropsychology. The background to both of these issues and their contemporary relevance will be considered in the following two sections. First, the evidence for localisation and lateralisation of function in the human brain will be considered. Second, the evidence for dissociation of function and the basic methodological approaches of contemporary neuropsychology will be introduced.

Localisation and Lateralisation of Function

Historical Background

In the early years of the nineteenth century the phrenologists Gall & Spurzheim (1809) speculated that the convoluted surface of the brain reflected the juxtaposition of a large number of discrete cerebral organs. Each organ was thought to subserve a particular psychological faculty (or in more contemporary terms, function). Individual differences in endowment for a specific faculty would result in different degrees of development of particular convolutions of the brain. By analogy with muscular development, they suggested that endowment with mental muscles would result in an increase in the size of cerebral organs. They further speculated that this endowment would be reflected in bulges on the skull. In support of their hypothesis they produced evidence from anthropological studies of races with supposed differences in intellectual endowment, and clinical post-mortem evidence from brain-injured individuals. One of their speculations, that the language faculty might be located in the anterior sectors of the brain, was tentatively supported by post-mortem evidence. This was corroborated in independent clinical studies conducted by the eminent French physician Bouillaud (cited by Benton, 1984). However other workers reported conflicting evidence of patients whose language abili-

ties were preserved despite damage to this part of the brain (e.g., Andral, 1834, cited by Benton, 1964).

In 1861, the anthropologist and physician Paul Broca reported the case of a patient who had lost the ability to utter a single word, but who had retained his ability to understand what was said to him. The patient, a **Monsieur LeBorgne**, was a long-term resident in an institution who had been nicknamed "**Tan**" by the staff because this was the only sound he ever uttered. Despite having no meaningful speech, "Tan" (although reportedly a difficult patient) was able to cooperate with staff and to assist in the care of other inmates. Broca argued that the patient's disorder was not one which affected the muscles which were necessary for speech, because he was able to eat and drink. The patient appeared to have a specific impairment of language. Broca suggested that the patient's disease had damaged a specific centre in the brain which was responsible for mediating articulate language, a deficit he termed "aphemie." In fact, the area of tissue loss or lesion in Tan was quite extensive, spreading from the frontal to the temporal lobes of the brain. Such large areas of damage would appear to pose considerable, if not intractable, problems for precise localisation. However, Broca drew on his clinical knowledge to infer the likely sequence of events which had led to the loss of language. On the basis of the progression of Tan's difficulties, Broca argued that the onset of language disturbance was attributable to damage in a critical and restricted area, namely the third frontal convolution of the left hemisphere. This part of the brain is still termed "**Broca's area**" in recognition of his pioneering attempts to localise and lateralise the site of damage responsible for disrupting speech (see Fig. 1.1).

Karl Wernicke (1874) described patients with the opposite pattern of

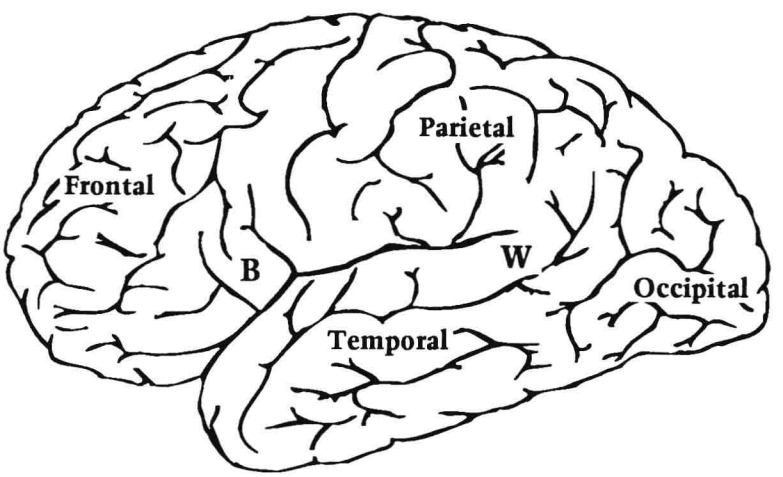

Figure 1.1 Lateral surface of the left hemisphere showing the four major lobes. B, Broca's area; W, Wernicke's area.

speech difficulty to Broca's cases—they could speak fluently, but they were unable to understand what was said to them. The patients' speech, though fluent, was by no means normal, indeed it was virtually unintelligible. They used words inappropriately and made errors in pronunciation which reflected the wrong choice of word sounds. These errors often resulted in words which were not part of the language, an error termed a *neologism* (literally, a "new word"). One patient died, and when her brain was studied at post-mortem she was found to have a lesion in the left temporal lobe, near to primary auditory cortex. However, the damage was not in the primary auditory cortex itself, but slightly more posterior, extending from the first temporal convolution into the parietal lobe (see Fig. 1.1).

Broca had also noted that damage to the *left* hemisphere appeared to be critical for language impairment. His observations were confirmed by other researchers and appeared to be valid even when the pattern of language deficit was not identical to that described in the original cases. The idea that the left hemisphere might play a special role in language function became widely accepted. This hypothesis has stood the test of time. Post-mortem studies of the brains of patients who had shown language disturbances in life indicated that damage to the left hemisphere was usually critical. This gave rise to the view that the left hemisphere was the "dominant" or leading side of the brain in most people. It is now universally accepted that the human brain has an asymmetric organisation of function. Language abilities are compromised by damage to the left hemisphere in the vast majority of people and appear to be unaffected by damage to the right side.

A strong emphasis on the lateralisation of language, rather than on the organisation of other cognitive skills, has been characteristic of many neurological and neuropsychological studies of patients. It is easy to understand why this has happened. Language deficits are obvious and a cause of considerable concern, making them somewhat easier to detect and investigate than other types of disorder. This should not blind one to the fact that damage to the right hemisphere of the brain may also have considerable effects on other types of cognitive function. The first to recognise that the right hemisphere might have specialised functions of its own was the English neurologist Hughlings Jackson (1876). On the basis of his clinical observations of a single patient he argued that whilst the left hemisphere might be important in language, the right hemisphere was critical in "visuoperceptual" abilities. The idea that the right hemisphere might be "dominant" for some types of ability was not followed up in any great detail at the time. The prevailing viewpoint was that the cerebral hemispheres existed in a "dominance" relationship with the left hemisphere being "in charge" in most people. It is only since the 1940s that systematic investigations of perceptual and spatial abilities have been conducted in patients with unilateral lesions. The results of these investigations have supported Jackson's original observations. It is now universally

recognised that the two cerebral hemispheres have complementary, but very different, specialisations. The term *cerebral dominance* still continues in modern usage, however, it now has a much more restricted meaning, namely "dominance for language."

Individual Differences?

Broca's view that the left hemisphere was necessarily dominant for language in all individuals was challenged by other neurologists. They argued that the cerebral organisation of speech would be directly related to hand preference (e.g., Wernicke, 1874). It was suggested that writing was intimately linked with spoken language and could even be considered as "parasitic" upon it and making use of the same brain centres. It would therefore be eminently reasonable if language and writing were organised in close proximity in the brain. Since control over motor function is primarily organised in a contralateral manner (with the left hemisphere controlling the movement of the right hand and the right hemisphere controlling the left hand), then the dominant hand would be contralateral to the dominant (language) hemisphere. Individuals with right-hand preference for writing would show left-hemisphere dominance for language, whereas left-handed people would show the opposite pattern and have dominant right hemispheres.

The view that language laterality and hand preference would be invariably linked became widely accepted and persisted for many decades. For the vast majority of right-handed people this "rule" does apply. However, since the majority of the population is right handed this could easily reflect a general population bias toward being "left brained" rather than a link between language laterality and hand dominance (Annett, 1985). Left-handed individuals provide the crucial test. Examples of language disorders following lesions to the left hemisphere in left-handed patients were sporadically reported in the literature up until the 1950s as examples of "crossed dominance." However, when systematic surveys of left-handed patients with unilateral lesions and language impairment were carried out this received wisdom was questioned (Zangwill, 1960). The occurrence of "crossed dominance" was by no means as rare as had been assumed (see Table 1.1).

There are two ways of interpreting these findings. The first, and the one which has been most frequently expressed in the literature, is that left-handed people have a bilateral organisation of language. This, it is argued, accounts for the similar numbers of left-handed patients with language difficulties following either right- or left-sided lesions. The inference that left handers have a bilateral organisation of language function is based on the assumption that all the individuals making up the clinical group of left handers can be considered to have the same fundamental organisation of function. This assumption is perhaps most clearly expressed by Hécaen & Sauget (1971): "If the bilaterality of cerebral dominance is the rule in left handed subjects . . . the

Table 1.1 Left-Handedness and Laterality[a]

	Lesion location	
Investigation	Right (*N*)	Left (*N*)
Conrad (1949)	7	10
Goodglass and Quadfasel (1954)	5	5
Hécaen and Ajuriaguerra (1963)	7	13

[a]Laterality of lesion site in left-handed patients with language disorders.

frequency of language difficulties should be about the same in both hemispheric (lesion) groups." There is, of course, a second way of interpreting this data: exactly the same pattern would be expected were some of these patients right brained and others left brained with respect to the organisation of language.

Another source of evidence for "bilateral" organisation of language function in left handers has been derived from estimates of the overall *risk* of language impairment following damage to the right or to the left hemisphere in this group. If language is bilaterally organised, then there should be either an increased or a decreased incidence of language disorders regardless of the location of the lesion. Thus if language was bilaterally organised then patients might be placed at greater risk of showing a disturbance following damage to either hemisphere. Alternatively, they might be "protected" from the effects of unilateral damage because function is duplicated in both sides of the brain. It has been suggested that bilateral organisation of language should result in a very different distribution of impairment than should unilateral organisation. At first sight the data in Table 1.2 appear to support this hypothesis, showing an increased incidence of language disorders in left-handed patients with unilateral lesions.

Overall, the left-handed patients have a higher incidence of language impairment, suggesting that there is an increased risk for left-handed people. However, the increased risk is not equivalent between the two hemispheres as would be expected on the bilateral hypotheses. The increased risk in left handers is entirely due to the comparatively large number of cases with language disorders following right-hemisphere

Table 1.2 Incidence of Aphasia in Left- and Right-Handed Patients[a]

	Left handers		Right handers	
Lesion site	Left	Right	Left	Right
Aphasia (N)	56	26	625	16
No aphasia (N)	46	63	422	879
% Aphasic	43		33	

[a]Data collated from 5 studies. Adapted from Zangwill (1967).

lesions. This pattern would also be consistent with there being a heterogeneity in the lateral organisation of language in left handers. Some might have right-hemisphere dominance and some might have left-hemisphere dominance, and there could possibly also be a small subgroup with bilateral language.

Surveys of patients with unilateral lesions are not really suitable for answering questions about bilateral organisation. Once damage has been done to the brain it is impossible to conclude whether remaining capacities are attributable to residual activity of the damaged side, to activity of the undamaged side, or to a contribution from both cerebral hemispheres. What is needed is a technique for temporarily "**blocking**" the activity of one side of the brain which can subsequently be applied to the other side. This would enable a direct comparison to be made between the language capacities of the two sides of the brain functioning in isolation.

Two such procedures have been reported. Milner and her colleagues (Milner, Branch, & Rasmussen, 1964; Milner, 1975) have used **Wada's intracarotid sodium amytal test** (Wada & Rasmussen, 1960), and Warrington & Pratt (1973) have used **unilateral electroconvulsive shock**. In the **carotid amytal study** patients with epilepsy who were about to undergo surgery were tested. In this procedure the barbiturate sodium amytal is introduced into the brain via the left or right internal carotid artery during the course of a standard preoperative radiological examination (angiography). This temporarily sedates one hemisphere and allows testing of the other side of the brain in relative isolation. In the Warrington & Pratt study, patients undergoing **electroconvulsive therapy (ECT)** as a treatment for depression were given unilateral shocks on alternate sides of the head on two different days. ECT has the effect of temporarily disrupting normal activity in the brain. When the shock is administered unilaterally (i.e., with the electrodes placed on one side of the head), then this disruption is largely confined to one cerebral hemisphere.

The results which have been obtained using these two techniques are in very good agreement (see Table 1.3). This is all the more impressive considering the differences in population and the relatively small numbers which are involved. The evidence appears consistent with the "mixed group" hypothesis, namely that left handers are a heterogeneous population with respect to their language laterality. The majority of left handers have unilateral representation of language, and a small proportion have bilateral organisation. If one considers the ratio of left-brained language organisation to right-brained and bilateral cases, both of these studies are also remarkably consistent with the surveys of unilateral lesion cases which were discussed above. It is therefore safe to conclude that for the vast majority of the population the left hemisphere is specialised for language processing. In left handers, the incidence of right-hemisphere and bilateral language organisation is increased.