HUMAN COGNITIVE NEUROPSYCHOLOGY

A Textbook with Readings

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Psychology Press
Taylor & Francis Group
HOVE AND NEW YORK

First published 1988
Reprinted 1989, 1990, 1991, 1993, 1994, 1995
Reprint of augmented edition 1997, 1998, 2000, 2002, 2003 and 2004

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Psychology Press Ltd, Publishers
27 Church Road
Hove, East Sussex
BN3 2FA
UK

http://www.psypress.co.uk

British Library Cataloguing in Publication Data

A catalogue record for this book is available from the British Library

ISBN 978-0-86377-715-8

Contents

This augmented edition of *Human Cognitive Neuropsychology* contains selected readings from books and journals, which are gathered together at the end of the book, starting on page 345. The readings are listed here below the chapter to which they relate.

Preface to the Augmented Edition

This book first appeared in 1988 and we are pleased that it has generally been well received and widely used. If there had been no other demands on our time, we would like to think that we would now be announcing a rewritten second edition. But there have been other demands. Both of us have endured spells as head of department since 1988. Then there are the pressures that dominate academic life in Britain in the 1990s: to win grants, maintain research teams, and publish journal articles. Writing textbooks is currently regarded as a somewhat self-indulgent pastime that distracts the individual from more the important business of writing the next grant application or submitting the next paper.

That is, we believe, an unfortunate and short-sighted attitude. Good textbooks summarise and organise material that is otherwise dispersed among countless individual articles, hence they can be of value even to researchers active in the field. Textbooks also serve as the introduction to an area for beginners, hopefully instilling in them an interest and enthusiasm for the topic. Without good textbooks, there is a serious danger that the stream of young talent that is vital to maintaining the vigour of an area will dry up. And successful textbooks help to pay for their authors' family holidays.

This is not the second edition we would like to be announcing. It is—and there is no point avoiding this fact—a stopgap, even if the gap it stops may prove to be a rather long one. We are indebted to Michael Forster of

Psychology Press (formerly Erlbaum, UK) for suggesting to us the idea of producing an augmented version of the first edition in which selected articles are used to introduce the reader to developments since 1988. The more we thought about this, the more positive we became. The addition of readings allows us to bring in material written by leading authorities in each field, and the inclusion of some primary-source case reports gives a much clearer idea of the work that goes into a really first-rate case study, bringing the topic literally and metaphorically to life. We may even continue the concept if and when we manage to write a true second edition.

The first part of this expanded edition of *Human Cognitive Neuropsychology* is, then, just the old first edition with the Further Readings at the end of each chapter revised, and a few minor errors corrected. The second part is a series of papers that expand upon, or bring up to date, the contents of each chapter. We have written a short Introduction to the one or two papers selected to complement each chapter, indicating how they relate to the material in the chapter. One of the reasons we warmed to this device is that we can imagine these additional papers being used as readings for seminars and tutorials in courses on neuropsychology and cognitive psychology. We have therefore deliberately stopped short of summarising the contents of each paper in a way that would eliminate the necessity for the student actually to read them.

We thank the authors and publishers of the reprinted papers for permission to use their work in this way. We are acutely aware that this is not the second edition that many people would have liked to see, but we hope they will find that it has genuine value.

Andy Ellis
York

Andy Young
Cambridge

June 1996

1 What is Cognitive Neuropsychology?

In any well-made machine one is ignorant of the working of most of the parts—the better they work the less we are conscious of them... it is only a fault which draws our attention to the existence of a mechanism at all.
Kenneth Craik, *The Nature of Explanation* (1943)

INTRODUCTION

On 5th August, 1982 a 19-year-old man who we shall refer to by his initials as PH was involved in an accident in which he was knocked off his motorcycle. He lost his right arm, and suffered a severe closed head injury. He was in a coma for over 12 days.

Like many head-injury patients PH has, with the help of rehabilitation services, made quite a good recovery. Some four years after his accident his language abilities seemed normal in conversation, and he could read without difficulty. His I.Q. on verbal tests (91) was probably close to what it was before the accident. His short-term memory abilities were normal and, although he scored poorly on formal tests involving long-term retention, he was able to remember the things that were important to his daily life without apparent difficulty.

However, one of PH's problems was most resistant to rehabilitation; he could not recognise people's faces. As soon as a familiar person spoke he would know who it was but, to PH, all faces seemed unfamiliar. He could tell if a face belonged to a man or a woman, an old or a young person, and he could describe the general appearance and facial features

reasonably accurately. But PH had no sense of recognising people who had previously been very familiar to him. In neuropsychological terms, his accident had left PH *prosopagnosic*—able to see, but unable to recognise many once-familiar faces (De Haan, Young, & Newcombe, 1987a).

EST was a well-educated, 65-year old man whose difficulties lay not in perception or recognition, but in speaking. His attempts to converse were hampered by the fact that he could no longer call to mind many words that had once been part of his ordinary, everyday vocabulary. The cause of EST's *anomia* (as his condition is known) was not a head injury, but a large slow-growing tumour in the left hemisphere of his brain which was successfully removed when he was 53 years old. Whereas normal people just occasionally find themselves caught in a "tip of the tongue" state, temporarily unable to remember a word, EST seemed to be trapped in such a state almost every minute because EST's word-finding problems extended to commonplace words like "piano", "spider", and "lamp". He knew perfectly well what such objects were, and what one could do with them, but was often unable to remember what they were called. His understanding of speech was good, and he could comprehend written words, though his attempts to read aloud were hindered by the same word-finding problems as affected his speaking (Kay & Ellis, 1987; Kay & Patterson, 1985).

The difficulties experienced by PH and EST are just two of the vast range of different problems that can be caused by brain injury. In this book we shall encounter many of them, though there are many more that we have not had space to include. Chapter 4, for example, reviews different forms of face recognition disorder, including the sort of prosopagnosia suffered by PH, whereas Chapters 5 and 9 examine disorders of speech production, including EST's type of anomia. Other conditions we shall look at include disorders affecting object recognition, spatial knowledge and orientation, speech comprehension, reading, writing, and memory.

Human cognitive neuropsychology is, however, much more than just a catalogue of the different problems that brain injury can give rise to. Cognitive neuropsychologists believe that by studying patients like PH and EST (with their co-operation and consent), fundamental insights can be gained into the way the human mind works. These insights should then feed back to provide a better understanding of the problems of brain-injured patients, and should lead in turn to the development of better therapies (e.g. Howard & Hatfield, 1987).

As an approach to understanding the mind and the brain, cognitive neuropsychology is both old and new—old to the extent that the issues it addresses are ones which have exercised the minds of philosophers,

psychologists, neurologists and others for hundreds, even thousands, of years; and new because it is only within the last 15 years or so that cognitive neuropsychology has become established and has articulated its distinctive approach. This chapter is intended to acquaint the reader with what cognitive neuropsychologists are trying to do. We shall discuss the sort of questions they ask, the methods they adopt in trying to answer them, the assumptions they make, and some of the potential pitfalls that await them along the way. In doing so we shall try to be brief for two reasons. First, we believe that the vigour and usefulness of cognitive neuropsychology is best established through illustrating its practical application in different areas: If any converts are to be made, they will be won over by the demonstrations in later chapters of how cognitive neuropsychology can illuminate the processes involved in human perception, language, and memory. Secondly, cognitive neuropsychology is an approach in evolution. Matters to be reviewed in this chapter, such as the appropriate methodology and the underlying assumptions, are matters of lively current debate, and we are only too well aware that opinion on these topics is likely to continue to evolve in the years to come. But the fact that we can make extensive use in the chapters to come of observations and conclusions made by earlier researchers whose theoretical viewpoints were different from our own shows that the main substance of the book has a fair chance of surviving a wide range of changes in theoretical fashion.

QUESTIONS AND POSSIBLE ANSWERS

Assuming we have spent some time investigating a case like the anomic patient EST mentioned earlier, two questions arise naturally:

1. What has happened to this patient to cause him to show the particular symptoms he does?
2. Can his pattern of impaired and intact capabilities teach us anything about the way the normal mind and brain are organised?

If we consider first the question of what has happened to EST to cause his anomia, then it soon becomes clear that the question can be answered in at least two very different ways. Brain scans have shown that the tumour which caused EST's anomia occupied a large area in his left cerebral hemisphere, affecting in particular the temporal and temporo-parietal areas (Kay & Patterson, 1985). As we have seen, the consequence of the resulting brain injury was that EST could no longer remember, or "find", many words which had once been well-established parts of his vocabulary. Is it better to say of EST, "He is anomic because of damage

to his left cerebral hemisphere" or "He is anomic because of damage to the psychological processes which mediate spoken word finding?" Although there are those who believe that one of these two modes of explanation is intrinsically superior to the other, we would suggest that they are both valid in their own way. Only the second explanation is a cognitive neuropsychological explanation, however. Accordingly, the emphasis in this book will be on *explaining the symptoms of brain-injured patients in terms of impairment to psychological operations which are necessary for normal, efficient perception, language and memory*, though we shall see that there are times when a knowledge of the relevant anatomy and physiology is of positive benefit when it would be churlish to ignore biological evidence.

Our main subject matter, however, is *cognitive* neuropsychology. Cognitive psychology (without the neuro- prefix) is the study of those mental processes which underlie and make possible our everyday ability to recognise familiar objects and familiar people, to find our way around in the world, to speak, read and write, to plan and execute actions, to think, make decisions and remember (Eysenck, 1984; Smyth, Morris, Levy, & Ellis, 1987). Neuropsychology is the study of how particular brain structures and processes mediate behaviour, and encompasses such things as appetites and emotions as well as cognitive aspects of mental life. As its name suggests, cognitive neuropsychology represents a convergence of cognitive psychology and neuropsychology. In Campbell's (1987a) words: "Neuropsychology is *cognitive* to the extent that it purports to clarify the mechanisms of cognitive functions such as thinking, reading, writing, speaking, recognising, or remembering, using evidence from neuropathology."

Cognitive neuropsychology has, then, two basic aims (Coltheart, 1986; Ellis, 1983). The first is *to explain the patterns of impaired and intact cognitive performance seen in brain-injured patients in terms of damage to one or more of the components of a theory or model of normal cognitive functioning*. Thus PH's prosopagnosia and EST's anomia might be explained in terms of damage to one or more of the processes required to effect normal face recognition and speech production, respectively.

The second aim of cognitive neuropsychology is largely responsible for the recent upsurge of interest in the approach. It is *to draw conclusions about normal, intact cognitive processes from the patterns of impaired and intact capabilities seen in brain-injured patients*. In pursuing this second aim, the cognitive neuropsychologist wishes to be in a position to assert that observed patterns of symptoms could not occur if the normal, intact cognitive system were not organised in a certain way. We shall make claims of this sort with respect to patients PH and EST in Chapters 4 and 5.

Dissociations and Associations

Assertions about the way the intact mind must be organised are often based on what are termed *dissociations*. If patient X is impaired on task 1 but performs normally on task 2, then we may claim to have a dissociation between the two tasks. For instance, if task 1 is reading words and task 2 is recognising famous faces, then we would state that patient X shows a dissociation between reading, which is impaired, and face recognition, which is intact. On such evidence alone, many cognitive neuropsychologists would feel justified in saying that the normal cognitive system must be organised with face recognition and written word recognition handled by different sets of cognitive processes, thereby allowing one set to be impaired while the other continues to function normally.

Other cognitive neuropsychologists might be more circumspect, however. They would point out that logically possible alternative accounts of patient X can be put forward. It might be, for example, that written word recognition is in some way easier than face recognition and that X's brain injury has rendered him incapable of difficult recognition tasks, while leaving him still able to perform easy ones. This type of alternative account could be ruled out, however, if a second patient, Y, could be discovered in whom written word recognition was intact whereas face recognition was impaired. That patient when contrasted with patient X would provide us with a *double dissociation* between face recognition and written word recognition. There is no doubt that double dissociations are more reliable indicators that there are cognitive processes involved in the performance of task 1 that are not involved in the performance of task 2, and vice versa (Shallice, 1979a; Teuber, 1955; Weiskrantz, 1968). Double dissociations can also be established without requiring that either patient should perform normally on either task: It would often be sufficient to show that patient Y performed reliably and significantly better on task 1 than on task 2 whereas patient X performed reliably and significantly better on task 2 than on task 1 [for the technically-minded, Jones (1983) discusses cases in which this would *not* be sufficient evidence].

There are times, however, when arguments based on such things as the relative simplicity of two tasks seem so implausible that cognitive neuropsychologists are willing to venture claims about normal cognitive organisation on the basis of single dissociations (where a patient performs well on one set of tasks but badly on another), and we shall encounter several examples of such reasoning later in the book. Also, it would be unwise to regard the search for double dissociations as some sort of royal road to understanding the structure of the mind. Having unearthed a double dissociation, there is a lot of work to be done in determining just what cognitive processes mediate aspects of tasks 1 and 2 independently,

and what processes, if any, the two tasks share in common. This requires intensive investigation of the patients in order to discover just why they perform badly when they do, and just where in the total cognitive system their breakdowns have occurred.

Much more problematical than arguments based on either double or single dissociations are arguments based on *associations* between symptoms. It is common in neuropsychology to discover that patients who are impaired on task 1 are also typically impaired on tasks 3, 4 and 5. Now, it might be that this association of deficits occurs because a cognitive process required for the successful execution of task 1 is also required for the successful execution of tasks 3, 4 and 5, so that a patient in whom that process is damaged will experience problems with all these tasks. Unfortunately, deficits can also tend to co-occur for reasons that are of neurological importance, but of less interest specifically to the cognitive neuropsychologist.

It could be, for example, that tasks 1, 3, 4, and 5 have no overlap in terms of the cognitive processes required for their execution, but that four discrete sets of cognitive processes are mediated by four adjacent areas of the brain. If this is so, then a brain injury which damages one of those areas will tend also to damage the others, so that deficits on the four tasks which depend on those four regions will tend to be associated. This point as applied to language disorders was well expressed by Lord Brain (1964, p.7) in the following passage:

> ...let us consider two aspects of language which we will merely call *a* and *b* to indicate that we habitually distinguish them in our own minds and give them different labels. Let us further suppose that they are both depressed [*i.e., impaired*] in a particular aphasic patient. There are several possible explanations of this. The primary disturbance may involve *a*, and the disturbance of *b* may be secondary to this, or conversely, we may implicate some general function *c* and say that both *a* and *b* are particular examples of disorder *c*. These are all functional or dynamic [*cognitive*] interpretations. But there is also the possibility that there is no functional relationship between *a* and *b*. They are involved together merely because their pathways, though separate in terms of neurones, run close enough together to be damaged by the same lesion.

Associations which occur for anatomical reasons rather than cognitive-psychological reasons will be encountered on several occasions in this book. They are revealed in their true colours when the exceptional patient is discovered whose lesion affects some but not all of the anatomically adjacent regions and which, therefore, affects some but not all of the cognitive tasks mediated by those regions. In sum, theoretical arguments based on observed associations between symptoms can be very appealing

because there are often good psychological reasons for expecting two or more deficits to co-occur as a result of damage to a single cognitive process, but such arguments should always be advanced with caution and are never as secure as arguments based on dissociations.

COGNITIVE NEUROPSYCHOLOGICAL METHODS

We have just seen that *differences* between patients play a very important role in the development of theories in cognitive neuropsychology. In contrast, similarities between patients, in the form of shared sets of associated symptoms, are viewed with caution if not suspicion. Several important dissociations between symptoms have been discovered in patients whom traditional neuropsychology would have grouped together as members of the same syndrome category.

This difference in emphasis is perhaps what most distinguishes modern cognitive neuropsychology from traditional neuropsychology. The latter approach used common co-occurrences of symptoms to group patients together into syndromes. Thus patients with language disorders following brain injury (aphasias) were grouped into categories labelled Broca's aphasia, Wernicke's aphasia, conduction aphasia, etc. on the basis of shared symptoms. The assumption made would be that patients with Broca's aphasia are effectively interchangeable, and quite strong claims would sometimes be made concerning symptom complexes that *had* to co-occur (if patient Z shows symptom q, she will also show symptoms r, s, and t, etc.).

It is now generally acknowledged in cognitive neuropsychology that traditional syndrome categories are too coarse-grained and often form groupings on the basis of symptoms that co-occur for anatomical rather than functional reasons (Poeck, 1983). This is understandable, because one of the original purposes of such syndromes was to assist in the determination of probable lesion sites in the days before more direct brain scanning techniques became available, but most cognitive neuropsychologists would now accept Caramazza's (1984) advice that "research based on classical syndrome types should not be carried out if the goal of the research is to address issues concerning the structure of cognitive processes".

The problem lies in deciding how best to proceed once one has acknowledged that classical syndromes are unsuitable for cognitive neuropsychological analysis. On this issue cognitive neuropsychologists fall into two broad camps. The first wishes to replace the old, broad groupings with newer, finer, more theoretically motivated categories. These could be developed by subdividing the old syndrome categories to take account of dissociations as they arise, or they could be developed *de novo* (as

with the classification of acquired reading disorders into "deep dyslexia", "surface dyslexia", "phonological dyslexia" etc.—see Chapter 8). Shallice (1979a) strongly advocates this approach, though acknowledging that it will inevitably lead to the postulation of ever more syndromes of increasing complexity and specificity.

Other cognitive neuropsychologists react to the manifest inadequacies of the classical syndromes by suggesting that there may simply be no need to group patients into categories in order to practise effective cognitive neuropsychology (e.g. Caramazza, 1984; 1986; Ellis, 1987). If it were possible to group patients into homogeneous categories, then that would represent a valuable saving because cognitive neuropsychologists would only need to produce an explanation for each syndrome, not each individual patient. Unfortunately, advocates of the revised syndrome viewpoint have not yet managed to come up with a single, lasting homogeneous category. Thus, the categories of acquired reading disorder mentioned earlier, which are only 10 or 15 years old at the time of writing, are already fractionating as theoretically important individual differences are found among patients in the same categories. As the rest of this book shows, similar fates are befalling all other attempts to delineate new syndrome categories.

One possible response to this situation is to argue that cognitive neuropsychologists should treat each patient as a unique case requiring separate explanation. Single patients could serve the same role in cognitive neuropsychology as single experiments do in experimental cognitive psychology—each is a separate test of cognitive theory (Ellis, 1987). This does not mean that all comparisons between patients are excluded: There are times in the book, for example, when we wish to highlight similarities between two or more patients. Typically, however, this happens because they share a single particular *symptom* which may be given the same explanation in each case. The point is that the other symptoms these patients show may be very different: The patients are alike in one respect but are different in several others and could not plausibly be combined into a syndrome category. In the remaining chapters of this book, we tend to retain traditional neurological terms (aphasia, dyslexia, agnosia, etc.) simply as a shorthand convenience for referring to particular broad classes of symptoms: We do *not* wish to imply that patients with a common symptom will necessarily show that symptom for the same reason.

We do not wish to labour this point about the usefulness or otherwise of syndrome groupings (which we see as just one of the teething troubles of a new scientific approach trying to establish how best to proceed). Advocates of new syndromes still talk to advocates of the single-patient approach. The two groups share the same theoretical models of reading,

object recognition, memory or whatever, and each uses the others' case studies to develop those theories. Although we have our own views on this issue, we have tried not to let them dominate this book, which we hope will find acceptance among cognitive neuropsychologists of all denominations.

Case Studies

What an increasing number of cognitive neuropsychologists now agree upon is that the approach is best served by intensive single-case studies of patients with deficits in different areas of cognitive processing. This stands in contrast to traditional neuropsychology where the dominant approach has often been one in which the performance on one or more tasks of a group of patients of a given type is contrasted either with the performance of another group of patients of a different type or with a group of normal "control" subjects. Such studies commonly report only the average score on each task for each group. Unfortunately, much potentially valuable information can be lost in such an averaging procedure, notably information about individual differences between patients assigned to the same groups (Shallice, 1979a).

Accordingly, even cognitive neuropsychologists who believe in the usefulness of syndrome groupings now tend to present data on each individual patient separately. Many publications in cognitive neuropsychology are devoted to presenting and interpreting data from just one patient with a disorder of particular theoretical interest. Generalisability of theories comes in two ways: First, a theory or model of a particular cognitive function is meant to account for *all* reported cases of disorder of that function, so that the theory is *not* a theory of a single patient; and secondly, these are theories of normal, intact cognitive functioning which are used to explain disorders. As such they must explain all the available data from experimental cognitive psychology as well as all the available neuropsychological data. Few areas of psychology place such exacting demands on their theories.

Shallice (1979a) made several recommendations about how single-case studies should proceed. He suggested, for example, that when comparisons between patients are appropriate they would be facilitated if "baseline" data from a range of standard neuropsychological tests were supplied. Beyond that point the particular tasks given to the patient are likely to be tailor-made and designed to evaluate a particular hypothesis as to the nature of the patient's disorder. Such tests should be given under conditions that are as controlled as possible, and their results should be analysed statistically using tests applied as standard in experimental cognitive psychology. Tasks which are of particular theoretical importance

should be given on more than one occasion to establish the replicability of their results, and theoretical conclusions should be supported wherever possible by data from more than one task.

In fact, although we have described traditional neuropsychology as being devoted to group studies, that is an over-simplification. Rare or exceptional disorders have always been reported as single-case studies, and in the decades between about 1870 and 1910 a succession of important case studies were published. As we shall see shortly, there are several respects in which modern cognitive neuropsychology can justly be regarded as a return to this turn-of-the-century approach, though the theories and methods employed have become more sophisticated.

MODULARITY

It has already been argued that if one patient shows an impairment of reading but not face recognition, whereas another shows an impairment of face recognition but not reading, then that double dissociation indicates that there are cognitive processes involved in recognising faces that are not involved in reading words, and vice versa. As the remainder of this book will reveal, such dissociations abound in cognitive neuropsychology. If we follow through the logic of our argument, that means that the cognitive skills of the sort we shall be discussing are mediated by large numbers of semi-independent cognitive processes or systems, each capable of separate impairment.

This view of how the mind and brain are organised has come to be known as the *modularity hypothesis*. According to the modularity hypothesis, our mental life is made possible by the orchestrated activity of multiple cognitive processors or *modules*. There may, for example, be one set of modules responsible for various aspects of face recognition, another set for recognising written words, a third set for maintaining our orientation in the geographical environment, and so on. Every module engages in its own form of processing independently of the activity in modules other than those it is in direct communication with. Modules are also distinct within the brain, so that brain injury can affect the operation of some modules while, at the same time, leaving the operation of other modules intact (hence a patient can, for example, experience difficulties in face recognition following brain injury without necessarily experiencing difficulties with reading).

Current interest in the modularity hypothesis stems in large part from the work of Marr (1976; 1982) and Fodor (1983). Building on his experience in both vision research and the simulation of complex human abilities in computers, Marr suggested that complex systems, like minds and brains, are very likely to evolve towards a modular organisation in

the course of their development. This is because it is easier, according to Marr, both to detect and correct errors and to improve complex systems whose organisation is modular. Thus, Marr (1976) writes:

> Any large computation should be split up and implemented as a collection of small subparts that are as nearly independent of one another as the overall task allows. If a process is not designed in this way a small change in one place will have consequences in many other places. This means that the process as a whole becomes extremely difficult to debug or to improve, whether by a human designer or in the course of natural evolution, because a small change to improve one part has to be accompanied by many simultaneous compensatory changes elsewhere.

An Analogy

An analogy may help at this point. Modern hi-fi systems are often highly modular, consisting of separate and separable record decks, cassette decks, radio tuners, amplifiers, speakers, headphones, and so on. In contrast, all-in-one "radiograms" of the sort seen in the 1950s were much less modular. One advantage of the modularity of a modern hi-fi is that it assists in tracing the source of a malfunction because disorders can be confined to particular modules leaving the operation of the others intact. Thus, if the record you are playing sounds dreadful, you can decide whether the fault lies in the deck, amplifier or speakers by trying a cassette, listening through headphones instead of through the speakers, and so on.

Many amplifiers have spare slots which allow you to add on new components as they come on the market (adding a compact disc player to an existing hi-fi for example). All that is required is that the new component should provide an output which is compatible with the requirements of the existing components. Similarly, the modular organisation of our minds and brains may allow us to develop new cognitive components and interface them with old ones to create new skills and capabilities. The development in childhood of modules for reading and writing would be an example; reading and writing have only become widespread in very recent history, yet we will see that they appear to be modularised within the brain. Finally, a new, improved type of record deck may come onto the market. If your system is modular you can simply replace your old deck with one of the new type without needing to touch any of your other components, thereby illustrating Marr's point about modular systems being easier to improve.

Diagrams and Diagram Makers

If you were in the position of wanting to assemble a hi-fi system from scratch then you might find it useful to sketch a simple diagram showing the components you need and how they will interconnect. Diagrams are very useful expository devices wherever modular systems are under consideration (Ellis, 1987; Morton, 1981). They were used extensively by the school of neuropsychologists which flourished between 1870 and 1910 (Morton, 1984).

Figure. 1.1 shows the diagram put forward by Lichtheim (1885) as a model of the recognition and production of spoken and written words. It comprises five different "centres" or modules interlinked in certain ways. Centre A is a module whose function is to recognise the spoken forms of words when listening to a speaker and also to provide spoken word-forms when you are speaking yourself. Centre B houses word concepts or meanings, and is similarly employed in both the production and comprehension of language. Centre O, the centre for visual word images, recognises written words and also makes their spellings available in the act of writing. Finally, Centre M holds "motor images" ready to guide the groups of muscles which will articulate the words.

Diagrams like this were used to explain different forms of language disorder in terms of damage either to the centres themselves or to the pathways connecting them. A patient who had problems understanding or producing both spoken and written words might, for example, be

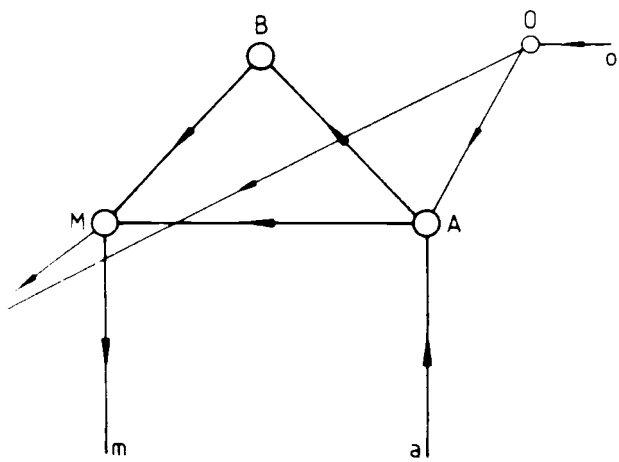

FIG. 1.1 Lichtheim's (1885) model of word recognition and production (reproduced with permission from Howard and Hatfield, 1987).

assumed to have suffered damage to centre B, whereas a patient whose problems lay in articulating words in speech would be said to have an impairment affecting centre M. A patient who could both understand and produce spoken words, but had difficulties in repeating heard words, might be interpreted as having suffered damage to the connection between A and M, and so on.

The popularity of diagrams like Lichtheim's must tell us something about their usefulness for neuropsychologists in practice. They did, however, have their problems (Marshall, 1982; Morton, 1984). First, the diagrams were only of real use in interpreting disorders that affected the comprehension, production or repetition of single words: They had little to say about disorders affecting, for example, grammatical processes involved in sentence construction. Secondly, the theorists had very little idea as to how their centres might actually work. They lacked the vocabulary of computational, information-processing concepts that now enables us to talk about the possible internal operations of the modules we postulate. Thirdly, whereas a modern cognitive theory has to account for data from experimental cognitive psychology as well as data from neuropsychology, the diagrams of the late nineteenth century were only constrained by neuropsychological evidence. The danger was that a diagram could be modified in a fairly *ad hoc* manner in order to fit the particular pattern of impairment seen in any particular patient. Some of these modifications were arbitrary and manifestly unsatisfactory. A theory that can "explain" any patient who comes along simply by redrawing the diagram is unfalsifiable. Unfalsifiable theories may seem attractive to the novice, but because they set no constraints on the claims or predictions that can be made, they are of little real use.

Finally, the majority of the turn-of-the-century diagrams were superimposed upon an outline of the left cerebral hemisphere of the brain. The diagrams therefore incorporated both a cognitive theory as to what the appropriate centres and their interconnections were, and a neuropsychological theory as to where the centres were located in the brain and where the tracts ran between them. If a patient was discovered whose symptoms were not those one would predict, given the site of the patient's brain injury, there was no way of knowing whether the cognitive component of the diagram—the proposed set of centres and connections—was at fault, or whether it was the proposed localisation of the centres and connections that was in error.

Today's cognitive neuropsychologists are much more careful to distinguish between theories as cognitive models and questions as to where a proposed set of modules may be localised within the brain. Some cognitive neuropsychologists refuse almost as a matter of principle to discuss issues of possible localisation; others consider such issues to be of interest but

acknowledge that they are separate from the evaluation of the model as a cognitive theory. Our own view is that many of the deficits we will discuss are related to damage to particular areas of the brain. Thus we will mention lesion sites from time to time, but we do *not* give them *explanatory* status.

In his influential book, *The Modularity of Mind*, Fodor (1983) acknowledged several antecedents of the notion of modularity, but curiously failed to mention the nineteenth-century diagram makers. Yet these theorists are arguably closer than anyone to current thinking, as Morton (1984) demonstrates in his point-by-point comparison of nineteenth-century diagrams with information-processing diagrams proposed by cognitive psychologists in recent times. We shall make quite extensive use of modular diagrams in this book to help us understand disorders shown by patients, though some of the weaknesses of nineteenth-century diagrams, such as their problems in giving an account of grammatical and other high-level disorders, remain.

Fodor's Proposed Attributes of Cognitive Modules

In *The Modularity of Mind* Fodor listed what he thought to be the properties of cognitive modules. Important among these was the property of *informational encapsulation*, meaning that a module must carry out its own form of processing in complete ignorance of, and isolation from, the processes going on elsewhere in the total cognitive system. If, for example, there is a module or set of modules which process the emotional expression on a face, and a separate module or set of modules which recognise the face and determine who the person is, then informational encapsulation demands that the modules processing the emotion on the face must operate independently of any activity within those modules processing the identity of the face.

According to Fodor, modules must also be *domain-specific*, meaning that each module can only accept one particular sort of input. For example, the module processing the emotional expression of faces would not also be able to process the emotional tone of voices; such processing would require a separate domain-specific module. As Shallice (1984) pointed out, if the assumptions of informational encapsulation and domain specificity are combined with an assumption of *neurological specificity*, whereby modules are distinctly represented within the brain itself, then cognitive neuropsychology becomes a viable enterprise, because the possibility arises that brain lesions will selectively impair certain modules while leaving the others intact and operating at normal, pre-injury levels of efficiency.

The notions of informational encapsulation and domain specificity are

ones which commentators upon Fodor's thesis have found easiest to accept (see, for example, the commentaries which accompany Fodor, 1985). Other properties of modules that Fodor proposed have received less unanimous acclaim. For example, Fodor argued that the operation of modules is *mandatory*. This means that modules are unstoppable—they are beyond voluntary control, and if the appropriate input is present a module will carry out its particular source of processing whether the owner of that module wishes it or not. We shall discover that many modules do indeed show the property of mandatory operation, particularly the modules involved in various aspects of recognition and processing of sensory information, but there may be modules whose operation is not mandatory. For example, the system from which the names of people and things are retrieved appears to have many of the required properties of modules, yet the retrieval of the name of a person or object seems to be more voluntary than mandatory. We cannot stop ourselves recognising a familiar person we see, but we do seem to have some voluntary control over whether or not we activate the module from which the person's name is retrieved. Conceivably, mandatoriness is more a property of input modules than of output modules.

Another property of modules which Fodor suggested but which has come in for some criticism is the notion that cognitive modules are of necessity *innate*, i.e. they are part of our genetic endowment. As Schwartz and Schwartz (1984) among others noted, some of the best cognitive neuropsychological evidence for the existence of modular systems comes from studies of acquired reading and writing disorders (dyslexias and dysgraphias). The skills of reading and writing appear to be made possible by the concerted and orchestrated activity of several cognitive modules each of which is capable of separate impairment, and those modules in the skilled reader and writer apparently behave just like any other cognitive modules. Yet reading and writing are artificial, culturally transmitted skills which until recently have only ever been acquired by a very small minority of people, and few psychologists are willing to entertain the notion that the modules required to read and write are part of our biological heritage (though see Marshall, 1987). It in no way threatens Fodor's general thesis to argue that modules can be established through a process of learning as well as being inherited genetically.

One of Fodor's most controversial suggestions was that whereas input processes to do with the perception of the external world (and possibly output processes to do with the control of action upon the world) are modular, there may also be central parts of the mind which are not modular in their organisation. Fodor suggests that higher-level thinking processes such as are involved in reasoning, decision making, the formation of beliefs, etc., are the product of operations which are not informa-

tionally encapsulated, not mandatory, not domain-specific, and so on. He even went so far as to suggest that because these central processes are not modular they are not amenable to scientific investigation. Readers interested in pursuing this aspect of Fodor's line of thought are directed to the commentaries in Fodor (1985).

From a cognitive neuropsychological perspective it is true that the dissociation methodology has largely been applied to input and output processes, and dissociations have not been sought among higher-level mental operations. If Fodor is right, then the quest for dissociations among input and output modules will be a quest directed at carving Nature at her joints, but trying to find dissociations among higher mental processes would be like trying to carve a meat loaf at its joints. That said, even if Fodor is right, there is still room for a cognitive neuropsychology of higher mental processes, because dissociations are not the only weapon in the armoury of the cognitive neuropsychologist. One of the techniques that cognitive neuropsychologists use in order to formulate hypotheses about the possible internal workings of cognitive systems is to look at the sorts of errors those systems make when they are partially but not completely disrupted. We shall look, for example, at the sorts of errors made by patients with speech production deficits or spelling impairments in order to try to understand something about the internal workings of modules which mediate the production of spoken and written words. One can equally look at the sorts of errors made by patients with disorders of higher mental functions and the sorts of difficulties they experience on different tasks in order to learn something about how those higher mental functions can be disrupted and impaired. One would hope in the process to learn something about how higher mental processes actually operate. The work of Shallice (1982) and Duncan (1986) represent steps in this direction.

SOME FURTHER ASSUMPTIONS OF COGNITIVE NEUROPSYCHOLOGY

The philosopher of science, Imre Lakatos, has argued that every science has at its core a set of assumptions which are not directly testable (Lakatos, 1974). These assumptions may be right or they may be wrong—the only way that the scientist working in a particular area will know whether its assumptions are right or wrong is by seeing whether the whole approach advances or flounders. As cognitive neuropsychology has become established in recent times, so its practitioners have sought to identify some of the core assumptions upon which it rests. The work of Shallice (1979a; 1981a), Saffran (1982) and Caramazza (1984; 1986) is of particular importance here.

Modularity is arguably one of the core assumptions of cognitive neuropsychology—something which can never be ultimately proved or disproved, but upon whose validity the enterprise as currently articulated rests. Another key assumption, following Shallice (1981a), is what we have already called *neurological specificity*, and what others have called *isomorphism*. This is the assumption that there is some correspondence between the organisation of the mind and the organisation of the brain. In the words of Lashley (1941): "The discovery that the various capacities which independently contribute to intellectual performance do correspond to the spatial distribution of cerebral mechanisms represents a step towards the recognition of similar organisation in neurological and mental events."

This assumption is not one that neuropsychologists in all periods have been willing to make. Brain (1964, p. 6) wrote that:

> The older neurologists, and even some today, thought that the different varieties of aphasia produced by lesions in different situations could be classified in psychological terms... but this presupposes first that in the nervous system speech is organised in such a way that anatomical centres correspond to psychological functions, and then that destruction of such a centre merely impairs a particular psychological element in speech. This view has largely been abandoned.

The view which Brain thought abandoned is one which has been revived by cognitive neuropsychologists and one which underpins much of the cognitive neuropsychological enterprise. This does not mean that it is right, and if it is wrong (as Brain thought) then the early promise of cognitive neuropsychology will not be borne out in its further development. We should note, however, that at minimum all that cognitive neuropsychology needs to claim is that impairments of cognitive processes can be selective. It may well be possible to see selective deficits following injury to systems in which the storage of information is "distributed" rather than being organised into physically discrete centres (modules) corresponding to psychological functions. If so, then cognitive neuropsychology can proceed with its research programme.

Another assumption of cognitive neuropsychology is the assumption of *transparency*, which requires that "The pathological performance observed will provide a basis for discerning which component or module of the system is disrupted" (Caramazza, 1984). That is, careful analysis of the pattern of intact and impaired performance and the pattern of errors shown by a patient after brain injury must be capable of leading us to valid conclusions about the nature and functions of the impaired processing components. To this end, Caramazza (1984) suggests that the

performance of a given patient will reflect four factors. These are:

1. The contribution attributable to the "true" effect of the hypothesised disruption of one or more processing components (modules).
2. Normal individual variation in performance.
3. The effects of compensatory operations.
4. Effects that result from disruptions to processing mechanisms other than the hypothesised component.

Clearly some of these factors present obstacles to the interpretation of the pattern of symptoms shown by a particular patient. Caramazza's mention of normal individual variation in performance highlights the fact that we are not all alike in so-called normality. Even within the normal population of individuals without brain injuries some people's modules work better for some things than do other people's, so that some people are naturally better than others at, say, verbal skills or spatial skills. Before one attributes a patient's poor performance on certain tasks to brain injury, it is necessary to satisfy oneself that the patient was not constitutionally poor on those tasks even before his or her brain injury. There is a real danger of diagnosing an impairment of geographical orientation in a patient who always used to get lost when he turned round twice, or an impairment of spelling in a patient who has never been able to spell at all well. The usual (but not infallible) way of preventing such possibilities is to show that the patient's performance is seriously impaired in comparison to appropriately chosen control subjects.

Caramazza's third factor, that of "compensatory operations", refers to the widely acknowledged fact that aspects of a brain-injured patient's performance may reflect cognitive systems working in ways rather different from those in which they worked before the brain was damaged. For example, some patients read words in a letter-by-letter manner, naming each letter before saying what the word is and often before understanding it (see Chapter 8). Letter-by-letter reading is not something that normal readers ever do, nor something that these patients would ever have done before their brain injury; rather it seems to be a way that non-damaged cognitive systems can operate so as to effect a form of reading in a new and unusual way when other parts of the system have been damaged. What matters for cognitive neuropsychology is not that old modules can be put to new uses, but that new modules should not be coming into existence following brain injury.

The important assumption that the performance of a brain-injured patient reflects the total cognitive apparatus minus those systems which have been impaired, is what Saffran (1982) termed the assumption of *subtractivity*. It is assumed—and there is as yet no good evidence to

cast doubt on this assumption—that the mature brain is not capable of sprouting new modules after brain injury. Only if we make this assumption can we use our models and other theoretical accounts of intact cognitive operations to interpret a patient's behaviour in terms of damage to the formerly intact cognitive system. As Caplan (1981) observes: "If the lesioned brain develops systems that are radically different than normal, that is an interesting and medically important fact, but not one relevant to normal functions." The injured brain may develop new *strategies* for coping in a particular task or situation, but it must do so using pre-existing *structures*. We shall encounter several examples of such strategies in the course of the book (e.g. patients who identify people using single salient visual features, or patients who read words by first naming all their letters). Such bizarre strategies need to be explained in terms of old modules and connections being put to new uses, though it is probably fair to say that such abnormal strategies are less helpful than other disorders when it comes to helping us understand the organisation of normal cognitive processes.

Caramazza's (1984) fourth requirement—that effects seen in brain-injured patients should not result from disruptions to processing mechanisms other than those hypothesised to be impaired—alludes to the fact that most brain injuries are substantial and cause damage to multiple processing components. There is a danger of ascribing to one component effects which are in fact due to a second, separate component which also happens to be damaged in that patient. In essence, this is the point made earlier in the chapter that co-occurrences of symptoms are much more hazardous things to base theoretical conclusions upon than are dissociations between symptoms. We will encounter several instances in this book where two or more symptoms, which at one time have plausibly been attributed to the impairment of one cognitive component, have later proved to be dissociable and must now be attributed to impairments to two separate cognitive components.

We should note that the assumption of transparency, whereby a patient's pattern of performance will provide a guide—albeit a complex one—to the nature of the underlying disruption, is one on which opinions have varied quite widely. Heeschen (1985, p. 209) quotes the neuropsychologist Kurt Goldstein as "emphatically pointing out over and over again that the brain-damaged patients' spontaneous behaviour never reflects the deficit itself, but rather the patients' reactions to the deficit". This does not undermine cognitive neuropsychology because "the true behavioural deficit shows up... under more carefully controlled and restricted formal testing conditions", but it suggests that we should be thinking in terms of an assumption of potential if sometimes clouded visibility rather than crystal transparency.

CONVERGING OPERATIONS

We have mentioned a number of similarities between today's cognitive neuropsychology and that practised by the "diagram makers" of the late nineteenth century. There are also, however, some important differences. One of these is that the diagram makers came to a cognitive neuropsychology from a background in medicine and neurology. In contrast, most of today's cognitive neuropsychologists either come from a background in experimental cognitive psychology or work in collaboration with mainstream cognitive psychologists. This means that cognitive neuropsychology is much closer in aims and in theories to experimental cognitive psychology than has ever been the case in the past. This is in part responsible for cognitive neuropsychology's current vigour.

It is noticeable that the subjects on which cognitive neuropsychology cut its teeth in the late 1960s and early 1970s were subjects for which there was a strong tradition in experimental cognitive psychology and for which there were viable theories of normal functioning. For example, Shallice and Warrington (1970) used existing theories of the organisation of short-term memory and long-term memory as a framework within which to interpret the performance of their patient KF in whom short-term verbal recall was impaired although long-term recall was preserved. Shallice and Warrington argued that such a pattern of performance was, in fact, incompatible with prevailing theories of memory structure, such as that put forward by Atkinson and Shiffrin (1968), but was interpretable in terms of a somewhat modified memory model.

Although Shallice and Warrington eventually disagreed with and sought to modify pre-existing memory models, nevertheless those models and techniques which derived from experimental cognitive psychology shaped and guided their investigation of patient KF. Similarly, Marshall and Newcombe (1973) were able to make use of models of normal reading performance which existed at the time in their analysis of different forms of reading disorder (acquired dyslexia). Once again their cognitive neuropsychological work led them to propose modifications to certain existing models, but their work was nevertheless closely guided and shaped by the theories and methods of cognitive psychology. Unlike the diagram makers, who had to devise their own theories of normal performance while simultaneously using those theories to explain different patterns of disorder, the more recent generation of cognitive neuropsychologists have often been able to begin their investigations of disorders in particular areas with reference to theories of normal performance put forward by mainstream experimental cognitive psychologists.

Modern-day cognitive neuropsychologists also bring to the study of patients the techniques of analysis developed in experimental psychology,

including techniques for the statistical interpretation of results. Indeed, many cognitive neuropsychologists retain a foot in the experimental camp, because it is not uncommon for work with a patient to generate predictions about how normal subjects will behave in particular tasks or under particular conditions. Cognitive neuropsychologists can find themselves alternating between the hospital ward or patient's home and the cognitive laboratory in their pursuit of the understanding of how a particular area of cognition works.

We noted above that cognitive neuropsychology rests on a number of fundamental assumptions. So, equally, does experimental cognitive psychology. But the assumptions of the two approaches are to some extent different. This means that a conclusion about the nature of cognition which is supported by evidence from both experimental and neuropsychological studies is more reliable than a conclusion which is supported by evidence from only one source, because the conclusion supported by two lines is that much less likely to be artifactual or to rest on a faulty assumption. Seeking support for a theoretical conclusion from two or more different sources is what Garner, Hake, and Eriksen (1956) termed *converging operations*, and the quest for converging operations has provided much of the vigour of cognitive neuropsychology in recent decades.

The quest for converging evidence is seen very clearly in the work of Shallice, McLeod, and Lewis (1985). They sought evidence for the independence of cognitive modules from experiments with normal subjects involving "dual-task" performance. They reasoned that if neuropsychological data suggests that two tasks are dissociable and therefore mediated by separate sets of cognitive modules, then in the normal subject it should be possible for those two sets of modules to sustain their separate tasks independently without detriment to either. Therefore, two tasks which each depend for their execution on different sets of modules should be capable of being executed together simultaneously almost as efficiently as either can be executed on its own. Shallice et al. (1985) tested this prediction in a dual-task experiment where normal subjects were required simultaneously to read aloud written words and to monitor a list of heard names for particular target names. They found that their subjects were capable of reading aloud while monitoring heard names almost as well as they could either read aloud alone or monitor heard names alone. This matches similar work, such as that of Allport, Antonis, and Reynolds (1972) who found that skilled pianists with a little practice could simultaneously repeat passages of prose they were hearing over headphones and sight-read music they had seen beforehand for only 10 seconds with little detriment to either task. This line of convergence promises to provide a good way of assessing whether modules which neuropsychological evi-

dence suggests are capable of functioning independently of one another can in fact do so in the normal, intact person.

A rather different form of convergence between data from patients and data from normal subjects comes when normal subjects display "symptoms" similar to those shown by brain-injured patients. We shall see in Chapter 5 how the word-finding difficulties of some "aphasic" patients closely resemble the occasional difficulties which normal people can experience when caught in a tip-of-the-tongue state. Similarly, in Chapter 7 we shall show how patients with certain forms of writing disorder can make habitual spelling errors which resemble the occasional spelling difficulties of normal subjects. Chapter 8 discusses a form of acquired reading disorder known as "attentional dyslexia" in which patients often report having seen words which are made up of letters taken from words actually present on the written page but rearranged to form a new word (for example, seeing the word *peg* when the words in front of them are *pad* and *leg*). Normal subjects will occasionally make this sort of error when reading (Cowie, 1985) but will make these same errors much more frequently if shown groups of words for very short intervals. That simple experimental manipulation greatly increases the number of errors to a level which can come close to that of "attentional dyslexics" (Allport, 1977). The crucial difference, of course, is that the "attentional dyslexics" make these errors when they have unlimited time to inspect words.

The importance of this sort of converging evidence lies in the support it provides for the subtractivity assumption—the assumption that what we see in a brain-injured patient is just the previous, intact cognitive system minus those components which have been lost or impaired through brain injury. Where the errors made by neurological patients resemble errors made by normal people then we feel confident in saying that the cognitive systems which are impaired in the patient, and give rise to the habitual errors of those patients, are the same systems which very occasionally malfunction in normals, or which can be made to malfunction more often when stressed by various experimental manipulations. We do not need to postulate the growth of new cognitive processes or even of new strategies in the patient in order to explain the occurrence of symptoms which have counterparts in normal behaviour and normal errors.

Converging operations are extremely important in present-day cognitive neuropsychology. The aim is to develop theories of normal, intact cognitive functioning which are also capable of accounting for the different patterns of disorder that can be seen in neurological patients. Sometimes the development of those theories will be better served by laboratory experiments with normal subjects; sometimes by careful study of brain-

injured patients. We should be willing to turn to either source of evidence as necessary. The continuing vigour of cognitive neuropsychology will depend to a large extent on whether or not it is able to keep abreast of developments in cognitive psychology. If cognitive neuropsychology were to lose touch with mainstream cognitive science, then it would be in real danger of losing much of its momentum.

We said at the outset of this chapter that we think the strength of cognitive neuropsychology is best appreciated through experience of its achievements in helping to unravel cognitive processes. Accordingly, we shall wind up our introduction at this point in order to turn to a consideration of specific applications of the cognitive neuropsychological approach. Each of the following chapters will end with an Overview, a Summary, and a list of Further Reading. The Overview will make some general theoretical points about the cognitive function under consideration, while the Summary will provide a précis of the main points. Because this entire chapter is, in a sense, an Overview, we shall forego such a section here.

SUMMARY

Cognitive neuropsychology has undergone a revival since around 1970. It is an approach which attempts to understand cognitive functions such as recognising, speaking or remembering through an analysis of the different ways those functions can be impaired following brain injury. More specifically, cognitive neuropsychology seeks to explain the patterns of impaired and intact cognitive performance seen in brain-injured patients in terms of damage to one or more of the components of a theory or model of normal cognitive functioning and, conversely, to draw conclusions about normal, intact cognitive processes from the observed disorders.

Dissociations, in which one aspect of performance is impaired whereas others are preserved, are taken to imply the existence of separate cognitive subsystems or *modules* responsible for different cognitive operations. The hypothesised organisation of these modules may (according to taste) be expressed in terms of an "information processing" diagram. Frequently, observed *associations* between deficits are harder to interpret because of the danger that they may arise for anatomical rather than functional reasons (e.g. cognitively distinct modules depend on adjacent regions of cerebral cortex and thus tend to be impaired together).

In contrast to traditional neuropsychology which tended to study groups of patients, cognitive neuropsychologists typically investigate single cases of theoretical importance. The results of these investigations are interpreted in terms of a set of assumptions which are still being articulated

and changed as the approach evolves. The assumption of *isomorphism* states that the cognitive structure of the mind is reflected in, and arises out of, the physiological organisation of the brain. The assumption of *transparency* holds that, given the wit and the time, it will be possible to deduce the nature of the underlying cognitive disorder in a patient from the pattern of preserved and impaired capabilities (including the pattern of errors). This process will be aided by the assumption of *subtractivity* according to which the performance of a brain-injured patient is explained in terms of the capabilities of the normal, intact cognitive system minus those components which have been lost as a result of the injury. In other words, the mature brain is assumed to be incapable of developing new cognitive structures following injury.

Cognitive neuropsychologists believe that we can draw general conclusions about the way the intact mind and brain work from studying neurological patients, but such conclusions can obviously also be drawn from observational and experimental studies of normal subjects. Some theoretical questions may be more easily resolved by the study of patients, others by the study of normals. The most reliable conclusions, however, will be those supported by independent evidence from the two separate lines of enquiry.

FURTHER READING

We can begin with the two papers that initially excited many people and awakened them to the possibilities of cognitive neuropsychology:

Shallice, T. & Warrington, E.K. (1970). Independent functioning of verbal memory stores: a neuropsychological study. *Quarterly Journal of Experimental Psychology, 22*, 261–273. Used data from patient KF to argue against the prevailing view of the organisation of short-term and long-term memory as sequential memory stores in favour of a parallel entry model (see Chapter 10).

Marshall, J.C. & Newcombe, F. (1973). Pattems of paralexia: a psycholinguistic approach. *Journal of Psycholinguistic Research, 2*, 175–199. A review of classical research on reading disorders plus new data supporting a "dual route" model of normal reading processes (see Chapter 8). Introduced, for better or for worse, the terms "visual dyslexia", "surface dyslexia", and "deep dyslexia". Still a joy to read.

Then we recommend three readings that examine the reasons why detailed investigations of individual patients are particularly important in cognitive neuropsychology:

Shallice, T. (1988). *From neuropsychology to mental structure* (Section I, pp. 1–37) Cambridge: Cambridge University Press. Introducing cognitive neuropsychology. More detailed introduction to topics discussed here.

Caramazza, A. & McCloskey, M. (1988). The case for single–patient studies. *Cognitive Neuropsychology, 5*, 517–528. Part of a special issue of the journal *Cognitive Neuropsychology*, which was devoted to methodology. The paper argues that only single-case studies allow valid

inferences about normal cognitive processes from acquired disorders and rejects clinical syndrome classifications.

Ellis, A.W. (1987). Intimations of modularity, or the modularity of mind: doing cognitive neuropsychology without syndromes. In M. Coltheart, G. Sartori, & R. Job (Eds), *The cognitive neuropsychology of language*. Hove, UK: Lawrence Erlbaum Associates Ltd. Queries the wisdom of replacing old neuropsychological taxonomies of patients with new cognitive neuropsychological ones. Argues that the results of case studies should be related directly to theoretical models without the intervention of syndrome categories.

Finally, here are two readings that look carefully at recent theoretical developments:

Shallice, T. (1988). *From neuropsychology to mental structure*. Cambridge: Cambridge University Press. Section III (pp. 203–266): Inferences from neuropsychological findings. Critique of ultra-cognitive neuropsychology and detailed discussion of inferences that can be made from neuropsychological dissociations.

Quinlan, P. (1991). *Connectionism and psychology: a psychological perspective on new connectionist research*. New York: Harvester Wheatsheaf. Chapter 5 (pp. 195–237): Higher-order aspects of cognition. Intelligent discussion of some connectionist accounts of neuropsychological phenomena.

2 Object Recognition

INTRODUCTION

The area of neuropsychology that has received the most attention, both from the traditional localisationalist approach and in the more recent studies in which the disorders are considered from a psychological perspective, is language use. There are several reasons for this, including the marked cerebral asymmetries in the control of language which seem well suited to investigation in terms of the localisation of functions in particular areas of the brain. The structural properties of language itself also offer a ready choice of factors to manipulate and investigate in more psychologically oriented studies. In addition, disorders of language are commonly encountered in stroke patients and in other patients with cerebral injuries, and can take remarkably specific forms.

The use of language, however, presupposes something to talk about. So let us begin by considering impairments in an individual's ability to understand the world around her or him; a world of objects and people. In doing this we will first consider the ability to recognise objects (this chapter), then broaden our discussion to examine a wider range of visual and spatial abilities (Chapter 3), and then consider the ability to recognise other people and to interpret their feelings and expressions (Chapter 4). These are vast topics, and in order to keep the range and quantity of material to a manageable level we will concentrate on the understanding of the visually perceived world, and on the face as a source of information used to identify people and interpret their feelings. Although the cognitive analysis of such impairments has not been nearly as widely pursued as

has the cognitive analysis of disorders of language, we think that it holds great promise and that there are exciting discoveries to be made. This is not, however, to underestimate the size of the obstacles that will be encountered along the way.

Before considering neuropsychological studies of object recognition, we examine briefly some of the factors involved in recognising objects, and develop a simple theoretical framework to describe the functional components (modules) involved.

UNDERSTANDING OBJECT RECOGNITION

Most people are able to recognise everyday objects with ease across quite wide ranges of distances, orientations and lighting conditions. This is necessary for normal life, because we encounter the objects concerned under many different circumstances. In pointing out that an object can usually be recognised despite such transformations we do not wish to imply that the transformations have no effect. Gross or unusual transformations of distance, lighting or orientation can, for instance, be used to make puzzles in which everyday objects become hard to recognise. Our point is only that the brain's object recognition system has the potential to cope with such transformations and that under everyday conditions their effects are not usually noticed.

We can also readily recognise depictions of objects on a two-dimensional surface in the form of photographs, coloured pictures, or line drawings that may or may not include implied pictorial depth. Realistic depictions of these types make use of some but not all of the cues that can be used to recognise real objects.

Two important points can be deduced from this preliminary consideration of object recognition. The first is that descriptions of the structures of all of the objects we know must in some sense be stored in the brain, so that we are able to recognise one we have met before even if it is seen from a new angle. Object recognition can thus be considered to involve a comparison of the structure of a seen object with the structures of objects that are already known. The second point is that although this comparison will often demand knowledge of the three-dimensional structure of the objects concerned, there are certain cases in which outline shape can be sufficient to effect recognition. Recognition from outline shape probably requires that the object concerned is both well known and has a particularly characteristic shape, and will often also require that it is seen (or depicted) from one of a limited range of viewing positions.

The most powerful theoretical analysis of object recognition to date was presented by Marr (1980, 1982). Marr took as his starting point the

assumption that vision involves the computation of efficient symbolic descriptions or representations from images cast by the world upon the retina. The basic questions he addressed were thus those of what types of representations are necessary for vision and what computational problems their construction poses. He suggested an analysis that proceeds through a sequence of three types of representation:

1. An initial representation, which Marr called the primal sketch. He thought that this would represent intensity (brightness) changes across the field of vision, and the two-dimensional geometry of the image. Such features as edges will usually produce abrupt intensity changes.
2. A viewer-centred representation, which Marr called the $2^1/_2$-D sketch. This would represent the spatial locations of visible surfaces from the viewer's position. Marr's idea was that conventional sources of information concerning depth and location (stereopsis, texture gradients, shading, and so on) are computed as part of the primal sketch and then assembled in the the $2^1/_2$-D sketch. The disadvantage of the $2^1/_2$-D sketch is that it lacks generality since it describes the object only from the observer's viewpoint.
3. An object-centred representation, which Marr called the 3-D model representation. This is a representation of the seen objects and surfaces which is independent of the viewer's position, and specifies the real shape of these objects and surfaces and how they are positioned with respect to each other.

Because the object-centred (3-D model) representation specifies the three-dimensional structure of the object in a relatively standard form, recognition by means of looking up this structure in some kind of store of all known object structures would then be possible.

A problem in understanding how objects are recognised that has often received comment concerns the fact that the level in the hierarchy of things in the world at which recognition is required can vary. A motor car might, for instance, be identified under different circumstances as a vehicle, a car, a Ford car, a Ford Escort, or as your friend's car. This point is important because it emphasises the flexibility of the human cognitive system.

We think, however, that the significance of our potential for flexibility of approach in object recognition can be overemphasised. Although it is certainly true that the car can be identified at any of the levels described by someone with the requisite knowledge, it does not follow either that all levels of recognition can be achieved with the same ease or that one level is not typical of everyday use. A particularly convincing case has been made by Rosch and her colleagues that categorisations of concrete

objects are not arbitrary, but determined by their natural properties into certain basic categories (Rosch, Mervis, Gray, Johnson, & Boyes-Braem, 1976; Rosch, 1978). The basic category for our example would be "car". These basic categories were found to exist at a level at which objects in different categories could be most readily differentiated from each other in terms of attributes and shapes; they were also the earliest categories to be sorted and named by children. Of particular importance to the present discussion is Rosch et al.'s (1976) finding that objects could be classified as members (or not members) of the basic category more quickly than they could be classified as members (or not members) of superordinate or subordinate categories. Thus you would be quicker to identify your friend's car as a car than as a vehicle (superordinate category) or as a Ford car (subordinate category). This suggests that identification as a member of superordinate or subordinate categories may often be achieved via an initial identification at the basic level.

A MODEL OF OBJECT RECOGNITION AND NAMING

A model of the functional components involved in object recognition and naming consistent with the points we have discussed is presented in Fig. 2.1. This is by no means the only possible theoretical model, but we believe that it is adequate for present purposes. The model makes use of Marr's idea that three levels of representation of the visual input can be distinguished; we have called these initial, viewer-centred and object-centred. It also makes use of the idea that recognition is effected by comparing viewer-centred and object-centred representations to stored structural descriptions of known objects. We have called these stored descriptions object recognition units, and they act as an interface between visual and semantic representations (see Humphreys & Riddoch, 1987b; Seymour, 1979; Warren & Morton, 1982, for related conceptions of object recognition). A visual representation (separated here into initial, viewer-centred and object-centred) describes what the object looks like, whereas a semantic representation specifies its properties and attributes. One recognition unit is held to exist for each known object. This recognition unit can access the object's semantic representation when the visual representation of a seen object corresponds to the description of the object stored in the recognition unit. The object recognition units can be "primed" by recent experience or by context to be more easily activated (i.e. to "expect" certain objects to occur). Like most contemporary theories of object and word recognition (see Seymour, 1979) we tend to think that any particular stimulus has a semantic representation that can be accessed by different types of input (object, picture, written

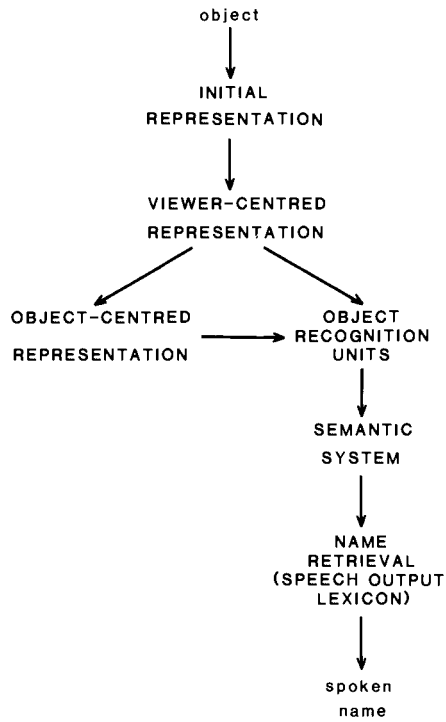

FIG. 2.1. Functional model for object recognition.

name, spoken name, etc.) rather than a different semantic representation for every type of input.

Object naming, which is a task commonly given to neuropsychological patients, is dealt with in our model by assuming that the semantic system does not contain the object's name, but can retrieve the name from a separate store or "lexicon", which we shall discuss in Chapter 5 under the heading of the "speech output lexicon". There is no direct link between the object recognition units and this name store; all retrieval of object names occurs via a semantic representation.

THE CONCEPT OF AGNOSIA

GL, an 80-year-old salesman, returned exhausted from one of his business trips after a severe storm had blown him against a wooden fence, knocking his head. He retired to bed for a few days, complaining that he was no longer able to see as well as before.

GL's problems were obvious when he got up. Although he could still see, he no longer recognised the things about him. Instead, he

looked around in a perplexed manner, as if everything was unfamiliar. He thought that pictures in his room were boxes, and tried to search in them for things he could not find. He mistook his jacket for a pair of trousers, and at mealtimes he could not recognise the pieces of cutlery on the table.

Detailed investigations showed that GL still had almost normal visual acuity for his age, and that he could draw quite accurate copies of seen objects that he could not recognise; thus his vision was in at least some respects intact. Neither had he lost his knowledge of objects; he referred to them appropriately in conversation, and he could recognise them immediately if he handled them, or from characteristic sounds. When shown a whistle, for example, he had no idea what it was, yet he recognised it straight away when it was blown.

GL's problems form a good example of *visual agnosia*. The term agnosia is derived from the Ancient Greek language, and roughly translates as "not-knowing". Use of the term agnosia is usually taken to imply that the recognition disorder is not a consequence of general intellectual deterioration, language impairment, or basic sensory dysfunction at the level we have described as the initial representation. The patient can still see things, but fails to recognise what they are. GL's case is, in fact, of particular interest because it was one of the first to be reported. His accident occurred in 1887, and his case was described in a classic paper by Lissauer (1890; see Shallice & Jackson, 1988).

Agnosias may be in visual (inability to recognise seen objects), tactile (inability to recognise felt objects) or auditory (inability to recognise heard objects) modalities (see Oppenheimer & Newcombe, 1978; Rubens, 1979; Vignolo, 1982). Within a particular modality they can occur for different classes of stimuli such as colours, objects or faces. Often, however, the same patient will be agnosic for more than one modality or for more than one class of stimuli within a particular modality. An excellent review of the clinical features of agnosic disorders is given by Rubens (1979). We will give most attention here to the nature of the underlying cognitive impairments found in cases of agnosia for visually presented objects. These are very disabling, and Humphreys and Riddoch (1987a) provide a sympathetic account of the impact of the condition on everyday life.

There seem to be several different causes of visual object agnosia. Lissauer (1890) himself realised this, and distinguished between what he termed "apperceptive" and "associative" agnosias. He proposed that visual recognition can be separated into apperceptive and associative stages, and that each when impaired has its own characteristic agnosia. The apperceptive stage would correspond to the final stage of purely "perceptual" processing; it was considered to be intact if the patient

could accurately copy items s/he could not recognise. The associative stage would give the percept meaning by linking it to previous experience.

Lissauer's apperceptive or associative distinction is still often used as a starting point in identifying the various types of agnosia, but we will not make much use of it here because, as we will see, the issues raised by modern studies of agnosia demand a richer type of theory.

Disconnection Hypotheses

Lissauer's explanation of apperceptive agnosia was in terms of *damage* to the perceptual mechanisms themselves, whereas his explanation of associative agnosia was closer to that of a *disconnection* between intact perception and stored associations. Damage and disconnection continue to be used as explanatory concepts in modern studies of agnosia, and disconnection explanations were given a particular boost by the work of Geschwind (1965a; 1965b), who demonstrated that certain neurological syndromes fit disconnection explanations very neatly.

Although it was not central to his argument, Geschwind (1965b) proposed that visual object agnosia can result from disconnection of areas of the brain responsible for vision and for speech. Such a disconnection could, for instance, happen when serious injury of the posterior part of the left cerebral hemisphere deprived the left hemisphere's speech areas of visual input by destroying simultaneously both the left hemisphere's own visual areas and the connections (via the corpus callosum) to the left hemisphere's speech area from the remaining visual areas of the right cerebral hemisphere. The patient would still have an intact (right hemisphere) visual area and an intact (left hemisphere) speech area, but these intact areas would be disconnected from each other (for most patients there is no right hemisphere speech area). This is actually an oversimplification of Geschwind's suggestion, but it is sufficient to establish the basic point that such disconnections are anatomically possible. Moreover, cases of difficulties in object recognition for which disconnection provides a plausible and appealing explanation have certainly been described (e.g. Mack & Boller, 1977; Newcombe & Ratcliff, 1974, case 3; Rubens & Benson, 1971). As we will see, however, disconnection cannot account for the problems of all agnosic patients, and the basic distinction of vision and speech is in any case too simple to cope with the complexity of the issues that emerge. The first cases we will discuss involve shape processing impairments. They are what Lissauer (1890) would have considered to be apperceptive agnosias (but see Warrington, 1987, for a different view), and are not susceptible to disconnection explanations.

SHAPE-PROCESSING IMPAIRMENTS

Benson and Greenberg (1969) reported their observations of a young soldier, Mr S, who had suffered accidental carbon monoxide poisoning. Mr S seems to have possessed an initial representation of visual stimuli that was at least to some extent intact. His visual fields were normal to 10 mm and 3 mm white objects, and he was able to maintain fixation. He could name colours and describe at least some other perceptual qualities; for instance he described a safety pin as being "silver and shiny like a watch or nail clipper". He was said to appear attentive to his surroundings, and he could navigate the hospital corridors successfully in his wheelchair. He was also able to distinguish small differences in stimulus brightness and wavelength on psychophysical testing, and could detect movements of small objects.

On any task requiring shape or form perception, however, Mr S was very severely impaired. His eye movements seemed random when he scanned pictures, and he was virtually unable to recognise objects, pictures of objects, body parts, letters, numbers, faces or geometrical figures from vision alone. He was unable to copy letters or simple figures and could not match a sample figure to an identical figure in a set of four. In marked contrast, he was able to identify and name objects from tactile, olfactory and auditory cues. No defects were noticed in his memory, spontaneous speech, or comprehension.

Mr S showed impaired ability to analyse visual form. We could interpret the case as being one of severe impairment in constructing the viewer-centred representation, because there is some evidence of sparing of the simple perceptual properties given by the initial representation despite almost total deterioration on tasks requiring shape information including copying, matching and identification. Efron (1968) provides further information on Mr S's shape-processing impairment.

A more recent case described by Abadi, Kulikowski, and Meudell (1981), and investigated subsequently by Campion and Latto (1985), however, suggests that impairment of the initial representation might contribute to this type of problem. Like Mr S, Campion and Latto's patient, RC, had suffered accidental carbon monoxide poisoning. He also showed an impairment of object recognition, being able to identify only 17 out of 27 objects with considerable difficulty. He could not copy line drawings, or even trace them with his finger. In contrast, he could negotiate obstacles, reach out for seen objects, name objects from touch or sound, and comment on the colour and texture of seen objects. His visual acuity was normal, yet he maintained that his vision was "not clear".

Initial investigation of RC's visual fields showed only that there was

an area of blindness in the lower right-hand portion. Such visual field defects are often found after neurological injury, but this alone could not account for RC's object recognition impairment, because many patients with worse visual field defects do not have the same problem. More careful testing revealed, however, that small areas of blindness were scattered across the whole of RC's field of vision. Campion and Latto (1985) suggest that this "peppering" of the visual field resulted from diffuse damage to the visual cortex of the brain: Because of their cortical origin, RC would not be aware of these numerous small areas of blindness when he looked at things (in the same way as we are not normally aware of the blind spot in our own field of vision where the optic nerve leaves the retina). Figure 2.2 gives an idea of how difficult this hypothesised scattering of blind areas throughout the visual field might make object recognition.

We are touching here on the much debated issue of the contribution of impairments of basic perceptual abilities to agnosias. RC's case makes clear that such impairments may contribute to at least some cases of disordered object recognition ability. The suspicion that all visual object agnosias may be a consequence of subtle (and, by implication, probably overlooked) alterations in perceptual function has been particularly difficult to shake off, but it is now widely accepted that sensory impairments show no necessary relation to object identification difficulties, and that many patients who are in no sense agnosic show greater sensory defects than those who are (Ettlinger, 1956; Young & Ellis, 1988). Moreover, as we will see, even the most careful testing has failed to reveal sensory defects in a few of the agnosic patients who have been described.

RECOGNITION OF DEGRADED STIMULI, AND IMPAIRMENTS OF OBJECT CONSTANCY

Degraded Stimuli

We have already commented on the facility with which normal people can recognise objects across a relatively wide range of perceptual transformations. Several studies of groups of patients with posterior lesions of the left or right cerebral hemispheres have, however, shown that patients with right hemisphere injuries do not show this facility to the same degree as normal people. These patients are not agnosic, in the sense that their everyday recognition abilities are not dramatically affected by the disorder, but they show clear impairments on certain types of task. They experience, for instance, disproportionate difficulty in identifying objects which are drawn overlapping each other (De Renzi & Spinnler, 1966), or in identifying objects from pictures degraded by the removal of some

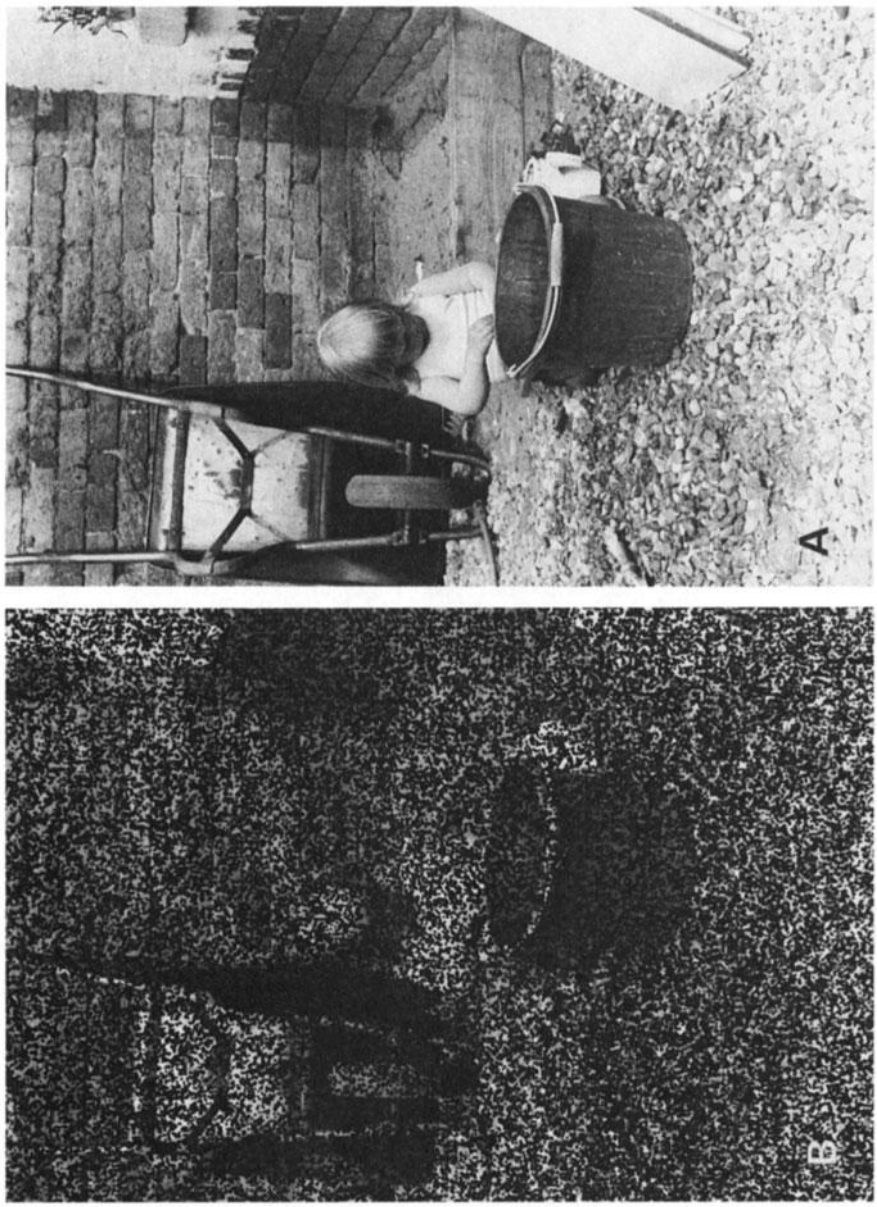

FIG. 2.2. A: Photograph of a scene, and B: the same photograph covered with a random dot mask to simulate what the world might look like to R.C. It is assumed that the patient would not actually be aware of the dots but would be left with the consequent disruption of contour necessity for form perception. Note the differential masking effect of the same mask on different objects. Thus, the child's face completely disappears, whereas the wheelbarrow remains visible. The bucket contour is disrupted to an intermediate extent. (Reproduced with permission from Campion, 1987.)

edge information (De Renzi & Spinnler, 1966; Warrington & James, 1967a).

A number of other examples of this type could be given. What they all share is the finding that in difficult object recognition tasks the performance of patients with right hemisphere injuries is more affected than the performance of normal control subjects or patients with left hemisphere injuries. Warrington (1982, p. 18) remarks that "the hallmark of this syndrome appears to be a difficulty in perceiving meaningful visual stimuli when the redundancy normally present within the figure is reduced or degraded".

This impairment in difficult recognition tasks does not seem to be a direct consequence of impairment at the level of what we have called the initial representation. Several examples of this point are given by Warrington (1982), who shows that despite their impairment on object identification tasks, patients with right posterior injuries can achieve what she calls an "adequately structured percept". Their impairments in visual sensory efficiency, figure-ground discrimination and contour discrimination are no greater than those of other patients (usually patients with left hemisphere injuries) who do not experience difficulty in object identification tasks.

The difficulties in identification tasks are thus thought to reflect impairments at a post-sensory level of visual information processing, such as the viewer-centred and object-centred representations. The deficits are seen as sufficient to interfere with difficult identification tasks while leaving performance on relatively simple identification tasks within normal limits. Comparable impairments in right hemisphere patients have also been shown for the recognition of degraded letters (Warrington & James, 1967a) and for deciding whether pictures of faces distorted by the exaggeration of lighting effects were those of a man, woman, old man, old woman, boy or girl (Newcombe, 1969; 1974). The parallel between these tasks and those causing impairment of object identification in right hemisphere patients is easy to see, though it should not be too quickly assumed that they all measure the same deficit, because Warrington (1982) has presented evidence for dissociations between comparable impairments with different types of visual stimulus material.

Unusual Views

In a series of papers Warrington (1982; Warrington & Taylor, 1973; 1978) has argued that object recognition requires some means of assigning equivalent stimuli to the same perceptual category, in order to cope with transformations of orientation, lighting, distance, and so on. It is this perceptual categorisation that she thinks defective in patients with pos-

terior injuries of the right cerebral hemisphere. In terms of our own model Warrington's idea of perceptual categorisation involves the combined action of the functional components described as viewer-centred representation, object-centred representation, and object recognition units (i.e. stored descriptions of the structures of familiar objects).

The evidence presented by Warrington and Taylor (1973; 1978) is intriguing. They showed patients photographs of objects taken from conventional and unusual views. Although Warrington and Taylor do not attempt to define what constitutes a conventional or an unusual view, the idea is not difficult to pick up, and an example of what is meant is given in Fig. 2.3. Warrington and Taylor (1973) point out that they chose the unusual views so that they were not necessarily unfamiliar views. Their unusual view of a bucket, for instance, involves looking almost directly into it; yet buckets are not uncommonly seen from this angle.

Two different versions of tests using conventional and unusual views have been devised by Warrington and Taylor. The first version (Warrington & Taylor, 1973) involved photographs of the same 20 common objects taken from both conventional and unusual views. Subjects were first required to identify the object shown in each of the unusual views, and then to identify the same objects from each of the conventional views. Their findings were that few errors were made from the conventional view, but a group of patients with posterior injuries of the right hemisphere were poor at identifying objects from an unconventional view.

We would suggest that at least part of this deficit in identifying objects from unconventional views may be explainable in terms of an impairment in constructing object-centred representations. As already stated, Warrington's idea of perceptual categorisation seems to encompass what we have described as the viewer-centred representation, object-centred representation and object recognition units. However, the unimpaired performance of the patients with right posterior injuries on conventional views suggests that the viewer-centred representation and object recognition units are relatively intact. The key feature of many (though by no means

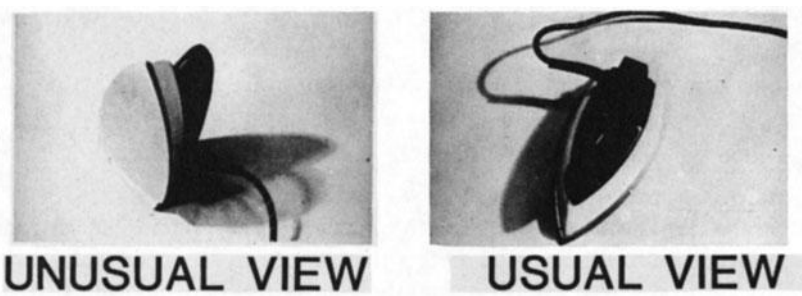

FIG. 2.3. Examples of usual and unusual views of an object. (Figure kindly supplied by Professor E. K. Warrington.)

all) of the unusual views used by Warrington and Taylor (1973) is most likely to be the foreshortening of the object's principal axis of elongation. This foreshortening would make it particularly difficult to derive an object-centred representation (Marr & Nishihara, 1978) and would thus highlight any impairment at this level.

In the second version of their conventional and unusual views test Warrington and Taylor (1978) presented pairs of photographs of objects, with one conventional view and one unusual view in each pair. The task was to decide whether the photographs in each pair were pictures of the same object; Warrington and Taylor (1978) describe this task as involving matching by physical identity. An impairment in a group of patients with posterior injuries of the right cerebral hemisphere was again found. This result is particularly striking because it implies that these patients cannot form an adequate representation of the object in the unusual view *despite* being able to derive an explicit hypothesis as to what it might be from their unimpaired performance with the conventional view photographs. When the same patients were later asked to identify the objects from photographs of conventional and unusual views presented one at a time, an impairment was again found for the unusual views.

It is clear, then, that patients with posterior (usually parietal lobe) lesions of the right cerebral hemisphere show impairments on some object recognition tasks which do not seem to be a direct consequence of an impairment in the initial representation of visual stimuli. Warrington has interpreted these difficulties as reflecting impaired perceptual categorisation, whereas we have preferred to emphasise the importance of object-centred representations. We see this as a variant of Warrington's explanation rather than a challenge to it. Both accounts locate the impairment at a level of visual information processing that can be described as post-sensory but pre-semantic.

Object Constancy

Humphreys and Riddoch (1984; 1985) have extended Warrington's work by carrying out investigations of individual patients with impairments of object constancy. By object constancy, Humphreys and Riddoch mean the ability to recognise that an object has the same structure across changes in view. They propose that we have two independent means of achieving object constancy; one by making use of an object's distinctive features, and the other by describing its structure relative to its principal axis of elongation. "Unusual" views might then impair object constancy either because they obscure a distinctive feature or because they foreshorten the object, making its principal axis of elongation more difficult to determine.

To disentangle these possibilities Humphreys and Riddoch used a matching task in which two photographs of the same object were presented together with a third photograph showing a visually similar distractor.

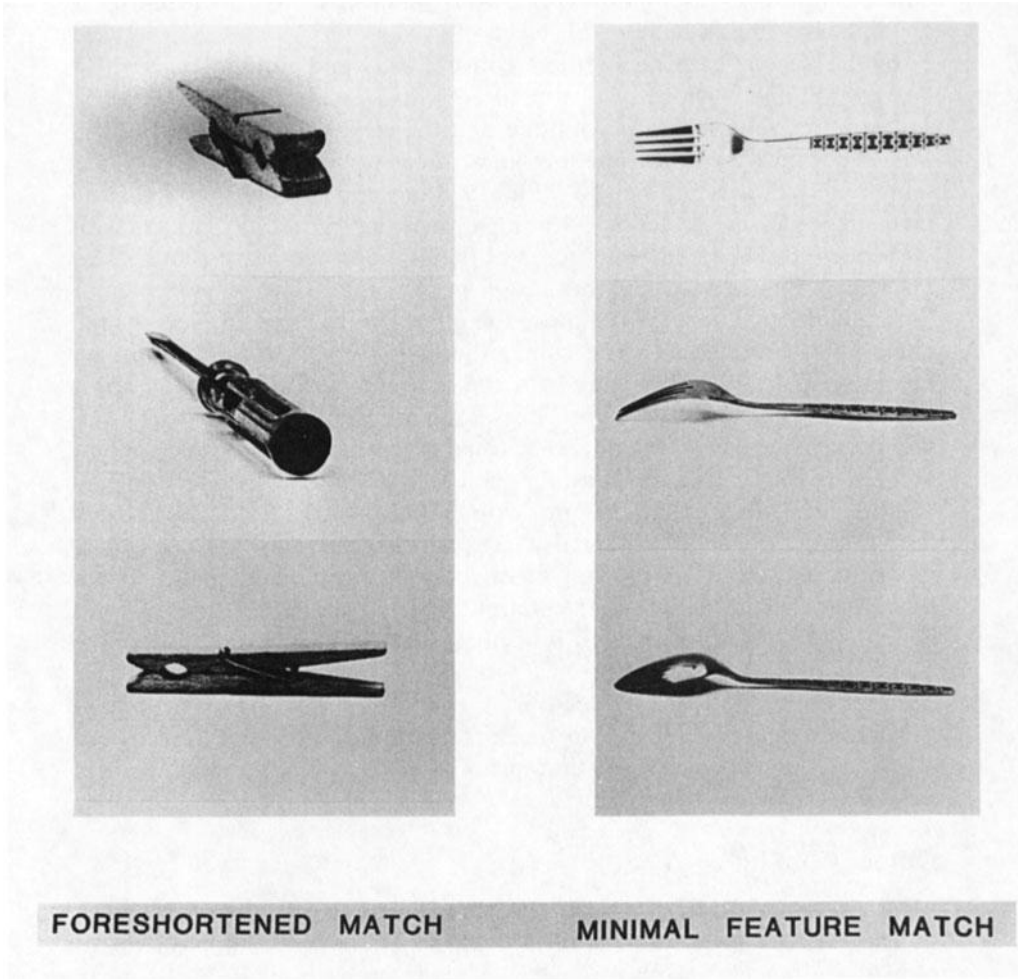

FIG. 2.4. Examples of the foreshortened and minimal-feature conditions of Humphreys and Riddoch's (1984) matching task. (Stimuli kindly supplied by Dr. G. Humphreys & Dr. J. Riddoch.)

Patients and control subjects were asked to pick the two photographs which showed the same object. The target objects were carefully chosen to have a primary distinctive feature and a principal axis of elongation. One member of the correct pair of photographs always showed the target object in a "prototypical" view, in which its distinctive feature and axis of elongation were clearly visible. The other member of the correct pair of photographs showed the same object with its axis of elongation foreshortened or with the saliency of its primary distinctive feature reduced; these are called the foreshortened and minimal-feature conditions, respectively. It is easy to get the idea by examining the examples shown in Fig. 2.4.

Humphreys and Riddoch (1984; 1985) present data for five patients—four of these had right posterior cerebral lesions and the fifth, HJA, had a severe visual object agnosia caused by bilateral occipital lobe lesions. Because the performances of the four patients with right hemisphere lesions were so similar we have selected one representative, JL. On tests of basic form perception involving length, orientation and position discrimination with two-dimensional shapes, both JL and HJA were within the normal range of performance.

Data from the matching task for JL, HJA and control subjects are shown in Fig. 2.5, together with data indicating how often JL and HJA could name successfully the objects used from the different views. It was only the foreshortened pictures that created serious problems for JL, and these problems were equally severe whether the task was one of matching or naming. Thus, as we suggested for Warrington's patients with right posterior lesions, JL seems to have difficulty deriving the object-centred representation that would be needed to make a successful match or a successful identification in the foreshortened conditions. Humphreys and Riddoch (1984) in fact note that JL tended to fail to utilise available depth cues with the foreshortened objects, treating them instead as if they were almost two-dimensional, and that JL's performance was improved if extrinsic depth cues (in the form of a textured background) were provided.

HJA's problems are clearly quite different. The most notable feature is that he was much better at matching the objects than at identifying them by name. His performance at matching the foreshortened views was much better than JL's, yet he was as poor as JL at naming them. Thus HJA seems to have access to some form of object-centred representation (because he can match foreshortened views quite well) yet he did not seem to be able to use this very successfully to identify the objects concerned. HJA's case is one of the most fascinating (and thoroughly documented) in the literature of visual agnosia, and we will return to consider the case in detail later in this chapter.

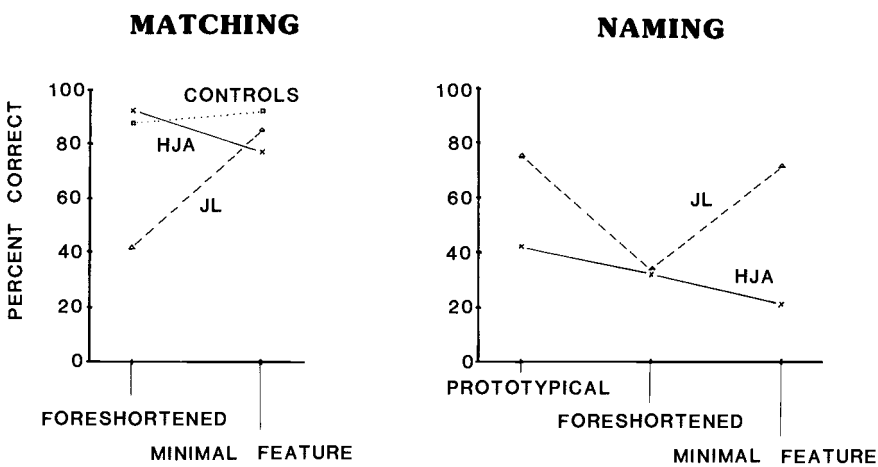

FIG. 2.5. Performance of JL, HJA and control subjects on object matching and naming tasks. Data from Humphreys & Riddoch (1984; 1985); the naming data consider omissions as errors.

SEMANTIC IMPAIRMENTS

Sometimes inability to recognise objects after brain injury can be caused by impairment to the semantic system itself. That is, knowledge of the object's category membership, functions, and so on, is degraded or inaccessible. This can be seen both in the findings of group studies and single-case studies.

Group Studies

Although patients with right hemisphere injuries can show impairments of object recognition to degraded or unusual views, De Renzi, Scotti, and Spinnler (1969) found that it was patients with left (rather than right) hemisphere injuries who were the most impaired when asked to match real objects to pictures of objects with very different appearances but the same names. This task, however, allows the match to be made at the level of object recognition units, semantic representations, or names. This problem of interpretation was eliminated by Warrington and Taylor (1978) who grouped stimuli, so that a photograph of an object was presented together with a photograph of a different object of a somewhat similar appearance and a photograph of another object with a similar function. When they were asked to pick the photograph of the object that matched the original object in function, patients with left and patients with right posterior injuries both showed significant impairments. For the patients with right posterior injuries, however, the impairments were

found to be linked to misidentifications of the photographed objects, whereas the patients with the left posterior injuries made few misidentifications but were still impaired on this matching by function task. Thus there is evidence of dissociable deficits of recognition and classification by function, or in Warrington's (1982) terms, of perceptual and semantic categorisation.

Single-case Studies

Single-case studies of recognition impairments due to damage to the semantic system were initially reported by Taylor and Warrington (1971) and Warrington (1975). The four cases described in these reports all involved progressive cerebral atrophy, and thus present particular problems of description and interpretation because of the changes in the patients' condition. Warrington (1975), however, points out that for these patients at least the pattern of abilities and disabilities remained constant as their condition deteriorated. In effect, what can be done with each patient is to present a "snapshot" of his or her abilities at a certain stage in the disease's progression. From these four cases we have selected that of a former civil servant, AB (Warrington, 1975), for discussion here because of the combination of relatively severe object recognition difficulties and at least some preserved intellectual abilities.

Initially, AB was referred to the National Hospital in London for investigation of his deteriorating memory. He did indeed show impairments on conventional memory tests, yet he obtained a verbal I.Q. of 122 (well above average). His understanding of verbal instructions was good and he was able to converse fluently using a somewhat impoverished vocabulary.

Performance on a variety of perceptual tests was within normal limits. AB's visual fields, visual acuity and hearing were normal. He was able to distinguish shapes, and to make figure-ground discriminations at various levels of background noise. He could also match pictures of different views of faces satisfactorily (25/28 correct), and on Warrington and Taylor's (1978) task of matching conventional and unusual views of objects his performance (17/20 correct) was also within normal limits. In our terms this would indicate a preserved ability to derive an adequate object-centred representation.

In contrast to this pattern of remarkably intact perceptual abilities, AB was severely impaired in recognition tasks. Although he could identify colours, numbers, and letters, he was unable to name successfully any of a series of 12 common objects, and could name only 2 of a set of 15 photographs of the faces of contemporary personalities. In addition to these visual object and face recognition problems he was also unable to

identify meaningful sounds such as a telephone ringing or a dog barking (2/12 correct). His comprehension of spoken words presented individually (i.e. without any supporting context to aid interpretation) was also impaired. Similarly, although he could read words he often did not understand what he had read, and he was also found to be poor at reading irregular words. He could, for instance, read "classification" but not "nephew". This combination of poor comprehension and difficulty with irregular words suggests that he relied a lot on spelling to sound correspondences in reading single words (see Chapter 8).

As well as being poor at object naming, AB was poor at naming pictures, and this deficit extended to both conventional (11/20 correct) and unusual (12/20 correct) views. When he could not identify an item he would usually state that it was "familiar" or that he had "forgotten" it. The other types of error mentioned by Warrington included being able to identify an object but not name it, being only able to place the item in a superordinate category (identifying a daffodil as "some kind of flower"; a hammer as "some kind of tool"), and substitutions of an incorrect item from the same category (identifying a donkey as a "horse"; a dog as a "cat").

When asked to identify the same items from pictures and spoken words (by naming the objects shown in the pictures or by describing the function of the same objects presented as spoken words) AB was impaired on both tasks, but was a little more successful with spoken words (27/40 correct) than with pictures (19/40 correct). The presence of an impairment on both tasks introduces the possibility favoured by Warrington (1975) that he is suffering from an impairment of semantic memory. This would correspond to an impairment of what we have called the semantic system and would imply some loss of knowledge of the "meanings" of objects (what they do, what they are made of, what category they belong to, where they are found, etc.).

In order to investigate this idea Warrington showed AB 40 photographs of animals and objects. These were given one at a time in random order, and he was asked to decide whether or not each was a photograph of an animal. The animal photographs were then presented one at a time and he was asked whether each one was a bird, whether each one was foreign, and whether each one was bigger than a cat. Similarly, with the object photographs he was asked whether each one was made of metal, used indoors, and heavier than a telephone directory.

The results of these tests are summarised in Table 2.1, together with the performance of five control subjects. AB is clearly impaired on most of the tests, but he is able to judge quite well whether or not photographs are of animals (37/40 correct). This confirms the observation that he could sometimes identify objects only to the level of a superordinate category

TABLE 2.1
Performance of Warrington's (1975) Patient AB on Semantic Judgement Tasks (Number of Items Correct)

		Items Presented as Photographs		*Items Presented as Spoken Words*	
Stimulus Items	*Task*	*AB's Performance*	*Mean Performance of Control Subjects*[a]	*AB's Performance*	*Mean Performance of Control Subjects*[a]
Animals and objects	Animal?	37/40	39.6	29/40	39.8
Animals	Birds?	13/20	19.6	15/20	19.4
	Foreign?	9/20	18.4	14/20	18.8
	Size?	11/20	16.0	13/20	15.2
Objects	Metal?	16/20	19.8	11/20	19.6
	Indoors?	18/20	19.4	15/20	19.2
	Weight?	8/20	14.8	12/20	15.8

[a](n=5)

(e.g. identifying a daffodil as "some kind of flower"). Further evidence of a selective semantic impairment comes from the finding from another task that AB was poor at defining low-frequency concrete words yet able to define low-frequency abstract words. He was, for instance, able to define abstract words like supplication ("making a serious request for help") and pact ("friendly agreement"), while being unable to define needle ("forgotten") or geese ("an animal but I've forgotten precisely").

AB thus presents a pattern of intact initial, viewer-centred and object-centred representations, together with impairments on semantic tasks, suggesting the possibility of a selective impairment of some aspects of semantic memory. Although his memory was also impaired in other ways these additional memory impairments are not in themselves a satisfactory explanation of his recognition difficulties, because even globally amnesic patients are not usually agnosic, as Warrington (1975) and Ratcliff and Newcombe (1982) point out.

Category-specific Semantic Impairments

A remarkable feature of semantic memory impairments is that, for some patients, they can be category-specific. The patient JBR from the series of four patients described by Warrington and Shallice (1984) forms a good example. Like Warrington and Shallice's (1984) other patients, JBR

was recovering from herpes simplex encephalitis, which causes extensive damage to the temporal lobes. His scores on intelligence tests were average (Verbal I.Q. 101, Performance I.Q. 103), though probably lower than his premorbid level (he had been an electronics undergraduate). He was amnesic, and disoriented in time and place. Like AB he could match conventional and unusual views well (20/20 correct), but performed poorly on tests of object recognition.

JBR's impairment was, however, particularly noticeable to living things. Table 2.2 shows data obtained when he was asked to identify 48 coloured pictures of animals and plants (living things), and 48 pictures of inanimate objects matched to the animals and plants for frequency of use as a word. He was then asked to define the same items when they

TABLE 2.2
Performance of Warrington and Shallice's (1984) Patient JBR at Identifying Objects from Coloured Pictures and Defining Them to Their Spoken Names (Percent Correct)

	Living Things	*Inanimate Objects*
Recognition from picture	6	90
Successful definition of spoken name	8	79

were presented to him as spoken words instead of pictures, and the data are again shown in Table 2.2. The superiority of inanimate objects over living things is most striking. JBR could define an item such as a compass ("tools for telling direction you are going"), yet produced the response "don't know" when asked what a parrot is. The category-specific impairment was found irrespective of whether JBR was tested on verbal description, naming, mimed responses, or picture–word matching. Often he could get superordinate information to living things (e.g. that a daffodil is a "plant"; a snail is "an insect animal"), but even in terms of access to the superordinate category he was still impaired in comparison with inanimate objects.

Warrington and Shallice (1984) are careful to make clear that the living/non-living distinction may not be the one that captures *every* aspect of these category-specific semantic impairments. JBR, for instance, was poor at identifying (inanimate) musical instruments yet good at (living) body parts. They suggest that semantic systems may be organised differently (and hence vulnerable to selective impairment) for things that have significance in terms of the way we use them (household objects, tools, etc.) and things that we know primarily in terms of their visual form (animals, plants, etc.). For those of us who are not musicians, musical

instruments, the inanimate category JBR was poor at identifying, are known primarily from their appearance whereas our own bodies, i.e. the parts of living things JBR could identify well, are used all the time.

PAUSE FOR CONTEMPLATION

So far, we have developed a model (Fig. 2.1) in which an initial representation of a seen object is used to construct viewer-centred and object-centred representations which have parallel access to stored descriptions of the structures of known objects (object recognition units), allowing access to semantic representations. We were then able to use this model to account for cases involving shape-processing impairments (which would traditionally be classified as involving apperceptive agnosia) in terms of impairment to the viewer-centred (and perhaps initial) representations, and to account for other object recognition impairments in terms of impairment to the semantic system itself. We were also able to account for some of the problems experienced by patients with right posterior cerebral lesions by proposing that they experience difficulties in constructing object-centred representations, and were able to explain why these patients are not agnosic (because access to object recognition units from viewer-centred representations remains unimpaired).

If we stopped discussing object recognition impairments now, everything would seem neat. But there are other cases in the literature that do not fit this tidy story so well. Because they force us to reconsider, and perhaps revise, our notions these cases are of exceptional theoretical interest. We will now discuss four such patients, two (MS and HJA) involving "higher-order" perceptual impairment, and two (JF and JB) involving a condition known as optic aphasia.

HIGHER-ORDER PERCEPTUAL IMPAIRMENT

MS had been a police cadet until he suffered a febrile illness. This left him blind in part of his former field of vision and with disturbed colour vision. He also had serious memory difficulties, but he was still able to achieve a verbal I.Q. of 101 (normal level). Object and face recognition were very poor. His case has been described by Newcombe and Ratcliff (1974, case 2) and Ratcliff and Newcombe (1982).

When shown a series of 36 line drawings of objects, MS was only able to name 8 of them correctly. This poor performance cannot be attributed solely to the area of blindness in his left visual field, because most other patients with comparable or even more severe visual field defects would experience little difficulty with a task of this type. For most (20) of these drawings he did not give any suggestions as to what the object might be,

but when he did make an error of identification it tended to resemble the stimulus. He thought, for example, that a drawing of an anchor showed an umbrella. The same types of error were evident to photographs and to real objects, though there were signs of improved performance for real objects (in a 10-item test he recognised 4 real objects, 1 photograph and 1 drawing). He was poor at describing the appearance of objects from memory. When asked to recognise objects presented in sense modalities other than vision, MS also showed an impairment in tactual recognition by both left and right hands, but his ability to recognise environmental sounds was within normal limits.

When Newcombe and Ratcliff (1974) asked MS to name each of the 36 objects used in their drawing naming task from a verbal description of its function or use he was able to name 20 correctly. This is certainly an improvement on the eight he could name from drawings, so that it does seem that any semantic or name retrieval problems are not sufficient to account for his agnosia. Further evidence for this conclusion comes from the finding (Ratcliff & Newcombe, 1982) that MS was much better able to make semantic judgements about printed words than pictures, which again suggests an impairment of object recognition over and above any semantic disturbance. However, MS did show uneven performance when asked to define objects himself. He could, for instance, explain successfully what an anchor is ("a brake for ships") but not what a nightingale is (Ratcliff & Newcombe, 1982); the possible parallel with Warrington and Shallice's (1984) patient JBR is obvious. These observations suggest that MS may also be experiencing some disturbance of the semantic system, but that (unlike JBR) it is not in itself sufficient to account for his agnosia.

The impairment of visual object recognition stands in marked contrast to some of MS' other abilities. In particular, we will draw attention to his preserved ability to read, and to his ability to copy drawings and to match identical stimuli. In each of these respects MS was quite unlike patients such as those described by Benson and Greenberg (1969) and Campion and Latto (1985).

On tasks involving naming printed words, MS was very accurate. This preserved ability to read is important because it suggests that the visual analysis needed to recognise words may be different to that required for object recognition, though Humphreys and Riddoch (1987a) have noted that reading *accuracy* is not a particularly sensitive measure, and that at least some agnosic patients may read accurately but letter-by-letter. The question as to the relation between reading impairments and the different types of visual agnosia may thus merit more systematic investigation.

MS also showed good ability to copy drawings and to match identical

stimuli. His copy of a picture of an anchor is shown in Fig. 2.6. This was an object he had identified as an umbrella, yet the copy is remarkably accurate. It was achieved, however, only by the use of considerable care and a line-by-line copying strategy; when drawing objects without a model to copy from, his attempts were poor (but not always unrecognisable). When asked to match objects as being the same or different to each other MS performed almost perfectly on visual, tactual and cross-modal (one object presented to vision and one by touch) matches.

His ability to copy and match stimuli successfully suggests that, in contrast to Benson and Greenberg's (1969) patient, MS was able to construct adequate viewer-centred representations of the objects he viewed. Despite the evidence of intact viewer-centred representations, however, MS failed to identify many objects and pictures (such as the anchor) for which shape alone would seem to provide a powerful cue. Thus MS would seem to have an impairment to the stored descriptions of the structures of known objects (object recognition units). This is also suggested by the fact that MS was poor at describing the appearance of objects from memory. In addition, however, object-centred representations seemed to be impaired for MS. This can be seen in his performance on Warrington and Taylor's (1978) task involving the matching of objects photographed from conventional and unusual views. On this task, MS's performance was close to chance level despite his good performance on simpler matching tasks, for which viewer-centred representations would be adequate.

MS's problems in object recognition seem to derive primarily from impairment of "higher" visual functions. We have argued that the principal causes of his visual agnosia might be a combination of impairments of object-centred representations and object recognition units. This view is consistent with those of Newcombe and Ratcliff (1974) and Ratcliff and Newcombe (1982), though our theoretical model differs from theirs in some respects. However, the second case of "higher-order" perceptual impairment we will consider, HJA, cannot be reconciled with our model so easily.

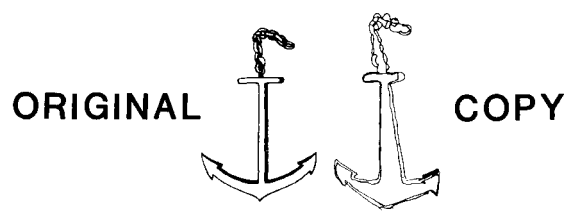

FIG. 2.6. Copy of a picture of an anchor by MS. He had misidentified the original as an umbrella. (Figure kindly supplied by Dr. F. Newcombe.)

Integrative Agnosia

HJA suffered a stroke when he had an appendicitis operation at the age of 61. Following this stroke he complained of loss of colour vision, impaired reading (initially, he could only read slowly and letter-by-letter), and severe problems in recognising objects and faces. Investigation of his visual fields revealed blindness in the upper-left and -right quadrants (i.e. he had lost the top half of the normal field of vision), but for the lower quadrants acuity was normal. Again, we must note that this visual field defect is not in itself a sufficient explanation of HJA's agnosia; there are other patients with the same visual field loss who can recognise objects without difficulty.

As well as having normal visual acuity, HJA showed normal discrimination of length, orientation and position. He was susceptible to visual illusions such as the Muller-Lyer and Ponzo illusions (which are often thought to depend on implied depth), and he could still see depth from disparity between images presented to each eye (stereopsis). Initial representation of perceptual qualities would thus seem intact.

HJA's object recognition impairments have been carefully investigated by Riddoch and Humphreys (1987a). He was better able to identify real objects (21/32) than photographs of the same objects from a prototypical view (12/32), and worst of all at recognising line drawings. When he could identify a stimulus it was usually only after careful, feature-by-feature examination, leading to response latencies of around 25 seconds for correct responses. All of his errors involved either misidentification as a visually similar item or omission (i.e. failure to come up with an answer). He could not mime the use of objects he could not identify.

Like MS, HJA could copy drawings of objects that he could not recognise. His copy of a drawing of an eagle, which he identified as "a cat sitting up", is shown in Fig. 2.7. To the extent that copying demands use of a viewer-centred representation, HJA's viewer-centred representations seem intact. Moreover, as Fig. 2.5 showed (using data from Humphreys & Riddoch, 1984), HJA's ability to match foreshortened to prototypical views of objects was also unimpaired; thus he seems able to construct some form of object-centred representation.

There was no evidence of any impairment to HJA's semantic system; he was easily able to define objects that he could not recognise. When asked what a duck is, for instance, he said that it:

> is a water bird with the ability to swim, fly and walk. It can be wild or kept domestically for eggs; when wild it can be the target of shooting. In the wild it has a wingspan between 15 and 18 inches and weighs about 2 or 3

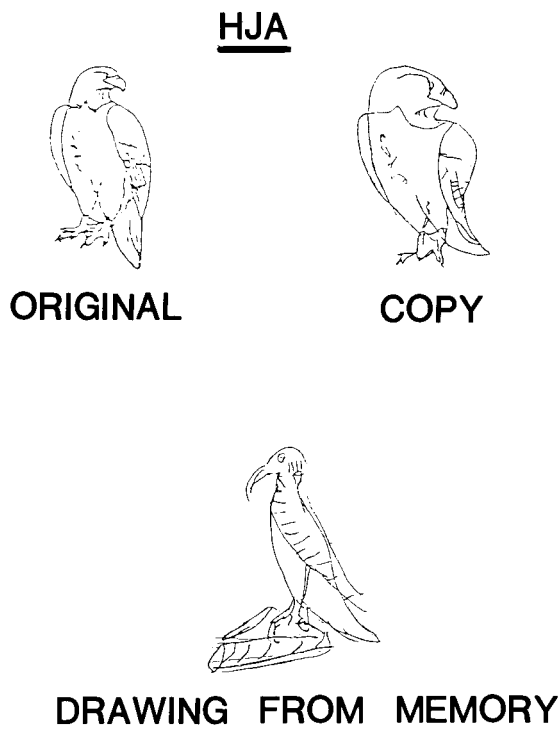

FIG. 2.7. HJA's copy of a picture of an eagle, and his drawing of an eagle from memory. (Reproduced with permission of Oxford University Press from Riddoch and Humphreys, 1987a.)

pounds. Domestic ducks are heavier, up to about 6 pounds perhaps. Wild ducks are multicoloured, mainly brown but with green and yellow breasts. Domestic ducks are white or khaki.

An interesting feature of HJA's object definitions was that, as above, they often included information about the *appearance* of the objects concerned. It seemed as if his stored knowledge of objects (i.e. object recognition units) was intact and accessible from the semantic system, and this was demonstrated convincingly by his ability to draw from memory. Figure 2.7 shows HJA's drawing of an eagle; clearly he remembers what eagles look like. Figure 2.7 is thus very informative about HJA's problems. He can see a drawing of an eagle well enough to copy it accurately, and he can remember what the bird looks like sufficiently well to draw it from memory, yet he cannot recognise an eagle when shown a picture of one. It is as if he can no longer achieve an accurate

mapping between his intact perception and his intact stored knowledge of the appearance of objects. We might argue, then, that object recognition units can no longer be accessed properly from viewer-centred or object-centred representations.

This does seem at least in part correct. HJA's ability to decide whether line drawings represented real objects or meaningless objects made by combining different real objects was at chance level (69/120; mean for control subjects 115.7/120). This finding conforms exactly to the view that he is no longer properly able to relate what he sees to his stored knowledge of visual forms.

We could fit HJA to our model by suggesting that intact object recognition units can no longer be accessed effectively from viewer-centred and object-centred representations which are themselves largely intact. To maintain that viewer-centred and object-centred representations are *intact* we would, however, have to overlook an aspect of his attempts at recognition which Riddoch and Humphreys (1987a) see as crucial—the laboured, feature-by-feature descriptions. These were sometimes accompanied by incorrect grouping of the local parts of objects. HJA thought, for instance, that a photograph of a paint-brush with a wooden handle might show "two things close together; a longish wooden stick and a darker, shorter object".

Riddoch and Humphreys (1987a) suggest that HJA finds it difficult to integrate local form information into a coherent overall visual description of the seen object. He picks up the details, but cannot "see" the whole properly. This idea is reminiscent of anecdotes in the literature in which agnosic patients complain that things they look at seem fragmented.

To support this "integration deficit" theory of HJA's difficulties, Riddoch and Humphreys (1987a) point out that his ability to recognise line drawings was reduced both by decreasing exposure duration and by overlapping one drawing on top of another. Moreover, he was better able to decide whether or not a silhouette (i.e. outline shape) represented a real or meaningless object than he was able to make the same decision about line drawings (63/88 silhouettes correct, whereas for line drawings he had been at chance level; mean for controls 77.73/88 silhouettes correct). It is as if the internal detail present in line drawings hindered HJA's performance instead of facilitating it; thus people with normal vision find line drawings easier than silhouettes in this task, but HJA found silhouettes easier than line drawings.

For Riddoch and Humphreys (1987a), then, HJA's perception is impaired, but it is impaired at the highest level of visual analysis. His is an integrative agnosia. He can pick up local features, shape cues, depth cues, and so on, but Riddoch and Humphreys think that he does not

readily integrate these into a coherent representation of what he is looking at.

This is an intriguing idea, and we note Young and Deregowski's (1981) suggestion that a similar process is quite generally implicated in picture perception, because under certain conditions children will pick up local depth and feature cues correctly but fail to integrate these into a coherent representation of the depicted object, leading to problems strikingly similar to those experienced permanently by HJA.

One possible way of accommodating the idea of an agnosic integration deficit to our model would be to propose that construction of an adequate object-centred representation involves at least two steps: (1) finding the object's axis of elongation; and (2) integrating local details correctly with respect to this. Patients with posterior lesions of the right hemisphere would then be impaired for the first step, but HJA only for the second step. We would still, though, have to propose that HJA has an additional impairment in accessing object recognition units from viewer-centred representations; perhaps integration is as important to the construction of effective viewer-centred representations as it is to object-centred representations.

More work will be needed before we can know how fruitful such speculations will be (see Humphreys & Riddoch, 1987b, for related suggestions). In the meantime, HJA illustrates well the challenges and insights that a well-documented case study can provide.

OPTIC APHASIA

Optic aphasia was first described by Freund in 1889. The key features of optic aphasia are problems in naming or verbally identifying visually presented objects, which are accompanied by the ability to demonstrate by miming their use that the objects have been recognised, and by unimpaired tactual naming. Thus we are faced in optic aphasia with a naming defect that is specific to the visual modality.

In January, 1970 JF, a retired French electrician, experienced visual disturbances and pins and needles on the right side of his body while driving his car. He had suffered a stroke involving the left posterior cerebral artery. At first he did not seek medical advice and resumed his normal life, though he had become blind in the right side of his former field of vision and unable to read. Some months later, however, he became suspicious and aggressive; he thought that his wife intended to poison him.

In the period following June, 1970 JF was examined at the Salpêtrière Hospital in Paris. He was found to have a complete loss of vision in his right visual field. No disturbances were evident in his spoken language or comprehension, and he obtained a verbal I.Q. of 93 on the Wechsler

Bellevue Test. He was also able to write adequately, both spontaneously and to dictation, and was able to draw well. He was, however, somewhat amnesic and showed a variety of memory difficulties on formal testing, with a Memory Quotient (M.Q.) of 77 on the Wechsler Memory Scale.

JF was found to have problems in naming visually presented stimuli which were carefully investigated by Lhermitte and Beauvois (1973). He was able to name objects presented tactually to his left or right hand (109/120 correct) and he could also name environmental sounds (24/25 correct). He was, however, impaired at naming colours, seen objects (23/30 corrrect), pictures (72/100 correct), and photographs of famous people's faces. He was also severely alexic, being able to name only a few letters and no words at all. When asked to define the spoken name of objects he had misnamed, however, he succeeded on 96 of 100 trials, thus clearly indicating that his object naming difficulty was not due to his impaired memory.

JF's poor performance on visual object naming tasks did not usually derive from failures to produce a name, but from the production of incorrect names. Various types of error were observed by Lhermitte and Beauvois. A number of these were perseverations, in which the name of a previously identified object was repeated; thus he called a fork a comb when it followed a comb in a series of objects, and he called a tomato a strawberry when it occurred a few trials after a strawberry in a series of fruits. He also made a lot of errors in which he produced the name of a semantically related object, including "shoes" for trousers and "grasshopper" for a slug. Some of his errors were visual in nature, such as "hazel nuts" for coffee beans, and there were others in which it was not entirely clear whether the production of the incorrect names was linked to visual or to semantic factors, or some combination of the two ("glass" for a bottle, "toothbrush" for a comb).

The most notable feature of the case, however, was that JF could indicate that he had understood what a visually presented object was, despite his inability to name it. He did this by miming its use. Thus, for instance, when shown a comb he correctly pretended to use it even though he called it a toothbrush. None of his mimes were found to be incorrect. When asked to draw an object he had just misnamed, he usually drew what he had seen rather than what he had called it. Although he seems to have been aware of his visual field defect and alexia, Lhermitte and Beauvois (1973) state that JF was apparently unaware of his problems in naming visually presented objects.

Optic aphasia poses a challenge to theories that postulate a common set of semantic representations for known objects that can be accessed from any sensory modality. The patient "knows" what seen objects are (in the sense of being able to gesture their use), and

knows what felt objects are, but can only name the ones that have been handled.

One way of resolving this paradox is to maintain that different parts of the semantic system can become disconnected from each other. Beauvois (1982) adopts this position. Her claim is that JF has more or less normal vision in the intact part of his visual field, and that visual semantic processes are normal. He also has normal speech and verbal semantic processes, but the visual semantic processes and the verbal semantic processes have become disconnected from each other, whereas tactile input still has access to verbal semantics.

The possibility of separation of visual and verbal semantic processes is supported by Schwartz, Marin, and Saffran's (1979) finding that a patient with a progressive dementing disease, WLP, could mime the use of objects despite a severe impairment of semantic memory. Her mimes were so precise that observers could distinguish easily her "use" of a depicted spoon or fork, pipe or cigarette, and so on. Yet WLP could not identify these objects verbally, or even show understanding of their names in a classification task.

Optic Aphasia as a Semantic Access Impairment

One problem with Beauvois' (1982) account of optic aphasia is how to make more clear the distinction between visual and verbal semantic processes. Work by Riddoch and Humphreys (1987b) has helped by suggesting a somewhat different basis for the distinction and a more precise hypothesis concerning a potential cause of optic aphasia.

Riddoch and Humphreys studied JB, who had sustained a left hemisphere injury in a road traffic accident. This left him unable to read or write, but with intact oral spelling. His speech was not affected, but he was initially amnesic. Although he had a right hemianopia, JB's vision did not seem to be otherwise much affected. He could copy simple drawings, and his ability to match prototypical to minimal-feature or foreshortened views of objects was within the range of control subjects (minimal-feature 26/26; foreshortened 20/26).

JB's ability to name seen objects was poor, and was unaffected by view (42% prototypical view; 40% foreshortened view; 43% minimal-feature view). Like JF, he made several semantic errors. He could, however, provide specific gestures indicating the uses of seen objects that he could not otherwise identify. Object naming from tactile presentation was better than naming from vision.

Thus far, the main difference between JB and the agnosic patient HJA is that whereas JB could mime the functions of the objects he saw, HJA could not. When JB was asked to decide whether line drawings repre-

sented real objects or meaningless objects made by combining different real objects, however, he was much better than HJA (HJA 69/120; JB 110/116—JB performed within the range of control subjects). Thus JB was able to access stored knowledge of object structures (object recognition units) from vision, whereas HJA could not do this. JB's performance on this object decision task remained in the normal range even when the task was made very difficult by deriving the meaningless objects from items in the same semantic category (for example, combining features of two different types of animal).

Riddoch and Humphreys (1987b) thus describe JB's impairment as involving semantic access from vision; although he can recognise objects as familiar, he is poorer at accessing semantic knowledge from pictures than from touch or from spoken names. This problem proved to be bidirectional: JB was equally poor at accessing knowledge of an object's appearance from semantics. His ability to draw objects from memory was poor, and he commented that "I know what it (the object he was asked to draw) is, but I just can't picture it."

For JB, then, the ability to mime the use of seen objects occurs in the context of intact access to object recognition units. It would seem that knowledge of how to use objects is linked to their structural rather than their semantic properties. Whether this would also have been true for Lhermitte and Beauvois' (1973) patient JF we do not, of course, know. It may be the case (as we are inclined to think) that intact access to object recognition units is *necessary* for correct gestures to be made, or it may be that there are different forms of optic aphasia. Only further detailed investigations of individual cases can reveal the answer.

Optic aphasia forces us to think more carefully about the different types of information we can access from seen objects. An outline of a possible account of optic aphasia is given in Fig. 2.8. Here we propose that intact miming reflects preserved access to a system of stored motor programs for object use, whereas the naming impairment reflects a disconnection between object recognition units and the (verbal) semantic system.

OVERVIEW

The basic lessons of cognitive neuropsychology, which will be encountered throughout this book, can be learnt from studies of disorders of object recognition.

The first lesson is that quite specific impairments can occur. There are patients who do not recognise the objects they see, yet have well-preserved language, memory, and other intellectual functions. These people are not blind and they can still use their vision effectively for many purposes,

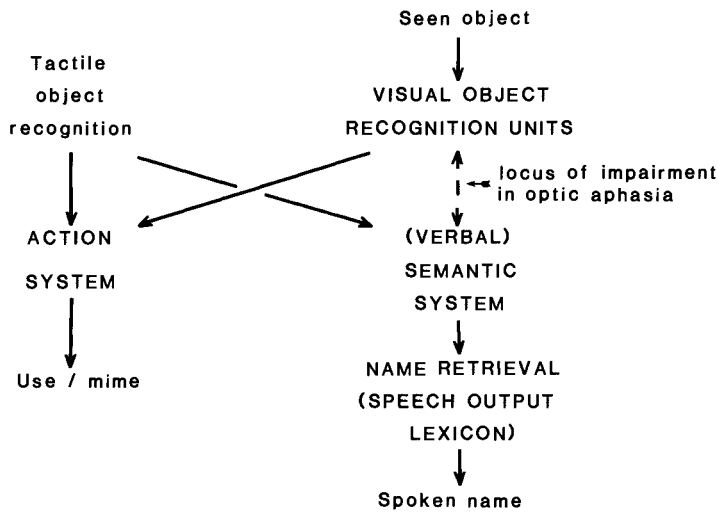

FIG. 2.8. A possible account of optic aphasia as a disconnection between visual object recognition and the verbal semantic system.

but the things they see have little meaning for them, or meanings that are only attained with difficulty. Such cases are rare, but this rarity is probably due to anatomical reasons (see Chapter 1). The key point is that they can exist at all.

Neuropsychological impairments do not, however, affect object recognition as if it were a single, homogeneous faculty. Instead, what we encounter are different *types* of recognition impairment. One person may be unable to perceive the shapes of seen objects properly, another can perceive shape but fails to form an effective integrated representation that combines local and global features, and another can recognise object forms as familiar and even mime their use, while remaining unable to give any verbal identification. The organisation of complex abilities such as object recognition seems to be into a number of separable functional components or modules, any one of which may be impaired selectively.

Of course, every neuropsychological patient is unique; exactly the same injury is no more likely to recur than people are likely to have the same fingerprints. But the patterns of impairment that are found do not turn out to be unlimited. There are, for instance, no accounts of patients who show impaired processing of shape information and yet recognise seen objects without difficulty. Thus, among the myriad of observed patterns, it is possible to discern a sense of order.

The easiest way to comprehend this orderliness of neuropsychological breakdown is in terms of an explicit theoretical model. A satisfactory model needs to explain what functional components are involved in the ability of interest, and how these are organised with respect to each other. It should be able not only to account for the patterns of impairment observed, but also for those that are *not* found. In our model (Fig. 2.1), for instance, it is clear that anyone who shows impaired shape processing *must* have impaired object recognition ability, because the viewer-centred representations which would include shape information have to be constructed before recognition can take place. The demonstration of one case in which this was not so would be sufficient to force revision (or abandonment) of a model of this type.

With a reasonably adequate theoretical model, then, it is possible to account for existing patterns of impairment, to predict new types of pattern that might be found, and to use newly discovered patterns that do not conform to revise our understanding. That is why cognitive neuropsychology can be so exciting.

SUMMARY

We can think of object recognition as requiring that viewer-centred and object-centred representations of seen objects are matched to stored descriptions of the structures of known objects (object recognition units) which then allow access to semantic representations (see Fig. 2.1). Cases in which there is a severe impairment of form perception and inability to copy seen objects may then be considered to involve impairment of the ability to construct viewer-centred representations. Problems with matching unusual or foreshortened views derive from impaired object-

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VIDEOS

Two videos in the series edited by E. Funnell & G.W. Humphreys: *Teaching programmes in cognitive neuropsychology* (Hove, UK: Lawrence Erlbaum Associates Ltd.) illustrate topics discussed in this chapter. They are *Peripheral agnosia: disorders of object recognition* and *Central agnosia: loss of knowledge about objects.*

3 Visual and Spatial Abilities

INTRODUCTION

In Chapter 2 we looked at what cognitive neuropsychological studies have revealed about how we recognise objects. However, for normal life we use vision for a much wider range of purposes. We can recognise other types of stimuli than everyday objects, such as printed words or people's faces, we can see colour and movement, and we can assess accurately the locations of seen objects both with respect to ourselves and in relation to each other.

Our visual experience has such a unified quality that it is initially surprising to discover that, as for object recognition, a range of quite specific impairments of visual information processing can be found after brain injury. The processes responsible for vision are organised into a number of functionally separable modules. This modular organisation makes good sense in terms of keeping the nerve connections involved as tidy as possible and in terms of allowing one process to be modified without affecting others that are unrelated to it (Cowey, 1985; Marr, 1982), and there is good agreement between studies of humans and studies of other species concerning the types of functional module that exist (Cowey, 1982; 1985; Ratcliff & Cowey, 1979; Zeki, 1978). None the less, one cannot help being puzzled as to how our visual experience can have its unified quality when it is produced by a number of separate processing modules. We will not attempt to answer this question here, but will concentrate instead on introducing some of the dissociable disorders that can be observed. Impairments of word and face processing are dealt with

elsewhere (Chapters 4, 5, 6, and 7), and in this chapter we examine some of the other deficits that are found. We will not discuss every possible disorder; for those seeking it, a more detailed listing can be found in Benton (1979). Instead, we will illustrate the range of problems that can arise. We begin with colour processing and movement perception, which may be considered to be fairly "basic" visual abilities. These are followed by a discussion of the phenomena associated with "blindsight", which have important implications for our conception of the relation between visual experience and perceptual mechanisms. We then turn to look at impairments affecting abilities that are best considered to be more "spatial" in nature—visual location, spatial attention, and spatial knowledge and thought.

Although modular organisation is a widely agreed feature of visual and spatial abilities, there is as yet no generally accepted theoretical framework that can specify the complete underlying pattern (see Young & Ratcliff, 1983). Obviously, the various processing modules must be interconnected into some kind of coherent system, but we do not yet know how. An important clue, however, is that mechanisms responsible for the recognition of different types of visual pattern seem to be organised into separate systems to those involved in space perception. Thus patients experiencing object recognition difficulties may be relatively unimpaired on spatial tasks, and patients who are spatially disorientated may yet remain able to identify objects without difficulty.

Such observations are borne out by more formal studies. Newcombe and Russell (1969), for instance, studied a group of men with brain lesions due to shrapnel and gunshot wound injuries sustained some 20 years previously. They found that men with right hemisphere injuries could show severe problems in spatial tasks (such as maze learning) or in more directly "visual" tasks likely to relate to pattern recognition, but that these deficits did not relate to each other. Newcombe, Ratcliff, and Damasio (1987) present detailed information on two cases from this series, one with each type of impairment. Such findings, and related studies of the monkey's brain, have been developed by Ungerleider and Mishkin (1982) into the view that there are effectively parallel visual systems in the cerebral cortex responsible for appreciation of an object's identity and its spatial location. Interestingly, Levine, Warach, and Farah (1985) have pointed out that the same dissociation occurs for impairments affecting mental imagery.

COLOUR PROCESSING

Impairments of colour processing due to brain injury can be grouped into three main types affecting what we might roughly describe as colour perception, colour knowledge and colour naming. Of course, many

patients experience what would have to be regarded as hybrid impairments under this classification scheme, but this seems justifiable as some relatively pure cases have been described.

Impairments of colour *perception* are called achromatopsias. Patients with cerebral achromatopsia complain that they cannot see colours, and that everything is like a black and white picture, or that colours have lost their brightness (Meadows, 1974b). In some cases the loss of colour perception relates only to part of the field of vision (Damasio, 1985). Even when the whole of the visual field is involved, however, everyday objects can still be recognised provided that colour is not a critical cue; there need not be any impairment of form perception itself. Pallis's (1955) patient, for instance, stated that he could usually identify everyday objects, but gave as examples where his loss of colour perception caused him problems the fact that his shirts all looked dirty and he could not tell them apart, and that he could not tell until he had opened a jar (and smelt or tasted the contents) whether it would contain jam or pickles. Warrington (1987) provides documented cases of dissociations between impairments affecting the processing of colours, locations and shapes.

The cerebral achromatopsias are quite different to the types of colour blindness found in the absence of brain damage, which are due to deficiencies in the retina of the eye (Mollon, 1982). Mollon, Newcombe, Polden, and Ratcliff (1980) showed, for instance, that the patient MS, who experienced achromatopsia as well as his object agnosia (see Chapter 2), none the less retained the usual three functional cone mechanisms with normal spectral sensitivities (cones are the retina's colour-sensitive cells). In threshold tasks MS was found to be able to respond to signals from any of the three classes of cone normally associated with colour blindness; his complete achromatopsia was thus due to a deficit affecting his ability to make proper *use* of the signals that the different types of cone continued to send. He could respond to wavelength without being able to match, sort, or name different hues.

Clinically, achromatopsias are often found in conjunction with problems in recognising familiar faces (prosopagnosia) and familiar places. In some cases, however, colour perception may be impaired without loss of ability to recognise faces or places and, conversely, it remains intact in some reported cases of prosopagnosia (Heywood, Wilson, & Cowey, 1987; Meadows, 1974a; 1974b; see Chapter 4 for further discussion of this point). Thus it seems that these functions are carried out by separate information processing modules, but that these lie in adjacent or even partially overlapping cortical areas that are often damaged simultaneously.

A quite different type of deficit seems to involve impairment of colour *knowledge*. For these patients colour perception is intact, but errors are made in tasks that demand use of stored information about colour.

Patients may be unable to answer from memory questions such as "What colour is a strawberry?", and may choose the wrong coloured crayons if asked to colour in line drawings of the objects concerned (Kinsbourne & Warrington, 1964; Oxbury & Humphrey, 1969, case 2). This deficit often occurs in the context of more general language difficulties (De Renzi, Faglioni, Scotti, & Spinnler, 1972), but, apparently, it can also occur in an isolated form (see Meadows, 1974b). Unlike achromatopsia, the impairment is not found only in "perceptual" tasks, because it is as great in what is apparently a purely verbal task such as stating the colour of a specified object—a task which would not cause an achromatopsic patient particular difficulty.

Impairments of colour knowledge need to be distinguished from problems affecting colour *naming*. Geschwind and Fusillo (1966) described a 58-year-old man who, following a stroke, became unable to name seen colours. This patient could state the usual colours of familiar objects like bananas, or the sky, but he could not put a name to the colour of objects (such as items of clothing) for which there was no learnt association to rely on. Similarly, he could not point to the correct colour when a particular colour name was specified.

These colour naming problems are often found in combination with forms of reading impairment involving letter-by-letter reading, or the complete inability to read. Geschwind and Fusillo's (1966) patient was also unable to read (alexic), and they interpreted his colour naming defects as demanding a similar explanation to his reading problems; in essence, their argument is that there is a disconnection between the brain's visual and language areas. Colour naming problems and alexia do, however, dissociate in rare cases which weaken Geschwind and Fusillo's (1966) argument. For example, Greenblatt (1973) described an alexic patient who could name colours, whereas Mohr, Leicester, Stoddard, and Sidman's (1971) patient could read but was impaired at colour naming. Davidoff and Ostergaard (1984) describe a patient with a colour naming impairment who was none the less able to point to named colours, which again suggests that the explanation of colour anomia is not as simple as disconnection theories often assume. It may prove more enlightening to link impairments of colour naming to other reports of naming disorders specific to certain semantic categories (see Chapter 5).

Colour processing impairments, then, both demonstrate the separability of colour processing from other aspects of vision, and form an interesting and reasonably coherent pattern. Much more work needs to be done, however, before an adequate model of the deficits can be proposed, because there are already signs that the simple classification scheme we have adopted need not always apply (see, especially, Beauvois & Saillant, 1985).