

INTRODUCTION TO NEUROPSYCHOLOGY

Introduction to
Neuropsychology

SECOND EDITION

J. GRAHAM BEAUMONT



THE GUILFORD PRESS
New York London

© 2008 The Guilford Press
A Division of Guilford Publications, Inc.
72 Spring Street, New York, NY 10012
www.guilford.com

All rights reserved

No part of this book may be reproduced, translated, stored in a retrieval system, or transmitted, in any form or by any means, electronic, mechanical, photocopying, microfilming, recording, or otherwise, without written permission from the Publisher.

Printed in the United States of America

This book is printed on acid-free paper.

Last digit is print number: 9 8 7 6 5 4 3 2 1

Library of Congress Cataloging-in-Publication Data

Beaumont, J. Graham.

Introduction to neuropsychology / J. Graham Beaumont. — 2nd ed.
p. cm.

Includes bibliographical references and index.

ISBN 978-1-59385-068-5 (cloth: alk. paper)

1. Neuropsychology—Textbooks. I. Title.

QP360.B413 2008

612.8—dc22

2007052765

. . . And I have felt
A presence that disturbs me with the joy
Of elevated thoughts; a sense sublime
Of something far more deeply interfused,
Whose dwelling is the light of setting suns.
And the round ocean and the living air,
And the blue sky, and in the mind of man.

—WILLIAM WORDSWORTH,
from *Lines Composed a Few Miles
above Tintern Abbey*

About the Author

J. Graham Beaumont, PhD, CPsychol, FBPsS, is Head of the Department of Clinical Psychology at the Royal Hospital for Neuro-disability in Putney, London, United Kingdom. Widely published, Professor Beaumont is a past Honorary General Secretary of the British Psychological Society and Chair of the Division of Neuropsychology, and holds an Honorary Chair at Roehampton University, London. He was formerly a Reader and Associate Dean for Combined Science at the University of Leicester and Professor and Head of the Department of Psychology at Swansea University.

Preface to the First Edition

This book was born out of my frustration at being unable to find a text to accompany the courses that I teach in neuropsychology to undergraduates, graduate clinical psychology trainees, and other medical and paramedical groups. Its first aim is therefore to provide a systematic and comprehensive introduction to the field of neuropsychology for those with some, perhaps not very advanced, knowledge of psychology.

At the same time, I wanted to produce a book that would make the current advances in neuropsychology accessible to the intelligent layman, without sacrificing critical standards of good science by “popularizing” the material. I wanted to communicate, if I could, some of the excitement that I feel about this area of investigation. Only you can judge whether I have succeeded in this ambitious aim.

In trying to cover, in a balanced way, what I consider to be the whole subject of neuropsychology within a fairly short book, I have naturally met problems. The limited space has dictated rather severe compression of the material in places, and more examples and further elaboration would probably have been helpful. However, every book has a purpose, and the purpose of this one is to provide a thorough but concise introduction to the subject. I hope that you will not feel that readability has suffered unreasonably in trying to fulfill this aim.

Throughout the book, there are both references and suggestions for further reading to enable the reader to pursue specific topics and particular areas of interest. This may help to fill some of the gaps that are inevitably left in a text of this length.

One limitation that has been accepted in preparing this text is that it deals only with adult neuropsychology. There is quite deliberately almost no reference to children. Sadly, it is not even possible to recommend an alternative text for those whose prime interest is the neuropsychological organization of children in normal or abnormal states. That is another book waiting to be written.

Another point—in one sense an apology—is that the text uses the male gender “he” throughout to imply male or female individuals. There comes a time when the syntactic maneuvers required to avoid any use of “he” or “she” become burdensome, and until some better neutral article than “s/he” is found, it seems reasonable to carry on using “he.” No particular stance on sexual politics is implied by this, and I hope that you will accept what has been written in good faith as simply a traditional (and I think fairly harmless) usage within the English language.

A word also about the figures. First, and most important, I should like to thank my talented friend Don Keefe for his excellent work in preparing these. Although all the anatomical figures are original drawings, they are inevitably in some debt to two quite exceptional sets of anatomical illustrations: the collections of Nieuwenhuys, Voogd, and Van Huijzen (1988), and Netter (1983) (see “Further Reading” on p. 41). The example items from test material are also, with some exceptions that are noted, original drawings. They are based upon real test material but, because it is considered unethical to expose this material unnecessarily since it may undermine the accuracy of the test in clinical practice, most of the examples have been slightly altered.

There are many others whom I want to thank for their help with the book. There are those people who have kindly given permission for the reproduction of figures. Dr. Jennifer Wakely was extremely helpful in arranging for the photographs of normal brain specimens. Professor Tony Gale read the manuscript and made many perceptive and constructive suggestions which have been particularly valuable. The publishers have been unfailingly patient, encouraging, and helpful, and I am especially grateful to them. Lastly, my family, colleagues, students, and friends have had to tolerate my reclusiveness and frequent ill humor during the preparation of the book, and they have been unreasonably understanding and kind.

It only remains to say that I hope that you not only find the book informative, but that you enjoy reading it. I especially hope that some readers may become sufficiently interested by this introduction to continue their study of neuropsychology, and may become those who will develop the subject toward solving some of the great and fascinating problems that remain in understanding the relationship between the brain and intelligent behavior.

Preface to the Second Edition

Just 25 years after writing the preface to the first edition of this book, I have now completed a thorough revision of the original text. The previous edition was embarrassingly out of date, but I am pleasantly surprised that it has remained in print, and this has encouraged me to give it a thorough overhaul. The first edition was probably the first concise general text on neuropsychology for undergraduates, at least in the United Kingdom, and despite the subsequent proliferation of other texts (the best of which are listed at the end of Chapter 1), there still seems to be a need for a concise and readable, but authoritative, introduction to neuropsychology. I hope that this is it.

In revising this text, it has been fascinating to reflect on the changes in neuropsychology over the past 30 years. In the 1970s, when I was a young academic, neuropsychology was a relatively new discipline in the universities and existed only as a clinical specialty. It is now an established field of psychological research in its own right, with rich links to other areas of psychology and cognate disciplines. Professionally, the specialty of clinical neuropsychology is now formally recognized, and training programs are available worldwide to prepare psychologists for this area of professional practice. In the last 30 years there has been a staggering growth in neuropsychology in parallel with the development of the neurosciences. It has been an inspiring and exciting period, but it has made revision of the text a rather daunting challenge. In the 1970s it was possible for one person to have a grasp of all the relevant literature in neuropsychology; today, it is no longer the case.

These developments in the discipline have been reflected in some major additions to the book. There is a new chapter on degenerative diseases and profound brain injury, hardly a topic within neuropsychology in 1980, and a major section has been introduced on imaging to reflect the very considerable impact that clever developments in medical physics are having on research in the discipline. Advances in clinical neuropsychological practice are reflected in substantial changes to the relevant chapter, and these changes reflect the increasing role of rehabilitation; it is an embarrassment to remember just how neglected this topic was until the 1980s. Neuropsychiatry has also become established as a recognized discipline since the first edition of this book, and its convergence with neuropsychology is reflected in the final chapter. The remainder of the text has been subject to detailed revision and updating, with substantial additions to the chapter on the frontal lobes to reflect more recent interest in executive functions and attention. The extensive references have also been updated. Sexist language is no longer acceptable and has been removed.

I make no apology for the fact that the text still contains a balance between clinical and experimental neuropsychology. Contrary to some of the advice that I have received, I still think it important that students are educated in both aspects of the discipline, even though the traditional experimental approaches are currently somewhat out of fashion (although there are signs of a sea change) and imaging studies are very much the vogue. For the same reason I have retained many of the older references that are still of relevance and importance. Too many of my students believe that nothing published before 2000 is worth reading; they are wrong. Psychology, generally, is in danger of losing contact with its rich intellectual heritage, and an appreciation of the history of neuropsychology is critical to a full understanding of contemporary concerns.

I must thank Rochelle Serwator at The Guilford Press for her sustained enthusiasm for the project and unfailing patience with my delays, and Jennifer DePrima and Louise Farkas for their efficient editing of the text. I wish to warmly thank Professor Andrew Mayes, Lisa Williams, MA, and Antonio E. Puente, PhD, for their perceptive and helpful comments on the manuscript. The Institute of Neuro-palliative Rehabilitation at the Royal Hospital for Neuro-disability permitted me some time during the tenure of an international fellowship to work on the text, for which I am grateful. I also owe a debt to my professional colleagues at the Royal Hospital, without exception a talented group of friends, for their inspiration and support. Roehampton University has provided an academic base and given me the opportunity for continued

exposure to the stimulus of undergraduate and postgraduate students. Most importantly, I must thank Pamela for inspiring me to complete this revision and for helpful and intellectually challenging discussions that have greatly enriched my knowledge and sustained me through difficult times.

The one thing that has not changed in 25 years is my wish to inspire students with an interest in neuropsychology to grapple with the intellectual questions that the discipline poses and to share the satisfactions that I have enjoyed from being a neuropsychologist. I hope this book continues to contribute toward these goals.

Contents

PART I. INTRODUCTION

| | |
|--|----|
| CHAPTER 1. The Discipline of Neuropsychology | 3 |
| <i>What Is Neuropsychology?</i> | 3 |
| <i>Branches of Neuropsychology</i> | 4 |
| <i>Comparative Neuropsychology</i> | 5 |
| <i>Conceptual Issues</i> | 6 |
| <i>Historical Background</i> | 8 |
| <i>Clinical Neuropsychology</i> | 11 |
| <i>Experimental Neuropsychology</i> | 14 |
| <i>Cognitive Neuropsychology</i> | 17 |
| <i>The Fringe</i> | 18 |
| <i>The Plan of This Book</i> | 19 |
| <i>Conclusion</i> | 20 |
| CHAPTER 2. The Structure of the Central Nervous System | 22 |
| <i>Terminology</i> | 23 |
| <i>The Environment of the CNS</i> | 24 |
| <i>The Spinal Cord</i> | 26 |
| <i>Divisions of the Brain</i> | 27 |
| <i>The Brain Stem</i> | 29 |
| <i>The Cerebellum</i> | 31 |
| <i>The Diencephalon—The Subcortical Forebrain</i> | 31 |
| <i>The Telencephalon—The Cerebral Cortex</i> | 35 |
| <i>Conclusion</i> | 40 |

PART II. CLINICAL STUDIES

| | |
|---|-----|
| CHAPTER 3. The Frontal Lobes | 45 |
| <i>Some Methodological Issues</i> | 45 |
| <i>Intelligence</i> | 48 |
| <i>Specific Functions</i> | 51 |
| <i>The Motor and Premotor Cortex</i> | 52 |
| <i>The Prefrontal Cortex</i> | 54 |
| <i>Broca's Area</i> | 61 |
| <i>The Orbital Cortex</i> | 61 |
| <i>Modern Theories of Frontal Lobe Function</i> | 64 |
| <i>Lateralization of the Frontal Lobe</i> | 67 |
| <i>Conclusion</i> | 68 |
| CHAPTER 4. The Temporal Lobes | 72 |
| <i>Audition</i> | 74 |
| <i>Vision</i> | 78 |
| <i>Attention</i> | 78 |
| <i>Memory</i> | 80 |
| <i>Personality</i> | 89 |
| <i>Conclusion</i> | 91 |
| CHAPTER 5. The Parietal Lobes | 95 |
| <i>Somatosensory Perception</i> | 96 |
| <i>Tactile Perception and Body Sense</i> | 98 |
| <i>Spatial Orientation</i> | 101 |
| <i>Spatial Neglect</i> | 104 |
| <i>Symbolic Syntheses</i> | 108 |
| <i>Apraxia</i> | 109 |
| <i>Intersensory Association</i> | 113 |
| <i>Short-Term Memory</i> | 113 |
| <i>Gerstmann Syndrome</i> | 113 |
| <i>Language</i> | 114 |
| <i>Conclusion</i> | 114 |
| CHAPTER 6. The Occipital Lobes | 118 |
| <i>Anatomical Divisions</i> | 118 |
| <i>Basic Visual Functions</i> | 121 |
| <i>Visual Perceptual Functions</i> | 123 |
| <i>Blindsight</i> | 127 |
| <i>Visual Prostheses</i> | 128 |
| <i>Conclusion</i> | 129 |
| <i>Integration of Cortical Functions</i> | 130 |
| CHAPTER 7. Language | 134 |
| <i>The Lateralization of Language</i> | 134 |
| <i>Varieties of Aphasia</i> | 136 |

| | | |
|--------------------------------|---|-----|
| | <i>Anatomical Structures</i> | 139 |
| | <i>The Forms of Aphasia</i> | 141 |
| | <i>Aphasia Assessment</i> | 150 |
| | <i>The Rehabilitation of Aphasics</i> | 151 |
| | <i>Conclusion</i> | 153 |
| CHAPTER 8. | Degenerative Diseases and Profound Brain Injury | 159 |
| | <i>Multiple Sclerosis</i> | 160 |
| | <i>Parkinson's Disease</i> | 162 |
| | <i>Huntington's Disease</i> | 165 |
| | <i>Profound Brain Injury</i> | 167 |
| CHAPTER 9. | The Subcortex and Psychosurgery | 180 |
| | <i>Neurosurgery</i> | 182 |
| | <i>Psychosurgery</i> | 185 |
| | <i>Conclusion</i> | 191 |
| | | |
| PART III. EXPERIMENTAL STUDIES | | |
| CHAPTER 10. | Split Brains and Dual Minds | 199 |
| | <i>The Commissurotomy Operation</i> | |
| | <i>and the Patients</i> | 200 |
| | <i>The Effects of Commissurotomy</i> | 202 |
| | <i>Consciousness Divided?</i> | 211 |
| | <i>Conclusion</i> | 215 |
| CHAPTER 11. | Divided Visual Field Studies | 220 |
| | <i>The Technique</i> | 221 |
| | <i>The Evidence</i> | 224 |
| | <i>The Theories</i> | 234 |
| | <i>Conclusion</i> | 239 |
| CHAPTER 12. | Dichotic Listening | 245 |
| | <i>The Technique</i> | 245 |
| | <i>Lateral Ear Asymmetries</i> | 247 |
| | <i>Attention</i> | 252 |
| | <i>An Index of Lateralization</i> | 255 |
| | <i>Conclusion</i> | 256 |
| | <i>Other Methods</i> | |
| | <i>in Experimental Neuropsychology</i> | 257 |
| CHAPTER 13. | Electrophysiology and Imaging | 263 |
| | <i>Electrophysiology</i> | 263 |
| | <i>Minor Physiological Techniques</i> | 279 |
| | <i>Brain Imaging</i> | 280 |
| | <i>Conclusion</i> | 285 |

| | |
|---|-----|
| CHAPTER 14. Individual Differences: Gender and Handedness | 292 |
| <i>Gender Differences in Cerebral Organization</i> | 292 |
| <i>Handedness</i> | 297 |
| <i>Conclusion</i> | 309 |
| | |
| PART IV. APPLICATIONS | |
| CHAPTER 15. Neuropsychological Practice | 317 |
| <i>Testing for Brain Damage</i> | 318 |
| <i>The Assessment of Specific Functions</i> | 323 |
| <i>Assessment in Practice</i> | 329 |
| <i>Rehabilitation</i> | 332 |
| <i>Conclusion</i> | 336 |
| CHAPTER 16. Neuropsychiatry and Neuropsychology | 340 |
| <i>Neuropsychiatry</i> | 341 |
| <i>Functional States and Laterality</i> | 345 |
| <i>Theoretical Models</i> | 350 |
| <i>Conclusion</i> | 354 |
| | |
| Author Index | 360 |
| Subject Index | 370 |
| Index of Tests and Procedures | 380 |

PART I

INTRODUCTION

The Discipline of Neuropsychology

WHAT IS NEUROPSYCHOLOGY?

The human brain is a fascinating and enigmatic machine. Weighing only about 3 pounds (1.36 kilograms) and with a volume of about 1,250 cubic centimeters, it has the ability to monitor and control our basic life support systems, to maintain our posture and direct our movements, to receive and interpret information about the world around us, and to store information in a readily accessible form throughout our lives. It allows us to solve problems that range from the strictly practical to the highly abstract, to communicate with our fellow human beings through language, to create new ideas and imagine things that have never existed, to feel love and happiness and disappointment, and to experience an awareness of ourselves as individuals. Not only can the brain undertake such a variety of different functions, but it can do more or less all of them simultaneously. How this is achieved is one of the most challenging and exciting problems faced by contemporary science.

It has to be said at the outset that we are completely ignorant of many of the things that the brain does, and of how they are done. Nevertheless, very considerable advances have been made in the neurosciences over the last decade or two, and there is growing confidence among neuroscientists that a real understanding is beginning to emerge. This feeling is encouraged by the increasing integration of the various disciplines involved in neuroscience, and a convergence of both experimental findings and theoretical models.

Neuropsychology, as one of the neurosciences, has grown to be a separate field of specialization within psychology over about the last 40 years, although there has always been an interest in it throughout the 120-year history of modern scientific psychology. Neuropsychology seeks to understand the relationship between the brain and behavior, that is, it attempts to explain the way in which the activity of the brain is expressed in observable behavior. What mechanisms are responsible for human thinking, learning, and emotion, how do these mechanisms operate, and what are the effects of changes in brain states upon human behavior? There are a variety of ways in which neuropsychologists conduct their investigations into such questions, but the central theme of each is that to understand human behavior we need to understand the human brain. A psychology without any reference to physiology can hardly be complete. The operation of the brain is relevant to human conduct, and the understanding of how the brain relates to behavior may make a significant contribution to understanding how other, more purely psychological, factors operate in directing behavior. Just how the brain deals with intelligent and complex human functions is, in any case, an important subject of investigation in its own right, and one that has an immediate relevance for those with brain injuries and diseases, as well as a wider relevance for medical practice.

BRANCHES OF NEUROPSYCHOLOGY

Neuropsychology is often divided into two main areas: clinical neuropsychology and experimental neuropsychology. The distinction is principally between clinical studies, on brain-injured subjects, and experimental studies, on normal subjects, although the methods of investigation also differ. The division between the two is not absolutely clear-cut but it helps to form an initial classification of the kinds of work in which neuropsychologists are involved.

Clinical neuropsychology deals with patients who have *lesions* of the brain. These lesions may be the effects of disease or tumors, may result from physical damage or *trauma* to the brain, or be the result of other biochemical changes, perhaps caused by toxic substances. Trauma may be accidental, caused by wounds or collisions; it may result from some failure in the vascular system supplying blood to the brain; or it may be the intended result of neurosurgical intervention to correct some neurological problem. The clinical neuropsychologist measures deficits in intelligence, personality, and sensory–motor functions by specialized testing procedures, and relates the results to the particular areas of the brain that have been affected. The damaged areas may be clearly circum-

scribed and limited in extent, particularly in the case of surgical lesions (when an accurate description of the parts of the brain that have been removed can be obtained), or may be diffuse, affecting cells throughout much of the brain, as is the case with certain cerebral diseases. Clinical neuropsychologists employ these measurements not only in the scientific investigation of brain–behavior relationships, but also in the practical clinical work of aiding diagnosis of brain lesions and rehabilitating brain-injured patients.

Behavioral neurology, as a form of clinical neuropsychology, also deals with clinical patients, but the emphasis is upon conceptual rather than operational definitions of behavior. The individual case rather than group statistics is the focus of attention, and this approach usually involves less formal tests to establish qualitative deviations from “normal” functioning. Studies in behavioral neurology may often sample broader aspects of behavior than is usual in clinical neuropsychology.

The distinction between clinical neuropsychology and behavioral neurology is not entirely clear, and it is further blurred by the historical traditions of investigation in different countries, particularly in the United States, the former Soviet Union, and Great Britain. Examples of clinical work in these countries are discussed below and in Chapter 15.

By contrast, *experimental neuropsychologists* work with normal subjects with intact brains. This is the most recent area of neuropsychology to develop and has grown rapidly since the 1960s, with the invention of a variety of techniques that can be employed in the laboratory to study higher functions in the brain. There are close links between experimental neuropsychology and general experimental and cognitive psychology, and the laboratory methods employed in these three areas have strong similarities. Subjects are generally required to undertake performance tasks while their accuracy or speed of response is recorded, from which inferences about brain organization can be made. Associated variables, including psychophysiological or electrophysiological variables, may also be recorded.

COMPARATIVE NEUROPSYCHOLOGY

Although the subject of this book is human neuropsychology, it should not be forgotten that much experimental neuropsychology has been conducted with animals, although this form of research is now in decline. At one time, the term *neuropsychology* was in fact taken to refer to this area, but it is now used more generally and the relative importance of the animal studies of comparative neuropsychology has decreased. The obvious advantage of working with animals, ethical issues apart, is that

precise lesions can be introduced into the brain and later confirmed by histology. Changes in the animal's behavior are observed and can be correlated with the experimental lesions. The disadvantages are the problems of investigating high-level functions using animals as subjects (the study of language is ruled out, to take the most obvious example) and the difficulty of generalizing from the animal brain to the human brain. Although it may be possible to discover in great detail how some perceptual function is undertaken in the brain of the rat, the cat, or the monkey, it may not necessarily be undertaken in the same way in the human brain. There are also basic differences in the amount and distribution of different types of cortical tissue in the brains of the various animals and of humans, which add to the difficulties of generalization.

Nevertheless, animal studies continue to be important, particularly with regard to the functions of subcortical systems—those functions located in the structures below the surface mantle of the brain that deal with relatively basic aspects of sensation, perception, learning, memory, and emotion. These systems are harder to study in humans, because damage to these regions may interfere much more radically with a whole range of behaviors, and may often result in death. One of the problems facing contemporary neuropsychology is to integrate the study of cortical functions and higher-level behaviors, which have generally been studied in humans, with the study of subcortical structures and more basic behavioral systems, which have been studied in animals. These have tended to be separate areas of research, although there are now signs of integration between the two. For example, intelligence is now being discussed not just in terms of human performance on intelligence tests, but also in terms of underlying basic processes of learning, attention, and motivation that are only understood, in neuropsychological terms, from animal studies. Sexual behavior is another area where the basic systems are only open to experimental study in animals, yet must be viewed within the context of socialized and cognitively controlled behavior in humans.

CONCEPTUAL ISSUES

Neuropsychology suffers philosophical and conceptual difficulties no less than other areas of psychology, and perhaps more than many. There are two problems in particular of which every student of the subject should be aware.

The first of these springs from the nature of the methods that must be used in neuropsychological investigation. Descriptions of brain organization can only be relatively distant inferences from the human perfor-

mance that is actually observed. The real states of the brain are not observed. Behavioral measures are taken, and by a line of reasoning that is based on background information about either the general arrangement of the brain (in the case of experimental neuropsychology) or about the gross changes in the brain of a particular type of patient (in the case of clinical neuropsychology), conclusions are drawn about what the correlation must be between brain states and behavior. The one exception to this general rule is in electrophysiological studies and studies of cerebral blood flow and metabolism through advanced scanning techniques, where actual brain states can be observed, albeit rather crudely, in “real time” alongside the human performance being measured. This makes these studies of special importance in neuropsychology. However, in general, neuropsychological study proceeds only by inference. It is important to remember this in assessing the validity of many of the findings claimed by neuropsychologists, and also to be particularly vigilant that the reasoning used in drawing inferences is soundly based and the data not open to alternative explanations.

The second problem is even more fundamental, and is that usually referred to as the *mind-body problem*. It is a subject far too complex to receive satisfactory treatment here, but in brief it is concerned with the philosophical difficulties that arise when we talk about mental events or “mind,” and physiological events or “body,” and try to relate the two. We first have to decide whether mind and body are, or are not, fundamentally different kinds of things. If they are, then there are problems in giving explanations that correlate the two. If they are not, then we have to be careful not to be misled by our everyday language and concepts, which tend to treat mind and body as if they *were* different kinds of things. The debate has gone on for some centuries, and is far from being resolved, but there is a general position accepted by most if not all neuropsychologists.

This position is known as “emergent materialism” or “emergent psychoneural monism.” It rejects the idea that mind and body are fundamentally different (hence it is “monist” rather than “dualist”) and proposes that all mental states are states of the brain. Mental events therefore exist but are not separate entities. However, mental states cannot be reduced to a set of physical states because the brain is not a physical machine but a biosystem, and so possesses properties peculiar to living things. The brain is seen as not simply a complex composition of cells, but as having a structure and an environment. The result is that there are “emergent” properties that include being able to think and feel and perceive. These properties are emergent just as the sweetness of an apple is an emergent property. There is nothing in the chemistry or physical structure of the apple that possesses sweetness. It is the whole object, in

interaction with the eater, that produces the quality of sweetness. Mind is therefore seen as a collection of emergent bioactivities, and this has implications for both theories and methods in neuropsychology. It means that it is sometimes quite proper and sensible to reduce explanations to lower levels of description, purely in terms of the physiology or the biochemistry involved. However, it also means that integration among these lower processes and their description in terms of higher-level concepts (concerning the emergent properties) are both feasible and valuable.

The student first taking an interest in neuropsychology should not be overly concerned about these philosophical issues; much, if not most, of neuropsychological work is conducted while ignoring them altogether. However, some position is always implied in any investigation or theoretical model, and it is wise not to lose sight of the implications of holding a particular position for a satisfactory understanding of how the brain works.

HISTORICAL BACKGROUND

It is intriguing to discover just how far back neuropsychological study may have been undertaken. Although the interpretation of the content is a little uncertain, there is an Egyptian papyrus dating from about 2500 B.C. that reports on some cases of trauma to the brain. Many primitive societies, and even some more developed societies, have from early times practiced the trepanning of skulls, although the evidence is usually in the form of artifacts and in human remains. In trepanation large or small openings are made in the skull that may or may not be subsequently repaired. The purpose of these, whether magical, medical, religious, or simply punitive, generally remains obscure, but the practice has been recorded over a large proportion of the globe, and into the last century.

In classical Greece there was, as one might expect, an interest in the brain and its functions, and Hippocratic writers clearly recognized the role of the brain in mental functions from careful study of their patients. However, while these ideas passed into the Arabic world, and were preserved there until the Renaissance, Greek ideas about the brain did not hold an important place in Western medieval thought. Galen, in the second century A.D. also made remarkable advances in observation of the anatomy of the brain, but his ideas on the functioning of the brain, and those developed from his work, do not form the basis of our understanding today.

Following the Renaissance, and the growth of practical human anatomy (rather than the dissection of animals, which had previously

been the practice), there were increasingly sophisticated and accurate descriptions of the anatomy of the brain, and by about the middle of the 19th century a number of factors came together to form the basis of modern neuropsychology. These factors included: the location of the cortex of the brain as the source of intelligent behavior; the accurate description of the neuroanatomy of the sensory and motor pathways; the idea that psychological processes could be analyzed and grouped into a number of associated sets of “faculties,” and the observation that specific areas of the brain could be identified that, if damaged, resulted in the loss of language abilities.

In the 1830s Gall, with Spurzheim, had founded the “science” of phrenology, which was based on the idea that development of the various mental faculties was associated with development of certain areas of the brain, and that this in turn was reflected in bumps on the skull overlying the relevant area. “Reading the bumps” was in fashion through much of the 19th century. Professor J. Millot Severn was providing “delineations” from the person or from photographs (“verbal statement” from two shillings and sixpence) at the Brighton Phrenological Institute up to World War I. Though the hypotheses both about brain development and its reflection in scalp topography were ultimately to be dismissed, phrenology at least planted the idea that psychological characteristics could be broken down into a number of components, and each component associated with a specific area of the brain.

Broca, in 1861, demonstrated that lesions of a specific area interfered with the production of speech, as Wernicke was later to do for the understanding of speech. From the 1860s, then, there was an intensive study of the cortical lesions of patients with psychological deficits in an attempt to map brain–behavior relationships. This approach is known as *localizationist theory* because it assigned a specific function to a precise part or location of the brain, and it had moderate, although not unqualified, success.

From the outset, however, there were those who supported an *equipotential theory*, believing that precise mapping of functions was impossible because damage at different sites in the brain could result in the same specific deficit. Equipotential theory states that while sensory input may be localized, perception involves the whole brain, and the effects of brain lesions depend not upon their location but upon their extent. It is not which cells are damaged that matters but how many. Equipotential theory has had many eminent supporters until well into the 20th century, including Kurt Goldstein, Henry Head, and Karl Lashley. However because it has, on the one hand, been possible to demonstrate some kind of a relationship between cortical locations and certain specific functions and, on the other hand, impossible to derive a

good index of brain damage that is independent of the site of the damage, equipotential theory has proved rather unsatisfactory.

A third position, which largely derives from the work of Hughlings Jackson, and is sometimes termed *interactionist theory*, proposes that higher functions are built up from a number of more basic component skills. These component skills are relatively localized, but because of the potential variety of complex ways in which the skills are linked to form intelligent functions, the higher-level behavior does not necessarily appear to be localized. That no single function or learning process is entirely dependent upon a particular area of cortex, and that each part of the brain plays an unequal role in different functions, would support this position. Interactionist theory, often linked with *regional equipotentiality*, which argues for equipotentiality but within relatively well-defined regions, is the position implicitly accepted by most contemporary neuropsychologists. Important modern examples of theories that express this position are those of Luria (see p. 323) and Geschwind, although Geschwind has emphasized the role of connections between the component elements rather than the elements themselves.

Clinical neuropsychology in the 20th century showed a steady accumulation of clinical reports and research investigations that have gradually refined the theoretical positions sketched above. World Wars I and II gave a particular impetus to research, and subsequently the conflicts in Southeast Asia, which provided cases of fairly circumscribed localized traumatic injuries. Great advances were made at these times both conceptually and in the sophistication of psychological descriptions, by Teuber, Luria, Zangwill, and a host of other neuropsychologists. This approach, of examining relatively discrete psychological deficits, together with the subsequent development by such neuropsychologists as Reitan of batteries of tests assessing a broad range of behavioral functions, forms the basis of contemporary clinical neuropsychology.

Experimental neuropsychology has rather more recent origins. It has grown from two independent, although related, sources. The first is general experimental psychology, and in particular that branch now described as cognitive psychology. From the 1940s, spurred on in part by wartime demands to investigate the performance characteristics of people operating complex equipment, the analysis has been developed of humans as information processing systems. Some of the forms of investigation—examining the responses of subjects to briefly presented visual stimuli (see p. 15) or to complex and competing auditory messages—have been seen as relevant to neuropsychology. It has been found that a neuropsychological model of the organization of the brain can be a powerful component in explaining certain aspects of human performance, and the methods of the experimental psychological laboratory have been employed to study hypotheses about the organization of

the brain. Just how these methods differ from those of clinical neuropsychology is explained in the next two sections of this book.

The realization of the significance of human performance research came about as a result of interest in the other source of experimental neuropsychology. This was the fortuitous revival (for the researcher at least) of the “split-brain” or “commissurotomy” operation in about 1960 (see Chapter 10). This operation, which divides the connections between the two hemispheres of the brain, was thought to produce two independent brain systems within the individual, and it enabled the study of the relative specialization of these two systems within split-brain patients. Its historical importance lies not only in the data collected in such studies, but also in the techniques developed in the course of the investigations, which could be employed in the laboratory with normal intact humans to draw inferences about cerebral organization. It is from the study of split-brain patients that modern experimental neuropsychology can be considered to date, and the very large number of studies in this area, particularly in the 1970s and 1980s, has established experimental neuropsychology as a significant element of modern psychology.

CLINICAL NEUROPSYCHOLOGY

Clinical neuropsychology relies almost exclusively on established correlation between damage to a particular region of the brain and a relatively specific deficit in psychological functions, indexed by a variety of more or less formal psychological tests. The collection of data by which such correlations are established allows models to be constructed of how systems of psychological functions are organized in the brain. It also allows knowledge of the deficits exhibited by a particular patient to be used to suggest where brain damage might be, as well as providing a psychological description of the handicap which that patient experiences. Within this central, essentially very simple, methodology there are different historical traditions that have influenced how clinical neuropsychology is practiced. These traditions are far from independent, but are characterized by different emphases in approach of which the student should be aware. The three main historical traditions may be said to be associated with work in North America, the former Soviet Union, and Great Britain.

Of all the historical traditions, that in North America has shown the most systematic approach. Allied to the general interest of American psychology in the assessment of individual differences, neuropsychological investigation has been linked with psychological models of human abilities. That is, the tests that are used (and that are illustrated in Parts II and IV) are more likely to be selected because they are believed to

measure some element of a scheme of psychological abilities. This is, of course, not always entirely explicit, but theories on the structure of human abilities have been influential in selecting which aspects of behavior are to be assessed. Because of this, it is only in America that systematic batteries (collections of tests) for the assessment of neurological patients have emerged. The most widely used of these is the Halstead-Reitan Neuropsychological Test Battery, which incorporates a range of tests covering all the principal elements of psychological abilities. It includes a full assessment of the intelligence of the patient, which yields information from the component subtests of the intelligence scale, a systematic assessment of the language abilities of the patient through a standard aphasia battery, and a range of more specialized tests to assess perceptual, psychomotor, memory, learning, and thinking abilities. This enables an analysis of the overall pattern of deficits and preserved abilities in the patient, which can lead to a diagnosis of any dysfunction. Attempts have been made to improve the diagnostic efficiency of this approach by the application of the specialized statistical techniques of multivariate and discriminant function analysis, and even to automate the interpretation of the test results by computerized key analysis. (These methods, together with the more recent Luria-Nebraska battery, are described more fully on pp. 326-327). Such approaches have achieved a considerable degree of success, although a penalty is imposed by the length of time required to administer the extensive tests.

By contrast, *Russian* neuropsychology, again reflecting the general character of psychology in that country, has tended to adopt a single case study approach linked to the prevailing theories about cerebral organization. These theories are expressed in terms of functional systems that can be referred to particular regions within the brain. Rather informal, and in general unstandardized, tests are used to assess the functional status of these systems, and only those tests that the clinician considers immediately relevant to a patient will be employed. Again reflecting the interests of Russian psychology, psychophysiological and electrophysiological measures may well be taken alongside those obtained from more typical psychometric instruments such as standard psychological tests. Attempts, not entirely successful, have been made to extract a systematic battery out of the investigative procedures employed in Russian neuropsychology. The difficulties that arise in such an exercise are largely due to the fact that the tests are often not formal, standardized procedures, not "tests" in the psychometric sense at all, but are general procedures for the assessment of some function that depends on the clinical skill and insight of the investigator.

British neuropsychology stands between these two approaches. This third tradition has drawn from both the strong history of British neurology and the empirical biases within British psychology. In Britain there is

a tendency to use standardized procedures, but for the selection of tests to be more pragmatic. Research in clinical neuropsychology tends to focus on some relatively discrete aspect of performance and to investigate it by controlled procedures in a homogeneous sample of patients selected according to some criterion, which is often the regional locus of their lesion. These procedures, originally employed in experimental investigations, have often evolved into relatively formal standardized tests that can be used to investigate the problems of individual patients. While research has tended, therefore, to concentrate on group data, the investigation of individual patients proceeds by the selection of appropriate tests, in a relatively standard form, that are combined to elicit a description of the deficits present in that patient. This tradition, while relying on data from standardized procedures, emphasizes the individual nature of each case, and is not averse to the construction of single case experimental investigations to clarify some aspect of the problems shown by a particular patient. A summary of the three traditions, with the strengths and weaknesses of each, is shown in Table 1.1, and more detailed discussion of all these approaches will be found in Chapter 15.

To describe the three historical traditions in this way underestimates the variety of approaches to be found in the practice of clinical neuro-

TABLE 1.1. Historical Approaches in Clinical Neuropsychology

| | |
|---|--|
| <i>North American:</i> Systematic collections of tests. | |
| Strengths: | Comprehensive coverage of functions. Based on psychological model of abilities. Allows use of scores that combine results from different tests. |
| Weaknesses: | Cumbersome, and perhaps wasteful, in use. Based on model of normal function, rather than abnormalities. May be insensitive. |
| <i>Russian:</i> Single case approach based on behavioral neurology. | |
| Strengths: | Relevant assessment instruments selected. Wide scope for application of clinical skill. Based on model of abnormal function. |
| Weaknesses: | Little use of standardized procedures or normative data. Depends heavily on clinical skill. Difficult to measure subtle changes in level rather than quality of performance. |
| <i>British:</i> Investigation of individual cases by selection of standardized tests. | |
| Strengths: | Focus on difficulties of individual patient. Can make use of statistical psychometric analysis. Allows development of a model of individual patient's disability. |
| Weaknesses: | Investigation may be fragmentary and unsystematic. Overemphasis on tests that happen to be available. Risk of overreliance on poor and inadequate test procedures. |

psychology, and the growing internationalism of the discipline. It is probably fair to say that the British approach is now becoming the dominant international style, particularly because of the growing influence of cognitive neuropsychology (see p. 17) and the adoption of the approach through Europe and Australasia.

However, all approaches rely to a certain extent on the clinical skill and insight of the investigator. Perhaps especially within the British approach, there is a strong element of scientific detective work in selecting appropriate tests and developing an appropriate investigation for each patient. There is a certain satisfaction in the exercise of a clinical skill that enables the clinical neuropsychologist to take information about the patient from other medical specialties, to evolve an investigation, to arrive at a relatively specific description of the patient's problem in psychological terms, and to contribute to the diagnosis of the patient's lesion, which may subsequently respond to treatment and rehabilitation provided by the neuropsychologist or other specialists. There is also a definite thrill in the successful solution of this kind of detective problem, which demands intellectual as well as clinical ability, and which may benefit a patient in danger, pain, or distress.

The relevance of clinical neuropsychology is not hard to see. Despite developments in other neurological fields, particularly the development of modern medical imaging or "brain scans" that enable far clearer visualization of changes in the structures within the head, neuropsychologists can still make a substantial contribution to the diagnosis and localization of lesions in individual cases. In addition, although it has been a field slow to develop, neuropsychology can play a central role in the rehabilitation of brain-injured patients. In order to encourage the relearning of lost skills and the development of compensatory strategies, and to promote appropriate social and occupational adjustment, an accurate description of psychological deficits is vital. This description needs, of course, to be integrated with the application of theories of learning, memory, language, and motor skill. Our developing knowledge of the brain's neuropsychological organization enables coherent plans to be formulated that may help individual patients compensate for their handicaps, as well as cognitive strategies that can enable them successfully to circumvent their difficulties.

EXPERIMENTAL NEUROPSYCHOLOGY

Experimental neuropsychology is best understood in terms of the various methods that have been used in research studies. These can be grouped into methods that rely upon the logic of stimulus presentation,

those in which lateral asymmetries in human performance allow more distant inferences, and those involving various specialized techniques.

By far the most numerous are the studies that rely upon the *logic of stimulus presentation*. These employ the techniques known as divided visual field presentation (see Chapter 11), dichotic listening (see Chapter 12), and lateralized tactile presentation. In all of them, the stimuli presented for subsequent cognitive analysis and response are “lateralized.” The brain (as is made clear in the following chapter) is divided into two lateral hemispheres that are only interconnected at the cortical level by a number of commissures, the most important being the corpus callosum. From clinical evidence, particularly that from split-brain patients, the hemispheres can be regarded as relatively independent systems with their own particular specializations. Sensory input, through vision, hearing, or touch, is lateralized to the contralateral hemisphere; that is, stimuli presented to one side of the body are received at the opposite side of the brain. This contralateral mapping is complete for vision, more or less complete for touch, and predominant for hearing. It also works in similar fashion for voluntary motor control. Therefore, if stimuli are presented at a selected location on one side of the body, it is to the opposite side of the brain that the information initially travels, and subsequent performance, generally in terms of accuracy or response latency, can be studied according to the initial reception of the information at one of the hemispheres. The use of the left or right hand to respond is an additional variable that can be included in the design. When visual stimuli are employed, the technique is that of divided visual field presentation; when auditory, it is that of dichotic listening, and when tactile, that of lateralized tactile presentation.

Although a very complex series of events, involving both hemispheres, follows the initial reception of information from lateralized presentation, it has been possible to establish a number of asymmetries in performance, which are taken to indicate different specializations of the cerebral hemispheres. These relate in part to the nature of the information given to the subject, whether verbal or nonverbal (e.g., words or faces), in part to the nature of the cognitive task (naming, matching, or evaluating), and in part to more general attentional variables. Despite a number of methodological problems, and some lack of clarity in the cognitive and neuropsychological models that are used in this area, there seems to be sufficient consistency in the data to enable conclusions about cerebral organization to be drawn from these studies. These techniques have led to very considerable advances being made in our understanding of the principles that underlie the operation of the brain for psychological function, and progress has been made in the construction of coherent models of neuropsychological function.

Associated with these more direct techniques (although it should not be forgotten that they still rely upon a rather indirect chain of inference about the brain) are some *other performance asymmetries*. One of these concerns lateral eye movements. Many subjects, when involved in solving a problem they have been given, avert their gaze to either the right or left, and this has been shown to be associated with the nature of the mental processes (either verbally or spatio-perceptually based) required to solve the problem. This is believed to reflect, in turn, the operation of lateralized brain systems. Some other performance asymmetries—direction of preferred movement in drawing circles, the extent of thumb rotation, and preferences in judging pictorial compositions, among others—have also been linked to hemisphere specialization, although the connection is rather less well established (see pp. 257–258).

More extensive research has been done on handedness. Individual differences have always been a theme of study in experimental neuropsychology, with handedness and, to a lesser extent, gender as the most important variables. It is clear from clinical and other experimental evidence that right and left handers differ in their brain organization. While there are groups of left handers who show a laterally reversed pattern of asymmetry, left handers in general seem to have less lateralized brains than right handers. Research into the relative performance of right and left handers has been undertaken to clarify these differences and to extend our understanding of brain organization in general.

Lastly there are some more *specialized techniques* that come closer to a direct association of brain events with psychological processes. In the Wada test, sodium amytal is injected into the left or right internal carotid artery, which is the principal blood supply to the brain. The effect is to suppress all activity on the side of the injection for a period of a few minutes. It is therefore possible to observe the functions of one half of the brain while its partner has been temporarily put out of action. Because of the risks involved in this injection, the test is not made on normal subjects, and is only performed as part of the assessment of patients who are to undergo certain types of neurosurgery (see p. 86). It has nevertheless yielded some valuable data.

The study of regional cerebral blood flow, or rCBF, and other metabolic processes (see Chapter 13) can, however, be undertaken with volunteer subjects. Here a quantity of radioactively labeled gas is inhaled, absorbed into the bloodstream, and then tracked by an array of detectors placed alongside the head as it passes through the brain; there are now a range of similar techniques, but they all share the same essential methodology. Subjects may be engaged in some mental task during this procedure, and more active regions of the brain, by demanding more

blood, show up at the recording detectors. Amounts of radiation are small, but the problem with the technique is its poor resolution. It takes some time for the radioactive elements to pass through the head and the temporal correlation with psychological events is therefore crude.

The last specialized technique of importance is the study of electrophysiological variables. The electrical activity of the brain has for many years been recorded for clinical, neurological purposes, but it has more recently attracted attention as a research technique in psychology. Electrical activity can be recorded while subjects engage in psychological tasks and can be analyzed in terms of the characteristic activity at certain sites during certain periods (“ongoing” EEG), or else in terms of the typical response of the brain to some stimulus event (average evoked potentials). While there are considerable technical and methodological difficulties in this area, it offers an opportunity to observe psychological events and the associated brain events simultaneously and with a fair degree of resolution. This is a fundamental advance, but although it has appeared to hold great promise for the future, it has yet to deliver the insights that were once anticipated.

COGNITIVE NEUROPSYCHOLOGY

Cognitive neuropsychology developed in the 1970s as a relatively distinct sub-branch of neuropsychology. There has always been a productive interchange between the fields of cognitive psychology and neuropsychology. Cognitive models of human abilities inform neuropsychological analysis, and neuropsychological findings have contributed to the development of the cognitive models. However, this became more formalized in cognitive neuropsychology by the explicit development of cognitive models on the basis of neuropsychological data, and the utilization of these models in the analysis of clinical neuropsychological problems.

A good example can be given in one of the most highly developed models, which is for single word reading. Based upon previous cognitive models of the reading process in normal individuals, cognitive neuropsychological models have been developed in which the various forms of neuropsychological reading disorders can be understood in terms of faults in one or more of the specific component processes, or in the connections among these processes. Visualized as a flowchart of the reading process, the reading disorder can be described as errors in the “boxes” or the “arrows” of which the chart is composed. The model can, of course, be tested in its ability to adequately characterize the difficulty that a particular patient experiences, alongside its coherence with the

experimental findings from normal individuals reading single words in the laboratory.

Two forms of cognitive neuropsychology have been identified: a “hard” and a “soft” form. The soft form accepts that some reference to what is known about the structure and neurological organization of the brain is appropriate, and that the model should be broadly consistent with this information. The anatomy of the brain may be a useful guide to the structure of the cognitive neuropsychological model. However, the hard form adopts the position that the model stands quite independently of anatomical considerations of brain structures. The analysis is purely at a psychological level and the descriptions couched in terms of psychological processes. It seems strange to many neuropsychologists to have a branch of neuropsychology which need make no reference to the brain at all; but this is where the logic of this approach leads.

Cognitive neuropsychology has been both prominent and influential in shaping the development of neuropsychology over the past 30 years. Particularly successful models have been developed not only for word reading but also for face recognition, object perception, and a number of other abilities. These models have also continued to make an impact upon the cognitive psychology of normal individuals. Nevertheless, it must be recognized that, other than through a generally increased sophistication of neuropsychological understanding, these models have made relatively little impact in clinical practice. The demands of performing the relevant cognitive neuropsychological analysis normally exceeds the time available to a clinician in dealing with a particular client. The contribution of cognitive neuropsychology—and it is a substantial contribution—has been in research and theoretical development rather than in the development of useful tools and procedures.

THE FRINGE

Neuropsychology, like many sciences, has its fringe elements, and because it has recently become fashionable, perhaps more than its share. Hemisphere lateralization has been the starting point for a whole range of theories about consciousness, cultural differences, and occupational and educational adjustment. It has been suggested that consciousness originated from processes in the right hemisphere that were originally attributed to the “voice of gods.” It has been proposed that differences in societal development between primitive cultures have their origins in the relative development of the specializations of the two cerebral hemispheres, and that Eastern and Western styles of thought reflect similar lateral differences. Occupational success has been attributed to “hemisphericity,” or the rela-

tive balance between the activities of the two sides of the brain, and questionnaires have been designed to measure this balance. Changes in educational curricula have even been proposed to develop the supposedly more creative aspects of right-hemisphere function.

Although many of these ideas are stimulating and exciting, it has to be said that they go well beyond the scientific evidence at present available, and may indeed be harmful if incorporated into our general cultural ideas, or translated into social policy. It is hoped that the student, having read this book and some of the recommended further reading, will be in a better position to make a balanced assessment of some of these ideas and their significance and value.

THE PLAN OF THIS BOOK

The following chapter gives a brief overview of the structure of the brain for those unfamiliar with neuroanatomy. It is necessarily rather cursory, but should be sufficient to enable the reader to understand the structures and systems that are referred to in the remainder of the text.

The fairly clear distinction, already made, between clinical and experimental neuropsychology is preserved in the arrangement of the main part of the book. Part II deals with clinical studies, which have been divided according to the main regions of the cortex, with separate sections on language and subcortical systems. In any study of neuropsychology, the student needs constantly to try to build an integrated scheme that maps the interrelations of psychological processes onto the anatomical systems of the brain. It is possible to approach the studies either in terms of the anatomical structures, or by subdividing the material according to psychological functions. There are advantages and disadvantages to both approaches. As it is expected that readers may be more used to thinking in terms of a psychological scheme of functions, the necessary integration may be easier if the material is presented according to an anatomical classification, so that the student makes the necessary links across functional categories. This is not an easy task, but is one of the exciting challenges of the study of neuropsychology.

Part III presents some of the principal areas of experimental neuropsychology, divided by the techniques of investigation employed, and enables reference to be made back to the findings with clinical patients described in the previous section. The final section presents in further detail some of the practical ways in which neuropsychology is applied, and suggests some of the advances that may soon be made in translating research studies into techniques that may be of value in both neurology and psychiatry.

Neuropsychology is a fascinating, and currently very exciting, field of study. At both the conceptual and theoretical levels, and the level of practical application, there are a variety of challenges and a number of mysteries. We do not know much about the brain and must suspect that it still holds many secrets. Dramatic developments are being made and there is the promise of more to come. I only hope that some of this fascination and excitement will be glimpsed by readers beginning their study of neuropsychology.

CONCLUSION

Neuropsychology can be divided into two branches. The first, clinical neuropsychology, includes behavioral neurology and deals with patients with cerebral lesions. Three historical traditions, loosely associated with North America, the former Soviet Union, and Britain, can be identified.

The second branch is experimental neuropsychology, which studies normal subjects in the laboratory by a range of techniques including dichotic listening, divided visual field, and lateralized tactile presentation, as well as more specialized physiological techniques. Studying the brains of animals, known as comparative neuropsychology, makes an important but distinct contribution.

There are fundamental conceptual issues raised by neuropsychology, which include the mind–body problem. The generally accepted position is emergent materialism or emergent psychoneural monism, which holds that all mental states are also states of the brain, but cannot be reduced to the properties of single cells. The historical origins of modern neuropsychology can be traced to the second half of the 19th century. Early theoretical positions included localizationist and equipotential theories, but most neuropsychologists now accept interactionist theory with its associated concept of regional equipotentiality.

FURTHER READING

Some general books on neuropsychology, perhaps more for reference purposes:

- Beaumont, J. G., Kenealy, P. M., & Rogers, M. J. C. *The Blackwell Dictionary of Neuropsychology* (Oxford, UK: Blackwell, 1999, paperback edition). More an encyclopedia than a dictionary, this is a comprehensive reference source with an eminent range of international contributors.
- Kolb, B., & Whishaw, I. Q. *Fundamentals of Human Neuropsychology* (Fifth revised edition, San Francisco: Freeman, 2003). Probably the most sound and widely recommended text at a rather more detailed level; a good “follow-up” from this book.

Two books that form a good introduction to some conceptual issues:

Bunge, M. *The Mind–Body Problem* (Oxford, UK: Pergamon Press, 1980). This is an excellent introduction to the philosophical issues, with extensive reference to the neurosciences. It summarizes all the major positions, assesses their strengths and weaknesses, and proposes a new development of emergentist materialism.

Shallice, T. *From Neuropsychology to Mental Structure* (Cambridge, UK: Cambridge University Press, 1988). An excellent, but advanced text; best used selectively.

Three books that concentrate primarily upon clinical neuropsychology:

Andrewes, D. G. *Neuropsychology: From Theory to Practice* (Hove, UK: Psychology Press, 2001).

Halligan, P. W., Kischka, U., & Marshall, J. C. *Handbook of Clinical Neuropsychology* (Oxford, UK: Oxford University Press, 2003).

Martin, G. N. *Human Neuropsychology* (Second edition, Harlow, UK: Pearson Education, 2006).

And, to stimulate your interest, two books that provide readable accounts of case studies in neuropsychology:

Campbell, R. (Ed.). *Mental Lives: Case Studies in Cognition* (Oxford, UK: Basil Blackwell, 1992).

Code, C., Wallech, C.-W., Joannette, Y., & Lecours, A. R. (Eds.). *Classic Cases in Neuropsychology* (Hove, UK: Psychology Press, 1996).

The Structure of the Central Nervous System

The human nervous system is conventionally divided into three parts: the central nervous system (CNS), the peripheral nervous system (PNS), and the autonomic nervous system (ANS). The ANS is a specialized system formed from components of both the CNS and the PNS, and is concerned with general activation, with emergency response, and with emotion, and is more particularly the study of the psychophysicologist.

The function of the PNS is to carry information from receptors distributed around the body into the CNS, and to carry information back out to effectors. By this system, sensations of light touch, pain, and temperature are detected in the skin; pressure, pain, and position sense are conveyed from subcutaneous and deep tissue; and pain is transmitted from the viscera. Similarly, instructions are carried outward from the CNS to effectors in muscles and glands. Although the PNS is more complex than this would suggest, for our purposes we can consider it simply as forming a communications link between the spinal cord and the periphery of the body. The only specialized parts of the PNS that we should note are the cranial nerves, which deal with the neck and head, and the special senses, but we shall return to these later. Perhaps the most significant difference between the PNS and the CNS is that, when damaged, parts of the PNS can be regenerated and will grow back into their original configuration. If the CNS is damaged, then there is no practical degree of regrowth possible, and the damage, in structural terms, is permanent.

TERMINOLOGY

The terminology for structures in the CNS can often seem difficult to students. Two points may help to reduce this difficulty. The first is to note that many structures have Latin, or rather medieval dog-Latin, names that were often given by anatomists on the basis of some visual reference or joke. This helps to make the terms easier to remember, and for those not familiar with Latin, translations of some of these terms will be given in parentheses when they are introduced. The second point is to understand how the general biological system of indicating direction is applied to the CNS. In this system, *rostral* means toward the head (a “rostrum” is raised up), and *caudal* toward the tail. *Dorsal* means toward the back, while *ventral* is toward the belly. *Lateral* is toward the side, and *medial* toward the midline (see Figure 2.1.).

The complication with respect to the human CNS is that in the course of evolution it is considered to have bent forward through a right angle at the level of the brain. We should therefore think of these terms as applying to the situation in which the figure is standing but with the head bent back and the eyes pointing toward the ceiling. This resolves

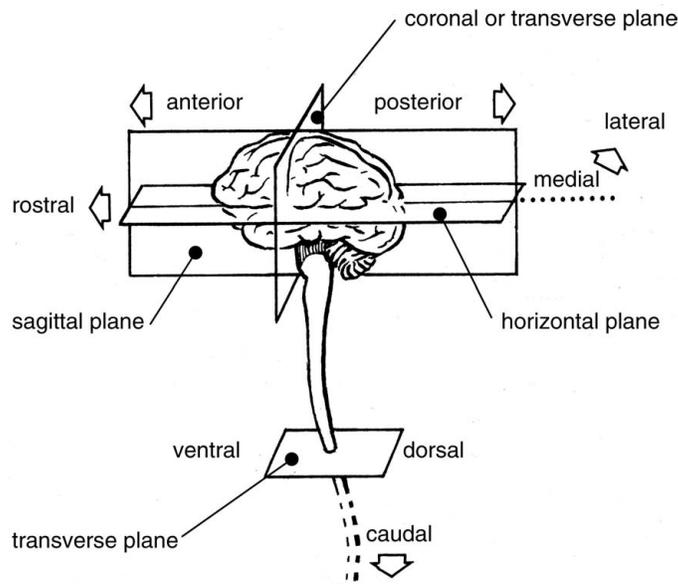


FIGURE 2.1. The terminology used to describe sections and relative locations in the central nervous system.

the puzzlement often generated by the description *coronal* (or transverse) applied to a section through the brain, which in a standing figure would appear to be a vertical section. You must imagine the head bent back before the “crown” is placed on top of the figure. A *sagittal* (cut by an arrow) or front-to-back section is fairly clear, although a *horizontal* section applies to the head placed in its actual everyday orientation. The terms *anterior* (toward the front), *posterior* (toward the back), *superior* (above), and *inferior* (below) as applied to the head also refer to it in the normal posture. There is sadly no alternative to learning how these terms are used, and in the early stages of study being prepared to look them up whenever you are unsure.

THE ENVIRONMENT OF THE CNS

The CNS, which is basically composed of the brain and the spinal cord, is contained within the bony case of the skull and the spinal column, no doubt because of the serious biological consequences of damage. Within that bony case it is further protected and supported by a series of membranes, or *meninges* (meningitis is inflammation of the meninges). These membranes are the *dura mater* (“hard mother”), the *arachnoid* layer (like a spider’s web), and the *pia mater* (“soft mother”). An extradural block is an anesthetic, used in obstetrics, placed outside the *dura mater* in the spinal column, and a subdural hemorrhage is bleeding in the space below the *dura*, but outside the brain itself.

The brain also has a very rich blood supply, taking about a fifth of the blood pumped by the heart. Models of the brain, or illustrations of the appearance of the brain, from which the arterial and venous structures have been cleared often allow this to be forgotten. The system extends around every part of the brain, and failure of part of this system in a cerebrovascular accident (CVA), either by part of the blood supply becoming blocked (as in a stroke), or by bleeding into the brain and surrounding tissue, is a common cause of pathological change.

Within the meninges and enmeshed in its vascular supply, the brain floats in a fluid known as the *cerebrospinal fluid* (CSF). The function of CSF is both to provide mechanical protection by absorbing some of the shocks to the CNS and to supply certain aspects of nutrition of the brain. It is generated within the brain itself, in the *lateral ventricles*, which are large chambers within the brain, and it circulates through narrow passages into the midline *third* and *fourth ventricles*, and then out into the spaces surrounding the brain and the spinal cord, until it is finally absorbed into the venous system (see Figure 2.2). CSF is generated in the ventricles under some pressure in order to maintain flow in

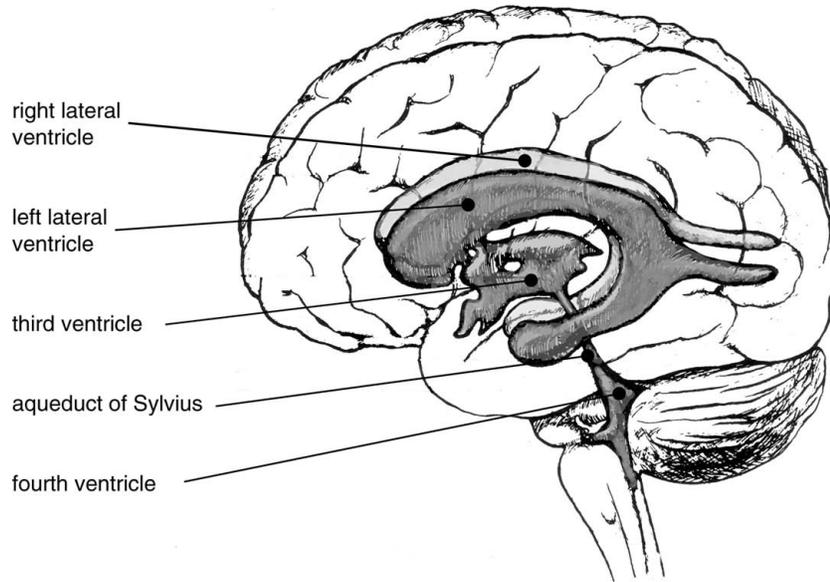


FIGURE 2.2. The ventricular system of the brain.

the system, and there are serious consequences if the passages in the brain become blocked. This is the cause of the condition known as *hydrocephalus* (water on the brain), and is the reason why the head circumference of infants is regularly measured. In some children the passages are congenitally blocked, the CSF is unable to circulate out of the ventricles, and thus both the brain and the unknit bones of the skull begin to swell. This can be corrected by the insertion of a bypass channel and valve (a shunt), and few long-term effects are apparent if the condition is detected at an early stage. The condition can also occur in adults and be successfully treated. However, because the bones of the skull cannot separate to allow the brain to swell, the condition may not be detected at such an early stage, and the consequences can be more serious.

The ventricular system can be made use of in diagnosis in two ways. In lumbar puncture, a needle is inserted in the lumbar region of the spine to withdraw a quantity of CSF for inspection, and in *ventriculography* or *air encephalography* a quantity of radio-opaque dye or a bubble of air is introduced into the ventricular system so that its outlines can be traced by x-ray photography. This can give information about either the displacement of structures inside the head or changes in the size of the ventricles that may indicate shrinkage of brain tissue.

THE SPINAL CORD

The spinal cord is often underrated by psychologists, but many aspects of behavior are in fact organized and integrated within it. It may be considered as composed of a series of layers, with each layer related to a spinal vertebra. At each layer there are spinal nerve roots, one ventral and one dorsal pair, by which the PNS links to the CNS, and each relates to an identifiable region on the surface of the body. Because of the resemblance of a map of these regions to a series of horizontal slices, they are known as *dermatomes*. The neurologist can make use of this mapping to determine the level of the spinal cord at which pathology is present.

Apart from the carrying of motor information down from the brain and out to the body, and receiving sensory information back in return, certain behaviors are organized entirely within the spinal cord, principally by means of *reflexes*. In these reflexes the sensory information enters at one layer of the spinal cord and links directly in a reflex arc to a motor nerve, which then exits back out to the PNS. This is the simplest form of reflex and is known as a two-neuron reflex, but slightly more complex reflexes may extend over several adjacent layers in the spinal cord, although the principle of their operation is exactly the same. All this occurs without the direct intervention of higher levels of the CNS. Higher levels of the brain may influence the speed with which reflexes operate, or how easily they may be triggered, but control is limited to this general kind of supervision.

Reflexes may be divided into *superficial*, *deep*, and *special* reflexes. Superficial reflexes may be elicited by stimulation of the skin. For instance, if the sole of the foot is scratched, then the toes flex. An example of a deep reflex is the knee jerk that follows the striking of the patella with a soft hammer. Here a tendon is stretched, which results in a reflex muscle contraction. There are other similar reflexes associated with the ankle, elbow, and wrist. Special reflexes often have a more complex organization, of which the constriction of the pupil in bright light is a clear example. Reflexes may be of developmental significance and, in abnormal form, of clinical value. For example, the Babinski reflex at the sole of the foot produces upward flexion of the toes in infants, but after infancy changes to downward flexion. Upward flexion in adults is therefore a sign of some pathological process.

One way to assess the contribution of some region of the CNS is to ask what functions remain when all other regions have been removed. This kind of experiment can of course only be carried out with animals. Clinical cases involving complete section at the very top of the spine are in any case very rarely seen because such damage is incompatible with survival. However, animal experiments have provided some useful infor-

mation about this. If the section is above the fourth cervical segment (the cervical section of the spinal cord is at the top, and is numbered from the top down), artificial respiration is required, but section below that is compatible with life. Following spinal shock there is, in motor terms, a progression from some reflex activity, through flexor spasms, to extensor dominance and mass extension with all the limbs rigidly stretched out. (Flexion is the action of bending a joint so that the angle between the parts it connects becomes smaller; extension is the opposite action of straightening the parts out at the joint.) The animal may even stand if placed upright on its extended limbs. The bladder will function and defecation may occur, as may integrated sexual activity following appropriate stimulation. Otherwise there is little that we would call behavior, although a description of this state may underestimate the contribution of reflex activity organized in the spine to the normal functioning of the intact system.

Locked-in syndrome is the one disorder occurring in humans that in part illustrates these phenomena. In the locked-in syndrome, which will be described more fully in Chapter 8, a stroke in the brain stem effectively disconnects the brain from the body. Locked-in patients lose all control over their motor functions with the exception of some extremely limited movements, typically vertical eye movements. They are unable to move or speak, and yet have full awareness and may retain normal comprehension, thinking, and memory, as can be demonstrated when they are provided with devices that can translate their limited movements into language and environmental controls.

DIVISIONS OF THE BRAIN

The brain, which is a rather loose term generally taken to be all of the CNS except the spinal cord, is conventionally divided into a number of regions. Outside a phylogenetic perspective, these divisions are not always very helpful, and are not of great importance for neuropsychologists. To add to the difficulty there is no complete agreement on their use. However, these divisions have been laid out in Figures 2.3 and 2.4 in order to show how they relate to some of the major structures in the brain, and we shall follow these basic divisions in describing the major components of the brain. It is worth noting, however, that the phylogenetic development of the brain does have some significance for understanding its structure, and thereby understanding the functional interrelationship of parts of the system. The brain has evolved as a series of layers wrapped around a central core formed by the spinal cord surmounted by the brain stem, and with each layer a “higher” level of func-

| | | | |
|-----------------------------------|-----------------------------|--------------------------------------|---|
| BRAIN | FOREBRAIN prosencephalon | TELENCEPHALON (end brain) (1) | cerebral cortex |
| | | RHINENCEPHALON | limbic system |
| | | DIENCEPHALON (interbrain) (2) | thalamus hypothalamus basal ganglia internal capsule |
| BRAIN STEM reticular formation | MIDBRAIN | MESENCEPHALON (midbrain) (3) | midbrain |
| | | | cerebellum (4) |
| | | METENCEPHALON (afterbrain) | pons (5) |
| | | MYELENCEPHALON (narrow brain) (6) | medulla oblongata |
| SPINAL CORD (7) | | | |

FIGURE 2.3. The regions into which the central nervous system is divided, and some principal structures in each. The numbers refer to Figure 2.4.

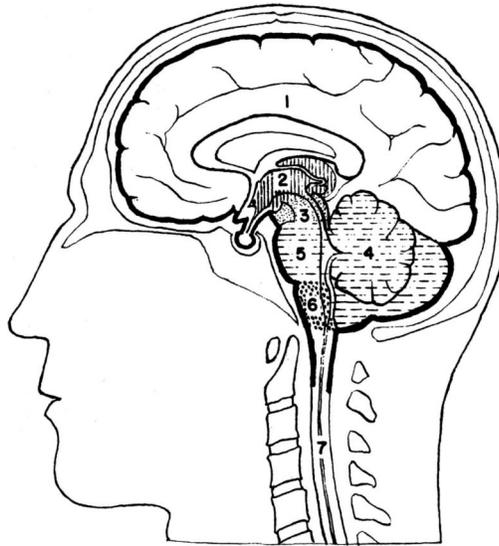


FIGURE 2.4. The regions of the brain. See Figure 2.3.

tion has developed, progressively extending the functional capacity of the system. Each of the divisions we consider represents a major development in which another layer was added in evolution to the total brain system.

THE BRAIN STEM

On entering the skull, the spinal cord becomes the *brain stem*, which is formed of three principal structures: in ascending order, the *medulla oblongata* (“oblong marrow”), the *pons* (“bridge”), and the *midbrain*. The *cerebellum*, which will be discussed shortly, sits astride this region and forms massive connections with it (see Figure 2.5).

The functions of the brain stem can most easily be considered if we again look at what happens when it is separated from higher control in the brain. In a *decerebrate* animal the cranial nerves (see below) are intact, allowing full sensory information into the system, and intact final motor outflow. The cerebellum is still contributing to behavior, but oth-

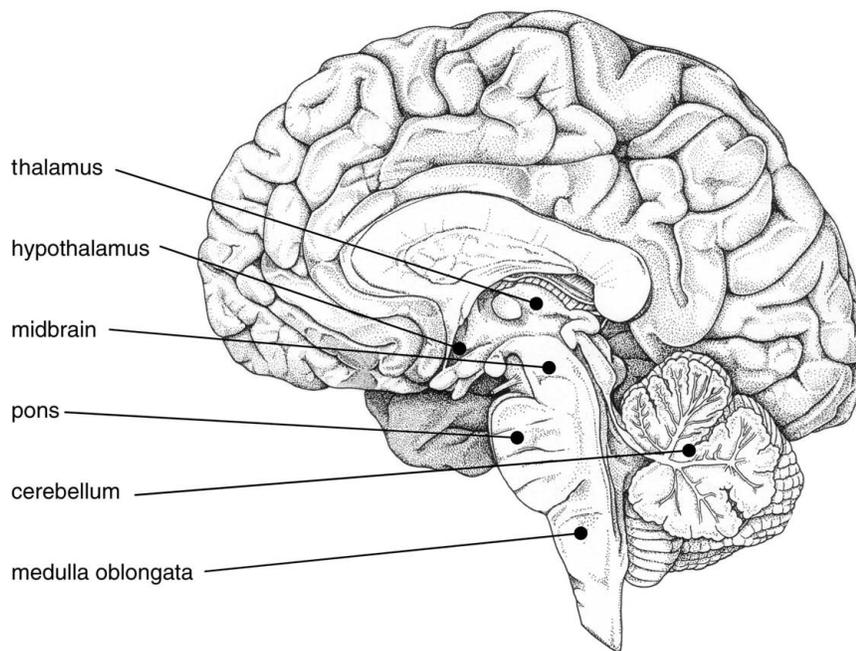


FIGURE 2.5. Principal structures of the brain stem and cerebellum.

erwise the system is divorced from higher levels of control. In motor terms, the decerebrate cat has strong extensor contractions that it will maintain (like the “spinal” animal), and its limbs will support its weight, but it is unable to move about. If pushed over, it cannot right itself. It must be fed by hand and, as temperature control is lacking, be kept at an appropriate stable temperature. Blood pressure is, however, maintained and respiration continues, slowly and deeply (as it does characteristically in many patients in coma). Certain reflexes that depend on sensory information carried by the cranial nerves also operate, so that the head and neck can be moved.

The midbrain in particular has some additional and specialized functions. Visual information is relayed to this part of the brain and some forms of visual reflex, for instance blinking, pupillary constriction, and certain eye movements, are organized here. Similarly, auditory reflexes, for example starting at a sudden noise, originate in this region.

Certain aspects of autonomic function are organized in the brain stem, so that salivation, control of blood pressure, respiration, and movements in the alimentary canal are influenced through the ANS from this region. These influences may include part of the control of vomiting.

An important structure that runs through the whole brain stem is the *reticular formation* (“like a little net”—as in *retiarius*, meaning a Roman gladiator who fought with a net). Cells in this formation communicate with cells throughout the spinal cord, cerebellum, and the higher regions of the brain. A particular feature is the *ascending reticular activating system* (ARAS). This system receives inputs from sensory pathways on their upward journey, and projects onto the cerebral cortex through a variety of intermediate centers. The result is a generalized arousal of the whole forebrain, and is an important component in wakefulness, in the maintenance of consciousness, and in attention. The ARAS thus forms a general vigilance apparatus that ensures the brain is ready to receive information.

As indicated above, the *cranial nerves* enter the brain at this level. They consist of a set of twelve pairs of nerves, some sensory, some motor, and some combining both functions, which almost entirely project to the head and neck. Apart from subserving sensation in this region, and providing motor control, the cranial nerves serve the special senses through the olfactory nerves (smell), the optic nerves (vision), the auditory nerves (hearing and equilibrium), and the facial, glossopharyngeal, and vagus nerves (taste). Eye movements are, of course, also directly controlled by the cranial nerves. The important feature is that these nerves enter the brain directly without passing through the spinal cord.

In summary, the brain stem is concerned with the special senses, with vital processes, and with other visceral and somatic functions, all of which may be modified by impulses entering by the cranial nerves, down from the cerebellum or from the forebrain.

THE CEREBELLUM

The *cerebellum* (“little brain”) is joined onto the brain stem and sits astride it, but this should not lead us to ignore the cerebellum’s important direct connections with both the spinal cord and the forebrain.

The cerebellum’s main role is to coordinate muscular activity, both in postural and locomotor mechanisms, all of which is carried out at a subconscious level. Perhaps because this happens without our awareness, we tend to forget what a massive control operation is required to maintain our posture and to effect both voluntary and involuntary motor movements. Most positions of the body are achieved by the antagonistic activity of opposed groups of muscles. These muscles, if not properly coordinated, are sufficiently powerful to break bones or at least to tear other muscles out of position. It is therefore vital that these groups of muscles be carefully coordinated. That we can achieve this feat of control so effortlessly, and not only avoid self-injury but also execute the most delicate, accurate, smooth, and graceful movements, is a tribute to the marvellous contribution of the cerebellum in motor control.

The cerebellum achieves this control by receiving information from the skin and from muscles, tendons, joints, and the semicircular canals (the organs of positional sense and balance near the ear), as well as from the visual and auditory systems. In turn, it discharges out to the cerebral cortex and spinal cord, executing control over the timing of the execution of motor events. It thus ensures smooth, controlled, and well-organized movement. Patients with damage to the cerebellum show jerky movements and intention tremor (tremor that only appears when a deliberate movement is made), walk in a broad stumbling gait, and show loss of both balance and position sense. In fact, deprived of visual feedback by closing the eyes, these patients readily topple to the ground.

THE DIENCEPHALON—THE SUBCORTICAL FOREBRAIN

When we reach this level of the CNS, things start to get more interesting for the psychologist, for structures in this region are centrally involved in motivation, emotion, and the ANS, and in states of awareness. We also

begin to consider the structures that, when damaged, lead to recognizable psychological signs in clinical patients, although it must be remembered that many patients with extensive diencephalic damage do not survive, and much of our information is based on inferences from comparative studies on animals. Nevertheless, because relatively isolated injury to the cerebral cortex (the most common form of clinical damage to be studied by neuropsychologists) cannot be considered without reference to the subcortical structures to which that cortex is related, an understanding of the arrangement and function of the diencephalon is of central importance.

It is convenient to consider the diencephalon in terms of three structures (see Figure 2.6): the *thalamus* (“inner room”), the *hypothalamus* (“lower room”), and the *basal ganglia*. “Basal ganglia” is a rather “rag-bag” term used differently by different writers, but it indicates centers of activity deep in the middle of the brain, and I am using the term in just that inexact way. Some writers include the hypothalamus in the basal ganglia.

While thinking phylogenetically often leads us to think of the cerebral cortex as the endpoint of the CNS, in a sense it is the thalamus that

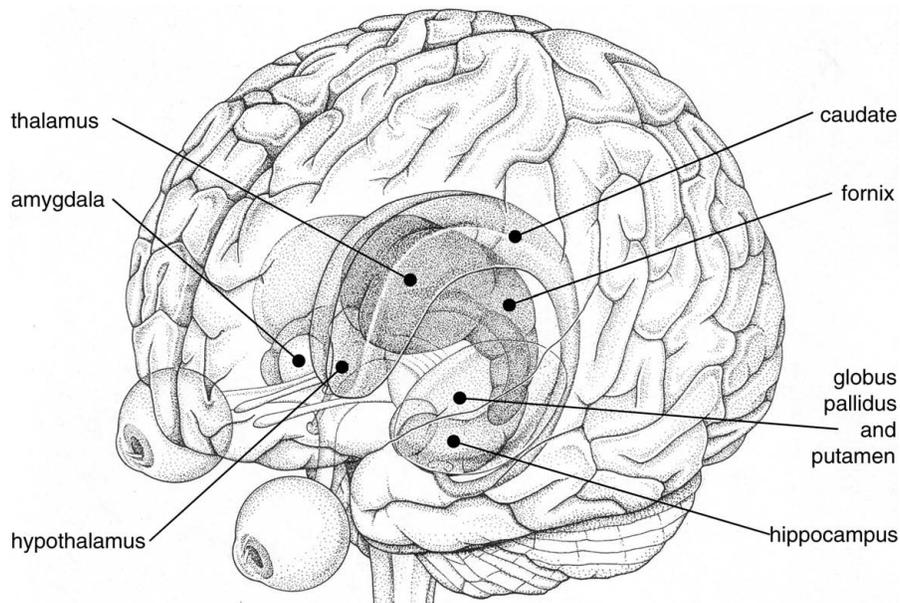


FIGURE 2.6. Principal structures of the diencephalon. (Drawing after W. J. H. Nauta and M. Freitag, *Scientific American*, 1979.)

is the main control center governing sensation and movement. Physiologically, all the pathways (except the direct voluntary “pyramidal” motor pathway) travel to and from the thalamus as their final destination, with an additional loop via the cerebral cortex. This loop no doubt allows more sophisticated operations to be performed with the sensory or motor information, and may be the circuit that gives us conscious awareness of sensory and motor events. However, it is unwise to neglect the primary role of the thalamus in the central registration of sensory information and the executive control of motor activity.

Righting reflexes (which enable a cat to land safely on its feet from whatever orientation it begins its fall) are organized in the thalamus, operating with visual and positional information from the head, neck, and body, but the thalamus also performs more general underlying motor functions, as a list of the motor disorders that may follow thalamic lesions indicates. Damage to the thalamus may produce resting tremor, *chorea* or rapid, jerky involuntary movements, and *dystonia* or uncontrolled movement. The syndrome of *Parkinsonism* is associated with thalamic lesions and includes rigidity in movement, a shuffling gait, loss of associated movements (such as arm swinging while walking), loss of emotional expression, and tremor. It is not uncommon to see Parkinsonian tremor in the elderly, and in such cases the presence of resting tremor is obvious in that it disappears when an intentional movement, such as drinking from a cup, is executed but reappears when the attention is allowed to wander, usually resulting in the contents of the cup being spilled.

Similarly, the thalamus is an important receiving center for sensation, although the sensation is not at this stage localized in terms of associated perceptions, but is in the form of general awareness of touch, temperature, or pain.

The hypothalamus (below the thalamus) is equally important behaviorally, and its functions have led certain writers to call it the “center of the brain.” It contains pairs of nerve centers that can be shown to influence eating and drinking, sleeping and waking, sexual behavior, organization for fight or flight and the rage reaction (in association with the ANS), and the response to reward and punishment. Although such studies are now considered to give too simplified a picture of the operation of the hypothalamus, studies of animals in which particular centers are either stimulated or extirpated (surgically removed) suggest that there are relatively discrete centers that turn these aspects of behavior on or off. Appropriate lesions can cause animals permanently to cease drinking, or else to drink continually and excessively. Animals will also work far more vigorously to achieve self-stimulation, in a conditioning paradigm by bar pressing or by running in a treadmill, if electrodes are

placed in certain hypothalamic centers than they will for conventional rewards such as food or water.

Perhaps the most dramatic of the demonstrations of the diencephalic control of behavior was performed by Delgado. He inserted electrodes in the appropriate regions of the brain of a fighting bull. Entering the bullring armed only with a cape and a radio-controlled telemetric stimulator, he showed that it was possible to halt the bull in midcharge. The rather confused bull merely turned and wandered away.

The remaining structures in the diencephalon, the basal ganglia, which include the *amygdala* (“almond”), *globus pallidus* (“pale sphere”), *caudate* (“having a tail”), and *putamen* (“husk”), have functions largely associated with those described for the thalamus and hypothalamus. There is, however, one important system that involves many parts of the basal ganglia, as well as parts of the telencephalon, and that is the *limbic system* (on the “borders” of the diencephalon). This is a relatively ancient system, sometimes known as the *visceral brain*, and is concerned with many aspects of emotion and behavior (see Figure 2.7). The principal structures in this system, which is coiled around the central structures of the thalamus and hypothalamus, are the *hippocampus* (“seahorse”), the *fornix* (“a vaulted chamber”), the *amygdala*, the *septal region* (“enclosing”), the *cingulate (“beltlike”) gyrus*, and the *mammillary (“breastlike”) bodies*, together with significant links into the thalamus and hypothalamus. The functions undertaken by parts of this system include memory and learning, taming and some aspects of

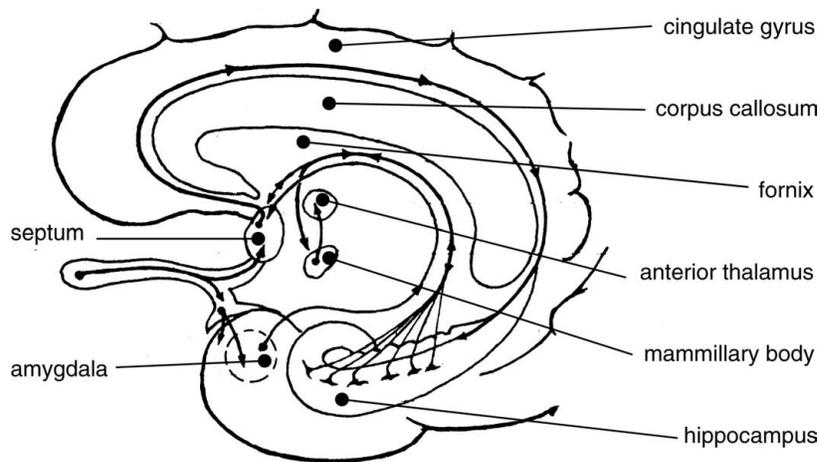


FIGURE 2.7. Some of the interconnections between structures in the limbic system.

aggression, some primitive features of object recognition, sexual and exploratory behavior, and, in animals, avoidance learning, connected with the effects of frustrative nonreward (the effects on subsequent learning of not receiving an anticipated reward for performance). As there is some similarity between these behaviors and those associated with the hypothalamus, it seems clear that the limbic system in general undertakes the organization of these behaviors and the control of their execution, while the hypothalamus is more concerned with their initiation in response to the current level of motivation.

Because the cortex of the temporal lobe has direct connections with the limbic system, we shall return to discuss certain aspects of this system, particularly those relating to memory and sexual behavior, in Chapter 4. We shall also meet the limbic system again when we discuss psychosurgery in Chapter 9.

THE TELEENCEPHALON—THE CEREBRAL CORTEX

The *cerebral cortex* (“bark” or “rind”; also called the *neocortex*) forms the surface of the brain. It is spongy and jellylike, and extremely convoluted or wrinkled. Man is the only animal to have a brain convoluted to this degree, and this is often considered an evolutionary response to the need to pack an ever-expanding amount of cortex in a head of given size. If the most intelligent aspects of function are organized in the cortex, and that seems a reasonable supposition, given that we have more cortex in relation to our body size than any other animal, then for us to evolve into a more intelligent animal, the cortex must grow larger. However, if this were to imply an ever increasing head size, there would be severe biological disadvantages, including significant loss of heat through the head, difficulties of balance and motor control of the head, and vulnerability of the head to damage, either accidental or aggressive. Presumably, folds in the cortex have made it possible to fit a larger cortex into an acceptable size for the human head.

The appearance of the brain has led to its description as “gray matter” (although in reality it has a distinctly yellow tinge) and, indeed, if the brain is sectioned, both gray and white regions are to be seen. The gray comes from the cell bodies of the neurons and indicates that nerve cells have their origins and are making rich interconnections in these regions. The white comes from the myelin that covers the bundles of fibers forming tracts passing about the brain. A section of the brain clearly shows the distribution of gray matter around the edge, forming the cortex, and indicates the subcortical centers that have been described above, leaving the areas in between to appear as white matter. The fur-

rows or grooves on the surface of the cortex are known as *sulci* (singular: *sulcus*, “furrow”) or fissures, and the bumps or islands in between as the *gyri* (singular: *gyrus*, “coil”). There is surprising regularity in the pattern of sulci between individuals (not always apparent on casual inspection).

Only some of the main features of the cortex need be remembered. Most of the topographical features of the cortex have been named in Figure 2.8, and photographs of a real brain are shown in Plates I and II for comparison. The most obvious feature is the horizontal line named the *lateral* or *Sylvian fissure* or sulcus, and the sulcus that runs down from the top of the brain to meet it, which is the *central* or *Rolandic fissure* or sulcus. These landmarks allow us, more or less completely, to map out the four lobes into which the cortex is conventionally divided: the *frontal*, *temporal* (behind the temples), *parietal* (across the brain), and *occipital* (to the back of the head) lobes.

It should not be forgotten that from the diencephalon up, we are in fact considering pairs of structures (two thalami, two hypothalami, and so on), and that the telencephalon is formed of two cerebral hemispheres separated by the *longitudinal fissure* and a large partition extending down from the meninges, the *falx* (“sickle”). The two cerebral hemispheres are only directly connected by a series of cerebral *commissures*: the *anterior commissure*, the *corpus callosum* (“hard-skinned body”), and the *posterior commissure*. Apart from these commissural links, the two cerebral hemispheres are quite independent at the cortical level, an intriguing feature that has implications for brain organization, as we shall see in later chapters.

The terms introduced so far, together with the general orientational terms, will enable us to refer to areas of the cortex with sufficient precision for elementary neuropsychological analysis. More precise taxonomic systems are currently of little use in human neuropsychology, although some are used, and students must refer to more advanced neuroanatomical texts if these are met in further reading. Perhaps the only system that should be mentioned here is the numbering system for areas based on Brodmann’s cytoarchitectonic maps (where areas are delineated by the types of cell they contain). This is thought to provide a surer basis for generalization across species, so that, for example, studies on area 17 (primary visual cortex) can be referred from cats and monkeys to man by reference to the area of cells in roughly the same structural location, but delineated by the presence of appropriate cell types.

The next chapters will examine what conclusions can be drawn from clinical studies of the function of each lobe of the cerebral cortex, and what follow here are some general remarks about the organization of function in the cortex.

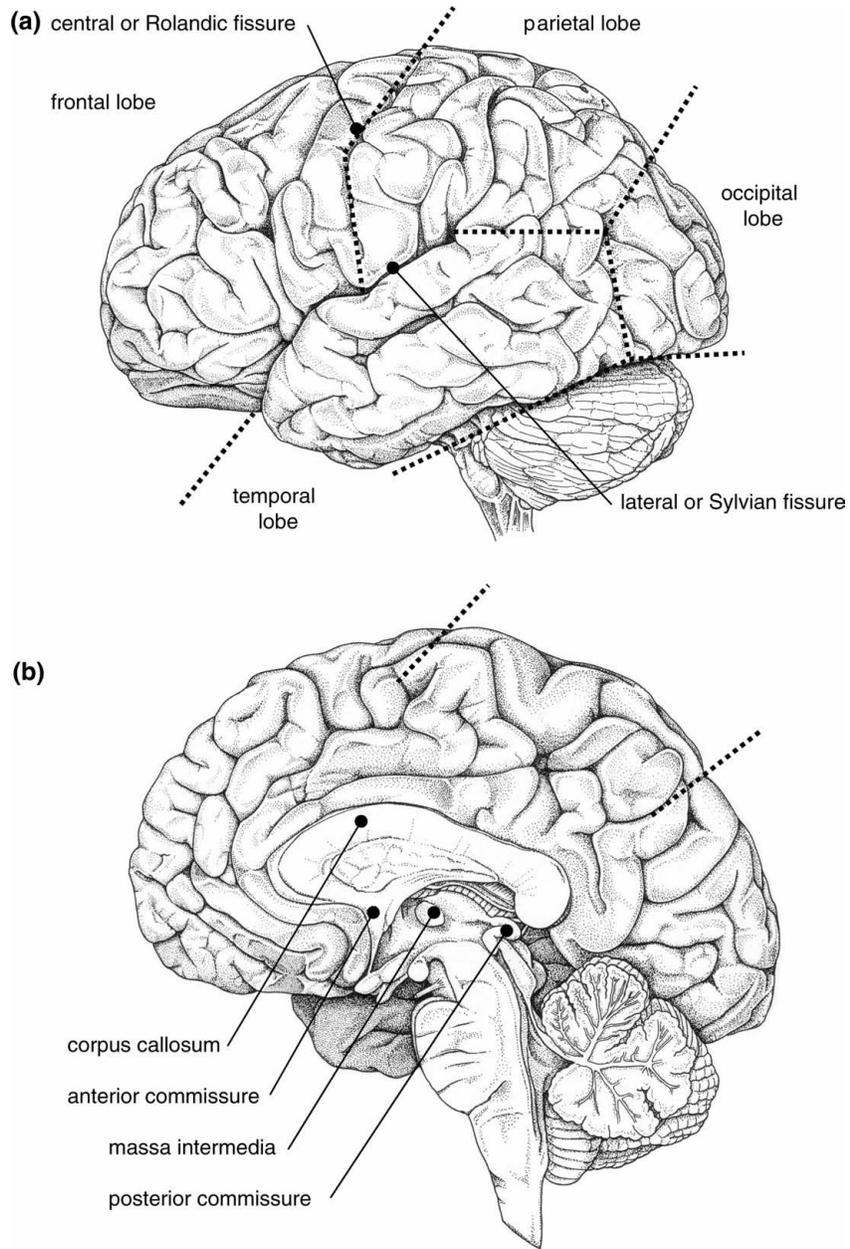


FIGURE 2.8. The cerebral cortex: (a) left lateral view showing the four lobes and two principal fissures; (b) medial view of the right hemisphere showing the interhemispheric commissures.

The first principle to note is that of *relative localization*. Neuropsychologists hold differing views on the extent of localization, but the majority assume that most functions can be assigned to a region of the cortex, but without precise localization to specific cells. There is certainly no accurate mapping of specific cells taking on specific functions, at least outside the areas in which primary sensory information is received. The degree of localization appears to vary according to how “high-level” a function is, with the higher levels that involve more integration of different types of information and serve more complex functions appearing to be less localized. An example of a “low-level” function with a high degree of localization might be sensory awareness in vision, where seeing light at a specific position in the visual field can be linked to a specific cell, or small group of cells, in the primary visual cortex. The lesser localization for higher functions is probably an artifact of the way basic functions are linked together to produce complex behavior; if we examine the effects on complex behavior of a system composed of basic, relatively well-localized units, the function will not appear to be well localized because aspects of it are distributed throughout the system. The higher the function, the higher the degree of integration and the less localized the system. Nevertheless, most neuropsychologists maintain that at the level of basic functional components there is a relative distribution of function within a region of the cortex.

The second principle to note is that of *plasticity*. This doctrine holds primarily that the brains of young children are plastic (less clearly localized), and that the degree of plasticity decreases with age. The corollary of this is that the brain of a young child is functionally resistant to damage, as a greater degree of compensation is possible in the young than in older patients. This is clearly illustrated by the amazing preservation and recovery of abilities in children who undergo hemispherectomy, or the removal of one entire cerebral hemisphere, for some pathological condition, in comparison with the severe and persistent handicap that may follow loss of certain very specific regions of cortex in adults, particularly if the handicap involves language. Children who undergo hemispherectomy at an early age, particularly below the age of about 5, develop cognitive abilities within the normal range and full bilateral motor function. It may take quite sophisticated testing to reveal the effects of their surgery in later life and they are without significant handicaps in their everyday activities. Whether the degree of plasticity seen in the young reflects true compensation, in which undamaged tissue actually takes over the function previously performed by the absent or damaged tissue, or whether there is radical functional rearrangement and new learning, which may be more easily achieved in the young brain, is a matter of debate. There is some evidence for both processes, although

the contribution of functional rearrangement, as opposed to structural compensation, may be the more important.

Lastly, it is helpful to consider the cortex as comprising three zones: primary, secondary, and tertiary cortex. (This follows the division made popular by Luria, although it does not necessarily imply his scheme of functional interpretation.)

In *primary cortex* are found the primary sensory and motor regions of the cortex, each with a relatively high degree of localization. Here primary visual stimulation is received near the pole of the occipital lobes, auditory stimulation in the superior temporal lobes, and somatosensory information in the sensory strip just posterior to the Rolandic fissure. Electrical stimulation of sites in these areas in the conscious subject results in reports of nonspecific but localized stimulation in the appropriate modality. The patient may report a flash of light (to which he may point), or a touch on the arm, or hearing a particular tone. Primary motor cortex, situated in the strip anterior to the Rolandic fissure, is similarly organized, and stimulation of points in this area may elicit discrete body movements. The area of the motor and somatosensory strips dedicated to each body region reflects the relative functional importance of these regions in a most illuminating way (see Figure 2.9). It should also be noted that the projection of sensory fields up to the cortex, and

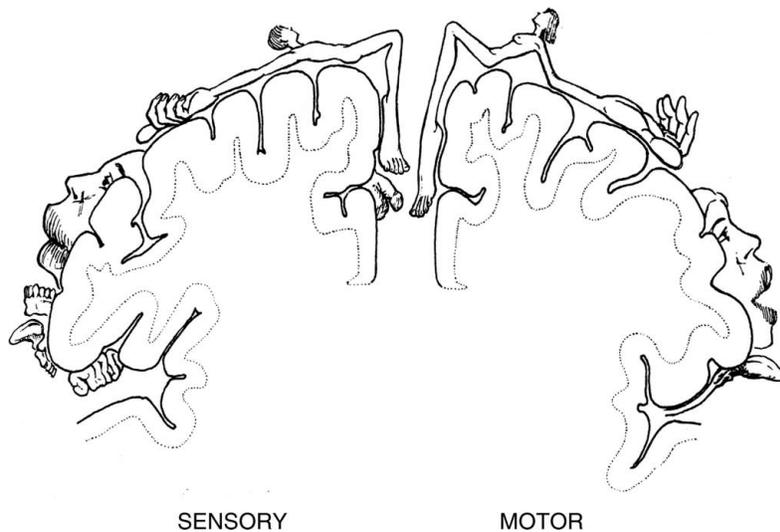


FIGURE 2.9. The relative representation of different parts of the body over the sensory and motor strips of the cortex as revealed by stimulation studies. (Drawing after N. Geschwind, *Scientific American*, 1979.)

motor control downward, is contralateral; that is, the left hemisphere receives sensory information from the right side of the body and sensory space, and controls the right side of the body, with the reverse arrangement for the right hemisphere.

Secondary cortex is formed by regions adjacent to primary cortex, so that there is, for example, secondary visual cortex anterior to primary visual cortex. Stimulation of secondary cortex results in the report of integrated percepts. The patient may describe a vivid visual scene, may say that his arm has been lightly brushed by velvet, or that he hears part of a popular melody. It is inferred from this that the secondary cortex is important in interpreting the sensations received in primary cortex and turning them into perceptions.

Apart from the specialized regions—those in the left hemisphere of right handed subjects, which deal with language functions and which will be discussed in Chapter 7—the remaining cortex can be considered as *tertiary* or *association cortex*. We do not have a clear idea of the real function of this cortex, which forms large areas of the cerebrum, but we infer that it performs functions that contribute to intellectual processes at the highest level. Evidence for this is given in the next chapters, but the nature of these processes is still very much a matter for speculation and debate.

The cerebral cortex may contain some of the most difficult puzzles for the neuropsychologist, but it also contains some of the greatest scientific challenges. In that the cortex holds the key to whatever it is that separates humans from other animals and confers intellectual power, insight, and foresight, it is the most fascinating of systems to investigate. In the next chapters we shall see how closer examination of the effects of damage to the cortex can give us clearer insights into how the brain is organized, and how it undertakes some of the amazing feats of which it is capable.

CONCLUSION

The brain can be divided into four areas: the brain stem, cerebellum, diencephalon, and telencephalon. The brain stem receives the input from the special senses and deals with vital processes and other visceral and somatic functions. The cerebellum plays a major role in the coordination of muscle activity. The diencephalon or subcortical forebrain maintains central control of sensation and movement as well as of appetitive behavior. Motivation, emotion, and the ANS are also served by this region and states of awareness maintained. The telencephalon or cerebral cortex supports high-level intelligent behavior and provides con-

scious experience. It can be considered as composed of three zones. Primary cortex deals with sensation and the initiation of voluntary motor activity. Secondary cortex produces perception and the integration of sensory and motor behavior. Tertiary or association cortex serves high-level thinking, planning, and problem solving. In addition, there are specialized regions of the cortex that deal with language.

FURTHER READING

Almost all general textbooks in physiological psychology carry an extensive introduction to the structure of the CNS. Among those that can be recommended are:

- Bloom, F. E., Nelson, C. A., & Lazaron, A. *Brain, Mind, and Behavior* (Cranbury, NJ: Worth, 2000).
 Carlson, N. R. *Physiology of Behavior* (Eighth edition, Boston: Pearson Education, 2004).

Three more specialized but readable texts:

- Crossman, A. R., & Neary, D. *Neuroanatomy: An Illustrated Colour Text* (Edinburgh: Churchill Livingstone, 2005).
 Garoutte, B. *Survey of Functional Neuroanatomy* (Mill Valley, CA: Mill Valley Medical Publications, 1994).
 Noback, C. R., Strominger, N. L., Demarest, R. J., & Ruggiero, D. A. *The Nervous System: Structure and Function* (Sixth edition, Totowa, NJ: Humana Press, 2002).

Finally, three superb collections of illustrations, including functional systems:

- England, M. A., & Wakely, J. *A Color Atlas of the Brain and Spinal Cord* (Second edition, New York: Harcourt Brace, 2006). Much the best illustrated of any anatomy text.
 Netter, F. H. *The CIBA Collection of Medical Illustrations: Vol. 1, Nervous System* (Summit, NJ: Novartis Medical Education Program, 1983).
 Nieuwenhuys, R., Voogd, J., & Van Huijzen, C. *The Human Central Nervous System: A Synopsis and Atlas* (Berlin: Springer-Verlag, 1988).

And a book that is fun, but nevertheless aids serious study:

- Diamond, M. C., Schiebel, A. B., & Elson, L. M. *The Human Brain Coloring Book* (New York: Harper Perennial, 1985).

PART II

CLINICAL STUDIES

The Frontal Lobes

The frontal lobes of the cerebral cortex are traditionally considered to be the seat of the “highest” mental functions and the center of those activities that make us characteristically human. This is largely because in evolutionary terms the frontal cortex has been the most recent to evolve, and humans happen to possess particularly large frontal lobes. However, it may also be that because of the relative difficulty of ascribing a clear set of functions to these areas, they have been attributed with intelligence by default. The large proportion of the cerebral cortex described as frontal lobe, up to about half of the total area of the cortex and an even higher proportion of the association cortex, makes it likely that significant aspects of intellectual activity are performed there, but, as we shall see, it is necessary to be a little more cautious about what functions we can with confidence ascribe to this region, which nonetheless remains one of the most interesting for neuropsychologists.

SOME METHODOLOGICAL ISSUES

Before considering just what aspects of intelligence may be associated with the frontal lobes, some points must be made about the specific problems that arise in carrying out research studies on the effects of focal damage to the cerebral cortex. These problems appear because we have to work with clinical material, which does not arise in a random way, and the points made here therefore apply not only to this chapter, but to all the chapters in this section on clinical studies. The logic of the

research design is to collect cases in which there is an identified lesion of some area, let us say in the frontal lobes, and to compare the performance of these patients with the performance of patients who have lesions in areas outside the frontal lobes. This determines whether the functions being studied are affected only by frontal lesions.

However, the essential point is that we have to control in some way for all the factors apart from the site of the damage that could contribute to any deficit observed in performance. These other factors include the type of lesion: what caused it, whether it is developing (“progressive”) or stable (“static”), and whether it was recently caused (“acute”) or is long-standing (“chronic”). For example, tumors are usually progressive, and may develop slowly or rapidly depending on type, while a gunshot wound can be considered, after the initial period following the injury, to be static. The age of the patient is also important, as is the extent or “mass” of the lesion and how far it extends below the cortex into subcortical tissue.

The main problem is that lesions of different types tend to occur in different areas, and in patients of different ages. Tumors of certain types grow in particular sorts of tissue, but may be fairly evenly distributed across age groups, while missile wounds obviously occur most frequently in young males injured during war or urban violence. Vascular accidents, in which either the blood supply to some region of the cortex is lost (as in a stroke) or some failure results in bleeding into the brain, tend to occur more commonly in older subjects. Studies that compare lesions of the frontal and parietal regions without controlling for the type of lesion may then end up by confounding the site of the lesion with its cause.

Even if the study is restricted to a comparison of lesions of one particular type, for example those caused by gunshot wounds, the lesions occurring at less usual sites may be in some way atypical. Wounds from modern high-velocity projectiles yield perhaps the best clinical material for the neuropsychologist, for the bullet, if not at close range, tends to punch a very neat hole straight through the head, causing remarkably little disturbance to regions not immediately affected, and producing a clean wound that is self-sterilized and cauterized by the heat generated as the bullet passes through. In such cases, the important issue for survival is whether the bullet passes through important central subcortical centers essential to life or fundamental aspects of behavior. If the entry and exit points are around the temporal and parietal regions, death is much more likely than if they are in the frontal and occipital regions. As a result, more soldiers arrive for neuropsychological assessment with frontal or occipital wounds than with temporal and parietal wounds, and the lesions of those with temporal and parietal injuries who do sur-

vive may be less extensive than those of their colleagues and, in a variety of ways, less serious.

An alternative example is studies that examine differences between the left and right members of a particular pair of lobes. Here the confounded variable may be the mass of the lesion. Someone with a developing tumor in the left or right frontal lobe will sooner or later notice some of its effects and will probably consult his or her general practitioner (GP). However, because of the much greater importance of verbal as opposed to spatial abilities in everyday life in our society, these patients are more likely to notice that they cannot remember the contents of the day's paper or an address just given to them, than that they cannot remember some drawing or route to be taken to a particular place. Since the failure in verbal memory usually results from a left lesion and in spatial memory from a right lesion, patients typically arrive for surgery with smaller tumors in the left than in the right hemisphere, where they have been allowed to grow unnoticed for longer. This can naturally confound the results of any study that compares the effects of tumors in the left and right sides of the head, because any differences found may not be due to the lateral site of the tumor but due to the mass of the lesion.

These examples illustrate the considerable difficulty of constructing sound scientific studies when it is necessary to work with incidentally occurring clinical material. The ideal study would involve equal amounts of the same kind of damage occurring in each cortical area, but the data are just not available for such a study. There are additional problems in that it is often assumed that the deficits observed are a reflection of more specific deficits in complex tasks that involve several basic unitary functions in their performance. The factors that contribute to methodological difficulties are summarized in Table 3.1.

It should also be realized that studies of the highest methodological standard are rather uncommon, owing to deficiencies in design and theoretical interpretation, and that many of the findings reported below are

TABLE 3.1. Methodological Difficulties in Interpreting Clinical Lesion Studies

| | | |
|---|---|--|
| 1. Variations in: | site lateralization extent cause age of patient stability acuteness | lobe or region left/right hemisphere mass progressive/static acute/chronic |
| 2. Inferring unitary deficits from performance on complex tasks | | |

subject to difficulties of interpretation that follow from research problems of the type just described.

INTELLIGENCE

From the latter part of the 19th century the frontal lobes have been associated with intelligent abilities, but a controversy raged through much of the 20th century as to whether these abilities may be associated exclusively with the frontal lobes. It may simply be that the frontal lobes are large, subserve many functions, and are as a result likely to affect “intelligent” behavior more than other lobes of the brain. Alternatively, there may be some general factors such as attention, or motivation, associated with the frontal lobes that have an impact upon all “intelligent” tasks. (Many psychologists would in any case say that “intelligence” is no more than the abilities that determine performance on intelligence tests.) To evaluate the arguments presented in this controversy, it is important to distinguish between quantitative and qualitative changes in intelligence.

In terms of quantitative deficits in intelligence, case reports from the beginning of the 20th century reported reduced intelligence following frontal lesions, and these findings were largely confirmed by the first important research studies by Rylander in 1939 and Halstead in 1940. The finding was simply that measured general intelligence was reduced after damage to the frontal lobes. The view was expressed most clearly in Halstead’s description of “biological intelligence” in 1947. He had formulated this concept from the results of a statistical analysis of a battery of tests that had been administered to a large sample of subjects with various focal cortical lesions. Among these tests, and showing the highest “loading” on biological intelligence, was the Category Test, which is a test of concept formation or categorization in which sets of graphical items are presented, and the patient has to indicate which of the numbers 1 to 4 may be associated with the set from the other three (see Figure 3.1). Patients with frontal lobe damage do badly on this test.

Although Halstead’s theory commanded much support through the 1940s and 1950s, it was criticized by Hebb, who, largely by studying the effects of deliberately placed experimental lesions in animals on abilities such as maze learning, argued that the mass of the lesion was more significant than its location. This view was confirmed in 1959 by Chapman and Wolff, who performed a reanalysis of much of Halstead’s data, introducing the factor of lesion size and adding new data of their own, and found that Halstead’s findings could be interpreted in terms of the effect of the mass of the lesion.

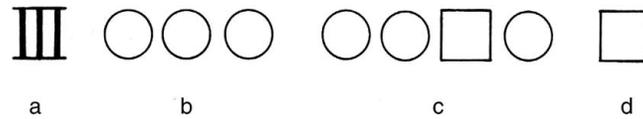


FIGURE 3.1. Examples of four items presented in four subtests of the Halstead Category Test. In each case the correct response would be to press the button marked “3.”

During the 1950s and 1960s Teuber, with colleagues, carried out an impressive series of studies on the war injured, which again tended to emphasize that deficits in general intelligence are not exclusively associated with frontal lesions, and that not all frontal lesions produce deficits of this type. The majority of recent studies, particularly those that have been careful in their experimental design, have supported this view, and a good example is the study of Black (1976) on veterans from the war in Southeast Asia. Even studies based on the modern version of Halstead’s own battery, developed by Reitan (see Reitan & Davison, 1974, and p. 325, of this volume), and including such tests as the Category Test, do not support the idea that “biological intelligence” is a property of the frontal lobes. There is therefore no good evidence to support the association of the degree of intelligence with the frontal lobes. But do frontal lobe injuries affect the quality or form of intellectual performance?

The change in the quality of thinking most commonly linked with the frontal lobes is the loss of *abstract thought*. This change, or the loss of the “abstract attitude,” is linked with the name of Kurt Goldstein, who published his ideas between 1936 and 1959. Goldstein considered there to be two forms of thinking: “concrete” and “abstract.” The abstract form was characterized by the ability to assume mental sets, to consider different aspects of a given situation, to dissect and synthesize the elements of some object, and to plan ahead and think symbolically; the concrete “attitude” was tied to the immediate sensory data that could be derived from the object. He employed a battery of tests that included various sorting tasks and a block design task in which colored blocks had to be arranged to match some design presented to the subject (see Figure 3.6 on p. 57). Goldstein claimed to demonstrate that frontal lobe lesions impaired the ability to adopt the abstract attitude, and thereby also caused a decline in conventionally measured intelligence. It should be noted, however, that Goldstein’s own work was not based upon the quantitative results of performance in his tests of abstract thinking. He did not, for example, present any quantitative data upon which a discrimination between frontal and more posterior lesions could be based. His arguments rested essentially upon the nature of qualitative

changes, despite the fact that they could be seen as providing the explanation for the quantitative changes in intelligence observed by some investigators.

The difficulty in assessing Goldstein's views arises from both general theoretical and specific methodological problems. The theoretical problem is with the formulation of abstract thinking and its distinction from concrete forms of thought. For instance, some researchers take the copying of a block design in the same color as a concrete task, and the copying of it in a different color as an abstract task. Others, in demanding a definition of the proverb "The sun shines upon all alike," would take "The sun shines on everybody" as a concrete response, and "All men are created equal" as an abstract response. The meaning of "abstractness" is clearly different in these two examples; the definition of this concept is a general problem in psychology. There is insufficient space to discuss this topic sensibly here, but few psychologists currently would accept the views implied in Goldstein's theoretical formulations.

The methodological problem arises from the nature of the tests used to assess the abstract attitude. Because the performance of subjects was not observed, recorded, scored, and analyzed according to the standards that we would now consider appropriate for the administration and interpretation of clinical tests, some doubt is cast upon the data collected by their application. The expectations of the examiner may have played some part in determining the results of Goldstein's tests, and it is known that their formal reliability (that is, the degree to which they yield stable and replicable measures) is unacceptably low. Normative data, by which the test results may be interpreted, are either not available or inadequate. For these reasons, the results of the tests of abstract thinking are not generally acceptable. It is also now clear that patients with posterior (nonfrontal) lesions may also fail on these tests.

In conclusion, it is fair to say that there may be qualitative changes in thinking following frontal lobe lesions but the data and arguments presented by Goldstein are not adequate evidence for such changes. It seems more profitable to inquire why patients may fail on certain tests, and to look at more specific deficits to provide a better explanation of the general difficulties experienced by frontal lobe patients.

The concept of impairment in abstract thinking is very important historically but it also continues to play a role in current theories of frontal lobe function. The idea that the frontal lobes are associated with underlying general intelligence persists. Duncan et al. (2000), taking account of the historical problems in investigations of this kind, argued that *g*, the general factor relating to intelligence that can be extracted from factor analyses of cognitive tests, is specifically associated with

frontal lobe function, and that a specific frontal system underpins the control of a broad variety of forms of behavior.

SPECIFIC FUNCTIONS

If we reject the idea that general aspects of intelligence can be specifically linked to the frontal lobes, then what specific aspects of behavior are controlled by them? There are, indeed, a variety of behavioral components that are affected by frontal lesions, but lacking any clear theory of the logical relationships among all these components (although some theories relating to regions of frontal lobe function are presented shortly), it seems sensible to discuss the frontal lobes by dividing them into four regions, and to treat these separately. It must be emphasized that the division into these four regions, and the association of specific behaviors with each region, is not at all clear-cut, but is a way of making sense of a rather bewildering collection of data.

The four divisions, shown in Figure 3.2, are the motor and premotor cortex; the prefrontal cortex (sometimes referred to as “frontal granular cortex” because of the type of cells predominant in this area, or as *dorsolateral* cortex); Broca’s area, which we assume to exist in the left frontal lobe only (at least for the right-handed—see Chapters 7 and 14); and the orbital (or *orbitofrontal*) cortex. We will examine the effects of lesions of each of these regions in turn.

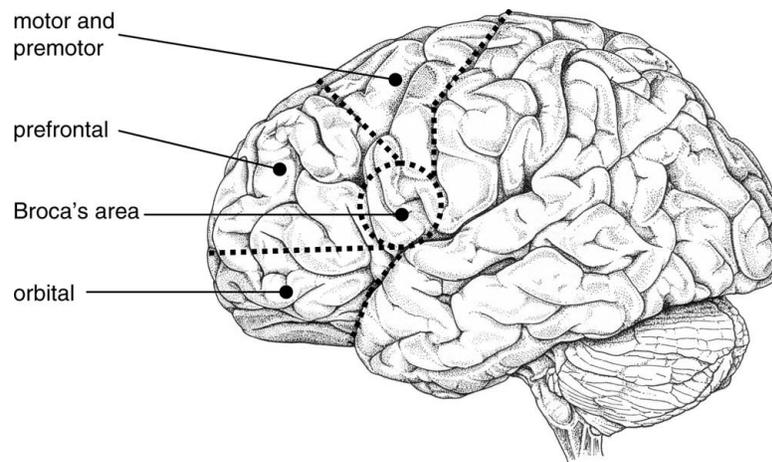


FIGURE 3.2. The four main divisions of the cortex of the frontal lobe.

THE MOTOR AND PREMOTOR CORTEX

When the organization of the cerebral cortex was introduced in Chapter 2, the model of three levels of control, of decreasing specificity and increasing integration, was suggested: primary, secondary, and tertiary. The frontal lobe control of motor function provides a clear illustration of the model.

The primary region is the *motor cortex*, or “motor strip,” which has already been described. As cells in this region connect directly with spinal motor neurons and motor nuclei in the cranial nerves, focal lesions of a specific area will lead to a loss of voluntary control over the precise area of the body that is “mapped” onto that area of the motor cortex. The general arrangement of this mapping in the form of an homunculus was illustrated in Figure 2.9. Although there is variation among individuals, the mapping is sufficiently precise for it to have been proposed, and demonstrated as a practical proposition (Prochazka, Mushahwar, & McCreery, 2001), that a prosthesis for spinal injuries might operate by picking up the signals that originate in the motor cortex and relaying them by wiring and a computer interface, past the damage in the spine, directly to the point at which they should be fed into the peripheral nervous system and on to the muscles (or to an artificial limb). Damage to the motor cortex results in chronic deficits in fine motor control, which may be seen most clearly in movements of the hands, fingers, and face, and in a reduction in the speed and strength of limb movements.

Adjacent and anterior to the motor cortex, the *premotor cortex* forms the *secondary level of motor control*. Cells in this region contribute to motor control by forming connections in various subcortical centers, particularly in the basal ganglia, and there seem to be distinct systems for limb movements and for whole body movements. Because the control is exerted by influencing the operation of these lower centers, the effects of lesions of this region are less specific and more subtle, for the basic aspects of control are still being carried out by centers in the basal ganglia, the thalamus, and elsewhere. In particular, lesions of the premotor cortex (with some contribution from parietal cortex) seem to impair the way in which separate movements of the limbs, the hands, and gross body movements are integrated into fluid sequences of action.

Among the signs of lesions of this region, apart from the specific effects on particular limb movements, are some changes of a reflex kind. In *gegenhalten*, which literally means “counterpull,” there is an involuntary resistance to movement, so that if the forearm, held in a certain

position quite loosely, is moved by the examiner, a marked resistance preventing movement of the limb may develop unintentionally. Similarly, there may be an involuntary grasp of a hand or object placed in the patient's hand, despite conscious attempts not to take hold of the object in this way. There may also be changes in gait (rather similar to those seen with damage to the cerebellum), so that the patient shows *marche a petit pas*, walking rather clumsily in little rapid steps.

The tertiary level of motor control is in the next region of the frontal lobes, the prefrontal cortex, which is discussed below, but there are some specific functions associated with the three levels of the motor cortex that should be mentioned here. These concern control of the face, and the data come from the study of patients who have had the motor and sensory cortex for the *face region* on one side of the head surgically removed (Taylor, 1979). These patients do not in fact suffer a lasting problem in controlling the face, or in receiving and interpreting sensation from it, largely because the motor and sensory connections to the head (via the cranial nerves) are bilateral, and are not contralaterally organized as in the rest of the body (via the spine). On recovery, the patient's face is normally expressive, and facial movements can be imitated on command. There are some expressive speech difficulties immediately following the operation but, apart from some slight residual difficulty, this clears within the first year after surgery. The patients, rather surprisingly, nevertheless show marked difficulties with verbal fluency, phonetic discrimination, spelling (especially after a left-sided operation), and design fluency (after a right-sided operation).

The verbal fluency deficit is seen when the patient is asked to give a series of words beginning with a particular letter, or belonging to a particular category such as "fruit and vegetables," within a given time limit, usually 1 minute. (This deficit is also seen with prefrontal lesions, but is reported to be less severe.) Design fluency is tested similarly, except that here the patient is asked to make as many nonrepresentational drawings as possible within a 5-minute period. While normal subjects may produce about 15 words beginning with, say, *s* in 1 minute, patients with a verbal fluency deficit may only manage 4 or 5. They also cannot identify, with normal accuracy, phonemes (the building blocks of spoken language, roughly equivalent to syllables) that have been embedded in nonsense words, and there is an associated impairment in spelling. These difficulties occur in the absence of any other significant problems with the expression or understanding of language, and it is presumed that there is some essential connection with the motor control of the face, or alternatively in inhibiting recently produced responses, although the real origin of these deficits remains something of a mystery.

THE PREFRONTAL CORTEX

A number of rather different functions are associated with the prefrontal cortex, and this is not surprising in view of its extensive area. Prominent among these functions are several linked with motor control, which form the tertiary level of the motor control system.

The *tertiary level of motor control* exerts its influence by operating upon all lower levels of the motor system, both in the cortex and at subcortical levels. The control is therefore not of specific components of movement, but rather of the planning and programming of motor acts and their flexible adaptation to particular circumstances. Monitoring of movement patterns ensures that behavior is appropriate and adaptive, and lesions of the prefrontal cortex therefore result in motor behavior becoming inflexible and stereotyped.

An example of this inflexibility can be seen in the Wisconsin Card Sorting Test. A set of four cards is placed before the patient, as shown in (a) at the top of Figure 3.3. The cards contain one, two, three, or four shapes, each in one of four forms and in one of four colors. The patient is then asked to sort cards containing similar stimuli (b) into piles below the initial set, but without being told the rule for sorting. The cards might thus be sorted according to the number of shapes, the type of form, or the color. The examiner tells the patient whether she or he is correct after each card is sorted, so that the patient has to discover the correct rule by which to sort the cards. Normal subjects, and also

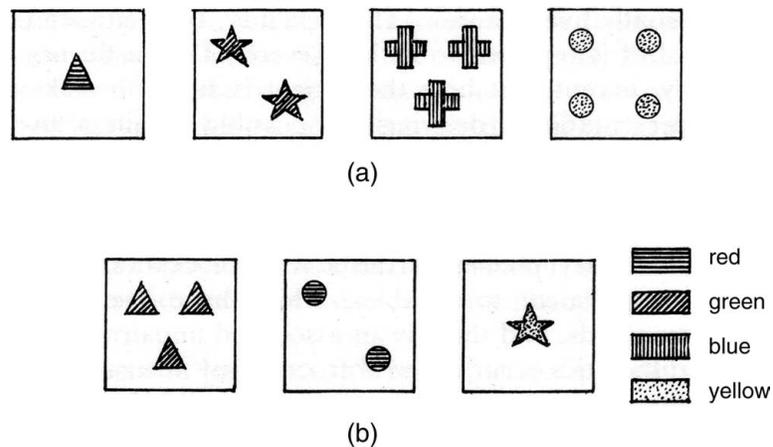


FIGURE 3.3. Wisconsin Card Sorting Test: (a) the four cards under which the test cards must be sorted and (b) examples of the test cards.

patients with frontal injuries, are able to discover this initial rule. However, after the sorting rule has been learned, the examiner changes the rule without warning the patient. This initially causes puzzlement and frustration in most subjects, but normal subjects and patients with nonfrontal lesions rapidly realize what has happened and search out the new rule; they readily adapt to subsequent changes of rule. However, frontal lobe patients are extremely slow to adapt to the new rule, and many do not manage it at all. They simply continue sorting according to the first rule, getting responses correct only by chance. This behavior, continuing with a response once it is no longer appropriate, is known as *perseveration*. The general problem seems to be one of impaired inhibition of cognitive responses; that is, once certain responses have been brought to the fore they cannot be replaced by more appropriate responses. A similar example of perseveration is seen in the patient who, asked to subtract 7 serially from 100, responds with “93 . . . 86 . . . 76 . . . 66 . . . 56 . . . ” instead of “93 . . . 86 . . . 79 . . . 72 . . . ”

A final example may be shown with the Stroop phenomenon. Here, subjects are asked to follow a list of words that are the names of colors but that are printed in ink of a contrasting color, and to name the color of the ink. Normal subjects take longer to go down such a list than a list of comparable words that are not color names, because the color names interfere with naming the ink colors. The interference is quite extreme in some frontal lobe patients, who find it impossible to inhibit reading the color names (Perret, 1974). The failure to inhibit associated but incorrect responses may also be seen in the responses to vocabulary tests, where the patient’s response is confused with that appropriate to a similar-sounding word. For example, the patient correctly reads *river*. The next word is *see* but the patient reads it as *ocean*, confusing it with the similar sounding word *sea*.

Another aspect of motor control in prefrontal cortex is the *programming and planning* of sequences of behavior. At the level of programming simple sequences, it is best demonstrated by the 1979 work of Kolb and Milner, as cited in Kolb and Whishaw (2003). They asked patients to imitate certain facial gestures (see Figure 3.4), and found that patients with prefrontal lesions were not impaired when imitating single gestures, but when asked to copy a series of three gestures, they showed significant impairment. A similar difficulty with planning is sometimes shown by patients who perform badly on paper and pencil mazes, such as the Porteus mazes (see Figure 3.5), because they are not able to build up a sequence of moves that will get them to the goal.

Patients with difficulties of this kind may also have difficulties with *problem solving* of a more general kind, including visuo-constructive problems. When asked to perform the block design task mentioned



FIGURE 3.4. Examples of the stimuli used by Kolb and Milner, showing gestures to be copied by the patients.

above, these patients may fail. Part of the difficulty may lie in the way in which the design to be reproduced is presented, for it does not clearly show how individual blocks will form parts of the design (see Figure 3.6). If this information is given to the patient he or she may be able to perform normally. The patient's difficulty again seems to be in building up a plan of component moves to attain a complex goal. In a similar way, patients may be unable to reproduce a complex nonfigurative drawing unless they are specifically taught to build it up in a series of discrete steps.

While patients with prefrontal lesions do not show difficulty with arithmetical computations (compare the effects of parietal lesions,

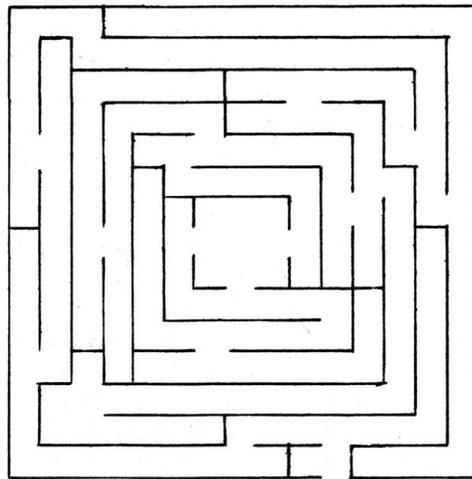


FIGURE 3.5. A maze similar to the Porteus mazes at the adult level of difficulty.

p. 108), they may have difficulty with arithmetical problems, especially if these are couched in the form of sentences, such as “There were 18 books on two shelves, and there were twice as many books on one as on the other. How many books were on each shelf?” The patient seems unable to abstract the elements of the problem, and then to arrange these into a plan for its solution (Christensen, 1975).

A final, and specialized, aspect of motor control is that of *voluntary eye movements*. Prefrontal cortex contains an area known as the *frontal eye fields*, in which eye movements related to scanning of the environment and the inspection of visual objects are controlled. This can be illustrated by comparing the recordings of eye movements of patients with damage to this area with those of normal subjects when shown a complex picture (see Figure 3.7). Normal subjects rapidly detect the picture’s most significant and informative elements and follow a series of glance paths between these elements when asked to extract meaning from it. In marked contrast, those with frontal lobe injuries show a disorganized series of movements that lack the adaptive articulation of normal subjects (Yarbus, 1967). This difficulty may underlie a number of more general problem-solving deficits in frontal lobe patients, as well as the poor performance in visual search tasks (locating a target item in a larger array of similar items) that is sometimes apparent. Alternatively, all these difficulties, including the eye movement problem, may be manifestations of a basic deficit in generating and operating strategies for collecting and processing information needed for intellectual tasks.

The motor difficulties of damaged frontal lobe patients may also show in reduced spontaneous behavior, and they may therefore be

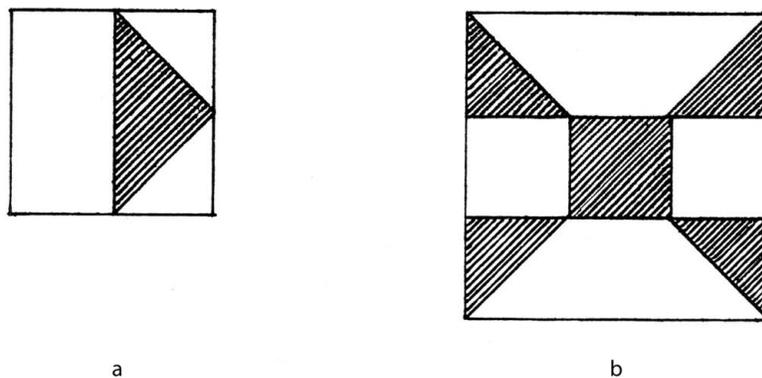


FIGURE 3.6. The type of pattern to be constructed in a block design task using four blocks (a) and nine blocks (b).

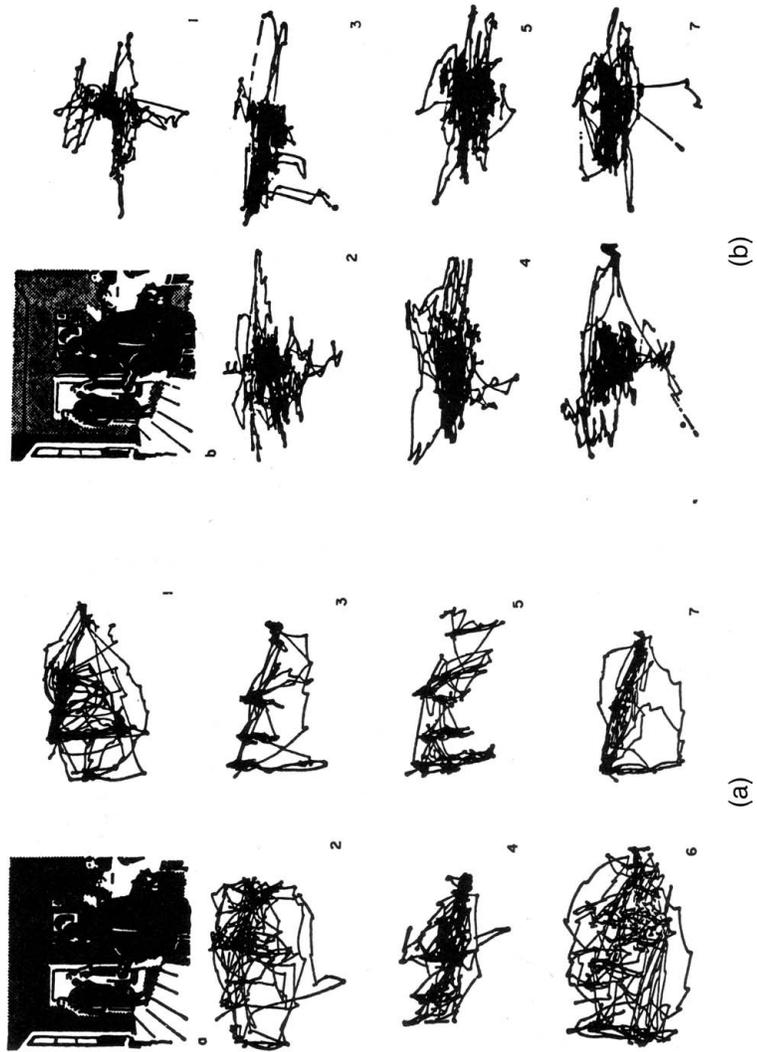


FIGURE 3.7. Eye movement patterns of (a) a normal subject and (b) a patient with a massive frontal lobe lesion while examining the picture shown top left: (1) free observation, and after the questions; (2) “Is the family rich or poor?”; (3) “How old are the people in the picture?”; (4) “What were they doing before the man entered the room?”; (5) “Can you recall how the people were dressed?”; (6) “How were the people and furniture placed in the room?”; (7) “How long had the man been away from his family?” (Reprinted from A. R. Luria, *The Working Brain*, Penguin, 1973.)

referred to as *pseudo-depressed*. Such patients sit around, have almost nothing to say, engage in little activity, and typically present a flat, emotionless expression. The deficit in verbal fluency (by which patients with frontal lesions are unable to generate a normal number of different words beginning with a given letter in a set period of time) is considered to be associated with the overall reduction in response emission (Milner, 1964). Some writers, notably Luria (Luria, Pribram, & Homskaya, 1964), have considered this and other frontal lobe motor deficits to be due to a failure in the verbal regulation of behavior.

The idea that underlies this concept of *verbal regulation* is that we use covert language to control complex motor activity. For example, you may be aware when learning some new motor skill, say driving a car, of “talking to yourself” to help sort out what to do, but with practice the skill becomes more automatic and the internal commentary is no longer necessary. It has certainly been shown by Luria that children can be helped to perform such tasks as a go/no-go problem (squeeze the bulb if the green light comes on, but do not squeeze if the red light comes on) at a younger age than would normally be possible if they are taught to use explicit verbal mediation (saying “press” or “don’t press” to the green or red light). The idea is that as a skill becomes practiced, the verbal mediation becomes covert and internalized, and the difficulties that frontal lobe patients have in motor control can thus be explained in terms of a deficit in the verbal regulation of behavior. While this hypothesis is widely quoted, Drewe (1975) tested frontal lobe patients with go/no-go learning tasks and failed to find clear support for it.

Some *perceptual deficits* are also associated with prefrontal cortex. One of these is perhaps unexpected and concerns making judgments about egocentric space. Semmes and colleagues showed patients diagrams of the human figure from the front and back with numbers indicating parts of the body: the palm of the left hand, the back of the right calf, and so on. The patients were given a number and asked to point to the appropriate part of their own bodies. Frontal lobe patients did poorly on this task in comparison with patients with other lesions (Semmes, Weinstein, Ghent, & Teuber, 1963). (If you think this is an easy task, try standing in front of a mirror with a child and asking the child to point to various parts of the body while looking in the mirror.)

Associated with this deficit is impaired performance in the Aubert task. In this task the patient is seated in a dark room in a chair that can be tilted to the left or right. In front he sees a luminous rod that is to be set to the vertical. Normal subjects show an effect of head and body tilt, so that the rod is misaligned away from the horizontal in the opposite direction to that of the subject’s tilt. Greater tilt produces greater error. The effect is much more pronounced in those with frontal lesions

(Teuber & Mishkin, 1954). Teuber (1964) has proposed that both of these perceptual deficits may be explained by impaired *corollary discharge*. Here, the idea is that when movements are executed, information is sent out to other parts of the system so that the effects of the movement can be anticipated and accounted for. The obvious example is that when you move your eyes the world does not appear to move but remains apparently stable. If your eyes are passively moved (try gently pushing your half-open eye with a finger on the eyelid) then the world does apparently move about. When you make active eye movements, the systems interpreting information from the eye are forewarned and make all the necessary adjustments, but these cannot be made when unexpected passive movements are imposed upon the eye. A gymnast performing on parallel bars would come dramatically unstuck if the world did not appear to remain stable as the body moved through the exercises. The perceptual world is in fact whirling about, but knowledge of the muscle movements allows all the necessary compensation to be introduced.

In the Aubert task, it is assumed that damaged frontal lobe patients fail to generate appropriate corollary discharges for the compensation in muscular tonus that occurs during tilt, leading to a faulty perception of where the vertical should be. By extension, this can be applied to the judgment of egocentric space. The patient fails to keep proper track of where his or her body is in space, and cannot accurately relate the external world to it.

One specific aspect of *memory* that is affected by prefrontal lesions is that of recency. In other words, if a patient is shown a series of items one at a time, and then shown two items, he or she may be able to recognize them correctly as having been in the list but may not be able to report correctly which of the items was presented more recently (Milner, 1971). This seems to be the only pure memory function to be affected by frontal lesions, although various aspects of learning may be affected in a secondary way by other frontal lobe deficits. In particular, *working memory* deficits, which reflect aspects of attention and concentration, may be associated with frontal lobe lesions. It is not uncommon to see patients who complain of memory problems and yet score at their premorbid level on formal tests of memory in the consulting room. The explanation is normally that in everyday life, unlike the consulting room, the patient fails to attend adequately to information so that the memories are not efficiently laid down. The memory processes function normally, but material does not get fed into these processes in a normal fashion, resulting in the problems that the patient experiences.

Owen, Evans, and Petrides (1996) proposed a two-stage model of working memory that involves, first, organization and sequencing, and

second, monitoring and manipulation. They linked the first stage to the ventrolateral frontal cortex, and the second to dorsolateral prefrontal cortex. Goldman-Rakic (1996) has proposed the concept of On-Line Memory, associated with dorsolateral prefrontal cortex, which confers the ability to hold separate pieces of information together and so blend otherwise unrelated memories into distinct lines of thought. This is a variant of the concept of working memory but with the distinct property of facilitating the association of otherwise unrelated thoughts and memories. It is attractive in explaining a range of the problems experienced by patients with frontal lobe disorders.

Deficits in attention feature in a number of models of frontal lobe function. Attention is a complex concept, but an influential contemporary model has been provided by Posner and Petersen (1990), although it is not without its critics. According to this model there are three main components in the attentional process: disengaging the current focus of attention (detection, a function of the posterior parietal cortex); shifting attention to a new location (orientation—frontal eye fields and related structures); reengagement at the new location (discrimination—thalamus). However, also involved in the two latter processes is the cingulate gyrus. The cingulate gyrus is on the medial surface of the frontal lobes, tucked into the midline division between the two hemispheres. In light of its protected position, it is rarely directly damaged in cerebral trauma, and therefore not well understood, but may well nonetheless be involved in the attentional deficits that then have a consequential impact upon other frontal lobe functions.

BROCA'S AREA

The third region of the frontal lobes is Broca's area, which is sited in the left frontal lobe and has the primary function of expressive speech. This, however, will be discussed with other elements of the language system in Chapter 7.

THE ORBITAL CORTEX

The final region of the frontal lobes subserves aspects of *personality* and *social behavior*. The classic example of the effects of lesions of this area is that of Phineas Gage, a construction worker on the American railroads who in 1848 suffered an accident in which an iron bar, over 3½ feet long and 1¼ inches thick, was blown through the front of his head, entering at the lower cheek and exiting from the upper forehead (see Fig-



FIGURE 3.8. The skull of Phineas Gage, after a contemporary drawing.

ure 3.8). Gage survived but underwent a marked change in personality. From being a capable foreman and an efficient worker (Harlow, who attended Gage and reported the case, says he was “energetic and persistent in executing all his plans of operation”), he became impulsive, willful, inconsiderate, and obstinate. He took to swearing, which had not previously been his habit, and continually changed his mind. Indeed, he was so dramatically altered that his “friends and acquaintances said that he was no longer Gage.”

Although this type of personality change generally follows only large, and usually bilateral, frontal injuries (often the result of road traffic accidents), it is not at all uncommon. The changes may also be accompanied by what can only be described as silliness, and patients may constantly sing, whistle, and repeat rather poor jokes. In fact there is a term for this puerile kind of jocularity: *witzelsucht*. An additional symptom may be a lack of the social graces; these patients engage quite freely in belching, picking their noses, and even less savory socially proscribed activities in public, without any apparent concern. A similar effect can sometimes be seen in the maze performance of frontal lobe patients. They may simply go through the walls of the maze, failing to pay attention to the rules governing performance. Alternatively, asked to learn a maze pathway in which the correct path is not apparent (e.g., the stylus maze; Milner, 1965) but in which feedback is given at each point, the patients do badly because they fail to obey the rules or make appropriate use of the feedback information given to them. An alternative pattern of response may be that of indifference, lack of initiative, and general loss of drive, part of the pseudo-depression already noted. These

patients say very little and exhibit almost no emotional expression. This may also be partly associated with *anhedonia*, a loss of the ability to experience pleasure.

Finally there may be associated changes in *sexual behavior*. These may be in terms of a loss of social inhibitions, resulting in exhibitionism and public masturbation, although the amount of sexual activity is not increased. However, the changes are more often in terms of a loss of libido (which may also be associated with prefrontal lesions). The capacity for sexual activity is not lost, but patients lose interest in it.

Orbital lesions may therefore result in personality and social behavior changes that may loosely be characterized by impulsiveness, face-tiousness, and mild euphoria; by diminished anxiety and concern for the future; and by lack of initiative and spontaneity. It was these observations that led to this area being the site of the prefrontal leucotomy, which will be discussed in Chapter 9.

Substantial injuries to the frontal lobe, particularly where there is both a change in personality and difficulties of planning and execution, can be among the most disabling of cerebral injuries. For obvious reasons, road traffic accidents not infrequently result in extensive frontal damage. It is common to see patients who, despite scoring normally on conventional tests of general cognitive ability, require 24-hour care and supervision. This is because, although their intellectual functions are retained, they are unable to apply these cognitive skills appropriately in their everyday life. Such patients may compromise their own safety by leaving the stove lit, the gas turned on, or taps running, or failing to lock doors. They are vulnerable to the persuasions of salesmen at their door and make ill-considered financial decisions; they are generally unable to budget their own money. They fail to plan meals or to shop and stock their larder sensibly, and may also neglect their own hygiene and fail to change or wash their clothes. Activities are impulsively conceived but rarely carried out, or if begun not completed. Domestic bills go unpaid.

In some of these individuals, particularly if there is some pre-accident history of aggressive behavior, there may be poor temper control and episodic aggression. Although there is usually some trigger for this aggressive behavior in the form of frustration or irritation, the response is disproportionate and poorly controlled and it can result in significant violence. Young men with this problem (and they are those most likely to be involved in road traffic accidents) can frequently end in trouble with the police as a result of their neurological injury. There are drugs that can assist with this problem, but the lack of insight that often accompanies the disorder makes behavioral change difficult to achieve.

In general, this *frontal lobe syndrome* is difficult to rehabilitate because of the lack of insight and awareness on the part of patients. If

frontal lobe patients were aware of their problem, they would no longer have the problem, retaining the cognitive abilities to perform the relevant tasks appropriately. It is this lack of insight, and the inability to weigh matters in the balance and appreciate the consequences of their actions, that makes the frontal lobe syndrome so disabling and leads to a substantial, and lifelong, need for care and supervision.

An interesting aspect of the frontal lobe syndrome is that it can be difficult to demonstrate and assess in the consulting room. In an environment where there are clear task demands and few distractions, these patients can perform cognitive tasks surprisingly well. Most psychological tests involve clear instructions and a procedure that is controlled by the examiner, and as a result the examiner “acts as the patient’s frontal lobes.” A number of tests have been devised that attempt to bring a more naturalistic element into the examination, most notably the Behavioural Assessment of the Dysexecutive Syndrome (BADS; Wilson, Alderman, Burgess, Emslie, & Evans, 1996). There are a number of tests in this battery, but most require some planning and organization on the part of the patient. For example, in Key Search patients must draw a route on the plan of a field to show how they would search the field to be sure to find keys they had lost somewhere in the field. In the Zoo Maps a visit to the zoo must be planned with the locations to be visited listed and a number of rules provided governing the route to be taken. Again, subjects draw their planned route once they have worked out what it should be.

In another test, the Cognitive Estimates Test (CET; Shallice & Evans, 1978), patients must estimate quantities, which they are unlikely to know, by reasoning from their real world experience. Typical questions are “At what speed do racehorses gallop?” and (my favorite question), “How many camels are there in Holland.” Minor inaccuracies are allowed; it is the extreme and “bizarre” estimates that are of clinical significance—the patient who considers that racehorses gallop at either 10 or 80 miles per hour. Such tasks are helpful in identifying frontal lobe problems, but the correspondence between poor test scores and problems in everyday life remains rather weak.

MODERN THEORIES OF FRONTAL LOBE FUNCTION

One difficulty, which will be apparent from the preceding discussion, is that there is a broad variety of behaviors associated with the functions of the frontal lobes. Rather than seeking an overall encompassing explanation of frontal lobe function, modern theories have rather concentrated on how diverse processes might be integrated.

The most influential of these theories has been Shallice's Supervisory Attentional System (SAS) model (Shallice, 1982, 2002; Shallice & Burgess, 1996). The basic model is illustrated in Figure 3.9.

The essential idea is that there are schema control units (a schema refers to a plan of action) that govern patterns of action, operating on the basis of information received from the perceptual system. There are inhibitory links among the schema control units that permit the most important activity to be dominant. The way they operate, and so govern the pattern of behavior, is controlled by a "contention scheduling system," which ensures that appropriate priority for behaviors is maintained. When a novel goal state arises (the individual needs to perform a novel behavior) then this nonroutine activity requires the intervention of the overarching supervisory attentional system. The model has at least two major advantages in that it explains distractibility, as strong cues cannot be overridden by the SAS, and it also explains perseveration by the dominance of particular schemas that the SAS is unable to displace. In subsequent versions the model has become very much more complex, incorporating attentional systems, among others. Nevertheless, it provides a useful framework for demonstrating how particular behaviors may be triggered by particular stimuli, and how automatic behaviors

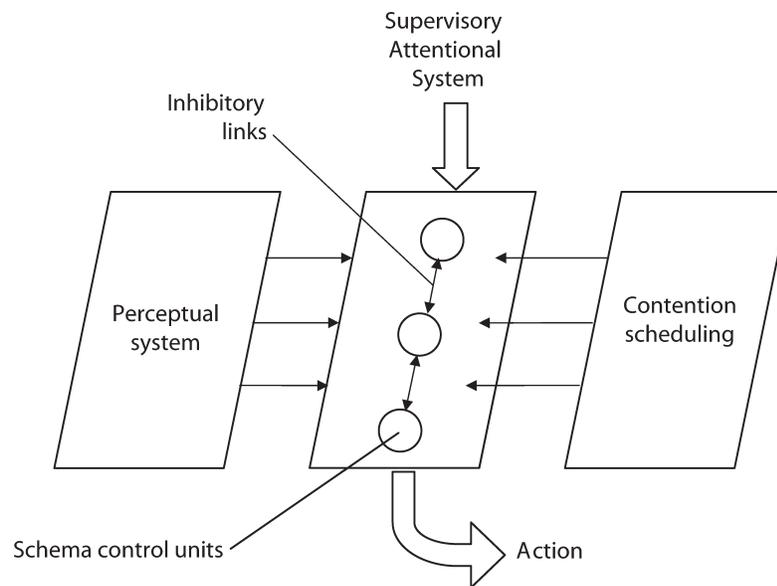


FIGURE 3.9. Shallice's model of the Supervisory Attentional System. (Redrawn after Shallice, 1982.)

may be initiated and terminated. It is less adequate in describing how new behaviors are formulated and selected in the prefrontal cortex.

Another influential model has been that of Stuss and Benson (Stuss & Benson, 1986; Stuss & Knight, 2002). In its fundamentals, this model sees the frontal cortex as involved in a series of processes controlling behaviors. In the first stage, *drive* and motivation are modulated by the orbital region, while medial structures are linked to the initiation of activity. Secondly, *sequencing* in both dorsolateral prefrontal cortex and orbital cortex creates and maintains temporal order. The third and fourth processes *anticipate* and *select* salient goals, to be followed by procedures for *preplanning* the behaviors required for potential outcomes. Finally, *monitoring* evaluates the success of these outcomes.

An alternative approach has been to recognize the relevance of emotion in behavioral control. Failures of emotional perception have now been recognized as associated with both the orbital and cingulate cortices. This deficit as now sometimes termed *alexithymia*, literally an inability to read emotions. Perceptual disorders of emotional recognition may relate to acknowledging one's own emotional state, but may also be expressed in a deficit of identifying emotions in the faces or voices of others. There are two important models broadly associated with this observation, associated with Rolls and with Damasio.

Rolls (2000) argued that the orbital cortex is deeply involved in the processing of both taste and olfaction, but that it also establishes the reward value for secondary reinforcers. Hence, this region of the frontal lobe provides a means of establishing secondary reinforcers on the basis of more primitive responses and providing a reward value for a variety of other environmental stimuli. This concept is a development, albeit a more detailed and sophisticated one, of the observation already noted, that frontal lobe patients may experience anhedonia and so fail to experience appropriate rewards as a consequence of their behavior; as a result the behaviors decline in frequency and such patients become relatively apathetic and inert. (Those interested in a brief but authoritative review of the brain systems underlying emotion should consult Dalgleish, 2004.)

Damasio's Somatic Marker Hypothesis (Damasio, 1997, but a popular and readable account is to be found in Damasio, 2006) recognizes that the brain cannot be considered to be independent of the body in which it is placed, the somatic environment. Specific stimuli trigger the reactivation of somatosensory patterns that act as markers of these stimuli. The reactivation may occur through neural interactions with the body ("body loops") or simply within the somatosensory maps of the cortex. The reactivation constrains reasoning and decision making as the somatic marker provides a biasing system, outside consciousness, that

facilitates either appetitive or avoidance behavior. As situations with personal or social significance are generally associated with reward and punishment, with pleasure and with pain, these somatic markers provide critical signals in many situations of reasoning and decision making, especially if some social context is involved. While Damasio's model does not permit the detailed predictions concerning cognitive behavior that are afforded by other models, it provides a salutary lesson in the poverty of considering the brain apart from its biological environment.

LATERALIZATION OF THE FRONTAL LOBE

A theme that runs through almost all of this book is that of cerebral lateralization. It has been clear throughout the history of neuropsychology that there are differences between the functional specializations of the two hemispheres. In terms of clinical evidence, this means that some deficits are more frequently associated with right sided lesions and some with left, and some deficits only appear with bilateral lesions (in both hemispheres). It should be emphasized that this is rarely a firm distinction, but is one of *relative specialization*. The evidence relating to frontal lobe injuries makes this clear.

Benton (1968) constructed a study in which he gave six tests to a group of patients with either left, right, or bilateral frontal lobe damage. Two of the tests (Verbal Fluency and Verbal Learning) were expected to be associated with left sided lesions, and indeed in both tests the patients with left lesions more frequently showed a deficit than those with right lesions. Those with bilateral lesions had difficulty as frequently as those with left lesions for Verbal Fluency, and more often for Verbal Learning. The "right hemisphere tasks" (Block Design and Design Copying) also produced the expected results: those with right lesions or bilateral lesions had a deficit more commonly than those with left lesions. The final two tests (Time Orientation—to see if patients know the time, day, and date and can locate themselves in time—and the Gorham Proverbs Test, in which the patient must explain the meaning of some well-known proverbs) showed that impaired performance was associated much more frequently with bilateral lesions than with unilateral lesions of either side.

These hemisphere specializations can loosely be divided into verbal and symbolic (left hemisphere) versus visuospatial (right hemisphere), although we shall question the validity of this distinction in Part III. In fact, evidence against such a simple dichotomy is already apparent in that failure in the Proverbs task only occurs commonly with bilateral lesions. Also, the test of identification of body parts in egocentric space

that has already been mentioned is associated with lesions of the left frontal lobe, and not the right, as might have been expected. This information on lateralization is important, not only because it helps in locating lesions in patients under examination, but also for the construction of neuropsychological models of the organization of the brain.

CONCLUSION

Some of the most important specific functions associated with the frontal lobes have been discussed, and are summarized in Table 3.2.

Although we have no space to deal with it here, there is very considerable research literature on the effects of frontal lobe lesions in animals. The evidence that this provides is extensive and complex, but given the difficulties of generalizing results even from apes to humans, the results are remarkably similar. Some deficits found in higher animals do not have clear parallels in humans, but there are few fundamental disagreements. (See the Further Reading section at the end of this chapter for the various reviews that are available.) With reference to the suggestion that the frontal lobes are in some way special in man, neither the evidence

TABLE 3.2. Some Specific Functions Associated with Regions of the Frontal Lobe

| |
|---|
| Motor and premotor cortex: |
| Primary and secondary levels of motor control |
| Verbal fluency and design fluency |
| Spelling |
| Prefrontal: |
| Tertiary level of motor control |
| Adaptability of response pattern |
| Programming and planning of sequences of behavior |
| Level of response emission |
| Verbal regulation |
| Problem solving |
| Voluntary eye movements |
| Perceptual judgment |
| Memory and attention |
| Broca's area: |
| Expressive speech |
| Orbital cortex: |
| Personality |
| Social behavior |

from animal studies (when compared with human clinical data) nor that from patients with brain damage would support this conclusion. We may still regard the frontal lobes as undertaking some of the highest intellectual tasks in humans, but there is no essential discontinuity between these abilities in humankind and those in the higher animals.

Is it possible to conceive of general neuropsychological systems that might account for the deficits found in the frontal lobes? Despite recent advances, the answer at present is no, which should hardly surprise us in view of the extent of frontal cortical tissue. Teuber's corollary discharge theory holds some, but not complete, explanatory power. Luria (1973) has suggested that the three special functions of the frontal lobes are the regulation of activation processes; the execution of verbally programmed behavior processes; and problem-solving behavior. This explanation is also incomplete, and cannot be supported in all its details by scientific evidence. A general theory that the frontal lobes operate by undertaking the temporal structuring of behavior (Fuster, 1980) is no more satisfactory. The more recent theories of Shallice and Burgess, Stuss, Rolls, and Damasio discussed above have all provided useful insights and new ways of conceptualizing the contribution of the frontal lobes, but have yet to succeed in providing a single integrated model of frontal functions. The frontal lobes are involved in many functions, and we are not yet at the stage where neuropsychological systems can be so explicitly summarized. Nor are many neuropsychologists sure whether they are seeking a single theory of frontal lobe function, or whether the ideal model would relate general functions to more circumscribed areas within the frontal lobes. The delineation of the frontal lobes is, in any event, a rather artificial construct (as for all the lobes) and it may be entirely unreasonable to maintain the ambition of a simple description of their functions.

We can conclude that the frontal lobes play a major role in the higher levels of motor control and in the planning and controlled execution of motor acts and skills. They contribute also to general problem-solving behavior, and the regulation of eye movements is an important aspect of this performance. Associated with these functions, in prefrontal cortex, are some specific verbal abilities, some perceptual functions, and some limited aspects of memory. Frontal regions are also involved in the regulation of attention, and in motivation and the regulation of behavior more generally. Lesions, especially in the orbital cortex, may lead to changes in personality and social behavior.

At about the turn of the 20th century, Hughlings Jackson described the frontal lobes as the "least organized" area of the cortex. It has always also been the least understood, and remains the most challenging to neuropsychologists.

FURTHER READING

There are some general texts that apply to all the chapters in Part II, and these have already been listed at the end of Chapter 1 (p. 20). Some texts that relate more specifically to the frontal lobes will be found among the references below.

REFERENCES

- Benton, A. L. Differential Effects of Frontal Lobe Disease, *Neuropsychologia*, 6 (1968), 53–60.
- Black, F. W. Cognitive Deficits in Patients with Unilateral War-Related Frontal Lobe Lesions, *Journal of Clinical Psychology*, 32 (1976), 366–372.
- Christensen, A.-L. *Luria's Neuropsychological Investigation* (Copenhagen, Denmark: Munksgaard, 1975).
- Dalgleish, T. The Emotional Brain, *Nature Reviews: Neuroscience*, 5 (2004), 582–589.
- Damasio, A. R. Towards a Neuropathology of Emotion and Mood, *Nature*, 386 (1997), 769–770.
- Damasio, A. *Descartes' Error: Emotion, Reason, and the Human Brain* (Revised edition, New York: Vintage Books, 2006).
- Drewe, E. A., No-No Go Learning After Frontal Lobe Lesions in Humans, *Cortex*, 11 (1975), 8–16.
- Duncan, J. D., Seitz, R. J., Kolodny, J., Bor, D., Herzog, H., Ahmed, A., et al. A Neural Basis for General Intelligence, *Science*, 289 (2000), 457–460.
- Fuster, J. M. *The Prefrontal Cortex* (New York: Raven Press, 1980).
- Goldman-Rakic, P. S. The Prefrontal Landscape: Implications of Functional Architecture for Understanding Human Mentation and the Central Executive, *Philosophical Transactions of the Royal Society B: Biological Sciences*, 251 (1996), 1443–1445.
- Kolb, B., & Whishaw, I. Q. (Eds.). *Fundamentals of Human Neuropsychology* (San Francisco: Freeman, 2003).
- Luria, A. R. *The Working Brain* (London: Penguin, 1973).
- Luria, A. R., Pribram, K. H., & Homskaya, E. D. An Experimental Analysis of the Behavioural Disturbance Produced by a Left Frontal Arachnoidal Endothelioma (Meningioma), *Neuropsychologia*, 2 (1964), 257–280.
- Milner, B. Some Effects of Frontal Lobectomy in Man, in J. M. Warren & K. Akert, eds., *The Frontal Granular Cortex and Behavior* (New York: McGraw-Hill, 1964).
- Milner, B. Visually-guided Maze Learning in Man: Effects of Bilateral Hippocampal, Bilateral Frontal, and Unilateral Cerebral Lesions, *Neuropsychologia*, 3 (1965), 317–338.
- Milner, B. Interhemispheric Difference in the Localization of Psychological Processes in Man, *British Medical Bulletin*, 27(1971), 272–277.
- Owen, A. M., Evans, A. C., & Petrides, M. Evidence for a Two-Stage Model of Spatial Working Memory Processing with the Lateral Frontal Cortex: A Positron Emission Tomography Study' *Cerebral Cortex*, 6 (1996), 31–38.

- Perret, L., The Left Frontal Lobe of Man and the Suppression of Habitual Responses in Verbal Categorical Behaviour, *Neuropsychologia*, 12 (1974), 323–330.
- Posner, M. I., & Petersen, S. E. The Attention System of the Human Brain, *Annual Review of Neuroscience*, 13 (1990), 25–42.
- Prochazka, A., Mushahwar, V. K., & McCreery, D. B. Neural Prosthesis, *Journal of Physiology*, 533 (2001), 99–109.
- Reitan, R. M., & Davison, L. A. *Clinical Neuropsychology* (New York: Wiley, 1974).
- Rolls, E. T. The Orbitofrontal Cortex and Reward, *Cerebral Cortex*, 10 (2000), 284–294.
- Semmes, J., Weinstein, S., Ghent, L., & Teuber, H.-L. Impaired Orientation in Personal and Extrapersonal Space, *Brain*, 86 (1963), 747–772.
- Shallice, T. Specific Impairments of Planning, *Philosophical Transactions of the Royal Society B: Biological Sciences*, 298 (1982), 199–209.
- Shallice, T. Fractionation of the Supervisory System, in D. T. Stuss & R. T. Knight, eds., *Principles of Frontal Lobe Functioning* (New York: Oxford University Press, 2002).
- Shallice, T., & Burgess, P. W. The Domain of Supervisory Processes and the Temporal Organization of Behaviour, *Philosophical Transactions of the Royal Society B: Biological Sciences*, 351 (1996), 1405–1412.
- Shallice, T., & Evans, M. E. The Involvement of the Frontal Lobes in Cognitive Estimation, *Cortex*, 14 (1978), 294–303.
- Stuss, D. T., & Benson, F. D. Control of Cognition and Memory, in E. Perecman, ed., *The Frontal Lobes Revisited* (Hillsdale, NJ: Erlbaum, 1986).
- Stuss, D. T., & Knight, R. T. (Eds.). *Principles of Frontal Lobe Function* (New York: Oxford University Press, 2002).
- Taylor, L. Psychological Assessment of Neurological Patients, in T. Rasmussen & R. Marino, eds., *Functional Neurosurgery* (New York: Raven Press, 1979).
- Teuber, H.-L. (1964). The Riddle of Frontal Lobe Function in Man, in M. Warren & K. Akert, eds., *The Frontal Granular Cortex and Behavior* (New York: McGraw-Hill, 1964).
- Teuber, H.-L., & Mishkin, M. Judgment of Visual and Postural Vertical after Brain Injury, *Journal of Psychology*, 38 (1954), 161–175.
- Wilson, B. A., Alderman, N., Burgess, P. W., Emslie, H., & Evans, J. J. *The Behavioural Assessment of the Dysexecutive Syndrome* (Flempton, UK: Thames Valley Test Company, 1996).
- Yarbus, A. L. *Eye Movements and Vision* (New York: Plenum Press, 1967).

The Temporal Lobes

The temporal lobes are rather better understood than the frontal lobes, although there are mysteries yet to be revealed. However, it is clear that the temporal lobes play the major role in dealing with all aspects of auditory perception, with certain higher aspects of visual perception, and in the receptive aspects of language. In addition, they are of central importance to psychological function through their contribution to affective, emotional, and personal experience.

The temporal lobes on each side of the brain are easily identified, and each forms a separate, forward-pointing protrusion rather like the folded wing of a bird. If the anterior pole of the temporal lobe is gently drawn away from the rest of the brain, then it is possible to see the cortex running right around the inner (*mesial*) surface of the lobe, and also an underlying cortical surface facing it which is part of the *insula*. The location of the inner surface of the temporal lobe is shown in Figure 4.1(b).

The lateral surface of the lobe, the part that is normally visible, is usually divided into three horizontal strips, separated by two clearly visible fissures, as shown in Figure 4.1 (a). These strips are termed the *superior*, *middle*, and *inferior temporal gyri*. If we were to continue the sequence of strips underneath the temporal lobe and up the mesial surface behind, we should next encounter the *fusiform* and *parahippocampal gyri*, and at the superior border, the *uncus*. These mesial areas of

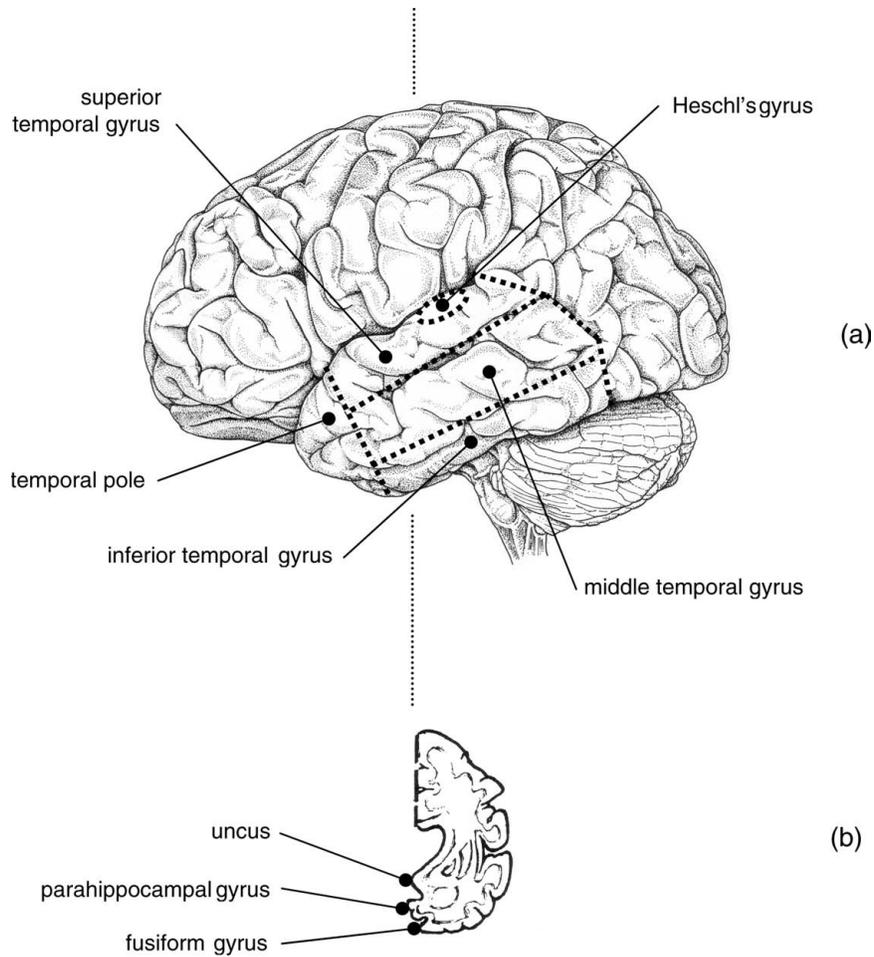


FIGURE 4.1. The principal areas of the cortex of the temporal lobe: (a) lateral view; (b) coronal section of the left temporal lobe showing medial divisions.

the temporal lobe have intimate connections with the limbic system (hence “parahippocampal”—close to the hippocampus), and because they belong to an older evolutionary system, they are sometimes referred to as *paleocortex*. The only other terminology which needs to be noted is that for the region within the superior temporal gyrus, at its superior and posterior end, and folding over into the lateral fissure, which is known as *Heschl's gyrus*.

AUDITION

Just as the frontal lobes include three levels of cortical organization for motor control, the temporal cortex includes three levels for audition.

The *primary auditory cortex*, which deals with the initial sensory reception of auditory stimulation, is located in the region of Heschl's gyrus extending into the insula. The projection of the auditory pathways up from the auditory nerve (the cranial nerve connected to the ear) contains both crossed and uncrossed pathways (see Figure 4.2); that is, stimulation received at one ear is conveyed to the auditory cortex of both temporal lobes. Within this system, however, the crossed (contralateral) pathway is dominant. It is both anatomically thicker and also preferred if competing information is being fed into the system from the left and right ears. The use of competing stimulation to study the organization of the brain is known as *dichotic listening*, which will be discussed at some

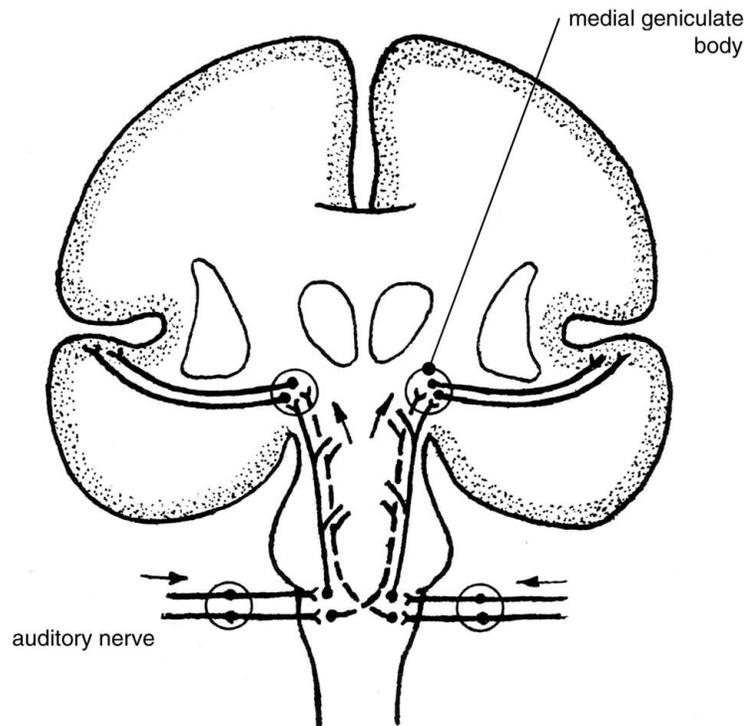


FIGURE 4.2. The auditory pathways to the temporal cortex.

length in Chapter 12. The evidence from such studies, with both clinical patients and normal subjects, points to the greater importance of the crossed pathway, although in normal hearing both pathways are almost certainly being used.

Because of the bilateral projection of auditory sensation, lesions in one of the pathways do not result in deafness (except in some specialized cases of brain stem lesions) because one of the pathways may carry information from both ears by one crossed and one uncrossed pathway. Damage to primary auditory cortex does, however, result in altered auditory detection thresholds in the contralateral ear, again illustrating the primacy of the crossed pathway. The evidence from lesion studies suggests that auditory sensation is represented in primary cortex according to the pitch of the sound, and to some extent according to the sound's location and the harmonics present.

This conclusion about the elements of auditory sensation is supported by the evidence from *stimulation* studies. Because the brain contains no sensory receptors, once the skull has been opened for surgery under general anaesthesia, it is possible to maintain the patient under local anesthetic, conscious, alert, and able to report the experiences that follow mild electrical stimulation of the cortex (see Figure 4.3). This enables the surgeon to gain some direct evidence of cortical localization, assisting decisions about the likely effects of removing a given area of tissue. The name most associated with this procedure is Penfield (see his 1975 review), and the mapping of the primary motor and somatosensory cortex (shown in Figure 2.9) was established by this method. If the primary auditory cortex is stimulated in this way, then the patient reports hearing tones of a certain pitch, perhaps seeming to come from a particular direction, and associated with certain harmonics.

The *secondary auditory cortex* is principally located in the superior temporal gyrus, including the part known as Heschl's gyrus. Stimulation studies of this region produce reports of fully formed auditory percepts; that is, patients report sounds that are meaningful to them. They may hear a very specific sound, such as part of a song, or an identifiable voice, which is associated with the memory of a specific auditory event, or they may hear more general sounds, such as a tap running, that nevertheless carry meaning for them.

At this level we find evidence for laterality in auditory processing. Lesions of the left temporal lobe, especially if toward the posterior part, may be associated with deficits in phonemic hearing, so that the patient may have difficulty in discriminating between similar phonemes such as "ba" and "pa," or "da" and "ta." This difficulty is often associated with other language problems. Lesions of the left temporal lobe are, for

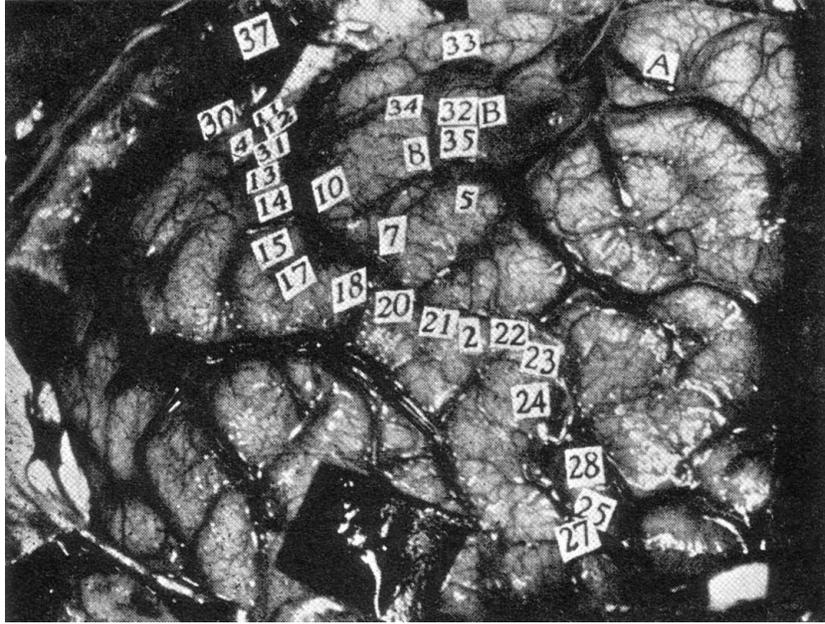


FIGURE 4.3. The exposed brain at surgery, showing the points where stimulation elicited a response. The line of tickets from 27 up to 11 follows the right Rolandic fissure from just above its junction with the lateral fissure. A tumor lies between points A and B. (From *The Cerebral Cortex of Man* by Wilder Penfield and Theodore Rasmussen, copyright 1950 by Gale, a part of Cengage Learning, Inc. Reproduced by permission. www.cengage.com/permissions.)

example, also associated with changes in the report of dichotic digits (different digits simultaneously presented to each ear). The patient's ability to report digits presented in this way may be affected, although it may be possible to report other dichotic stimuli. If, in addition, primary auditory cortex is affected, then these changes may be in the order of report of the digits, indicating changes in the normal superiority of the crossed pathways (see Chapter 12).

The right temporal lobe has, by contrast, been clearly associated with musical perception. The classic tests for musical abilities are the Seashore Tests, which involve presenting varying musical stimuli from gramophone recordings for matching or identification. Two of these tests show clear deficits resulting from right, but not left, lesions: the tests of tonal memory and timbre (which involves the perception of harmonics) (Milner, 1962). This handicap may extend to difficulty with the

location of sources of sound in space. These data clearly indicate that lateral specialization is a feature of cerebral organization at this level of auditory analysis.

It is a little difficult to separate the secondary and tertiary auditory cortices since *tertiary auditory cortex* is also situated around the superior temporal gyrus, but it is more anterior, and extends into the middle division of the temporal lobe. The deficits that follow lesions to this region are known as the *auditory agnosias*—“agnosia” means loss of knowledge—and such loss may affect a single modality, so that a bell might be recognized by sight or touch but not by its sound, or may affect several modalities together. It is generally, but not universally, accepted that this term should be reserved for nonspeech sounds and not applied to difficulties in the perception of speech stimuli, although both deficits are associated with these regions of the temporal lobe. (Speech sounds will be discussed with language functions in Chapter 7.)

Auditory (or “acoustic”) agnosia may be evident in the inability to interpret the meaning of nonspeech sounds, so that the patient can neither name the sound, nor signify anything meaningful about it. The patient cannot, for example, name the sound of running water or indicate that it might imply drinking or washing or swimming. The agnosia may alternatively be evident in lack of musical abilities, producing *amusia*, so that the patient is tone or melody “deaf,” or unable to recognize or discriminate particular rhythms, tempos, or musical measures (Critchley & Henson, 1977). There is again evidence of lateralization from studies by Vignolo (1969). He showed that there was a dissociation between performance on a Meaningful Sounds Identification Test, impaired by left temporal lesions, and a Meaningless Sounds Discrimination Test, impaired by right temporal lobe lesions. On the basis of an analysis of the errors made by auditory agnosic patients, he also proposed two types of auditory agnosia: one concerned with the perception and discrimination of sounds, and one concerned with the associations generated by sounds. All these deficits are, of course, intimately bound up with difficulties in the reception of spoken language, which will be discussed later.

Functions connected with language are discussed in Chapter 7, but it should be noted here that the temporal lobes make a major contribution to the reception and comprehension of language. In particular, systems in the superior temporal gyrus and in the region at the junction of the temporal, parietal, and occipital lobes (Wernicke’s area and the angular gyrus) supply the essential elements for understanding speech and written language. These systems are highly lateralized, to the left temporal lobe in most individuals.

VISION

Although the temporal lobes are more important for hearing than for vision, two aspects of visual function should be noted.

The first is that as the visual pathways pass back toward the occipital cortex, they run underneath the surface of the temporal lobes. Lesions of the temporal cortex, if they extend down below the cortex into the tissue below, may interrupt the visual fibers (here forming the *optic radiation*). The result is a loss of vision in the upper homonymous quadrants, that is, a loss in the upper half of vision on one side (the opposite side of vision to the side of injury), for both of the eyes. This emphasizes the importance of remembering that most lesions do not simply affect the cortex, but extend more or less radically into subcortical tissue. Lesions are three-dimensional.

The second aspect of visual function is that the *tertiary visual cortex*, located principally in the occipital lobes, extends into the middle and inferior gyri of the temporal lobes. It is more convenient to discuss these regions in the context of the complete system for vision in Chapter 6, but it should be noted that visual agnosia for objects (affecting the naming, recognition, and appropriate use of objects) or for drawings (affecting recognition) may be associated with temporal lesions. Indeed, one visual deficit, *prosopagnosia*, which means the loss of the ability to recognize faces, is produced principally by such lesions, although it is more commonly seen following lesions of the right temporal lobe. This difficulty seems to affect not the perception and recognition of the component elements in faces, but the way in which associative meaning is attached to the face, and the problem may well lie in memory components of facial recognition. Although the full form of prosopagnosia is fairly rare, it may be so extreme that the patient fails to recognize close relatives, and may even have difficulty recognizing his or her own face in a mirror.

ATTENTION

As the highest levels of analysis of both auditory and visual stimulation occur in the temporal lobes, it is not surprising that lesions of many parts of the lateral surface of the lobe can result in deficits in selective attention to visual or auditory input. That is, there is an inability to attend deliberately to one source or type of stimulation while filtering out others, or there is a generally reduced awareness of some particular form of stimulus. In the auditory modality this is shown by the changes in dichotic listening performance already mentioned. There is a parallel

finding in the visual modality, so that the ability to report stimuli presented in the lateral visual fields (see Chapter 11) may be affected. Left hemisphere lesions may affect the report of items in the right visual field, while right lesions affect the report from both visual fields. Perhaps the deficits in performance shown by the McGill Picture Anomalies Test (Figure 4.4) may be associated with these problems. In this test, fairly detailed pictures that contain some incongruous element are shown to the patient, who must spot which is the unexpected feature (Shalman, 1961). It is not clear whether the difficulty experienced by temporal lobe patients, especially those with right-sided lesions, results from a problem with the higher levels of visual perception, or from a problem with attention.

The integration of stimuli perceived in different sensory modalities is also considered a function of the temporal lobes. The primary evidence for this is clinical, coming from the abnormal experiences reported by patients with temporal lobe epilepsy (see pp. 86 and 185), where there seem to be errors in the way in which sensory data obtained in one modality are transposed into sensory experiences in another. Although the frontal lobes are concerned with controlling the overall alerting system that maintains attentional tone in the cortex (shown principally by studies of the electrical activity of the brain), the temporal lobes also contribute to the cognitive control of attention, dependent upon aspects of the stimulation that relate to higher levels of meaning. Note that spatial attention, or unilateral neglect, is related to these deficits and, being



FIGURE 4.4. The type of stimulus presented in the McGill Picture Anomalies Test.

associated with the adjacent regions of the parietal lobe, will be discussed in Chapter 5.

MEMORY

Besides its major roles in auditory perception and language comprehension, the temporal lobe is known to be involved in memory. Patients with memory dysfunction show a rather different picture depending on whether their lesion is bilateral or unilateral.

Bilateral Lesions—The Amnesic Syndrome

Bilateral temporal lobe lesions may lead to one of the most remarkable states seen by neuropsychologists. A famous example is that of HM, an intensively studied patient, who in 1953, when in his mid-20s, underwent an operation for severe epilepsy in which the mesial part of the temporal lobes, with parts of the hippocampus, amygdala, and uncus, were removed on both sides of the brain. The operation dramatically improved HM's epilepsy, but left him with severe *anterograde amnesia*—a loss of memory for all events forward in time. HM literally remembered nothing that happened after 1953, although his memory for events before that time was at least as good as one might expect. Although of normal intelligence (in fact his intelligence increased by 13 IQ points following the operation, presumably in part because of the reduction in his anticonvulsant medication), he needed continual care and protection. At his reassessment in 1967, he was able to draw a sketch plan of the bungalow in which he had been living for 8 years, but was unable to describe anything about the routine sheltered work that he had been doing regularly for the previous 6 months, or the place in which he did the work (Milner, Corkin, & Teuber, 1968). In the hospital at the time of this reassessment, he was reported to have rung repeatedly for the nurse during the night to ask where he was and why he was there. He described his experience as “like waking from a dream,” aware of events but only half able to make sense of them because they were out of the context of whatever went before.

On formal testing, HM's attention span was normal. He had the normal digit span of about seven items, and showed the normal decay of primary memory. However, in any task in which there was an interval between presentation and recall, that is, any learning task, HM failed completely. Extensive series of test trials were conducted, with no evidence of learning or memory whatsoever. This result applied to words, prose, digits, faces, shapes, or pictures. In fact early studies concluded

that there was no learning of any kind in HM. It is now clear that there were two very limited exceptions to this conclusion. Asked to learn the visually guided stylus maze (see p. 62), HM failed completely. However, with a much simplified form of the maze, in which the number of choice points was within his immediate memory span, he learned the solution in 155 trials, showed clear savings (a reduced time to relearn) after a week, and some savings even after 2 years. Second, HM showed remarkably good motor learning. An example of a motor learning test is the mirror drawing task (see Figure 4.5), in which subjects repeatedly trace the outline of a star, but are only able to visually guide a pencil by watching it in a mirror. The rate of improvement, in speed and errors, can be recorded as an index of learning. HM showed almost normal learning on this task, and also showed an effect of learning that carried over days, even though he was unable to recall having performed the task before. (For a good summary of our knowledge about HM, see Ogden & Corkin, 1991.)

It is clear from other clinical evidence that the crucial element in producing HM's dramatic amnesia was the bilateral loss of the hippocampus. However, another much-quoted case with an amnesic disorder of almost equal severity shows that it is not the hippocampus alone that is associated with the amnesic syndrome. NA was unlucky enough to be injured by a miniature fencing foil, or rapier. He swung around in his chair as his roommate was making a mock lunge at him and the foil went up his nose, passed through the base of his skull, and penetrated his brain. NA, like HM, developed a severe anterograde amnesia, with only limited retrograde amnesia (for about 2 years before the accident). NA did have some limited memories for public events following his

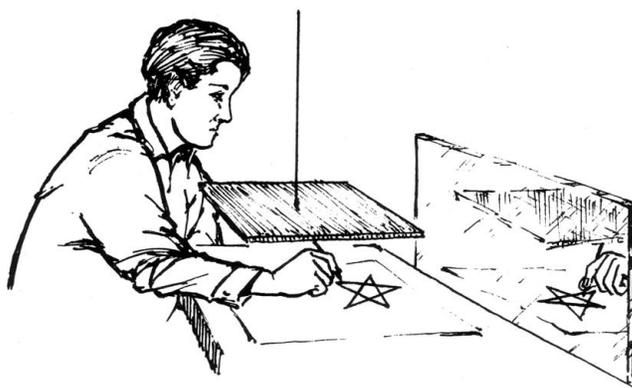


FIGURE 4.5. The mirror drawing task.

injury in 1960, and also retained some memory for spatial locations. His initial learning was normal, but he forgot more rapidly, so that within 40 minutes he would forget what he had learned.

One of the difficulties with the case of NA is that there is a dispute about exactly which parts of his brain were injured, but it is fairly certain that midline structures were affected, and not the hippocampus. Early discussions localized the damage to the frontal orbital cortex and the left side of the thalamus, besides other structures such as the corpus callosum and the neostriatum, and the lesion was therefore described as “dorso-medial.” It was then argued that this damage could not have occurred without the path of the foil traveling through the mammillary bodies. Subsequent attempts to replicate the injury on cadavers created no lesion in the mammillary bodies, but an MRI scan of NA conducted in 1989 did confirm that NA had damage to the left thalamus, part of the dorso-medial nucleus, but that his mammillary bodies were missing (and therefore atrophied through injury). A critical review of the neuropsychological history of the case of NA can be found in Parkin (2003).

Another form of the amnesic syndrome, known as *Korsakoff's psychosis* (or, increasingly, Korsakoff's disease), confirms that other structures besides the hippocampus are almost certainly of importance, including the amygdala, mammillary bodies, and anterior thalamus. Korsakoff's psychosis is commonly a consequence of chronic alcoholism, although there are other causes, including infections and a diffuse disease of the brain called Wernicke's encephalopathy, when the ensuing memory disorder is known as Wernicke-Korsakoff syndrome. Research has demonstrated some differences between the alcoholic and non-alcoholic forms, and more recent evidence emphasizes the importance of the anterior thalamus over the mammillary bodies, previously thought to be critically involved in Korsakoff's disease. Whatever the cause, these disorders are characterized by a severe anterograde amnesia. Not all the patients show an amnesia as dense as that of HM, but many are similarly handicapped. They fail to learn their way around hospital wards, can continually reread the newspaper as if they had never seen it, and have no recall for people, places, or events. It is a curious experience to spend considerable time with such a patient, to leave the room for a short period, and return to be greeted as an entirely unfamiliar person. These patients may also deny their disability and may confabulate, that is, invent details to fill the gaps created by their absent memories. Like HM, these patients show some limited savings on certain tasks. On the Gollin Incomplete Figures, where a highly incomplete drawing is shown to the patient, followed by increasingly complete versions until the drawing is recognized (Figure 4.6), normal subjects show rapid learning, identifying the item earlier and earlier in the sequence on repeated trials.



FIGURE 4.6. Examples of the Gollin Figures. (Redrawn after E. S. Gollin, *Perceptual and Motor Skills*, 11 (1960), 290.)

Amnesic patients show some very slight savings on this task (although their performance is still well below normal), as well as on a parallel task with sets of fragmented words that become successively less fragmented (Warrington & Weiskrantz, 1968).

A case of the amnesic syndrome following brain infection leading to herpes simplex encephalitis is that of CW (Wilson & Wearing, 1995). CW is interesting not only because of his profound anterograde amnesia, and his extensive retrograde amnesia, but because his previous high level of musical ability was essentially retained. CW, like HM, reported each moment as seeming to be the point of awakening, the first time of being aware (and also wrote this repeatedly in the diary he compulsively kept), and he had a profound deficit in acquiring new information. He had a severe loss of autobiographical memory although he did know that he was a musician and recognized his wife. As with many patients with Korsakoff's disease, CW confabulated. He had some loss of semantic knowledge, for example being unable to recall the differences among jam, honey, and marmalade; he was observed to eat a whole lemon, peel and all. However, previously a very talented academic musician, CW retained the ability to play fairly straightforward musical pieces from the 16th through to the 20th centuries, and had one fixed passage of improvisation, although he was unable to read large musical scores. He had

some personality change, which included euphoria as well as some aggressive dyscontrol, but this may be related to frontal lobe damage rather than his temporal lesions. The areas of the temporal lobe affected in CW were on the left side and in the medial temporal lobe on the right, but in both cases involving the hippocampus (see Figure 4.7). Enlargement of the third ventricle suggests that the thalamus was also involved, but CW had no significant damage to the mammillary bodies.

The case of CW can be contrasted with the case of SS (Cermak & O'Connor, 1983). SS also developed herpes simplex encephalitis and the amnesic syndrome. In SS there was also retrograde amnesia, but this showed a temporal gradient, so that older events from the 1930s were remembered better than more recent pre-illness events. However, SS did retain general knowledge about himself, as opposed to specific episodic memories, and did not confabulate. The lesion that developed in SS was more extensive on the left than on the right, but on both sides involved anterior and medial temporal cortex, with the amygdala, hippocampus, and other structures (putamen and lateral globus pallidus) affected. SS also had some damage to his frontal lobes, especially in the orbital region.

Patients with Korsakoff's disease have been an important source of research data, although a hotly argued debate continues over the precise explanation for the difficulties that they show. If we adopt a model of memory involving the stages of encoding, storage, and retrieval, then one possibility is that the patients encode memories normally, but are

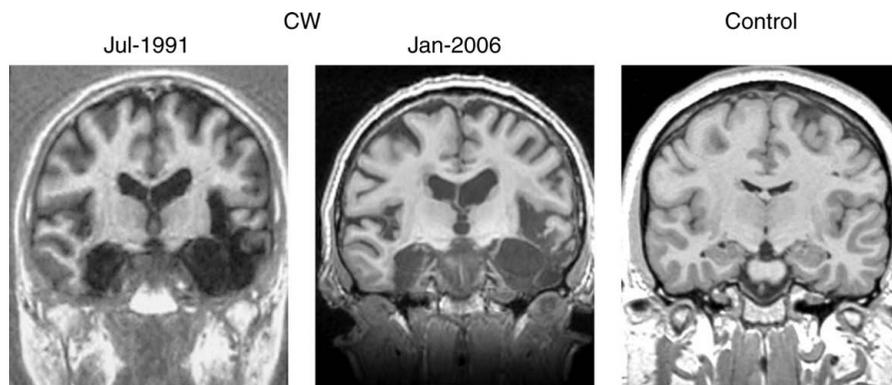


FIGURE 4.7. Images of the brain of CW (left and center) and a normal control for comparison (right). CW's lesion clearly involves the hippocampus on both sides, and is more fully described in the text. (After B. A. Wilson, M. Kopelman, and N. Kapur, *Neuropsychological Rehabilitation*, in press. Reproduced by permission.)

unable to retrieve them. Supporters of this position have argued that if one provides certain types of cue, for instance contextual information, at the time recall is demanded, the performance of these patients may be improved. The alternative view is that there is a failure of encoding, perhaps as a result of increased sensitivity to interference, so that memories are not laid down normally in the first instance. The evidence here is that deliberately aiding patients in the use of strategies that should improve encoding also improves later recall (Butters & Cermak, 1980; Mayes, 1988; Parkin & Leng, 1993). The argument is a technical one, with many of the experimental results disputed, especially as it is difficult to distinguish experimentally between, for instance, a failure of storage and a failure of retrieval; if the item is stored, but inaccessible, then how do you find out if it is stored? This debate is not entirely academic, in that the techniques that should be taught to patients to help them overcome their handicap depend upon which theory is the valid one. What is not in doubt is that the hippocampus, the associated areas of medial temporal cortex, and some other limbic structures, especially the anterior thalamus, are of central importance in the laying down and retrieval of long-term memory.

Unilateral Lesions—Temporal Lobectomy

Unilateral lesions of the hippocampus and its associated cortex may also produce effects upon memory, and these effects depend upon the side of the lesion. Lesions on the right side may, as well as affecting the learning of mazes, whether performed under visual or tactual guidance, affect memory for spatial information. This has been shown experimentally using the Corsi block tapping task (also, in a similar form, Spatial Span in the Wechsler Memory Scale, third edition—WMS III), in which the patient must learn to tap a sequence of locations on an array of irregularly arranged blocks (Figure 4.8). The number of blocks to be tapped just exceeds the span of the patient's immediate memory, but in the Corsi version the sequence of trials includes a particular sequence every third trial. Normal subjects learn this repeating sequence fairly easily, but not the patients. An associated deficit may also appear in memory for pure spatial position when patients are asked to reproduce the position of a point placed along a line. In contrast, left hippocampal lesions affect the recall of nonsense syllables (like *zot* or *pel*) or the digit span tested under conditions similar to the block tapping task.

More general unilateral temporal lesions may also have effects upon memory, although not of so dramatic a nature as in the amnesic syndrome. The most complete experimental evidence comes from studies of patients who have undergone temporal lobectomy, studies principally

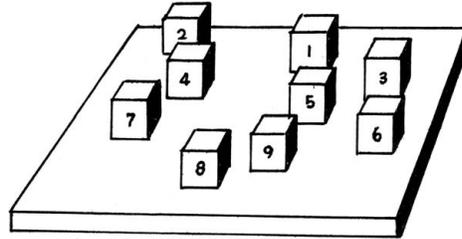


FIGURE 4.8. The Corsi block tapping task. This is the examiner's view; the subject does not see the numbers that aid the examiner to determine the sequence. (After B. Milner, *British Medical Bulletin*, 27 (1971), 275.)

carried out by Milner (1972). *Anterior temporal lobectomy* is a neurosurgical procedure carried out for certain types of epilepsy in which the epileptogenic focus is in the anterior temporal lobe, of which the surgeon removes the entire anterior portion (see p. 185). By studying these patients before and after surgery, it is possible to gain a very clear idea of the functions undertaken by this area of the brain, but it should be remembered that even before surgery these patients have abnormal brains, and we must be cautious about generalizing from them to normal individuals.

An associated procedure, that of testing by *intracarotid sodium amytal*, sometimes known as the *Wada technique*, may also be mentioned here. In most individuals verbal memory functions are associated with the left temporal lobe, and nonverbal memory functions with the right. Since deficits in verbal memory are very much more disabling in everyday life than nonverbal, a surgeon performing a temporal lobectomy operation is particularly anxious to limit the amount of tissue removed from the "verbal" side. The Wada technique provides a way of checking which is the patient's "verbal" side. Sodium amytal, which depresses the activity of nervous tissue, is injected into the internal carotid artery of one side of the head, and is carried to the anterior and central parts of the brain on that side, where it temporarily suppresses all activity in that hemisphere. The drug has no harmful effects, but the risks attached to the injection only justify the use of the technique in cases being assessed for surgery. The procedure can be carried out on the two sides on separate occasions, but when the drug is injected on the "verbal" side, the patient loses the ability to speak or understand language. We shall return to this in later chapters. For the present, it should be emphasized that although the procedure seems to be neat and well controlled the period of time during which testing is possible is very

brief, and it is carried out under adverse conditions with patients who are anxious and possibly confused or distressed, and upon brains that are not normal.

As mentioned previously, the effects of temporal lobectomy on memory are lateralized, and in most subjects left lesions affect verbal memory, while right lesions affect spatial memory; in both cases there is a delay between presentation and recall. It can be shown that information is initially encoded, because it may be reproduced in immediate recall. The evidence from focal traumatic lesions, and from the Wada test, supports these conclusions. Verbal memory, and the effect of left lesions, is usually tested by the Logical Memory subtest of the Wechsler Memory Scale. In this test the patient is read a short prose passage, rather like a brief newspaper article, and asked to reproduce it immediately following presentation. The patient is scored not for precise reproduction but for recall of the semantic elements in the passage, and it is acceptable to substitute words of similar meaning, rephrase parts of the story, or revise the order. At this stage temporal lobe patients perform normally. After an interval, usually half an hour, the subject is asked again to reproduce the passage. Scoring on this occasion is calculated as a percentage of immediate recall. Patients with left temporal lesions are handicapped at this task; postoperative scores were only about 33% of immediate recall, while right temporal lobectomy patients averaged a fairly normal 68%.

There is a corollary deficit shown by patients with right-side lesions when tested on memory for the Rey–Osterrieth Figure (see Figure 4.9). In this case the patient copies the figure (and all patients do as well as normal subjects, so they perceive the figure, have attended to it, and can draw it), and after half an hour are asked, unexpectedly, to draw it

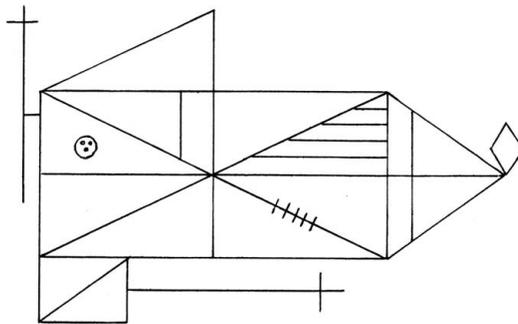


FIGURE 4.9. A figure similar to the Rey–Osterrieth figure.

again. Scoring is calculated by expressing delayed recall as a percentage of immediate copying, and normal subjects, generally to their own surprise, can reproduce the figure quite well. Right temporal lobectomy patients, however, show a deficit that is not seen in left temporal lobectomy cases.

Another facet of this memory deficit is in novelty tasks, in which the subject, after being shown a list of items, is then presented with two items, one of which has occurred in the list and one of which is novel, and is asked to indicate which is the novel item. Again there is a lateralized dissociation, with left lesions resulting in difficulty when the stimuli are words, and right lesions when the stimuli are abstract paintings.

Effects of temporal lobectomy can also be seen in *paired associate learning*, where the patient must learn a series of associated pairs of items and, on being given one member of the pair, be able to respond with the other. Patients who have undergone left temporal lobectomy show marked impairment in learning verbal material, whether the paired associates are presented in the visual or the auditory modality.

Difficulty with this task may persist for over a year after operation, although improvement may continue, albeit slowly, for up to 6 years afterward. Studies have also shown that slowing the rate of presentation may produce an improvement in the performance of this task by affected patients. This may be because it is helpful to use verbal mediators (perhaps by making up a sentence linking the two paired elements), and a slower rate of presentation provides more opportunity for these to be generated (Jones-Gotman & Milner, 1978).

Finally, although there is very clear evidence for lateralization of memory abilities, we should beware of too simple a view of their localization. Studies using the Wada technique have shown that when the injection of sodium amytal is on the same side as the lesion, recognition for *both* verbal and nonverbal material is relatively intact (although recall may be affected), and only when the injection is on the opposite side to the lesion are there substantial effects on recognition (Milner, 1975).

MEDIAL TEMPORAL STRUCTURES

It has been clear for some time that there are also a complex set of systems that support declarative memory—the memories referring to events and knowledge to which we have conscious access—and that importantly include medial temporal structures. The combination of data obtained from clinical lesion studies and from imaging investigations points to the involvement of frontal cortex and areas of the parietal cor-

tex in association with various elements of the basal ganglia in addition to the medial parts of the temporal lobe, which include the hippocampus. There are various competing theories about the precise operation of these systems, but a general acceptance of some system that incorporates these elements (Eichenbaum, 2001; Hannula and Ranganath, 2008, Mayes, Montaldi and Migo, 2007, Squire, Stark and Clark, 2004). One suggestion has been that the role of medial temporal lobes has been to bind complex memories, relying on the convergence of information in this region.

Although this is not strictly a clinical study, research by Maguire and colleagues (Maguire et al., 1998) is relevant to the role of right temporal regions in spatial awareness and navigation. It had been established for some time from studies with animals that the right hippocampus plays an important role in the mapping of spatial information, although there has been little comparative work with humans. Maguire and her colleagues undertook imaging studies (see Chapter 13) with London taxi drivers. The special quality of London taxi drivers is that they have to pass a rigorous test of their memory for the streets of London before they are granted a license; this is known as acquiring “the knowledge.” These cabbies showed increased activation of the right hippocampus when recalling routes around London, but not when recalling London landmarks or scenes from unrelated films. However, Rosenbaum, Gao, Richards, Black, and Moscovitch (2005), taking evidence from late-life dementias in cab drivers, have argued that the parahippocampal regions (around the hippocampus) are actually those of critical importance. Be that as it may, this area of research has shown that the right hippocampus is clearly associated with the acquisition and retention of spatial information in the short term, although its role in longer-term retention, or with environments from the more distant past, is less certain.

PERSONALITY

Temporal lobe epilepsy is associated with certain changes in personality and with altered states of experience. These changes do not invariably occur, and are more or less limited to this particular form of epilepsy, but the link is sufficiently clear for us to conclude that the temporal lobes are involved.

Those who suffer from epilepsy stemming from a focus in the temporal lobes, which may not produce the full-blown generalized (in older terminology “grand mal”) seizures but perhaps merely short “absences,” blackouts, or periods of automatic behavior, are often

described as having a particular sort of personality. These patients seem to talk about themselves a great deal, especially about their personal problems. They may be peculiarly pedantic in their speech, a little paranoid in their ideas, and the sort of person it is difficult to get away from in social encounters. There may be a tendency for aggressive outbursts, and it is reported that among this group of patients sudden religious conversion occurs more frequently than would be expected. Not all these traits occur in any one patient, but one or more is likely to be present.

Sufferers from temporal lobe epilepsy may also experience *illusions*, *hallucinations*, and other abnormal states, in association with their seizures. The hallucinations may be very like the complex auditory hallucinations experienced by schizophrenics, in which they hear voices conversing, but may alternatively be visual or olfactory. The illusions are usually distortions of perception so that things appear larger or smaller, or sounds louder or softer, than they should. The patient is usually aware of the distortion, which may apply to specific objects, or may in some way apply to the whole environment. Normal individuals sometimes report similar experiences when they are very tired, perhaps after periods when a lot of sleep has been lost. The illusions and hallucinations are not always pleasant, and the olfactory hallucinations in particular are usually reported as particularly offensive.

Linked with these disorders of perception are more general disorders of consciousness. The patients often report *out-of-body* experiences, or *depersonalization*, in which they seem to be able to watch themselves from "outside," often at a distance and at a height above themselves. Alternatively they may suffer *déjà vu*, in which novel places, objects, or sounds have a familiar feeling about them, or *jamais vu*, where familiar situations suddenly seem as though they are entirely novel. Again, most people have experienced the feeling that they recognize a place they cannot have visited before. Such experiences seem particularly powerful and compelling for patients suffering from temporal lobe epilepsy, although paradoxically they may at the same time be aware that their experiences are false. These phenomena lead us to regard the temporal lobe as playing some role in governing correct perception of the self located within an experiential framework.

There are also changes in sexual activity associated with temporal lobe epilepsy. As with most brain disorders the change is toward reduced sexual activity and interest, although very occasionally changes in sexual orientation, perhaps linked with an increase in activity, or disinhibition with inappropriate sexual overtures are reported. The number of such

cases is small and may not be of great significance. However, it is known that dramatic changes in sexual activity can follow extensive bilateral temporal lesions. An increase in sexual drive, in the form of *hypersexuality*, should be distinguished from the sexual disinhibition associated with the frontal lobes. Temporal lesions producing hypersexuality occur rarely in humans, but there are a number of well-documented cases, and research on monkeys provides parallel evidence. The result of such lesions is known as the *Klüver-Bucy syndrome*, in which, together with deficits in language and memory, there is grossly aberrant sexual behavior. Sexual activity increases dramatically and is quite indiscriminate in its orientation; homosexual activity is as prevalent as heterosexual activity, and sexual acts may also be directed toward inanimate objects. This occurs in a context of relatively flat emotions. In addition, there may be a tendency for patients to explore objects by putting them into their mouths, and an insatiable appetite, even for inappropriate objects. As in the case of memory, it is the association between temporal cortex and the underlying limbic structures that leads to cortical lesions having such radical effects upon behavior.

CONCLUSION

The temporal lobes provide the apparatus for the reception of auditory stimulation, its interpretation into perceptual elements, and the subsequent extraction of meaning. They also include elements for the higher-level interpretation of visual stimulation, and the syntheses that must be attained between these two sensory modalities. A specialized aspect of these perceptual systems is the reception of language stimuli.

Bilateral temporal lobe lesions may, in association with limbic structures, produce severe anterograde amnesia in which almost all capacity for long-term memory and learning is lost. Unilateral temporal lobe lesions may also affect memory for material that must be stored over significant intervals of time. The effects may be specific to the type of material (verbal or spatial), although not the modality, and while the effects may be lateralized, it is important to distinguish recall from recognition tasks.

There are also noncognitive functions that may be associated with the temporal lobes. Temporal lobe abnormality may produce personality changes, abnormal states of experience, and changes in sexual behavior. A summary of the functions associated with the temporal lobes appears in Table 4.1.

TABLE 4.1. Some Specific Functions Associated with the Temporal Lobes

| | |
|-------------------------|---|
| Audition | Reception of auditory stimulation (Heschl's gyrus) Perception of auditory stimuli (superior temporal gyrus) Cognitions relating to auditory events (anterior, superior, and middle temporal gyrus) Musical abilities (right temporal lobe) |
| Vision | Tertiary visual function (middle inferior temporal gyrus) Perception of faces (right inferior temporal gyrus) |
| Language | Reception and comprehension of speech and writing (left superior temporal gyrus and temporal–parietal–occipital junction) |
| Attention | |
| Cross-modal integration | |
| Memory | Amnesic syndrome (bilateral mesial temporal lobe) Verbal long-term memory (left temporal lobe) Spatial long-term memory (right temporal lobe) Paired associate learning (anterior temporal lobe) |
| Personality | Experiential perception (anterior temporal lobe) Sexual behavior (anterior, especially bilateral) |

FURTHER READING

The general texts for this section, given at the end of Chapter 1, provide the main source of further reading for this chapter (see, especially, Andrewes, 2001, for neuropsychological models), but these additional texts on memory may be of interest.

- Baddeley, A. D., Kopelman, M., & Wilson, B. A. *The Essential Handbook of Memory Disorders for Clinicians* (Chichester, UK: Wiley, 2002).
- Campbell, R., & Conway, M. A. (Eds.). *Broken Memories: Case Studies in Memory Impairment* (Oxford, UK: Blackwell, 1995).
- Mayes, A. R. *Human Organic Memory Disorders* (Cambridge, UK: Cambridge University Press, 1988).
- Mayes, A. R., & Downes, J. J. *Theories of Organic Amnesia: A Special Issue of Memory* (Hove, UK: Psychology Press, 1997).
- Parkin, A. J. *Case Studies in the Neuropsychology of Memory* (Hove, UK: Psychology Press, 1997).
- Squire, L. R., & Schacter, D. L. *Neuropsychology of Memory* (Hove, UK: Psychology Press, 2003).

An excellent brief overview of the neuroanatomy of structures relating to memory is to be found in:

Markowitsch, H. J. Functional Neuroanatomy of Memory and Learning, in P. W. Halligan, U. Kischka, & J. C. Marshall, eds., *Handbook of Clinical Neuropsychology* (Oxford, UK: Oxford University Press, 2003).

And two texts on the neuropsychology of sexual behavior:

Boiler, F., & Frank, E. *Sexual Dysfunction in Neurological Disorders: Diagnosis, Management and Rehabilitation* (New York: Raven Press, 1982).

LeVay, S. *The Sexual Brain* (Cambridge, MA: MIT Press, 1993).

REFERENCES

- Butters, N., & Cermak, L. C. *Alcoholic Korsakoff's Syndrome* (New York: Academic Press, 1980).
- Cermak, L. S., & O'Connor, V. The Anterograde and Retrograde Retrieval Ability of a Patient with Amnesia Due to Encephalitis (SS), *Neuropsychologia*, 21 (1983), 213–234.
- Critchley, M., & Henson, R. A. (Eds.). *Music and the Brain: Studies in the Neurology of Music* (London: Heineman Medical Books, 1977).
- Eichenbaum, H., The hippocampus and declarative memory: Cognitive mechanisms and neural codes, *Behavioural Brain Research*, 127 (2001), 199–207
- Hannula, D. E. & Ranganath, C. Medial Temporal Lobe Activity Predicts Successful Relational Memory Binding, *Journal of Neuroscience*, 28 (2008), 116–124.
- Jones-Gotman, M., & Milner, B. Right Temporal Lobe Contribution to Image-mediated Verbal Learning, *Neuropsychologia*, 16 (1978), 61–71.
- Maguire, E. A., Burgess, N., Donnett, J. G., Frackowiak, R. S. J., Frith, C. D., & O'Keefe, J. Knowing Where and Getting There: A Human Navigation Network. *Science*, 280 (1998), 921–924.
- Mayes, A. R. *Human Organic Memory Disorders* (Cambridge, UK: Cambridge University Press, 1988).
- Mayes, A. R., Montaldi, D., & Migo, E. Associative Memory and the Medial Temporal Lobes, *Trends in Cognitive Sciences*, 11 (2007), 126–135.
- Milner, B. Laterality Effects in Audition, in V. B. Mountcastle, ed., *Inter-hemispheric Relations and Cerebral Dominance* (Baltimore: Johns Hopkins Press, 1962).
- Milner, B. Disorders of Learning and Memory after Temporal Lobe Lesions in Man, *Clinical Neurosurgery*, 19 (1972), 421–446.
- Milner, B. Psychological Aspects of Focal Epilepsy and Its Neurosurgical Management, *Advances in Neurology*, 8 (1975), 299–321.
- Milner, B., Corkin, S., & Teuber, H.-L. Further Analysis of the Hippocampal Amnesic Syndrome: 14-year Follow-up of HM, *Neuropsychologia*, 6 (1968), 215–234.

- Ogden, J. A., & Corkin, S. Memories of HM, in W. C. Abraham, ed., *Memory Mechanisms: A Tribute to G. U. Goddard* (Hillsdale, NJ: Erlbaum, 1991).
- Parkin, A. J. Low-Velocity Intra-Nasal Penetrating Head Injury: Case NA, in C. Code, C.-W. Wallesch, Y. Joannette, & A. R. Lecours, eds., *Classic Cases in Neuropsychology, Volume 2* (Hove, UK: Psychology Press, 2003).
- Parkin, A. J., & Leng, N. R. C. *Neuropsychology of the Amnesic Syndrome* (Hove, UK: Erlbaum, 1993).
- Penfield, W. *The Mystery of the Mind* (Princeton, NJ: Princeton University Press, 1975).
- Rosenbaum, R. S., Gao, F., Richards, B., Black, S. E., & Moscovitch, M. "Where to?" Remote Memory for Spatial Relations and Landmark Identity in Former Taxi Drivers with Alzheimer's Disease and Encephalitis. *Journal of Cognitive Neuroscience*, 17 (2005), 446–462.
- Shalman, D. C. The Diagnostic Use of the McGill Picture Anomalies Test in Temporal Lobe Epilepsy, *Journal of Neurology, Neurosurgery and Psychiatry*, 24 (1961), 220–222.
- Squire, L. R., Stark, C. E. L., & Clark, R. E. The Medial Temporal Lobe, *Annual Review of Neuroscience*, 27 (2004), 279–306.
- Vignolo, L. A. Auditory Agnosia: A Review and Report of Recent Evidence, in A. L. Benton, ed., *Contributions to Clinical Neuropsychology* (Chicago: Aldine, 1969).
- Warrington, E. K., & Weiskrantz, L. A Study of Learning and Retention in Amnesic Patients, *Neuropsychologia*, 6 (1968), 283–292.
- Wilson, B. A., Kopelman, M. D., & Kapur, N. Prominent and Persistent Loss of Past Awareness in Amnesia: Delusion, Impaired Consciousness or Coping Strategy? *Neuropsychological Rehabilitation* (in press).
- Wilson, B. A., & Wearing, D. Prisoner of Consciousness: A State of Just Awakening Following Herpes Simplex Encephalitis, in R. Campbell & M. A. Conway, eds., *Broken Memories: Case Studies in Memory Impairment* (Oxford, UK: Blackwell, 1995).

The Parietal Lobes

Any difficulty in describing the functions of the parietal lobes comes not from the complexity of ideas about their function, but from the quite bewildering range of symptoms that may be shown by those with parietal lesions. In fact, the parietal lobes seem simply to deal with the perception of somatosensory events, and then to undertake functions with some spatial element that combine the somatosensory information with information in other modalities. There is a very broad range of functions that meet this description and that may therefore be affected by damage to the parietal lobes.

The area that we call the parietal lobe is delimited in a rather arbitrary way, and we should not pretend that its posterior boundary is exact. Some of the complex subdivisions found in textbooks have little functional significance, and for our purposes we can simply divide the parietal lobe into an anterior and posterior section, as in Figure 5.1. The anterior region, the *postcentral gyrus*, lies posterior to the Rolandic fissure and is sometimes referred to as the *sensory strip*. Behind this region and its associated secondary cortex, the posterior region is composed almost entirely of tertiary cortex. Some other landmarks, the superior and inferior parietal lobules, the angular gyrus, and the supramarginal gyrus, are sometimes referred to in research reports. Precise localization of function to these specific areas is, however, not yet possible.

We shall take the anterior region first, and its major functions of somatosensory perception, tactile perception, and body sense. In the posterior region we will consider language, spatial orientation and neglect, symbolic syntheses, apraxias, cross-modal matching, and memory.

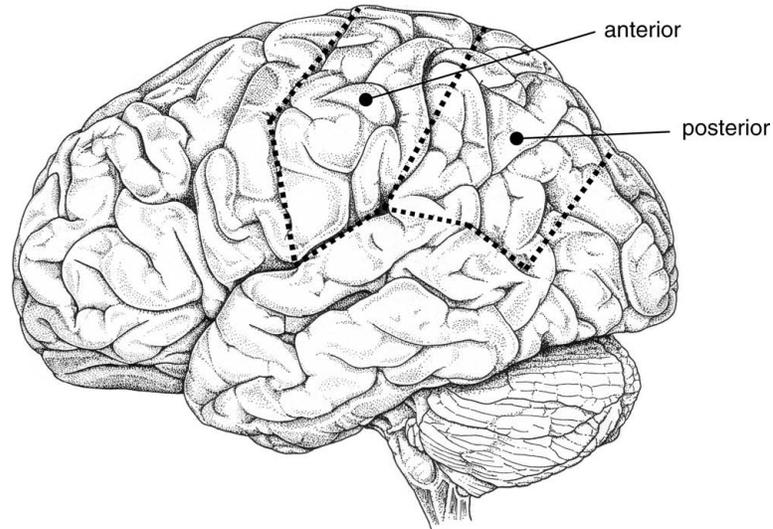


FIGURE 5.1. The principal divisions of the parietal lobe.

SOMATOSENSORY PERCEPTION

As already mentioned in Chapter 2, there is a clear point-to-point localization in the post-Rolandic sensory strip, which has been demonstrated by stimulation studies (see Figure 2.9). As with motor representation, the amount of cortex devoted to any particular body area seems to be more or less proportional to the sensory acuity of that area. Regions with very acute somatosensory perception, such as the tips of the fingers or the lips, are associated with large amounts of cortex, while insensitive areas like the back have little cortex devoted to them. The sensory acuity of a region of the skin can be tested with an instrument with two sharp points (try a pair of dividers—carefully!). Either one or two points are placed onto the skin, out of the subject's sight, and the distance between the points is varied until the smallest distance at which the number of points can be reliably discriminated is found. This is the *two-point threshold*.

The representation in this area of *primary somatosensory* cortex is mainly contralateral, although there are both contralateral and ipsilateral (uncrossed) sensory projections. In fact, there is good evidence that there are two independent sensory systems, operating by different anatomical pathways, one (the *lemniscal*) dealing with touch sensation, and the other (*extralemniscal*) dealing with temperature and pain. How-

ever, stimulation of the secondary somatosensory cortex, which is behind the primary cortex and in an area at the inferior end of the sensory strip (just above the Sylvian fissure), results in simultaneous bilateral reports of sensory perceptions.

Lesions of both the primary and secondary somatosensory cortex result in changes in normal *sensation from the body*. These changes are more the interest of the neurologist, and they include loss or alteration of sensation (anesthesia or dysesthesia) for parts of the body, which may be total or confined to certain types of sensation (touch, pressure, temperature, etc.). Not only sensation from the skin, but also kinesthetic information about the position of limbs and their movement may be affected. In turn this may lead to primary errors in spatial perception because the patient has impaired position sense and may be misinformed about the position of her or his own limbs. There may also be changes in sensory acuity, not only in terms of the two-point threshold but also in terms of the threshold for the intensity that can be perceived. The study of changes of this kind, a full account of which can be found in most textbooks on neurology, may sometimes seem a little dull to the psychologist. They are important, however, first because they can be the basis of more complex disorders seen in higher functions, and second because, in considering cognitive models of higher dysfunction, it is often important to rule out the effects of primary somatosensory loss. For instance, if we were to postulate a specific function for dressing movements (see p. 110), it would be important to show that difficulties in dressing could not be explained entirely by impaired position sense, although there is likely to be some contribution from this more basic function.

An interesting debate exists about whether there is an asymmetry in the form of representation of these functions in the left and right hemispheres. In an extensive study of soldiers with missile wounds, Semmes, Weinstein, Ghent, and Teuber (1960) found that when the lesion was in the left hemisphere, only lesions of the postcentral gyrus produced somatosensory deficits. On the other hand, lesions in the right hemisphere over a much wider area could produce such deficits. This conclusion was not supported by another major study by Corkin, Milner, and Rasmussen (1970), who found changes in position sense, intensity and two-point threshold, pressure sensitivity, and point localization to be affected only by the contralateral postcentral gyrus. Effects were seen most clearly in the hand contralateral to the lesion. The disagreement between these studies is difficult to resolve, but an important factor is the difference in the patients studied: surgical cases as against the war-injured. The size and specificity of the lesions involved, the premorbid state of the patients, and the degree of trauma undoubtedly differed between the studies and this may well account for the differing conclusions.

Because sensory feedback is important in motor control, some motor functions are also affected by lesions of primary and secondary somatosensory cortex. The loss of normal sensory feedback can result in clumsy movements, particularly of the fingers, and if the sensory face area of the left hemisphere is involved, there may be difficulty in speaking resulting from problems in articulatory control (*dysarthria*).

TACTILE PERCEPTION AND BODY SENSE

The deficits of tactile perception and body sense are known as somatosensory *agnosias* (literally a “loss of knowing”). These deficits result from damage to the secondary somatosensory cortex, and may or may not be associated with primary sensory deficits. It is convenient to divide these agnosias into those that relate to external objects, and those that relate to the patient’s own body.

Loss of the ability to recognize objects by touch is known as *astereognosis*. The patient is unable by touch alone to name objects, describe them, or demonstrate their use or significance. The term *asymbolia* is essentially equivalent. Astereognosis may occur in the presence or absence of a primary sensory deficit. When the sensory deficit is absent, it is usually the ability to recognize shape, rather than other attributes of the object, that is affected (Semmes, 1965). Apart from the difficulties that a patient may show in informal clinical testing, it may be possible to demonstrate this deficit by impaired performance on the tactile version of the Seguin-Goddard Formboard. This formboard consists of a series of simple geometrical shapes cut from a wooden sheet that must be fitted back into their appropriate holes as rapidly as possible. When required to do this blindfolded, using touch alone, patients with parietal injuries may find the task difficult or impossible.

When the somatosensory agnosia is not for external objects, but relates to the patient’s own body, then it is termed *asomatognosia*. There are two principal forms of asomatognosia: anosognosia and autotopagnosia (although, as is so often the case in clinical neurology, there are a host of other less commonly used terms).

Anosognosia is one of the most remarkable symptoms seen in neurological patients, and more frequently occurs with right-sided lesions, which therefore usually affect the left side of the body. The patient becomes unaware of the disposition of his or her limbs and, when these limbs are affected by sensory or motor losses, may deny the handicaps associated with them. In the most striking cases, where the condition may be referred to as *somatoparaphrenia*, the patient will disown his or her own limb, and cases are recorded in which patients have repeatedly

called the nursing staff to complain of the strange arm or leg that is in their bed. The affected arm or leg is quite commonly allowed to hang out of the bed without concern for its comfort or safety, exactly as if it did not belong to the patient and was none of his or her concern. Like so many neuropsychological deficits, this is a powerful reminder of the great range of functions we continually perform automatically for our own biological survival.

Conceptually similar to anosognosia is a particularly troublesome handicap, which is *asymbolia* for pain. Patients with this dysfunction fail to correctly interpret pain sensations from areas of the body, and are usually indifferent to pain. As a result, considerable damage can occur to some limb or body part because the defective perception of pain does not lead to remedial action. If linked to a more general anosognosia, then it may mean that an arm, for example, might be left on a hot stove or a hot radiator until the smell of burning flesh alerts either the patient or someone else to what is happening.

The second form of asomatognosia, more commonly seen following left-side injuries, is *autotopagnosia*. This is an impairment in the localization or naming of parts of the patient's own body. The patient is unable to point to parts of the body named by the examiner, or to move them on command, and may not be able to identify them on the examiner's body or on a diagram. That the problem may be extended to moving a body part a short time after the examiner has touched it shows that the deficit is not fundamentally related to the verbal aspects of the task.

A particular form of autotopagnosia, to which much significance is often attached, is *finger agnosia*. Although the disorder is the specific difficulty of naming and identifying the fingers, it is often tested in two particular ways. In one (the in-between test), two fingers are touched simultaneously, and the patient is asked to report the number of fingers that lie in between the two that have been touched. The other is the two-point finger test, where two touches are made upon the fingers, and the patient must decide whether they are on the same finger. However, tests for finger agnosia may also extend to naming the fingers, moving particular fingers on command, or in some other way indicating that the fingers can be identified and that their spatial arrangement is known and understood.

Demonstrating just how complex is the interrelationship of deficits of cognitive ability, astereognosis may occur in association with *visual object agnosia*, which is an impairment of visual object recognition. This may initially seem a little strange when we have so far concentrated upon the somatosensory modality in discussing the parietal lobes. However, if the difficulty in astereognosis arises from some fundamental difficulty in forming and manipulating spatial-shape representations of

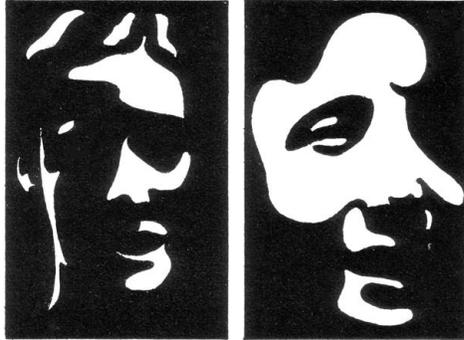


FIGURE 5.2. Mooney Closure Faces Test. (After C. M. Mooney, *Canadian Journal of Psychology*, 2 (1957), 219–226.)

objects, then the same kind of process may be required in some aspects of visual object recognition and may be performed by related structures. This view of how these associated symptoms are integrated is reinforced by the tests that are often used to demonstrate visual object agnosia: the Gollin Figures, the Mooney Closure Faces Test, and the Unconventional Views of Objects Test. The Gollin Figures were described in the last chapter (see Figure 4.6), and the Mooney Closure Faces Test is similar in that it requires that a whole percept be formed from a series of cues that do not make up a normal partial form of the figure (see Figure 5.2). The Unconventional Views of Objects Test (see Figure 5.3), devised by Warrington and Taylor (1973), consists of photographs of common objects taken from an unusual viewpoint. Again the task requires that a set of cues not normally experienced for an object be integrated and

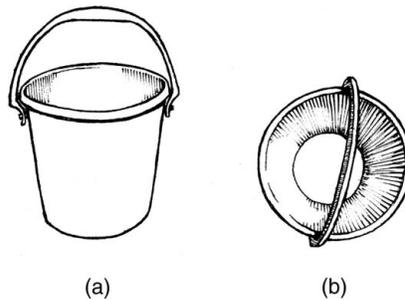


FIGURE 5.3. Unconventional Views of Objects Test: An everyday item is presented in (a) conventional and (b) unconventional representations.

turned into a representation from which meaning can be extracted. The parietal lobe patient, especially if the injury is to the right parietal lobe, may have particular difficulty with this task. Modern assessments of visual object agnosia commonly make use of the Visual Object and Space Perception Battery (VOSP; Warrington & James, 1991).

SPATIAL ORIENTATION

Underlying many of the functions of the parietal lobe is spatial representation. We have seen this in the somatosensory agnosias, and it is even more clear in *visuospatial agnosia*. This is an impairment of spatial location and orientation, either the orientation of objects with respect to the observer, or the relative orientations between objects. It is not difficult to imagine the kind of problems that a patient with this dysfunction experiences, but the tests that have been used to demonstrate visuospatial agnosia make it completely clear. A sample item (Figure 5.4) is shown from the Pool Reflections Test. In this test, the patient is shown a sample pattern and is asked to select from a multiple choice array the pattern that is a reflection of the sample rotated on its horizontal axis (Butters, Barton, & Brody, 1970). A similar test, but one that requires some con-

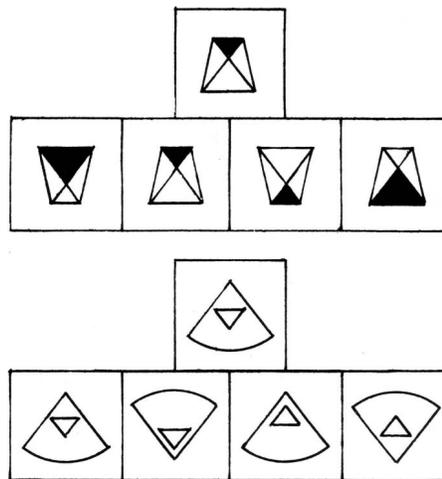


FIGURE 5.4. Pool Reflections Test. In these two sample items the subject selects from the four alternatives in the bottom row the one identical to the standard figure rotated 180° in its fronto-parallel plane (as reflected in a pool). (After Butters, Barton, and Brody, 1970.)

structional ability (see below) is the Stick Test (Butters & Barton, 1970). Here, the patient is asked to reproduce a simple pattern made from matches that the examiner has demonstrated forming. The patient must first perform the test sitting alongside and then again opposite the examiner so that some translation of the spatial elements of the construction is necessary. A more complex spatial task has also been used in which the subject sees the model of a village and is asked to judge which of a set of photographs is of the model, rejecting photographs of the same elements but in different positions.

There are aspects of memory for spatial position that are also sometimes impaired together with these functions. The task that may be set the patient is essentially the same but will involve some of the spatial information being retained in memory. The Tactual Performance Test, which forms part of the Reitan Battery, may be used to assess this aspect of spatial function. It is, in fact, an extension of the Seguin–Goddard Formboard, in which, after the blindfold test, the patient is asked, unexpectedly, to draw the shapes and their locations. The Benton Visual Retention Test (see Figure 5.5), which demands the memory retention of relatively complex, abstract, spatial figures, is an alternative way of testing impairments of this kind.

Disturbances of spatial location and orientation can also be shown on less abstract tasks, more like the situations encountered in everyday life. Just as the patient's report of familiar surroundings may be inaccurate or abnormally lacking in spatial detail, and he or she may experience particular difficulties in following a route, it is possible to see deficits on formal tests of route following ability. The most widely used form of assessment of this ability is the Locomotor Map Following Task (also known as the Semmes Maps, or Weinstein Maps; see Semmes,

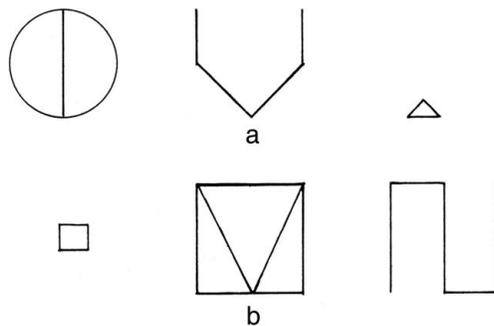


FIGURE 5.5. Two items similar to those found in the Benton Visual Retention Test.

Weinstein, Ghent, & Teuber, 1955). Nine large dots are placed upon the floor, and the patient is handed a “map” showing a simple route around these points (see Figure 5.6). The patient must follow the route by walking between the dots, but keeping the map in the same orientation to his own body throughout. Turning the map to preserve its orientation with respect to the points on the floor is not allowed, so that translation of spatial relationships between the map and the points in space is required for correct performance on the task. Difficulties on this and related tasks are associated with parietal lesions on the right side of the head.

The final aspect of this collection of spatial functions is left–right discrimination. Although many normal adults show a marked degree of left–right confusion, patients with right parietal lesions may show an even more pronounced dysfunction. It may be demonstrated by informal clinical evaluation or by more formal assessments, which might include the Money Road Map Test. This test demands that the patient decide at each turn of a route, which is marked on a schematic street plan, whether the route turns to the left or to the right with respect to its previous direction (see Figure 5.7).

We have to be careful about the aspects of lateral differentiation that are affected in a particular patient, because, as we have already noted, the left frontal lobe plays some role in orientation to egocentric space. The right parietal injuries are, by contrast, more likely to affect extrapersonal space. (If you are uncertain about the variety of abilities involved in spatial orientation, try sitting down and drawing a rough map of your daily route to work or college, or draw a plan of your home, trying to observe the mental activities in which you must engage.)

It is interesting that when normal subjects show directional confusion (you must have had the experience of being directed by someone who tells you to turn to the right, while pointing away to the left), then it is usually the verbal label that they get wrong. They rarely point in the wrong spatial direction. The parietal lobe patient will, however, show much grosser confusion about which side is which in space, and the fact that the two sides cannot be related to the lateral verbal labels stems

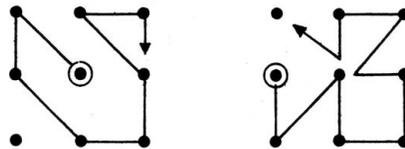


FIGURE 5.6. Two examples of the Weinstein Maps. The subject must follow the routes indicated around nine points laid out on the floor.

hemi-inattention (see Robertson & Halligan, 1999, for an excellent review of this whole topic). The disorder is, however, also seen in isolation from sensory or other deficits, which indicates that it can exist as a purely attentional disorder. The patients suffering from this impairment will often appear with bruising and scratches down one side of the body, because they constantly bump into objects and scrape along walls and hedges on the unattended side. Given tasks to perform they will neglect information that would normally appear to the one side in space, and may, characteristically, write or draw neglecting one side of the paper or the figure being copied (see Figure 5.8). They may shave only on one side of the face and may forget to complete dressing one side of the body. Most dramatic, and a common symptom, is to eat only the food on the right half of the plate; if the plate is then rotated 180 degrees the patient will resume eating and complete the meal. It is as if these patients simply do not consider one half of space to exist at all, although once their attention is explicitly drawn to that side, they can see what has been neglected and perform the necessary adjustments.

There has been considerable interest in this condition over recent years, with some surprising results. One interesting finding was that the neglect applied to imagined scenes as well as to real-life situations. Bisiach and Luzzatti (1978) asked a number of their patients familiar with the city of Milan to describe standing in the main square facing east toward the Cathedral, the Piazza del Duomo. They described the center of the square, the west façade of the Cathedral, but only the buildings on their (imagined) right side, to the south of the square. They were then asked to imagine standing on the steps of the Cathedral facing the square and again to describe what they “saw.” They repeated their description of the central area of the square and also reported the buildings ahead of them at the west end of the square; but they now described the buildings on the north side of the square (again, on their imagined right) and neglected to mention the buildings to the south, previously described.

Research has also led to a distinction between “within-object” neglect and “between-object” neglect. The issue arises as follows. If we ask the patient to copy a simple, largely symmetrical, drawing of a flower, much as a child might draw it with a stem, a leaf on each side arising from the base, and a circular flower head with radiating petals, then the patient with left neglect typically draws the stem, the right-hand leaf, and only the petals on the right side of the flower head. But, what will happen if we present two of these stylized flowers side by side? Will the patient copy only the right-hand flower, and if so, will this be complete? Or will the patient copy only the right-hand side of each flower? The answer is that either may happen; some patients do one thing, and

I
Установили у него
он болевой синдром
Зачем же
Тисонь Баренс рас
Решение
репы и чинил
не рубли
в Сангопен
H. Luria



specimen



specimen



performance



performance

FIGURE 5.8. The writing and drawing performance of a patient showing left unilateral neglect. (Reprinted with permission from A. R. Luria, *The Working Brain*, Penguin Books, 1973.)

some the other. This is explained by the hypothesis that some patients construct their drawing by reference to the independent objects, considering each separately and so drawing only the right half of each object. This is “within-object” neglect. However, some patients consider the entire array as their attentional focus and so their copy drawing, showing “between-object” neglect, is missing the left half of the page, rather than the left half of each object.

It is also now clear that patients gain some semantic information from the neglected side of space that is somehow outside of their awareness. This has been elegantly demonstrated by Marshall and Halligan (1988). What they did was to show patients with left unilateral neglect pairs of simple line drawings such a house or a glass tumbler. The right-hand halves of the pair of stimuli were identical, but in the left-hand half one of the pair had some defect. Flames issued out of the house; the rim of the tumbler was broken. Asked to detect any differences between the members of each pair, the neglect patients reported no differences, as they neglected the side containing the defect. So far, this is just what we should expect. However, asked to give their preference for one of these apparently identical stimuli, the patients chose the version without the defect: the house not on fire, or the intact drinking glass. Therefore, at some level, the patients had processed the information to the left of the stimulus, even though they were not explicitly aware of the fact.

Efforts to find an effective treatment for unilateral neglect have produced further surprises. Early efforts concentrated on ways to make the neglected half of space more salient, or to somehow attract attention to stimuli in the neglected area. This, however, proves relatively ineffective, although patients can learn to use compensatory strategies, such as very deliberately inspecting this area of space. The most effective treatment has been through contralateral limb activation; here, “contralateral” means contralateral to the side of the lesion. Regular movement of an arm or a leg on the same side of space as the neglect does reduce the degree of neglect, even when this occurs out of vision. This has been interpreted as generating some form of cerebral activation that assists in counteracting the neglect. While of practical value, this form of therapy is often limited by the fact that patients with neglect also have hemiplegia on the same side (as a result of more anterior parietal involvement), and so are unable to move the limbs as required. The treatment has been shown to work if the patient simply imagines moving the relevant limbs, but this approach has not been widely applied (McCarthy, Beaumont, Thompson, & Pringle, 2002).

Because the symptoms of neglect can in many cases spontaneously remit, the impairment has been described as passing through two phases.

The first is *allesthesia*, where attention is paid to the spatial elements on the previously neglected side, but they are treated as if they were on the unaffected side. The patient consequently completely misdirects his or her responses to the unaffected side of the body. This may be followed by the second stage, in which only *simultaneous extinction* is to be observed; that is, the patient responds normally and attends effectively to both sides of space until there is simultaneous competing stimulation of both the sides. Then neglect of one of the two stimuli will reappear and only one will be attended and responded to.

It is interesting to consider whether spatial neglect is normally a purely attentional condition, or whether spatial orientational factors, perhaps in relation to internal spatial representations, are inevitably linked with the disorder. In one case a bus driver was referred for investigation after he repeatedly drove up onto the sidewalk when drawing to a halt at a bus stop, to the consternation of those waiting to board the bus. Here, elements of both errors of spatial perception on the left of the body and attentional neglect to this side, resulting from a right parietal tumor, contributed to his alarming behavior. Defective spatial perception may be a feature of many cases, and it is important to rule out primary visual field defects. Just as with the temporal lobes, the visual projection passes behind the inferior parts of the parietal lobes, and lesions extending below the cortex can interfere with this pathway to produce loss in the lower half of vision on the contralateral side (lower quadrant homonymous hemianopia). The relation of all these factors to spatial neglect is an issue that cannot be resolved at the present time, and illustrates how complex and fascinating it can be to attempt to unravel the basis of such cognitive impairments. In most patients it is almost certainly the case that a number of factors contribute to a deficit of a high-level function like hemispatial attention.

SYMBOLIC SYNTHESSES

Symbolic (quasi-spatial) syntheses is the rather grand term sometimes given to certain difficulties that at first appear to be purely symbolic but are considered to stem from a basic spatial dysfunction. One of the clearest of these is *acalculia*, or loss of the ability to perform arithmetical calculations. It can involve such simple calculations as $2 + 2$, but it is more usual for the problem to arise with calculations that are a little more complicated. The reason that this condition may be considered to involve spatial elements is that analysis of the errors made by patients doing arithmetic indicates that many people use spatial representations

to solve such problems. It is as if they write the problem down, as they were originally taught in primary school, and then proceed to solve it by working on an internal representation of the numbers involved. The errors arise because patients muddle up the order of the digits representing a number, or fail to “carry one,” or forget that the lower item is being subtracted from the upper item within their mental representation. This is not to say that this is how all subjects tackle mental arithmetic, but many certainly do adopt this strategy. This is not the only reason for arithmetic errors—simple failure to read numbers, or neglect of one side of the problem may be others—but it is one illustration of how pervasive spatial representations are in our thinking, and how they may be disturbed by parietal lesions. A similar failure of symbolic synthesis may underlie some difficulties in reading, where regular spatial scanning of a text is important. (For a very readable account of the neuropsychological aspects of mathematics, see Butterworth, 1999.)

APRAXIA

Certain *apraxias* (the loss of intentional movements) may arise from parietal lesions. These may occur in the absence of paralysis or any other impairment of sensory or motor function. They may relate to almost any kind of purposeful movement, although gross proximal (closer to the central axis of the body) movements of the body and limbs are more commonly affected. However, imitational movements of the hands and face, tapping, and complex manual sequences do not escape impairment (Kimura, 1977). The patient is unable to organize some motor task if she or he must start from the most abstract description of that task. In other words, it may be perfectly possible to carry out some movement automatically, or in the context of everyday life, such as drinking from a cup or striking a match, but when asked to demonstrate how to drink from a cup or strike a match, the patient is quite unable to do so. Sometimes the patient may find it possible if the relevant object is present to act as a cue, but will fail if asked to perform the action without a cup or a match to act as a trigger.

Certain forms of this apraxia are given terms such as ideomotor or ideational apraxia. *Ideomotor apraxia* generally refers to a disorder of the execution of simple gestures, such as saluting or waving good-bye, either to command or by imitation. The ability to carry out more complex acts may well be retained, and it is sometimes said that “the patient knows what to do, but not how to do it.” By contrast, *ideational apraxia* is a failure to carry out a well-ordered sequence of behaviors to

perform an action, such as taking a match from a box and striking it. It is the integration of the component elements into an ordered sequence that fails; elements may be omitted, performed in incorrect order, or there may be perseveration of elements. However, the distinction between these forms is not universally accepted, and the terms are not even used consistently. It is clear that precise testing is crucial in determining exactly what deficits are seen, and the whole question awaits more thorough scientific investigation.

One theory about apraxia, promoted by Geschwind (1974) but originally derived from Liepmann, is that there is a system that links the left posterior parietal region with the left frontal lobe (through a tract called the arcuate fasciculus), in which speech mechanisms may be involved in verbal regulation (see p. 59). Motor control then proceeds by the left motor cortex for the right hand, and via the corpus callosum and the right motor cortex for the left hand. This would neatly explain the reported bilateral apraxias following left lesions, but unilateral left hand apraxia following right lesions, as well as making anatomical sense. However, these observations are not generally accepted, and the theory is the subject of continuing debate.

There are two special forms of apraxia that ought to be mentioned. The first is *dressings apraxia*, which is a particular deficit in putting on clothes. Patients may become entirely confused about the sequence of dressing, or how a particular garment should be put on, often with bizarre results. Why this can apparently occur as an isolated form has never been satisfactorily explained, but it may be that dressing is unusually difficult and that relatively mild forms of apraxia are only evident in this demanding context. Dressing is a challenging task because we are required to relate a highly plastic object, the garment, which changes its configuration so radically in external space, to our map of our own personal space, our body, and then perform appropriately adapted motor sequences to put on the garment. When thought about in this way, dressing becomes a quite remarkable achievement, and yet one we perform quite unthinkingly on a daily basis.

The second, and more important, form is *constructional apraxia*. This can be a difficult concept to grasp, but it involves the idea of an activity in which the relationships between component elements must be clearly understood so that they can be brought together and properly synthesized. It is most commonly demonstrated by drawing, or by simple constructional tasks, although it is sometimes also demonstrated by route-finding tasks of the kind already mentioned. The four tasks used in a study by Benton (1969) probably represent the core functions involved in constructional apraxia. These tasks were the Benton Visual

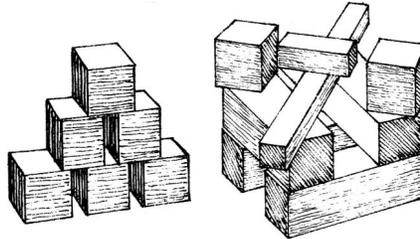


FIGURE 5.9. Three-Dimensional Constructional Praxis: A simple, and a more complex, model to be copied by the patient. (After A. L. Benton and M. L. Fogel, *Archives of Neurology*, 7 (1962), 349.)

Retention Test (see Figure 5.5), the Stick Construction Test, the Kohs block design task (Figure 3.6), and the Benton Test of Three Dimensional Constructional Praxis. In this last test, a variety of increasingly complex arrangements must be made of a collection of wooden blocks (see Figure 5.9). The aim of Benton's study was in fact to see if there was a unitary constructional apraxia that affected all four tasks. He did not find uniformity in performance across the tasks, but he did find a reasonable degree of association between the last three tests, and concluded that there may be separate syndromes that affect graphical and constructional ability. More severe constructional apraxia was more usually found to follow right lesions, and this has been supported by subsequent studies (Benson & Barton, 1970).

A final word should be added about difficulties in drawing. There are characteristic drawing errors that may be linked with the parietal lobe and are unlike those seen with occipital lesions. With parietal injury, it is the arrangement and interrelation of the elements of the drawing, rather than more basic visuoperceptual production, that are affected (see Figure 5.10). Even within parietal drawing deficits, it has been suggested that there are qualitative differences which are associated with the laterality of the lesion (Warrington, 1969). The drawings of right parietal cases are often fragmented, with component parts put in the wrong place and orientation, and with a variety of lines subsequently added in an attempt to correct the drawing. Left lesion patients, by contrast, often simplify their drawings, probably because they cannot formulate plans to execute them effectively, so that the result is a correct but laborious drawing notably lacking in detail. These qualitative differences are difficult to quantify, but make sense in terms of our overall view of the functions of the parietal lobes.

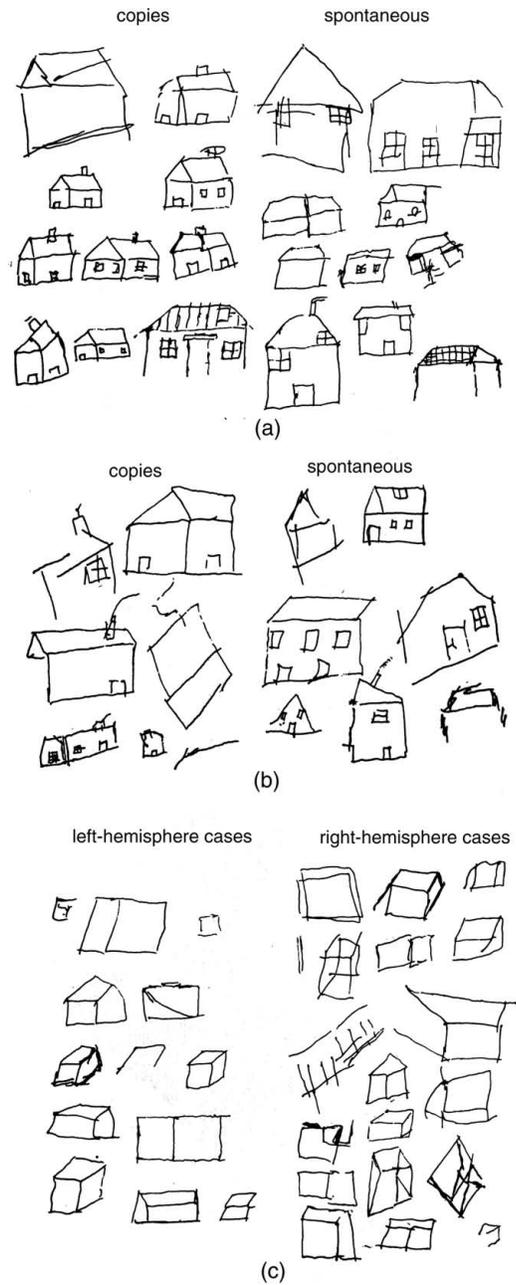


FIGURE 5.10. Drawings by patients with parietal lesions: Drawings of a house by patients with (a) left-hemisphere lesions and (b) right-hemisphere lesions, and (c) copies of a cube. (Reproduced with permission from M. Piercy, H. Hécaen, and J. De Ajuriaguerris, *Brain*, 83 (1960), 234, 236.)

INTERSENSORY ASSOCIATION

A difficulty in intersensory association, or cross-modal matching, may well contribute to certain of the deficits shown by patients with posterior parietal lesions, such as those described above. Lesions of the parieto-temporal junction in particular may result in difficulties with integration of this kind, which is really an extension of the kind of function seen in neighboring temporal zones as visual–auditory association. In the parietal lobe, the deficit is more likely to be in tactile–visual crossmodal integration. If an object is palpated by the patient, he or she may then have difficulty in selecting that object from an array of visually presented objects (Butters et al., 1970). The difficulty may, however, be shown between any or all combinations of auditory, visual, and tactile stimuli. There may be some asymmetry in that the left hemisphere deals more particularly with the “what” (identity and semantic associations), while the right deals more with the “where” (location, form, and visuospatial properties), but the division is not absolute. Undoubtedly, these difficulties may contribute to higher-level impairments, such as constructional apraxia.

SHORT-TERM MEMORY

There is little evidence for the localization of short-term memory in the cerebral cortex (providing this is distinguished from working memory, which has already been linked to the frontal lobes), except that a small number of patients have been described with difficulties in short-term auditory memory following parietal lesions (Shallice & Warrington, 1974). These patients, in particular a patient called Kenneth (KF), showed abnormally rapid forgetting of auditorily presented verbal information (spoken lists of words or numbers), and were especially susceptible to the effects of distractor items. This forgetting could not be accounted for by problems in either speech or hearing. Whether this functional impairment is really restricted in all cases to the auditory modality is in dispute, as is whether it should be considered an aspect of more general language disturbances.

GERSTMANN SYNDROME

Gerstmann syndrome must be mentioned because of the amount of attention it has attracted, although current opinion rejects it as a unitary syndrome. Gerstmann proposed that four symptoms were associated in

a single syndrome: agraphia (loss of the ability to write), acalculia, right–left confusion, and finger agnosia. He also proposed that this collection of symptoms indicated a specific lesion of the angular gyrus in the left parietal lobe. A considerable amount of investigation points to the conclusion that these four symptoms do not occur together as a unitary syndrome, although large lesions of the posterior parietal lobe may well produce all four symptoms (Benton, 1961; Heimberger, Demeyer, & Reitan, 1964). These large lesions also, however, tend to produce general language difficulties that make it difficult to decide whether the symptoms are present in “pure” form, and although patients can be found with one, two, three, or all of the symptoms, their presence is not strong evidence for specific lesions of the angular gyrus as opposed to lesions of the surrounding region.

LANGUAGE

The whole subject of language function will be discussed in Chapter 7, but it should be noted here that the more posterior areas of the parietal lobes play an important role in language function, as do the frontal and temporal lobes. Particularly, the left parietal lobe in right-handed subjects is concerned with aspects of the reception of spoken language, and also with reading.

CONCLUSION

The variety of symptoms that may be produced by parietal lesions can be confusing, especially as many are referred to by terms that do not immediately convey the nature of the impairment. The laterality of functional specialization may also seem rather more complex than in the frontal and temporal lobes, particularly if we try to account for left- as well as right-handed patients (see Critchley, 1953). Some effects are only seen with bilateral lesions, some with unilateral lesions of either side, and some with only left- or right-side lesions. In each case there is rarely a firm division, but only a relative frequency of association, perhaps because the high-level functions that are normally examined involve such a range of more basic cognitive processes.

The classic study of McFie and Zangwill (1960) is a useful example of the asymmetries that may be found. They gave a set of seven tasks to patients with parietal injuries and found only three tasks to be completely lateralized. The cube-counting and paper-cutting tasks were affected only by right-side lesions, and the test of right–left discrimina-

tion only by left-side lesions. Three further tests, of unilateral neglect, dressing ability, and topographical perception, were seen more commonly in patients with right-side injuries but were also seen in those with left lesions. The final task, the Weigl Sorting Test, showed impairment much more often with left lesions. In general, however, tasks with purely spatial elements tend to be affected by right-side lesions, but when verbal processes are involved (perhaps as a strategy for solving spatial problems), the function is more likely to be represented on the left. Some of the confusion that exists about lateralization may result from the adoption by different patients of different cognitive strategies as their preferred cognitive mode.

The specific functions associated with the parietal lobes are summarized in Table 5.1. In general the parietal lobes in the anterior regions are concerned with somatosensory sensation and perception, and are associated with a number of visual and tactile agnosias in which spatial representations are involved. In the posterior regions of the parietal lobe, there is further integration of this kind of information with information from other modalities in order to perform spatial and spatially related functions. These include certain forms of intentional movement, including complex skilled constructions, understanding and manipulation of the spatial environment with respect to both the patient's own body and the extrapersonal world, and certain tasks in which spatial representa-

TABLE 5.1. Some Specific Functions Associated with the Parietal Lobes

| |
|------------------------------------|
| Anterior |
| Somatosensory perceptions |
| Tactile perception |
| Body sense |
| Visual object recognition |
| Posterior |
| Language |
| Reception of spoken language |
| Reading |
| Spatial orientation and attention |
| Route following |
| Left-right discrimination |
| Symbolic syntheses |
| Calculation |
| Intentional movement |
| Constructional ability |
| Drawing |
| Crossmodal tactile-visual matching |
| Short-term auditory memory |

tions may be more symbolically involved, including calculation and a variety of language functions (discussed in Chapter 7). This range of spatial processes also involves the integration of somatosensory information and spatial operations with visual and auditory processes (again especially with respect to language). It is the pervasive nature of so many of these functions in our general cognitive abilities that leads to the variety and complexity of the handicaps that may follow from lesions of the parietal lobe.

FURTHER READING

The general references at the end of Chapter 1 are, again, the main texts for further study. In addition it may be worth consulting a textbook of neurology to learn more about primary somatosensory impairments. Classic reference texts of this kind include:

Bannister, R. (Ed.). *Brain and Bannister's Clinical Neurology* (Seventh edition, Oxford, UK: Oxford Medical Publications, 1992).

Another classic text, although over 50 years old, is also still of value:

Critchley, M. *The Parietal Lobes* (London: Arnold, 1953).

And more specialized texts that cover aspects of this chapter:

Butterworth, B. *The Mathematical Brain* (London: Macmillan, 1999).

De Renzi, E. *Disorders of Space Exploration and Cognition* (New York: Wiley, 1982).

Robertson, I. H., & Halligan, P. W. *Spatial Neglect: A Clinical Handbook for Diagnosis and Treatment* (Hove, UK: Psychology Press, 1999).

Spatial Neglect: A Representational Disorder, special issue, *Cortex*, 40, part 2 (2004).

REFERENCES

Benson, F. D., & Barton, M. I. Disturbances in Constructional Ability, *Cortex*, 6 (1976), 19–46.

Benton, A. L. The Fiction of the “Gerstmann Syndrome,” *Journal of Neurology, Neurosurgery and Psychiatry*, 24 (1961), 176–181.

Benton, A. L. Constructional Apraxia: Some Unanswered Questions, in A. L. Benton, ed., *Contributions to Clinical Neuropsychology* (Chicago: Aldine, 1969).

Bisiach, E., & Luzzatti, C. Unilateral Neglect of Representational Space, *Cortex*, 14 (1978), 129–133.

Butters, N., & Barton, M. I. Effect of Parietal Lobe Damage on the Performance of Reversible Operations in Space, *Neuropsychologia*, 8 (1970), 205–214.

- Butters, N., Barton, M. I., & Brody, B. A. Role of the Right Parietal Lobe in the Mediation of Cross-modal Associations and Reversible Operations in Space, *Cortex*, 6 (1970), 174–190.
- Butterworth, B. *The Mathematical Brain* (London: Macmillan, 1999).
- Corkin, S., Milner, B., & Rasmussen, T. Somatosensory Thresholds, *Archives of Neurology*, 23 (1970), 41–58.
- Critchley, M. *The Parietal Lobes* (London: Arnold, 1953).
- Geschwind, N. *Selected Papers on Language and the Brain* (Boston: D. Reidel, 1974).
- Heimberger, R. F., Demeyer, W., & Reitan, R. M. Implications of Gerstmann's Syndrome, *Journal of Neurology, Neurosurgery and Psychiatry*, 27 (1964), 52–57.
- Kimura, D. Acquisition of a Motor Skill after Left Hemisphere Damage, *Brain*, 100 (1977), 527–542.
- Marshall, J. C., & Halligan, P. W. Blindsight and Insight in Visuospatial Neglect, *Nature*, 336 (1988), 766–767.
- McCarthy, M., Beaumont, J. G., Thompson, R., & Pringle, H. The Role of Imagery in the Rehabilitation of Neglect in Severely Disabled Brain Injured Adults, *Archives of Clinical Neuropsychology*, 17 (2002), 407–442.
- McFie, J., & Zangwill, O. L. Visual Constructive Disabilities Associated with Lesions of the Left Cerebral Hemisphere, *Brain*, 83 (1960), 243–260.
- Robertson, I. H., & Halligan, P. W. *Spatial Neglect: A Clinical Handbook for Diagnosis and Treatment* (Hove, UK: Psychology Press, 1999).
- Semmes, J. A Non-tactual Factor in Astereognosis, *Neuropsychologia*, 3 (1965), 295–315.
- Semmes, J., Weinstein, S., Ghent, L., & Teuber, H.-L. Spatial Orientation in Man after Cerebral Injury. I: Analysis by Locus of Lesion, *Journal of Psychology*, 39 (1955), 227–244.
- Semmes, J., Weinstein, S., Ghent, L., & Teuber, H.-L. *Somatosensory Changes after Penetrating Brain Wounds in Man* (Cambridge, MA: Harvard University Press, 1960).
- Shallice, T., & Warrington, E. K. The Dissociation between Short-term Retention of Meaningful Sounds and Verbal Material, *Neuropsychologia*, 12 (1974), 553–555.
- Warrington, E. K. Constructional Apraxia, in P. J. Vinken & G. W. Bruyn, eds., *Handbook of Clinical Neurology*, Vol. 4 (Amsterdam: New Holland, 1969).
- Warrington, E. K., & James, M. *Visual Object and Space Perception Battery (VOSP)* (Bury St. Edmunds, UK: Thames Valley Test Company, 1991).
- Warrington, E. K., & Taylor, A. M. The Contribution of the Right Parietal Lobe to Object Recognition, *Cortex*, 9 (1973), 152–164.

The Occipital Lobes

Because the occipital lobes principally contain primary and secondary cortex subserving vision, they are almost exclusively concerned with elementary aspects of visual sensation and perception. As a result, in recent years they have seemed to be of less interest to many neuropsychologists, who have been attracted by the study of higher cortical functions. However, at least two recent developments, the study of “blindsight” and the interest in computational models of visual function, have revived interest in this region of the cortex, and we can probably expect to see a continuing increase in research into the functions of the occipital lobes.

ANATOMICAL DIVISIONS

The occipital lobes are much less clearly demarcated by anatomical landmarks than the other three pairs of lobes, but they are usually considered to comprise three regions, characterized by the type of cell contained in each. These cytoarchitectonic regions are Brodmann’s areas 17, 18, and 19, and these numbers are often used to refer to the three occipital regions. More common, perhaps, is the use of the terms *striate* (because of its striped appearance when sectioned), *parastriate*, and *peristriate*, for regions 17, 18, and 19 respectively (see Figure 6.1).

The delineation of these regions by the type of cortical cell that they contain is particularly valuable in this lobe because it allows some generalization to be made from studies of the visual cortex in animals to the visual cortex in humans. There is no good reason to suspect that fundamental mechanisms differ between the cat or the monkey and the

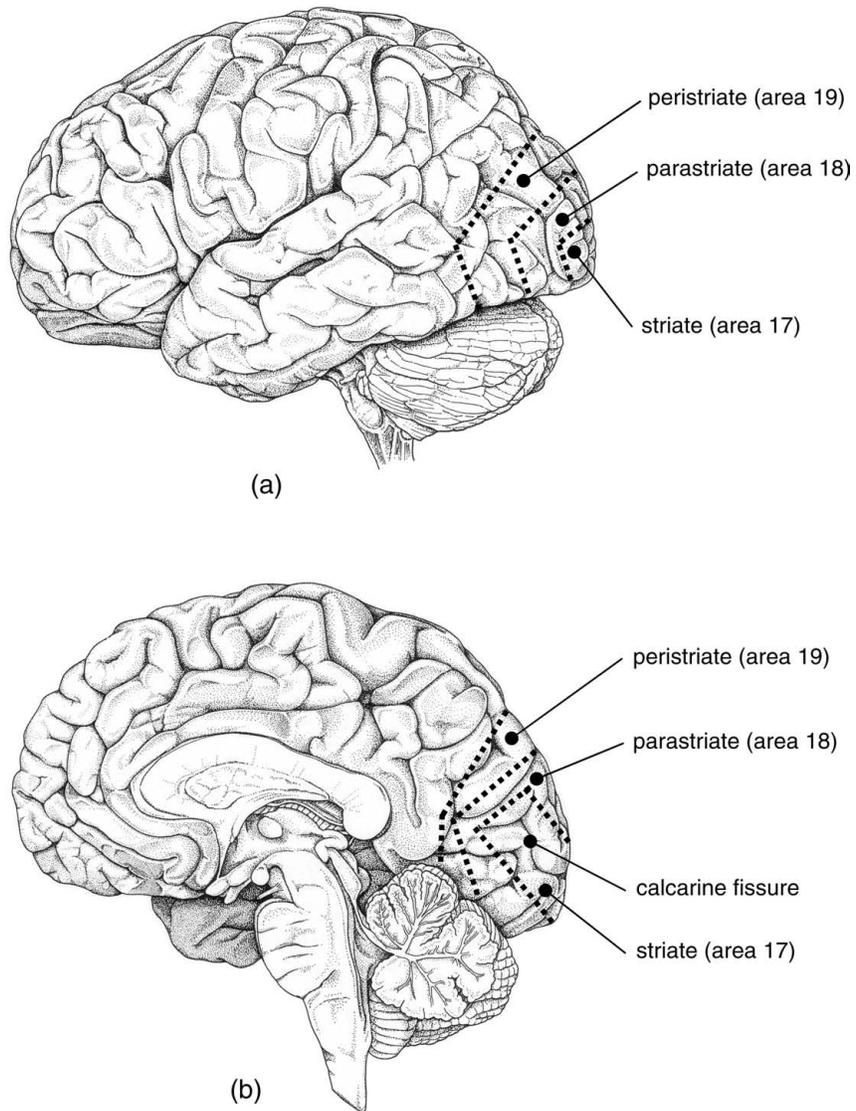


FIGURE 6.1. Anatomical divisions of the occipital lobe in (a) lateral and (b) medial views.

human, and we can therefore assume that regions made up of similar cells will perform the same elementary functions in humans as in the animals studied. A great deal of highly sophisticated physiological work has been done on the functions of the visual cortex of higher mammals, of which that of Hubel and Wiesel (1979) is a particularly successful example, and this has enabled a remarkably clear picture to be built up of the basic mechanisms involved in vision.

At each level in the visual system, there is a clear spatial mapping of the visual world. It is possible to identify cells at each level that respond to stimuli at a certain point in space, and these cells are arranged in a way that corresponds to the relationships of the points in external space, although with some minor distortions. This principle should not be extended too far, because it is not a simple representation of the visual world that is being duplicated in the brain, and within the spatial mapping of the system, a variety of forms of analysis is operating to extract different features and dimensions of the visual stimulation before the information reaches the cortex of the brain. Nevertheless, within these subsystems, which operate at various levels within the visual pathways, a certain amount of spatial information is preserved by spatial mapping of the anatomical cell arrangements.

The spatial mapping of visual stimulation extends right up to *primary visual cortex*, which is the striate region or area 17. This is principally on the medial surfaces of the hemispheres around the *occipital pole* and extending up and around the *calcarine fissure* (see Figure 6.1). Because this region is so well tucked up inside the medial surface of the hemispheres, it is relatively well protected from damage, and the most common cause of large lesions of the area is a failure of blood supply, usually as a result of *cerebral ischemia*, or a narrowing and blockage, leading to a “stroke,” of the posterior cerebral artery, which provides the entire blood supply to this area.

From the striate cortex, the visual information is passed out, by a reflection of the spatial map, to the parastriate region, area 18, which surrounds it. This forms the secondary visual cortex, and also has rich interhemispheric connections through the posterior part of the corpus callosum, known as the *splenium*. From area 18, the information may pass on to the peristriate region, area 19, as well as forward into the inferior and middle temporal gyri.

An important qualification should be made to what has been said so far about the visual system. It is now clear that there are two systems involved in mammalian vision: the *geniculostriate* and the *tectopulvinar* systems (Masterton, 1978). These systems are sometimes referred to as the *primary* and *secondary visual systems* (and the secondary as the “subcortical” visual system). What we have discussed so far is the pri-

mary geniculostriate system, which runs through the lateral geniculate nucleus of the thalamus back to the occipital cortex, and can be considered roughly to subserve the perception of forms, patterns, and color. By contrast the tectopulvinar system runs by the superior colliculus to the pulvinar and the lateral posterior regions of the thalamus before projecting out to the inferior and middle gyri of the temporal lobes. This system, which may in some ways be more primitive than the primary system, probably deals with visual location and to some extent movement, although the division of function between the two systems is not entirely clear. What is clear, as we shall see, is that this system is increasingly considered to play a significant role in human vision. For the present, however, we shall continue to discuss the primary geniculostriate system, which certainly plays the more important part in human visual perception.

BASIC VISUAL FUNCTIONS

In line with the three-level model of cortical functions we have followed so far, lesions of the primary visual cortex result in loss of visual sensation, while lesions of the secondary cortex result in perceptual impairment.

Damage to the striate cortex simply results in loss of vision. If the lesion is extensive, this may extend over the whole of the visual field, and if confined to a single hemisphere, it will affect vision in the contralateral visual field, that is, on the opposite side of space to the side of the lesion. Smaller areas of damage will result in gaps in vision known as *scotomas*, which may be surrounded by regions of partial deficit that are described as *amblyopic*. Even if the whole visual field is affected, there may be sparing of the very central, *macular*, region of vision, which leaves the patient with a small central region in which some vision is preserved. This macular sparing may come about because this region, which is normally that of highest visual acuity, has a double blood supply from the middle and posterior cerebral arteries, or because it receives bilateral projections from the left and right, visual half-field systems.

By contrast, stimulation of the primary visual cortex in patients whose visual system may be damaged but whose cortex is intact results in reports of points or flashes of light, and sometimes of simple shapes, such as triangles, squares, or circles. These images are usually small and discrete and have a specific spatial location, so that the patient can point to where they seem to appear. They can be regarded as elementary visual sensations, and when resulting from artificial stimulation are termed *photisms* or *phosphenes*. It is possible that you have experienced these

following a sharp blow to the head, particularly from behind, and “seeing stars” is the apt common phrase often used to denote such after-effects. What probably happens is that the blow results in the brain coming up rather sharply against the inside of the skull, with the result that abnormal mechanical stimulation follows, producing phosphenes. A blow to the front rather than the back of the head may have the same result, because it may lead to a kind of “whiplash” effect inside the head, so that the major contact of the brain with the skull is at the occipital pole. In severe cases, this kind of blow may lead to serious damage of the cortex opposite to the site of the blow, and it is known as a *contre-coup* injury.

An interesting and rather surprising aspect of many cases of loss of vision following occipital injury is the patient’s lack of awareness of the deficit. It is not uncommon to see patients who may have effectively lost half of their vision—all vision to one side of their central midline—and yet be apparently unaware that they have any disability. This is more likely to be the case if the loss results from a slowly developing lesion so that the loss is progressive, perhaps from the periphery inward.

This lack of awareness can be attributed to two factors. The first is that of *completion*. This is a very powerful mechanism that operates in normal vision to fill in gaps in the visual information available. It no doubt results from the systems that work so effectively to provide us with a stable, rich, and complete visual perception of the world from the series of jerky glimpses that our constantly moving and dithering eyes provide. Visual perception is essentially a reconstruction of the visual world “out there” from the information at our disposal, and completion is just one part of that process. Its clearest demonstration is in the way it fills in the normal “blind spot” in vision that results from the gap in the retina of the eye through which blood vessels and neural wiring pass. (If you have never located your own blind spot, close one eye and hold a finger up at arm’s length, facing a fairly plain background. Now fix your gaze on the background and, keeping your eyes still, move your finger out to the same side as the open eye, about six inches to the side and perhaps a little down. You should easily locate the point where the tip of the finger apparently just disappears.) We are never normally aware of the blind spot because completion compensates for the missing portion, just as it may for the scotoma (blind region) of the brain-injured patient. Completion can compensate for remarkably large areas of scotoma, and there seems no difficulty in its operation across the visual midline.

The second factor that may result in lack of awareness of the deficit is that associated with *denial* of the disability. This can sometimes be part of Anton’s syndrome, of which denial of blindness (often with unconvincing confabulation—“There’s no light in here”) is a central ele-

ment. It is probably a mistake to think of this simply as the patients trying to cover up their difficulty, especially as there are studies showing that patients with visual loss and hemiplegia may deny the loss of vision but not the loss of motor abilities. It is better to consider that the visual functions have become disconnected from the rest of the cognitive systems, so that these patients do not see the gaps in their vision because they are not seeing at all. This is the same problem, conceptually, for most of us as conceiving the experience of the congenitally blind. It may be therefore that some of these patients do not report their visual handicap because they have stopped seeing altogether in certain regions of space.

A final point about basic visual processes that cannot be overstressed is the difficulty of accurately delimiting visual field deficits (see Milner & Teuber, 1968, for an excellent discussion; and also Frisén, 1980). First, this is because the extent of vision depends crucially on the type of stimulation. Whether the stimulus is stationary or moving, neutral or brightly colored, large or small, contrasting or blending with the background, will all determine whether the patient can see it. Second, the functional state of many patients is highly unstable, and the extent of any visual disability may vary from day to day, with fatigue and perhaps with psychological state. It is therefore a grave mistake to think of visual field defects as being simple, easily delineated, and represented on a chart of the visual fields. Certainly, there will be common regions where the defect is relatively dense and constant, but around these regions there will be a great deal of variability. This raises considerable problems for research studies, where we may simply need to know “Does this patient see the stimulus material, or not?” and may be unable to obtain an entirely satisfactory answer. Whether considering research studies or individual clinical cases, descriptions of visual field defects, especially if based on simple confrontational testing or perimetry, should be treated with great caution.

VISUAL PERCEPTUAL FUNCTIONS

The job of *secondary visual cortex* is to translate the assembled visual sensations from primary cortex into meaningful percepts, and to pass this perceptual information on to the tertiary association cortex, where it can be integrated with information from other modalities and other cognitive data. Area 18 of the occipital lobe performs the secondary elaboration and synthesis of visual percepts, while area 19, in association with regions of the temporal cortex, performs intersensory integration and higher-level processing.

Lesions of the occipital secondary visual cortex therefore tend to result in deficits in simple perceptual functions. The classic work in this area was performed on brain-injured combatants of the Second World War by Teuber's group (Teuber, Battersby, & Bender, 1960), and has been little extended in recent years. Patients have difficulty in discriminating objects that differ in shape, size, orientation, or color, and in addition may be poor at bisecting lines or judging their length or orientation. This may be evident in patients' drawings (see Figure 6.2), in which the deficit is qualitatively different from that seen with parietal lesions (compare Figure 5.10). Here it is not simply the articulation of the elements that results in defective performance, but the elementary processes of judging the relationships and distances between elements, forming straight lines, angles, and curves, and relating drawing movements to the elements already put down on the paper.

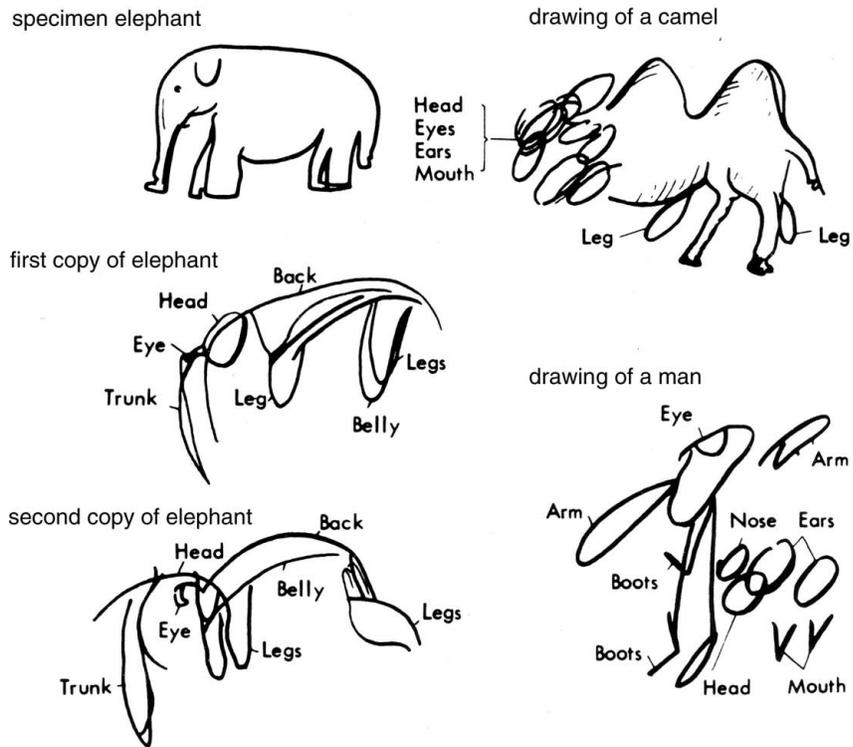


FIGURE 6.2. Copies of an elephant (the second with verbal assistance) and drawings of a camel and a man by a patient with an occipital lesion. (Reprinted with permission from A. R. Luria, *The Working Brain*, Penguin Press, 1973.)

Patients with occipital lesions may also show gross distortions of perception, seeing things as too small (micropsia), too large (macropsia), further away (porropsia), closer (pelopsia), at a great distance (teleopsia), or repeated (polyopsia). Boundaries of objects may become distorted or displaced, and there may be fluctuations in the intensity or clarity of vision. These *metamorphopsias* by which the shape of things becomes changed are difficult to imagine and clearly have radical implications for all kinds of psychological skills. In addition, visual events may curiously perseverate (palinopsia) and remain longer than they should or, even more bizarrely, be repeated after a short interval. For example, the sight of someone walking past the bed may be “replayed” over again after a brief interval, and even repeated a number of times. Some of these more dramatic symptoms are relatively rare, but their varied and extensive nature shows just how disturbing and disabling can be the malfunction of the normally powerful systems that govern visual perceptual processes. Lesions of secondary visual cortex therefore result in widespread disturbances in temporal and spatial accuracy and stability of visual perception.

Some interesting work has been carried out on the recovery of function that may follow certain vascular accidents that affect the occipital cortex (Cloning, Cloning, & Hoff, 1968). There seems to be a fairly clear functional sequence in the recovery process, with initial darkness being relieved first by photisms, and then by undifferentiated light. Primitive movement detection follows, at first without clear perception of direction or speed, followed by contours of increasing stability, and finally with the addition of color. It may be wrong to think of this as a hierarchy of visual functions, but there is some reason to believe it reflects functional levels of the visual perceptual system.

Beyond the elementary perceptual processes, there are of course higher cognitive functions, impairments of which result in *visual agnosia* (a term introduced by Sigmund Freud during his early career). Most of these, including visual object agnosia, and agnosia for drawings and facial recognition, involve regions of the temporal lobe together with the occipital lobes, and have already been discussed (see p. 78). Some other functions rely more heavily on the occipital contribution, although not without some involvement of temporal and parietal cortex (Milner et al., 1991; Sparr, Jay, Drislane, & Venna, 1991).

One group of these functions involves color perception. In *color agnosia* the patient may be able to discriminate color accurately by simple matching but may be unable to link appropriate colors to objects or to sort colors into groups. The patient may also be unable to operate symbolic color-based codes or to extract meaningful associations from colors. Whether there is also a deficit in naming colors (*color anomia*)

will depend on whether certain language systems around the temporo-parieto-occipital junction are affected (Damasio, McKee, & Damasio, 1979). Whether color anomia is an independent deficit from color agnosia is still a matter of debate. Linked to these deficits is *achromatopsia*, in which the patient perceives the world as colorless, so that it appears in shades of gray or occasionally shades of a single color (Zeki, 1990).

Visual spatial agnosias may also result from occipital lesions, particularly if they involve stereoscopic vision. As might be expected, the deficit results in a perceptual world that seems limited to two dimensions, or where sensible interpretations cannot be made of depth relationships. This no doubt arises from interference with the important interhemispheric splenial connections that link areas 18 and 19 in the two hemispheres and serve to tie together the two halves of visual space, integrating information about the two visual fields with information about images from the two eyes. Spatial agnosias that involve topographical concepts or the neglect of visual space are more likely to have significant parietal involvement.

Associative visual agnosia is sometimes distinguished as an independent entity (Albert, Reches, & Silverberg, 1975; Mack & Boller, 1977). Here the patient may copy and draw objects quite accurately, either from the original or a model drawing, but still be unable to name or indicate their proper use or associations (although the object may automatically be taken up and used as part of a habitual sequence of action). Deep lesions of the occipital lobes have been found to produce this deficit, although temporal lesions may also do so.

The final important form of visual agnosia that should be mentioned is *simultanagnosia*. This dysfunction results in the patient being unable to formulate simultaneously more than one percept. It is usually demonstrated on tasks that involve hidden or embedded figures (see Figure 6.3), of which there are a number of popularly used sets for testing. The true simultanagnosic patient will pick one percept from among the overlaid outlines of such a stimulus and then be unable to determine any others. On subsequent occasions of testing, the same single percept is likely to be elicited, with no additional responses. As a similar deficit, the ability to find figures embedded in complex perceptual displays may also be affected, although there is some dispute as to whether these problems occur in the absence of significant primary visual field defects (Bisiach, Capitani, Nichelli, & Spinnler, 1976). There is considerable difficulty in completely dissociating most of these agnosic deficits from primary perceptual disorders on the one hand, and language mediated mechanisms on the other (Spinnler, 1971).

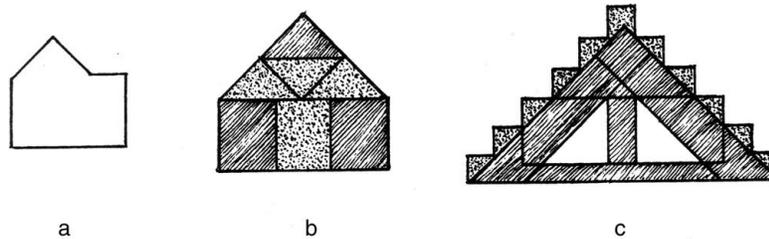


FIGURE 6.3. Embedded figures. The task is to find the form (a) hidden in figures (b) and (c).

A passing reference should also be made to a rare but significant condition known as *alexia without agraphia*, *agnosic alexia*, or *pure word blindness*. In this condition the patient cannot read although may write spontaneously and to dictation, and may speak normally. Such patients may also be unable to read their own writing once its content has been forgotten. There is some dispute about the existence of this condition in a “pure” form, but some have argued that it results from lesions of the occipital lobe and the splenium of the corpus callosum, which effectively disconnect the visual perceptual mechanisms of reading from other functional language systems (Geschwind, 1965; Benson, 1976).

BLINDSIGHT

One dramatic development that has served to enliven research into the neuropsychological functions of the occipital lobes has been the studies of “blindsight.” This phenomenon was reported in the early part of this century but then largely neglected until the last decade. It was noted that patients, although reporting themselves entirely blind, did possess some aspects of visual responsiveness. They could make certain visual judgments but without having any perceptual experience.

In the modern studies, these abilities have been found to be quite extensive, whether following damage from trauma, from stroke, or from surgical lesions introduced either early or later in life. The abilities preserved in the “blind” areas of these patients have of course varied but they include: orientation of the eyes toward the source of some flashed stimulus (Poppel, Held, & Frost, 1973), accurate pointing and reaching responses to some point source of light, differentiation of the orientation

of lines, differentiation between large Xs and Os, and an acuity (established by the discrimination of fine gratings) only a little poorer than regions of preserved normal vision (Cowey, 2004; Weiskrantz, 1986, 1999). Even more extensive stimulus identification and discrimination have been reported by Torjussen (1978). Although these patients have constantly to be encouraged to perform, because they have no conscious awareness of the stimuli and no idea of whether they are performing accurately or not, they can still show quite considerable ability.

One of the most dramatic demonstrations of this phenomenon is contained in a brief film of a monkey, Helen, with the occipital visual cortex entirely removed (Humphrey, 1995). Taken into a forest where food has been scattered on the ground, Helen moves about purposively, avoiding the trees and accurately identifying and picking up food objects while successfully rejecting similar and equally small nonfood objects. All this is achieved as a result of blindsight.

The explanation for these preserved abilities is in the operation of the secondary tectopulvinar visual system mentioned above. These functions are precisely those which from animal studies we might expect to be undertaken by such a system. It may continue to operate once the primary system has been damaged, and provide visual perception to a limited degree, but without the conscious visual experience that is a feature of the primary system.

It is hoped that these discoveries may lead to rehabilitative strategies for some of the patients who suffer cortical blindness, and although the opportunities must naturally be somewhat limited, they nevertheless provide some hope that retraining and the use of new behavioral strategies may ameliorate the handicap. It has to be said that although blindsight has been recognized for a number of years, efforts to harness it for the benefit of cortically blind patients have met with very limited success (Zihl & Kennard, 1996). This in turn provides evidence of the critical importance of conscious awareness in the control of skilled human action.

VISUAL PROSTHESES

In the first edition of this book, almost 25 years ago, I wrote about what seemed at the time to be a dramatic development, then in the initial tentative research stage, in the form of prostheses for those who are blind following damage to the eyes or to the visual tract, but whose visual cortex is intact. The idea, which is artificially to stimulate the visual cortex so as to give the appropriate sensations of vision, had been tried in at

least two facilities (Dobelle, Quest, Antunes, Roberts, & Girvin, 1979; Rushton & Brindley, 1977). In each patient, an array of tiny electrodes was placed over the visual cortex with connections out through the skull so that patterns of stimulation could be delivered directly to the cortex. The aim was to work toward some device that would perhaps incorporate a camera mounted on top of the head that would then transmit to a large array of very closely spaced stimulators over the cortex, giving a useful degree of artificial vision.

The results seemed at least encouraging. Following stimulation, these patients experienced phosphenes that were spatially relatively stable and enabled them to recognize simple patterns, including letters, transmitted to them. One patient had even been able to read “visual braille” at a rate that was faster than was possible by touch, which was a remarkable achievement.

There are bound to be limits to what can be accomplished by such methods, and the initial excitement was not followed by the development of useful prostheses. There are bioengineering problems in constructing stimulation arrays of sufficient density to form a long-term implanted prosthesis; and there are biological problems, too, in the response of the system to repeated and prolonged stimulation, and in the amount of useful cognitive information that can be derived from such a crude input without all the associated links with eye and head movement systems, general postural and orientational senses, and intersensory integration circuits. Nevertheless, the basic concept remains a valid approach, and it still seems possible that research could one day result in a practical visual prosthesis.

It is tempting to draw a parallel with the current enthusiasm for the development of neuromuscular “brain computer interfaces.” Here the logic is similar: to collect activity recorded at the motor cortex and artificially conduct instructions to muscles at the periphery, in patients with spinal lesions. There are early demonstrations of the feasibility of this approach (Santhanam, Ryu, Yu, Afshar, & Shenoy, 2006) and much excitement about the promise of this new technology. It will be interesting to see, in a further 30 years, whether the outcome has been more successful than for visual prostheses.

CONCLUSION

The occipital lobes subserve the functions of primary visual sensation and visual perception based upon these sensations. These functions are summarized in Table 6.1. They cooperate with neighboring regions in

TABLE 6.1. Some Functions of the Occipital Lobes

| |
|--|
| Primary visual sensation (points of light, simple forms) |
| Completion |
| Visual perception |
| Contours |
| Magnitude |
| Orientation |
| Depth |
| Stereopsis |
| Brightness |
| Color |
| Movement |
| Semantic connotation of visual objects |
| Reading |

the high-level cognitive functions that involve vision, for the interpretation and semantic processing of visual objects and representations. Many of the deficits associated with the occipital lobes are variable, complex, and unstable in character, and visual field defects are difficult to delineate. The contribution of primary perceptual dysfunctions, and the role of language systems in determining the presence of visual agnosias, is as yet unclear.

INTEGRATION OF CORTICAL FUNCTIONS

Now that we have looked at the functions of the four cerebral lobes, although we have yet to discuss the language system, this seems an appropriate point to introduce some comments about the complexity and integration of functional cortical systems. While in order to understand the organization of the cerebral cortex we have to try to dissociate the functions that may be linked with particular areas from other functions in other areas, and this information is in turn useful to the clinician in diagnosing lesions and planning the rehabilitation of patients, this should not lead us to lose sight of the complexity of cognitive functions in everyday life. It is easy to adopt a model of the cortex in which some area does a particular task, and then another area takes over to do something else, and so on, but the reality is clearly different. Most of the brain is involved, most of the time, in contributing to everyday intelligent behavior.

As I sit at this laptop typing the text you are reading, I am using verbal systems to generate the symbolic form of the semantic content I wish

to convey. At the same time I keep reading my notes, looking at the screen to see what has been typed, and planning how I must move my fingers to type the words, introducing little routines for capitals, new paragraphs, and so on. I have to maintain a map of where the keyboard, the screen, my notes, and my limbs are all placed relative to each other, and all the while I am listening for the telephone, trying to ignore that I feel thirsty, and carrying on a separate debate in my head about how much nicer it would be on a beach in the Maldives. Periodically I mutter things to myself, scratch my head, and notice that one of my teeth is aching a little. Not many of the functional elements mentioned in our tour of the lobes are missing from this sample of a few seconds of my behavior.

Take an even simpler sample of behavior: a tennis player about to hit the ball. A motor program is certainly being executed for that stroke, but many other operations are also being simultaneously performed. The body has to be moved to the right place on the court, and balance and control maintained. The ball is being watched, as is the opponent's position. A map is being maintained of spatial positions within the court, and constantly updated. The sound of the ball striking the racket will be attended to in order to check that the stroke was well executed, and feedback will be obtained through the arm at all stages of the stroke. At the same time future strategy is being planned, previous positions and plays are being remembered, the score is being maintained and calculations being made about points won and lost. Motivational systems are at work, feelings about the present position are being monitored, and an inner verbal dialogue (if not explicit speech) is being conducted.

This is perhaps laboring a point that is obvious. In everyday life most complex intelligent activities involve a great variety of cognitive skills and processes, all of which are interrelated and integrated within the overall operation of the brain. It is unreasonable to expect any of us to be able to grasp the actual complexity of this system, but at least in principle it is important to be aware of it, and not to allow the functional dissection that neuropsychological localization involves to blind us to how elaborate the operation of the brain actually is. No neuropsychologist can specialize in the study of only one area of the brain. The regions of the brain are not like "bolt-on parts," although the way that much of the research has been conducted might encourage you to think so. The brain, extending beyond only the cerebral cortex, and the body in which it is located *must* be seen as a whole, and considered as acting within the framework of a single comprehensive system of behavioral control.

FURTHER READING

As in the preceding chapters, the principal texts for further reading are given at the end of Chapter 1. In addition, there are an unusual range of more specialized texts concerning vision, among which the following may be of particular value:

- De Valois, K. K. (Ed.). *Seeing* (Second edition, San Diego, CA: Academic Press, 2000).
- Farah, M. J. *The Cognitive Neuroscience of Vision* (Oxford, UK: Blackwell, 2000).
- Goodale, M., & Milner, A. D. *Sight Unseen: An Exploration of Conscious and Unconscious Vision* (Oxford, UK: Oxford University Press, 2004).
- Humphreys, G. W. (Ed.). *Case Studies in the Neuropsychology of Vision* (Hove, UK: Psychology Press, 1999).
- Milner, A. D. Streams of Consciousness: Visual Awareness and the Brain, *Trends in Cognitive Sciences*, 2 (1998), 25–30.
- Zeki, S. *A Vision of the Brain* (Oxford, UK: Blackwell Scientific Publications, 1993).
- Zihl, J., & Kennard, C. Disorders of Higher Visual Functions, in T. Brandt, L. R. Caplan, J. Dichgans, H. C. Diener, & C. Kennard, eds., *Neurological Disorders, Course and Treatment* (San Diego, CA: Academic Press, 1996.)

REFERENCES

- Albert, M. L., Reches, A., & Silverberg, R. Associative Visual Agnosia without Alexia, *Neurology*, 25 (1975), 322–326.
- Benson, F. D. Alexia, in J. T. Guthrie, ed., *Aspects of Reading Acquisition* (Baltimore: Johns Hopkins University Press, 1976).
- Bisiach, E., Capitani, E., Nichelli, P., & Spinnler, H. Recognition of Overlapping Patterns and Focal Hemisphere Damage, *Neuropsychologia*, 14 (1976), 375–379.
- Cloning, I., Cloning, K., & Hoff, H. *Neuropsychological Symptoms in Lesions of the Occipital Lobe and Adjacent Areas* (Paris: Gauthier-Villars, 1968).
- Cowey, A. Fact, Artefact, and Myth about Blindsight, *Quarterly Journal of Experimental Psychology*, 57A (2004), 577–609.
- Damasio, A. R., McKee, J., & Damasio, H. Determinants of Performance in Color Anomia, *Brain and Language*, 7 (1979), 74–85.
- Dobelle, W. H., Quest, D. O., Antunes, J. L., Roberts, T. S., & Girvin, J. P. Artificial Vision for the Blind by Electrical Stimulation of the Visual Cortex, *Neurosurgery*, 5 (1979), 521–527.
- Frisén, L. The Neurology of Visual Acuity, *Brain*, 103 (1980), 639–670.
- Geschwind, N. Alexia and Color-naming Disturbance, in G. Ettliger, ed., *Functions of the Corpus Callosum* (London: Churchill, 1965).
- Hubel, D. H., & Wiesel, T. N. Brain Mechanisms of Vision, in *Scientific American: The Brain* (San Francisco: Freeman, 1979).
- Humphrey, N. Blocking Out the Distinction Between Sensation and Perception:

- Superblindsight and the Case of Helen, *Behavioural and Brain Sciences*, 18 (1995), 257–258.
- Mack, J. L., & Boller, F. Associative Visual Agnosia and Its Related Deficits: The Role of the Minor Hemisphere in Assigning Meaning to Visual Perceptions, *Neuropsychologia*, 15 (1977), 345–350.
- Masteron, R. B. (Ed.). *Handbook of Behavioural Neurology: Vol. 1, Sensory Integration* (New York: Plenum Press, 1978).
- Milner, A. D., Perrett, D. I., Johnston, R. S., Benson, P. J., Jordan, J. R., Heeley, D. W., et al. Perception and Action in Visual Form Agnosia, *Brain*, 114 (1991), 405–428.
- Milner, B., & Teuber, H.-L. Alteration of Perception and Memory in Man: Reflections on Methods, in L. Weiskrantz, ed., *Analysis of Behavioral Change* (New York: Harper and Row, 1968).
- Poppel, E., Held, R., & Frost, D. Residual Visual Function after Brain Wounds Involving the Central Visual Pathways in Man, *Nature*, 243 (1973), 295–296.
- Rushton, D. N., & Brindley, G. S. Short- and Long-Term Stability of Cortical Electrical Phosphenes, in F. C. Rose, ed., *Physiological Aspects of Clinical Neurology* (Oxford, UK: Blackwell Scientific Publications, 1977).
- Santhanam, G., Ryu, S. I., Yu, B. M., Afshar, A., & Shenoy, K. V. A High Performance Brain–Computer Interface, *Nature*, 442 (2006), 195–198.
- Sparr, S. A., Jay, M., Drislane, F. W., & Venna, N. A Historic Case of Visual Agnosia Revisited after 40 Years, *Brain*, 114 (1991), 789–800.
- Spinnler, H. Deficit in Associating Figures and Colours in Brain-Damaged Patients, *Brain Research*, 31 (1971), 370–371.
- Teuber, H.-L., Battersby, W. S., & Bender, M. B. *Visual Field Defects after Penetrating Missile Wounds of the Brain* (Cambridge, MA: Harvard University Press, 1960).
- Torjussen, T. Visual Processing in Cortically Blind Hemifields, *Neuropsychologia*, 16 (1978), 15–21.
- Weiskrantz, L. *Blindsight: A Case Study and Implications* (Oxford, UK: Oxford University Press, 1986).
- Weiskrantz, L. *Consciousness Lost and Found* (Oxford, UK: Oxford University Press, 1999).
- Zeki, S. A Century of Cerebral Achromatopsia, *Brain*, 113 (1990), 1721–1777.
- Zihl, J., & Kennard, C. Disorders of Higher Visual Functions, in T. Brandt, L. R. Caplan, J. Dichgans, H. C. Diener, & C. Kennard, eds., *Neurological Disorders: Course and Treatment* (San Diego, CA: Academic Press, 1996).

Language

The discussion of language functions has been deferred until now because, while it is reasonable to assign most functions to one of the four central lobes (although it inevitably involves some distortion by oversimplification), the language system involves sites spread across a large part of the cortex. We will now examine that system and its disorders, which are known as *aphasias*.

THE LATERALIZATION OF LANGUAGE

When lateralization of function has been mentioned in previous chapters, it has been qualified as relating to right handed subjects. The reason is that it has long been clear that individuals differ in their cerebral organization, and that one of the variables most clearly associated with this is handedness. This is a complex topic that will be treated in more detail in Chapter 14, but it is of particular relevance to studies of language.

An early piece of evidence for differing speech organization in right- and left-handed people came from the observation that right-side focal lesions rarely produce disorders of speech in the right-handed, but frequently do so in the left-handed. A number of studies have collected data on the relative frequency of aphasia in right- and left-handed patients, and there has been considerable debate over their significance. It became rapidly clear that only a few, if any, left handers have a “reversed” pattern of organization from right handers. Many have left-sided speech representation, which is the typical pattern in right handers, while others seem to have more bilateral representation. Left handers thus have a less

clear lateralization of speech, with both cerebral hemispheres contributing to the processing of language. The relative frequency of these different patterns of organization, and how they can be identified, is the subject of debate.

Evidence from a number of techniques of investigation is pertinent to this question (see Part III), but taking here only the data on the frequency of aphasia, a fairly clear conclusion has emerged from the work of Paul Satz. He reviewed the studies between 1935 and 1973 in some detail and analyzed the mathematical fit of the data to some of the models that have been proposed for left-handed speech organization. The results of these analyses (Carter, Hohenegger, & Satz, 1980; Satz, 1979) clearly support a model in which many left handers have speech in both hemispheres. The best fitting model was that in which 76% of left handers have bilateral speech representation, 25% left lateralized, and none right lateralized. For right handers the best model was: 95% left lateralized, none bilateral, and 5% right lateralized.

Another clear piece of evidence that right and left handers differ in the way in which language is organized in their brains comes from the Wada test, in which intracarotid sodium amytal is injected to depress temporarily the function of one of the two hemispheres (see p. 86), a technique developed to provide surgeons with information on speech lateralization. A report of the accumulated data from patients in Montreal who were tested by this method was published by Rasmussen and Milner (1975) and is shown in Table 7.1.

Of the patients without early left-hemisphere brain damage, the vast majority of right handers had left-sided speech representation, as did 70% of the left handers, and the remaining 30% of left handers were divided equally between right and bilateral speech. The proportions were different for those with early left-hemisphere damage, where it is

TABLE 7.1. Speech Representation in Patients of Different Handedness on the Wada Test

| | N | Speech representation (%) | | |
|---------------------------------|-----|---------------------------|-----------|-------|
| | | Left | Bilateral | Right |
| Without early left-sided damage | | | | |
| Right handers | 140 | 96 | 0 | 4 |
| Left/mixed handers | 122 | 70 | 15 | 15 |
| With early left-sided damage | | | | |
| Right handers | 31 | 81 | 6 | 13 |
| Left/mixed handers | 78 | 30 | 19 | 51 |

Note. Data from Rasmussen and Milner (1975).

believed that the young brain's plasticity allows some relocation of language function, and where a higher proportion of patients with right or bilateral speech is therefore to be expected. However, the proportions here differed quite dramatically from those inferred by Satz. This is partly accounted for by differences in the methods used to infer speech representation, partly by differences in criteria for "bilateral" representation, and partly by different ways of classifying handedness, but none of these considerations convincingly accounts for the difference. Resolution of this debate must await further evidence and further development of analytical techniques and theoretical models.

There are, of course, practical problems in finding out about the brain lateralization of individual patients. Knowing the handedness of a patient does not allow us to infer the patient's speech lateralization. If the Wada evidence is correct, then we should expect nearly everyone to have left speech representation, and if we accept Satz's analysis of the aphasia data, then we should expect most left handers to have bilateral speech representation. The fact is that neither study has allowed the construction of a satisfactory model of the relation of the lateralization of speech to handedness and associated variables. We will, however, return to models of handedness in Chapter 14. It should also be noted here that other methods of determining individual speech lateralization, based upon dichotic listening, seem to hold some promise in clinical applications (see Chapter 12).

A final point about left handers and aphasia is that left handers are considered to suffer more severely from lesions that affect speech in the initial stages of the illness, but then make more rapid and complete recovery from aphasia than do right handers. It is presumed that this is due to the relative bilateralization of their language representation, so that an undamaged hemisphere is more likely to be able to take over the functions previously performed by its damaged partner.

With this qualification about the language lateralization of left handers, we can return to consider the forms of aphasia, and will again assume the typical right handed pattern of left hemisphere speech lateralization.

VARIETIES OF APHASIA

The classification of aphasias has been one of the most hotly contested issues in the history of neuropsychology, and there is as yet no firm agreement. Not only has the terminology differed markedly from scheme to scheme, but the level of complexity of different systems has also varied greatly. Among those who have been willing to subdivide aphasias—

and there has not been complete agreement that they differ in anything other than severity—some have considered there to be only two forms, which might be termed as *receptive* and *expressive* (Weisenberg & McBride, 1935); *fluent* and *nonfluent* (Howes & Geschwind, 1964); or *anterior* and *posterior* (Benson, 1967). At the other extreme there are very detailed classifications, often associated with esoteric and unhelpful terminology, that are more commonly found in neurology textbooks. Each scheme of classification has its particular strengths and weaknesses, and useful tabulations of the various common classifications appear in Benson (1979), Kertesz (1979), and Wallesch, Johanssen-Horbach, and Blanken (2003).

The scheme used here is the one that seems to be most widely accepted at present, and is known as the Boston classification. It has developed from the work of Geschwind (1970) and Goodglass and Kaplan (1972), and while a number of variants of the classification are used, it forms perhaps the best basis for students of aphasia. This classification divides aphasias into the following six categories (with some roughly equivalent terms given in parentheses):

1. Broca's (motor, nonfluent) aphasia
2. Wernicke's (sensory) aphasia
3. Conduction (central) aphasia
4. Anomic (amnesic) aphasia
5. Transcortical motor aphasia
6. Transcortical sensory aphasia (isolation syndrome)

To these forms must be added *global* aphasia, in which there is massive and severe disturbance of language functions across a number of these categories. *Alexia* and *agraphia*, specific disorders of reading and writing respectively, are also included within the classification by some. (Note that while "a-" should imply total loss and "dys-" partial loss, these prefixes are used rather imprecisely in many of these terms.) It should also be recognized that there are other, more peripheral forms of speech pathology, which affect articulation, for example, but are not of primary interest to the neuropsychologist (see Murdoch, 1998).

The background to the Boston classification is the work of Wernicke and Lichtheim at the end of the 19th century, which resulted in a model revived and developed by Geschwind in the 1960s; hence the model is sometimes referred to as the Wernicke–Lichtheim–Geschwind model, or more commonly the Wernicke–Geschwind model. The basic idea is of a series of processes that lead from sensory input in the form of written language or speech through to output in speech or writing. Wernicke's area is the essential area for obtaining meaning from language, while

Broca's area organizes the output of language. The two are connected by the arcuate fasciculus (see the following section of this chapter for an anatomical description of these structures), although there are also other, less direct routes between the two. The angular gyrus acts to translate information between visual and auditory forms. The actual model is, of course, a little more detailed and complex, but this is the essential basis for the analysis of aphasic problems.

There are a number of reasons for accepting the Boston classification, in particular that there is some agreement that it is clinically valuable and allows sensible distinctions to be made among patients. One aspect of this is that it can be related, at least in broad terms, to lesions at particular sites on the cerebral cortex, and it therefore has some diagnostic validity. It has also proved possible to relate it to the results of various aphasia test batteries, so that the results of these tests can be expressed in terms of the classification. In fact, one of the strongest arguments in its support is that a taxonomic analysis of the results of one of these batteries (the Western Aphasia Battery) on a large group of patients produced a classification that maps very neatly onto the Boston scheme (Kertesz, 1979). This is impressive support, but it should be remembered that the battery was constructed in a milieu in which the Boston scheme was the accepted model of the aphasias, and the result is therefore a little less surprising than at first appears.

One disadvantage of the Boston scheme is that it does not formally recognize the neurolinguistic analyses of aphasias that have become of increasing importance in recent years. Rather than classifying aphasias in terms of performance on various tasks in various modalities, studies have been based upon a linguistic analysis of dysfunction, concentrating on the linguistic structures that have been affected, on distinguishing syntactic and semantic processes, and the parts of speech and characteristics of the language elements that are abnormally processed. Many neuropsychologists are relatively inexpert at linguistic analysis, and neurologists even more so, but the development of aphasiology to include linguistic parameters is logical, and may be of considerable value.

Although aphasias are described here in terms of the Boston classification, there are other valuable ways to subdivide them. One example is the scheme proposed by Luria and Hutton (1977), which is much more in the tradition of the Russian work, and another attractive system has been suggested by Brown (1976). Brown divides aphasias first into anterior and posterior disorders, and then into various levels of linguistic involvement. The anterior series progresses from complete mutism through selective mutism to agrammatism and finally anarthric aphasia (in which the difficulty is making the movements to produce speech),

thus moving from global action to more discrete facio-vocal activity and finally to speech articulation. The posterior series moves from semantic through nominal to phonemic disorders, being a progression toward increasing specificity in the selection of particular words. Each of these series reflects a structural progression from limbic transitional cortex through generalized neocortex to focal neocortex. Brown's theory is not easy to grasp when stated so starkly, but the nature of some of the functions mentioned should become clearer as the different forms of aphasia are discussed in more detail. An advantage of his scheme is that it does make explicit reference to linguistic parameters, while still being directly linked to anatomical structures. An added attraction of the scheme is that it treats the cortex as a three-dimensional structure and considers the depth of lesions in subcortical tissue. This aspect of cerebral lesions is too often ignored, and the cortex treated as if it had only two dimensions, relatively divorced from what lies beneath. Brown's scheme has yet, however, to gain wide acceptance.

ANATOMICAL STRUCTURES

Before proceeding to describe the various forms of aphasia, it may be helpful to look again at some of the anatomical locations thought to be involved in the language system (shown in Figure 7.1). Beginning anteriorly, the most important structure is *Broca's area* in the inferior posterior frontal cortex (of the left hemisphere, of course). This was the first

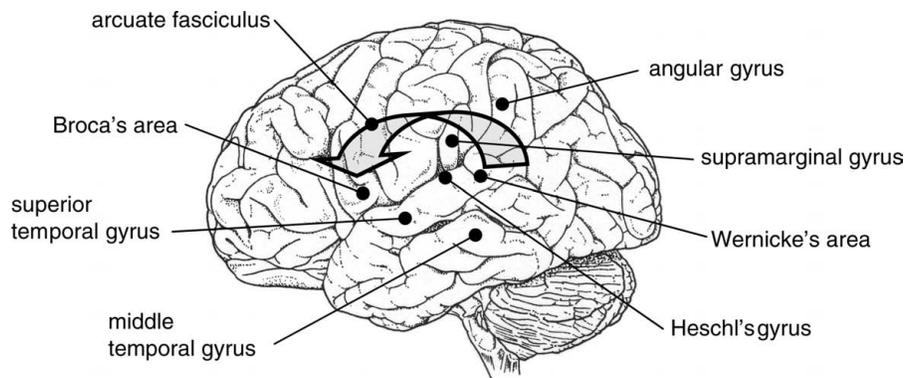


FIGURE 7.1. The principal structures in the left hemisphere associated with language functions.

location to be clearly associated with language function, in Broca's report of 1861 supported by postmortem findings (although Dax may have some claim to have established it earlier; see Critchley, 1979). Also in the frontal lobe, although separate from Broca's area, is a region of *anterior mesial cortex* that, as we shall see, is associated with transcortical motor aphasia.

Posteriorly, there is a more complex group of structures. *Wernicke's area* is in the superior middle and posterior regions of the temporal lobe, not far away from Heschl's gyrus, which is involved in auditory reception (see p. 73). Wrapped around the posterior end of the Sylvian fissure is an area of association cortex (*peri-Sylvian association cortex*), and moving posteriorly there are also the areas known as the *angular gyrus* and the *supramarginal gyrus*. The region referred to as the angular gyrus may extend rather beyond its strict topographical bounds into the middle posterior temporal region. The only other structure that we must denote is the *arcuate fasciculus*, which is an important tract of cortico-cortical (connecting two regions of cortex) fibers running approximately from the region around the posterior end of the Sylvian fissure forward to the posterior regions of the frontal lobe, and thereby serving as a direct link, so it is inferred, between the posterior and anterior language zones.

Before describing how all these different structures contribute to the language system, it should be pointed out that there is some danger of overlooking the considerable variability in the system by abstracting information from it that allows us to make sense of localization. The system should not ideally be described in such neat terms, with apparently accurate cortical localization. Figures presented by Kertesz, Lesk, and McCabe (1977) are derived from isotope scans of lesions producing various forms of aphasia and show that the lesions do no more than center on a particular region. In Figure 7.2, for example, the lesions producing anomic aphasia involve the angular gyrus and middle posterior temporal lobe, and it is clear from the figure that they may commonly involve a variety of other regions.

Not only the isotope scans, but also studies of stimulation of the exposed cortex at surgery have raised considerable doubts about the precise localization of language functions (Ojemann & Whitaker, 1978; Ojemann & Mateer, 1979). Others have more generally questioned the localization of structures involved in language, even to questioning the identification of such well-established landmarks as Wernicke's area (Bogen & Bogen, 1976; Zangwill, 1975). Care should therefore be taken to recognize both the variation among individuals and the degree of imprecision that may in reality characterize apparently precise accounts of the language system.

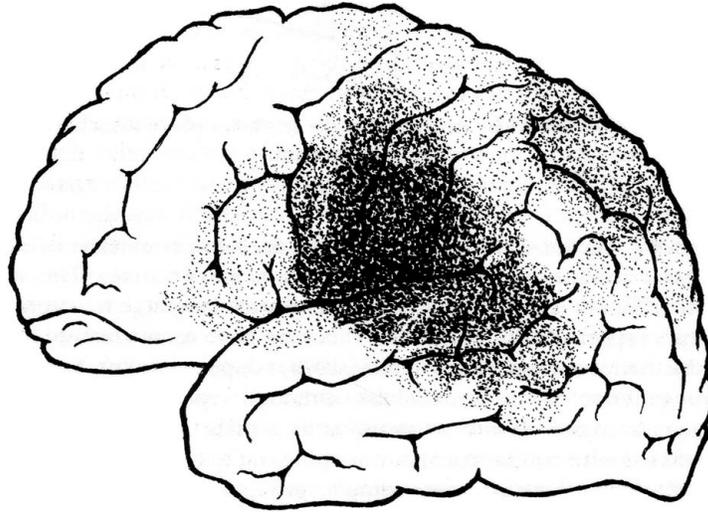


FIGURE 7.2. The regions of the brain associated with anomic aphasia. (Redrawn after Kertesz, Lesk, and McCabe, 1977.)

THE FORMS OF APHASIA

Broca's Aphasia

This type of aphasia is associated with lesions of Broca's area in the inferior posterior frontal lobe. It is the most generally recognized form of aphasia, and is the classical nonfluent form. The principal sign is that the patient's speech output is severely impaired. Most likely to be affected are articles, adverbs, adjectives, and other function words, so that speech tends to be reduced to nouns and verbs only. In the most extreme cases there is muteness, and in severe cases nouns may be restricted to their singular form, and verbs to the infinitive or a participle. This kind of speech is sometimes described as "telegraphic." The meaning is often evident, and words tend to be in the correct order, but it is as if speech has been reduced to its essential elements by agrammatic deletions. Short automatic phrases, of the kind common in "small talk," may well be inserted, and in fact used excessively and inappropriately.

There may also be errors in the actual production of words. *Paraphasias*, as these are known, are relatively common, but are of the phonemic variety where the target word is usually identifiable. Some error in the selection or production of sound elements seems to occur, but as the word produced usually approximates to the target word, and as the context may also provide some clues, it is generally possible to tell

what the patient is intending to say. There is, in addition, some difficulty with repetition and with naming, although prompting by sounds or context can be of significant help with the latter. However, it is probably best to see these particular difficulties as secondary to the problems with speech output, limiting the performance that might otherwise be attained. This is probably not a complete explanation, because written output may well be affected in the same way as speech, presumably by sharing certain output mechanisms, and the difficulty therefore seems to lie in language production generally rather than specifically with speech.

All these problems occur, however, with language comprehension intact. Patients understand perfectly what is said to them, and read as well as they comprehend speech. They often show extreme frustration at their own inability to communicate by speech, while understanding what they read and hear, although not all of them are aware of their speech limitation.

Wernicke's Aphasia

The lesion causing this dysfunction is to be found in Wernicke's area, as originally described in 1874, but, as has already been noted, there is some dispute about how precisely this area may be identified. This form of aphasia is, in contrast with Broca's, fluent and is characterized by a severe deficit in auditory comprehension. Patients may be able to tell speech from nonspeech, but they are able to extract little if any meaning from it. There is usually some related impairment in both reading and writing, parallel with auditory comprehension and speech output respectively. Naming is often also impaired to some degree, and repetition is always affected.

"Fluent" does not mean that speech is normal, and the most striking feature of patients suffering from this type of aphasia is their verbal behavior. Although they produce normal, and sometimes excessive, amounts of speech, they are generally quite unintelligible. The speech is filled by paraphasias that are in this case of a semantic nature. The patient substitutes different words for the ones that he presumably intends to utter. These may be semantically related to the target words (*red* for *green*, *table* for *chair*), but they may also be neologisms, resulting in *jargon aphasia*. This jargon is usually meaningless, as for example in this response of a patient asked what a pen was used for: "This is a tape of brouse to make buke deproed in the auria" (Kertesz & Benson, 1970). The form and often the intonation makes such speech sound like sensible English, even though it is nonsense. Again, short phrases, partic-

ularly if relatively “automatic” or well used, may be preserved. In the following example, from Kertesz (1981), a patient was asked by the examiner to describe a picture:

PATIENT: Dahnay. Enambalsay. Fack-anadee. Whynowneea. Oldeea. Eggerferma gerfriend.

EXAMINER: What’s he doing?

PATIENT: Coin’ tagowi. She’s got a rabliun. I think I wanta . . . Oh he . . .

EXAMINER: Do you know what this is over here?

PATIENT: No. Balky. I-isetinga.

One way to understand this problem in speech is to see it as caused partly by the comprehension deficit. It may be that the mechanisms for monitoring speech output overlap with those for interpreting incoming language, so that the patient loses control of his language expression by being unable to check on what is being produced. The result is often “word salad.”

Conduction Aphasia

There is less agreement about this form of aphasia than the previous two, but it is also an aphasia where repetition is disordered, and this provides the central characteristic. Patients with conduction aphasia comprehend both speech and writing normally. They will also produce more or less normal speech; it is at least reasonably fluent, the meaning is clear and appropriate, and the syntax is usually correct. Occasionally phonemic paraphasias may be introduced, but these are relatively minor and the target word is generally clear both from the context and from the form of the actual word produced. Naming may be mildly affected, but again the impairment tends to be connected with phonemic paraphasias.

However, although these patients can understand language normally, and generally produce acceptable speech, they are severely impaired if asked to repeat material that is spoken to them. The same problem may be evident if they attempt to read aloud, even though they comprehend the written material. This remarkable behavior probably arises from some disconnection between the posterior systems for language reception and the anterior systems for language production, and there are a number of models of how this might occur, although none is widely accepted.

Reflecting this disconnection, the critical lesion site producing conduction aphasia is often considered to be in deeper tissue and affecting the arcuate fasciculus, which connects anterior and posterior language centers. This makes for a neat model of how the “conduction” from reception to expression might be interrupted, but it is by no means universally accepted. Further, the role of short-term memory dysfunction in association with this aphasia has never been properly clarified.

Anomic Aphasia

This form of aphasia is generally considered to be produced by lesions of the angular gyrus, and of the middle posterior temporal lobe. It is perhaps the most common of the aphasias, and may exist as a residual form following recovery from one of the other types. Both comprehension and expression are essentially intact, and repetition is normal. There is a specific difficulty, nevertheless, with finding the names for objects (as “anomic” implies).

This specific deficit in word finding, particularly prominent for nouns, has a limited effect in speech output, although it may not be immediately apparent. There is often some blocking or hesitation in speech as a noun is being searched for, and careful testing often shows that words are substituted to avoid the problems raised by a word that cannot be found. This formally constitutes a semantic paraphasia, but as the substitutions are often appropriate and acceptable, it may go undetected in spontaneous speech. However, the kind of circumlocution that some patients employ to evade their handicap is more marked, and in severe cases the speech may be described as “empty” because the content lacks principal noun elements.

Although common, this form of aphasia is poorly understood. Word finding is clearly a complex process. To name an object, its essential characteristics must first be abstracted for identification, and then the semantically correct word retrieved and translated into a form that can be produced in speech. At precisely which stage the difficulty occurs is not clear, and indeed it may differ among cases.

Prompting is often not of great assistance to the anomic patient, and neither is providing the context in which the word occurs, nor its initial sound, nor a rhyming word. Curiously, these patients can often use a related verb, which may be the same word, to explain how they cannot find the correct noun. For example, unable to get the word “comb,” a patient might suddenly say, “I know, you use it when you want to comb your hair,” and still be unable to say that it was a comb. Perhaps the classic example is the patient who says, “No, doctor, I just can’t say ‘no!’”

Transcortical Motor Aphasia

The lesion associated with this disorder is generally to be found in the frontal association cortex, which is anterior or superior to Broca's area and assumed to be linked to it. In both transcortical motor and sensory aphasias a very curious phenomenon appears. In the most extreme form, which combines both motor and sensory elements and is relatively rare, the patient does not understand speech and cannot read. He or she is also totally nonfluent, speaks only if spoken to, and usually cannot write. Nevertheless, the patient can repeat what is heard with almost no impairment. This ability to repeat language, in the presence of an otherwise dense aphasia, has more descriptively been called *echolalic* aphasia. The only other preserved language ability reported in these patients is to complete proverbs, well-known sayings, or simple sentences. Singing, the production of automatic phrases, and swearing are also preserved, as in almost all of the aphasias.

In transcortical motor aphasia the impairment in speech output predominates. The patient therefore preserves some auditory comprehension and understands what is read, but speech is normally fluent only during repetition of what is heard, and writing is impaired along with spontaneous speech. Giving cues to assist with naming or to promote more fluent speech helps most of these patients.

Transcortical Sensory Aphasia

The lesion associated with this aphasia is usually found in peri-Sylvian association cortex, around the junction of the parietal and temporal lobes. In this type of transcortical aphasia, language reception is usually severely affected, but the ability to repeat is preserved. It is sometimes known as the *isolation syndrome*, implying that the speech cortex has been isolated from other elements of the language system, but beware because this term is not used consistently and is sometimes applied only to complete transcortical aphasia, and sometimes only to the motor form. All these aphasias are called "transcortical" because it is thought that the lesion allows transmission across the cortex of information involving language reception and production, but links with mechanisms that subserve comprehension, or links between the formulation of language and speech output, are not available.

In the sensory form of transcortical aphasia, patients can repeat what is said to them, but understand little of either what they repeat or what they read. They nevertheless produce fluent speech output, although it is often little more intelligible than that produced by the patients with Wernicke's aphasia. Because, however, the response to a

question may involve the direct repetition of elements that were in the question, some patients may produce slightly more recognizable and meaningful answers. In general, however, the speech of these patients is filled with jargon and paraphasias.

Alexia and Agraphia

Both reading and writing disorders, alexia and agraphia, may occur as relatively isolated syndromes in which there is relatively little loss in the comprehension of spoken language or in speech production, although there is often some evidence for subtle linguistic deficits in modalities other than written language. Both may be associated with lesions in the region of the angular and supramarginal gyri.

In discussing alexia or dyslexia, we must be careful to distinguish between *developmental dyslexia*, in which there is a failure to develop the ability to read, and *acquired dyslexia*, which is the loss of the ability already acquired, usually as the result of cerebral lesions. The same applies to *dysgraphia*, although developmental dysgraphia without an associated dyslexia is extremely rare. As this section is concerned with the effects of lesions on the abilities of adults, developmental dyslexia will not be discussed here, even though a great deal of research is being conducted into the disorder in a neuropsychological context. A general introduction to this work is to be found in Beaton (2004).

The study of acquired dyslexias is one area where there has been a fruitful interchange between neuropsychologists and cognitive psychologists interested in theoretical models of the process of reading. Not only has the study of clinical cases allowed some of the models to be tested, but it has also stimulated the development of improved models of how reading is normally carried out. In return, there is now a better understanding of the deficits shown by clinical patients, with the prospect that this may result in more effective approaches to rehabilitation. Patterson's 1981 review, which incidentally sets the dysfunction firmly in the context of theories of normal reading, provides a clear illustration of this exchange. She classified the acquired dyslexias into four forms: deep dyslexia, phonological dyslexia, letter-by-letter reading, and surface dyslexia (see also Hanley & Kay, 2003).

Before presenting these four forms, it is perhaps helpful to refer to current models of normal reading. A highly simplified form appears as Figure 7.3. More detailed models are to be found in Ellis and Young (1996), Coltheart, Rastle, Perry, Langdon, and Ziegler (2001), and Behrmann and Patterson (2004).

In understanding this model we must remember that, as young children, we initially learn to recognize a limited vocabulary of simple

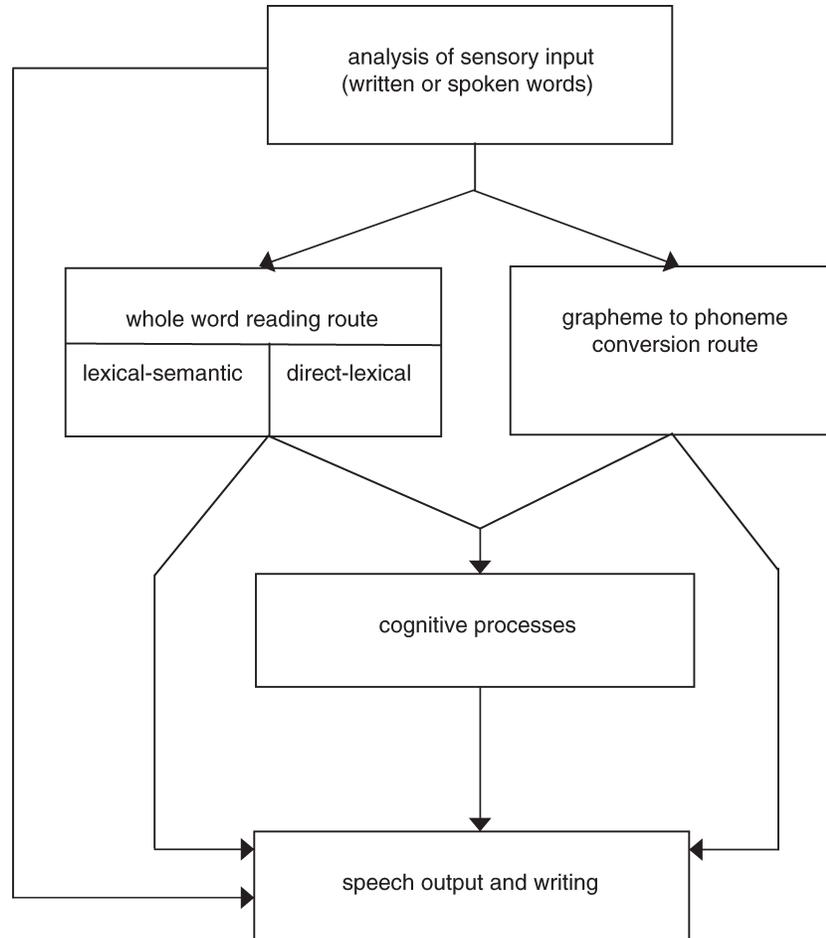


FIGURE 7.3. A simplified model of the cognitive processes involved in reading.

words, but that we then progress to abstract the rules by which words are pronounced: the grapheme–phoneme rules that govern the reading of most words. Graphemes are the elements of written words, and phonemes the elements of spoken words. However, in some languages (especially English, but much less so in Italian, Spanish, or Welsh) there are a large number of “irregular” or “exception” words (*salmon* is not pronounced “sal-mon”; *steak* is not like “streak” with the *r* omitted, which would be “steek”), the correct pronunciation of which simply has to be learned. If you doubt the complexity of English, consider the variety of ways in which the ending “-ough” is pronounced in *rough*, *though*,

slough (two forms), *cough*, and *thorough*. However, novel words or nonwords (collections of letters with no meaning in the language) have to be read by grapheme to phoneme conversion.

However, although we naturally use this grapheme–phoneme route as adult readers, the evidence is also clear that skilled readers do not need to go through this process for the majority of words, but simply recognize familiar whole words and know their meaning. This process involves a “logogen” that is simply like a large internal dictionary by which words can be identified and their meaning and pronunciation known. It is, in fact, necessary to hypothesize two versions of the whole word reading route. One, the “lexical-semantic” route, simply confers meaning but not necessarily pronunciation (used in silent reading), and a “direct-lexical” route that provides pronunciation on the basis of the word’s visual form. Skilled readers probably make much more use of whole-word reading processes than the grapheme–phoneme route in their normal reading. Whichever route is employed, and for normal reading both must be available, the abstracted word can then be passed on for higher-level cognitive manipulations, and a response passed to speech output or writing. The model also must have a direct route from input analysis to output (you can copy and speak words whose meaning you do not know), and also routes that bypass the cognitive system (as you may repeat what you do not understand, or read novel words or nonwords).

Acquired dyslexias can be understood in terms of a model of this kind, and the analysis can become quite complex. Analysis of a single case (Howard & Franklin, 1989), in terms of single word reading alone, revealed a possible eight separate failures in either the component processes or the connections among them. While theoretically interesting, but time-consuming, such an analysis is not of great clinical utility, and this demonstrates both the strengths and the weaknesses of cognitive neuropsychological models.

To return to the classification of acquired dyslexias:

Deep dyslexia is a condition in which relatively severe difficulty in reading is characterized by semantic errors (*lawn* for *grass*, *lift* for *elevator*), visual errors (*wine* for *wire*, *space* for *pace*), function word substitution (*up* for *down*, *with* for *or*), and derivational errors (*clothing* for *clothes*, *teacher* for *teach*). The reading of “nonwords” (such as “brod” or “pake”) is impossible, and ability to read is also affected by what part of speech a word is, and how imageable it is. Patients also have greater difficulty with function words than content words. There has been a great deal of research on this condition (which is also sometimes called *phonemic dyslexia*), despite its relative rarity, and an extensive review has been published by Coltheart, Patterson, and Marshall (1980) that

illustrates the breadth of the debate about it. Deep dyslexics appear to have damage to both the grapheme–phoneme route (as they cannot read nonwords) and the direct-lexical route, being forced to rely on the lexical-semantic route, from which they are prone to extract slightly inexact meanings.

Phonological dyslexia appears rather more straightforward (perhaps because it has received less attention and is relatively rare). These patients read words remarkably well, but do very poorly when asked to read nonwords. In this difficulty with nonwords they are rather similar to deep dyslexics, although the difference between the effects of words and nonwords is much greater in phonological dyslexics. However, they also have difficulties in reading generally, from content words, which they find the easiest, to function words, which cause the greatest difficulty. They also may misread word endings. It is not certain how clearly this dyslexia may be dissociated from deep dyslexia, although the reading of content words by patients with phonological dyslexia is markedly better than the (relatively good) performance of those with deep dyslexia. Imageability of the word has not been reported to have any effect on phonological dyslexic errors. However, it seems that the phonological route in reading is implicated in both disorders. Phonological dyslexics (in different cases) may be reading by either the lexical-semantic or the direct-lexical route, but others have argued that the problem could be explained by partial failure in both the whole word reading and the grapheme–phoneme reading routes (Hillis & Caramazza, 1991; Caccapolo-van Vliet, Miozzo, & Stern, 2004).

Letter-by-letter reading, also known as “visual word-form dyslexia,” is a form of reading disorder that may be seen in alexia without agraphia, or “pure” alexia. Such reading is relatively accurate, but slow, and it seems that the patient must spell out the individual letters, and often does this overtly, before being able to read the word. The actual linguistic characteristics of the word seem to have little effect on reading speed or accuracy. Whether errors occur largely depends on whether the patient accurately identifies each letter and then remembers it long enough to integrate the series of letters into a word. The deficit is assumed to be at the early stages of analysis of the language input.

Surface dyslexia, also termed “semantic” dyslexia, is much more like the reading that one expects from a developmental dyslexic or a child in the early stages of learning to read. Errors are more likely with longer than with shorter words, and there is particular difficulty with words that are irregularly spelled. It is as if the patient breaks the word down into phonemic elements, then does the grapheme-to-phoneme conversion to assemble the word. Errors occur either in integrating the elements, or in not applying the many complex modifications to the con-

version rules demanded by everyday English. Short words may be managed as single elements and read as a whole, but longer words have to be broken down, thus allowing errors to enter into the reading process. There is much confusion in applying the normal reading model to surface dyslexia. Some patients have problems in gaining access to the meanings of irregular words; others appear to have a deficit associated with the lexical-semantic route, while some may have a word identification problem. A simple deficit in one of the reading routes cannot account satisfactorily for surface dyslexia (Patterson, Coltheart, & Marshall, 1985).

Agraphia has been much less well studied than alexia, and is generally regarded as combining features that are seen in speech output performance with processes related to graphemic translation (and hence its association with alexia). The work has clear parallels with the study of acquired dyslexia, and a review is to be found in Beeson and Rapcsak (2003).

APHASIA ASSESSMENT

Aphasia is generally assessed by means of a battery of subtests that are designed to cover comprehensively the major aspects of language function. These would typically include conversational speech; oral and written expression, with tests of repetition, naming, and fluency; and auditory comprehension and reading. A number of such batteries have been produced—a notable review (Kertesz, 1979) discusses 13 of them—although not all are in common use. The value of a particular battery depends upon how well it covers language performance, how long and difficult it is to administer, how reliable are the results derived from its application, and how valid are the conclusions that may be drawn from these results. It will also depend on the kind of sample for which normative data are available, and ultimately on how useful they are in accurately assisting in diagnosis, management, and treatment.

Three batteries seem to be in relatively common use: the Schuell Short Examination for Aphasia (SSEA), the Porch Index of Communicative Abilities (PICA), and the Boston Diagnostic Aphasia Examination (BDAE). The SSEA is itself the short form of a larger battery, taking the most useful elements from the Minnesota Test for Differential Diagnosis of Aphasia, but still providing a relatively broad assessment of language abilities. The PICA is a more modern battery that includes 18 subtests, even extending to the gestural modality. It provides quite a useful amount of diagnostic information but does not include any assessment of conversational speech, and only three common types of aphasia are

differentiated. To these should be added the Frenchay Aphasia Screening Test, which is a brief clinically oriented battery for nonspecialist use, and the PALPA (Psycholinguistic Assessment of Language Processing in Aphasia), offering the most detailed assessment among these instruments.

Probably the most widely used is the BDAE, which is specifically related to the scheme of aphasia classification used in this chapter. This test has an even larger number of subtests than the PICA, providing even greater analytic detail of the patient's performance, but at the expense of quite lengthy administration. A useful feature is the provision of supplementary tests that allow certain areas to be explored in greater detail.

A battery known as the Western Aphasia Battery (Kertesz, 1979) was developed with the aim of improving on others currently available. It concentrates on fluency, comprehension, repetition, and naming, but takes account of recent psycholinguistic research. This has now been widely adopted and has proved to be a useful and popular battery.

Apart from the formal batteries, there are some other tests that deserve mention. The Halstead–Wepman Aphasia Screening Test is still widely used as a relatively brief test instrument to check for the presence of aphasia, and the Token Test, and associated Reporter's Test, are also widely used. The Token Test examines speech comprehension by requiring the subject to perform simple operations with a number of elementary tokens. The tasks are of graded difficulty running from "touch the red circle" up to such problems as "pick up the rectangles, except the yellow one" or "after picking up the green rectangle, touch the white circle." The Reporter's Test requires the converse activity, so that the patient must describe the operation that is being carried out upon the tokens by the examiner. Both of the tests are quick and easy to administer, and the Token Test in particular has gained considerable popularity.

THE REHABILITATION OF APHASICS

Aphasia most commonly results from either traumatic injuries or strokes, the latter being more frequent. The extent of recovery depends on a number of factors, but the most important are probably the cause of the dysfunction and the severity of the handicap. The outcome is generally much better following trauma than following stroke, with more than half of the patients fully recovering their premorbid level of function. This difference is, however, confounded with age, since strokes occur more commonly among the elderly, and this probably accounts for the poorer prognosis. The greatest spontaneous recovery happens within the first three months following the damage, with further significant

improvement up to six months. After that, progress tends to slow down, and there are few gains beyond a year after the damage has occurred (Benson & Ardila, 1996; Kertesz, 1993).

Despite there being few well-controlled studies of aphasia therapy, there is some evidence that it is effective in aiding recovery. Most importantly, there is evidence that therapy is more effective if it is begun as early as possible after the injury. A wide variety of approaches to treatment are used by speech therapists with aphasic patients, but most have developed from Wepman's idea that the patient should be systematically stimulated by appropriate materials and environments (Wepman, 1951). This approach was developed by both Schuell (Schuell, Jenkins, & Jimenez-Pabon, 1964) and Taylor (1964). Typical therapy sessions will involve the acting out of everyday situations and working with classes of objects such as food or clothing, or with similar sounding groups of words. There is a continual attempt to elicit words and stimulate responses, and extensive use is made of repetition and auditory stimulation.

Recently, therapy that aims to reestablish language by specific teaching programs has become more popular. This approach allows a more systematic structure for the therapy program, which may be based on an explicitly linguistic approach, and may also employ behavioral learning techniques such as operant reinforcement. An example of this kind of approach is to be found in Weniger, Huber, Stachowiak, and Poeck (1980). Specific information about the patient's linguistic deficits is translated into a training program designed to teach these deficient aspects of language, with progress at each stage carefully monitored and the programme adapted accordingly (Marshall, 2003). In some programs, specific rewards will be given for success in producing particular responses, according to a formal and explicit system.

There are a variety of other, less popular, forms of therapy. Among these are Deblocking Therapy (Weigl, 1968), which aims to use the intact aspects of language function to assist in the redevelopment of the damaged areas, and Compensation Therapy (Holland, 1977), which teaches the patient strategies to enable him or her to circumvent the difficulties by using the intact areas of function. Melodic Intonation Therapy (Sparks, Helm, & Albert, 1974) capitalizes on the fact that many aphasics maintain the ability to sing when they cannot speak, and so can learn to "intone" the speech they wish to generate. Finally, Visual Communication Therapy (Gardner, Zurif, Berry, & Baker, 1976) has translated some of the methods used to teach language to chimpanzees, based upon simple figural and geometric symbols, into a method that seems of particular value with patients who have severe global aphasia.

Therapy can be of considerable value to many patients, and aphasics often regain a significant degree of language function. There are, however, a large number of patients whose handicap persists, and who form a major rehabilitational challenge. What are needed are more extensive and better-designed studies of the value of particular therapies so that treatment can be applied more effectively and economically to those with language dysfunction.

CONCLUSION

The study of aphasia is currently a fruitful area of interchange between academic psychologists and clinical neuropsychologists, resulting in a better understanding of both normal and abnormal language processes. There are problems in classifying the aphasias, but there is now a widely accepted scheme, based upon the Boston classification, that divides aphasias into six types (see Table 7.2). Each of these forms of aphasia may be associated with a particular region of the cortex, although this correspondence is not as clear as is sometimes supposed. The cortical areas serving language are lateralized, being in the left hemisphere of almost all right handers and the majority of left handers, although left handers typically have a more bilateral representation. Disorders of reading and writing are naturally associated with major disorders of comprehension and expression of speech. There are a variety of forms of therapy currently practiced with aphasics, most of which appear effective, although there are few comparative and controlled studies of treatment.

TABLE 7.2. A Classification of the Aphasias with Their Associated Symptoms, as Described in the Text

| | Lesion | Fluency | Speech output | Repetition | Naming | Auditory comprehension | Written output | Reading |
|----------------------------|--|-----------------------------|--|-------------------|----------|---|----------------|---------------------------|
| <u>Aphasias</u> | | | | | | | | |
| Broca's (motor; nonfluent) | Inferior posterior frontal | Mild to severe impairment | Phonemic paraphasias; agrammatic deletion; word order and meaning normal | Limited | Limited | Normal; function words limited | As speech | As comprehension |
| Wernicke's (sensory) | Superior, middle, and posterior temporal | Normal to hyperfluent | Normal to jargon; word order normal to impaired; meaning irrelevant, vague | Impaired | Impaired | Mild to severe impairment; can tell if speech | As speech | As comprehension |
| Conduction (central) | Arcuate fasciculus and deep | Mild to moderate impairment | Some phonemic paraphasias; word order normal; meaning appropriate | Severely impaired | Impaired | Normal | As speech | Normal to mild impairment |

| | | | | | | | | | |
|---|--|-------------------------------|---|----------|----------|--|------------------------------|----------------------------|--|
| Anomic (amnesic) | Angular gyrus; posterior middle temporal | Normal (except noun blocking) | Normal except nouns may be omitted, substituted, or paraphrased | Normal | Impaired | Normal | May be as speech | As comprehension | |
| Trans-cortical motor | Anterior mesial frontal | Normal when repeating | Normal when repeating | Normal | Limited | Normal | Impaired | Normal to mild impairment | |
| Trans-cortical sensory (isolation syndrome) | Peri-Sylvian association cortex | Normal | Normal to jargon as Wernicke's | Normal | Impaired | Severely impaired; can tell syntactic errors | Impaired | Impaired | |
| Global | | Impaired | Impaired | Impaired | Impaired | Impaired | Impaired | Impaired | |
| Other language disorders | | | | | | | | | |
| Alexia, agraphia | Supra-marginal gyrus; angular gyrus | Normal | Normal | Normal | Normal | Normal | Severely impaired (agraphia) | Severely impaired (alexia) | |

FURTHER READING

The general texts listed at the end of Chapter 1 are probably the best way to pursue further study of this topic. Additional, more specific, references are, of course, contained within this chapter and listed below. However, some useful general texts more specifically concerned with language disorders are:

- Behrmann, M., & Patterson, K. (Eds.). *Words and Things: Cognitive Neuropsychological Studies in Tribute to Eleanor M. Saffran* (Hove, UK: Psychology Press, 2004). (Reprint of *Cognitive Neuropsychology*, vol. 21, nos. 2–4.)
- Berndt, R. S. *Language and Aphasia*, Handbook of Neuropsychology series (Amsterdam: Elsevier, 2001).
- Funnell, E. *Case Studies in the Neuropsychology of Reading* (Hove, UK: Psychology Press, 1999).
- Hillis, A. *Handbook of Adult Language Disorders* (Hove, UK: Psychology Press, 2002).

A general introduction to many aspects of dyslexia is to be found in:

- Beaton, A. A. *Dyslexia, Reading and the Brain: A Sourcebook of Psychological and Biological Research* (Hove, UK: Psychology Press, 2004).

REFERENCES

- Beaton, A. A. *Dyslexia, Reading and the Brain: A Sourcebook of Psychological and Biological Research* (Hove, UK: Psychology Press, 2004).
- Beeson, P. M., & Rapcsak, S. Z. Neuropsychological Assessment and Rehabilitation of Writing Disorders, in P. W. Halligan, U. Kischka, & J. C. Marshall, eds., *Handbook of Clinical Neuropsychology* (Oxford, UK: Oxford University Press, 2003).
- Behrmann, M., & Patterson, K. (Eds.). *Words and Things: Cognitive Neuropsychological Studies in Tribute to Eleanor M. Saffran* (Hove, UK: Psychology Press, 2004).
- Benson, F. D. Fluency in Aphasia: Correlation with Radioactive Scan Localization, *Cortex*, 3 (1967), 373–394.
- Benson, F. D. Aphasia, in K. M. Heilman & E. Valenstein, eds., *Clinical Neuropsychology* (New York: Oxford University Press, 1979).
- Benson, F. D., & Ardila, A. *Aphasia: A Clinical Perspective* (Oxford, UK: Oxford University Press, 1996).
- Bogen, J. E., & Bogen, G. M. Wernicke's Region—Where Is it?, *Annals of the New York Academy of Sciences*, 280 (1976), 834–843.
- Brown, J. W. The Neural Organization of Language: Aphasia and Lateralization, *Brain and Language*, 3 (1976), 482–494.
- Caccappolo-van Vliet, E., Miozzo, M., & Stern, Y. Phonological Dyslexia: A Test Case for Reading Models, *Psychological Science*, 15 (2004), 583–590.

- Carter, R. L., Hohenegger, M., & Satz, P. Handedness and Aphasia: An Inferential Method for Determining the Mode of Cerebral Speech Specialization, *Neuropsychologia*, 18 (1980), 569–574.
- Coltheart, M., Patterson, K. E., & Marshall, J. C. (Eds.). *Deep Dyslexia* (London: Routledge and Kegan Paul, 1980).
- Coltheart, M., Rastle, K., Perry, C., Langdon, R., & Ziegler, J. DRC: A Dual Route Cascaded Model of Visual Word Recognition and Reading Aloud, *Psychological Review*, 108 (2001), 204–256.
- Critchley, M. *The Divine Banquet of the Brain* (New York: Raven Press, 1979).
- Ellis, A. W., & Young, A. W. *Human Cognitive Neuropsychology: A Textbook with Readings* (Hove, UK: Psychology Press, 1996).
- Gardner, H., Zurif, E. B., Berry, T., & Baker, E. Visual Communication in Aphasia, *Neuropsychologia*, 14 (1976), 275–292.
- Geschwind, N. The Organization of Language and the Brain, *Science*, 170 (1970), 940–944.
- Goodglass, H., & Kaplan, E. *The Assessment of Aphasias and Related Disorders* (Philadelphia: Lea and Febiger, 1972).
- Hanley, J. R., & Kay, J. Neuropsychological Assessment and Treatment of Disorders of Reading, in P. W. Halligan, U. Kischka, & J. C. Marshall, eds., *Handbook of Clinical Neuropsychology* (Oxford, UK: Oxford University Press, 2003).
- Hillis, A. E., & Caramazza, A. Mechanisms for Accessing Lexical Representations for Output: Evidence from a Category Specific Semantic Deficit, *Brain and Language*, 40 (1991), 106–144.
- Holland, A. L. Some Practical Considerations for Aphasia Rehabilitation, in M. Sullivan & M. S. Kommers, eds., *Rationale for Adult Aphasia Therapy* (Omaha: University of Nebraska Press, 1977).
- Howard, D., & Franklin, S. *Missing the Meaning?: Cognitive Neuropsychological Study of Processing of Words by an Aphasic Patient* (Cambridge, MA: The MIT Press, 1989).
- Howes, D., & Geschwind, N. Quantitative Studies of Aphasic Language, in D. M. Rioch & E. A. Weinstein, eds., *Disorders of Communication* (Baltimore: Williams & Wilkins, 1964).
- Kertesz, A. *Aphasia and Associated Disorders* (New York: Grune and Stratton, 1979).
- Kertesz, A. The Anatomy of Jargon, in W. Brown, ed., *Jargonaphasia* (New York: Academic Press, 1981).
- Kertesz, A. Recovery and Treatment, in K. M. Heilman & E. Valenstein, eds., *Clinical Neuropsychology* (Third edition, Oxford, UK: Oxford University Press, 1993).
- Kertesz, A., & Benson, F. D. Neologistic Jargon: A Clinicopathological Study, *Cortex*, 6 (1970), 362–386.
- Kertesz, A., Lesk, D., & McCabe, P. Isotope Localization of Infarcts in Aphasia, *Archives of Neurology*, 34 (1977), 590–601.
- Luria, A. R., & Hutton, J. T. A Modern Assessment of the Basic Forms of Aphasia, *Brain and Language*, 4 (1977), 129–151.

- Marshall, J. C. Treatment of Spoken Language Disorders, in P. W. Halligan, U. Kischka, & J. C. Marshall, eds., *Handbook of Clinical Neuropsychology* (Oxford, UK: Oxford University Press, 2003).
- Murdoch, B. (Ed.). *Dysarthria: A Physiological Approach to Assessment and Treatment* (Cheltenham, UK: Stanley Thornes, 1998).
- Ojemann, G. A., & Mateer, C. Human Language Cortex: Localization of Memory, Syntax and Sequential Motor-phoneme Identification Syndromes, *Science*, 205 (1979), 1401–1403.
- Ojemann, G. A., & Whitaker, H. A. Language Localization and Variability, *Brain and Language*, 6 (1978), 239–260.
- Patterson, K. E. Neuropsychological Approaches to the Study of Reading, *British Journal of Psychology*, 72 (1981), 151–174.
- Patterson, K. E., Coltheart, M., & Marshall, J. (Eds.). *Surface Dyslexia: Neuropsychological and Cognitive Analyses of Phonological Reading* (Hove, UK: Erlbaum, 1985).
- Rasmussen, T., & Milner, B. Clinical and Surgical Studies of the Cerebral Speech Areas in Man, in K. J. Zülch, O. Creutzfeldt, & G. C. Galbraith, eds., *Cerebral Localization* (Berlin: Springer-Verlag, 1975).
- Satz, P. A Test of Some Models of Hemispheric Speech Organization in the Left and Right Handed, *Science*, 203 (1979), 1131–1133.
- Schuell, H., Jenkins, J., & Jimenez-Pabon, E. *Aphasia in Adults* (New York: Harper & Row, 1964).
- Sparks, R., Helm, N., & Albert, M. L. Aphasia Rehabilitation Resulting from Melodic Intonation Therapy, *Cortex*, 10 (1974), 303–316.
- Taylor, M. L. Language Therapy, in H. Burr, ed., *The Aphasic Adult: Evaluation and Rehabilitation* (Charlottesville: Wayside Press, University of Virginia, 1964).
- Wallesch, C.-W., Johanssen-Horbach, H., & Blanken G. Assessment of Acquired Spoken Language Disorders, in P. W. Halligan, U. Kischka, & J. C. Marshall, eds., *Handbook of Clinical Neuropsychology* (Oxford, UK: Oxford University Press, 2003).
- Weigl, E. On the Problem of Cortical Syndromes, in M. L. Simmel, ed., *The Reach of Mind* (New York: Springer, 1968).
- Weisenberg, T., & McBride, K. *Aphasia* (New York: Commonwealth Fund, 1935).
- Weniger, D., Huber, W., Stachowiak, F.-J., & Poeck, K. Treatment of Aphasia on a Linguistic Basis, in M. T. Sarno & O. Hook, eds., *Aphasia: Assessment and Treatment* (Stockholm: Almqvist and Wiksell, 1980).
- Wepman, J. M. *Recovery from Aphasia* (New York: Ronald, 1951).
- Zangwill, O. L. Excision of Broca's Area without Persistent Aphasia, in K. J. Zülch, O. Creutzfeldt, & G. C. Galbraith, eds., *Cerebral Localisation* (Berlin: Springer-Verlag, 1975).

Degenerative Diseases and Profound Brain Injury

So far we have concentrated principally on the cerebral cortex, and this does reflect the main preoccupation of human neuropsychology. However, there is a great deal more to the brain than its cortex, as can be demonstrated by the disorders discussed in this chapter. First to be considered will be multiple sclerosis, in which diffuse lesions can occur almost anywhere in the nervous system, including both the cortex and the subcortex of the forebrain. Multiple sclerosis serves as the most common example of a number of degenerative diseases that affect the nervous system and have neuropsychological consequences. The other prominent example, not discussed here, is motor neuron disease, or amyotrophic lateral sclerosis, also known as Lou Gehrig's disease in the United States.

The next two conditions to be described, Parkinson's disease and Huntington's disease, are disorders that primarily affect subcortical centers, particularly in the basal ganglia, and are sometimes referred to as subcortical dementias. This distinguishes them from the cortical dementias, of which the most common are Alzheimer's disease and multi-infarct dementia, normally disorders of older people, which again, are not discussed here.

The final sections of this chapter deal with severe and profound brain injury in its various forms, in which lesions can be considered to have occurred in many regions of the brain. As a result the presentation of these disorders is very different from the discrete cortical lesions that are most commonly studied by neuropsychologists but no less fascinat-

ing for what they can reveal about the organization of psychological processes in the human brain.

MULTIPLE SCLEROSIS

Multiple sclerosis (MS) is the most common of the neurological diseases, affecting about 1 per 1,000 of the population. It is also one of the most intriguing as its presentation and course are so variable. Although most people's image of an MS patient is of someone in a wheelchair, fewer than 10% of those with MS ever need a wheelchair and the majority live out their normal life expectancy and die of a cause unrelated to their disease. At the other extreme there are a small number who become profoundly disabled by their MS, becoming doubly incontinent, highly dependent for all their physical care, able to communicate only through aids, and cognitively disabled, besides needing a wheelchair. The course is equally variable. Some patients develop a mild form of MS that gradually worsens through their lives, but they never develop any major handicap. Some have a much more steady decline into significant disability as the disease progresses. Some, although this is rarer, very rapidly develop very severe disability, which then persists over many years. All patterns seem possible and are seen in MS.

Although the course is so variable, there are two broad patterns that can be identified: a relapsing–remitting form (RR) and a chronic progressive form (CP). In RR there are episodes in which the MS patient experiences an increase in symptoms for a period, but this is then followed by a period of relative recovery. The duration of the relapses and periods of remission, and their frequency, are of course highly variable. Remissions have been recorded, however, up to 25 years. First presentation is most likely to be the RR form, although the illness may begin with the CP form. The RR form may, but does not always, develop into the CP form.

The reason for this variability is in the nature of the pathology. The axons that constitute the network of the nervous system are (in most places) contained within a myelin sheath, which might be considered the insulation on the wiring. In MS this “insulation” breaks down in the process of *demyelination* so that the associated neuron becomes dysfunctional. This process can occur anywhere in the nervous system, and be relatively localized or very diffuse. The symptoms that afflict the patient naturally reflect the distribution of these lesions. Although the lesions are commonly quite widespread, there do appear to be two broad types of pattern: a form that primarily affects the spine and is associated with

greater physical disability, and a form that primarily affects the brain and leads to greater cognitive impairment. However, in particular cases the situation is usually not so clear cut.

Why this demyelination occurs is more of a puzzle. The main line of research is that MS is an autoimmune disease and that, for some reason, the body fails to recognize the myelin as its own and so destroys it. There are many sophisticated hypotheses about the precise mechanism by which this occurs but none, as yet, can be regarded as a satisfactory explanation of the cause of MS. There is, however, a genetic element, with about 10% of cases being familial. More women than men are affected, and the disease is commoner in temperate climates worldwide, although this may reflect genetic pools rather than an essential environmental factor. Onset is generally between 20 and 40, most commonly between 30 and 40, although the disease may rarely arise in children or in older adults. A precipitating factor may be present but is more commonly a physiological stress than a psychological one.

The early symptoms of MS are of the effects of single or multiple discrete lesions. Optic neuritis (inflammation of the optic nerve resulting in blurring of vision, loss of acuity, and pain), weakness, especially of the legs, numbness, double vision, and urgency to urinate are common early symptoms. Patients with advanced cases may be dependent on a wheelchair or special seating system, produce staccato and slurred speech that may be very difficult to understand, have abnormal eye movements, upper limb weakness and ataxia, severe spastic paraplegia with spasms, and sensory loss, and be doubly incontinent. They may be completely dependent for all their physical care. As already stated, the variability of both the course of the disorder and the pattern of symptoms cannot be overstated. The diagnosis of MS used to be a “diagnosis by exclusion,” that is, MS was diagnosed when no other explanation for the patient’s symptoms could be found. Nerve conduction studies (electromyography) were next used to aid in diagnosis, but it is now possible to have a positive identification of the disease by the pattern of lesions observed on MRI scan (see Chapter 13).

The degree of cognitive impairment in MS is poorly correlated with physical disability—reasonably, as different lesions are almost certainly involved. It is generally considered that visuo-spatial abilities are more affected than verbal abilities, but this may be an artifact of visual problems and motor handicap. Dynamic, conceptual, problem-solving abilities tend to be more affected than overlearned routinized skills, so standard measures of intelligence may not be fully sensitive to the difficulties. The most consistent effect reported is a reduced speed of mental information processing and impaired attention. There are also promi-

nent effects in memory with an impaired ability to learn and recall and more rapid forgetting with interference, with recognition less affected than recall. The underlying problem may be a retrieval deficit due to faulty search strategies (DeLuca, Barberi-Berger, & Johnson, 1994). Individuals with MS may also have difficulty forming concepts, shifting sets, and responding to feedback. For cognitive factors there is the same variability as seen in other aspects of the disease (Rao, Leo, Bernardin, & Unverzagt, 1991; Zakzanis, 2000).

MS patients also have disturbances of mood, and while the rate varies considerably across different studies, it is typically found to be between 40% and 60%. A classic inappropriate euphoria is often described for MS and, while this certainly can occur in some cases, it has probably been overemphasized; the most common affective reaction is depression (Arnett, Higginson, & Randolph, 2001). The suicide risk in MS may be up to 15%. Psychologically, it is probably sensible to think of the experience of MS as a series of repeated minor losses that are unpredictable and each a challenge to the individual.

There is, as yet, no effective treatment for MS, although there are drugs such as beta-interferon that can slow the progression in the earlier stages. It is a matter of dealing with the symptoms as they arise, when they can be alleviated. Even in a progressive disease of this kind there is a role for rehabilitation interventions to reduce the accumulating impact of the disease, if not to achieve any absolute improvement. Cognitive rehabilitation may ameliorate the impact of the disease and disability counseling clearly has a role in supporting individuals with the disorder.

PARKINSON'S DISEASE

Parkinson's disease (PD), originally described by Parkinson in 1817 and previously known as "paralysis agitans," is a degenerative disorder of the subcortical areas in the basal ganglia that serve nonvoluntary (extrapyramidal) movement. The principal features of the disorder are tremor, slowness of movements ("bradykinesia"), and rigidity in movement. Its prevalence is also around 1 per 1,000 of the population, making it nearly as common as MS. PD should not be confused with *Parkinsonism*, where similar symptoms occur as a secondary result of other factors, which may include psychotropic medication and also environmental toxins. The mechanism that causes the disorder is, however, essentially the same.

The classic, untreated, form of the disease has a number of distinctive characteristics. Patients adopt a stooped posture and tend to walk

with little steps, “*marche à petits pas*.” They also tend to walk in straight lines and may have difficulty in turning, even to the extent of using a lamp post as a pivot around which to turn. Their movements are also rather jerky, and testing reveals “cogwheel rigidity” at the major joints as rotational movement occurs in a series of steps. Perhaps the most marked symptom is the tremor. This is resting tremor, which means that it is abolished by voluntary movement. (Tremor during the execution of movements is a symptom of lesions of the cerebellum.) Patients who sit passively and show significant tremor in the arms and hands may, surprisingly, manage a cup and saucer remarkably well, but only as long as they concentrate upon what they are doing. The motor aspects of speech are also affected so that patients find it increasingly difficult to make themselves understood as a result of their dysarthria, abnormal speech resulting from motor problems with speech movements, and speech may be “hypophonic,” of low volume. It is now relatively rare to see the untreated form of the disorder in its full presentation, but it is within the memory of my generation to see patients seriously disabled by this disease.

Although it is agreed that PD is associated with abnormalities in centers in the basal ganglia, particularly in the substantia nigra, which rely on the neurotransmitter dopamine, the precise neuropathology is still to some extent uncertain. It was once thought that the disorder originated in the thalamus, but the putamen and the caudate nucleus are now thought to be more important. A particularly influential suggestion has come from Alexander, Crutcher, and DeLong (1990), who proposed that there are five circuits that interlink the basal ganglia with the frontal lobes, serving motor control, cognition, the control of eye movements, and emotion. Among these “Alexander circuits,” it is the circuit for motor control that is affected in PD.

One approach to the treatment of PD has been neurosurgery (see Chapter 9), but while it is effective, most patients are now treated by medication in the form of dopaminergic replacement therapy (levodopa). This can be highly successful, although with prolonged use some patients suffer from abrupt fluctuations between the symptoms being present and absent (Obeso, Olanow, & Nutt, 2000).

An interest in the neuropsychological aspects of PD has, as with MS, been a relatively recent development (see, for example, Litvan, Mohr, Williams, Gomez, & Chase, 1991; Pillon, Dubois, Ploska, & Agid, 1991), and there is still much research to do to clarify the precise nature of the impairments. Further, there seem to be three subgroups of PD patients: those with no or minimal cognitive deficit; those with specific neuropsychological problems; and those who suffer a generalized

dementia that shares some characteristics with other dementias of old age. As with other subcortical dementias, and MS, there is only a very weak relationship between the degree of motor handicap and the extent of cognitive impairment.

The specific neuropsychological problems evident in PD sufferers fall into three areas: executive function, memory, and visuo-spatial impairment. Executive functions were discussed in Chapter 3, and PD patients may do poorly on many of the tests described there. Performance on the Wisconsin Card Sorting Test reveals problems in concept formation, and PD patients also have difficulty in changing mental set. There may be problems in recalling the order of events, and there are associated problems in initiation and word fluency. One hypothesis about the basis of this dysexecutive behavior is that PD patients have problems when forced to rely on internal cues, although they are able to perform normally when given external cues. This observation (Brown & Marsden, 1990) has led to the suggestion that PD patients have a restriction on central processing resources that affects the normal operation of internally generated supervisory processes. This is a sensible suggestion, but difficult to test empirically.

With respect to their memory problems, PD patients have particular difficulty with episodic or “context-bound” information. They have relatively normal recognition memory, but are poor at recalling a short story or learning sets of paired associates. This suggests that encoding and storage processes may be operating normally, but that the processes of retrieval, especially if this requires some effort or organization, may be defective. Implicit memory—that is, memory without awareness—has also been found to be impaired in some PD patients.

There has been more debate about visuo-spatial impairments in PD. It was initially thought that PD patients had difficulty relating objects in space and performing spatial mental transformations. However, this problem has been considered by some to rather reflect a dysexecutive problem—a difficulty in shifting sets or in planning. While this may be a valid interpretation, it is certainly the case that visuo-spatial problems become severe in PD patients who develop a general dementia, and a primary specific visuo-spatial impairment cannot be ruled out in patients less severely affected (Boller & Muggia, 1999).

Disorders of mood are also seen in PD, with widely varying estimates as to its prevalence, between 20% and 90%. However, it seems generally agreed that about 20% of PD patients suffer from a major depression with about a further 20% experiencing a more minor depression. The factors that dictate into which group of cognitive severity patients will fall, or the course of the disease, have yet to be identified.

HUNTINGTON'S DISEASE

Huntington's disease (HD) is the classic example of an hereditary neurodegenerative disease, inherited by Mendelian dominance. That is, if one of your parents has the disorder, there is a 50% chance that you will also suffer from the disorder. Although this has been understood for some time, one reason why the disease has persisted is that the onset is typically between ages 35 and 40, that is, after the childbearing years.

HD was formerly known as "Huntington's Chorea" from the dramatic gyrating movements of the limbs (in neurological terms: choreiform) that characterize the initial phases of the disorder, first described by George Huntington in 1872. The disease affects about 1 in 20,000 of the population, but 1 in 3,000 has a greater than 10% chance of becoming affected. As would be expected, males and females are equally affected.

The genetic abnormality producing HD has now been identified and lies on chromosome 4, associated with an abnormal number of repeats of the bases C, A, and G. Normal chromosomes have a sequence of between 11 and 34 repeats of the CAG combination, while HD is associated with 40 or more (and sometimes very many more) repeats. Interestingly, a higher number of repeats is linked to earlier onset of the disease. This discovery has permitted development of a highly reliable test that can determine whether an individual at risk will develop the disease. However, whether to take the test raises considerable personal, psychological, and ethical issues, and many at-risk individuals elect not to take it. It must be remembered that most people at risk have had the experience of a close relative suffering, and dying from, a devastating disorder.

The disease appears with the insidious onset of involuntary movements, usually in middle life, but in a minority there is an early onset before the age of 20. Problems making speech movements (dysarthria), difficulties of upper-limb movement and an abnormal gait follow, with progressive loss of mental functions within a few years. Eventually, problems of chewing, swallowing, and breathing lead to physical deterioration and death within about 15 to 20 years.

The chromosomal abnormality produces a protein (huntingtin) that affects the neurotransmitters in the basal ganglia, and especially the striatum, which play a role in both voluntary and involuntary movement. HD is therefore one of the subcortical dementias, as well as being a movement disorder.

Global intellectual impairment occurs in only about two-thirds of cases, but all have a degree of neuropsychological abnormality, in addi-

tion to the functional handicap caused by the movement disorder. The early effects are typically in memory, in verbal fluency, and in visuospatial performance, but this is generally followed by a general decline with particular difficulties in organizing, sequencing, and planning, together with lack of initiation. The effects on memory are complex and not easily summarized. It would seem that different processes are affected at different stages of the disease, but general features include an increased sensitivity to proactive interference, failures of encoding strategies, and a marked retrieval deficit. The retrieval deficit results in there being an abnormally large advantage for recognition over recall (Butters, Wolfe, Martone, Granholm, & Cermak, 1985; Zakzanis, Leach, & Kaplan, 1999).

Visuospatial functions in HD are harder to analyze because of the impaired motor function, and this extends to the control of eye movements (and so scanning of test materials) as well as arm and hand movements. Language function is, however, relatively well preserved, although speech output is seriously affected by the motor problems in speaking (Poldroll, Caspary, Lange, & Noth, 1988). In general, HD is a classic psychomotor disorder with a general failure of the integration of cognitive and motor activities. Progression of the disease leads to more widespread, and therefore more diverse, effects.

There are also significant psychological aspects of HD. In the initial phases of the disease the picture is dominated by the classic choreiform involuntary movements, although other functions begin to be affected. However, the choreiform movements tend to subside in the middle stages of the disorder, when there is commonly a phase of psychiatric disturbance before, in the late stages, increasing physical disability leads to death. In the middle stages, psychiatric disorder occurs in between one-third and three-quarters of patients. In 4–12% there are schizophrenia-like symptoms of a variety of types, although persistent and induced delusions are particularly common. Patients are frequently disinhibited, both with respect to sexual activity and also in aggressive outbursts. Clinical depression is found in up to half of cases, and there is evidence that this is not simply a secondary psychological reaction to having developed the disease, but the depression may have an underlying organic cause. Suicide is commoner than in the general population, and the rate is raised by comparison with the general population not only for patients but also for those at risk. In terms of personality change, those with HD may become withdrawn and neglect their personal care (Caine & Shoulson, 1983).

HD is one of the most unpleasant of neurological diseases, partly because of its relentless course to death, and partly because those who develop it generally have personal knowledge of the fate that awaits

them. Although the mechanism of the disease is now well understood, there is as yet no cure, and treatment consists of alleviating the symptoms.

PROFOUND BRAIN INJURY

Neuropsychologists generally work with a model of normal performance and deviations from that model. In the behavioral neurology approach (see Chapter 1) there is a clear distinction between what a normal person can perform but the patient cannot, and the neuropsychologist searches for failures in performance that would never be seen in a normal person. In the individual normative approach (the British tradition) there is a statistical definition of the range of normal performance and neuropsychological assessment seeks to identify performance that falls outside this range, and is therefore statistically abnormal. Neuropsychologists look for “holes” (deficits) in the pattern of normal function (see Figure 8.1).

However, there are some conditions for which the impairment is so severe, or so widespread, that this is no longer a helpful and appropriate model. In these states of profound brain injury, a model that assumes no intelligent function at all, but in which “islets” of retained function are present, seems more appropriate (see Figure 8.2). The task of the neuropsychologist is to chart these islets and to attempt to extend their size and form bridges among them.

Patients with profound brain injury are often highly unresponsive, and may make no intentional responses whatsoever. Their awareness of



FIGURE 8.1. The conventional model of specific neuropsychological deficits (white areas) in a context of normal preserved abilities (gray areas).

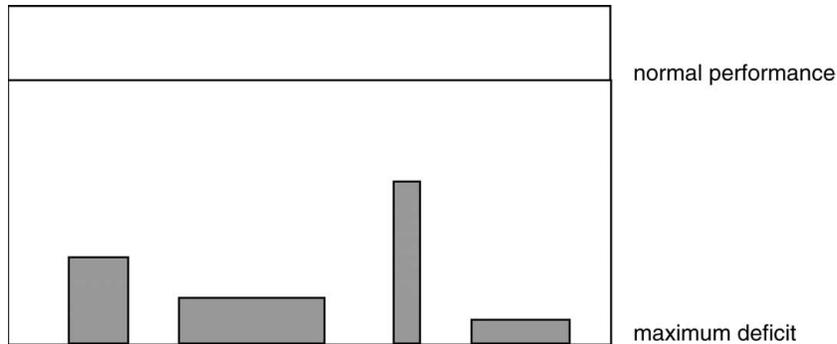


FIGURE 8.2. The neuropsychological model in profound brain injury; only islets of ability (gray areas) remain and are below the normal level of performance. The area of functional disability is shown as white.

their environment may be in question, and they show little spontaneous behavior. They are generally highly dependent for all their physical care; they may or may not be able to communicate even their most basic needs. While these states are relatively rare, advances in emergency medicine have resulted in increasing survival from very severe trauma, so the number of individuals who survive in a state compatible with ongoing life (without artificial support), but with profound neurological impairment, has increased. This has brought new challenges, not only in care and rehabilitation, but also in the moral and ethical issues that these patients raise, which will briefly be discussed at the end of this chapter.

There are four states of profound brain injury that it is sensible to differentiate: coma, vegetative state, minimally conscious state, and locked-in syndrome (Royal College of Physicians, 2003).

Coma

Coma is of relatively little interest to neuropsychologists, but it is important to distinguish between coma and the other states described here. In coma, patients do not open their eyes, either spontaneously or in response to stimulation. They do not make any response to even simple commands (such as “open your eyes” or “raise your hand”) and do not utter, or even mouth, recognizable words. There are no *intentional* movements. There may be some movements as a result of reflexes, and these can be difficult to distinguish from intentional movements, but intentional movements do not occur in coma. A sensible parallel, with which most readers will be familiar, is the deceptive “smiling” seen in

infants. A small baby may appear to smile, even in response to the “coo-coo” noises directed to it, but the experienced parent knows that the smiling is only associated with wind (gas) and is ready with the terry towel when the baby burps and spits up some of its last feeding. Patients in coma may react to certain stimuli, such as painful stimuli, but the response is only a reflex action of withdrawal, or even of a movement that attempts to remove the source of the pain.

An important instrument used in the identification of coma is the Glasgow Coma Scale (GCS), which is a simple, internationally used scale that assesses the level of consciousness. It is even used by paramedics at the scene of an accident, and is routinely recorded on admission to accident and emergency facilities. The GCS has three component subscales that relate to eye opening, motor function, and verbal behavior; the details are given in Table 8.1.

As can be seen, the maximum score, calculated over all three scales, is 15, and that is what a normal person, you or I, would score at the present. The minimum score is actually 3, and a person with this score is regarded as being in “deep coma.” The criterion for the diagnosis of coma is a score of 8 or less.

Because patients in coma produce no intentional behavior, and do not communicate, they are really of little interest to neuropsychologists, except in so far as it may be important to monitor when the level of consciousness improves and a patient emerges from the coma.

Vegetative State

The vegetative state (VS) is identical to the state of coma, but with one critical difference. The patient in VS opens his or her eyes spontaneously and shows what is termed a “sleep-wake cycle.” This does not really mean that there are periods in which the patient is actually, in normal terms, asleep or awake, but that there are prolonged periods in which the eyes are kept open, and periods when they are closed. These cycles commonly follow a daily rhythm typical for the patient, but the cycle is

TABLE 8.1. Glasgow Coma Scale (GCS)

| E: eye opening | M: motor function | V: verbal |
|-----------------|----------------------------|-----------------------|
| 1 None | 1 None | 1 None |
| 2 To pain | 2 Extends to pain | 2 Grunts |
| 3 To sound | 3 Abnormal flexion to pain | 3 Inappropriate words |
| 4 Spontaneously | 4 Normal flexion to pain | 4 Confused |
| | 5 Localizes pain | 5 Oriented |
| | 6 Normal | |

not necessarily tied to day- and nighttime in the normal human diurnal pattern. The cycles are, reasonably, believed to relate to periods of increased and decreased arousal, although the eyes-open state does not necessarily indicate awareness, for the patient, as the individual in coma, shows no intentional movement and no response to command.

The issue of awareness in VS is a difficult one. These states are often referred to as “states of low awareness” as the question of any degree of awareness is still open—and may simply be unknowable. As the patient is apparently unresponsive (other than through reflex activity), there is no way of telling whether any awareness is present. The only relevant information we possess comes from those who have emerged from VS and recovered sufficiently to report their experience. Such cases are infrequent and the evidence anecdotal. However, there are sufficient reports to lead us to think that there may be some limited awareness in certain patients, sufficient for care staff to be required to treat patients as if they were aware. The “first words” spoken by at least two patients of whom I am aware are telling: one is reported to have said, “Leave me alone”; another, agnostic, young man, around whom his mother and her friends had held lengthy daily religious meetings, exclaimed, “F—off, Mum.” Notwithstanding this slender evidence, it is likely that VS patients are not aware in any way that we would recognize.

The terminology associated with VS has tended to become more confusing, although it aims to clarify the distinction between those who, for a short period, pass through VS on the route from coma toward recovery and those for whom VS is a long lasting condition; the longest survival in VS is certainly in excess of 20 years and may be over 40 years. The distinction used to be made between VS and PVS—persistent vegetative state, a state of VS extending beyond 6 months in the view of some, or beyond 1 year in the view of others. PVS was considered to carry a poor prognosis. This is likely to be the meaning of the term in any publication before about 2003. However, in 2003 an influential report of Britain’s Royal College of Physicians (2003) recommended a distinction between vegetative state (VS), persistent vegetative state where VS extended beyond 4 months (*not* “PVS”), and permanent vegetative state that might be the condition diagnosed after a longer, but not clearly defined, period (PVS). This classification is being widely adopted, but the lack of a clear definition by which PVS (as a permanent state) should be diagnosed leaves some residual confusion.

This confusion is supported by different views as to whether VS can ever be described as “permanent” given evidence about “late” emergence from VS. It is certainly the case that emergence from VS after 1 year is very unlikely. However, there are some patients who do emerge

after a period of years in VS. The evidence is difficult to evaluate, as many of the reports are anecdotal and it is not always clear that the diagnosis of VS in the earlier period was accurate, misdiagnosis being not uncommon (Andrews, Murphy, Munday, & Littlewood, 1996), but there is reason to think that emergence after up to 5 or 6 years may occur.

A considerable impact has been made by recent reports from the Impaired Consciousness Research Group in Cambridge, United Kingdom, that aspects of language comprehension, as well as covert awareness, are retained in a patient in a vegetative state, and that this can be demonstrated by functional magnetic resonance imaging (see Chapter 13; Coleman et al., 2007; Owen et al., 2007). For example, when instructed to imagine either playing a game of tennis or moving about a familiar room, their patient, who is apparently unresponsive, produces brain activity detected by fMRI that distinguishes between these different instructions. This is certainly a startling finding if it proves to be repeatable and observable in at least a proportion of vegetative patients, and challenges our current beliefs about the state of consciousness in which these patients exist. It is unfortunate that the patient cannot tell us whether any conscious awareness accompanies this brain response, but this is clearly destined to be an active area of research in the near future.

Minimally Conscious State

The patient emerging from VS moves into a minimally conscious state (MCS), which is a much more poorly defined condition in which there is evidence of some limited awareness and some limited responsiveness (Giacino et al., 2002). The formal definition of MCS is that there should be meaningful, if often inconsistent, behavioral responses that are significantly stimulus linked, and that have been observed at least once during formal assessment.

Some neuropsychologists feel that MCS is an unfortunate term, as it carries implications about the state of consciousness, which is largely unknowable. More accurate might have been “minimally responsive state,” which seems more descriptive of the behavior that is observed, but for the present we are stuck with the term in the interests of cross-discipline agreement.

The condition covers a wide territory in that at the boundary with VS, patients may emit only a very occasional and inconsistent (non-reflexive) response to some stimulus. As an indication of the difficulty of making the diagnosis in some patients, a specialist team with which I am associated considers that it requires 3 months of meticulous, systematic

daily observations before a valid diagnosis can be made. At the upper boundary of MCS the condition merges into a more normal, although still highly impaired, state in which there is reasonably consistent responsiveness to command and some limited communication on the part of the patient.

The neuropsychologist clearly has a role in MCS, and it may assist in giving a “feel” for the state of patients in MCS to describe the “lowest” level of intelligent responsiveness that neuropsychologists can detect. In the CAVE (Cognitive Assessment by Visual Election), two stimuli are held up to the left and right in front of the patient, who is instructed to “Look at _____,” one of the stimuli. The stimuli may be two patches of color, two numerals, two letters, two small objects such as a toy car and a toy cow, line drawings of these objects, or the names of the objects. If the patient is reliably, according to a statistical definition, able to shift his or her gaze toward the nominated object then there is evidence for comprehension of the instruction and appropriate behavior in response. Of course, the patient must be able to hear the instruction, and have sufficient vision to discriminate the stimuli. Whether a patient has the required sensory functions is not always easy to determine, and failure on the task is not necessarily evidence of a failure of cognition, but this is about the simplest task that has yet been devised to assess patients in MCS.

The pathology of coma, VS, and MCS is not fully understood, but it is almost universally the case that there is widespread failure of the cerebral cortex to function. In enduring states of VS there is widespread, general damage to the cortex. Some have considered that extensive damage to the thalamus, as the principal organizing center of information flowing to, and away from, the cortex, may be the more critical aspect, but whether or not this is the case, the result is a globally malfunctioning cortex.

Locked-in Syndrome

Locked-in syndrome (LIS) is behaviorally similar to VS, but is fundamentally different both in its pathology and in the prospects for successful rehabilitation. The patient with LIS is almost completely unable to make any voluntary or involuntary movement (although some reflex movements will again be retained) and this includes movements of the face and head, and so precludes eye movements and speech. In many patients, one simple movement may be retained, most commonly vertical eye movements or voluntary blinking, rarely a flicker of muscle movement elsewhere in the body. This single retained movement is the key to rehabilitation of these patients, who otherwise have no way of control-

ling, or communicating with, the world. They are completely dependent for their care, and appear in almost every respect to be in VS.

However, in LIS, the cortex has not been extensively damaged, and may be functioning quite normally, but has been disconnected from the body, most commonly by a widespread stroke in the brain stem. As a consequence the cortex may be supporting a normal mental life with perceptions, thoughts, feelings, and internal language, but a mind that cannot exert any control over the body or communicate with the outside world. Hence the term “locked-in syndrome” and what, for many, is one of the most frightening prospects of what may befall a person.

It is for obvious reasons that differential diagnosis between VS and LIS is so important, for in LIS, with expert assistance, that single retained voluntary movement can be used to provide a means of artificial communication, often supported by computer-based devices, and some control over the world can be exercised through assistive technology. I know of a patient, misdiagnosed as in VS for many years and regarded as unaware, who came to our unit, was rapidly diagnosed as in LIS (with no credit to me), and within 2 weeks was writing letters to his wife. This seems a good outcome. Such a dramatic transformation is rare, but with skilled rehabilitation from physiotherapists, occupational therapists, biomedical engineers, speech and language therapists, and, of course, neuropsychologists, supported by the broader neurorehabilitation team, the majority of LIS patients can regain useful function through artificial means.

An eloquent and moving account of the experience of being in LIS is to be found in *The Diving Bell and the Butterfly*, written by Jean-Dominique Bauby (1997), previously editor of a well-known fashion magazine, after suffering this condition and being provided with the technology that enabled him to write the book. A successful outcome, of course, partially depends on there being no other coincidental areas of damage to the patient’s brain, but where this has not occurred the patient’s neuropsychological functions can be demonstrated to be almost entirely intact, as I can also testify from very challenging games of Scrabble played with patients in LIS.

A useful overview and review of the conditions of coma, VS, MCS, and LIS is provided in Table 8.2, which is based on the recommendations of the RCS report referred to earlier (Royal College of Surgeons, 2003).

Rehabilitation

Rehabilitation of profound brain injury, other than through assistive technology in LIS, remains a significant challenge. There is frankly

TABLE 8.2. Overview of Coma, VS, MCS, and LIS

| Condition | Vegetative state | Minimally conscious state | Locked-in syndrome | Coma |
|-----------------------------|---|---|---|---|
| Awareness | Absent | Present | Present | Absent |
| Sleep-wake cycle | Present | Present | Present | Absent |
| Response to noxious stimuli | +/- | Present | Present (in eyes only) | +/- |
| Glasgow Coma Scale score | E4, M1-4, V1-2 | E4, M1-5, V1-4 | E4, M1, V1 | E1-2, M1-4, V1-2 |
| Motor function | No purposeful movement | Some consistent or inconsistent verbal or purposeful motor behavior | Volitional vertical eye movements or eyeblink preserved | No purposeful movement |
| Respiratory function | Typically preserved | Typically preserved | Typically preserved | Variable |
| EEG activity | Typically slow wave activity | Insufficient data | Typically normal | Typically slow wave activity |
| Cerebral metabolism (PET) | Severely reduced | Insufficient data | Mildly reduced | Moderately to severely reduced |
| Prognosis | Variable: if permanent, continued vegetative state or death | Variable | Depends on cause but full recovery unlikely | Recovery, vegetative state, or death within weeks |

Note. Based on Royal College of Physicians (2003).

nothing, at present, that has been demonstrated to promote emergence from VS, or improvement in the level of responsiveness in MCS.

A decade or so ago there was still enthusiasm for the benefits of sensory stimulation. Initially, this was for multimodal and complex stimulation, but when the evidence did not support the efficacy of this approach, simpler stimulation in a single modality was the approach adopted (Wilson, Powell, Elliot, & Thwaites, 1993). The modality selected was preferentially one in which some limited responsiveness might already be evident. However, these approaches have now largely been abandoned as of limited value, although the idea persists that stimuli familiar to the patient, or those that could elicit an emotional response (pictures of family, favorite music, video of the football team the patient supports) might somehow “awaken” the patient. There is no scientific support for this idea.

There have, however, been advances in the assessment and monitoring of these states and, in particular, the SMART (Sensory Modality Assessment and Rehabilitation Technique; Gill-Thwaites & Munday, 1999) provides a highly systematic procedure for the regular appraisal of any responses that the patient may emit. In MCS and through the early stages of recovery the WHIM (Wessex Head Injury Matrix; Shiel, Wilson, McLellan, Horn, & Watson, 2000) also provides a coherent system for recording the behaviors that the patient exhibits.

One of the reasons why states of low awareness may be so difficult to treat is that it is not only cognitive function that is disordered. In most neuropsychological rehabilitation, we are dealing with relatively specific deficits of cognitive processes in an individual who is, more or less, normally aroused and adequately motivated. Contemporary psychology is relatively successful at understanding cognition, and to a lesser degree emotion, but it has seriously neglected motivation as a topic. A traditional division of the subject matter of psychology was cognition, affect, and conation or “will.” We are very good at describing *how* people do things, but rather poor at understanding *why* they do these things. The problem with most patients in states of low awareness is that they emit so little behavior. They initiate almost no spontaneous behavior, and appear inadequately motivated or insufficiently aroused to emit more than the little they may do. I suspect that until we have a more adequate psychology of motivation, in contemporary terms, we will make little further progress in the rehabilitation of these disorders.

Ethical Issues

These patients, particularly those in VS, raise very difficult ethical, moral, and social issues. The central issue is whether, in a patient who

has been in VS for many years, who shows no signs of awareness or recovery, life should be prolonged. The difficulty is that the VS state is compatible with life, given nutrition and hydration and basic physical care. This issue is not like that of the artificially ventilated patient on a life support system; it is not a matter of whether to “turn the patient off.”

The issue affects not only the person in VS, if an unaware and unresponsive person can be so affected, but also family members who face the prospect of perhaps years of caring for, or visiting, a loved one who is in every way absent and lost apart from the presence of their physical body. A further concern may be the cost of caring for an individual in VS when scarce health care resources might be better dedicated to hip replacements or eye surgery.

The reaction to this issue has been rather different in Britain and in the United States. In Britain relatives have no legal influence in the matter, and the legal issue has been whether it would be lawful for the medical consultant to withdraw nutrition and hydration and so permit the patient to die, although in all cases at the instigation of the relatives. There have been a number of such cases, although each case must be considered on its individual merits by the High Court. In each case the individual has been in prolonged VS and the prospects for any recovery have been considered effectively nil.

In the United States relatives have legal rights, and nutrition and hydration have been withdrawn in cases where there has been clear agreement among the relatives and medical professionals that this was the most appropriate course of action. The courts have been less involved, although they have been required to arbitrate among relatives holding different views about the course to be taken.

If I may be permitted a personal view in what is a very complex issue, I think it a sign of a civilized society that we respect and maintain life, even when the prospects for recovery are so poor. To draw a line between those who have no prospects and who may be “allowed to die” and those who are “worth keeping alive” is a dangerous precedent and could lead to a slippery slope that we might all agree would be better avoided.

FURTHER READING

Further reading in this area tends to be written for patients and their families, and is therefore insufficiently technical, or else is written for specialist professionals and tends to be rather too detailed for students. However, here are some technical texts that may be useful for further reading:

Multiple Sclerosis

- Brassington, J. C., & Marsh, N. V. Neuropsychological Aspects of Multiple Sclerosis, *Neuropsychological Review*, 8 (1998), 43–77.
- Cook, S.D. (Ed.). *Handbook of Multiple Sclerosis* (Third edition, New York: Dekker, 2001).
- Feinstein, A. *The Clinical Neuropsychiatry of Multiple Sclerosis* (Cambridge, UK: Cambridge University Press, 1999). A very useful and accessible review.
- Freedman, M. *Multiple Sclerosis and Demyelinating Diseases* (Philadelphia: Lippincott Williams & Wilkins, 2005).

Parkinson's Disease

- Ebadi, M., & Pfeiffer, R. F. (Eds.). *Parkinson's Disease* (Boca Raton, FL: CRC Press, 2005).
- Pfeiffer, R. F., & Bodis-Wollner, I. (Eds.). *Parkinson's Disease and Nonmotor Dysfunction* (Totowa, NJ: Humana Press, 2004).

Huntington's Disease

- Bates, G., Harper, P., & Jones, L. (Eds.). *Huntington's Disease* (Third edition, Oxford, UK: Oxford University Press, 2002).
- Folstein, S. E. *Huntington's Disease: A Disorder of Families* (Baltimore: Johns Hopkins University Press, 1989).
- Knowles, J. *Huntington's Disease* (New York: Rosen Publishing Group, 2006).
- Wexler, A. *Mapping Fate* (Berkeley: University of California Press, 1995). A personal account of the search for the gene responsible for Huntington's disease.

Profound Brain Injury

- Beaumont, J. G., & Kenealy, P. M. The Incidence and Prevalence of the Vegetative and Minimally Conscious States, in M. R. Coleman, ed., *The Assessment and Rehabilitation of Vegetative and Minimally Conscious Patients* (Hove, UK: Psychology Press, 2005).
- Dolce, G., & Sazbon, L. (Eds.). *The Post-Traumatic Vegetative State* (Stuttgart, Germany: Thieme, 2002).
- Jennett, B. *The Vegetative State: Medical Facts, Ethical and Legal Dilemmas* (Cambridge, UK: Cambridge University Press, 2002).

Locked-In Syndrome

- Doble, J. E., Haig, A. J., Anderson, C., & Katz, R. Impairment, Activity, Participation, Life Satisfaction, and Survival in Persons with Locked-In Syndrome

- for Over a Decade: Follow-Up on a Previously Reported Study, *Journal of Head Trauma Rehabilitation*, 18 (2003), 435–444.
- Smith, E., & Delargy, M. Locked-In Syndrome, *British Medical Journal*, 330 (2005), 406–409.

REFERENCES

- Alexander, G. E., Crutcher, M. D., & DeLong, M. R. Basal Ganglia-Thalamocortical Circuits: Parallel Substrates for Motor, Oculomotor, “Prefrontal” and “Limbic” Functions, in H. B. M. Uylings, C. G. Van Eden, J. P. C. De Bruin, M. A. Corner, & M. G. P. Feenstra, eds., *The Prefrontal Cortex: Its Structure, Function and Pathology* (Amsterdam: Elsevier, 1990).
- Andrews, K., Murphy, L., Munday, R., & Littlewood, C. Misdiagnosis of the Vegetative State: Retrospective Study in a Rehabilitation Unit, *British Medical Journal*, 313 (1996), 13–16.
- Arnett, P. A., Higginson, C. H., & Randolph, J. R. Depression in Multiple Sclerosis: Relationship to Planning Ability, *Journal of the International Neuropsychological Society*, 7 (2001), 665–674.
- Bauby, J.-D. *The Diving-Bell and the Butterfly* (London: Fourth Estate, 1997).
- Boller, F., & Muggia, S. Non-Alzheimer Dementias, in G. Denes & L. Pizzamiglio, eds., *Handbook of Clinical and Experimental Neuropsychology* (Hove, UK: Psychology Press, 1999).
- Brown, R. G., & Marsden, C. D. Cognitive Function in Parkinson’s Disease: From Description to Theory, *Trends in Neuroscience*, 13 (1990), 21–29.
- Butters, N., Wolfe, J., Martone, M., Granholm, E., & Cermak, L. S. Memory Disorders Associated with Huntington’s Disease: Verbal Recall, Verbal Recognition and Procedural Memory, *Neuropsychologia*, 23 (1985), 729–743.
- Caine, E. D., & Shoulson, I. Psychiatric Syndromes in Huntington’s Disease, *American Journal of Psychiatry*, 140 (1983), 728–733.
- Coleman, M. R., Rodd, J. M., Davis, M. H., Johnsrude, I. S., Menon, D. K., Pickard, J. D., et al. Do Vegetative Patients Retain Aspects of Language Comprehension?, *Brain*, 130 (2007), 2494–2507.
- DeLuca, J., Barberi-Berger, S., & Johnson, S. K. The Nature of Memory Impairment in Multiple Sclerosis: Acquisition versus Retrieval, *Journal of Experimental and Clinical Neuropsychology*, 2 (1994), 183–189.
- Giacino, J. T., Ashwal, S., Childs, N., Cranford, R., Jennett, B., Katz, D. I., et al. The Minimally Conscious State: Definition and Diagnostic Criteria, *Neurology*, 58 (2002), 349–353.
- Gill-Thwaites, H., & Munday, R. The Sensory Modality Assessment and Rehabilitation Technique (SMART): A Comprehensive and Integrated Assessment and Treatment Protocol for the Vegetative State and Minimally Responsive Patient, *Neuropsychological Rehabilitation*, 9 (1999), 305–320.
- Litvan, I., Mohr, E., Williams, J., Gomez, C., & Chase, T. N. Differential Memory and Executive Functions in Demented Patients with Parkinson’s and

- Alzheimer's Disease, *Journal of Neurology, Neurosurgery and Psychiatry*, 54 (1991), 25–29.
- Obeso, J. A., Olanow, C. W., & Nutt, J. T. Basal Ganglia, Parkinson's Disease and Levodopa Therapy, *Trends in Neuroscience*, 23 (2000), S1–S126.
- Owen, A. M., Coleman, M. R., Boly, M., Davis, M. H., Laureys, S., & Pickard, J. D. Using Functional Magnetic Resonance Imaging to Detect Covert Awareness in the Vegetative State, *Archives of Neurology*, 64 (2007), 1098–1102.
- Pillon, B., Dubois, B., Ploska, A., & Agid, Y. Severity and Specificity of Cognitive Impairment in Alzheimer's, Huntington's and Parkinson's Diseases and Progressive Supranuclear Palsy, *Neurology*, 41 (1991), 634–643.
- Poldroll, K., Caspary, P., Lange, H. W., & Noth, J. Language Functions in Huntington's Disease, *Brain*, 111 (1988), 1475–1503.
- Rao, S. M., Leo, G. J., Bernardin, L., & Unverzagt, F. Cognitive Function in Multiple Sclerosis. I. Frequency, Patterns and Prediction, *Neurology*, 41 (1991), 685–691.
- Royal College of Physicians, *The Vegetative State: Guidance on Diagnosis and Management* (London: Royal College of Physicians, 2003).
- Shiel, A., Wilson, B. A., McLellan, L., Horn, S., & Watson, M. *Wessex Head Injury Matrix—WHIM* (London: Harcourt Assessment, 2000).
- Wilson, S. L., Powell, G. E., Elliot, K., & Thwaites, H. Evaluation of Sensory Stimulation as a Treatment for Prolonged Coma: Seven Single Experimental Case Studies, *Neuropsychological Rehabilitation*, 3 (1993), 191–202.
- Zakzanis, K. K. Distinct Neuropsychological Profiles in Multiple Sclerosis Subtypes, *Archives of Clinical Neuropsychology*, 15 (2000), 115–136.
- Zakzanis, K. K., Leach, L., & Kaplan, E. *Neuropsychological Differential Diagnosis* (Lisse, The Netherlands: Swets & Zeitlinger, 1999).

The Subcortex and Psychosurgery

It is easy to forget when reading texts on human neuropsychology that most cerebral lesions extend more or less radically into the subcortical tissue. As the conditions discussed in Chapter 8 demonstrate, lesions below the level of the cortex may have neuropsychological consequences. However, lesions of the subcortex alone are relatively rare in humans, except in the case of certain neoplasms (tumors), when surgery normally involves disturbance of the cortex. As traumatic damage to subcortical tissue is likely to have fatal consequences, or to have such radical effects on behavior that study of the higher aspects of human abilities is precluded, there has been relatively little study of such damage in humans, and much of our knowledge about the functions of subcortical structures comes from animal studies. However, what clinical evidence is available suggests that generalizations from animals to man with respect to lower functions of the subcortex are not likely to be grossly inaccurate. Information about these functions is to be found in most textbooks on physiological psychology, and therefore only a general summary is given here.

First, subcortical lesions may interfere with general activating systems—for example the ascending reticular activating system (ARAS) in the brain stem (see p. 30)—and thereby affect the levels of consciousness, attention, and awareness. Such changes may well be reflected in the responsiveness of bodily reflexes, and this is one reason why reflexes are carefully tested by the neurologist. Lesions in the lower brain stem may

lead to sudden attacks of deep unconsciousness, and to changes in the pulse and the pattern of breathing. "Cheyne-Stokes breathing," in which the pattern is slow, deep, and perhaps periodic, is characteristic of some coma states. By contrast, lesions of the upper brain stem and around the thalamus produce states more like sleep. Sleep may also be short and fitful.

Subcortical lesions can also interrupt the afferent (toward) and efferent (away from) pathways as they pass to and from the cerebral cortex, so that lesions involving the internal capsule or the pyramidal tracts (the major descending pathway from the cortex to the lower brain stem and spinal cord) may have primary effects on somatosensory and motor performance, even though the cortex is not itself involved.

Midbrain lesions in the area of the thalamus, besides leading to disturbances of consciousness, can result in motor disabilities, such as resting tremor, choreiform movements, and the uncontrolled movements of dystonia, as discussed in Chapter 8. Parkinsonian symptoms are typical of lesions in this area. There is some evidence that thalamic lesions may also result in changes in verbal performance, particularly verbal fluency, and in face recognition and face matching, and that these deficits may vary with the laterality of the lesion (Vilkki, 1978). Stimulation of the region of the pulvinar through chronically implanted electrodes may produce transient dysphasia, memory loss, and poor visual discrimination and recognition (Fedio & Van Buren, 1975). It has even been suggested that the absence of the *massa intermedia*, which joins the two thalami in some but not all individuals, may be associated with higher intelligence in males (Lansdell & Clayton-Davies, 1972). Not all these functional relationships can, however, be regarded as well established.

Hypothalamic lesions have effects (which animal studies would lead one to expect) on basic drives, such as eating and drinking, sleeping and wakefulness. There are often, in addition, signs of dysfunction of the autonomic nervous system. Changes in sexual behavior may also be seen, and the impotence that sometimes afflicts those who have pursued a long career in boxing is sometimes attributed to damage to the hypothalamic region resulting from repeated blows to the head.

Lesions of the basal ganglia, as noted in Chapter 4, are closely linked with the cortical functions of the temporal lobe, including memory, aggression, and the appropriate direction of sexual behavior. Finally, lesions of the cerebellum produce the effects that might be expected on motor coordination, and on balance and position sense. Patients with damage to the cerebellum show tremor and jerkiness when executing intentional movements, and may walk in a rather drunken fashion with a very broad-based gait. It has been argued that there are also significant nonmotor functions of the cerebellum that should not be neglected

(Watson, 1978), but these are far less prominent than the classical motor functions in the deficits shown by patients.

These more basic subcortical functions are generally of less interest to human neuropsychologists, although they are naturally of considerable importance in assessing the overall neurological and psychological status of a brain damaged patient, and the rest of this chapter is devoted to the current debate about “surgery on the mind”: the procedures and outcome of neurosurgery and psychosurgery. Insofar as is possible, a balanced summary of the scientific arguments for and against these treatments will be presented.

NEUROSURGERY

A clear distinction is usually made between *neurosurgery* and *psychosurgery*. Neurosurgery is regarded as uncontroversial and acceptable, while psychosurgery is the subject of intense debate on scientific, ethical, and legal grounds. When closely examined, however, the distinction between the two is not so clear. It is sometimes suggested that the basis of the distinction is that in neurosurgery pathological diseased tissue is being removed or some gross physiological abnormality is being corrected, while in psychosurgery tissue that is not grossly abnormal (and is therefore “healthy” and “normal”) is being destroyed. This is not strictly accurate, because some neurosurgical procedures, for the relief of intractable pain or the correction of spastic limbs, for example, involve the destruction of healthy tissue. An alternative basis for the distinction is that neurosurgery is for physical conditions, while psychosurgery attempts to change mental symptoms. However, setting aside the question of how we might distinguish “mental” from “physical” conditions, the symptoms of some tumors may only be apparent in cognitive, and therefore mental, changes. Pain is a purely mental phenomenon.

The distinction in reality is probably a false one, based on what is generally accepted as opposed to what is controversial. It may be helpful not to regard psychosurgery as in any way fundamentally different from routine neurosurgery, and indeed some writers (proponents of psychosurgery) have suggested the substitution of the term “psychiatric surgery.”

Whatever view you reach on psychosurgery, there are a large number of routine surgical procedures, mostly interventions in clearly pathological states, that are not in question. Surgery to remove neoplasms, to repair cerebrovascular accidents, to ameliorate spasticity and other movement disorders, to correct hydrocephalus, to relieve chronic pain, to correct neuroendocrine disorders, or remove the irritant cause of focal

epilepsy is widely practiced. Many of these conditions are life threatening, all cause significant distress and suffering, and there is almost no prospect of spontaneous recovery or alternative effective treatment. The ability of the patient to consent to treatment is usually not in doubt. Many of the procedures are technically difficult and hazardous, but their justification is rarely in question, and in considering psychosurgery we should not neglect the significant, and in some ways more important, area of general functional neurosurgery, as the examples given below may illustrate.

It should also be pointed out here that the frequent assumption that drug treatment is a less powerful treatment than surgery, because of the more dramatic nature of the latter, is in some ways a misconception. Many psychotropic drugs are extremely powerful and yet not very selective in their effects, and many bring unpleasant and unwanted effects that must be corrected by further medication. It is therefore inaccurate to contrast drugs with surgery on the assumption that the former are inherently safe, gentle, and reversible in their effect, while the latter is crude, dangerous, and irreversible. It often surprises students to learn that in cases where there is a history of coronary disease, and in older adults, it is often considered safer to give electroconvulsive therapy than to administer certain antidepressant drugs. Surgery is relatively hazardous, and irreversible, but the drugs are also extremely potent, and may also have irreversible effects.

Parkinson's Disease

One particular application of the development of stereotactic surgery has, historically, been used in the treatment of Parkinson's disease, which was discussed in Chapter 8. Stereotactic surgery involves the insertion of probes under general anesthetic by reference to landmarks on the skull, brain maps, and images of the patient's brain. The insertion can be guided by further imaging studies. Once the probes are inserted, the patient can be brought back to consciousness and the probes used to study the functional nature of the site of the probe before irreversible lesions are deliberately created by high frequency coagulation, freezing, or radiation.

As Parkinson's disease involves the dysfunction of sites in the basal ganglia, stereotactic lesions may be placed in this area, including in the ventrolateral nucleus of the thalamus, the globus pallidus, and parts of the internal capsule, among other structures (Mawdesley, 1975). When a number of the treated brains became available for postmortem examination, the exact location of these lesions proved to be rather less accurate than had been expected, but there were nevertheless therapeutic gains.

In about nine out of ten patients, the primary tremor and rigidity that so disable Parkinson's disease patients were abolished, with a concomitant increase in motor proficiency. The surgery has been less successful, however, at improving the impoverishment of motor behavior and the difficulties in the initiation of motor acts that many of these patients also have. Disequilibrium and akinesia were also often not significantly improved. Nevertheless, mortality from the procedure is low, and despite some troublesome side effects in speech production for a number of patients, daily function could be considerably improved. A large number of these operations were performed in the 1960s and into the 1970s, but the advent of new drugs for the treatment of this disorder, notably levodopa, among a variety of others (see Pincus & Tucker, 1978), resulted in a decline in popularity of the operation. Because its results in everyday function were not more impressive (Funkiewiez et al., 2006; Pereira & Aziz, 2006), and certain cognitive functions were affected, albeit not seriously, drug treatment is now usually preferred to surgery. There has, however, been a more recent development of deep brain stimulation for Parkinson's disease with promising results (Parsons, Rogers, Braaten, Woods, & Troster, 2006; Schupbach et al., 2006; Weaver, Follett, Hur, Ippolito, & Stern, 2005), and also considerable interest in cell transplants (Winkler, Kirik, & Bjorklund, 2005). Stereotactic surgery and related techniques for Parkinsonism remain a good example of a discrete, controlled, and successful treatment for a condition with neuropsychological aspects.

Focal Epilepsy

Certain forms of epilepsy, where there is an identified "focus" in the cortex that is considered to trigger the epileptic seizures, can be treated by surgical removal of the epileptogenic tissue (Rasmussen, 1979; Sweet, 1977). The results in terms of relief from seizures caused by lesions other than tumors are in general good, with typically about two-thirds of the patients showing a marked reduction in seizures, including about a third who become immediately or eventually seizure free, and the remaining third generally showing a moderate or small reduction in seizure frequency. There is a very low operative mortality and few reports of significant unwanted effects. The figures are naturally less good when the lesion is a tumor, largely because of the tendency of tumors to recur. These results are impressive (particularly as they come from a large number of patients: 1,277 in the Canadian series), and doubts about the efficacy of the treatment must be set against the side effects of anti-convulsive medication. Drugs administered for epilepsy can be seriously

disabling, particularly in retarding cognitive function, which adds to the attraction of the neurosurgical alternative in appropriate cases.

Another particular form of surgery for epilepsy is anterior temporal lobectomy. (In *lobectomy* the lobe, or part of it, is removed; in *lobotomy* the connections to the lobe are severed; in *leukotomy* more discrete interruptions in fiber tracts are made.) The operation is indicated when pathological tissue has been demonstrated in the anterior temporal lobe. The use of the Wada technique to establish speech lateralization in connection with this procedure has already been mentioned (see p. 86). The results on seizure frequency are generally good. Of particular interest, however, are the deficits in cognitive performance that follow temporal removals, which were discussed in Chapter 4 (see also Milner, 1975). The operation has been used successfully with children and adolescents, and has been considered by some to be particularly valuable where abnormalities of the electrical activity of the temporal lobe are associated with violent and aggressive outbursts (Sadler, 2006; Sanyal et al., 2005; Shimazu, Kawai, Sunaga, Sugano, & Yamada, 2006; Smith, VanderGriff, & Fountas, 2004).

PSYCHOSURGERY

Prefrontal Leukotomy

Recent debate over psychosurgery has been strongly influenced by the history of the leukotomy operation. Although there were earlier experiments, it was Moniz who introduced the operation in 1936 for serious psychiatric illnesses, including schizophrenia. It was soon adapted into its most common form, the “standard leukotomy,” by Freeman and Watts, and popularly known as “frontal lobotomy.” In this form, burr holes were drilled at each side of the head, and a cutting instrument inserted and swiveled up and down in a broad arc, so severing a large number of the connections coming up into the frontal lobe (see Figure 9.1). There were several refinements of this operation, including “orbital undercutting,” developed by Scoville in 1948 (Figure 9.2), but all used relatively crude techniques with the object of disconnecting centers in the frontal lobe from subcortical circuits in the limbic system. An enormous number of these operations were performed, with estimates of up to 100,000 worldwide, and an extensive account of the history of the operation will be found in Valenstein (1980).

As with many treatments in psychiatry, it was taken up with fervent enthusiasm, and a proper evaluation of its effectiveness was too long delayed (see Clare, 1980, for a discussion of this and other shortcomings

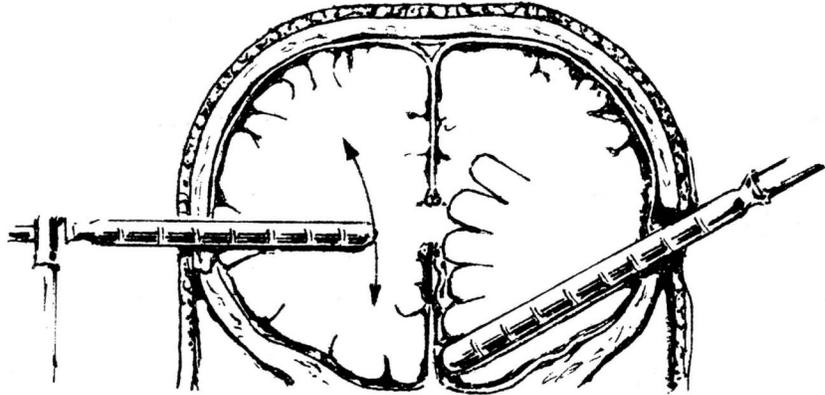


FIGURE 9.1. The leukotomy procedure developed by Freeman and Watts. (After Freeman and Watts, 1963.)

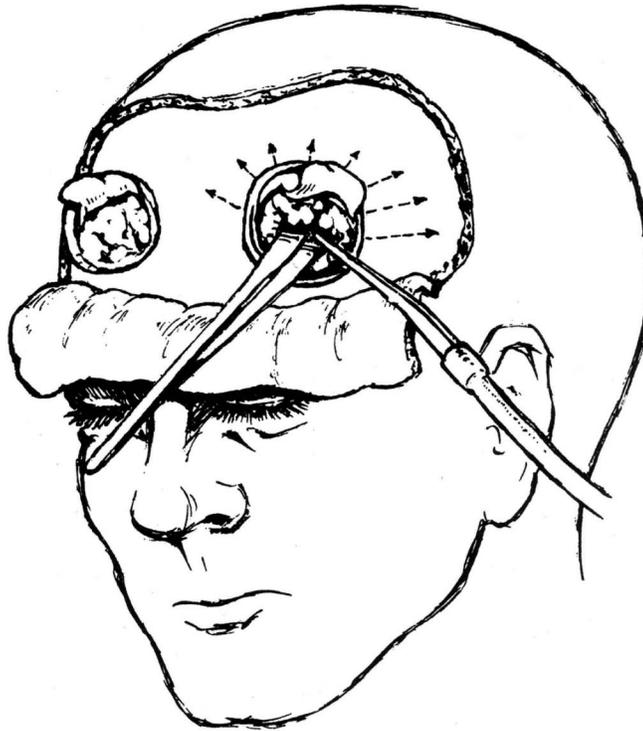


FIGURE 9.2. The orbital undercutting operation developed by Scoville. (After Asenjo, 1963.)

in the history of psychiatry). While there were earlier reviews, the first major study was that of Tooth and Newton (1961), who examined 10,365 cases in the United Kingdom in the years 1942–1952. Forty-six percent were discharged from hospital (although this does not mean fully recovered), but 4% died, 3% had major undesired effects, including chronic epilepsy, and 2% became significantly worse. It should be noted that the assessments of outcome were often not independent or objective, and the criteria for improvement were fairly minimal. On the other hand, we should recognize that many of the patients would now be regarded as most unsuitable for this kind of operation, and that many had an extremely long history of psychiatric illness (41% had been ill for more than 6 years).

More recent assessments have not been more encouraging about the success of the operation. Post, Linford-Rees, and Schurr (1968), examining patients who had undergone a modified leukotomy operation, found undesirable permanent side effects in 59% of the patients, and troublesome symptoms in a further 21%. Such symptoms included various degrees of epilepsy, incontinence of urine and feces, and motor impairments. Nevertheless, it was concluded that in 40% of patients the operation initiated lasting changes for the better, particularly if the illness was chronic depression or anxiety rather than schizophrenia.

Robin and Macdonald (1975) also reported on a series of studies, both retrospective and prospective, of the effects of different leukotomy operations. They attempted to match control subjects within their studies and found no better prospect of discharge from hospital after leukotomy for either schizophrenia or depression, and inconclusive results for severe phobic and obsessional disorders. Although there were some differences in improvement within the institutional setting, drugs were as effective as the surgical operations. Surgery might in some cases affect certain symptoms but did not result in general clinical improvement. Postoperative deaths were still found to be rather high (2%), and the rate of epilepsy was as high as 5% over a long follow-up period. They also reported intellectual deficits in the surgical patients.

There have been many other studies, but there is now general agreement, even among the proponents of psychosurgery, that leukotomy operations at best fell short of success, and at worst are a scandal in the history of psychiatry. If they were to be successful (as some related operations now are), they should preferably have been undertaken on chronically depressed and anxious patients, and certainly not on schizophrenic patients. It should, however, be remembered that the operation was introduced in the 1950s, in the period before the revolution brought about by psychotropic drugs, when psychiatrists were desperate to prevent the decline of psychotic patients into a chronic retarded state, a

progress they were largely powerless to halt. Nevertheless, despite the apparent scientific rationale that was used to justify the operation, it was not well founded theoretically, the operative procedure was primitive and crude, and it was applied to large numbers of unsuitable patients. The history of leukotomy should rightly teach us a lesson about the care that must attend the introduction of novel treatments, but we should also not allow the failure of these earlier techniques to cloud our judgment of modern psychosurgical techniques (Lerner, 2005).

Modern Psychosurgery

There are a variety of operative procedures used in modern psychosurgery, with certain operations being favored in certain centers, but they fall roughly into four groups.

The first of the contemporary procedures is *stereotactic subcaudate tractotomy*, which is essentially a much-refined version of the earlier frontal lobe operations. In devising it, Knight developed a method of stereotactically placing tiny ceramic rods containing radioactive yttrium at the site for the lesion, which is in the white matter beneath and in front of the caudate nucleus (below the septal region), extending into the frontal central white matter (see Figure 9.3 and Figure 2.6). The yttrium decays quite rapidly, as its half-life is only 62 hours, and leaves a lesion of about $25 \times 15 \times 5$ millimeters in the standard operation with six rods. Introducing the rods can be undertaken with relative accuracy

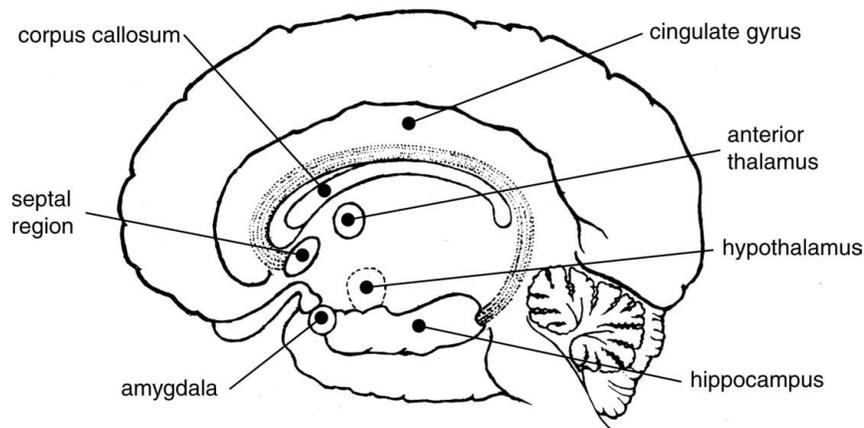


FIGURE 9.3. Some of the subcortical sites that are targets in psychosurgery.

and causes very little trauma, and the operation may be performed entirely under general anesthetic. Assessments of the effectiveness of this operation (Dalglish et al., 2004; Woerdeman, Willems, Noordemans, Berkelbach van der Sprenkel, & van Rijen, 2006) show that there is a “good response” in about 60% of severely depressed patients, and in about 50% of those with chronic anxiety or obsessive–compulsive disorder. Unintended effects on the personality were reported in about 5%, but these were by and large not considered “socially disabling.” The number of patients developing seizures was low, and there was only one fatality among the 418 cases reported. The clinical assessments of outcome are supported by some objective psychological test data, and there was a dramatic decline in the number of suicide attempts in comparable periods before and after the operation.

A second contemporary operation is *limbic leukotomy* and is very similar to subcaudate tractotomy (Dean, 2002). The lesions are usually created by freezing or electrocoagulation, but are placed by stereotactic means at two sites. One site is in the subcaudate area, as in the tractotomy, although the lesion is smaller; the other is in the cingulum bundle. This operation has been performed less commonly than the tractotomy, but the results suggest that the effectiveness of the two operations is roughly comparable. It has been suggested that limbic leukotomy is more effective for obsessional neurosis and less good in anxiety states, although there is some controversy over this point. Unlike the tractotomy, however, it has been performed in highly selected cases of schizophrenia, with a surprisingly good outcome.

The third group of operations is aimed at the *cingulate region* alone. In a prospective study of 85 patients (Corkin, 1980), which included extensive follow-up data and careful objective assessment, no lasting neurological or behavioral deficits were found. There was therapeutic improvement in many although not all of the patients, the best outcome being in chronic pain or depression. Some of the earlier studies had suggested that this operation was less safe than the others just mentioned, but the latest developments of the technique seem to have avoided the major side effects seen earlier (Kim et al., 2003).

Most controversial is the fourth group of operations, aimed at the *amygdala* or the *posterior hypothalamus*. This operation is considered for cases of severe, pathological, and uncontrollable aggressiveness, particularly if there are clear signs of a neurological abnormality, usually evidenced by abnormal electrical activity of the brain. Much of the opposition to this operation springs from its earlier use in the United States with violent offenders, and the resulting argument that a behavioral rather than a neurological disorder was being treated by the operation. This resulted in very strict legal controls on the operation in the

United States, although it is performed in at least one center in the United Kingdom. It appears effective in reducing emotional excitability and improving the patient's social adaptation without significant effects on cognitive abilities.

There are other operative procedures, including methods by which arrays of electrodes are implanted in frontal lobe sites, so that small serial lesions can be produced over a period of time while the therapeutic progress is being monitored. The variety of operations that have been performed in the United States is also wider than this account perhaps suggests, but the above four groups contain the essential elements to be found in any of these procedures.

Some general points should be made about the modern operations. The first is that they are performed only as a "treatment of last resort," that is, after all other treatments have been tried and failed. Some critics of psychosurgery have raised the quite reasonable point that if it is believed to be effective, why should it be reserved in this way? While a valid point, it is a little unfair, for the caution exercised by most psychosurgeons is urged upon them by the operations' opponents. However, it does mean that the patients who undergo the operations have severe and intractable conditions, and have generally been seriously ill for a protracted period. That the outcome statistics are as good as they are is perhaps more remarkable in the light of this, given the severity of the illnesses being treated. Nevertheless, the cases are still highly selected as to their suitability for surgery, which tempers the force of this point.

The number of psychosurgical operations performed each year is, in any case, extremely small. In the early 1970s the number of operations in the United Kingdom was about 200–250 each year, as against about 400 in the United States, the operations therefore being relatively more common in the United Kingdom. Since this time there has been a decline in numbers, with about 150 each year in the United Kingdom by 1976, and a comparable decline in the United States. Most contemporary operations are performed in a small number of units that specialize in particular operations. A statement that clearly lays out the indications for the treatments practiced at certain of these centers was published by Bartlett, Bridges, and Kelly (1981).

Taking an overview of these modern operations, it seems that objective (although not always independent) assessments show them to be effective for chronic depression, anxiety, neurosis, obsessionality, somatic complaints, tension, and phobias. Undesirable cognitive effects are only rarely manifested, and tend to be in learning and memory or language. These deficits were most commonly associated with thalamic operations, which are now much less frequently performed. The psychiatric and neurological ratings of improvement are generally favorable, if

schizophrenia and drug addiction are excepted, and the rate of postoperative complications has declined steadily over the years, so that it is now acceptably low. The suggestion that psychosurgery merely produces overall indiscriminate emotional blunting and cognitive slowing, with the result that the patient becomes an incomplete and damaged person, is not really supported by any of the studies of the modern operations. One concern, however, is the extent of (scientifically) unreported psychosurgery, particularly in the United States, where as many as three out of four surgeons have not published their results in the literature (Valenstein, 1980). This is to be regretted.

There are undoubtedly ethical problems that attach to psychosurgery, particularly the problem as to whether a patient can really consent to any procedure the outcome of which he has no way of knowing in advance. As all of the patients have relatively severe psychiatric disorders, this problem is doubly difficult. However, it is also important to balance the distress experienced by these patients, and the very real risks from suicide in many cases (perhaps 15%), if they are untreated, against the limits of the effectiveness of the operations. There is also something in the criticism that these operations are still relatively crude and are not based on any clear scientific rationale. However, if they are treated as experimental medicine and employed with great caution, the information that we have suggests that the operations should continue to be used in highly selected cases, with careful independent assessment of outcome and full reporting of the results. What, of course, is needed is a sound controlled trial of psychosurgery, but for ethical and methodological reasons it is probably impossible to achieve. Patients cannot ethically be randomly assigned to treatment and no-treatment control groups; the treatment cannot be applied to a control group not possessing the normal indications for the therapy; it is very difficult to have an independent yet thorough assessment of postoperative state by an examiner who is "blind" as to whether the operation has been performed or not; and it is certainly impossible for the patient to remain ignorant about whether the treatment has been applied (as is the case with placebo controls for drug therapy). In the meantime, psychosurgery probably has a place in neuropsychiatry, while more is discovered about both the disorders and the full effects of the operations.

CONCLUSION

Almost all lesions of the brain have subcortical as well as cortical effects, although these have historically been much neglected by human neuropsychologists. This is, however, one area where comparative neuropsychy-

chology can contribute invaluable evidence from studies of animals that help us to understand the subcortical systems of man. The effects of major subcortical lesions are often so radical that the cognitive deficits are either extremely gross or unimportant in comparison with the major functional impairments of consciousness or basic drives.

In considering the subcortex, neurosurgery and psychosurgery are of some interest. There is much routine and uncontroversial neurosurgery practiced with effects upon behavior, often demanded by one of a number of life-threatening processes. Some examples have been given, however, where neurosurgery is not an emergency procedure, where it is aimed at behavioral deficits among others, and where the rationale and success of the methods have clearly justified their use.

Psychosurgical methods have also been described. While the historical operations fell far short of real success, the modern operations are by contrast well controlled and relatively successful. They raise serious ethical and scientific issues, but an unbiased appraisal of the evidence suggests that they should have a place in contemporary medicine, even if that place is under continual critical review.

FURTHER READING

There are many excellent texts on physiological psychology that review the functions of subcortical structures, but two that can be recommended are:

Carlson, N. R. *Physiology of Behavior* (Sixth edition, Boston: Pearson Education, 1997). This takes the reader to a more advanced level.

Wickens, A. *Foundations of Biopsychology* (Second edition, Harlow, UK: Pearson Education, 2005).

Books on neurosurgery tend to be rather technical, but interested students may find these useful:

Greenberg, M. S. *Handbook of Neurosurgery* (Sixth edition, New York: Thieme, 2005).

Haines, S. J., & Walters, B. C. *Evidence-Based Neurosurgery: An Introduction* (New York: Thieme, 2007).

More specifically addressed to psychosurgery are the following:

Clare, A. *Psychiatry in Dissent* (Second edition, London: Tavistock Publications, 1980). This volume also covers a range of controversial topics in psychiatry.

Pressman, J. D. *Last Resort: Psychosurgery and the Limits of Medicine* (Cambridge, UK: Cambridge University Press, 1998). A critical assessment of historical aspects.

- Rodgers, J. E. *Psychosurgery: Damaging the Brain to Save the Mind* (New York: HarperCollins, 1992).
- Valenstein, E. S. (Ed.). *The Psychosurgery Debate: Scientific, Legal and Ethical Perspectives* (San Francisco: Freeman, 1980). This is undoubtedly the best single book on psychosurgery. It contains excellent coverage of the historical and current scientific status of the various operations, and represents critical opinion fairly. It also includes discussion of the ethical and legal position (although primarily with respect to the United States), and an account of the reports submitted to the U.S. National Commission for the Protection of Human Subjects of Biomedical and Behavioral Research.

Students may also find useful a pair of papers that present an informative debate:

- Crossley, D., & Freeman, C. Should Neurosurgery for Mental Disorder Be Allowed to Die Out?: Against, *British Journal of Psychiatry*, 183 (2003), 196.
- Persaud, R. Should Neurosurgery for Mental Disorder Be Allowed to Die Out?: For, *British Journal of Psychiatry*, 183 (2003), 195–196.

REFERENCES

- Asenjo, A. *Neurosurgical Techniques* (Springfield, IL: Charles C. Thomas, 1950).
- Bartlett, J., Bridges, P., & Kelly, D. Contemporary Indications for Psychosurgery, *British Journal of Psychiatry*, 138 (1981), 507–511.
- Clare, A. *Psychiatry in Dissent* (Second edition, London: Tavistock Publications, 1980).
- Corkin, S. A Prospective Study of Cingulotomy, in E. S. Valenstein, ed., *The Psychosurgery Debate: Scientific, Legal and Ethical Perspectives* (San Francisco: Freeman, 1980).
- Dagleish, T., Yiend, J., Bramham, J., Teasdale, J. D., Ogilvie, A. D., Malhi, G., et al. Neuropsychological Processing Associated with Recovery from Depression after Subcaudate Tractotomy, *American Journal of Psychiatry*, 161 (2004), 1913–1916.
- Dean, C. E. Limbic Leucotomy in Self-Mutilation, *Journal of Clinical Psychiatry*, 63 (2002), 1181–1182.
- Fedio, P., & Van Buren, J. M. Memory and Perceptual Deficits During Electrical Stimulation in the Left and Right Thalamus and Parietal Subcortex, *Brain and Language*, 2 (1975), 78–100.
- Freeman, W., & Watts, J. W. *Psychosurgery in the Treatment of Mental Disorders and Intractable Pain* (Second edition, Springfield, IL: Charles C. Thomas, 1963).
- Funkiewiez, A., Ardouin, C., Cools, R., Krack, P., Batir, A., Chabardes, S., et al. Effects of Levodopa and Subthalamic Nucleus Stimulation on Cognitive

- and Affective Functioning in Parkinson's Disease, *Movement Disorders*, 10 (2006), 1656–1662.
- Kim, C. H., Chang, J. W., Koo, M. S., Kim, J. W., Suh, H. S., Park, I. H., et al. Anterior Cingulotomy for Refractory Obsessive-Compulsive Disorder, *Acta Psychiatrica Scandinavica*, 107 (2003), 283–290.
- Lansdell, H., & Clayton-Davies, J. Massa Intermedia: Possible Relation to Intelligence, *Neuropsychologia*, 10 (1972), 207–210.
- Lerner, B. H. Last-Ditch Medical Therapy—Revisiting Lobotomy, *New England Journal of Medicine*, 14 (2005), 119–121.
- Mawdesley, C. Parkinson's Disease, in W. B. Matthews, ed., *Recent Advances in Clinical Neurology*, No. 1 (Edinburgh, UK: Churchill-Livingstone, 1975).
- Milner, B. Psychological Aspects of Focal Epilepsy and Its Neurosurgical Management, in D. P. Purpura, J. K. Penry, & R. D. Walter, eds., *Advances in Neurology*, Vol. 8 (New York: Raven Press, 1975).
- Parsons, T. D., Rogers, S. A., Braaten, A. J., Woods, S. P., & Troster, A. I. Cognitive Sequelae of Subthalamic Nucleus Deep Brain Stimulation in Parkinson's Disease: A Meta-analysis, *Lancet: Neurology*, 5 (2006), 578–588.
- Pereira, E. A., & Aziz, T. Z. Surgical Insights into Parkinson's Disease, *Journal of the Royal Society of Medicine*, 99 (2006), 238–244.
- Pincus, J. H., & Tucker, G. J. *Behavioral Neurology* (Second edition, New York: Oxford University Press, 1978).
- Post, F., Linford-Rees, W., & Schurr, P. An Evaluation of Bimedial Leucotomy, *British Journal of Psychiatry*, 114 (1968), 1223–1246.
- Rasmussen, T. Cortical Resection for Medically Refractory Focal Epilepsy: Results, Lessons and Questions, in T. Rasmussen & R. Marino, eds., *Functional Neurosurgery* (New York: Raven Press, 1979).
- Robin, A., & Macdonald, D. *Lessons of Leucotomy* (London: Kimpton, 1975).
- Sadler, R. M. Failure of Surgery for Temporal Lobe Epilepsy: A Review of Selected Aspects, *Advances in Neurology*, 97 (2006), 97–104.
- Sanyal, S. K., Chandra, P. S., Gupta, S., Tripathi, M., Singh, V. P., Jain, S., et al. Memory and Intelligence Outcome Following Surgery for Intractable Temporal Lobe Epilepsy: Relationship to Seizure Outcome and Evaluation Using a Customized Neuropsychological Battery, *Epilepsy and Behaviour*, 6 (2005), 147–155.
- Schupbach, M., Gargiulo, M., Welter, M. L., Behar, C., Houeto, J. L., Maltete, D., et al. Neurosurgery in Parkinson's Disease: A Distressed Mind in a Repaired Body?, *Neurology*, 66 (2006), 1811–1816.
- Shimazu, H., Kawai, K., Sunaga, S., Sugano, H., & Yamada, T. Hippocampal Transection for Treatment of Left Temporal Lobe Epilepsy with Preservation of Verbal Memory, *Journal of Clinical Neuroscience*, 13 (2006), 322–328.
- Smith, J. R., VanderGriff, A., & Fountas, K. Temporal Lobotomy in the Surgical Management of Epilepsy: Technical Report, *Neurosurgery*, 54 (2004), 1531–1534.
- Sweet, W. H. (Ed.). *Neurosurgical Treatment in Psychiatry, Pain and Epilepsy* (Baltimore: University Park Press, 1977).
- Tooth, G. C., & Newton, M. P. Leucotomy in England and Wales 1942–1954,

- Reports on Public Health and Medical Subjects*, no. 104 (London: HMSO, 1961).
- Valenstein, E. S. (Ed.). *The Psychosurgery Debate: Scientific, Legal and Ethical Perspectives* (San Francisco: Freeman, 1980).
- Vilkki, J. Effects of Thalamic Lesions on Complex Perception and Memory, *Neuropsychologia*, 16 (1978), 427–437.
- Watson, P. J. Nonmotor Functions of the Cerebellum, *Psychological Bulletin*, 85 (1978), 944–967.
- Weaver, F., Follett, K., Hur, K., Ippolito, D., & Stern, M. Deep brain stimulation in Parkinson's Disease: A Meta-analysis of Patient Outcomes, *Journal of Neurosurgery*, 103 (2005), 956–967.
- Winkler, C., Kirik, D., & Bjorklund, A. Cell Transplantation in Parkinson's Disease: How Can We Make it Work?, *Trends in Neuroscience*, 28 (2005), 86–92.
- Woerdeman, P. A., Willems, P. W., Noordmans, H. J., Berkelbach van der Sprenkel, J. W., & van Rijen, P. C. Frameless Stereotactic Subcaudate Tractotomy for Intractable Obsessive-Compulsive Disorder, *Acta Neurochirurgia*, 148 (2006), 633–637.

PART III

EXPERIMENTAL
STUDIES

Split Brains and Dual Minds

Split-brain patients, or in more formal terms those who have undergone *cerebral commissurotomy*, provide a natural link between the clinical and experimental studies of the organization of the human brain. These patients are clinical subjects who have undergone neurosurgery, and yet the data they provide are treated as if they were derived from an experimental procedure. The focus of the research is on understanding the brain, not on helping the patient. Historically, split-brain studies also provided a particular stimulus for the development of laboratory techniques for use with intact human subjects.

The commissurotomy operation was introduced by Van Wagenen in 1940 as a treatment for certain severe and intractable forms of epilepsy, characterized by a focus in one hemisphere triggering a major seizure that spreads across the corpus callosum to involve both hemispheres. The idea was, simply, that cutting the corpus callosum and the other forebrain commissures would restrict the seizure to one hemisphere. Ultimately, it was found that even better relief from the epilepsy could be obtained, and that the frequency as well as the extent of seizure activity could be dramatically improved by the operation. A series of patients was operated upon, but interest in the procedure was lost until the early 1960s when Sperry, in collaboration with the neurologist Joseph Bogen and the surgeon Philip Vogel, instituted a new series of operations. (For a full treatment of the theoretical background to this work, see Bogen, 1979.)

Sperry had been working in the 1950s on experimental split-brain preparations in animals, and it was he who saw the significance of the operation for theories about the organization of the brain. A curious fact

about the earlier series was that almost no handicaps in everyday life had been reported, despite the radical nature of the surgery. Relatively sophisticated experimental investigations, for the time, were also carried out without significant deficits being revealed.

However, Sperry, working with Michael Gazzaniga, was able to show by elegant techniques that the patients in the Bogen and Vogel series did suffer from certain subtle deficits that were not apparent in their everyday activity. These deficits are described in the following pages. Following the Bogen and Vogel series, a further series of patients was operated upon by Wilson, and a number of patients also received commissurotomy for the treatment of intraventricular and midline tumors, and for abnormalities of the cerebrovascular system.

THE COMMISSUROTOMY OPERATION AND THE PATIENTS

The operation in its full form involves complete section of the corpus callosum, the anterior and hippocampal commissures, and the massa intermedia (see Figure 10.1). It therefore divides all direct cortical links between the two cerebral hemispheres, isolating the cortex of each hemisphere from its partner. From this division comes the term “split brain,” and also the importance of these patients in helping to construct models of the function of the right and left halves of the human brain. A number of patients have also undergone partial commissurotomy (incomplete section of the corpus callosum), and while data from these patients are of considerable importance, in the interests of clarity only the “complete splits” will be discussed in this chapter. The related operation of hemispherectomy, in which an entire hemisphere is removed, and the naturally occurring cases of absence of the corpus callosum (“callosal agenesis”) are also relevant but will not be treated here. A review may be found in Lassone and Jeeves (1994).

Before we go on to examine the effects of commissurotomy, several points should be made clear. The first is that although split-brain patients are treated as a single group, the operations performed in the three series do differ in significant ways (and this may partly account for the failure to find in the first series some of the deficits that were observed in the later series). In particular, not all the operations involved cutting the anterior commissure, which is now known to be capable of transferring quite considerable amounts of information between the two hemispheres (Risse, LeDoux, Springer, Wilson, & Gazzaniga, 1978). Within the Wilson series, there are two subseries, the anterior commissure being sectioned in the first, but not the later, set of patients (Wilson,

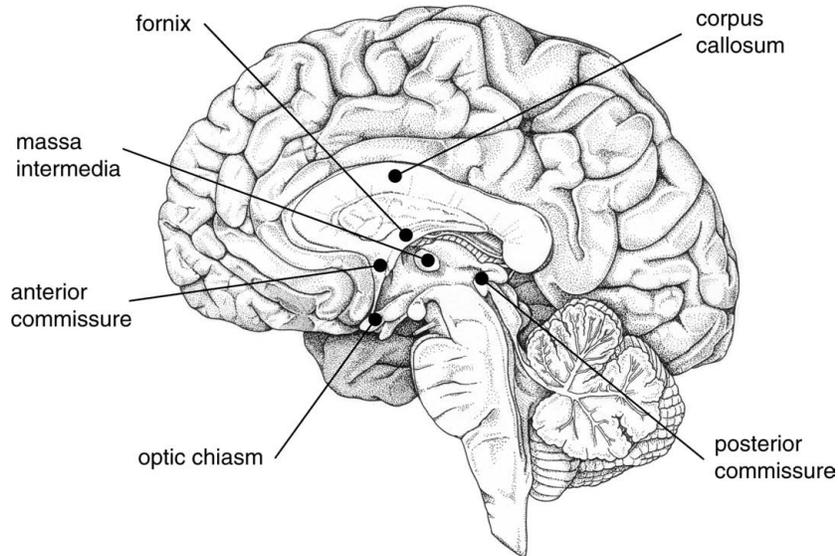


FIGURE 10.1. The major interhemispheric cerebral commissures.

Reeves, & Gazzaniga, 1978). The operation performed by Van Wagenen was undertaken in stages, while later series used a single operation. There is insufficient space here to go into the significance of these differences, but a careful study of the effects of commissurotomy should take account of these factors.

Perhaps more important is a word of caution about the patients. The number of patients operated upon is small, and the number reported in the (very extensive) literature is a minority of these. The total number of patients available is probably no more than about 30, and the vast bulk of the literature concerns no more than about half a dozen of these. Particularly as these patients differ greatly in their performance on the experimental tasks set for them, it is dangerous to regard them as a homogeneous group. Beware of any conclusion based on the mean performance of a group of these patients, as it is likely to be misleading. In fact, the data from the patients are increasingly reported as a set of single case studies, which seems more reasonable, but this practice has not been universally adopted.

It is also tempting to treat the data as if split brain patients presented us with neat controlled experimental results. This is not so. These are surgical lesions in *abnormal* brains. The patients all have long and complex neurological histories, reflected in their pre- and postoperative

neurological status. Many have lesions in other parts of the brain; some have had lesions from birth (with the suspicion that the organization of the brain has developed atypically); and some have had the operation at a relatively early age, when a degree of plasticity still remains in the brain. A detailed illustration of how these factors make interpretation of the data from split-brain patients extremely difficult has been provided by Whitaker and Ojemann (1977).

All these factors—the small number of patients, their different pre- and postoperative histories and response to the operation, the precise nature of the surgery they have received—as well as the effects of their current anticonvulsive medication point to caution in treating the patients as a single group, and in treating the findings as if they came from experimental lesions in a previously normal brain.

THE EFFECTS OF COMMISSUROTOMY

The most striking result of commissurotomy remains the lack of handicap experienced by most patients. There is an interesting film of one female patient doing domestic tasks, like beating eggs, and also swimming, activities that involve integration between the two sides of the body, and doing them quite successfully. Nevertheless, by ensuring that information coming from the external world is restricted to one side of sensory space, and external means of communication between the two sides of the body are prevented (more of this later), the independence of the two cerebral hemispheres, and consequent deficits in integration between the two sides of the body, can be demonstrated.

The clearest example of this is when visual information is presented in the *left or right visual half field*. Stimuli that appear to the left of the point on which a subject is fixating are projected initially only to the right occipital cortex, while those to the right of fixation (in the “right visual field”) are projected to the left occipital cortex. Therefore, if the subject’s fixation is controlled, this “divided visual field” technique can be used to present visual information to either the left or the right hemisphere alone. While in normal subjects the information is then distributed through the entire brain (see Chapter 11), in split brain patients it is confined to the hemisphere of original reception (see Figure 10.2).

If a visual stimulus is shown in the right visual field, and projects to the left hemisphere, which possesses speech (see Chapter 7), then the stimulus can be verbally identified, and can be picked out from a set of possible responses by the right hand (also controlled by the left hemisphere). It cannot, however, be selected by the left hand (controlled by the right hemisphere). Conversely, a stimulus presented in the left visual

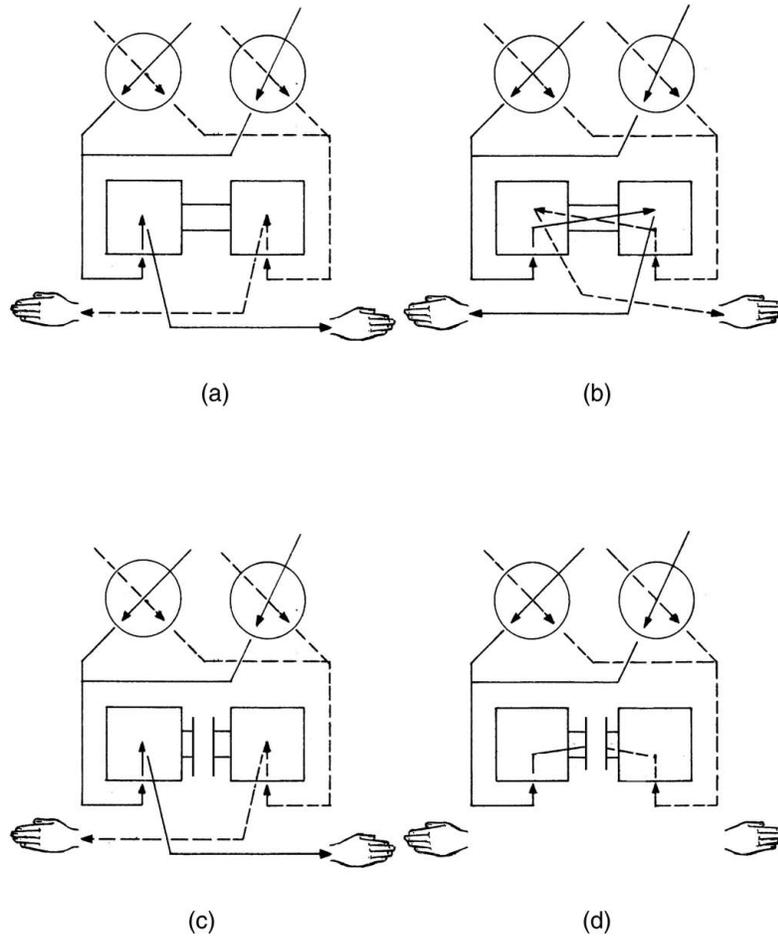


FIGURE 10.2. Connections between the two hemispheres and the hands. In normals, with the direct pathway (a), the right hand responds to the right visual field (solid line) and only one hemisphere, the left, is involved. With the indirect pathway in normals (b) the left hand responds to the right visual field (solid line) and both hemispheres and the corpus callosum are involved. In split-brain subjects the direct pathway still operates (c) but the indirect pathway is unable to function (d). (After Beaumont, 1983.)

field will go to the right hemisphere and can be indicated by the left hand, but not by the right hand, or by speech. The information is restricted to the hemisphere of reception, and can be linked only to response processes that are controlled by that hemisphere. This basic pattern of response to visual stimuli occurs whether the task is to match a visual picture to a real object, to another picture, or to a word, or to try to name it; or whether it is to match a word to another word, to a picture, or an object, or to speak it.

Some curious phenomena can be observed as a result of this dissociation between the hemispheres. If a word with two component parts, for example *heart* (*he* + *art*), is presented very briefly so that the subject is fixating between the *e* and the *a*, then patients will say that they saw *art* while with their left hand they will, at the same time, pick out (from among an array of possible responses) *he* as the stimulus. This behavior can be seen at its clearest in the “chimeric figure” experiments (Levy, Trevarthen, & Sperry, 1972). Chimeric figures are constructed of two half figures arranged so that patients fixate upon the vertical division between the two half stimuli (see Figure 10.3). These stimuli may be constructed from outline drawings or more complex figural stimuli like

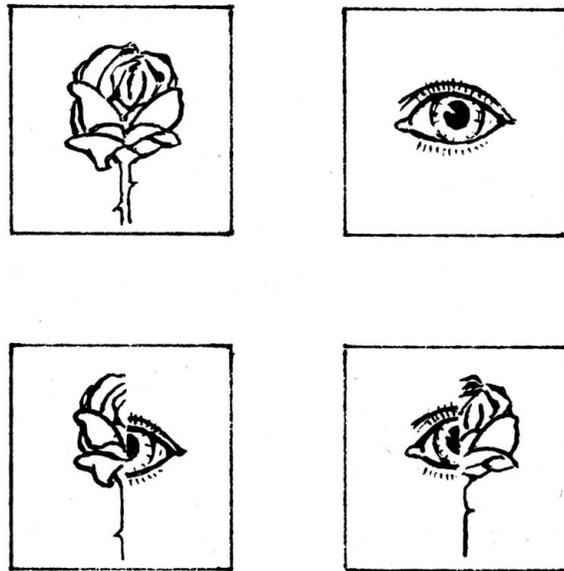


FIGURE 10.3. Chimeric figures. The chimeric figures (below), constructed from whole stimuli (above), are used to test recognition. (After Levy, Trevarthen, and Sperry, 1972.)

faces. As we might expect, shown a chimeric figure composed of the left half face of an old man with a beard wearing a hat and the right half face of a young blonde, patients say that they have seen an attractive young woman, as the left speaking hemisphere only knows about the right half of the stimulus. Incidentally, the patients do not know of the chimeric nature of the stimuli and report nothing unusual about what they have seen. At the same time, the left hand will select the old man from among a selection of whole faces that include the complete faces of the old man and the blonde. If the anomaly of the response is pointed out to the patient, he or she may well appear confused, and may make some comment such as that the hairstyle looked rather like a hat, in an attempt to resolve the confusion.

Stimuli may also be lateralized by *somaesthetic presentation*, following the same logic as divided visual field presentation. The patient may be touched at some point on the body and asked to report (in speech) the location of the stimulation or its nature (light touch, pinprick, and so on), or asked to point to the spot with either the right or the left hand. Objects may be placed in one hand (out of sight) and haptically explored for naming, or for matching to another object among an array using either the same or the opposite hand. Hand postures may be set up on one hand (again out of sight) to be reproduced by the opposite hand, or a representation of a gesture presented in one of the visual fields to be reproduced by the ipsilateral or contralateral hand. There is a rich variety of possibilities for the ingenious investigator, and many of the changes have been rung on this kind of experiment. What the results boil down to is, however, that split brain patients have difficulty integrating information across the body midline: pointing with one hand to places on the opposite side of the body, passing information from one hand to the other, or relating visual information on one side of the body to movement and sensation on the other. Further, there is difficulty in expressing the results of right hemisphere processing or left-sided stimulation in speech.

This conclusion concerning somaesthesia has to be qualified a little. The lateralization of somaesthesia is most complete in relation to the more distal parts of the body, especially if finely controlled or skilled movements are required. The effects are seen most clearly, therefore, in the hands and especially the fingertips. Taking more gross body movements, and sensation from the more proximal body areas such as the trunk, the lateral differentiation is less clear. This is almost certainly because of the existence of ipsilateral connections to these areas for both sensation and motor control (see Gazzaniga & LeDoux, 1978).

Audition turns out to be rather less straightforward than vision. This is because the projections from the ears to the brain are not exclu-

sively contralateral, but are to some extent bilateral (see p. 74). Nevertheless, the dominance of the crossed contralateral pathway can be observed in the suppression that follows dichotic (bilateral simultaneous) presentation. Split-brain patients can report stimuli presented to either ear if the stimuli are presented singly, but if two stimuli are presented at the same time to the two ears, there will be a massive bias to report the right ear stimulus (which passes to the left hemisphere by the dominant crossed pathway). It is assumed that the ipsilateral projection from left ear to left hemisphere is suppressed in dichotic presentation, although, while the spoken response may favor the right ear, there may be evidence of reception of the left ear stimulus in response by the left hand (Gordon, 1980; Springer, Sidtis, Wilson, & Gazzaniga, 1978).

While the question of speech function in the two hemispheres is clearly answered by the commissurotomy evidence, the question of the extent of *right hemisphere language* is not. The left hemisphere clearly has the capacity to generate speech, but there is no evidence to suggest that in the normally developed and lateralized brain this capacity is also present in the right (outside automatic speech and swearing). In early studies there was a tendency to confuse "speech" and "language," and to assume that language functions were as completely lateralized. There has since been a gradual and continuing revision of this assumption, with the right hemisphere being credited with increasingly sophisticated linguistic abilities, short of speech output.

Early reports tended to suggest that the comprehension of language stimuli presented to the right hemisphere was very limited, and that beyond the recognition of simple and common concrete nouns there was little linguistic ability present. It is now clear that while an asymmetry undoubtedly exists, in that the right hemisphere is simply not as good at performing language functions as the left, the right hemisphere nevertheless possesses a significant degree of linguistic skill. Various studies have now shown that the right hemisphere can understand abstract words and a variety of syntactic structures including verbs, sentence transformations, and long nonredundant and semantically abstract references; is competent at semantic matching; can perform rhyme matches; and can understand mental associations based upon semantically coordinate, contingent and superordinate relationships. In addition, they have shown that the inability to name objects held in the left hand is only an inability to generate the name; it does not imply a failure to comprehend the nature of the object, or to construct semantic associations with it that may be manipulated and expressed in language, although not in speech. A degree of writing is possible for the left hand, although some of the patients show difficulty with this (in contrast with left-hand performance in drawing and spatial manipulation, which is superior to that

with the right hand). The precise extent of right hemisphere language is still a matter of debate, despite extensive and elegant studies (notably by Eran Zaidel). Reviews of the work on right hemisphere speech are to be found in Beeman and Chiarello (1997), Code (1987), and Wray (1992). Perhaps the most interesting and telling finding is that of Eran Zaidel (1979), who also compared the performance of the separated hemispheres with the performance in “free vision” (allowing the hemispheres to cooperate), and found that the two hemispheres together produced better performance than either alone.

Relevant to this issue of the abilities of the right hemisphere are the most elegant *metacognition* experiments of Levy and Trevarthen (1976). These studies employed chimeric figures that were outline drawings cleverly selected so that the half stimuli could be matched to complete stimuli on the basis of identity, or physical similarity, or by semantic functional association (see Figure 10.4). It was found, as had been expected, that the right hemisphere was relatively proficient at, and generally assumed control of, appearance matches, while the left hemisphere controlled function matches. From these findings come the theoretical arguments for a difference between the two hemispheres that is based upon modes of processing rather than upon the nature of the material or of the response that is demanded. It is suggested that it is the nature of the cognitive operations required that determines the relative proficiency of the two hemispheres. However, we must be careful to note that this result is only one of *relative* specialization. The data show that while there is an asymmetry between the performance of the two hemispheres, there was a significant number of trials in which the “wrong” hemisphere assumed control of the match. In other words, while the right hemisphere may prefer, or be more competent at, appearance matches (for whatever reason), it is not entirely without the ability to undertake function matches. Dissociations between controlling hemisphere and the strategy employed occurred on a significant number of trials, and therefore the asymmetry is certainly far from absolute.

As reviews of the effects of commissurotomy have tended to emphasize the division and independence of the two hemisphere systems, and to maximize the asymmetries in performance that may be observed, some evidence that points to the limitations of this view should also be noted. I have argued elsewhere (Beaumont, 1981) that the published literature has significantly misrepresented the nature of the effects of commissurotomy by presenting an oversimplified and overgeneralized picture, and I am wary of repeating the error myself here. The problem, historically, was undoubtedly the influence exercised on Sperry’s thinking by the initial one or two patients in the Bogen and Vogel series. These turned out to be not entirely typical for a variety of reasons, and

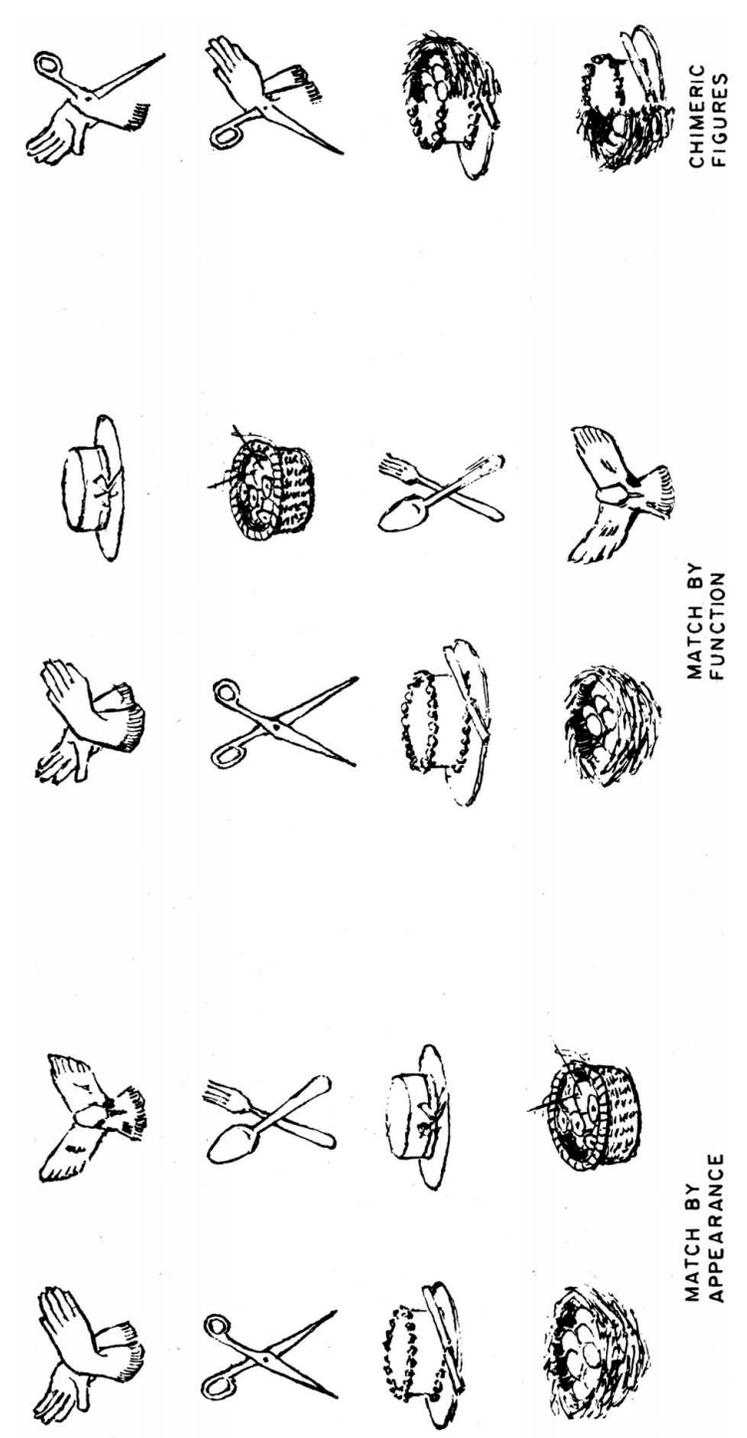


FIGURE 10.4. Chimeric stimuli (see Fig. 10.2) in metacontrol experiments. The whole stimuli implied by the chimeric figures (right) can be matched by appearance (left) or by function (center). (After Levy and Trevarthen, 1976.)

the rather bold and dramatic conclusions that he drew have had to be tempered in a number of ways.

First, there are clear exceptions to the general statements made above about the effects of the operation. For instance, some of the patients are able to indicate with a given hand points stimulated on the opposite side of the body. (The variation in the degree of left hand agraphia has already been noted.) There are also opportunities for the interhemispheric transfer (obviously by routes other than the commissures that have been sectioned) of certain types of information, transfer of the size-weight illusion being just one illustration (Gandevia, 1978). Other evidence has emphasized the continuity of the integrated and articulated operation of the total cerebral system. LeDoux, Risse, Springer, Wilson, and Gazzaniga (1977) examined the performance of one of the patients on a complex, high-level concept attainment and hypothesis testing task, in which there seems no doubt that the cooperation of both hemispheres was required for satisfactory performance. The patient was assessed both pre- and postoperatively, and no postoperative deficit was found, but rather an improvement in performance. They conclude that complex cognitive processes do not depend on the function of the corpus callosum.

A problem in assessing the significance of some of the exceptions to the “expected” findings (although the last-named study seems free of this suspicion) is that many of the patients develop subtle cross-cueing strategies, that is, they develop ways to circumvent their handicap by using external routes for the lateral transfer of information. For example, sound cues may be passed from objects in the left hand to the left hemisphere via the right ear. Also, emotional signals may transfer easily by subcortical routes, so that by feeling a generalized arousal reaction a correct response may be deduced. The left hemisphere may conduct a speech commentary on the responses of the left hand, which it can see in free vision in the right visual field, and so transmit guidance to the right hemisphere. But perhaps the most subtle strategy concerns eye movements, which can transfer subcortically, so that even the extent of eye movement required to inspect a stimulus may give the essential clue to stimulus identity if the range of possibilities is sufficiently small (Gazzaniga, 1970; Gazzaniga & Hillyard, 1971). The degree to which the cerebral hemispheres cannot be considered in isolation from the subcortical centers to which they remain connected (and which are not divided) has been an important theme introduced by Sergent (1986).

These examples alone show how carefully controlled the experiments must be, and how vigilant the experimenter, if the possibility of this kind of external transfer is to be ruled out. Also, it becomes less surprising that the patients suffer little handicap in everyday life, particu-

larly as they can manipulate what appears in which field of vision and employ both hands in order to implement strategies to share information between the hemispheres.

There is one further factor that potentially disturbs the rather neat explanation of the effects of commissurotomy. This is the role of attention, a factor that will feature more prominently in a related context in Chapter 12. The potential intervention of attentional factors is demonstrated by a beautifully elegant experiment by Gazzaniga, Ladavas and colleagues (Gazzaniga & Ladavas, 1987; Ladavas, De Pesce, Mangun, & Gazzaniga, 1994; Luck, Hillyard, Mangun, & Gazzaniga, 1994). They performed the standard experiment with stimuli in the left and right visual fields and obtained the expected effects, as described above. But, they then asked their participants to tilt their head 90 degrees so that one of their ears was touching their shoulder; the rest of the body was kept in the normal position. The experiment was then repeated. What we should expect is that any effects of lateral presentation of the stimuli will disappear. Although the stimuli appear to the left and right of the participant's body, they are now above and below the fixation point with respect to the orientation of the participant's head. What actually happened, amazingly, was that the lateral asymmetry was substantially preserved. This cannot be explained adequately with respect to the neural projection of the stimuli into the left and right hemispheres. It suggests the role of some internal representation of extra-bodily space, and processes that direct attention to the "left" or "right" of this space. (For a discussion of the processing of spatial relations in commissurotomy, see Sergent, 1991.) This important finding does not fundamentally undermine our understanding of the effects of commissurotomy, but it does demonstrate that our understanding is incomplete and that there are psychological aspects yet to be determined.

There are numerous reviews of this field in the literature; among the most useful from the period when research was most active are Bogen (1979), Dimond (1972), Gazzaniga and LeDoux (1978), Levy (1980), and Sperry (1973, 1974). References to more recent reviews are given at the end of this chapter. Excellent discussions of the theoretical models employed may also be found in Trevarthen (1975) and E. Zaidel (1978). As has already been made clear, care should be exercised in evaluating the accuracy of some of these reviews. It may nevertheless be concluded that split-brain patients have a deficit in the cross-integration of information, at least under experimental conditions. In a limited sense it is possible to accept the conclusion of Sperry that the patients exhibit "two independent streams of conscious awareness" that are out of contact with each other, each with its own "separate and private sensations; its own perceptions; and its own impulses to act" (Sperry, 1968, p. 724). Whether this really implies that the consciousness of these patients is

divided or in some way doubled, and whether this holds implications for the processes that normally underlie consciousness, are questions to which we now turn.

CONSCIOUSNESS DIVIDED?

Typically for certain writings in this area, Charles Furst has claimed that “split brain research has opened a new frontier of scientific investigations into the physical basis of consciousness. The vistas which lie beyond this frontier will perhaps revolutionize our traditional ways of understanding the human mind” (1979, p. 161). Is this neuroscience or neurofantasy?

The separation between the cognitive activities of the two hemispheres has led various writers to suggest that dividing the cerebral commissures produces two independent minds. This notion was taken up most energetically by Roger Sperry, who proposed that the split brain patient has two separate minds within the one body, each with its own will as well as its own perceptions and memories (Sperry, 1976). This model, with minor variations, has been the most popular, and it is possible to find several examples of it in the literature (see Beaumont, 1981).

The only major opponent of this theoretical position has been Eccles (1977). He has suggested that, while commissurotomy divides higher-level functions between the two hemispheres, only the left hemisphere contains the seat of consciousness. The right hemisphere is considered as an automatically acting subordinate partner with rather less than normal human attributes. He bases this position largely on the inability of the right hemisphere to produce speech, which he regards as the necessary test of the possession of conscious awareness.

It may have occurred to you to wonder what is meant by “consciousness.” Indeed, much of the debate might be resolved if those who construct theories about this topic were themselves more clear. Some writers, particularly those who espouse double mind theories, include any evidence of intelligent responsiveness, attention, and awareness as indicating consciousness. Eccles, however, employs the very much more restricted requirement that the organism must declare itself conscious by speech. I do not intend to try to resolve this matter here, but it seems to many people that Eccles’s definition is too restrictive. It demands, for instance, that we remain agnostic about whether the congenitally mute are conscious. It also implies that it is not possible to know if a person who has lost his or her voice from a sore throat possesses consciousness. This is patently absurd. On the other hand, “any intelligent cognitive act” seems too loose a definition, for it forces us to attribute consciousness even to lowly animals if they show that they can learn some simple

experimental task, even though we are in a wider context unsure that this demands “consciousness.” Possibly a solution can be found by demanding some evidence of self-awareness and purposeful cognitive activity, although this is inadequate as a formal definition. A fuller discussion of this problem may be found in Savage (1976).

By now it may also be clear to you that the debate is embroiled in philosophical mind–body issues (see p. 7). There are many fascinating discussions of these problems by both neuroscientists and philosophers, including the prospect of transplanting separated hemispheres into different bodies and then recombining them at a later date (Bell, 1975; Globus, 1976; Nagel, 1971; Puccetti, 1981, 1985). Such issues raise fundamental questions about our conception of personal identity, but they have to be sidestepped here for want of space. However, if my argument is accepted that it is not sensible to regard the minds of commissurotomy patients as divided, many of the questions become academic. (For excellent discussions of consciousness, with some reference to the split-brain patients, see Rose, 2006, and Zeman, 2001.)

To return to the scientific evidence, there are five central questions that we should ask before deciding on the structure of consciousness in split-brain patients.

1. *Does the right hemisphere possess language?* This question has already been broached, with the conclusion that the right hemisphere may be effectively mute, and is certainly less proficient at most linguistic tasks, but that it does possess a remarkable variety of linguistic skills. As a result, the right half brain of a split-brain patient cannot be considered to lack language. It is, of course, possible that this evidence is in part a consequence of the patient’s early neurological history, and that it does not imply that language is similarly represented in the right hemisphere of a normal brain. The speech developed in association with the right hemisphere by a patient known as PS, who was operated upon at the age of 15 and whose neuropsychological development was almost certainly not normal, provides an illustration of how atypical organization may occur in these patients (Gazzaniga, Volpe, Smylie, Wilson, & LeDoux, 1979). However, the point still stands that split-brain patients have some right hemisphere language capability.

2. *Are there other abilities that the right hemisphere may lack?* While there is much evidence for the relative specialization of the two hemispheres, with some functions better performed by the right and some better performed by the left, no abilities other than speech and language have been proposed as absent from the right hemisphere. In fact, the trend has increasingly been to credit the right hemisphere with sophisticated abilities (Perelman, 1983; Ardila & Ostrosky-Solis, 1984). The most striking example is the demonstration by Sperry, Zaidel, and

Zaidel (1979) of self-recognition and social awareness in the right hemisphere of two of the patients. Arrays of pictures and photographs that included some with personal and emotional significance among similar neutral stimuli were presented. The detailed accounts of the patients' responses leave little doubt that while the patients have great difficulty in describing their thoughts about the stimuli through speech, a characteristic social, political, personal, and self-awareness, such as we should expect for the left, was present in the right hemisphere. For example, a woman was shown (to her right hemisphere) four photographs of men, which included one of her son. She readily picked out her son as the one she recognized, but could not say who it was. She reported good feelings about the image and suggested the names of her daughter and husband, but remained confused. Following further questions to which she indicated further positive feelings ("Yeah, it's fine, it's beautiful"), she pointed to the picture of her son and said, "The best-looking one there."

EXAMINER: Is it you? Your husband? (*Patient does not respond.*)
 . . . your son?

PATIENT: (*in a very definite decisive tone*) Yeah.

Thus helped to overcome the handicap of limited speech output, the right hemisphere showed every sign of being characteristically human.

It has also been suggested that there is a lack of awareness of right hemisphere processes, based on some rather slender evidence, such as the denial of left-hand control, the failure to recognize the anomaly in chimeric figures (although normal subjects may also miss the anomaly under certain circumstances), and the maintenance of independent response probabilities by the two hemispheres in a learning task. This evidence is not sufficiently strong to deny awareness of right hemisphere processes, particularly in view of the kinds of demonstration above.

3. *Is there any cross-integration between the disconnected hemispheres?* It has already been hinted that the separation of the hemispheres is not as complete as is sometimes suggested. There is inter-manual transfer of maze learning, accurate target-directed reaching across the midline, and completion across the midline for incomplete geometric figures, pictures, and words. The ambient space around the body, in vision outside the central field, is not divided for the perception of space relations or motion in space (Trevarthen, 1974). Bimanual skills are relatively preserved (Preilowski, 1975).

It is clear that the assumption that cross-integration across the hemispheres is impossible for these patients is incorrect. It is possible to demonstrate deficits in this function in a rigorous experimental situation and on a particular task, but for most cognitive activities (certainly in

the unrestricted patient) there are mechanisms that allow substantial cross-integration.

4. *Are the separated hemispheres ever in conflict?* It is more difficult to decide the answer to this question because the relevant evidence is anecdotal. There are various accounts of conflicting activity by the two hands: one hand pulling trousers up, while the other pulled them down; one hand seeking help from the patient's wife while the other pushed her away. At one time it seemed that most of these accounts came from one patient, and in the early period following surgery, but they have been amplified and extended by a range of personal accounts collected by Dimond (1979). One patient said: "You wouldn't want to hear some of the things this left hand has done—you wouldn't believe it. It acts independently a lot of times. I don't even tell it to—I don't know it's going to do anything. . . . It seems to have a mind of its own" (p. 213).

It is still difficult to know what to make of such accounts, particularly as many of the patients are not ignorant of the literature written about them. I feel that these are rare incidents that are not fundamentally different from a variety of the silly errors we all occasionally make (unwrapping a candy, holding the wrapper and candy in opposite hands, and then throwing the candy in the trash and placing the wrapper in your mouth). Experimental attempts to document the conflict have failed, and great significance should not be attached to such anecdotal reports.

5. *Are there qualitative changes in thinking?* This is another question that is difficult to answer. Again, there are intriguing reports, but little firm evidence. Different strategies in performance have been assigned to the two hemispheres on the basis of performance on cognitive tasks, but other reports are essentially anecdotal. It may well be that split-brain patients do suffer changes in the nature of their thinking, but as yet such changes have not been satisfactorily demonstrated.

In view of the answers to our five questions, can we regard the consciousness of commissurotomy patients as abnormal? I do not think so. It is first of all clear that the right hemisphere does not seem in any way basically different from the left, except in speech production. The patients appear to possess high-level abilities, conceptualization, and self-awareness within the right hemisphere and to be aware, although perhaps to a limited extent, of right hemisphere processes. It seems wrong, in the face of this evidence, to deny consciousness to the right hemisphere, and we can therefore reject Eccles's model.

If consciousness resides in both hemispheres, is it divided? The answers to our questions again suggest that outside very special, highly controlled, and artificial circumstances, it is not. It may be possible, par-

ticularly with cognitive tasks in the laboratory, to demonstrate some dissociation between the two hemisphere systems. This is no more remarkable than the fact that I can talk while holding a cup of coffee in one hand and scratching my ear with the other. There is extremely slight evidence for conflicting wills in the two hemisphere systems, and a fair amount of evidence for cross-integration between the two lateral systems. There is none for significant qualitative changes in the thinking, everyday responsiveness, or awareness of these patients. On consideration, this is hardly surprising. Both cortical systems are fundamentally bound onto a subcortical substrate with massive integrated bilateral structures, and they cannot sensibly be considered independently from this substrate. The two hemispheres in split-brain patients are no more disconnected than are my two hands. Under certain circumstances they may be observed to be undertaking different but simultaneous activities, and they may retain certain properties of their own, but they are part of a more general system that maintains the integrity of the mind, just as the hands are joined to the arms and trunk, so maintaining the integrity of the body.

Split-brain patients have prompted a valuable debate about the nature of consciousness and its relation to mental processes. However, it seems a mistake to think that the state of consciousness of these patients has been altered in any radical fashion, or to consider that the studies should lead us to revise our traditional model of a single and unitary mind associated with the entire organ of the brain, forming an integral part of a single physical body.

There has been a decline in the appearance of new literature on the commissurotomy patients over the past decade or so. This is partly because the operation is now rarely performed, except as a treatment for midline or ventricular tumors, and then the “split” is unlikely to be complete. Apart from the increasing success in controlling seizure activity by medication, there has been an increasing reluctance to perform the surgical operation. Ironically, this may in part be a consequence of the fact that certain of the split-brain patients made use of the research literature to sue their surgeons. The very success of the experimental literature has been the cause of its closure as an active area of research.

CONCLUSION

Considerable significance has been attached to the studies of patients who have undergone cerebral commissurotomy. It has been considered that they provide essential evidence about the independence of the two cerebral hemispheres and their lateral specialization. The evidence sug-

gests that, while there is a degree of disconnection between the two hemispheres in these patients, so that under laboratory conditions it can be shown that information cannot be transferred between the two sides of the brain, we should be cautious about overstating the importance of these findings. Split-brain patients do, however, possess cortical hemispheres each of which can independently perceive, remember, think, and respond, to some degree outside the awareness of the other.

It has also been suggested that these patients show evidence of divided consciousness. From a brief review, it is concluded that this view cannot be supported. Certain unusual phenomena may be demonstrated in these patients, but there is no good reason to believe that they possess two independent minds as a result of the operation, or that they tell us very much about the normal operation of consciousness.

The true significance of split brain studies is the dramatic stimulus that they have given to the development of neuropsychological techniques and theoretical models, which are discussed in later chapters.

FURTHER READING

It seems appropriate to introduce here some general books that are appropriate for all the experimental neuropsychology to be discussed in this and the following chapters in Part III. All of the following provide useful, and generally readable, summaries of the area:

- Bradshaw, J. L. *Hemispheric Specialization and Psychological Function* (Chichester, UK: Wiley, 1989).
- Benson, F. D., & Zaidel, E. *Dual Brain: Hemispheric Specialization in Humans* (New York: Guilford Press, 1985).
- Hellige, J. B. *Hemispheric Asymmetry: What's Right and What's Left* (Cambridge, MA: Harvard University Press, 1993).
- Hugdahl, K., & Davidson, R. J. (Eds.). *The Asymmetrical Brain* (Cambridge, MA: MIT Press, Bradford Books, 2004).
- Iaccino, J. F. *Left Brain–Right Brain Differences: Inquiries, Evidence, and New Approaches* (Mahwah, NJ: Erlbaum, 1993).
- Springer, S. P., & Deutsch, G. *Left Brain, Right Brain: Perspectives from Cognitive Neuroscience* (Fifth edition, San Francisco: Freeman, 1998).

A brief evaluation of contemporary research approaches is also to be found in:

- Beaumont, J. G. Future Directions in Laterality Research, *Neuropsychology Review*, 7 (1997), 107–126.

The most accessible reviews of the effects of commissurotomy are to be found in:

- Gazzaniga, M. S., & LeDoux, J. E. *The Integrated Mind* (New York: Plenum Press, 1978).

Sergent, J. A. A New Look at the Human Split Brain, *Brain*, 110 (1987), 1375–1392.

Discussions of consciousness and split brains are (besides specific references in the text) to be found in:

Beaumont, J. G. Split Brain Studies and the Duality of Consciousness, in G. Underwood & R. Stevens, eds., *Aspects of Consciousness*, Vol. 2 (London: Academic Press, 1981).

Sperry, R. W. Consciousness, Freewill and Personal Identity, in D. A. Oakley & H. C. Plotkin, eds., *Brain, Behaviour and Evolution* (London: Methuen, 1979).

REFERENCES

Ardila, A., & Ostrosky-Solis, F. (Eds.). *The Right Hemisphere: Neurology and Neuropsychology* (New York: Gordon and Breach Scientific Publishing, 1984).

Beaumont, J. G. Split Brain Studies and the Duality of Consciousness, in G. Underwood & R. Stevens, eds., *Aspects of Consciousness*, Vol. 2 (London: Academic Press, 1981).

Beeman, M., & Chiarello, C. (Eds.). *Right Hemisphere Language Comprehension: Perspectives from Cognitive Neuroscience* (Mahwah, NJ: Erlbaum, 1997).

Bell, G. A. The Double Brain and Mind Brain Relationship, *Journal of Behavioral Science*, 2 (1975), 161–167.

Bogen, J. E. The Callosal Syndrome, in K. M. Heilman & E. Valenstein, eds., *Clinical Neuropsychology* (New York: Oxford University Press, 1979).

Code, C. *Language, Aphasia and the Right Hemisphere* (Chichester, UK: Wiley, 1987).

Dimond, S. J. *The Double Brain* (Edinburgh, UK: Churchill-Livingstone, 1972).

Dimond, S. J. Symmetry and Asymmetry in the Vertebrate Brain, in D. A. Oakley & H. C. Plotkin, eds., *Brain, Behaviour and Evolution* (London: Methuen, 1979).

Eccles, J. C. *The Understanding of the Brain* (Second edition, New York: McGraw Hill, 1977).

Furst, C. *Origins of the Mind* (Englewood Cliffs, NJ: Prentice-Hall, 1979).

Gandevia, S. C. The Sensation of Heaviness after Surgical Disconnection of the Cerebral Hemispheres in Man, *Brain*, 101 (1978), 295–306.

Gazzaniga, M. S. *The Bisected Brain* (New York: Appleton-Century-Crofts, 1970).

Gazzaniga, M. S., & Hillyard, S. A., Language and Speech Capacity of the Right Hemisphere, *Neuropsychologia*, 9 (1971), 273–280.

Gazzaniga, M. S., & Ladavas, E. Disturbance in Spatial Attention Following Lesion or Disconnection of the Right Parietal Lobe, in M. Jeannerod, ed., *Neurophysiological and Neuropsychological Aspects of Spatial Attention* (Amsterdam: North-Holland, 1987).

- Gazzaniga, M. S., & LeDoux, J. E. *The Integrated Mind* (New York: Plenum Press, 1978).
- Gazzaniga, M. S., Volpe, B. T., Smylie, C. S., Wilson, D. H., & LeDoux, J. E. Plasticity in Speech Organisation Following Commissurotomy, *Brain*, 102 (1979), 805–815.
- Globus, G. G. Mind, Structure and Contradiction, in G. G. Globus, G. Maxwell, & I. Savodnik, eds., *Consciousness and the Brain: A Scientific and Philosophic Enquiry* (New York: Plenum Press, 1976).
- Gordon, H. W. Right Hemisphere Comprehension of Verbs in Patients with Complete Forebrain Commissurotomy: Use of the Dichotic Method and Manual Performance, *Brain and Language*, 11 (1980), 76–86.
- Ladavas, E., De Pesce, M., Mangun, G. R., & Gazzaniga, M. S. Variations in Attentional Bias in the Two Disconnected Cerebral Hemispheres, *Cognitive Neuropsychology*, 11 (1994), 57–74.
- Lassone, M., & Jeeves, M. A. (Eds.). *Callosal Agenesis: Natural Split Brain?* (New York: Plenum Press, 1994).
- LeDoux, J. E., Risse, G. L., Springer, S. P., Wilson, D. H., & Gazzaniga, M. S. Cognition and Commissurotomy, *Brain*, 100 (1977), 87–104.
- Levy, J. Cerebral Asymmetry and the Psychology of Man, in M. C. Wittrock, ed., *The Brain and Psychology* (New York: Academic Press, 1980).
- Levy, J., & Trevarthen, C. Metacognition of Hemispheric Function in Human Split-Brain Patients, *Journal of Experimental Psychology: Human Perception and Performance*, 2 (1976), 299–312.
- Levy, J., Trevarthen, C., & Sperry, R. W. Perception of Bilateral Chimeric Figures Following Hemispheric Disconnection, *Brain*, 95 (1972), 61–78.
- Luck, S. J., Hillyard, S. A., Mangun, G. R., & Gazzaniga, M. S. Independent Attentional Scanning in the Separate Hemispheres of Split Brain Patients, *Journal of Cognitive Neuroscience*, 6 (1994), 84–91.
- Nagel, T. Brain Bisection and the Unity of Consciousness, *Synthese*, 22 (1971), 396–413.
- Perelman, E. *Cognitive Processing in the Right Hemisphere* (New York: Academic Press, 1983).
- Preilowski, B. Bilateral Motor Interaction: Perceptual-Motor Performance of Partial and Complete “Split-brain” Patients, in K. J. Zülch, O. Creutzfeldt, & G. C. Galbraith, eds., *Cerebral Localization* (Berlin: Springer-Verlag, 1975).
- Puccetti, R. The Case for Mental Duality: Evidence from Split-Brain Data and Other Considerations, *The Behavioral and Brain Sciences*, 4 (1981), 93–123.
- Puccetti, R. Experiencing Two Selves: The History of a Mistake, *The Behavioral and Brain Sciences*, 8 (1985), 646–647.
- Risse, G. L., LeDoux, J., Springer, S. P., Wilson, D. H., & Gazzaniga, M. S. The Anterior Commissure in Man: Functional Variation in a Multi-sensory System, *Neuropsychologia*, 16 (1978), 23–31.
- Rose, D. *Consciousness* (Oxford, UK: Oxford University Press, 2006).
- Savage, C. W. An Old Ghost in a New Body, in G. G. Globus, G. Maxwell, & I.

- Savodnik, eds., *Consciousness and the Brain: A Scientific and Philosophic Enquiry* (New York: Plenum Press, 1976).
- Sergent, J. A. Subcortical Coordination of Hemisphere Activity in Commissurotomed Patients, *Brain*, 109 (1986), 357–369.
- Sergent, J. Processing of Spatial Relations Within and Between the Disconnected Cerebral Hemispheres, *Brain*, 114 (1991), 1025–1043.
- Sperry, R. W. Hemisphere Deconnection and Unity in Conscious Awareness, *American Psychologist*, 23 (1968), 723–733.
- Sperry, R. W. Lateral Specialization of Cerebral Function in the Surgically Separated Hemispheres, in F. J. McGuigan & R. A. Schoonover, eds., *The Psychophysiology of Thinking* (New York: Academic Press, 1973).
- Sperry, R. W. Lateral Specialization in the Surgically Separated Hemispheres, in F. O. Schmitt & F. G. Worden, eds., *The Neurosciences: Third Study Program* (Cambridge, MA: MIT Press, 1974).
- Sperry, R. W. Mental Phenomena as Causal Determinants in Brain Function, in G. G. Globus, G. Maxwell, & I. Savodnik, eds., *Consciousness and the Brain: A Scientific and Philosophic Enquiry* (New York: Plenum Press, 1976).
- Sperry, R. W., Zaidel, E., & Zaidel, D. Self-recognition and Social Awareness in the Deconnected Minor Hemisphere, *Neuropsychologia*, 17 (1979), 153–166.
- Springer, S. P., Sidtis, J., Wilson, D. H., & Gazzaniga, M. S. Left Ear Performance in Dichotic Listening Following Commissurotomy, *Neuropsychologia*, 16 (1978), 305–312.
- Trevarthen, C. Analysis of Cerebral Activities That Generate and Regulate Consciousness in Commissurotomy Patients, in S. J. Dimond & J. G. Beaumont, eds., *Hemisphere Function in the Human Brain* (London: Elek Science, 1974).
- Trevarthen, C. Psychological Activities after Forebrain Commissurotomy in Man: Concepts and Methodological Hurdles in Testing, in F. Michel & B. Schott, eds., *Les Syndromes de Disconnexion Calleuse chez L'Homme* (Lyon, France: Hôpital Neurologique, 1975).
- Whitaker, H. A., & Ojemann, G. A. Lateralization of Higher Cortical Functions: A Critique, *Annals of the New York Academy of Sciences*, 299 (1977), 459–473.
- Wilson, D. H., Reeves, A., & Gazzaniga, M. S. Division of the Corpus Callosum for Uncontrollable Epilepsy, *Neurology*, 28 (1978), 649–653.
- Wray, A. *The Focusing Hypothesis: The Theory of Left Hemisphere Lateralized Language Re-Examined* (Amsterdam: Benjamins, 1992).
- Zaidel, E. Concepts of Cerebral Dominance in the Split Brain, in P. A. Buser & A. Rougeul-Buser, eds., *Cerebral Correlates of Conscious Experience* (Amsterdam: North-Holland, 1978).
- Zaidel, E. Performance on the ITPA following Cerebral Commissurotomy and Hemispherectomy, *Neuropsychologia*, 17 (1979), 259–280.
- Zeman, A. Consciousness, *Brain*, 124 (2001), 1263–1289.

Divided Visual Field Studies

The perception of briefly presented lateralized stimuli has been studied by experimental psychologists throughout the last century and into this one. Much work was done in the 1950s to investigate the superior recognition of words presented in the right visual field, but while reference was made to “cerebral dominance,” other factors such as reading habits and postexposural scanning were considered important in explaining the findings. It was the study of split brain patients in the early 1960s by Sperry and Gazzaniga (see Chapter 10) that led to the technique of divided visual field presentation being used to investigate the cerebral organization of normal intact subjects in the laboratory, and laid the foundation for contemporary experimental human neuropsychology. Since the mid-1960s, and especially between 1970 and 1985, an enormous number of studies have used the technique, which now forms the most important method of investigation in experimental neuropsychology. A far from comprehensive review around 1980 contained a bibliography of more than 1,000 references, mostly published within the previous 10 years, and this is some indication of the importance of the technique. If the experimental studies were unanimous in their findings, it would be possible to report dramatic progress in this field, but in fact it is difficult to draw firm conclusions from them. This is partly because of the indifferent scientific quality of many of the studies, and partly because of the complexity of the system under study. Nevertheless, some fairly clear conclusions may be drawn.

THE TECHNIQUE

The logic of the technique of divided visual field presentation is simple and elegant, and has been referred to in the preceding chapter: if a visual stimulus is presented in the left visual field, then it is projected initially only to the right occipital cortex; if presented in the right visual field, it is projected only to the left occipital cortex; and therefore if the subject's fixation is controlled, stimuli can be injected into one or the other hemisphere. Because the technique usually involves binocular vision, and the stimulus in a given visual field travels through both eyes and via one crossed and one uncrossed pathway to the contralateral hemisphere, differences between the eyes and the visual pathways are balanced out (see Figure 11.1). The subject's performance in terms of accuracy of report or speed of response can then be studied as a function of the hemisphere of initial presentation, even though processes following initial reception may result in the stimulus information being distributed to both hemispheres of the brain.

The technique relies on accurate control of fixation, and this is usually achieved by directing the subject's fixation to a desired point. The stimulus is then presented for a brief duration, too short to allow the subject voluntarily to orient the eyes to the stimulus location. It is difficult to determine just how long an exposure may be before such eye movements can be brought into play, but a conservative estimate suggests 150 msec, and exposures up to 200 msec may be acceptable. A tachistoscope (or its computer simulation) is the most common method for presentation of stimuli, but other methods have been used (see, for example, Dimond and Beaumont, 1974). The use of half-occluding contact lenses to limit vision to one visual field for longer periods, allowing the presentation of a wider variety of forms of stimulation including films and very complex detailed displays, was one exciting development, although the technique may not be entirely reliable and has not been widely taken up (Dimond, Bures, Farrington, & Brouwers, 1975; Zaidel, 1975).

It is obviously wise to maintain a check on the direction of fixation at the time of stimulus presentation, although this has been done less commonly than one might wish. However, both direct video and electro-oculographic monitoring of eye movements are possible and have been used with success (Dimond & Beaumont, 1972; Young, Bion, & Ellis, 1980). It has also generally been considered advisable to present stimuli unilaterally, that is, in one field at a time, and in a random sequence so that the subject cannot anticipate where the next stimulus will occur. This guards against the loss of accurate central fixation, and might be

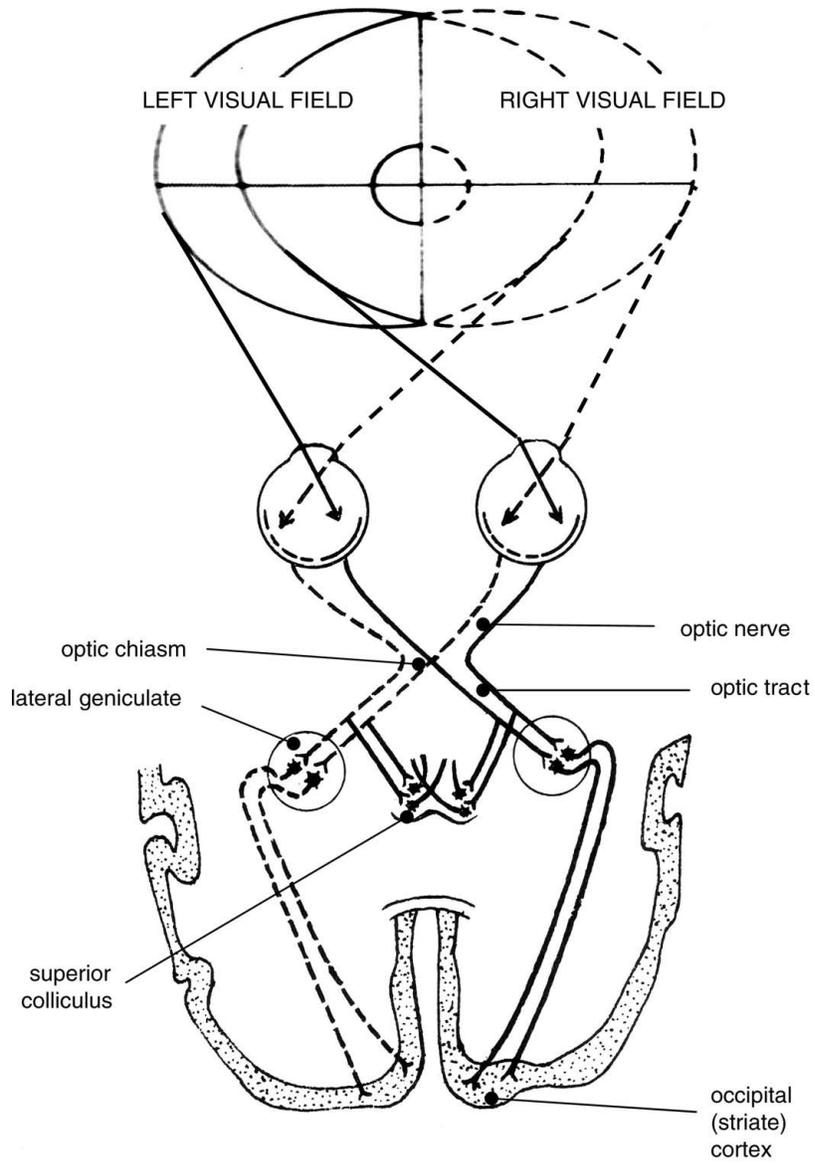


FIGURE 11.1. Anatomy of the visual pathways. Note how the left visual field projects to the right visual cortex (solid lines) and the right visual field to the left visual cortex (broken lines).

especially important where concurrent monitoring of fixation is not undertaken. However, quite a number of studies have used bilateral presentation, or “blocked” unilateral presentation, in which a series of stimuli all appear at the same side. There is remarkably good agreement between the bilateral and random unilateral studies, particularly where these involve some additional control over fixation, and where the bilateral studies report the order in which stimuli have been presented (either from the right, or left, visual field first). This is surprising in view of the general level of disagreement among published results and the heated debate that has surrounded the topic.

One extension of the technique, particularly with bilateral studies, has been the presentation of a “neutral” stimulus at central fixation, which is reported before the lateral stimuli as an aid to ensure accurate central fixation. It has been feared that this addition might affect the subsequent perception of the lateral stimuli because of the need to process and report the stimulus in the center, and although such an effect has never been clearly demonstrated, the method has become less popular as a result of this concern.

A final methodological question concerns the degree of overlap down the central meridian of the visual fields, and the eccentricity of the stimuli necessary to ensure lateralized presentation. We noted in Chapter 6 how there was an area of bilateral projection around central fixation that was often spared following unilateral lesions. There is also an area served with direct interhemispheric links through the splenium of the corpus callosum, around central vision. The precise extent of these two areas is not clearly established for humans, and it seems wise to avoid the central three degrees of vision around fixation when placing lateralized stimuli, although studies have reported visual field asymmetries with less eccentric stimuli.

Despite the fact that the method is in principle simple and elegant, some of the methodological problems connected with it are more than trivial (see Beaumont, 1982a; Young, 1982; Bourne, 2006). There has even been doubt cast over the whole field at various times, with suggestions that the findings might be due to something quite other than cerebral organization. However, none of these suggestions has ever satisfactorily explained away the very impressive body of evidence that has been accumulated. While the widespread adoption of the technique has been accompanied by some methodological laxity, the relatively coherent results that have been obtained in scores of different laboratories suggest that the technique is robust and generally reliable. It is now accepted as a valid method of investigating cerebral organization for performance in normal humans.

THE EVIDENCE

With such a large body of evidence to review, it is difficult to know how to divide it up sensibly for clear presentation. However, one of the earliest variables thought to underlie the appearance of a left or right visual field advantage was the verbal or nonverbal nature of the stimulus material, and this remains a useful way in which to classify the studies and the one that will be used here. Nevertheless, it should not be taken to indicate that this is the basis on which the observed hemisphere asymmetries rest. It is now clear, as we shall see, that a whole host of variables may influence any lateral advantage that emerges.

A comment is necessary about the terms *visual field asymmetry* and *advantage*. An asymmetry may be shown by either a right or a left visual field advantage (some people prefer to speak more accurately about the *hemifields*, and a *hemifield advantage*). A right visual field advantage, for example, is when performance shows either more accurate or more rapid response following stimulus presentation to the right of fixation than to the left. It is inferred that reception of the stimulus by the left cerebral hemisphere confers some advantage for the speed or accuracy of subsequent processing. Just how and why this occurs is the subject of the theoretical models that we shall discuss shortly. It should be noted as well that the reported lateral advantage or *lateral asymmetry* is usually based upon the mean results of a number of subjects whose data have been grouped together for statistical analysis. While an asymmetry may be clear from such data, it does not mean that these average results will be typical of each subject. There are wide individual differences (see Chapter 14), and this variability has been of concern to some writers (Colbourn, 1978).

Nonverbal Stimuli

Poffenberger in 1912 is generally credited as the first to show that there are asymmetries in the detection of simple undifferentiated stimuli such as patches of light. His results have been confirmed by modern experiments, and consist of two main findings. The first is that for a stimulus on a given side of the body, there will be a faster response with the hand on the same side than with the opposite hand. This is interpreted as reflecting the difference between the direct pathway in which detection and response initiation can be performed by the same hemisphere (see Figure 11.2, which already appeared as part of Figure 10.2), and the indirect pathway in which information must be passed across the corpus callosum after stimulus detection for response initiation by the opposite hemisphere. Some investigators have suggested that this model enables

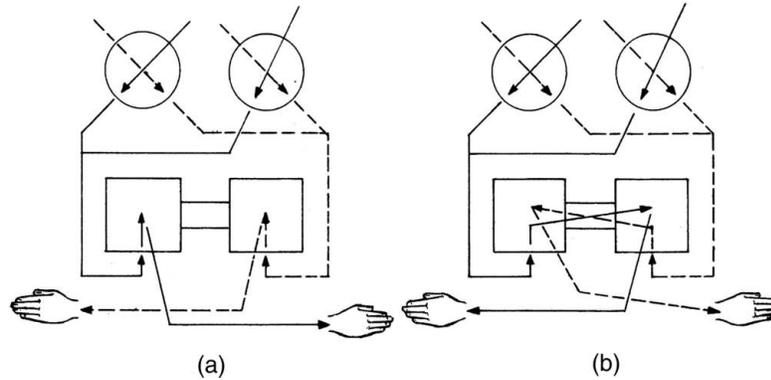


FIGURE 11.2. (a) Direct pathway: the right hand responds to the right visual field (solid line), and only one hemisphere, the left, is involved. (b) Indirect pathway: the left hand responds to the right visual field (solid line), and both hemispheres and the corpus callosum are involved.

an estimate to be made of the time taken to transfer information across the callosum (IHTT: interhemispheric transfer time), with a typical result of about 4 msec, for a simple experiment of this kind. However, it is clear that the model involving a single callosal transfer is too simple, particularly with more complex stimuli and tasks, and that is why firm values for IHTT have not been obtained. Incidentally, the values for IHTT of up to 50 msec, which are often found, are very much greater than would be expected from what is known about physiological rates of neural transmission across the commissures (Cherbuin & Brinkman, 2006).

It might well be pointed out that *compatibility* between stimulus and response is an established phenomenon in human performance, and that to respond with the hand on the same side as the stimulus is a more compatible response. Whether this might explain the finding has been elegantly tested by Berlucchi and co-researchers, by the simple expedient of requiring subjects to undertake trials in which the forearms were crossed and the hands therefore crossed over, thus associating the compatible alternative with the indirect, not the direct, route (Berlucchi, Crea, DiStefano, & Tassinari, 1977). Their results show that while response compatibility is a factor in determining the differences between hands, the anatomical differences between direct and indirect routes are more important.

The second main finding to come out of these experiments is that superimposed upon the advantage of direct over indirect routes is a *lat-*

eral asymmetry, with the advantage for stimuli in the left visual field. This finding applies not only to flashes of light, but also to other simple stimuli to be detected, such as a solid filled dot among an array of unfilled dots. The finding is not universal (almost none is, using this technique), but there seems general agreement on a right hemisphere advantage for simple stimulus detection.

A similar advantage has been found for sustained attention in a *vigilance* paradigm by Dimond (1979), who has suggested that the right hemisphere might contain the primary mechanisms for watch keeping (a task such as continuous monitoring of a radar display), although not all investigators have agreed with this interpretation. Similar fundamental differences have been suggested by others to account for asymmetries in elementary perceptual processes. The masking and visual persistence investigations that experimental psychologists use to study the initial stages of perception have been undertaken in this context. During such stages, stimuli have to be selected from a barrage of sensory stimulation, and processed to be ready for categorization and subsequent processing for recognition and storage. There is some evidence that these pre-categorical mechanisms, whereby stimuli are detected but not yet fully identified, may differ between the hemispheres, favoring the right, but there is also criticism of the experiments that have been performed (Cohen, 1976; Hellige & Webster, 1979).

Hemisphere asymmetries have also been sought for a variety of *simple perceptual variables*. Judgment of brightness has been shown to result in reports of greater brightness associated with the right hemisphere. Similarly, color perception seems to be better performed by the right hemisphere, although only if a relatively difficult discrimination is demanded. This last aspect is a general feature of these studies. It seems that the task must be perceptually demanding, either because the stimuli are hard to discriminate or because they are relatively complex, if the right hemisphere advantage is to be observed. Another aspect of this is the extent to which stimuli might be named, which brings us directly to the fundamental problem with our scheme: it is very difficult to decide when a stimulus is "verbal" or "nonverbal." Stimuli that are figural and meaningful are often relatively easy to name or to "verbalize," which brings verbal characteristics into our "nonverbal" stimulus. Conversely, even words have outlines, length, and so on, and although primarily verbal they have nonverbal attributes. It is sometimes suggested that for a right hemisphere advantage to be clearly observed, stimuli should be perceptually complex and difficult to verbalize, and that this is what is implied by "nonverbal."

Not only brightness and color, but also depth perception, especially in binocular vision, motion perception, and stimulus enumeration, seems

generally to be associated with a right hemisphere advantage. Detection of the length of lines is very difficult to test if a verbal response is to be avoided (so as not to encourage verbalization) and other lateral asymmetries in perception are not to interfere with the result. The findings for stimulus duration are similarly rather unclear because of some difficult procedural and methodological problems. It seems at least possible, however, that some aspects of the perception of duration are linked with a left hemisphere advantage. Two final stimulus variables, localization and orientation, have been associated with mixed results, although once again some sense can be made of the data by considering verbal codability. In general a right hemisphere advantage has been observed, and when a left hemisphere advantage has been found it can be explained by the inclusion of a frame or some other background that might aid coding. Even with stimuli that are (arguably) quite codable, such as a clock face, or with horizontal or vertical stimuli, a left visual field superiority has been found.

Form perception has produced unclear results. White in his review (1972) found no evidence of an asymmetry, and the picture is no less uncertain now. To some extent the outcome depends on the complexity of the stimuli, as studies that have used the classic Vanderplas and Garvin figures in identification or matching tasks (see Figure 11.3) have shown. Even so, the results are not simply that more complex figures (with more corners) produce a right hemisphere advantage, or that the “association value” of the stimuli (which is an index of how verbalizable or codable they are) predicts the asymmetry observed. What is clear is that when complex representations of nameable objects are presented, which are presumably readily associated with a name, then a left hemisphere advantage normally ensues (Andresen & Marsolek, 2005).

Some interesting results have been found with the signs used by the deaf. Adults who cannot “read” them, and for whom they are a meaningless configuration, generally show a left visual field advantage when they are shown, but hearing adults who understand them show, like the deaf, a right visual field advantage (Davidoff, 1982). This fits with the idea that it is semantic or linguistic properties that are linked with a left hemisphere advantage.

Faces have been much investigated as stimuli. In general they yield a right hemisphere advantage, although the results are rather complex. Interestingly, schematic faces produce a much less clear asymmetry than do photographs of real faces. However, the right hemisphere seems better at recognizing and matching faces than the left, and also at recognizing emotions. It was suggested at one time that the hemispheres, right and left, might be linked with negative and positive emotions respectively, but it now seems more likely that the right hemisphere is superior

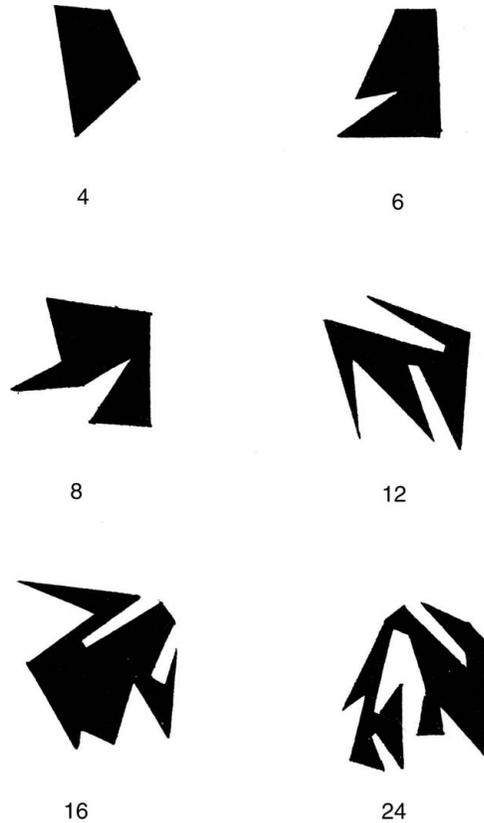


FIGURE 11.3. Examples of the Vanderplas and Garvin random shapes, with the number of corners as shown. (After J. M. Vanderplas and E. A. Garvin, *Journal of Experimental Psychology*, 57 (1959), 147–154.)

at emotional perception from faces, whatever the emotion (Burton & Levy, 1991; Bourne & Hole, 2006).

If the face is to be named, what should we expect: a right hemisphere advantage because it is a face, or a left hemisphere advantage because it has to be named? In fact, researchers have disagreed, although the balance of evidence seems slightly in favor of a right hemisphere advantage. The duration of presentation may be important here. In some experiments the faces were presented for less time than it normally takes to recognize them in central vision, with the result that basic perceptual aspects of the stimuli may have been emphasized at the expense of the properties that would normally be attended to. Upright and inverted

faces have been contrasted in an attempt to control for the perceptual aspects of the stimuli, but just what cognitive processes are invoked by an inverted face seems unclear (Leehey & Cahn, 1979; Young & Bion, 1981).

An intriguing study by Chabris and Hamilton (1992) examined chess masters, who are known to be superior at encoding and recalling the arrangement of a position during a game of chess; they possess a particular semantic structure of the “chess domain.” The study found the right hemisphere of the chess masters to be superior at understanding “sensible” chess positions, but the left hemisphere to be better at grouping pieces in unorthodox positions. The right hemisphere appears critical for chess skill.

It is difficult to summarize the above information on visual field advantages without doing grave injustice to it. Those interested in this topic are advised to consult the extensive review by Davidoff (1982). However, repeating the warning that there are many contrary findings, it seems that if basic low-level perceptual functions are examined, then some, but not all, may yield visual field asymmetries. When an asymmetry is found, it tends to be a right hemisphere advantage. Stimulus complexity and duration (with shorter durations producing perceptually more difficult discriminations) are implicated in determining a right hemisphere superiority. With more complex stimuli, familiar drawings with names produce a left hemisphere advantage, while faces and certain shapes may be associated with a right hemisphere advantage. The verbalizability of the stimuli may be an important variable, as may their symbolic content.

Verbal Stimuli

The results of studies using “verbal” stimuli have been less contradictory than the “nonverbal” studies. Certainly if stimuli such as words, strings of letters, and single letters or digits are presented for identification or matching, a strong left hemisphere (right visual field) advantage is normally found (Beaumont, 1982b). In cases where the effect has not been found, there is often reason to suspect that the stimuli have been processed on their perceptual rather than their verbal attributes. When words or letter strings are presented, the right hemisphere superiority (in responding to stimuli in the left visual field) is found whether the presentation is horizontal or vertical, although the horizontal orientation tends to produce the stronger effect. This demonstrates that while left-to-right scanning patterns, perhaps based upon reading habits, may have some influences on the left field superiority, they cannot be a complete explanation (Day, 1979).

What is surprising about these studies in view of the conclusions we drew previously is that *linguistic parameters* do not seem to relate directly to the left hemisphere advantage. The use of concrete nouns contrasted with abstract nouns, the manipulation of imageability, nouns opposed to verbs, or the effects of word frequency do not seem to relate in any coherent way to the left hemisphere superiority (Day, 1977; Bradshaw & Gates, 1978; Cousin, Peyrin, & Baciú, 2006). Once a lateral asymmetry for the recognition of linguistic stimuli had been found, it was confidently expected that the more “verbal” and less “nonverbal” a stimulus (for example, abstract versus concrete nouns), then the stronger would be the lateral advantage. This has not been clearly demonstrated. Even with strings of letters, or “nonsense” words, it does not seem to be the case that there is a left visual advantage if the string is more pronounceable (“BOV” rather than “ZVH”), or more similar to meaningful English words (Leiber, 1976). This very much undermines the assumption that left hemisphere superiority is determined by the operation of purely linguistic processes.

Although identification of stimuli for simple report or matching has been the task most commonly employed, certain more demanding tasks have also been used. A particularly important one has been the presentation of letter stimuli for physical and nominal matching (Cohen, 1972; Hellige, Cox, & Litvac, 1979; Ledlow, Swanson, & Kinsbourne, 1978). This is a task much studied in cognitive psychology, and requires the subject to determine whether two letters are identical (physical match: “AA but not Aa”), or similar in name (nominal match: “AA or Aa, but not AB”). Specific instructions are given to the subject as to which type of match to perform, and thus determine the cognitive demands of the task. In general, the requirement to make a physical match is associated with a right hemisphere advantage, at least for the “same” responses, although this has not universally been found. Where a nominal match is required, then a left hemisphere superiority is almost always found, although it is again clearer for “same” responses.

It is interesting that the distinction between nominal and physical matching may reflect on tasks where the type of match required is not clearly specified. Although the type of matching may be inherently clear in the form of stimuli presented, subjects may nevertheless adopt different *cognitive strategies* (Bryden, 1978); that is, they will adopt some preferred or habitual approach to solving the problem of determining a match. If different subjects select different strategies, then this may account for some of the variability found in the research reports. Also, the fact that a left hemisphere advantage is generally found for simple word or letter matching implies that although a physical matching strategy might be sufficient, subjects nevertheless generally adopt a nominal

matching strategy. The extent to which very subtle aspects of the experimental instructions and procedure may influence the adoption of cognitive strategies is also of concern to some investigators.

A delay may be introduced between the elements to be matched in the physical/nominal matching paradigm, in order to investigate the role of memory components in determining lateral asymmetry. Some researchers have found that introducing a delay in physical matching produces a shift from right to left hemisphere advantage, implying the necessity of verbally encoding the stimulus, but this has been contested in recent reports (McCarthy, 1980). Nevertheless, what these experiments clearly demonstrate is that purely cognitive factors may influence lateral asymmetry, so that even though the stimuli and presentation conditions may be identical, the task demanded may determine whether a left or right hemisphere advantage is found.

An obvious question that arises is the part played by the *response* in producing a lateral asymmetry. If a left hemisphere advantage were only to be found when a vocal or right hand response was demanded (both initiated presumably by the left hemisphere), this might well explain the phenomenon. The problem is that it is very difficult to change the response without to some degree changing the task. However, when a manual response is required, most investigators have demanded responses with each of the hands in a balanced order, and some have subsequently analyzed the effect of this variable (Besner, Grimsell, & Davis, 1979; Segalowitz, Bebout, & Lederman, 1979). The results are conflicting, and this may reflect the suspicion that quite subtle aspects of the response, such as how fine a movement is required (with more distinctly contralateral control), may be crucial variables.

It is more difficult to compare manual with vocal responses than to compare responses with the two hands, although in general the evidence is that vocalization in itself is not critical in determining the left hemisphere advantage. This has been shown by contrasting a meaningful response with a vocal, but meaningless, response (such as “pring”) in a go/no-go paradigm where the subject either responds or refrains from responding. Bradshaw and Gates (1978), studying lexical decision (“Is the stimulus a word?”), found a stronger left hemisphere advantage with overt naming than with manual response. Most researchers have been content to assume that response systems are relatively independent of other cognitive operations and, beyond balancing hand of response, have not taken much account of response variables.

Another factor that, it has been suggested, is important in determining lateral asymmetries is the *size of the stimulus set* (Hardyck, Tzeng, & Wang, 1978). This conclusion was based on a series of experiments in which matching was performed between various combinations of Eng-

lish words and Chinese characters by English and bilingual Chinese subjects. Part of the aim of the experiment was to compare the lateral advantage for perceptually complex characters when they were meaningless (English subjects) or possessed semantic properties (Chinese subjects). It was found, however, that asymmetries appeared only when a very restricted set of possible stimulus items was employed. The suggestion is that the subject must know the range of possible stimuli, or rapidly deduce it, and then adopt a strategy that involves “referencing a table of known values,” if a left hemisphere advantage is to be found with verbal stimuli (see also Chiarello et al., 2006). However, there has been no formal analysis of the literature to support this hypothesis, and many examples can be found where new information is presented on each trial and a right visual field superiority is nevertheless found.

Memory of course plays some role in almost any experiment, but does it play some fundamental role in lateral asymmetry? Studies that have explicitly required a target item or a target set to be held in memory, and to which later stimuli must be matched, have generally yielded a left hemisphere advantage. This might be expected on the basis of the verbal material alone. However, that requiring memory for verbal items does not necessarily produce a left hemisphere advantage is shown by the experiment of Klatzky and Atkinson (1971), who asked subjects to hold verbal items in memory and then presented either single letters or pictures of objects. The initial letter of the depicted object, or the letter presented, were to be matched to items in memory. A left hemisphere advantage emerged for the pictures, but not the letters. All this brings us back to the problem of strategies. We cannot know that subjects will necessarily adopt semantic coding for verbal items, for they might alternatively attend to the spatio-perceptual elements of the stimulus, and attempts to manipulate such strategies directly have not generated clear results (Metzger & Antes, 1976). Memory components are clearly implicated in lateralized cognitive processing, but there is no reason to believe that they are entirely responsible for lateral visual asymmetries.

Practice is another variable that complicates the investigation of hemisphere specialization (Jonides, 1979; Ward & Ross, 1977). Practice clearly does have some effect, but just how is not yet clear. The nature of the stimuli and the task, the difficulty of the task, the familiarity of the material and the experimental paradigm, the compatibility of the response, and the pattern of test trials and rest pauses all interact with the effects of practice. Two factors may be at work, however. One is that with increasing familiarity with the stimuli and task, there is a shift from attention to the spatial and configurational properties of the stimulus material to a processing mode with important verbal and semantic com-

ponents. This would be reflected in a shift from right hemisphere advantage to left hemisphere advantage. At the same time, increasing familiarity with the task might reduce the task's difficulty and consequently the strength of lateral asymmetry. These two factors will work to some extent in opposition, and interacting with the host of other variables may result in the very complex results that have been reported.

Finally, we might note that *additional tasks* have sometimes been added to the primary visual field task in an attempt to elucidate the nature of the lateralized processes (Kinsbourne & Hicks, 1978). In general the addition of a concurrent verbal task to a divided visual field task has been found to reduce or reverse the left hemisphere advantage normally observed. There are difficulties, however, in interpreting such studies (Cohen, 1979; Hellige, Cox, & Litvac, 1979). One simple problem is the ambiguity of *activation* and *interference*. Interpretations have suggested that the secondary task may activate one hemisphere and so make its contribution to processing the divided visual field task more prominent. On the other hand, it may interfere with processing, and so cause greater participation of the opposite hemisphere. Such activation and interference will result in opposite effects on lateral asymmetry, but there is no way of knowing which will apply, so that although with increasing secondary task difficulty there may be a shift from activation to interference, there is no independent way of knowing when this will occur. These concepts have tended to result in most unsatisfactory post hoc explanations of experimental results, which have done little to further our understanding of lateralized cognitive processes.

In conclusion, a clear left hemisphere advantage is generally found in studies that require identification of "verbal" stimuli, such as words, letters, and letter strings or digits, although this should not lead us to deny that the right hemisphere is entirely without such functions (Querné, Eustache, & Faure, 2000). The role played by semantic and linguistic parameters remains unclear but does not at present satisfactorily explain the left hemisphere advantage. If physical matching is required, a right hemisphere advantage may be found, depending upon cognitive variables inherent in the task. These results are relatively independent of response mode, although both this and mnemonic processes may play some part in determining lateral asymmetry. Practice is a complex variable that deserves more study, and the whole picture is further complicated by other subject and task variables. Nevertheless, over a very considerable number of experiments, the stability and importance of the left hemisphere advantage in association with verbal stimuli remain clear. (A more detailed and fully referenced review is to be found in Beaumont, 1982b.)

THE THEORIES

There has been a tendency for divided visual field studies to be reported independently of any explicit theoretical foundation. Visual field differences have thus been reported as if they were little nuggets of fact that had simply been dug up and deposited on a mound of knowledge. Such an approach is limited. At best, data accumulate and can constitute a resource for those who want to understand the processes that underlie performance asymmetries. At worst, the hypotheses and assumptions made in collecting the data are never made explicit and yet play a part in determining the findings that are reported. An associated problem, deriving from the ease with which divided visual field experiments may be carried out, is the large number of small-scale isolated studies that report on a particular task under a single set of conditions. What the theory builder needs are extended studies in which conditions are systematically varied in order to clarify the mechanisms that produce the phenomenon under study.

While several theories accounting for lateral asymmetries have been proposed, there has been little critical conceptual analysis of the various theoretical positions. However, in addition to Bryden's 1978 review, there is also an invaluable analysis by Cohen (1982). Indeed, the classification of theoretical models of hemisphere specialization suggested below (and see Figure 11.4) owes a great deal to her insightful and creative review.

Structural Models

This has been the most common variety of model adopted in divided visual field researches. If a model is implicit, then it is usually a structural model that is assumed. The general idea behind these models is that psychological functions may show lateral asymmetries, as we know from the behavioral data, and these functions may be localized in cerebral structures that are lateralized to one of the cerebral hemispheres. In this the models reflect current concepts in clinical neuropsychology.

Structural models do not necessarily exclude a variety of other factors that may contribute to lateral asymmetries, such as postexposural scanning or the influence of reading habits. They do, however, assume that when such factors are not themselves a function of cerebral lateralization, they are of minor importance by comparison with the structural cerebral factors.

This raises a whole host of conceptual issues about the allocation of *cognitive* functions to *physiological* structures, and accounts for the

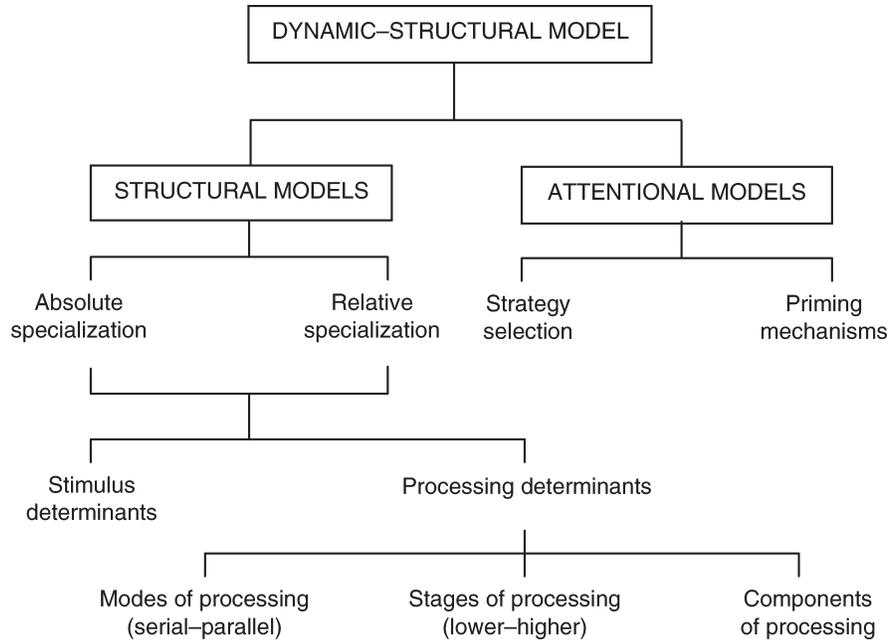


FIGURE 11.4. Theoretical models of hemisphere specialization. (Modified from Cohen, 1982.)

rather unsatisfactory status of some of the concepts used in building structural models. There is insufficient space to deal with the topic here (see Beaumont, 1982c), but greater attention should be devoted to this philosophical problem.

Structural models may be based upon either absolute or relative specialization. Where the specialization is thought to be *absolute*, some mechanism must be proposed to effect the transfer of stimulus information if it arrives at the “wrong” hemisphere. This transfer might be automatic, so that all information is transferred and is available to both hemispheres, but only the appropriate hemisphere takes up the processing. Transfer might alternatively be conditional, either following assignment by some initial sorting mechanism, or following the failure of the receiving hemisphere to complete the required processing. A further alternative is that transfer may occur and be followed by both hemispheres processing the material, with the specialized hemisphere reaching a conclusion faster, or some mechanism arbitrating the results. Davis and Schmit (1973) provide an example of such a model. There are prob-

lems with all these alternatives. They all seem to rely on mechanisms (for allocation of processing, for deciding when processing has failed, or for arbitrating between solutions) that have an unspecified location and for which there is little evidence. Moreover, in certain cases the systems are wasteful and implausible, such as dual simultaneous processing.

There is also difficulty in squaring absolute specialization models with the split-brain evidence we noted in the last chapter, which suggests (albeit in abnormal patients) that both hemispheres possess some capacity for most functions. Differential error rates may be attributed to “degradation” in callosal transfer, which seems implausible in the large degree often claimed, when other transmission pathways, from primary up to tertiary cortex, are used with much higher fidelity. The failure to establish good stable estimates of IHTT and to account for within-individual shifts in laterality also argues against the adoption of an absolute specialization model.

On the other hand *relative specialization* models, in which both hemispheres are believed to be capable of performing most functions but with different levels of efficiency, have the problem that they are too imprecise. They fit very well with most of the data from a variety of sources, but it is very difficult to derive firm predictions from them. Even the inference that increasing difficulty should be associated with greater asymmetry cannot generate firm hypotheses because of the inadequate definition of difficulty, and this imprecision is in turn reflected in confusion among the findings. Thus, although the relative specialization model in many ways seems more acceptable than the absolute model, it only gains its acceptability by a serious loss in explanatory power.

Structural models may be based on either stimulus or processing determinants. We have already seen how inadequate was the division of stimulus type into “verbal” and “nonverbal” as a predictor of lateral asymmetry. It nevertheless figured prominently in early theories and still underlies the assumptions in many procedures designed to exercise left or right hemisphere functioning.

It is now more fashionable to consider *processing determinants*. These were initially considered in terms of a number of dichotomies that assigned serial, sequential, temporal, or analytic processing to the left hemisphere and parallel, holistic, or gestalt processing to the right. Many have treated these terms as roughly equivalent, which they are not. The description of the processes has frequently been post hoc, and firm criteria for the identification of such processes have almost always been omitted. Although experimental techniques exist for the identification of such processes, they have rarely been employed, and when they

have been used, the results have not always been clear (Cohen, 1973; Patterson & Bradshaw, 1975). Of the mode of processing schemes, none accounts well for the experimental data.

The most recent formulations feature *stages of processing*. These may be in terms of component operations (Cohen, 1977) or information-processing models (Moscovitch, 1979).

The component operations model features the contribution of a number of elementary components in cognitive processing, each of which may be characterized by hemisphere specialization. Lateralization will then be determined by the specialization inherent in visual analysis, phonological analysis, or semantic analysis as each is called into operation. This approach is useful in that it allows for some flexibility in the operation of the contributory processes, and it can account for task variability if this is mediated by strategy selection. It suggests a way forward in the attempt to identify and specify the exact nature of the contribution of each element. However, like relative specialization, it is weak at generating firm and testable hypotheses, and perhaps only succeeds in shifting the phenomenon of lateralization to another part of the model without explaining it.

By contrast the information-processing model is more precise. It assumes a model with a clear sequence of processes from perception through categorization to memory and response (from low-level through to high-level). Lateralization is presumed to be characteristic of certain of these stages (mid- and high-level) and is carried through to later stages. The attraction of the model is that it has clear reference to general models in cognitive psychology, although cybernetic models now seem a little old fashioned in this context. The problem is that while the model is precise and relatively well formulated (although Cohen is highly critical of certain aspects), it does not always fit well with the data, and is for this reason not entirely acceptable.

Two more recent developments are worth noting. The first concerns the differentiation of categorical processes, in that a left hemisphere advantage has been demonstrated for “what” (lightness) and “where” (location) systems, while a right hemisphere bias was found for “whatever” (coordinate processing) systems (Parrot, Doyon, Démonet, & Cardebat, 1999). The second is the HERA model: hemispheric encoding/retrieval asymmetry. In this model the prefrontal cortices of the left and right hemispheres are respectively involved in episodic encoding and retrieval in memory. Developed originally from imaging data (see Chapter 13), the model has been supported by divided visual field data (Blanchet et al., 2001), although how these different processes are related to lateralized modes of processing is not yet entirely clear.

Attentional Models

Attentional models are a more recent development and have not gained such wide popularity. The idea underlying them is that the cerebral system has a certain capacity that can be allocated in a flexible way. Arousal, activation, and expectancy will result in attention and associated processing capacity being dedicated (and divided between the hemispheres) in a particular way. The most notable exponent of this form of model has been Kinsbourne (1975).

Such models do not deny the existence of structural differences but claim that these are small in relation to the effects of attentional variables. These effects can be best seen, and are accessible for study, in the mechanism of *priming*. Priming may be determined by stimulus location, by stimulus type, or by a concurrent task. When the stimulus location is predictable (or when the subject adopts a strategy of expecting stimuli at a given location, even if presentation is random), then the subject may direct attention (although not necessarily his or her gaze) to that area of space, resulting in better perception of stimuli at that location.

The stimulus type may prime the hemispheres either by putting the “appropriate” hemisphere in readiness for a certain type of stimulus, or by directing attention to the side of sensory space opposite the appropriately primed hemisphere. Both these mechanisms would result in better performance in the lateral visual field opposite the more specialized hemisphere. The degree to which such a mechanism is supported by the evidence is uncertain. Some experiments using mixed trials find effects that could be explained in terms of stimulus priming (Berlucchi, Brizzolara, Marzi, Rizzolatti, & Umiltà, 1974), but others have failed to find any specific effect of stimulus type expectancy, even when the expectancy can be shown to have an effect on overall performance. Beaumont and Colley (1980), for example, presented subjects with a mixed sequence of shapes or words that appeared randomly in the left or right visual field. In a given set either shapes or words were more common, and the subject was clearly informed about this. While subjects responded more rapidly to the more common type of stimulus in any set, whether a stimulus type was more or less common did not affect the asymmetry for that stimulus type, as attentional models would predict. The effects of perception in the unprimed hemisphere are not, however, always as would be expected from attentional models: presenting pairs of stimuli that could be any pairing from among words, faces, and shapes, presented in balanced order to the two hemispheres, Hines (1975) found only limited support for an attentional explanation of the results.

These models have been based upon the hypothesis of attentional bias, but variants of the attentional model have been suggested by Fried-

man, Polson, Dafoe, and Gaskill (1982) based upon the independent allocation of attentional resources within the two hemispheres, and also through differing allocation of attentional resources, by Nicholls and co-investigators (Nicholls, 1994; Lindell & Nicholls, 2003).

Reference has already been made to the use of concurrent tasks and the difficulties of interpreting the results of such studies. Nevertheless, some support for attentional models comes from such work. Kinsbourne (1973) required subjects to report the location of a small gap in the outline of a square while performing a concurrent task. When this task was to hold a string of words in memory, a left hemisphere advantage was found, but when remembering a melody, a right hemisphere advantage. Such studies are difficult to explain without recourse to attentional mechanisms, but models that rely on secondary task performance are nevertheless generally inadequate in their explanatory power.

The Dynamic-Structural Model

Despite its rather grand title, this model really does no more than accept that at present there is good evidence for both structural and attentional models. Both have weaknesses and strengths. While the dynamic-structural model has not yet been clearly formulated, the approach is implicit in much current research, and seems the only sensible way to proceed for the moment. Most researchers would regard the structural aspects as more important than the attentional processes, but few wish to deny the real contribution of attentional mechanisms. We are left, therefore, with a model that combines structural features with attentional processes. The structural components confer relative specialization determined by the contribution of particular cognitive processes. The attentional mechanisms include priming in association with cognitive strategy selection. We cannot pretend that this is a satisfactory model of cerebral specialization, but it seems to be the best sense that we can make of the experimental findings that have been accumulated to date.

CONCLUSION

The divided visual field technique has been the principal method of investigation in experimental human neuropsychology, and has been employed in a large body of research. Somewhat confused findings have resulted but nevertheless, out of this mass of data, a number of conclusions have emerged. These conclusions are now accepted as showing

that there are lateral differences in the specialization of the cerebral hemispheres for psychological functions. These differences seem, in general, to reflect the form of cerebral organization that has been inferred from the study of clinical patients.

The mechanisms that produce these lateral asymmetries are still unclear. It is not possible to say why, for example, the recognition of words is associated with superior right visual field performance. However, it is reasonable to believe that certain aspects of cognitive processing, as yet poorly specified but conceptualized in terms, perhaps, of linguistic or phonological or semantic analysis, are more effectively performed in the left cerebral hemisphere. The lateral advantage observed in such processes may be modified by the operation of attentional variables.

This may seem a rather weak conclusion in view of the research effort devoted to the topic. Nevertheless it comprises a significant advance in understanding a very complex dynamic system that, until the introduction of methods such as the divided visual field technique, remained a dark mystery, largely hidden to scientific investigation.

FURTHER READING

In addition to the general references given at the end of Chapter 1, and references generally relevant for experimental neuropsychology at the end of Chapter 10, a rather dated but comprehensive and useful review is:

Beaumont, J. G. (Ed.). *Divided Visual Field Studies of Cerebral Organisation* (London: Academic Press, 1982).

An updated review of methodological considerations can be found in:

Bourne, V. J. The Divided Visual Field Paradigm: Methodological Considerations, *Laterality*, 11 (2006), 373–393.

Similarly, still useful and relevant contributions will also be found in:

Dimond, S. J., & Beaumont, J. G. (Eds.). *Hemisphere Function in the Human Brain* (London: Elek Science, 1974).

Kinsbourne, M. (Ed.). *Asymmetrical Function of the Brain* (Cambridge, UK: Cambridge University Press, 1978).

REFERENCES

Andresen, D. R., & Marsolek, C. J. Does a Causal Relation Exist Between the Functional Hemispheric Asymmetries of Visual Processing Subsystems?, *Brain and Cognition*, 59 (2005), 135–144.

- Beaumont, J. G. Methods for Studying Cerebral Hemisphere Function, in A. W. Young, ed., *Functions of the Right Cerebral Hemisphere* (London: Academic Press, 1982a).
- Beaumont, J. G. Studies with Verbal Stimuli, in J. G. Beaumont, ed., *Divided Visual Field Studies of Cerebral Organisation* (London: Academic Press, 1982b).
- Beaumont, J. G. Neuropsychology and the Organisation of Behaviour, in A. Gale & J. Edwards, eds., *Physiological Correlates of Human Behaviour* (London: Academic Press, 1982c).
- Beaumont, J. G., & Colley, M. Attentional Bias and Visual Field Asymmetry, *Cortex*, 16 (1980), 391–396.
- Berlucchi, G., Brizzolara, D., Marzi, C. A., Rizzolatti, G., & Umiltà, C. Can Lateral Asymmetries in Attention Explain Interfield Differences in Visual Perception?, *Cortex*, 10 (1974), 177–185.
- Berlucchi, G., Crea, F., DiStefano, M., & Tassinari, G. Influence of Spatial Stimulus-Response Compatibility on Reaction Time of Ipsilateral and Contralateral Hand to Lateralized Light Stimuli, *Journal of Experimental Psychology: Human Perception and Performance*, 3 (1977), 505–517.
- Besner, D., Grimsell, D., & Davis, R. The Mind's Eye and the Comparative Judgement of Number, *Neuropsychologia*, 17 (1979), 373–380.
- Blanchet, S., Desgranges, B., Denise, P., Lechevalier, B., Eustache, F., & Faure, S. New Questions on the Hemispheric Encoding/Retrieval Asymmetry (HERA) Model Assessed by Divided Visual-Field Tachistoscopes in Normal Subjects, *Neuropsychologia*, 39 (2001), 502–509.
- Bourne, V. J. The Divided Visual Field Paradigm: Methodological Considerations, *Laterality*, 11 (2006), 373–393.
- Bourne, V. J., & Hole, G. J. Lateralized Repetition Priming for Familiar Faces: Evidence for Asymmetric Interhemispheric Cooperation, *Quarterly Journal of Experimental Psychology*, 59 (2006), 1117–1133.
- Bradshaw, J. L., & Gates, E. A. Visual Field Differences in Verbal Tasks: Effects of Task Familiarity and Sex of Subject, *Brain and Language*, 5 (1978), 166–187.
- Bryden, M. P. Strategy Effects in the Assessment of Hemispheric Asymmetry, in G. Underwood, ed., *Strategies of Information Processing* (London: Academic Press, 1978).
- Burton, L. A., & Levy, J. Effects of Processing Speed on Cerebral Asymmetry for Left- and Right-Oriented Faces, *Brain and Cognition*, 15 (1991), 95–105.
- Chabris, C. F., & Hamilton, S. E. Hemispheric Specialization for Skilled Perceptual Organization by Chessmasters, *Neuropsychologia*, 30 (1992), 47–57.
- Cherbuin, N., & Brinkman, C. Efficiency of Callosal Transfer and Hemispheric Interaction, *Neuropsychology*, 20 (2006), 178–184.
- Chiarello, C., Kacinik, N. A., Shears, C., Arambel, S. R., Halderman, L. K., & Robinson, C. S. Exploring Cerebral Asymmetries for the Verb Generation Task, *Neuropsychology*, 20 (2006), 88–104.
- Cohen, G. Hemisphere Differences in a Letter Classification Task, *Perception and Psychophysics*, 11 (1972), 139–142.

- Cohen, G. Hemispheric Differences in Serial Versus Parallel Processing, *Journal of Experimental Psychology*, 97 (1973), 349–356.
- Cohen, G. Components of the Laterality Effect in Letter Recognition: Asymmetries in Iconic Storage, *Quarterly Journal of Experimental Psychology*, 28 (1976), 105–114.
- Cohen, G. *The Psychology of Cognition* (London: Academic Press, 1977).
- Cohen, G. Comment on “Information Processing in the Cerebral Hemispheres: Selective Attention and Capacity Limitations” by Hellige, Cox and Litvac, *Journal of Experimental Psychology: General*, 108 (1979), 309–315.
- Cohen, G. Theoretical Interpretations of Lateral Asymmetries, in J. G. Beaumont, ed., *Divided Visual Field Studies of Cerebral Organisation* (London: Academic Press, 1982).
- Colbourn, C. J. Can Laterality be Measured?, *Neuropsychologia*, 16 (1978), 283–289.
- Cousin, E., Peyrin, C., & Baciú, M. Hemispheric Predominance Assessment of Phonology and Semantics: A Divided Visual Field Experiment, *Brain and Cognition*, 61 (2006), 298–304.
- Davidoff, J. Studies with Nonverbal Stimuli, in J. G. Beaumont, ed., *Divided Visual Field Studies of Cerebral Organisation* (London: Academic Press, 1982).
- Davis, R., & Schmit, V. Visual and Verbal Coding in the Interhemispheric Transfer of Information, *Acta Psychologica*, 37 (1973), 229–240.
- Day, J. Right Hemisphere Language Processing in Normal Right Handers, *Journal of Experimental Psychology: Human Perception and Performance*, 3 (1977), 518–528.
- Day, J. Visual Half Field Word Recognition as a Function of Syntactic Class and Imageability, *Neuropsychologia*, 17 (1979), 515–519.
- Dimond, S. J. Symmetry and Asymmetry in the Vertebrate Brain, in D. A. Oakley & H. G. Plotkin, eds., *Brain, Behaviour and Evolution* (London: Methuen, 1979).
- Dimond, S. J., & Beaumont, J. G. On the Nature of the Interhemispheric Effects of Fatigue, *Acta Psychologica*, 36 (1972), 443–449.
- Dimond, S. J., & Beaumont, J. G. Experimental Studies of Hemisphere Function in the Human Brain, in S. J. Dimond & J. G. Beaumont, eds., *Hemisphere Function in the Human Brain* (London: Elek Science, 1974).
- Dimond, S. J., Bures, J., Farrington, L. J., & Brouwers, E. Y. M. The Use of Contact Lenses for the Lateralisation of Visual Input in Man, *Acta Psychologica*, 39 (1975), 341–349.
- Friedman, A., Polson, M. C., Dafoe, C. G., & Gaskill, S. J. Dividing Attention Within and Between Hemispheres: Testing a Multiple Resources Approach to Limited-Capacity Information Processing, *Journal of Experimental Psychology: Human Perception and Performance*, 8 (1982), 625–650.
- Hardyck, C., Tzeng, O. J. L., & Wang, W. S.-Y. Cerebral Lateralization of Function and Bilingual Decision Processes: Is Thinking Lateralized?, *Brain and Language*, 5 (1978), 56–71.
- Hellige, J. B., Cox, P. J., & Litvac, L. Information Processing in the Cerebral

- Hemispheres: Selective Hemispheric Activation and Capacity Limitations, *Journal of Experimental Psychology: General* 108 (1979), 251–279.
- Hellige, J. B., & Webster, R. Right Hemisphere Superiority for Initial Stages of Letter Processing, *Neuropsychologia*, 17 (1979), 653–660.
- Hines, D. Independent Functioning of the Two Cerebral Hemispheres for Recognizing Bilaterally Presented Visual Half Field Stimuli, *Cortex*, 11 (1975), 132–143.
- Jonides, J. Left and Right Visual Field Superiority for Letter Classification, *Quarterly Journal of Experimental Psychology*, 31 (1979), 423–439.
- Kinsbourne, M. The Control of Attention by Interaction between the Cerebral Hemispheres, in S. Kornblum, ed., *Attention and Performance IV* (New York: Academic Press, 1973).
- Kinsbourne, M. The Mechanism of Hemispheric Control of the Lateral Gradient of Attention, in P. M. A. Rabbitt & S. Dornic, eds., *Attention and Performance V* (London: Academic Press, 1975).
- Kinsbourne, M., & Hicks, R. E. Functional Cerebral Space: A Model for Overflow, Transfer and Interference Effects in Human Performance: A Tutorial Review, in J. Requin, ed., *Attention and Performance VII* (Hillsdale, NJ: Erlbaum, 1978).
- Klatzky, R. L., & Atkinson, R. C. Specialization of the Cerebral Hemispheres in Scanning for Information in Short-Term Memory, *Perception and Psychophysics*, 10 (1971), 335–338.
- Ledlow, A., Swanson, J. M., & Kinsbourne, M. Reaction Times and Evoked Potentials as Indicators of Hemispheric Differences for Laterally Presented Name and Physical Matches, *Journal of Experimental Psychology: Human Perception and Performance*, 4 (1978), 440–454.
- Leehey, S. C., & Cahn, A. Lateral Asymmetries in the Recognition of Words, Familiar Faces and Unfamiliar Faces, *Neuropsychologia*, 17 (1979), 619–635.
- Leiber, L. Lexical Decisions in the Right and Left Cerebral Hemispheres, *Brain and Language*, 3 (1976), 443–450.
- Lindell, A. K., & Nicholls, M. E. Attentional Deployment in Visual Half-Field Tasks: The Effect of Cue Position on Word Naming Latency, *Brain and Cognition*, 53 (2003), 272–277.
- McCarthy, R. A. Visual Field Differences in Sequential Letter Classification, unpublished PhD dissertation, University of Leicester (1980).
- Metzger, R. L., & Antes, J. R. Sex and Coding Strategy Effects on Reaction Time to Hemisphere Probes, *Memory and Cognition*, 4 (1976), 157–171.
- Moscovitch, M. Information Processing and the Cerebral Hemispheres, in M. S. Gazzaniga, ed., *Handbook of Behavioral Neurobiology*, Vol. 2: *Neuropsychology* (New York: Plenum Press, 1979).
- Nicholls, M. E. The Non-Contribution of Attentional Biases to Visual Field Asymmetries for Temporal Discrimination, *Neuropsychologia*, 32 (1994), 209–220.
- Parrot, M., Doyon, B., Démonet, J. F., & Cardebat, D. Hemispheric Preponderance in Categorical and Coordinate Visual Processes, *Neuropsychologia*, 37 (1999), 1215–1225.

- Patterson, K. E., & Bradshaw, J. L. Differential Hemispheric Mediation of Non-verbal Stimuli, *Journal of Experimental Psychology: Human Perception and Performance*, 1 (1975), 246–252.
- Querné, L., Eustache, F., & Faure, S. Interhemispheric Inhibition, Intrahemispheric Activation, and Lexical Capacities of the Right Hemisphere: A Tachistoscopic, Divided Visual-Field Study in Normal Subjects, *Brain and Language*, 74 (2000), 171–190.
- Segalowitz, S. J., Bebout, L. J., & Lederman, S. J. Lateralization for Reading Musical Chords: Disentangling Symbolic, Analytic and Phonological Aspects of Reading, *Brain and Language*, 8 (1979), 315–323.
- Ward, T. B., & Ross, L. E. Laterality Differences and Practice Effects under Central Backward Masking Conditions, *Memory and Cognition*, 5 (1977), 221–226.
- White, M. J. Hemispheric Asymmetries in Tachistoscopic Information-processing, *British Journal of Psychology*, 63 (1972), 497–508.
- Young, A. W. Methodological and Theoretical Bases of Visual Hemifield Studies, in J. G. Beaumont, ed., *Divided Visual Field Studies of Cerebral Organisation* (London: Academic Press, 1982).
- Young, A. W., & Bion, P. J. Accuracy of Naming Laterally Presented Known Faces by Children and Adults, *Cortex*, 17 (1981), 97–106.
- Young, A. W., Bion, P. J., & Ellis, A. W. Studies toward a Model of Laterality Effects for Picture and Word Naming, *Brain and Language*, 11 (1980), 54–65.
- Zaidel, E. A. Technique for Presenting Lateralized Input with Prolonged Exposure, *Vision Research*, 15 (1975), 283–289.

Dichotic Listening

The technique of dichotic listening is the auditory parallel to divided visual field presentation. Like the latter, it was first developed within experimental psychology, and its relevance to neuropsychology was recognized only later. It was Kimura (1961) who first pointed out that the asymmetry that had already been observed could be attributed to cerebral lateralization, thus introducing the technique into human experimental neuropsychology at about the same time as the divided visual field technique was being explored with split brain patients.

Although dichotic listening historically has not attracted as much attention as divided visual field presentation, perhaps because of the added technical difficulty of creating the stimuli, the ability to more easily manipulate stimuli using laboratory computers has resulted in increasing use of the technique in recent years.

THE TECHNIQUE

The logic that underlies dichotic listening is exactly the same as that of the visual technique. Stimuli presented to the right ear are considered to be directed to the left hemisphere, and via the left ear to the right hemisphere. Subsequent performance measured by accuracy or response latency can be taken to reflect the operation of systems lateralized to the two hemispheres. The only significant difference is that in dichotic listening stimuli are presented to the two ears *simultaneously*, although associated procedures of lateralized auditory presentation may be also

used, in which stimuli are not bilateral and simultaneous. These are not, however, properly termed "dichotic."

As has already been pointed out in Chapter 4, although the primary projection of the visual system is completely lateralized, that of the auditory system is not (see Figure 4.2). For this reason, the technique is not quite as simple as divided visual field presentation. Dichotic listening relies on the dominance of the crossed contralateral pathways over the ipsilateral uncrossed pathways. The clinical evidence supports this contralateral dominance, at least under conditions of bilateral competition, as does the evidence from split-brain patients (discussed in Chapter 10). There are also physiological differences in the size of the auditory pathways, the crossed tracts being larger.

The extent of the crossed dominance in audition has, however, been questioned. Some workers have shown that the suppression effect seen in split-brain patients (see p. 206) operates only for dichotic speech stimuli and not for pure tones (Efron, Bogen, & Yund, 1977). It has also been suggested that the apparent suppression of left ear material may result from spectral-temporal overlap between stimuli, and so may be a more peripheral perceptual phenomenon than has generally been thought (Springer, Sidtis, Wilson, & Gazzaniga, 1978). These studies indicate that the dominance of the crossed pathway is not so clearly established as was once thought, but for the present we will accept that crossed dominance applies because it enables us to make sense of the experimental findings. Nevertheless, we should remember that if crossed dominance did not apply to a certain auditory stimulus, the logic of the technique used with normal human subjects would be completely undermined.

Careful attention must be paid to the construction of stimulus tapes or computer-generated stimuli for dichotic listening. The characteristics of the stimuli that arrive at the two ears must be carefully balanced, and the onset of the two stimuli carefully aligned. It has become increasingly common to use computers to generate and control the presentation of dichotic stimuli, and this allows very precise control of not only the onset but also the duration of the stimuli.

The way in which subjects are asked to respond is also very important, although it has been little studied. Given that the subject will hear pairs of simultaneous stimuli, whether he or she is asked to report one or both the items, and which is to be given first if both are to be reported, will greatly affect the results. Many workers have simply allowed free report in which subjects must report as many items as they can. Others have directed attention to one or the other ear, or controlled the order of reporting from the two ears. The findings from what little study there has been of the effect of response mode are equivocal. However, while

certain forms of response are more appropriate to specific experimental designs, controlled report must in general be superior to free report.

Much study has been made of how to score the results, which is a much less simple problem than it might appear. The asymmetry observed is usually described in terms of a left or right *ear advantage*, and this description must account for the effects of overall accuracy and the effects of guessing in forced choice response modes. The simplest solution of taking the difference between the scores at the two ears and dividing by the total score ($[\text{Right} - \text{Left}] \div [\text{Right} + \text{Left}]$) is not really satisfactory. The difficulty partly hinges on whether we consider that a performance of 20% in the right ear and 10% in the left ear represents the same asymmetry as 80% in the right and 70% in the left; and what if the scores are 100% in the right and 95% in the left: Is that the effect of a “ceiling” on performance? Decisions about how to treat such scores will have a fundamental effect on the interpretation of the results of the experiment (see Berlin & Cullen, 1977). Various solutions have been proposed and the most generally accepted is a slightly complicated index (the e and e_g coefficients) developed by Repp (1977). Few studies have, however, employed the more sophisticated scoring methods.

A more detailed discussion of the problems in the dichotic listening technique will be found in Beaumont (1982). For the present, we must recognize that there are some methodological difficulties inherent in it. The production of stimuli may be critical, as may the method of scoring, and there is some niggling uncertainty about the physiological substrate of the technique. Nevertheless, as we shall see, findings using dichotic listening have been remarkably consonant with those from other neuropsychological methods, and this must increase our confidence in its use.

LATERAL EAR ASYMMETRIES

In the classic dichotic listening experiment, three or four pairs of stimuli, one of each pair to each ear, are presented at a rate of one pair every one or two seconds. At the end of the series the subject must report the stimuli. The ear associated with more correct responses (or occasionally the earlier responses) is described as showing an ear advantage. Kimura's 1961 study had used digits as stimuli and found a right ear advantage. Such stimuli have been the most consistent in producing a clear right-side advantage, but the effect is not limited to numbers. A right ear advantage is also generally observed for words, and not only for meaningful but also nonsense words. Even artificially generated consonant-vowel (CV) syllables yield a stable right ear advantage, indicating that left hemisphere processes are not associated only with linguistically

meaningful material. Studies have proceeded to investigate most linguistic aspects of verbal stimuli. These studies are important in revealing, by the degree of lateral asymmetry that may be observed, the processes involved in producing the lateral advantage. They also provide important information for those investigating speech perception and language comprehension. The common factor, however, is that most speechlike and language-related stimuli are reported more accurately from the right ear if presented in dichotic pairs.

By contrast, certain stimuli are associated with a left ear advantage. These include melodies, sonar signals, environmental sounds, and “non-verbal vocal tract sounds.” Environmental sounds might be a running tap, traffic, or teeth being brushed, and vocal tract sounds might be coughs, hums, or grunts. These stimuli seem to have in common that they are patterned and nonverbal. In particular they are not at all speechlike, and this may be an important feature in determining the involvement of the right hemisphere. More recently, both emotional tone (Sim & Martinez, 2005), and the detection of deception (Malcolm & Paul, 2005), have been shown to moderate ear asymmetries and have some association with the right hemisphere.

The simplest model that can be set up for dichotic listening performance looks very much like that for divided visual field performance. Speech and language stimuli are associated with a left hemisphere advantage, while nonverbal stimuli are associated with a right hemisphere advantage.

Before looking at how this model has been developed, it is worth asking whether there is an asymmetry in response to very simple stimuli, reflecting findings in the visual modality. Obviously we cannot perform a parallel dichotic experiment, but we can test the reaction time, shown by the hands, to simple auditory tones presented to the right and left ears. From this test it seems there is some evidence that faster reaction times are associated with the “direct” route from ear to ipsilateral hand, in contrast with the crossed indirect pathway from ear to contralateral hand (Provins & Jeeves, 1975). A study by Elias, Bulman-Fleming, and McManus (2000) has also shown that the Poffenberger effect, illustrating interhemispheric transfer (see p. 224), can be demonstrated in the auditory modality. However, asking subjects to attend only to one ear modifies this effect: if subjects attend to the right ear, the difference in reaction times between hands disappears, if to the left ear, the left hemisphere advantage is retained and there is a faster reaction shown by the left hand (Spellacy & Wilson, 1978). This emphasizes the importance of central, cortically mediated mechanisms in modifying performance through selective attention.

Left Hemisphere Processes

What precisely underlies the left hemisphere advantage? An early idea was that the left hemisphere might be particularly equipped to deal with speech perception. Speech perception is more difficult to study than might at first appear, as is reflected in the slow progress being made with automatic systems for speech recognition. The difficulty is that the sounds which go to make up a word are not strung together simply. Many of the component sounds arrive in parallel, so that the difficulty in speech perception is to unravel parallel components, a process referred to as *drastic restructuring* (see Liberman, 1974; Springer, 1979). This difficulty is increased because a perceived phonetic segment may result from different acoustic cues in different contexts, that is, the same (perceived) sound may come from differing physical stimuli.

Drastic restructuring has been linked with right ear advantage when hard-to-decode stimuli have been contrasted with easy-to-decode stimuli. Stop consonants (/b/,/d/,/g/,/p/,/t/,/k/) need more restructuring than isolated vowels, and stop consonants with an added /a/ vowel produce a right ear advantage, while synthetic vowels do not. In nonsense words, if the consonants are contrasted (“bip” versus “gif”) then a larger right ear superiority will result than if the vowels are contrasted (“bip” versus “bap”). Sounds that require intermediate levels of restructuring, such as liquid consonants (/r/,/l/), semivowels (/y/,/w/), and fricatives (/s/,/v/,/f/,/z/), have been reported to produce an intermediate right ear advantage.

Darwin (1971) showed the importance of the presence of formant transitions (certain features of speech stimuli) for speech perception, thus supporting the restructuring hypothesis. Other linguistic factors have also been shown to be related to right ear superiority: if grammatical structure is present, even in a sentence of nonsense words (“The wak jud shendily” has grammatical structure, “Bul hudky gu nee” does not; Zurif & Sait, 1970), then it may produce the right ear superiority. Linguistic tone (intonation) may also be involved; if a language like Thai is used (with Thai speakers as subjects) in which the same sound with different intonation can have quite disparate meanings (*naa* can mean “aunt” or “field”), then a right ear advantage can be seen for stimuli that differ only in tone.

However, that the right ear advantage reflects the involvement of speech perception can only be a partial explanation. For one thing, this advantage may be seen for language stimuli that do not involve the effect of formant transitions. Some very elementary aspects of stimuli, such as intensity, time, and frequency, may also moderate the right ear advantage (Berlin, 1977; Brancucci, Babiloni, Rosini, & Romani, 2005); and

using vowels, but masking them by adding white noise, has also been shown to produce a right ear superiority (Weiss & House, 1973). Both these results indicate that purely acoustic factors may also be linked to left hemisphere processes. Some of these processes may still be linked to language perception, but it cannot be language perception alone that is lateralized to the left hemisphere.

Cutting (1974) compared performance using CV stimuli with that using appropriate or inappropriate formant transitions. Both types of stimuli yielded right ear advantages. However, only the stimuli with appropriate formants produced a discrimination curve that would be expected for language-related perception. This seems to point to a purely auditory component in the processing of CV syllables with inappropriate formants, yet they produced a right-sided advantage. Godfrey (1974) has systematically manipulated such features as the signal-to-noise ratio, acoustic-phonetic distinctness, and vowel duration, and his results also suggest that the effects are being created at the auditory rather than the phonetic level of processing.

This literature can be rather technical for those not expert in linguistics and speech processing. However, most workers now accept that there may be two types of processor associated with left hemisphere specialization (Bub & Whitaker, 1980; Springer, 1979). One of these is related to the restructuring and encoding of speech stimuli, and operates at a phonetic level. The other operates at a purely acoustic level, and will function in any difficult acoustic discrimination, even if nonlinguistic in character. It has been suggested that this second processor may be set to detect rapidly changing frequency information, or that it performs temporal order judgments. None of these hypotheses can be regarded as a very satisfactory explanation of the data, and it may be that some better model can yet be found. Nevertheless, it is clear that, while the left hemisphere is implicated in speech and language processing, this is not a sufficient explanation of many of the right ear advantages that have been reported.

Right Hemisphere Processes

That certain non-speechlike sounds can produce a left ear advantage is undisputed; but what processes actually determine right hemisphere lateralization? From what has been said of the left hemisphere, it seems unlikely to be a matter of nonverbal auditory discrimination.

This question has been most commonly tackled through the study of musical stimuli. Certain kinds of musical stimuli produce a clear left ear advantage, and it is possible to manipulate different aspects of such stimuli to discover just what determines the advantage. At least, that has

been the strategy behind research in this area. Unfortunately, the results have not yet enabled any clear conclusions to be drawn about the processors resident in the right hemisphere (for reviews, see Craig, 1979; Damasio & Damasio, 1977; Gates & Bradshaw, 1977a; Mitchell & Crow, 2005; Wyke, 1977).

Kimura, in 1964, described a left ear advantage for dichotic melodies, but this result has not always been supported by later similar experiments. For instance, Gordon (1970) found no lateral asymmetry for melodies, although he did find an effect for dichotic chords. Taking even simpler stimuli, the ear advantage for pitch (or frequency) has not been clearly established. There is some suspicion that this may be because pitch is also critically involved in speech perception, and the context of the stimuli may therefore be important in determining whether they are treated as being more or less speechlike. Other factors that have been shown to be relevant are the delay between the stimulus and a subsequent comparison stimulus; the complexity of the stimulus; and the subject's expectation as to which ear will receive the stimulus. The results with timbre are equally inconsistent, and time and rhythm have received little attention.

It is possible to make some sense of the data, however, by considering just how subjects are likely to treat the stimuli. One difficulty of decomposing complex stimuli into their components is that subjects may not process them in the same manner. As "musical" stimuli become more and more simplified, they are less and less distinct from simplified speech sounds, and it becomes more likely that subjects will process them as if they were speech sounds. This is supported by two pieces of evidence: that, by and large, the right hemisphere advantage is found more readily with structured musical passages, and that characteristics of the subjects themselves can have a profound effect on the lateral advantage observed.

Some of the conflict, between the findings of Kimura and Gordon with respect to melodies, for example, can be resolved by reference to the subjects employed. A number of studies have specifically examined the effects of the musical training and experience of the subjects. For example, in a study by Johnson and colleagues (1977) musicians were asked to recognize conventional melodies or random note sequences. All subjects showed a left ear advantage for the random note sequences. With the melodies, however, the results were especially interesting, for trained musicians who could transcribe music showed a right ear advantage, but the trained musicians who could not transcribe music showed a left ear advantage. The interpretation of these findings is that complex musical stimuli are processed by right hemisphere mechanisms unless the stimuli are both meaningful and can be encoded in a formal symbolic

way. If they can be so encoded, as they may be by those who can transcribe music, then left hemisphere mechanisms may be called into play.

This finding has an interesting parallel in an experiment with morse code (Papçun, Krashen, Terbeck, Remington, & Harshman, 1974). Morse code was presented dichotically to morse code operators and to these unfamiliar with morse code. The operators showed a consistent right ear advantage, but subjects unfamiliar with morse code only showed a right ear advantage if there were seven or fewer pairs of elements. With longer lists, which they were presumably unable to process semantically, or which could not be handled by the sequential analysis of components of the stimulus train, there was a left ear advantage, reflecting a switch to right-hemisphere-based functions.

However, not all studies with trained musicians have found a right ear advantage for complex stimuli: both Gordon (1980) and Zatorre (1979) found a left ear advantage and no effect of the degree of musical training. Also, in an elegant study of the parallel effects of verbal and musical components of the stimulus, which used either numbers superimposed upon piano notes or digits sung in a tonal pattern, a separate right ear superiority for the verbal elements and left ear superiority for the musical elements was maintained. Certain paradoxical effects have also been reported when comparing dichotic tasks with dual task interference studies employing musical stimuli (Lim, Lambert, & Hamm, 2001).

No doubt the solution to this conflicting evidence lies in the precise nature of the experimental task and the expectations of the subjects. Gates and Bradshaw (1977b), following a complex and thorough series of experiments on the detection of pitch, rhythm, and harmony changes, emphasized the importance of the different strategies adopted by subjects, as well as the familiarity of the type of material employed. Musical perception may well rely on contributions by both the cerebral hemispheres, in different forms in different subjects, and this makes it difficult to determine just what is the unique contribution of the right hemisphere.

ATTENTION

Both the structural and attentional models can be applied to dichotic listening data, in the same way that they were applied to divided visual field research (see p. 234). We have already seen how the structural model can be used to interpret a lateral ear advantage, but is it sufficient to explain all the results? Various forms of evidence are relevant in answering this question.

The first is whether competition is necessary to establish a lateral asymmetry; that is, can an ear difference be established using monaural instead of dichotic presentation? While the asymmetry is smaller and less stable with monaural stimuli, it seems that it can be observed (Henry, 1979). The study by Kallman (1977) serves as a good example of an experiment that was parallel to the usual dichotic procedure, and yet used monaural presentation. Both speech and nonverbal stimuli of the form used in classic dichotic experiments were used and the procedure was identical with the exception that the stimuli were presented to a single ear. The interactions expected in a dichotic paradigm, between the type of stimulus and performance at the two ears, were found. Belmore (1980) even managed to demonstrate an asymmetry using monaurally presented sentences, although only when the task demanded attention to the meaning of the stimulus material. Results of this kind support the importance of a structural explanation.

There are, however, findings that do not fit so neatly into a structural model. One is that monaural competing stimuli presented to the same ear can yield a right ear advantage (Bradshaw, Farrelly, & Taylor, 1981). This could be interpreted as a simple lateral advantage, but does suggest that the effects of competition do not necessarily arise from interference between different lateral input channels.

That the laterality effect does not depend on physical separation of the stimuli is demonstrated most clearly by the fact that a right ear advantage can still be found even when the stimuli are not presented through headphones but through loudspeakers at the left and right of the subject (Morais & Bertelson, 1973). The effect must in this case be due to the physical location of the stimuli in space, to the left or right, and not to the arrival of the stimuli by different anatomical channels, because the stimuli at each side enter by both ears. Even more remarkable is that the laterality effect can be produced by *apparent* rather than real physical location to one side of space. We determine the location of sounds by various cues, the most important of which are intensity and time differences. A stimulus to our left, for example, will be louder and will arrive earlier at the left than at the right ear, and this difference in intensity and time of arrival tells us the location of the stimulus. It is therefore possible, by simply manipulating the time and intensity differences between two stimuli, to produce an apparent origin for the sounds that they do not have in reality. That a sound is apparently located to the right side of the body is sufficient for it to be associated with superior performance (Morais & Bertelson, 1975).

Findings of this kind are difficult to explain by a structural model, and support the importance of attentional factors. The contribution of such factors has been assessed in a related phenomenon, the *ventrilo-*

quism effect (Morais, 1975). Subjects were seated with four loudspeakers visible to them, at 45 degrees and 90 degrees to the left and right of the direction in which they were facing. Behind each of these visible speakers, which were dummies, was a curtain and beyond that an active speaker. The subjects were told to which speakers they should attend for subsequent recall of the stimuli. Simultaneous messages, that might or might not come from the locations expected by the subject, were then presented. Among some rather complex results, the right side advantage was only found when subjects expected the message to be 90 degrees to the right and it was actually located there. This finding clearly shows that neither the structural nor the attentional model is sufficient in itself, both must be involved. If the structural model is a sufficient explanation, the subject's attention would be irrelevant; there would be a right side advantage whenever the stimulus was at that side. Similarly, if the attentional model were a sufficient explanation, the actual location would be unimportant, and the subject's expectation would alone determine the advantage.

A similar experiment, but actually using dichotic presentation, examined the effects of ear of entry and subjective location by directing different formants of the stimulus to different ears (Darwin, Howell, & Brady, 1978). Again, the ear of entry was in itself a significant factor, but an additional right ear advantage was attributed to the effects of subjective location (see also Hiscock, Inch, & Ewing, 2005).

A useful discussion of the competing claims of the structural and attentional models was presented by Studdert-Kennedy in introducing a special issue of *Brain and Language* in 1975 devoted to dichotic listening. He sensibly suggested that an experiment with mixed materials in a random sequence might help to resolve the issue. A structural model would predict that the usual asymmetries for each type of material would be preserved, while an attentional model would predict reduced lateral advantages. An experiment of this type has been reported by Kallman (1978). The speech stimuli produced the expected right ear advantage, while the musical stimuli that were mixed with them tended to give a left ear advantage. The results again indicate that attentional factors, if involved, are not themselves sufficient to explain the lateral asymmetries.

Finally, the fact that the subject's *response strategy* can affect the results is pertinent to any model of the processes involved. Both Bryden (1978) and Freides (1977) have shown this to be the case, and the conclusion is supported by Hiscock, Inch, and Kinsbourne (1999). Both of the earlier researchers contrasted a free recall condition with conditions in which attention and report order were more strictly controlled. Both found that the report instructions influenced which ear advantage was

found. However, they differed in the significance that they placed upon the result. Freides strongly made the point that if response strategy is such an important determinant of the lateral advantage, then competitive methods such as dichotic listening might be merely measuring the subject's response strategy rather than something more fundamental about brain organization. Bryden, on the other hand, emphasized how the subject's strategy reflected the operation of attentional and cognitive factors within the processing system. Whichever view is taken, the effects of cognitive strategy, in the context of our general models of cerebral lateralization, illustrate the contribution of variables other than those related directly to structural mechanisms (Voyer & Flight, 2001). Just as with the visual studies, the evidence clearly points to a dynamic-structural model.

AN INDEX OF LATERALIZATION

Many writers have been keen to point out that the right ear advantage for digits, while associated with cerebral laterality, cannot be used as an index of lateralization (Berlin, 1977; Colbourn, 1978; Teng, 1981). The problem arises from the variability both of different subjects and of any one subject over time. It is easy to be misled by the clear average ear advantage shown by group data, and assume that dichotic performance could be an index of speech lateralization in individual subjects. Studies continue to appear in which dichotic listening data are used in this way.

The evidence is clearly against the use of such an index, with one notable exception: the Dichotic Monitoring Test of Geffen, Traub and Stierman (1978). This test involves the dichotic presentation of monosyllabic word pairs, that include the target word *dog* among eight "noise words" in each channel that share two phonemes in common with the target (e.g., *dig* or *log*), as well as 52 dissimilar words. The subject must detect the target word, and both reaction time and accuracy are recorded in response to occurrences of the target word. The test was given to 4 patients who had undergone the Wada test to establish speech lateralization and to 31 patients whose speech laterality had been assessed by the presence of (temporary) dysphasia following the unilateral administration of electroconvulsive therapy (ECT), and extremely good agreement about language lateralization was shown between the physical tests and the dichotic procedure. An extension of this study by Geffen and Caudrey (1981) reported an agreement of 95%, and a test-retest study for reliability found a shift in assessed laterality in only 3 of 86 subjects. This procedure appears more reliable and more valid than previous attempts to develop an index of speech lateralization that could

be used in normal subjects but it has not been widely adopted in a clinical context.

If either dichotic listening or divided visual field methods could yield an index of cerebral lateralization, we should expect a correlation between the asymmetries found using the two techniques in any individual subject. Fennell, Bowers, and Satz (1977) examined just this issue, using a sequence of four tests of dichotic listening to concrete words and divided visual field presentation of letters. They found both the expected right visual field and right ear advantages. The dichotic asymmetry was relatively stable across the four occasions of testing, although the visual asymmetry was significant only after the first occasion. The correlation between the two modalities was also found to be reliable and stable, particularly on the final two occasions of testing. Clinical applications have been discussed by Voyer and Rodgers (2002) and Voyer, Russell, and McKenna (2002).

This study has now been subjected to extremely searching criticism in terms of the general methodological problems inherent in the measures, the composition of the samples, and the statistical treatments used (Berenbaum & Harshman, 1980). The authors of this criticism urge skepticism about the findings of the study by Fennell et al. (1977), and it seems at least prudent to await further investigation of the issue. Other data, collected with a primary interest in the effects of the subject variables of sex and handedness, also suggest that there can be a significant dissociation between the asymmetries observed in different modalities (Searleman, 1980).

CONCLUSION

While dichotic listening studies have made a major contribution to experimental neuropsychology, they have been less numerous than studies in the visual modality. This is due both to the technical demands of the technique and to the added problems that the bilateral nature of auditory projection brings to interpretation of the results. In consequence, studies in recent years have concentrated more on linguistic variables than on the general principles underlying cerebral lateralization.

The studies have nevertheless provided evidence of lateral specialization in the brain. The left hemisphere seems to be associated with two kinds of processing: one phonetic, the other acoustic. The phonetic processor deals with speech and speechlike stimuli in terms of their linguistic composition. The acoustic processor deals more generally with complex auditory stimuli, and may be involved in temporal order perception. It does not deal merely with speech stimuli.

The right hemisphere has been associated with “nonverbal” sounds and with complex musical stimuli. Not all aspects of music are processed preferentially in the right hemisphere, and music perception (like most real-life tasks) involves the contribution of both the hemispheres.

It will by now be apparent that the research undertaken in a purely experimental context is generally of a much higher standard than that performed with clinical patients (discussed in Part II). This mainly reflects the difficulty of doing scientifically sound research in a clinical context, but the general level of both methodological sophistication and theoretical discussion has been superior in experimental neuropsychology.

Direct comparison between experimental and clinical findings is difficult, partly because clinical work has concentrated on localization, which has been ignored in experimental research in favor of lateralization. Also, many of the tests and tasks employed in clinical research cannot be presented, for procedural reasons, in experimental paradigms. Integration of the two approaches has not been attempted in any systematic fashion, although there seem to be few fundamental disagreements between the two areas of research. The evidence from dichotic listening agrees remarkably well with both clinical findings and data from divided visual field studies.

The only model that at present accounts well for the whole body of dichotic research is the dynamic–structural model, which is also the model supported by visual studies. Attentional and cognitive factors play a significant role in auditory as in visual lateralization.

Dichotic listening has proved neither sufficiently reliable nor sufficiently stable to allow the determination of speech lateralization in individual subjects. Recent research may, however, show that an index of this kind can be developed.

OTHER METHODS IN EXPERIMENTAL NEUROPSYCHOLOGY

While divided visual field and dichotic listening techniques far outweigh all other methods in importance, some of the alternative methods should be briefly mentioned. A review of all these methods is to be found in Beaumont (1982).

Lateralized stimulus presentation has also been used in *tactile perception*. Stimuli can be presented to the right and left hands and subsequent performance recorded. It has been possible to demonstrate lateral asymmetries in this way, but only when fairly fine manipulation or tactile exploration is required. The projection of tactile information to the

cortex (see p. 96) involves both ipsilateral and contralateral pathways, and this means that it is difficult to be sure that stimulus presentation has been appropriately lateralized. However, a “dichhaptic” technique originally developed by Witelson (1974) and involving bilateral simultaneous stimulation has been used in a number of studies and produced interesting results.

Lateral eye movements have also attracted some interest. Kinsbourne (1972) sparked off this work by the observation that solving “verbal” problems was often accompanied by conjugate gaze deviation to the right, and solving “spatial” problems by gaze deviation to the left. He explained this phenomenon in terms of his attentional model, hemisphere activation being considered to produce eye movement toward the opposite side of space. Following much debate about the reliability and validity of such observations, recent reviews have suggested that lateral eye movements can be reliably elicited and that they do reflect hemisphere lateralization (Ehrlichman & Weinberger, 1978). Whether the evidence justifies their use as an index of an individual’s cerebral organization, as is sometimes done, seems doubtful.

Some lateral asymmetries are also to be observed in *free vision*; for example, there are lateral asymmetries in aesthetic composition that seem to relate to hemisphere specialization, although the mechanism behind such asymmetries is under dispute. It has also been reported that the left side of the face has been more commonly presented in formal portraiture, and this side of the face is also judged as more emotionally intense. Asymmetries in spatial orientation and judgment, as seen in map reading and direction finding, have also been demonstrated.

Finally, there are the *lateral performance asymmetries*, of which the most important is handedness. Differences between left and right handers are discussed at length in Chapter 14, but even in right handers the difference between the preferred and nonpreferred hands can be studied as one aspect of cerebral organization. Various aspects of manual skill have been measured, and there has been a renewal of interest in tapping performance. As with tactile presentation, inferences about cerebral lateralization are complicated by the bilateral nature of certain aspects of neural organization. The Torque Test, which measures the direction of drawing tendencies as clockwise or anticlockwise, has been much discussed but has also been subjected to criticism.

All of these methods can contribute to our overall model of neuropsychological organization but because of methodological or theoretical complications none is as satisfactory or important as divided visual field and dichotic listening research. All experimental data have nevertheless some part to play, and the development of techniques in neuropsychology may well spring from refinements of these alternative methods.

FURTHER READING

There is an unfortunate dearth of good general accounts of dichotic listening research. Reviews of relatively specific aspects have been mentioned in the text, but study might best proceed with a look at the following:

Hugdahl, K. (Ed.). *Handbook of Dichotic Listening: Theory, Method and Research* (Chichester, UK: Wiley, 1988).

REFERENCES

- Beaumont, J. G. Methods for Studying Cerebral Hemispheric Function, in A. W. Young, ed., *Functions of the Right Cerebral Hemisphere* (London: Academic Press, 1982).
- Belmore, S. M. Depth of Processing and Ear Differences in Memory for Sentences, *Neuropsychologia*, 18 (1980), 657–663.
- Berenbaum, S. A., & Harshman, R. A. On Testing Group Differences in Cognition Resulting from Differences in Lateral Specialisation: Reply to Fennell et al., *Brain and Language*, 11 (1980), 209–220.
- Berlin, C. I. Hemispheric Asymmetry in Auditory Tasks, in S. Harnad, R. W. Doty, L. Goldstein, J. Jaynes, & G. Krauthamer, eds., *Lateralization in the Nervous System* (New York: Academic Press, 1977).
- Berlin, C. I., & Cullen, J. K., Jr. Acoustic Problems in Dichotic Listening Tasks, in S. J. Segalowitz & F. A. Gruber, eds., *Language Development and Neurological Theory* (New York: Academic Press, 1977).
- Bradshaw, J. L., Farrelly, J., & Taylor, M. J. Synonym and Antonym Pairs in the Detection of Dichotically and Monaurally Presented Targets: Competing Monaural Stimulation Can Generate a Substantial Right Ear Advantage, *Acta Psychologica*, 47 (1981), 189–205.
- Brancucci, A., Babiloni, C., Rosini, P. M., & Romani, G. L. Right Hemisphere Specialization for Intensity Discrimination of Musical and Speech Sounds, *Neuropsychologia*, 43 (2005), 1916–1923.
- Bryden, M. P. Strategy Effects in the Assessment of Hemispheric Asymmetry, in G. Underwood, ed., *Strategies of Information Processing* (London: Academic Press, 1978).
- Bub, D., & Whitaker, H. A. Language and Verbal Processes, in M. C. Wittrock, ed., *The Brain and Psychology* (New York: Academic Press, 1980).
- Colbourn, C. J. Can Laterality be Measured?, *Neuropsychologia*, 16 (1978), 283–289.
- Craig, J. D. Asymmetries in Processing Auditory Nonverbal Stimuli?, *Psychological Bulletin*, 86 (1979), 1339–1349.
- Cutting, J. E. Different Speech-Processing Mechanisms Can Be Reflected in the Results of Discrimination and Dichotic Listening Tasks, *Brain and Language*, 1 (1974), 363–375.
- Damasio, A. R., & Damasio, H. Musical Faculty and Cerebral Dominance, in

- M. Critchley & R. A. Henson, eds., *Music and the Brain* (London: Heineman Medical Books, 1977).
- Darwin, C. J. Ear Differences in the Recall of Fricatives and Vowels, *Quarterly Journal of Experimental Psychology*, 23 (1971), 46–62.
- Darwin, C. J., Howell, P., & Brady, S. A. Laterality and Localisation: A “Right Ear Advantage” for Speech Heard on the Left, in J. Requin, ed., *Attention and Performance VII* (Hillsdale, NJ: Erlbaum, 1978).
- Efron, R., Bogen, J. E., & Yund, E. W., Perception of Dichotic Chords by Normal and Commissurotomed Human Subjects, *Cortex*, 13 (1977), 137–149.
- Ehrlichman, H., & Weinberger, A. Lateral Eye Movements and Hemispheric Asymmetry: A Critical Review, *Psychological Bulletin*, 85 (1978), 1080–1101.
- Elias, L. J., Bulman-Fleming, M. B., & McManus, I. C. Linguistic Lateralization and Asymmetries in Interhemispheric Transmission-Time, *Brain and Cognition*, 43 (2000), 181–185.
- Fennell, E. B., Bowers, D., & Satz, P. Within-modal and Cross-modal Reliabilities of Two Laterality Tests, *Brain and Language*, 4 (1977), 63–69.
- Freides, D. Do Dichotic Listening Procedures Measure Lateralization of Information Processing or Retrieval Strategy?, *Perception and Psychophysics*, 21 (1977), 259–263.
- Gates, A., & Bradshaw, J. L. The Role of the Cerebral Hemispheres in Music, *Brain and Language*, 4 (1977a), 403–431.
- Gates, A., & Bradshaw, J. L. Music Perception and Cerebral Asymmetries, *Cortex*, 13 (1977b), 390–401.
- Geffen, G., & Caudrey, D. Reliability and Validity of the Dichotic Monitoring Test for Language Laterality, *Neuropsychologia*, 19 (1981), 413–423.
- Geffen, G., Traub, E., & Stierman, I. Language Laterality Assessed by Unilateral ECT and Dichotic Monitoring, *Journal of Neurology, Neurosurgery and Psychiatry*, 41 (1978), 354–360.
- Godfrey, J. J. Perceptual Difficulty and the Right Ear Advantage for Vowels, *Brain and Language*, 1 (1974), 323–337.
- Gordon, H. W. Hemispheric Asymmetries in the Perception of Musical Chords, *Cortex*, 6 (1970), 387–398.
- Gordon, H. W. Degree of Ear Asymmetries for Perception of Dichotic Chords and for Illusory Chord Localisation in Musicians of Different Degrees of Competence, *Journal of Experimental Psychology: Human Perception and Performance*, 6 (1980), 516–527.
- Henry, R. G. Monaural Studies Eliciting an Hemispheric Asymmetry: A Bibliography, *Perceptual and Motor Skills*, 48 (1979), 335–338.
- Hiscock, M., Inch, R., & Ewing, C. T. Constant and Variable Aspects of the Dichotic Listening Right-Ear Advantage: A Comparison of Standard and Signal Detection Tasks, *Laterality*, 10 (2005), 517–534.
- Hiscock, M., Inch, R., & Kinsbourne, M. Allocation of Attention in Dichotic Listening: Effects on the Detection and Localization of Targets within Lists, *Journal of Clinical and Experimental Neuropsychology*, 21 (1999), 265–278.

- Johnson, R. C., Bowers, J. K., Gamble, M., Lyons, F. M., Presbrey, T. W., & Vetter, R. R. Ability to Transcribe Music and Ear Superiority for Tone Sequences, *Cortex*, 13 (1977), 295–299.
- Kallman, H. J. Ear Asymmetry and Monaurally Presented Sounds, *Neuropsychologia*, 15 (1977), 833–836.
- Kallman, H. J. Can Expectancy Explain Reaction Time Ear Asymmetries?, *Neuropsychologia*, 16 (1978), 225–228.
- Kimura, D. Cerebral Dominance and the Perception of Verbal Stimuli, *Canadian Journal of Psychology*, 15 (1961), 166–171.
- Kimura, D. Left–Right Differences in the Perception of Melodies, *Quarterly Journal of Experimental Psychology*, 16 (1964), 355–358.
- Kinsbourne, M. Eye and Head Turning Indicates Cerebral Lateralization, *Science*, 176 (1972), 539–541.
- Liberman, A. M. Ear Differences and Hemispheric Specialization, in F. O. Schmitt & F. G. Worden, eds., *The Neurosciences: Third Study Program* (Cambridge, MA: MIT Press, 1974).
- Lim, V. K., Lambert, A., & Hamm, J. P. A Paradox in the Laterality of Melody Processing, *Laterality*, 6 (2001), 369–379.
- Malcolm, S., & Paul, K. J. Hemispheric Asymmetry and Deception Detection, *Laterality*, 10 (2005), 103–110.
- Mitchell, R. L., & Crow, T. J. Right Hemisphere Language Functions and Schizophrenia: The Forgotten Hemisphere?, *Brain*, 128 (2005), 963–978.
- Morais, J. The Effects of Ventriloquism on the Right Side Advantage for Verbal Material, *Cognition*, 3 (1975), 127–139.
- Morais, J., & Bertelson, P. Laterality Effects in Dichotic Listening, *Perception*, 2(1973), 107–111.
- Morais, J., & Bertelson, P. Spatial Position Versus Ear of Entry as Determinant of the Auditory Laterality Effects: A Stereophonic Test, *Journal of Experimental Psychology: Human Perception and Performance*, 1 (1975), 253–262.
- Papçun, G., Krashen, S., Terbeck, D., Remington, R., & Harshman, R. Is the Left Hemisphere Specialized for Speech, Language and/or Something Else?, *Journal of the Acoustical Society of America*, 55 (1974), 319–327.
- Provins, K. A., & Jeeves, M. A. Hemisphere Differences in Response Time to Simple Auditory Stimuli, *Neuropsychologia*, 13 (1975), 207–212.
- Repp, B. H. Measuring Laterality Effects in Dichotic Listening, *Journal of the Acoustical Society of America*, 62 (1977), 720–737.
- Searleman, A. Subject Variables and Cerebral Organization for Language, *Cortex*, 16 (1980), 239–254.
- Sim, T. C., & Martinez, C. Emotion Words Are Remembered Better in the Left Ear, *Laterality*, 10 (2005), 149–159.
- Spellacy, F., & Wilson, A. Directed Attention and Perceptual Asymmetry to Monaurally Presented Tones, *Cortex*, 14 (1978), 71–77.
- Springer, S. P. Speech Perception and the Biology of Language, in M. S. Gazzaniga, ed., *Handbook of Behavioral Neurobiology, Vol. 2: Neuropsychology* (New York: Plenum Press, 1979).
- Springer, S. P., Sidtis, J., Wilson, D. H., & Gazzaniga, M. S. Left Ear Perfor-

- mance in Dichotic Listening Following Commissurotomy, *Neuropsychologia*, 16 (1978), 305–312.
- Studdert-Kennedy, M. Dichotic Studies II, *Brain and Language*, 2 (1975), 123–130.
- Teng, E. L. Dichotic Ear Difference Is a Poor Index for the Functional Asymmetry Between the Cerebral Hemispheres, *Neuropsychologia*, 19 (1981), 235–240.
- Voyer, D., & Flight, J. Gender Differences in Laterality on a Dichotic Task: The Influence of Report Strategies, *Cortex*, 37 (2001), 345–362.
- Voyer, D., & Rodgers, M. A. Reliability of Laterality Effects in a Dichotic Listening Task with Nonverbal Material, *Brain and Cognition*, 48 (2002), 602–606.
- Voyer, D., Russell, A., & McKenna, J. On the Reliability of Laterality Effects in a Dichotic Emotion Recognition Task, *Journal of Clinical and Experimental Neuropsychology*, 24 (2002), 605–614.
- Weiss, M. S., & House, A. S. Perception of Dichotically Presented Vowels, *Journal of the Acoustical Society of America*, 53 (1973), 51–58.
- Witelson, S. F. Hemispheric Specialization for Linguistic and Nonlinguistic Tactile Perception Using a Dichotomous Stimulation Technique, *Cortex*, 10 (1974), 3–17.
- Wyke, M. Musical Ability: A Neuropsychological Interpretation, in M. Critchley & R. A. Henson, eds., *Music and the Brain* (London: Heineman Medical Books, 1977).
- Zatorre, R. J. Recognition of Dichotic Melodies by Musicians and Non-musicians, *Neuropsychologia*, 17 (1979), 607–617.
- Zurif, E. B., & Sait, P. E. The Role of Syntax in Dichotic Listening, *Neuropsychologia*, 8 (1970), 239–244.

Electrophysiology and Imaging

ELECTROPHYSIOLOGY

At the time of the first edition of this book, the introduction of electrophysiological techniques into neuropsychology was one of the most exciting developments of recent years. For the first time it was, in principle, possible to observe in “real time”—that is, as they happen—cognitive processes and the physiological events that are believed to be associated with them. A technique was available that might make it possible to construct a bridge between mental and physiological events. If so, then there was a solution to the problem that has dogged so much of neuropsychology, that of directly investigating mind–body relationships.

Twenty-five years later, electrophysiology has failed to fulfill its promise, partly because of the methodological complexity and technical demands of the technique (discussed below). One has to feel a little sorry for electrophysiology. Its clinical importance in identifying the location of lesions was supplanted in the 1970s by the introduction of CT (computerized tomography) scans, and its anticipated role in neuropsychological research, which seemed to offer a new area of application, was never fully established, and was in turn supplanted by more advanced imaging methods (such as fMRI) of the 1990s. Nevertheless, this should not lead us to discount electrophysiology. It remains a clinically important technique, particularly in the analysis of seizure disorders, and it continues to have a place among the resources available to experimental neuropsychologists. In light of its recent history, it is difficult to predict

with confidence how it may next be employed. However, its potential in addressing mind–body relationships, and at considerably less cost than advanced imaging systems, remains.

Before being carried away by the exciting possibilities of electrophysiology, one must recognize that there are a number of difficult technical problems yet to be solved with these methods of investigation. This area of research is probably the most technically demanding in human neuropsychology, and there continue to be myriad experimental problems, as I hope to show. Nevertheless, despite the difficulties and some disappointments, we should not lose sight of what the techniques offer: the opportunity to observe subjects engaged in some intelligent task, and at the same time to observe the processes within the brain that are the physiological aspect of that mental activity. The degree to which other imaging techniques also offer this opportunity is discussed below.

Before introducing the techniques themselves, the distinction between clinical and research electroencephalography should be noted. Clinical electroencephalograms, or EEGs, are recordings of electrical activity in the brain used to investigate suspected pathological processes within the head. They are performed in a relatively standard way, using a variety of appropriate techniques, and play an important role in the physical investigation of neurological and psychiatric patients, particularly if a seizure disorder (epilepsy) is suspected. Research electroencephalograms, however, are used to investigate brain activity in normal subjects, and involve a much less well defined set of techniques that have been derived from, and are associated with, the clinical techniques.

Electroencephalographical techniques fall into two principal groups: recording “ongoing” activity, and recording evoked potentials. The procedures involved in both groups are described first, followed by some typical experiments and findings.

EEG Recording

The recording of the continuous electrical activity of the brain, or *ongoing* EEG, has been practiced since the 1930s. However, advances during the 1970s in amplifier design, and the involvement of laboratory computers in analyzing recordings, have enabled the technique to be used more widely as a research tool.

If two electrodes, traditionally small silver cups coated with silver chloride and filled with a conducting gel, are glued at two positions on the scalp, with a third electrode attached elsewhere on the head to ground them, it is possible to detect a fluctuating difference in electrical potential between the two electrodes. This constantly changing potential difference can be amplified (about 20,000 times) and written out on a

moving paper chart to form the EEG trace. It is usual to record from more than a single pair of electrodes (a single *channel*) and a typical recording might have four, eight, or many more channels simultaneously recorded (up to 24 in clinical EEGs). The chart forms a convenient way of inspecting the recording, although the data written out on the chart will be fed directly into a laboratory computer for analysis. As the computer will sample the trace for each channel at least 128 times a second, yielding 61,440 numbers for each minute of activity across eight channels, a great deal of data is generated by even a relatively short period of recording. An example of a normal human EEG is shown in Figure 13.1.

The first problems—of what exactly is being recorded and how to interpret the trace—arise when the sites for placing the electrodes are selected. Since the electrical activity of the brain is being recorded through the skull, scalp, and other tissues, the electrode is picking up activity from a relatively large area of underlying cortex, and presenting a rather distorted aggregate image of what is going on in an area of about 1 square centimeter. However, it is known from comparing scalp recordings with recordings made directly on exposed brain tissue that there is some validity in the method. Even if it is imperfect, it is all we have. Matters are sometimes further confused in that the electrode may pick up more activity from distant sites than from the directly underlying

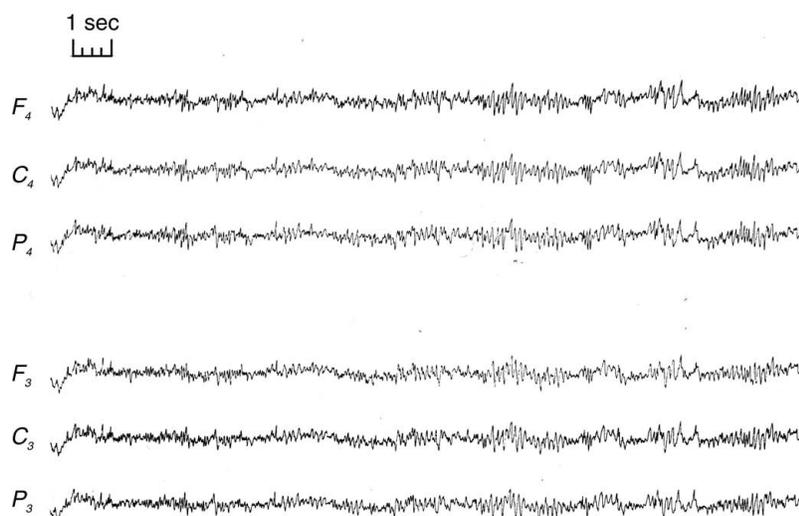


FIGURE 13.1. Normal human EEG: three channels recorded from the right (above) and left (below) hemispheres; common extracerebral reference. Bursts of alpha activity may be seen, especially to the right of center.

tissue, but these problems, caused by the directional nature of the propagation of the electrical activity, need not worry us for the present.

The electrodes are usually placed directly over the area of the brain that is of interest, and usually at points located with reference to a system of labeling known as the *10–20 system* (because it divides distances on the head in terms of 10% and 20% of the distance between fixed landmarks), which is shown in Figure 13.2. Modern systems commonly employ a “cap” that fits snugly over the head and is intended to automatically locate the electrodes, although its success in accurately achieving this is somewhat in doubt. A set of electrode positions is referred to as a *montage*.

Although, as mentioned previously, we cannot directly record the activity from one electrode, but only the difference between two electrodes, we can accept that we are recording the difference between two “active” electrodes (both recording electrical activity from the brain) and so make a *bipolar* recording. Such recordings are quite common in clinical work, but they present difficulties in research. When the potential difference between electrodes changes, which electrode has changed? Almost certainly both will have changed, and it is impossible to unravel the contribution of each to the final observed change in the EEG.

In order to get around this problem we may choose to make a *monopolar* recording, where one of the two electrodes is relatively

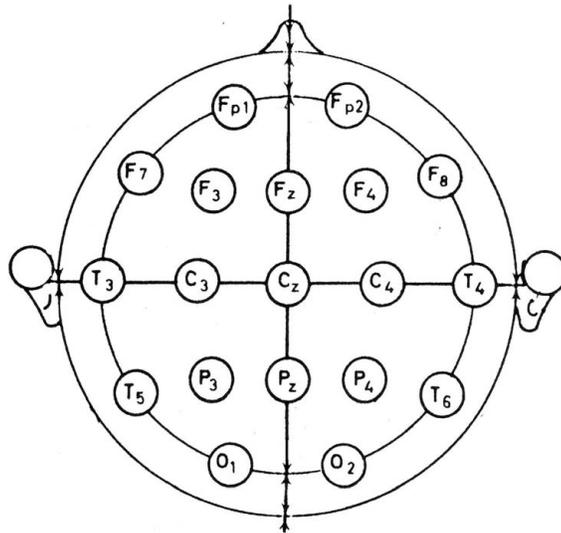


FIGURE 13.2. Principal points on the 10–20 system of electrode placement.

“inactive.” However, it is almost impossible to find sites on the head at which brain electrical activity will not be recorded, including the tip of the nose or the tongue, and we must also avoid recording eye movements, muscle potentials, and other artifacts. We might move our inactive electrode off the head (extracerebral reference), but then we find the activity of the heart and other bodily processes contaminating our recording, although there are techniques that allow these to be filtered out. The usual solution is therefore to choose a site that will pick up some EEG activity but will be relatively inactive with respect to the activity in which we are interested. Such sites are often on the midline, and frequently the *vertex* (Cz) or midpoint on top of the head is chosen. The mastoid processes (behind the ears) and other sites are also used, sometimes in linked pairs to cut out any lateral bias (although linking is probably ineffective in doing this).

Further, it is common to employ a single reference for all the active electrodes being used, in a *common reference* recording. The reference may not be truly inactive, but at least when a comparison is made across channels, its contribution to each channel will be the same, and differences between the channels associated with active electrodes will reflect differences between the activity at those sites.

There are other more technical problems associated with a selection of electrode site and recording montage, especially if lateral asymmetries are to be investigated, which has been the aim of much of the research in this field (see Beaumont, 1982; Donchin, Kutas, & McCarthy, 1977). Many of these problems can be solved by careful experimental design but one fundamental problem, as yet unsolved, is that of underlying asymmetries of the brain. We know that the brain is asymmetrical, particularly in certain regions, and there is now fairly good evidence that these asymmetries have functional significance, but electrodes are applied to symmetrical points on the scalp. If we detect asymmetries in the EEG at homolateral points, do these represent lateral asymmetries in the activity of homolateral regions or merely indicate that we are not recording from anatomically homolateral points on the brain? This is an extremely vexing problem, which, until we can determine individual differences in anatomical asymmetries, is insoluble.

Once we have battled with all these problems, have selected the task to set the subject (also fraught with difficulties that may produce artifactual results in the EEG), and have made our recording, the problems are not over. A form of data reduction and analysis must be selected. The difficulties here spring, quite simply, from ignorance: if we really understood what we see in the EEG trace, decisions about how to analyze it would be less arbitrary. While much is known about the origins of the EEG (see Thatcher & John, 1977, for a good introduction),

we still do not know how a complex pattern of changes relates to mental events. It is usual however to analyze the frequencies present in the recording, and to divide these up into several frequency bands:

| | |
|---------|-------------------------------|
| Delta: | 0 to 3.5 cycles/second (Hz) |
| Theta: | 4 to 7.5 cycles/second (Hz) |
| Alpha: | 8 to 12.5 cycles/second (Hz) |
| Beta 1: | 13 to 19.5 cycles/second (Hz) |
| Beta 2: | 20 to 29.5 cycles/second (Hz) |

These bands are not, however, precisely fixed, and there is some variation in the limits applied to them. Research initially concentrated almost exclusively on alpha activity, although all the bands are potentially of interest and there is now a recognition that equally interesting results may be found in other bands, especially theta and beta 1.

The *power* of a frequency band, or of a specific frequency, is usually calculated, and represents an overall measure of the strength of that component in the EEG during a specified period. Mental activity usually results in a decrease in the power of alpha activity, and an increase in the power of other frequency bands, especially beta 1. The former effect is referred to as alpha *attenuation* or alpha *abundance*, and the latter as *enhancement*. Power, however, confounds the amplitude with the duration of the component, that is, a very powerful component present for part of the time may result in the same overall power as a weaker component present for all of the period. These power analyses, however, are a useful if very crude way of quantifying the activity.

Online computers have enabled a variety of more complex analyses to be performed, in particular certain forms of frequency analysis. One of the more promising of these is *coherence analysis*. This analysis takes a pair of channels and describes the amount of shared activity at the two channels at each frequency, but independently of the power of that frequency in the two channels (Shaw, 1981). The particular value of this form of analysis, apart from its independence from power, is the fact that it can be calculated from relatively short periods of EEG recording, down to about 0.5 seconds, although a number of such short *epochs* have to be combined to provide a reliable estimate of the coherence. It is possible to identify some period when a particular cognitive process might be active, and then to examine the coherence between sites during that period. We can therefore begin to build up a dynamic map of the brain changes associated with relatively specific cognitive events, and to do this separately for each frequency across the range of the whole band of the EEG. Often associated with the *coherence spectrum*, as the plot of the coherence across the whole band of frequencies is called, is a *phase*

spectrum, which can indicate which channel leads the other when the two share a significant amount of activity at some given frequency.

There are of course difficulties with coherence, technically and in interpretation. It is, for example, still uncertain whether we should expect coherence to increase or decrease with task-related activity in some region. It seems that there is a local increase, but a decrease with reference to more distant sites, and much more fundamental work is required before we can appreciate what this and other recently developed techniques can tell us.

In the EEG, we do have a fairly direct record of the activity of the brain, and while we may have difficult decisions to make about how to analyze the complexity of what is recorded, we are at least dealing with brain processes in a relatively immediate way. We may still expect advances in understanding the EEG, and the brain processes that it reflects, in the future.

Recording Evoked Potentials

Evoked potentials (EPs), also known as averaged evoked potentials (AEPs), averaged evoked responses (AERs), and event-related potentials (ERPs), are an alternative way of studying psychological processes in the electrical activity of the brain. The common use of these abbreviations, further complicated by the addition of the modality of stimulation (hence visual, auditory, and somatosensory evoked response: VER, AER, SER), can be somewhat confusing, but they all indicate measures based on the same basic technique.

This technique relies upon computer averaging, and has only been possible since the advent of relatively inexpensive laboratory computers. The idea behind it is that there is a relatively invariant response by the brain to a given sensory event. It is not possible to see this response in the EEG because it is masked by the large amount of background noise in which the response is embedded. However, if the noise is considered to be random, and if a number of examples of the response to a given event are collected and added together, then the noise at any time will tend to cancel out, allowing the event to emerge out of the background noise. Calculating the average, rather than simply adding to find the total, allows the number of examples collected to be taken into account. An illustration of the principle behind the technique, using an artificial waveform, is shown in Figure 13.3.

In practical terms, a simple sensory stimulus that can be accurately reproduced is selected, and this is presented a large number of times (commonly between 64 and 512) to the subject. Time-locked to the stimulus presentation, a short epoch of EEG is collected (between 500 and

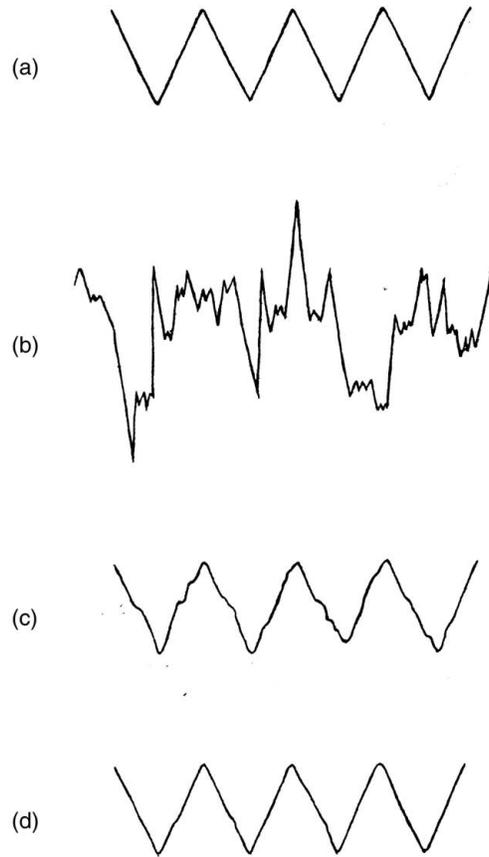


FIGURE 13.3. Illustration of the principle of waveform averaging: (a) the underlying waveform; (b) with added random noise; (c) after averaging 32 samples of the noisy signal; (d) after averaging 512 samples of the noisy signal, showing that the waveform has been accurately extracted.

1,000 msec, around the stimulus), and stored in the computer. These epochs or samples are averaged as they are collected, and usually displayed during data collection. Subsequent analyses can then be performed. Examples of typical evoked potentials from different sensory modalities are shown in Figure 13.4. (A note of warning here: physiologists prefer their graphs negative up, while engineers and psychologists generally adopt positive up, so take care about which way up an EP has been plotted.)

The recording electrodes and initial stages of the recording are just as for an ongoing EEG and many of the problems surrounding electrode

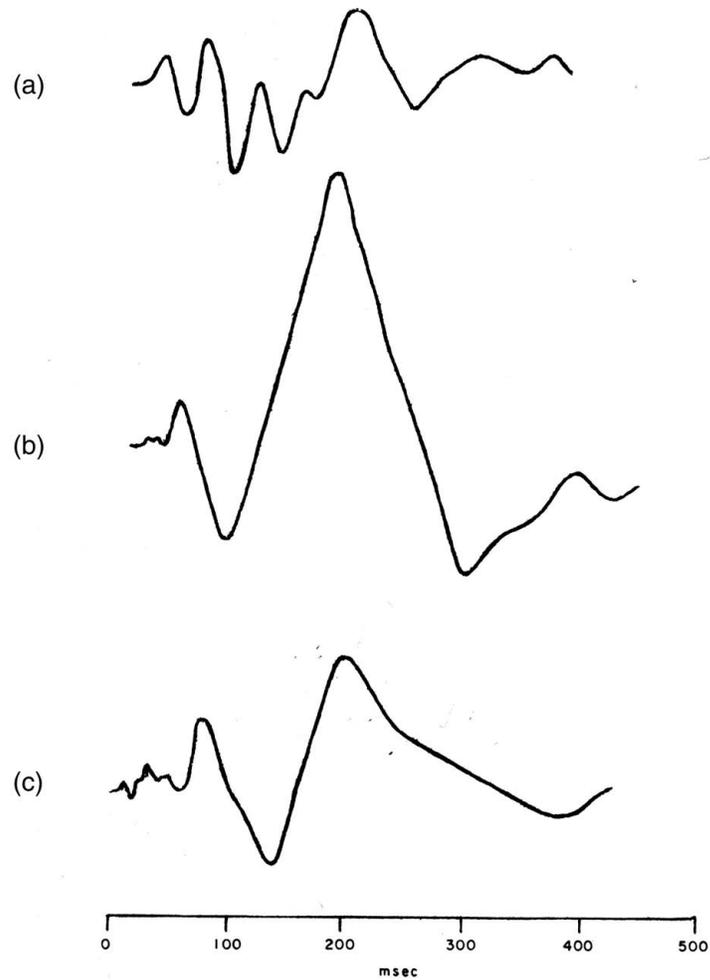


FIGURE 13.4. Typical average evoked potentials (positive up) in: (a) visual, (b) auditory, and (c) somatosensory modalities.

placement apply equally to EP recording. A rather different set of problems arises, however, when we come to analyze the evoked potential records. Even though the absolute amount of data will be less than in an EEG record, there is still a formidable problem of data reduction. Again, our difficulties in deciding how to extract the most important features from the EP stem from our relative ignorance of what it is that we are looking for.

Most EPs contain a series of peaks and troughs, known as *components*, which presumably relate to significant brain events. It is usual to

identify these components and to measure their location in time with respect to the stimulus event (*latency*), together with the *amplitude* of the response (commonly in the range of ± 15 microvolts). We can then compare the amplitude and latency of given components across different recording sites and in different experimental conditions.

Things are rarely so simple, however. Although most subjects show a similar response pattern to a fairly clearly defined stimulus, a given component may simply not be present in the record from a certain subject. There may be two peaks of equal amplitude very close together: which is the component we are measuring, and should we average the latencies? One component may be superimposed upon another, and it may be difficult to extract the “true” amplitude and latency information about both from the record that has been obtained. Also, the amplitude of one component may be influenced by the preceding component, and should this be taken into account? The use of peak-to-peak measurements of amplitude is one attempt to account for these sequential effects. What should be our baseline for measurement: is it zero volts, the average voltage of the whole response, or some prestimulus baseline?

None of these problems is readily soluble, although there are conventions that have been adopted to deal with most, and a number of computer algorithms that may help with the description of the components present in a set of evoked responses. There is, however, a further problem: that of *latency jitter*. One of the assumptions of the averaging technique is that the underlying response is invariant. If, however, there is some basic variability in the latency of a component, this variability will only be seen in a reduction of the amplitude of the averaged compo-

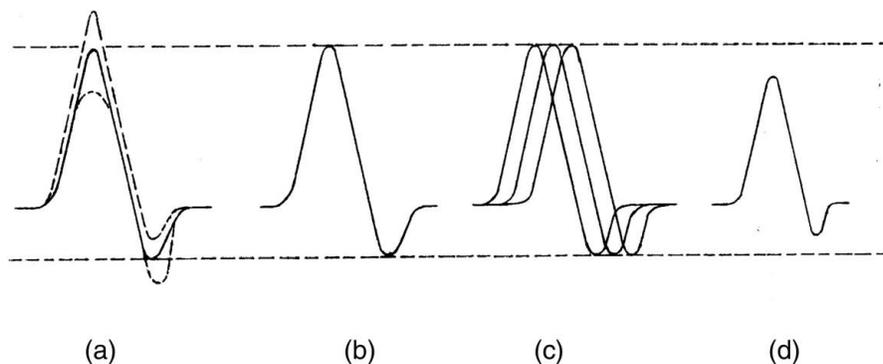


FIGURE 13.5. Latency jitter: averaging samples of differing amplitude but constant latency (a) yields the average (b); averaging samples of the same constant amplitude as (b) but differing latency (c) yields the average (d), which is of lower amplitude than (b).

nent (see Figure 13.5). Care must therefore be taken to ensure that amplitude differences between sites or conditions represent true amplitude differences and are not the effect of variable latency in the component. Inspection of the variability of the samples that make up the average, together with filtering techniques that correct for the latency of components, can help to deal with this problem. (Important discussions of EP methodology are to be found in Callaway, Tueting, & Koslow, 1978; Desmedt, 1977; and Swick, Kutas, & Neville, 1994.)

There are some conventions about the labeling of commonly occurring components seen in EPs but none of them is universally accepted. One approach is to number the components sequentially, so that P1 is the first main positive peak and N1 the first negative inflexion, followed by P2, N2, P3, and so on. This, however, tends to be rather confusing as the components tend to have different latencies with stimuli in different modalities. An alternative is to label the components as N100, P300, and so on, to indicate their polarity and approximate mean latency. An extension of this is that P300 comes to mean the theoretical component associated with P300, even if its latency is perhaps as long as 450 msec from stimulus onset.

Unfortunately but inevitably this is confusing. However, it is worth noting that the two components that are perhaps most discussed are N1, which occurs in the visual modality at about 90–120 msec from stimulus onset, and P300, which has a latency of 300–450 msec. Both these components have been clearly linked to cognitive processes. In general, the early components, up to about 100 msec from stimulus onset, are thought of as being *exogenous* components, determined by the sensory reception and perception of the stimulus. The middle-range components that follow (100–300 msec) and the subsequent late components (up to 1,000 msec) are thought of as *endogenous* and related to cognitive manipulation, stimulus evaluation, and decision making. In one special case, the brain stem evoked potential, it has been possible to identify components associated with each stage of the sensory transmission up to the cortex. With later endogenous components, things are much less well understood, although there are a number of theories about the cognitive significance of various components.

Mention should finally be made of the so-called slow potentials, generally seen as slow negative changes in the EEG preceding an expected event. These responses, or CNVs as they are sometimes called (contingent negative variation), can be recorded by similar averaging methods, and are thought to represent preparatory processes within the brain.

Evoked potential recording has been perhaps more successful than direct EEG studies in eliciting phenomena of psychological significance. Particularly where the stimulus event and task are relatively simple and

clearly defined, the technique is extremely valuable. With complex tasks and stimuli, it becomes more difficult to use the technique, and the need for a relatively large number of repetitious trials enforces a certain artificiality upon the experimental design. Despite this, and the numerous methodological problems and pitfalls, evoked potential recording has become an important specialty within neuropsychology.

Lateral EEG Asymmetries

As the bulk of research with ongoing EEG measures has been directed to establishing lateral asymmetries in the EEG, a review of this topic will serve to illustrate the neuropsychological research using such measures.

All the substantial reviews (Beaumont, 1982; Donchin et al., 1977; Kertesz, 1994; Marsh, 1978) have commented upon the methodological shortcomings of the studies that have been carried out. In fact it would be fair to say that all studies (including those in which I have been involved) can be regarded as having methodological limitations. The view that one takes of the literature depends upon a judgment of the seriousness of the methodological shortcomings.

The approach in most research designs has been to set the subject some task or tasks to “engage” the left or right hemispheres, and then to look for asymmetries in the accompanying EEG. These asymmetries might be seen by comparing the two hemispheres directly (between hemisphere effects) or by comparing intrahemispheric (within hemisphere) effects occurring on the left and on the right. The better studies have also recorded behavioral task performance alongside the EEG. This not only enables an examination of the relationship between EEG and performance parameters but, more mundanely, helps to ensure that the subject actually carries out the task that he has been set. It can also help to provide an independent measure of whether tasks selected to engage the left and right hemispheres have been satisfactorily matched in difficulty.

We might ask first whether there are relatively “good” studies that do find an effect of cognitive task. Davidson, Taylor, Saron, and Snyder (1980), for example, gave subjects tasks such as an embedded figures task, the Kohs blocks, and verbal analogies and reading comprehension tests, and found an asymmetry in right-handed subjects, although only for those with sinistrals (left-handed individuals) in the family. Ehrlichman and Wiener (1979, 1980) also used various verbal and spatial tasks and asymmetries in integrated alpha power, finding a significant reduction in power (which is what would be expected) over the engaged hemisphere. They repeated their experiment using covert as well as overt responses, because of the suspicion that overt responding may

introduce artifactual asymmetry into the EEG, and found their earlier results supported. It should be noted that their tasks, as with those in all similar studies, are only *expected* to engage one of the hemispheres preferentially; there is no independent evidence to demonstrate that they will do so (apart from clinical investigations).

Tasks such as counting the words *the* or *a* in an auditory passage, or listening to music, were used by Moore (1979), and similar tasks but without counting were used by Moore and Haynes (1980), resulting in different asymmetries for the two tasks, as was predicted. Parallel results have come from Ornstein, Johnstone, Herron, and Swencionis (1980) and Rebert and Low (1978). The latter study also involved listening to verbal material, but had as the spatial component a block construction task performed entirely in imagination, which seems rather odd. Beaumont and Rugg (1979) used two memory tasks, carefully matched for difficulty, for which there was clinical evidence of association with left and right anterior lobes; the tasks involved judging which of a pair of either words or abstract paintings had been previously presented in a sequence of similar items. A task asymmetry in intrahemispheric coherence (see p. 268), at the expected cortical sites, was found. There are a number of other studies that would also support the existence of task-related asymmetries on various EEG variables.

We must also ask, however, whether there are “good” studies that have failed to find the expected asymmetry. The study by Beaumont and Rugg just mentioned found the effects on coherence that were predicted, but found quite anomalous enhancement of alpha power with the cognitive task; further data on coherence are provided by Weiss and Mueller (2003). In the study by Ornstein et al. (1980), although task effects were found at centrally sited electrodes, they were not found at parietal placements. Rebert (1977) has also found alpha enhancement, rather than the expected attenuation, in the right hemisphere with a dot detection task. Rebert and Mahoney (1978) found no task effects at all in raw power measures taken during verbal and nonverbal target detection tasks. Visuospatial tasks designed to be analytic or synthetic in their cognitive demands failed to produce clear task-related effects (Tucker, 1976), and although Dolce with others (Dolce & Waldeier, 1974; Dolce & Decker, 1975) found complex and interesting effects in the beta, theta, and delta ranges, no task effects upon alpha activity were seen. Asymmetries in relation to mood and cognition have been reported by Gotlib, Ranganath, and Rosenfeld (1998), and Bell and Fox (2003). McCarthy and Donchin (1978) used a task derived from the “chimeric metacontrol” studies with split-brain patients, in a laudable attempt to produce well-matched “left and right hemisphere tasks,” but found no task-dependent asymmetries in alpha power.

Finally, we must ask whether there are “good” studies that find effects but suggest that the effect may be artifactual. The studies of Gevins and co-investigators (Gevins, Zeitlin, Yingling, et al., 1979; Gevins, Zeitlin, Doyle, Schafler, & Callway, 1979; Gevins, 1981) are particularly relevant here. They conducted a series of experiments in which particular efforts were made to control for stimulus characteristics, the effects of eye and limb movements, and other performance-related factors, such as the subject’s ability and task engagement. Their conclusion was quite clear: when such factors were effectively controlled, task-related asymmetries, which had been otherwise observed, disappeared.

Other artifactual sources of asymmetry are suggested by the work of Amochaev and Salamy (1979) and Haynes (1980). In the first study, different sites for the reference electrode were compared, and they found the stability of the task effect, using a variety of tasks, to be related to the site chosen. The study by Haynes, in which subjects listened to sentences that were either to be imitated or followed by the construction of a new sentence, found left hemisphere activation only when imitation was demanded. This was interpreted in terms of the preparatory phases of motor programming for ensuing speech, and suggests that even when EEG is recorded during a period of inactivity, subvocal preparation, or preparation for other response processes, might be contaminating the EEG.

What are we to conclude? If we adopt a fairly relaxed criterion of methodological rigor, then the bulk of studies point toward task-related asymmetries in the EEG. The asymmetries are those that would be predicted from the divided visual field and dichotic listening literature. That is the general trend of the results. However, the failure of a number of reputable studies to find effects, and the clear demonstration of a number of possible sources of artifact, must mean we have to conclude that clear and reliable task-related effects have not as yet been demonstrated. It does not mean that the search for them should be abandoned, and improvements in methodology should lead to a clearer answer to whether asymmetries are to be observed in ongoing EEG.

Evoked Potential Studies

When employing the evoked potential methodology to answer neuropsychological questions, there are two major strategies available. The stimuli that are used to elicit the EP may be task relevant (and may or may not be lateralized) or else may be incidental to the task. If task-relevant stimuli are used, there are problems about the control of such necessarily complex stimuli and also about obtaining strong potentials. On the other hand, it is not clear that if EPs are evoked incidentally during per-

formance of some cognitive task by simple “irrelevant” stimuli, the response to these “probe” stimuli will reflect the concurrent task engagement. Studies have been reported using both approaches, and have tended to be in either the visual or auditory modalities.

Taking visual EPs first, a good example of a study using task-relevant lateralized stimuli is that of Ledlow, Swanson, and Kinsbourne (1978). They used the nominal/physical letter-matching task (see p. 230) and collected performance data together with the EPs in order to establish independent evidence of lateral asymmetry in cognitive processing during the task. Their results are complex, but they found an overall difference in the amplitude of left and right hemisphere potentials, and for some components the direct pathway (when stimuli were presented to the hemisphere contralateral to response: see Figure 11.2) was associated with responses of smaller amplitude but reduced latency. At P300 there were differences between direct and indirect stimulation that also varied with type of match (nominal or physical), and also an effect of same or different cases at P130 and N170.

A similar experimental design was used by Rugg and Beaumont (1978). Letters were employed as stimuli throughout, but subjects were asked either to respond to letters containing an “ee” sound (verbal task) or to letters containing a right angle (spatial task). Variations in the amplitude of middle latency components with field of presentation were found that differed according to the cognitive task. A lateral asymmetry was found for late components, but only with the spatial task.

By contrast, Friedman, Simson, Ritter, and Rapin (1975) collected responses to sequentially flashed words that made up one of three sentences: “The wheel is on the axle,” “The heel is on the shoe,” “The peel is on the orange.” On some trials, the first part of the second word was not presented, so that from “-eel” the subject could not tell which sentence was being shown until the final word arrived. The second word therefore might deliver information or be indeterminate. The latency of P300 was longer for words delivering information, and “syntactic closure” was also shown to affect P300 amplitude. However, no lateral asymmetries were found between the hemispheres.

McCarthy and Donchin (1978), as already indicated, have used the structural–functional matching task first used with split-brain patients and have collected not only EEG data but also EP recordings. There was not only a visual EP to the stimulus figures, but also a warning tone that in some conditions served to indicate the type of match required and that evoked an auditory response. Although there was no effect of mode of matching on the EEG, during the preparatory period following the warning tone a component was seen that was sensitive to the mode required, and that had an asymmetrical distribution across the electrode sites employed.

The alternative of incidentally collecting EPs to flash stimuli during an ongoing left or right hemisphere task was developed by Galin and Ellis (1975). They found changes in the asymmetry of the potentials recorded at left and right temporal and parietal areas that parallel task-dependent asymmetries in both EEG alpha power and in performance measures. These effects have, however, not always been confirmed by other researchers (Beaumont & Mayes, 1977; Mayes & Beaumont, 1977).

In the auditory modality, the “identical stimuli but variable task” design has also been used, for example by Wood (1977). Reaction times and EPs were collected during tasks that focused upon various phonetic characteristics of the stimuli, with the result that differences were found in the left, but not the right, hemisphere between 60 and 140 msec after stimulus onset. This was taken to support the independence of auditory and phonetic processes. Tanguay, Taub, Doubleday, and Clarkson (1977) presented monaural voiced stop consonants (e.g., /ba/, /da/, /ga/) and found increased amplitude of response contralateral to the ear of presentation, but no hemisphere differences in latency or amplitude. Nevertheless, using musical chord stimuli, Taub, Tanguay, Doubleday, Clarkson, and Remington (1976) found lateral asymmetries as a function of hemisphere stimulated, in middle-range components and at sites close to Wernicke’s area.

The auditory parallel to the visual ambiguity task, described above, has been extensively investigated by Brown and Marsh with others (see Brown, Marsh, & Smith, 1979). They used sentences that contained an element with a given sound but with different meanings according to the context, for example, the words *led* and *lead* in “The horse was led” or “The metal was lead,” and *fire* in “Ready, aim, fire” or “Sit by the fire.” The results from these studies are complex, but effects have been found that are sensitive to parts of speech, as well as lateral asymmetries in certain components, some of which suggest the involvement of left anterior cerebral processes.

Task irrelevant probes with an ongoing auditory task have been used by Shucard and Shucard (1979) and found to be associated with engagement in either musical or verbal tasks. Musical performance has also been examined by Harris and Silberstein (1999) and by Aaron and Tobias (2004). Both auditory and visual stimuli have been examined by Davis and Wada (1978), who performed spectral analysis on their EPs and found greater coherence in the left hemisphere with click stimuli, while flash stimuli produced this effect in the right. Not only were these hemisphere effects observed, but they were also found to vary with the speech lateralization of the subjects, which had been independently assessed.

Attentional variables have been assessed through evoked potential recording, and in both auditory and visual modalities. There is considerable debate about the effects of attention on EPs, especially upon the N1 component (see Näätänen & Michie, 1979), with various groups of researchers contributing elegant experiments. Most of these experiments rely upon the presentation of lateralized auditory material, with attention being directed to one of the ears. Lateralized recording sites have been included as well as central sites, and some but not all of these have revealed asymmetrical components. Notably, Buchsbaum and Drago (1977) have reported clear asymmetries in an N140 component with visual presentation, which could be manipulated by attentional variables. The importance of these experiments lies not in the precise results, but in the demonstration that manipulation of the cognitive variable of attention may have effects that can be seen in electrophysiological recordings.

Research has not been limited entirely to the visual and auditory modalities, and somatosensory potentials (Barrett, Halliday, & Halliday, 1978; Saron, Foxe, Schroeder, & Vaughan, 2003) as well as movement-related effects (Kutas & Donchin, 1978; McCallum & Curry, 1979) have been shown to exhibit lateralized components.

The experiments mentioned above serve as examples of the kind of research that has been conducted using evoked potentials. The research literature is quite extensive, the pattern of results reported is extremely complex, and, as already indicated, methodological difficulties raise considerable problems in considering many of the studies. Happily there are several good reviews of the literature (Hillyard & Woods, 1979; Marsh, 1978; Rugg, 1982a, 1982b), and there seems no reason not to accept what is the general opinion of these reviews: that EPs appear sensitive to both the locus of cognitive processes and to factors that reflect the mode of stimulus presentation, and the resulting effects upon EPs are the product of a complex interaction of such factors.

MINOR PHYSIOLOGICAL TECHNIQUES

This seems an appropriate point at which to note that, while the major electrophysiological techniques employed in experimental neuropsychology have now been considered, there are a number of less widely used physiological techniques that have contributed to our present knowledge.

Psychophysiological measures such as skin conductance, muscle tension, and finger pulse volume have also been used, but the results, while interesting, are not entirely clear. The interpretation of results is

complicated by the ipsilateral efferent projection of the autonomic nervous system, and it is difficult to predict the direction of change in autonomic variables.

Anatomical investigations are also of interest to neuropsychologists. It is now clearly established that there are certain marked anatomical asymmetries in the topography of the cerebral cortex, that these are present from birth, and that there is some reason to associate them with functional specialization (Galaburda, LeMay, Kemper, & Geschwind, 1978).

BRAIN IMAGING

If there is a revolution occurring in neuropsychology, it is in the contribution of medical imaging. Indeed, things are changing so fast that, whatever I write may be substantially out of date before I have even finished writing this chapter, let alone before the book appears in print. All that I can do is provide a rather rough guide to the techniques and their uses. Beyond that, students are strongly encouraged to take a trip to the library or, more likely, sit down at their electronic literature search facility to ascertain the current “state of the art.”

Computerized Tomography

Although computerized tomography (CT; or in the earlier literature, computer-assisted tomography, CAT) now seems rather mundane, its introduction in the 1970s was quite remarkable and initiated the modern revolution in medical imaging, earning its developers, Godfrey Hounsfield and Allan Cormack, a Nobel Prize in 1979.

The technique employs X-rays directed at the body from a large number of orientations, with the X-ray source and detector continuously positioned opposite each other, and the body in between. The information from each of these images, which can be obtained using very low doses of radiation, is combined by computational methods to reconstruct “slices” through the body. A CT scan of the brain produces a set of images through consecutive planes of the brain in which, as in conventional or “plain” X-ray images, bone and other dense material appears white, air and water appear dark, and the brain substance is represented as varying shades of gray. The image is a representation of the structural state of the brain.

CT scans (see Plate III for an example) now appear very crude by comparison with more recently developed techniques, but they are relatively inexpensive and widely available, and are an important clinical tool for the neurologist and the trauma specialist. CT scans of the head

are now routinely performed following head injury, or to investigate suspected neurological abnormalities, and are an important clinical tool. However, CT is not capable of imaging all abnormalities, has relatively low definition, and is no longer of significance in neuroscience research.

Magnetic Resonance Imaging

A far more detailed image of the anatomical structure of the brain can be obtained by employing magnetic resonance imaging (MRI; also less commonly known as nuclear magnetic resonance imaging, NMR). Essentially, the body is placed in a very powerful magnetic field so that the atomic particles of the tissues being scanned are aligned in the same direction. Radio waves directed at the body then give off radio signals that differ according to the type of tissue; as in CT, “slices,” or 3-D reconstructions of the brain can be reconstructed as images. By comparison with CT scans, MRI images possess extraordinary detail and clarity (see Plate IV), and this form of imaging has become the gold standard for the visualization of normal and abnormal anatomical structures, in the head or in any other part of the body. More expensive than CT scanning, it is nevertheless widely available and routinely applied, but it can provide no more than a picture of structures in the body; it cannot tell us what these structures do (Perani & Cappa, 1999).

Functional MRI

Since the early 1990s the MRI technique has been developed in order to provide images, not only of structure, but also of function. Functional magnetic resonance imaging (fMRI) achieves this by detecting the flow of glucose and oxygen in the blood to areas where the neurons are most active and therefore consuming these substances. It is possible to acquire four or more images every second and therefore to obtain a dynamic, if rather crude, picture of the brain in action as it deals with cognitive processes.

An important aspect of fMRI technology has been the use of the *subtraction technique*. This is based on the idea of comparing the activity of the brain engaged in a particular task with the state of the brain “at rest,” or performing a neutral task. The differences between the two sets of images are calculated by simple subtraction, revealing the areas of activity that are particularly associated with task performance; it is an image of these differences that is displayed (for an example, see Plate V).

Functional magnetic resonance imaging has replaced the earlier technique of recording regional cerebral blood flow (rCBF) in which short-lived radioactive isotopes were attached to molecules in the blood through an injection, and their progress through the head, and areas of

local concentration, detected. Although employed within limits of safety, rCBF was limited by the number of scans that could be obtained and by how often it could be repeated. In contrast, fMRI is without such safety concerns and there are no problems in performing repeated or multiple scans. Although expensive, and only available in a small number of specialized centers, fMRI has become the most important of the functional imaging techniques in experimental neuroscience.

An example of the way that fMRI is currently used within neuropsychology is the study of Boronat and colleagues (2005). They were interested in the idea that conceptual knowledge is distributed through the various sensory, motor, and verbal-declarative attribute domains in a way that reflects the activity of these systems at the time the knowledge was acquired. Answering questions about this knowledge should then reactivate these systems. In their study, answering questions about object manipulation was predicted to activate the sensory-motor system employed in object use, and questions about object function to reactivate the verbal-declarative system. Participants viewed picture or word pairs identifying manipulable objects and were asked to decide either if the objects were manipulated similarly, or if they had a similar function; fMRI demonstrated that decisions about manipulation were associated with greater activation of the left inferior parietal lobe, more especially with pictures than with words. This is the region that is thought to support skilled object use, according to more traditional neuropsychological studies. No brain regions were found to be particularly associated with decisions about object function. Their results therefore partly supported the hypothesis concerning distributed representations.

There is now a large research literature employing fMRI, although the contribution it has made to neuropsychological understanding, to date, has not been proportionate to the research effort involved. Attempts continue to be made to develop increasingly powerful machines, involving magnets weighing many tons, in order to improve the clarity and specificity of the images, but the technique has an inherent limit of its temporal resolution, and also requires participants to be tested in a rather unusual environment. Although the scanning environment with the most modern machines is less frightening than the claustrophobic tubes into which participants were previously inserted, there is evidence that the assessment environment in itself may change the nature of cognitive functioning and, for example, impair memory performance in some individuals (Gutchess & Park, 2006).

Positron Emission Tomography

A precursor to fMRI, which is still applied, is positron emission tomography (PET). PET, which is a direct development of rCBF, is an alterna-

tive method of dynamically imaging the function of the brain. Although the images are exceptionally clear (and often quite beautiful), the technique is expensive, participants may only be subject to about 10 scans within a 12-month period, and it takes about 30 seconds to acquire each scan. There is therefore little hope of identifying specific cognitive processes from PET scans.

Nevertheless, PET scans continue to be of some importance, and an interesting example is provided by a previous student of mine, Eleanor Maguire (Maguire, Frackowiak, & Frith, 1997). She studied London taxi drivers, who are required to pass a stringent test of their knowledge of London streets (known as “the Knowledge”) before gaining their license, and naturally access this information in their memory on a daily basis. Experienced London taxi drivers showed an activation of the right hippocampus together with a network of other structures when recalling complex routes around the city, in comparison to baseline and nontopographical memory tasks. Nontopographical memory retrieval involved the left inferior frontal gyrus, with no activation of temporal regions (associated with the hippocampus). Incidentally, for those who might dismiss standard MRI as a neuropsychological technique, this group has also shown that the hippocampus is structurally more developed in these taxi drivers than in comparable controls without “the Knowledge.” Of course, whether this is a development consequent upon acquiring these special topographical skills, or whether individuals with greater potential based upon brain structure self-select to enter this form of employment, is an open question.

A further illustration of the way in which PET has been employed to investigate neuropsychological models is to be found in Peigneux et al. (2004), who investigated the components of upper limb apraxia by requiring participants to undertake tasks of pantomiming familiar gestures to verbal command, imitating familiar gestures, imitating novel gestures, and matching objects for functional use. They were able, on the basis of their results, to refine and extend an existing model of apraxia based upon the clinical neuropsychological literature.

Magnetoencephalography

The most exciting, and latest, development in imaging is magnetoencephalography (MEG). MEG at last offers the prospect of both high spatial and temporal resolution in functional mapping, is entirely non-invasive, and promises to make a considerable impact in neuroscience (Preissl, 2005; and see Plate VI).

This technique can be thought of as similar to EEG, but rather than detecting the electrical fields associated with groups of neurons, MEG detects the magnetic signals that accompany brain activity. These mag-

netic fields are extremely weak, and recording them has been compared to trying to detect the sound of the footsteps of an ant in the middle of a rock concert, but they can be detected by an array of superconducting coils. The efficiency of the device is increased by operating it in a special room shielded from magnetic and radio frequency signals and bathing the coils in liquid helium at -269 degrees Celsius, within an insulated cryogenic vessel known as a dewar. This is therefore a demanding and expensive technique and MEG machines are not yet widely available.

The remarkable potential of MEG is that it is capable of identifying specific areas of activity as small as 2 mm and with a temporal resolution of 1 ms. In this it dramatically outperforms any of the available imaging and electrophysiological techniques—at least, this is what is claimed by the proponents of MEG, although the degree to which the technique can precisely identify particular locations is still rather controversial. An example of the use of MEG can be found in the study of Kaehkönen, Rossi, and Yamashita (2005) in which MEG was used alongside traditional electrophysiological recording of auditory evoked potentials (AEP). (The use of different imaging techniques in combination has been a general strategy to improve the information that can be obtained, and to aid interpretation.) They examined the effects of alcohol on the auditory processing of frequency changes, presenting infrequent deviant frequency tones and novel sounds in a double-blind, placebo-controlled, design. Alcohol was shown to decrease the amplitude of AEP potentials, but not their latency, and to have a differential effect for tones, frequency change, and novel sounds. The MEG results confirmed these changes but provided additional and more detailed information not revealed by the AEPs.

Tractography

A final recent innovation deserves a brief mention. This is an alternative development of MRI into *diffusion-tensor* imaging (DTI) and fiber tractography (FT) (Mori & van Zijl, 2005). These are anatomical, rather than functional, techniques and are likely to be of greater importance clinically than as a research tool for investigating cerebral function. The technique relies upon the fact that water diffusion occurs differently in the gray matter of the cortex and the white matter of the “wiring.” Essentially, water diffusion occurs more rapidly along a white matter tract than across the boundary between the tract and surrounding tissues. Magnetic resonance imaging has been adapted to detect these patterns of diffusion and so create 3-D images of the fiber tracts within the brain. The images are often superimposed upon “standard” MRI images to give context and orientation. The images are striking, and reveal a

complexity to these pathways that belies our simplistic notion of regions of activity rather directly connected by simple bundles of fibers; but then the brain is undoubtedly more complex than the ideas that we can hold about it. An example of the application of tractography to an understanding of blindsight (see p. 127) can be found in Leh, Johansen, and Ptito (2006).

CONCLUSION

Having briefly reviewed the techniques employed in electrophysiological investigations and studies employing brain imaging, and the kinds of experiments that have been performed, let us consider what general result has emerged. On the one hand it would seem that there is remarkably good evidence, given the technical demands of the techniques, for the identification of cerebral processes that mirror those inferred from human performance studies. The results appear to fit remarkably well with what might be expected from divided visual field and dichotic listening research.

On the other hand, it is worth noting that there are considerable differences in the kinds of activity studied in clinical and experimental research. The experimental techniques, and especially the electrophysiological methods, demand a more passive involvement on the part of participants, and it has to be recognized that rather different domains of behavior are being studied by each of the different approaches and techniques.

It must also be remembered that there are considerable methodological difficulties in both the EEG and EP literature. Close inspection of the reported results also reveals little unanimity about the precise effects to be observed, and there is little replication of experimental findings. Much of the interpretation of results is uncomfortably post hoc, and our ignorance about just what to look for in either the EEG or in EP components means that it is almost impossible to construct sound experimental tests of precise neuropsychological hypotheses.

Brain imaging is currently the most fashionable area of experimental neuropsychology. I have a tendency to feel, as a personal view, that it has yet to fulfill its promise or, at least, to deliver the outcomes that were promised for it. The inherent limitations of the various techniques have not always been fully acknowledged or accepted, and a consequence has been that less glamorous, but more soundly established, techniques have been relatively neglected. Almost 20 years ago, a very level-headed friend who works in imaging told me that he thought it would take 20 years before imaging made a real contribution to neuropsychology. It

starts to look as though he was entirely right. After a period in which too many grand claims—that could not really be supported—were made for imaging, a more recent wave of advances employing more sophisticated research designs is beginning to reveal novel information about the neural bases of psychological processes. Examples have already been given in the areas of memory (see p. 89) and altered states of consciousness (see p. 171). Substantial advances in the future are likely to lie in combining image data with data from clinical lesion studies (currently neglected) and allowing each approach mutually to inform the other.

All these techniques described still hold considerable promise, and it may well be that the bridge will yet be established enabling us to link, precisely and accurately, the processes that we can observe in task performance with the associated cerebral events that we can observe in electrophysiological recordings and in functional imaging.

FURTHER READING

The best source for further information on EEG methodology, although now rather outmoded, is:

Cooper, R., Ossleton J. W., & Shaw, J. C. *EEG Technology* (Third edition, London: Butterworth, 1980).

And for EP techniques:

Callaway, E., Tueting, P., & Koslow, S. H. (Eds.). *Event-Related Brain Potentials in Man* (New York: Academic Press, 1978).

Desmedt, J. E. (Ed.). *Progress in Clinical Neurophysiology*, Vols. 1 and 3 (Basel, Switzerland: S. Karger, 1977).

Luck, S. J. *An Introduction to the Event-Related Potential Technique* (Cambridge, MA: MIT Press, 2005).

A useful introduction to recent findings can be found in the preceding volumes, as well as in:

Kertesz, A. (Ed.). *Localization and Neuroimaging in Neuropsychology* (San Diego, CA: Academic Press, 1994).

More specific reviews of the literature include:

Beaumont, J. G. The EEG and Task Performance: A Tutorial Review, in A. W. K. Gaillard & W. Ritter, eds., *Tutorials in ERP Research-Endogenous Components* (Amsterdam: North-Holland, 1982).

Donchin, E., McCarthy, G., & Kutas, M. Electronencephalographic Investigations of Hemispheric Specialization, in J. E. Desmedt, ed., *Progress in Clinical*

- Neurophysiology, Vol. 3: Language and Hemispheric Specialization in Man: Cerebral Event-related Potentials* (Basel, Switzerland: Karger, 1977).
- Hillyard, S. A., & Woods, D. L. Electrophysiological Analysis of Human Brain Function, in M. S. Gazzaniga, ed., *Handbook of Behavioral Neurobiology, Vol. 2: Neuropsychology* (New York: Plenum Press, 1979).
- Jennings, J. R., & Coles, G. H. (Eds.). *Handbook of Cognitive Psychophysiology* (Chichester, UK: Wiley, 1991).
- Marsh, G. R. Asymmetry of Electrophysiological Phenomena and Its Relation to Behaviour in Humans, in M. Kinsbourne, ed., *Asymmetrical Function of the Brain* (Cambridge, UK: Cambridge University Press, 1978).
- Rugg, M. D. The Relationship between Evoked Responses and Lateral Asymmetries of Processing, in A. W. K. Gaillard & W. Ritter, eds., *Tutorials in ERP Research: Endogenous Components* (Amsterdam: North-Holland, 1982).

There is a shortage of good general introductions to imaging techniques. This is partly because technical developments occur so rapidly that any publication would be too rapidly outdated. What sources there are tend to be highly technical. However, the interested student may find these references useful:

- Gillard, J. H., Waldman, A. D., & Barker, P. B. (Eds.). *Clinical MR Imaging: Diffusion, Perfusion and Spectroscopy* (New York: Cambridge University Press, 2005).
- Hillary, F. G., & DeLuca, J. (Eds.). *Functional Neuroimaging in Clinical Populations* (New York: Guilford Press, 2007).
- Papanicolaou, A. C., Rogers, R. L., & Baumann, S. B. Applications of Magnetoencephalography to the Study of Cognition, *Annals of the New York Academy of Sciences*, 620 (1991), 118–127.
- Perani, D., & Cappa, S. F. Neuroimaging Methods in Neuropsychology, in G. Denes & L. Pizzamiglio, eds., *Handbook of Clinical and Experimental Neuropsychology* (Hove, UK: Psychology Press, 1999).

REFERENCES

- Aaron, W., & Tobias, E. Memory Structures for Encoding and Retrieving a Piece of Music: An ERP Investigation, *Cognitive Brain Research*, 22 (2004), 36–44.
- Amochaev, A., & Salamy, A. Stability of EEG Laterality Effects, *Psychophysiology*, 6 (1979), 242–246.
- Barrett, G., Halliday, A. M., & Halliday, E. Asymmetries of the Late Evoked Potential Components and P300 Associated with Handedness and Side of Stimulus Delivery in a Somatosensory Detection Task, *Electroencephalography and Clinical Neurophysiology*, 44 (1978), 791.
- Beaumont, J. G. The EEG and Task Performance: A Tutorial Review, in A. W. K. Gaillard & W. Ritter, eds., *Tutorials in ERP Research: Endogenous Components* (Amsterdam: North-Holland, 1982).

- Beaumont, J. G., & Mayes, A. R. Do Task and Sex Differences Influence the Visual Evoked Potential?, *Psychophysiology*, 14 (1977) 545–550.
- Beaumont, J. G., & Rugg, M. D. The Specificity of Intrahemispheric EEG Alpha Coherence Asymmetry Related to Psychological Task, *Biological Psychology*, 9 (1979), 237–248.
- Bell, M. A., & Fox, N. A. Cognition and Affective Style: Individual Differences in Brain Electrical Activity during Spatial and Verbal Tasks, *Brain and Cognition*, 53 (2003), 441–451.
- Boronat, C. B., Buxbaum, L. J., Coslett, H. B., Tang, K., Saffran, E. M., Kimberg, D. Y., et al. Distinctions between Manipulation and Function Knowledge of Objects: Evidence from Functional Magnetic Resonance Imaging, *Cognitive Brain Research*, 23 (2005), 361–373.
- Brown, W. S., Marsh, J. T., & Smith, J. C. Principal Component Analysis of ERP Differences Related to the Meaning of an Ambiguous Word, *Electroencephalography and Clinical Neurophysiology*, 46 (1979), 709–714.
- Buchsbaum, M., & Drago, D. Hemispheric Asymmetry and the Effects of Attention on Visual Evoked Potentials, in J. E. Desmedt, ed., *Progress in Clinical Neurophysiology, Vol. 3: Language and Hemispheric Specialization in Man: Cerebral Event-Related Potentials* (Basel, Switzerland: S. Karger, 1977).
- Callaway, E., Tueting, P., & Koslow, S. H. (Eds.). *Event-related Brain Potentials in Man* (New York: Academic Press, 1978).
- Davidson, R. J., Taylor, N., Saron, C. D., & Snyder, M. Individual Differences and Task Effects in EEG Measures of Hemispheric Activation. II: Effects of Familial Sinistrality, *Psychophysiology*, 17 (1980), 312.
- Davis, A. E., & Wada, J. A. Speech Dominance and Handedness in the Normal Human, *Brain and Language*, 5 (1978), 42–55.
- Desmedt, J. E. (Ed.). *Progress in Clinical Neurophysiology, Vol. 1: Attention, Voluntary Contraction and Event-Related Cerebral Potentials* (Basel, Switzerland: S. Karger, 1977).
- Dolce, G., & Decker, H. Application of Multivariate Statistical Methods in Analysis of Spectral Values of the EEG, in G. Dolce & H. Künkel, eds., *CEAN: Computerised EEG Analysis* (Stuttgart, Germany: Fischer Verlag, 1975).
- Dolce, G., & Waldeier, H. Spectral and Multivariate Analysis of EEG Changes during Mental Activity in Man, *Electroencephalography and Clinical Neurophysiology*, 36 (1974), 577–584.
- Donchin, E., Kutas, M., & McCarthy, G. Electrocortical Indices of Hemispheric Utilisation, in S. Harnad, R. W. Doty, L. Goldstein, J. Jaynes, & G. Krauthamer, eds., *Lateralization in the Nervous System* (New York: Academic Press, 1977).
- Ehrlichman, H., & Wiener, M. S. Consistency of Task-related EEG Asymmetries, *Psychophysiology*, 16 (1979), 247–252.
- Ehrlichman, H., & Wiener, M. S. EEG Asymmetry during Covert Mental Activity, *Psychophysiology*, 17 (1980), 228–235.
- Friedman, D., Simson, R., Ritter, W., & Rapin, I. The Late Positive Component (P300) and Information Processing in Sentences, *Electroencephalography and Clinical Neurophysiology*, 38 (1975), 255–262.

- Galaburda, A. M., LeMay, M., Kemper, T. L., & Geschwind, N. Right-Left Asymmetries in the Brain, *Science*, 199 (1978), 852–856.
- Galin, D., & Ellis, R. R. Asymmetry in Evoked Potentials as an Index of Lateralised Cognitive Processes: Relation to EEG Alpha Asymmetry, *Neuropsychologia*, 13 (1975), 45–50.
- Gevins, A. S. The Use of Brain Electrical Potentials (BEP) to Study Localisation of Human Brain Function, *International Journal of Neuroscience*, 13 (1981), 27–41.
- Gevins, A. S., Zeitlin, G. M., Doyle, J. C., Schafner, R. E., & Callaway, E. EEG Patterns during “Cognitive” Tasks. II: Analysis of Controlled Tasks, *Electroencephalography and Clinical Neurophysiology*, 47 (1979), 704–710.
- Gevins, A. S., Zeitlin, G. M., Yingling, C. D., Doyle, J. C., Dedon, M. F., Schafner, R. E., et al. EEG Patterns during “Cognitive” Tasks. I: Methodology and Analysis of Complex Behaviors, *Electroencephalography and Clinical Neurophysiology*, 47 (1979), 693–703.
- Gotlib, I. H., Ranganath, C., & Rosenfeld, J. P. Frontal EEG Alpha Asymmetry, Depression and Cognitive Functioning, *Cognition and Emotion*, 12 (1998), 449–478.
- Gutchess, A. H., & Park, D. C. fMRI Environment Can Impair Memory Performance in Young and Elderly Adults, *Brain Research*, 1099 (2006), 133–140.
- Harris, P. G., & Silberstein, R. B. Steady-State Visually Evoked Potential (SSVEP) Responses Correlate with Musically Trained Participants’ Encoding and Retention Phases of Musical Working Memory Task Performance, *Australian Journal of Psychology*, 51 (1999), 140–146.
- Haynes, W. O. Task Effect and EEG Alpha Asymmetry: An Analysis of Linguistic Processing in Two Response Modes, *Cortex*, 16 (1980), 95–102.
- Hillyard, S. A., & Woods, D. L. Electrophysiological Analysis of Human Brain Function, in M. S. Gazzaniga, ed., *Handbook of Behavioral Neurobiology*, Vol. 2: *Neuropsychology* (New York: Plenum Press, 1979).
- Kaehkönen, S., Rossi, E. M., & Yamashita, H. Alcohol Impairs Auditory Processing of Frequency Changes and Novel Sounds: A Combined MEG and EEG Study, *Psychopharmacology*, 177 (2005), 1–7.
- Kertesz, A. (Ed.). *Localization and Neuroimaging in Neuropsychology* (San Diego, CA: Academic Press, 1994).
- Kutas, M., & Donchin, E. The Effects of Subject Strategies on the Lateralisation of Movement Related Potentials, *Electroencephalography and Clinical Neurophysiology*, 45 (1978), 29P.
- Ledlow, A., Swanson, J. M., & Kinsbourne, M. Reaction Times and Evoked Potentials as Indicators of Hemispheric Differences for Laterally Presented Name and Physical Matches, *Journal of Experimental Psychology: Human Perception and Performance*, 4 (1978), 440–454.
- Leh, S. E., Johansen, B. H., & Ptito, A. Unconscious Vision: New Insights into the Neuronal Correlate of Blindsight Using Diffusion Tractography, *Brain*, 129 (2006), 1822–1832.
- Maguire, E. A., Frackowiak, R. S. J., & Frith, C. D. Recalling Routes Around London: Activation of the Right Hippocampus in Taxi Drivers, *Journal of Neuroscience*, 17 (1997), 7103–7110.

- Marsh, G. R. Asymmetry of Electrophysiological Phenomena and Its Relation to Behavior in Humans, in M. Kinsbourne, ed., *Asymmetrical Function of the Brain* (Cambridge, UK: Cambridge University Press, 1978).
- Mayes, A. R., & Beaumont, J. G. Does Visual Evoked Potential Asymmetry Index Cognitive Activity?, *Neuropsychologia*, 15 (1977), 249–256.
- McCallum, W. S., & Curry, S. H. Hemisphere Differences in Event Related Potentials and CNV's Associated with Monaural Stimuli and Lateralized Motor Responses, in D. Lehmann & E. Callaway, eds., *Human Evoked Potentials: Applications and Problems* (New York: Plenum Press, 1979).
- McCarthy, G., & Donchin, E. Brain Potentials Associated with Structural and Functional Visual Matching, *Neuropsychologia*, 16 (1978), 571–585.
- Moore, W. H., Jr. Alpha Hemispheric Asymmetry of Males and Females on Verbal and Nonverbal Tasks: Some Preliminary Results, *Cortex*, 15 (1979), 321–326.
- Moore, W. H., Jr., & Haynes, W. O. A Study of Hemispheric Asymmetry for Verbal and Nonverbal Stimuli in Males and Females, *Brain and Language*, 9 (1980), 338–349.
- Mori, S., & van Zijl, P. MR Tractography using Diffusion Tensor MR Imaging, in J. H. Gillard, A. D. Waldman, & P. B. Barker, eds., *Clinical MR Imaging: Diffusion, Perfusion, and Spectroscopy* (New York: Cambridge University Press, 2005).
- Näätänen, R., & Michie, P. T. Early Selective-Attention Effects on the Evoked Potential: A Critical Review and Reinterpretation, *Biological Psychology*, 8 (1979), 81–136.
- Ornstein, R., Johnstone, J., Herron, J., & Swencionis, C. Differential Right Hemisphere Engagement in Visuospatial Tasks, *Neuropsychologia*, 18 (1980), 49–64.
- Peigneux, P., Van der Linden, M., Garraux, G., Laureys, S., Degueldre, C., Aerts, J., et al. Imaging a Cognitive Model of Apraxia: The Neural Substrate of Gesture-Specific Cognitive Processes, *Human Brain Mapping*, 21 (2004), 119–142.
- Perani, D., & Cappa, S. F. Neuroimaging Methods in Neuropsychology, in G. Denes & L. Pizzamiglio, eds., *Handbook of Clinical and Experimental Neuropsychology* (Hove, UK: Psychology Press, 1999).
- Preissl, H. *Magnetoencephalography* (London: Academic Press, 2005).
- Rebert, C. S. Functional Cerebral Asymmetry and Performance. I: Reaction Time to Words and Dot Patterns as a Function of Electroencephalographic Alpha Asymmetry; II: Individual Differences in Reaction Time to Word and Pattern Stimuli Triggered by Asymmetric Alpha Bursts, *Behavioral Neuropsychiatry*, 8 (1977), 90–98, 99–103.
- Rebert, C. S., & Low, D. W. Differential Hemispheric Activation during Complex Visuomotor Performance, *Electroencephalography and Clinical Neurophysiology*, 44 (1978), 724–734.
- Rebert, C. S., & Mahoney, R. A. Functional Cerebral Asymmetry and Performance. III: Reaction Time as a Function of Task, Hand, Sex, And EEG Asymmetry, *Psychophysiology*, 15 (1978), 9–16.
- Rugg, M. D. The Relationship between Evoked Responses and Lateral Asymme-

- tries of Processing, in A. W. K. Gaillard & W. Ritter, eds., *Tutorials in ERP Research: Endogenous Components* (Amsterdam: North Holland, 1982a).
- Rugg, M. D. Electrophysiological Studies of Divided Visual Field Stimulation, in J. G. Beaumont, ed., *Divided Visual Field Studies of Cerebral Organisation* (London: Academic Press, 1982b).
- Rugg, M. D., & Beaumont, J. G. Interhemispheric Asymmetries in the Visual Evoked Response: Effects of Stimulus Lateralisation and Task, *Biological Psychology*, 6 (1978), 283–292.
- Saron, C. D., Foxe, J. J., Schroeder, C. E., & Vaughan, H. G., Jr. Complexities of Interhemispheric Communication in Sensorimotor Tasks Revealed by High-Density Event-Related Potential Mapping, in K. Hugdahl & R. J. Davidson, eds., *The Asymmetrical Brain* (Cambridge, MA: MIT Press, 2003).
- Shaw, J. C. An Introduction to the Coherence Function and Its Use in Signal Analysis, *Journal of Medical Engineering and Technology*, 5 (1981), 279–288.
- Shucard, D. W., & Shucard, J. L. Auditory Evoked Potentials as Probes of Lateralized Information Processing in Adults and Infants, in D. Lehmann & E. Callaway, eds., *Human Evoked Potentials: Applications and Problems* (New York: Plenum Press, 1979).
- Swick, D., Kutas, M., & Neville, H. J. Localizing the Neural Generators of Event-Related Brain Potentials, in A. Kertesz, ed., *Localization and Neuroimaging in Neuropsychology* (San Diego, CA: Academic Press, 1994).
- Tanguay, P. E., Taub, J. M., Doubleday, C., & Clarkson, D. An Interhemispheric Comparison of Auditory Evoked Responses to Consonant-vowel Stimuli, *Neuropsychologia*, 15 (1977), 123–131.
- Taub, J. M., Tanguay, P. E., Doubleday, C. N., Clarkson, D., & Remington, R. Hemisphere and Ear Asymmetries in the Auditory Evoked Response to Musical Chord Stimuli, *Physiological Psychology*, 4 (1976), 11–17.
- Thatcher, R. W., & John, E. R. *Foundations of Cognitive Processes* (Hillsdale, NJ: Erlbaum, 1977).
- Tucker, D. M. Sex Differences in Hemispheric Specialisation for Synthetic Visuospatial Functions, *Neuropsychologia*, 14 (1976), 447–454.
- Weiss, S., & Mueller, H. M. The Contribution of EEG Coherence to the Investigation of Language, *Brain and Language*, 85 (2003), 325–343.
- Wood, C. C. Average Evoked Potentials and Phonetic Processing in Speech Perception, in J. E. Desmedt, ed., *Progress in Clinical Neurophysiology, Vol. 3: Language and Hemispheric Specialization in Man: Cerebral Event-Related Potentials* (Basel, Switzerland: S. Karger, 1977).

Individual Differences

Gender and Handedness

Happily, we are not all alike, and one of the major historical themes of experimental psychology has been the investigation of just how and why we differ. This interest has inevitably been reflected in human neuropsychology.

There is a whole range of variables upon which individuals differ and which may be relevant to neuropsychological function. They include sensory function and perceptual abilities; metabolic and endocrine factors; motor skills and manipulative ability; intellectual performance and cognitive style. Even emotional traits, personality differences, and anxiety bear some relation to cerebral function. However, two particular characteristics of the individual subject have come under extensive scrutiny, and they are gender and handedness or sinistrality.

GENDER DIFFERENCES IN CEREBRAL ORGANIZATION

The evidence in general psychology about gender differences in cognitive ability has pointed to a superiority among males for spatial and mechanical skills, and a superiority among females for verbal skills (Hutt, 1972; Maccoby & Jacklin, 1974). While this dichotomy has not been accepted uncritically, it is generally recognized that there are some differences between the genders along these lines. Whether such differences can be attributed to biological factors, or result from social and cultural influences, is still hotly debated. It seems reasonable that both are involved, although the extent of the contribution of each is unclear.

It was almost inevitable that these ideas would be carried across into neuropsychology, particularly considering the battle to explain the very considerable degree of variability shown by subjects in performing experimental tasks. The kind of dichotomy proposed for gender differences also appeared to reflect, appealingly, contemporary ideas about the dichotomy between the specializations of the two hemispheres.

The work of Buffery and Gray (1972) gave a significant impetus to research by suggesting a model in which males were considered to possess less cerebral lateralization than females. The male brain was seen to have language represented more bilaterally, which in turn implied that spatial abilities must also be represented more bilaterally to occupy the remaining capacity. The result was, for males, a relative deficit in verbal ability, which suffers as a result of bilateral representation, and a relative advantage in spatial ability, which might benefit from ambilaterality.

Subsequent research and theorizing have almost unanimously failed to support the Buffery and Gray hypothesis. Nevertheless, it illustrates the kind of argument that has led to current theories. These, when they have accepted the presence of gender differences, have proposed that the female brain is more bilaterally organized than that of males. The current debate is whether the evidence justifies such a conclusion. Before considering the evidence, some methodological issues must be raised.

There is a particular difficulty about the manner in which much of the gender difference data have been collected. Particularly because of the general feeling that gender differences might be important, an experimenter will typically include equal numbers of males and females in the participant sample, and will probably include the factor of gender in the statistical analysis of the results. If there is a significant effect of gender, it will be reported; if there is no significant effect, it may well not be reported at all. Reference may not even be made to gender in the report of the analysis and discussion of the results. Even if such a bias were not present in the reporting of results, the incidental way in which information has accumulated, from studies not specifically designed to investigate gender differences, must be regarded as unsatisfactory.

The question as to whether there are differences between the genders in the effects of clinical lesions raises another problem, which is the preponderance of males in clinical samples. Where comparable groups of male and female brain-injured patients are studied, there are almost inevitably differences between the two gender groups in terms of the cause of the lesion, the age of the patient, the severity of the lesion, and so on (see p. 47). McGlone (1980), from the evidence of her 1978 study, and from her review of similar studies, reports that while males show effects of the laterality of the lesion on the Verbal-Performance IQ discrepancy (left lesions affecting Verbal, and right lesions Performance, IQ

on the Wechsler Adult Intelligence Scale, or WAIS), these effects are not to be found in females. It is also claimed that the aphasic effects of left hemisphere lesions are less severe in females, who also recover more rapidly, and this hypothesis has been more recently supported by Frith and Vargha-Khadem (2001) with respect to literacy skills. The commentaries that accompany McGlone's 1980 review together with the additional data presented there, and the data set out in Fairweather (1982), show that while some studies support the conclusion of female bilaterality on the basis of clinical evidence, there are also a number that do not.

Experimental neuropsychology has yielded a very confused set of results on gender differences. To illustrate this confusion, in the early studies using the divided visual field technique and verbal stimuli, some researchers found the predicted greater right visual field advantage among male subjects (Bradshaw & Gates, 1978; Kail & Siegel, 1978). However, there are also well-conducted studies in which no evidence of a gender-related effect was apparent, for example, that of Hannay and Boyer (1978). Other researchers found differences, and then failed to replicate them (McKeever & Van Deventer, 1977). A similar picture emerged from the studies employing nonverbal stimuli, with both positive (Davidoff, 1977; Rizzolatti & Buchtel, 1977; Sasanuma & Kobayashi, 1978) and negative (Bryden, 1976; Kail & Siegel, 1978) findings.

Merely tallying up the number of studies that find a certain result, irrespective of their methodological adequacy, may be a dangerous procedure, but it is interesting that Fairweather (1982) calculated that among 49 studies with verbal stimuli, 42 found no gender effect, 5 found more lateralization among males, and 2 more lateralization among females. With nonverbal stimuli, of 62 studies, 13 showed greater lateralization for males and 4 for females. It is particularly striking that of the 111 studies reporting on gender differences, 87 found no effect of that variable. Fairweather concluded that there was no evidence for gender differences on verbal divided visual field tasks, and slender evidence for it on nonverbal tasks. The only hint of a consistent finding was in facial recognition, stemming from the work of Italian laboratories (Umilta, Brizzolara, Tabossi, & Fairweather, 1978). McGlone's (1980) review of the same material was much more selective, and reached the conclusion that the most parsimonious explanation for the reported findings was that there is less functional brain asymmetry in females.

Matters are complicated by the presence of differences in sensory and perceptual performance between the genders, and by the suggestion that there may be differences between males and females in cognitive strategy. The idea here is that females prefer to adopt "verbal strategies," and males "nonverbal" ones (Bryden, 1978). Incidentally, Metzger

and Antes in 1976 showed that instructing subjects explicitly to adopt a certain strategy had a general effect on performance, but it did not affect the basic pattern of cerebral asymmetry inferred to underlie task performance. However, the concept of differential strategy utilization is still often discussed as either a cause or an effect of fundamental differences in cerebral lateralization. Strategy adoption, as a gender-linked factor, clearly could have the observed effects on task performance (and criticisms of the mechanism have often confused within-individual effects with the effects that might be seen when the results of a group of subjects are collated). However, the operation of this mechanism has yet to be experimentally demonstrated, rather than proposed as a post hoc explanation.

Fortunately, there have been a series of more recent meta-analyses that have helpfully reviewed the accumulating evidence. Zaidel, Aboitiz, Clarke, Kasier, and Matteson (1995) reported an analysis of 31 divided visual field experiments, and Voyer (1996) also reported on an extensive series of studies, finding results that essentially supported McGlone's (1980) view. However, a considerable service has been provided by Hiscock and colleagues who, between 1994 and 2001, published four major papers reviewing the literature on, in turn, dichotic listening, divided visual field studies, tactile asymmetries, and dual task experiments (Hiscock, Inch, Jacek, Hiscock-Kalil, & Kalil, 1994; Hiscock, Israelian, Inch, Jacek, & Hiscock-Kalil, 1995; Hiscock, Inch, Hawryluk, Lyon, & Perachio, 1999; Hiscock, Perachio, & Inch, 2001). The first two papers reviewed 140 and 217 studies respectively, in which gender differences were analyzed, and the studies were further classified in terms of the stringency of their design and methodological controls. For dichotic listening, 9 of the 11 "stringent" studies found increased hemisphere specialization in males by comparison with females, although only 21 of the 140 found a weaker but consistent effect. A similar pattern was found for divided visual field studies: 17 of 23 studies meeting the stringent criteria demonstrated the increased specialization for males, but of the less stringent only 27 studies found a consistent parallel effect, and 6 studies reported contrary findings. The similarity of overall findings in these two meta-analyses suggests that this is a real effect, but the conclusion can be no more than that there is a weak population-level difference between the genders in the degree of lateralization, with greater lateralization seen in males. This is certainly not of a magnitude from which predictions might be made about individual cases. The overall contribution of gender differences to the variance of lateralization is no more than 1 to 2%, and this cannot be regarded as of real significance.

The two later meta-analyses of tactile and dual-task studies were

performed with smaller numbers of studies (30 and 51 respectively) and the number demonstrating unambiguous gender differences was small. No clear conclusions could be drawn from either of these studies alone, but the findings do not contradict the earlier, larger studies and so lend some support to the hypothesis of increased bilateralization of functions in females.

Two more recent studies are worth a mention. Sappington and Topolski (2005) analyzed SAT scores for mathematics in a very large number of American students. They found that being male and non-right-handed and having an earlier onset of puberty were all associated with higher math scores, while being female, being non-right-handed, and having a later onset of puberty were linked to very poor scores. On the assumption that greater lateralization confers an advantage for formal intellectual tasks, such as mathematics, this finding is consistent with the general view now being adopted. However, employing fMRI, Clements et al. (2006) found increased left lateralization for males on a phonological task with more bilateral activity on a visuospatial task (as might be expected), but for females bilateralization on the phonological task (also expected) with increased right lateralization on the visuospatial task.

An intriguing sideline to the investigation of gender differences has been the study of sexual orientation and laterality. Again, there is a fairly recent meta-analysis to assist us (Lalumière, Blanchard, & Zucker, 2000). Analysis of 20 studies revealed that individuals with a homosexual orientation had 39% greater odds of being non-right-handed and, by inference, having a less lateralized cerebral organization. The effect was much greater for women, where the increase in odds was 91%, while for men it was only 34%. It is reasonable to suspect that the basis for this effect lies in early neurodevelopment, through prenatal exposure to sex hormones, to a maternal immunological reaction, or simply in developmental instability. However, the precise mechanism that creates this effect remains unclear.

What view are we therefore to take? Is it that there really are gender differences in brain organization, but that they are subtle and often masked by other sources of variability in task performance? The within-gender variability is certainly large in comparison with the mean between-gender difference, and the more substantial effects of individual differences in memory function may also swamp any other effects that might be present. That so few studies are designed with the intent of studying gender differences may also explain why so few find these effects on subsequent analysis. We may adopt the position of McGlone and Hiscock and colleagues and believe that the effects are elusive, although real, and that when they can be demonstrated they show that

females have a less lateralized brain. Alternatively, we may follow Fairweather and consider that the evidence is too thin and inconsistent, or the contribution of the effect too small, to allow us to include gender in any theory of cerebral laterality (or laterality in any theory of gender differences). My personal inclination, formerly for the latter position, is to accept that the differences are real but that they are not of any (real-world) significance. Students should read the literature and arrive at their own opinion. However, significant gender differences have not to date been shown convincingly as present either in the effects of clinical lesions or in the neuropsychological performance of normal subjects. We have to await the accumulation of further evidence to prove this view too cautious.

HANDEDNESS

Handedness is one of the most obvious ways in which the performance of some people differs from that of the majority. Although the precise figure depends on how left-handedness is defined, most surveys find that between about 8% and 12% of the population is left-handed. Although there is some geographical variability (Raymond & Pontier, 2004), this figure seems typical of most contemporary cultures and even traditional societies (Faurie, Schiefenhövel, Le Bomin, Billiard & Raymond, 2005). The Eipo, who live in the forests of Irian Jaya, an area of Papua New Guinea, wear a plug in their earlobe on the opposite side to the hand they use for shooting with a bow; with this knowledge it has been possible to establish handedness rates from photographs where the ears are visible. The rate also seems to have been invariant through recorded history. There is even evidence that in prehistoric humans roughly 10% were sinistral, or left-handed.

The study of handedness is a major topic in its own right, with sociological, cultural, and even religious aspects. There have been many theories about the origins of left-handedness, but ever since asymmetries in the nervous system were first recognized, the major theories have all linked handedness in some way with cerebral laterality (see Herron, 1980; Coren, 1990).

Against this background, the contribution of genetic factors to the determination of handedness has never been denied. If you have one left-handed parent, you are 2–3 times more likely to be left-handed than if both your parents are right-handed; if both your parents are left-handed, the probability is 3–4 times (Bryden, Roy, McManus, & Bulman-Fleming, 1997). Left-handed children are therefore more commonly, although not exclusively, the children of left-handed parents, and it can

be shown that this is not merely due to cultural transmission. In fact, it has been found that left-handed parents may more strongly discourage sinistrality in their children because of their awareness of its disadvantages. In recent years there has been a debate between two opposing theories: that of Annett (2002) and that of Levy and Nagylaki (1972). Much of this debate is relatively technical, and the Levy–Nagylaki model is well specified. However, although a more specific model, it seems to account less well for all the available data than Annett’s model. The latter is known as the “right shift” theory, because it involves the inheritance of a single factor that shifts handedness, which would otherwise be determined by chance, toward right preference. Of individuals who do not inherit the factor, half will be of each handedness by random chance variation; among those who do inherit the factor, most, although not all, will be right-handed. One of the simplest predictions of this model is that of the children of two left-handed parents (who in most cases cannot inherit the right shift factor), almost half will be left-handed. This, and more complex, predictions seem to be borne out fairly well by the data. Nevertheless, there continue to be some who argue that this model is too simple (McManus & Bryden, 1993; Ecuyer-Dab, Tremblay, Joannette, & Passini, 2005), and that handedness may be an X-linked characteristic (McKeever, 2000) which would be incompatible with Annett’s theory.

Much of the research on handedness assumes that the measurement of handedness is a simple matter. It is not. There is no true dichotomy between right and left handers. Although the majority of people perform all skilled actions preferentially with the right hand, the remainder of the population forms a continuum out to “pure” left-handedness. Much of the discussion about handedness fails to take this adequately into account. It is, however, recognized in the questionnaires generally employed to assess handedness (Annett, 1970; Bryden, 1977; McFarland & Anderson, 1980).

As a final general point, it should be remembered that handedness occurs in a general setting of biological asymmetries. Although the link with some of these may not appear very direct, for example the hair-whorl on the crown of the head, fingerprints, nostril size, and the arrangement of the male testicles, the association with others, such as sighting dominance (which eye you use for a telescope) or footedness (with which foot you step out, or kick a ball), may be more clear. However, while all these phenomena show a statistical relationship with handedness due to an underlying general biological factor, none shows a very direct relation. For example, ocular dominance has less connection with handedness than is often assumed. The association, if any, is a very complex one (Birkett, 1977; Porac & Coren, 1976).

Handedness and Cerebral Dominance

It is now unfashionable to speak of “cerebral dominance” as a general factor determining laterality, although contemporary theories continue to use the idea quite freely under a number of guises. They may speak of “dominance for speech” or “dominance for phoneme discrimination” or whatever specific function is being considered, but in fact all theories involving handedness imply a general factor determining the overall pattern of laterality. There are four ways in which this factor might be linked with handedness:

Pathological Left-Handedness

This idea has a very long history and is simply that stress or trauma in early life, especially around birth, may result in left-handedness. The observation of an unusually high rate of left-handedness among many groups of abnormal children and adults, especially those with some dysfunction of the central nervous system, has always been used as an argument to support this idea. If we accept that intelligence is normally distributed in the population (and psychologists arrange that measured IQ follows this theoretical statistical distribution), there are more left-handed people at the lower end of the range than would be expected by statistical variation. This can be explained by the depression of intelligence as a result of accident or pathology. In the same way, handedness is thought to be affected by early damage or injury. This argument reveals the cultural bias that has often linked sinistrality (“sinister”) to weakness and even evil, and we might well question whether the depression of intelligence can be considered a parallel to a shift from right- to left-handedness. However, pathology is still an important factor in some explanations of left-handedness, and there is no doubt that severe abnormal states can be associated with disorders of the nervous system and may result in a variety of atypical patterns of handedness. There is much more doubt whether less severe birth stress and trauma, which do not produce any overt abnormality, can be considered to lead to a shift in hand preference. The evidence about this is discussed below.

The Contralateral Rule

This concept is much more simply stated and underlies many popular accounts and discussions of handedness. The concept is that speech dominance is always located in the hemisphere contralateral to the preferred hand. Stated another way, if almost all right handers have left hemisphere speech dominance, then left handers must have right speech dominance. Curiously, although it has never received much support

from scientific research, the idea often crops up in discussions by anthropologists, palaeontologists, and biologists of the development of language, tool use, and manual skill, when speech lateralization in the evolution of language is linked to asymmetries of skilled performance. To be fair, it is not necessarily implied that the relation between speech lateralization and skilled manual performance must also hold for left handers, but such a relation is often assumed, so that the mirror image nature of left preferent performance is extended to a mirror image model of the nervous system.

Bilateralization

This is probably the most popular position adopted, but is also the most vague. It states that left handers, including “mixed” handers either implicitly or explicitly, have a lesser degree of lateralization than the right-handed, just as we have seen argued for females. Occasionally the small group of “pure” left handers is excluded (and then follows the contralateral rule), but this is not generally the case. The reasons for this relative bilateralization are rarely clearly stated, and it is more common for this position to emerge from a review of the available evidence on lateralization of function and handedness.

The Right Shift Model

Annett (2002) has argued that the right shift model of genetic determination of handedness (see p. 298) predicts a pattern of cerebral asymmetry for the left-handed that is distinct from the bilateralization model. Annett very clearly demonstrates how the strictness of the criterion for what we would consider left-handedness has an important effect on the pattern of cerebral asymmetry to be expected for that group. If the criterion is strict, so that only the more extreme sinistrals are included, then very few will have the right shift factor, and just over half will have left hemisphere speech. As the criterion becomes more lax, more individuals will be included who have the right shift factor, and the proportion with left hemisphere speech will rise as a result since the right shift factor is, of course, considered not only to increase the probability of right-handedness, but, at the same time, of left hemisphere speech. The direct association between speech lateralization and hand preference is still evident, if less directly determined.

All these models make predictions about the pattern of asymmetry to be observed in clinical phenomena and in laboratory performance. The predictions to be made from the pathological model are least clear, but are generally taken to imply some reversal, perhaps with a degree of

disorganization, of observed laterality. The contralateral rule predicts reversed patterns of asymmetry, and the bilateralization model that no asymmetry will be observed. Annett's model predicts a reduced degree of the normal pattern to be found with right handers. It would seem, therefore, easy to test between these different predictions. In practice it is not. The pathological model admits results indicating a weak degree of asymmetry, especially if they are in the "reversed" direction. The bilateralization model admits all results that show any weak degree of laterality in either direction, allowing for sampling bias, errors of measurement, and so on. The right shift model is compatible, on the same grounds, with no asymmetry and even weak reversal, and with a strict criterion of sinistrality it becomes practically indistinguishable from the bilateralization model. Nevertheless, we must try to evaluate these models in the light of the available evidence.

Three final factors must be mentioned before we turn to the evidence. One is familial sinistrality, that is, whether the individual has any relatives who are left-handed. This factor is often included as an added dimension in studies of laterality, although its theoretical significance is not always clear; it should, of course, distinguish between "inherited" and "pathological" handedness, if such a distinction were to prove valid. It is sometimes linked to the second factor, the strength of handedness. This variable can be extracted from questionnaire responses or from measures of lateral dexterity or performance speed. Although Searleman, Tweedy, and Springer (1979), for example, have shown strength of handedness and familial sinistrality to be unrelated, it is still often assumed that left handers with sinistral relatives are purely or extremely left-handed, and hence right handers with sinistral relatives must be less clearly right-handed than those without. It is expected that these variables will be found in the laterality observed on experimental tasks.

The third and last factor is that there is a potential interaction between gender and handedness when considering bilateralization. As studies of gender in handedness almost unanimously show a greater frequency of left-handedness among males (see Annett, 2002), a more bilateral brain might be associated with both male gender and left-handedness. Or is hand preference a confounding factor in studies of gender, and vice versa? These are questions without, as yet, clear answers in the scientific literature.

The Evidence about Left-Handed Brains

The first question to be answered is whether the concept of pathological left-handedness receives any significant support. Bakan (1977) has been

one of the foremost proponents of this idea, and has defended it against a number of criticisms. He has also shown that there is an excess of left handers among first and late (fourth or later) positions in birth order, which he claims links sinistrality with birth stress. He goes further and specifically proposes that the left-handedness results from damage to the left hemisphere pyramidal motor pathways following perinatal anoxia. Nevertheless, other investigators have failed to support Bakan's findings. For example, Hicks, Elliott, Garbesi, and Martin (1979) constructed a study that they considered would give the greatest chance of replicating the Bakan results. They found, however, that the relationship between handedness and a combination of factors known to be associated with high-risk birth was "trivial." Annett and Ockwell (1980) found only the weakest relationship for daughters but none for sons, while Leiber and Axelrod (1981) found the slightest of links for sons but none for daughters. The current situation is that the gravest doubt is cast upon the validity of a contribution of birth stress to the determination of handedness.

Turning to the clinical evidence, two methods have already been introduced (see p. 86). The Wada technique by which sodium amytal is introduced into the carotid artery, so interrupting function in the hemisphere on the same side as the injection for a brief period, has given us some data on the speech lateralization of left handers. Rasmussen and Milner's 1975 data, which have already appeared in Table 7.1, show that of the left handers studied, about 70% had left-hemisphere speech, 15% right hemisphere speech, and 15% had speech bilaterally represented. Isaacs, Barr, Nelson, and Devinsky (2006) have recently reported similar findings; 9% of strong right handers had atypical language lateralization, while in the ambidextrous it was 46%, and 69% in strong left handers. Further support comes from a recent fMRI study (Szaflarski et al., 2002).

The second method mentioned in Chapter 7 was the study of the frequency and severity of aphasia following unilateral lesions of the left or right hemisphere. This is a complicated issue when studied in detail, but Satz's analysis of the studies reported between 1935 and 1973 is probably the clearest summary for our purposes (but also see Coren, 1990). In its most recent form (Carter, Hohenegger, & Satz, 1980), it suggests a model in which 24% of left handers have left-sided speech, none right-sided speech, and 76% bilateral speech.

A third, although less important, method might also be mentioned: the study of unilateral ECT (Warrington & Pratt, 1973, 1981). ECT (electroconvulsive therapy) for depression is now often administered unilaterally to avoid some of the undesired aftereffects of bilateral treatment, which may include interference with verbal memory. Such inter-

ference is minimized if the shock is given only on the side that is not dominant for speech. The studies that established this aspect of the unilateral method, and the early treatments that may be given to either side in order to establish speech laterality, also yield information that is of relevance for our question. What the studies suggest is that left-sided speech is to be found in about 70% of left handers, in line with the Wada technique results. Incidentally, Warrington and Pratt found that neither dichotic listening indices nor the hand used for writing could be used to determine speech laterality with any confidence.

What are we to make of this clinically derived evidence? There are problems associated with each of the methods discussed. All are concerned with abnormal subjects, and the general problems of comparing the effects of clinical lesions in different groups apply to the aphasia evidence. The Wada technique is also highly stressful for the patient and imposes severe practical limitations upon testing. The inferences drawn from the study of ECT are particularly indirect, and depressed patients may well have an abnormal pattern of cerebral lateralization (see Chapter 16). Criteria for left-handedness also differ between studies. Finally, it is probable that the nature of the testing and the way that responses are scored make a definite finding of right or left lateralization more likely for the Wada and ECT methods than for the aphasia assessments. So, bearing in mind this point, we might conclude that very few, if any, left handers have right hemisphere speech, at least a third have clear left hemisphere speech, and the remainder, between a half and two-thirds, have relatively bilateral speech in which the greater contribution comes from the left hemisphere.

What about the evidence from experimental neuropsychology? The work of Geffen and colleagues on a dichotic listening test that would predict speech laterality, and their encouraging results, were discussed in Chapter 12 (see p. 255). The studies of this group have included left handers, and have shown that a left ear advantage or no advantage is more common among left handers than right handers, although handedness did not affect the relative magnitude of the asymmetry. Familial sinistrality in male left handers increased the probability of finding a right ear advantage, and therefore left hemisphere speech (Geffen & Traub, 1979, 1980).

There are a very considerable number of studies using the dichotic listening and divided visual field techniques that have included handedness as a subject variable. Rather, as with gender, it has been easy to add handedness as a variable in a study never designed to investigate this factor and the result has been equally unsatisfactory. Happily, there are some valuable reviews of this rather baffling literature: Annett (1982, 2002), Hardyck and Petrinovich (1977), Levy (1980), and Coren

(1990). Both Annett and Hardyck include a tabulation of the most prominent studies and their results. Unhappily, however, the reviews do not entirely agree in their conclusions.

Annett (1982) shows that the usual kind of divided visual field study, using verbal stimuli and obtaining a right visual field advantage for right handers, generally produces no asymmetry in left handers. Of the 15 studies quoted, only one found a left field superiority, while two found the right field to be superior. The picture was much the same for the 13 nonverbal studies cited. Most showed a left field advantage for right handers, but no asymmetry for left handers. For this group, two studies found a left, and three a right, visual field advantage. Familial sinistrality was reviewed independently in 19 studies and the variable was considered to add "nothing substantial" to the results. It was occasionally found to reduce the lateral advantage of right handers, and to be linked with a trend to reversed asymmetry in left handers, but these effects were neither strong nor clear. Annett concludes that on visual tasks, left handers show no overall asymmetry, especially if left-handedness is strictly defined.

Hardyck, by contrast, considers familial sinistrality of significance, and his review leads to a model in which the right-handed without sinistral relatives are the most strongly lateralized. Left handers with sinistral relatives are the most bilateral. Right handers with left-handed relatives and left handers without left-handed relatives are intermediate and show weak (but not reversed) lateralization.

Levy reaches yet a different conclusion. She considers that a few sinistrals have strong right hemisphere language (although this is based principally upon the clinical evidence), but that the majority show weaker left hemisphere language in comparison with right-handed subjects. (This is having discounted the small number of sinistrals whose left-handedness can be attributed to major early disruption of the left hemisphere, and whose speech functions are then controlled from the right hemisphere.)

In so far as all these studies can be seen to present a common view, they suggest that very few, if any, left handers have right hemisphere speech. When the latter is observed in individual subjects, it is more commonly in a clinical rather than in an experimental context. It may well be that the small number of cases of reversed dominance results from abnormal processes, and that in normal subjects right-hemisphere speech is a rare phenomenon. All the reports agree that the majority of left handers will show weak or relatively bilateral left hemisphere speech lateralization. (The proportion of these individuals whose tendency to left-sided speech will be sufficiently pronounced to consider them to have clear left speech dominance will depend on the criteria for domi-

nance, and will probably also be influenced by the criteria adopted for left-handedness.) There seems a clear agreement, therefore, that left-handedness is in general associated with a reduced degree of cerebral lateralization.

Having reached this conclusion it is not surprising that there are reports, as we have already noted, of a weak relationship between handedness and speech laterality as assessed by dichotic listening (Searleman, 1980); that left handers show greater variability across occasions of testing (Hines, Fennell, Bowers, & Satz, 1980); and that for left handers there is no clear correlation between the results of visual and auditory tests (Hines & Satz, 1974).

The conclusion concerning familial sinistrality is less clear. While it seems to have been agreed that the existence of sinistral relatives is likely to be associated with a shift away from strong left hemisphere speech, it has not been agreed how important is the contribution of this variable.

In terms of the four models with which we started, pathological processes appear to provide a significant explanation in only a very small number of cases, and they cannot be considered relevant to any general explanation of left-handedness. There is also no evidence to support the operation of the contralateral rule. However, the evidence is broadly in agreement with both the bilateralization and the right shift models. As we noted, it is very difficult to devise a critical test between these two models given present techniques. The clinical evidence would marginally favor the bilateralization model because of the large proportion reported to have clear left sided speech. However, it could be argued that this is not entirely incompatible, given certain methodological considerations, with the right shift model. Annett has shown that the experimental evidence can fit very well with the right shift model, and if this model is accepted to explain the inheritance of handedness, then it also seems to serve fairly well as a way of interpreting observations of cerebral laterality in different handedness groups. It certainly seems that this, or some other version of the general bilateralization model, provides a good description of how left-handed brains differ from right-handed ones.

Differences in Cerebral Mechanisms

While most researchers have been content to present the empirical evidence of performance differences between left and right handers, a few have asked how these differences might be expressed in cognitive operations. Semmes (1968) developed an influential model of hemispheric function, suggesting that organization was “focal” in the left hemisphere and “diffuse” in the right hemisphere, with consequent advantages for

verbal and spatial processes respectively. She admitted, however, that the model could not account for the cerebral organization found in many left handers.

Levy (1974) developed a model based upon a different dichotomy, between "analytic" and "global" processing. Within this model, left handers are specifically taken into account, and are considered to have bilateral representation of the functions based upon analytic processes normally located in the left hemisphere in right handers. However, while speech, language, writing, and calculation are represented in both hemispheres, there is no corresponding bilateralization of "normal" right hemisphere functions. These suffer in left handers by being crowded out by the verbal functions. Levy has expanded this model to include gender differences in cerebral organization and orientation of writing hand (see p. 308; Levy, 1980; Levy & Gur, 1980). This theory has become quite complex. Nevertheless, it still contains the idea that left-handedness generally indicates a bilateralization of certain functions, which is in turn seen in a superior level of these abilities. The functions for which no fully specialized hemisphere exists will show a deficiency in performance. Handedness is, however, only one factor among others, including birth stress, genetic factors, and fetal gender hormones, which may be linked with a depression in the level of ability.

A different model was proposed by Beaumont (1974). This was that left handers showed a general diffuseness of cerebral organization in all functions in comparison with the relative focal organization of the right-handed. This was considered to lead to an advantage on certain cognitive functions, but a deficiency on others. The more complex the integration required by the task, the greater the advantage for the left hander, irrespective of whether the task was "verbal" or "spatial." This model was supported by evidence from divided visual field studies.

These last two models have been reviewed by Hardyck (1977). He finds more support for Beaumont's than for Levy's model, but rightly criticizes the lack of an independent criterion of task complexity. His second criticism, that a more diffuse system must imply longer processing times for complex tasks, does not really follow from the model. However, he himself develops a new form of the model, which does have certain advantages over its predecessor. Hardyck's idea is that the most bilateral individuals, left handers with sinistral relatives, will employ more interhemispheric processing. This idea can be extended to generate specific predictions about certain tasks, for instance, that left handers will have an advantage for identity judgments in divided visual field performance, and will make more errors with mirror image stimuli. This model fits well with much of the data, and deserves more attention than

it has attracted. In particular, tests of some of the specific predictions would be welcome.

Are Left Handers Less Able?

An issue related to that of the cognitive mechanisms that may distinguish left and right handers is whether left handers are less proficient in certain abilities. Levy (1969) had reported that sinistrals were inferior on non-verbal or “performance” intelligence subtests, such as are found in the Wechsler Adult Intelligence Scale (WAIS), and this finding was confirmed by Miller (1971), using different tests. That left handers could have such a fundamental handicap was greeted with some skepticism, and it was argued that the populations studied were in some way unusual. In both studies they were students, who are generally selected for their verbal abilities. This might have biased the results in that the left handers examined may have been in some respects atypical of the majority of left handers in the pattern of their abilities.

The doubt cast on these studies was strengthened by the findings of three large-scale studies of the general population: the Oxfordshire Villages Survey in Britain (Newcombe & Ratcliff, 1973); a study of 7,119 schoolchildren in the United States (Roberts & Engle, 1974); and a survey of 7,688 children tested by Hardyck, Petrinovich, and Goldman (1976). These studies found no evidence that handedness was in any way related to the pattern of verbal and performance abilities. In view of the serious implications that such a handicap would have for left handers, this has been thought to be the more reasonable result.

Nevertheless, some evidence continues to appear of such differences between handedness groups. Bradshaw, Nettleton, and Taylor (1981) again found the WAIS Performance deficit among sinistral students, particularly among those with left-handed relatives. Hicks and Beveridge (1978) confirmed some of their earlier work by finding that left handers were inferior to right handers on a general factor of intelligence, and although Heim and Watts (1976) did not find differences in the level of ability on three intellectual factors, they did find a specific deficit in numerical ability. This last study was of a large sample of 2,165 nine- to twenty-year-olds. Lastly, a specific deficit in reading abilities was reported by Cohen and Freeman (1978). Left handers were found to be poorer at silent reading, although they had adequate comprehension. They also suffered more, while reading out loud, from visual distortion introduced into the text, although they were less affected by linguistic distortion. This suggests a different strategy in the left-handed for read-

ing, based more on visual analysis, which in some situations may prove less efficient.

One aspect of Annett's recent research has been to show that there may be a disadvantage associated with inheriting the right shift gene from both parents; the heterozygote (a single right shift gene) confers an advantage. The disadvantages of a more "extreme" right shift can have an impact upon spatial and mathematical reasoning, and have implications for abilities in art, music, surgery, and sport (Annett, 2002).

It is a common observation that there is an excess of left handers (above the 10 to 12% that we might expect) among the unusually gifted, among high-achieving sportsmen (Grouios, Tsorbatzoudis, Alexandris, & Barkoukis, 2000), and in certain professions. A recent study in a tradition of studies examining different occupations found an excess of left handers among architects and lawyers and a dearth among orthopedic surgeons, mathematicians, and librarians; psychiatrists tended to be ambilateral (Schachter & Ransil, 1996).

It therefore appears that while there is no good evidence for a general and serious deficit in the intellectual abilities of left handers, we cannot discount that in certain groups of sinistrals there may be a limited range of functions that are impaired in comparison with right handers; however, there are also situations where left-handedness may be an advantage. How widespread and significant such handicaps and benefits may be has not yet been satisfactorily determined.

Writing Posture

A final topic that, for a time, attracted a great deal of interest is that of writing posture. Levy and Reid (1978) reported that speech lateralization, as shown by a divided visual field task, could be predicted by the hand and posture used for writing. They divided hand postures into the "normal" and the "inverted." Although the criteria for this division have never been quite clear, in the normal position the pen points to the top of the page while writing. In the inverted posture it points toward the bottom. Inverted postures are rare in right handers, but are common among the left handed. In terms of speech lateralization, the normal posture was associated with speech control contralateral to the hand of writing, while the inverted posture indicated ipsilateral speech localization. Right handers with normal posture and left handers with inverted posture therefore must have left-sided speech, while left handers with normal posture must have right-sided speech. If this model were supported, then it would be of tremendous value in providing a simple means of assessing speech laterality. Levy and Reid's data were most encouraging.

Unhappily, as seems the normal course in this area, subsequent studies did not unanimously support the model. Two clearly supported it, and two failed to provide any support, and indirect evidence has been found to support the model in only two out of three relevant studies. Finding partial support for the model, McKeever and Hoff (1979) attempted to develop it by introducing the idea of intrahemispheric disconnection of left hemisphere visual and manual motor areas. An additional problem is that the model does not seem to work when lateralization is assessed in the auditory modality by dichotic listening, rather than in vision by a divided visual field task (Beaumont & McCarthy, 1981; McKeever & Van Deventer, 1980). This is at best an embarrassment for the model.

While there may well be something of significance about writing posture that can provide information about handedness and cerebral organization, the main findings have not as yet been clearly established. The difficulty of measuring writing posture has not helped to generate clear replicable results. What is clear is that on current evidence writing posture does not constitute a valid index of cerebral lateralization in individual subjects. Its initial exciting promise has yet to be fulfilled.

CONCLUSION

The study of individual differences in cerebral organization is an important area of research, particularly in view of the practical necessity in clinical applications of being able to establish the pattern of laterality in an individual patient. Of the two principal variables investigated, the findings with respect to the first, gender, are too inconsistent to allow for definite conclusions and the cautious reader would accept that clear gender differences have yet to be demonstrated. Nevertheless, abandoning caution a little, there seems to be some body of evidence that supports a greater degree of cerebral lateralization in females.

The second variable, handedness, shows more consistent findings in that left-handedness appears to be associated with an abnormal pattern of cerebral lateralization, with most left handers having a relative bilateralization of function. This may be mediated through a relatively diffuse functional system that requires greater interhemispheric intercommunication in the left-handed. Although there is still some suspicion that left handers may suffer distinct cognitive handicaps, large studies have generally failed to support this conclusion. Writing posture seems to reflect certain aspects of cerebral function, but cannot be used as an index of speech lateralization at the present time.

FURTHER READING

The best introduction to the topic of gender differences in cerebral organization is to be found in:

McGlone, J. Sex Differences in Human Brain Asymmetry, *Behavioral and Brain Sciences*, 3 (1980), 215–263. This includes associated commentaries, together with the series of four papers published by Hiscock and colleagues cited in the references below (Hiscock, Inch, Jacek, & Hiscock, 1994; Hiscock, Israelian, Inch, & Jacek, 1995; Hiscock, Inch, Hawryluk, Lyon, & Perachio, 1999; Hiscock, Perachio, & Inch, 2001).

On handedness, further reading might include:

Annett, M. A. *Handedness and Brain Asymmetry: The Right Shift Theory* (New York: Psychology Press, 2002).

Coren, S. (Ed.). *Left-Handedness: Behavioural Implications and Anomalies* (Oxford: North-Holland, 1990).

Hardyck, C., & Petrinovich, L. F. Left-Handedness, *Psychological Bulletin*, 84 (1977), 385–404.

Herron, J. (Ed.). *Neuropsychology of Left-Handedness* (New York: Academic Press, 1980).

REFERENCES

- Annett, M. A. A Classification of Hand Preference by Association Analysis, *British Journal of Psychology*, 61 (1970), 303–321.
- Annett, M. A. Handedness, in J. G. Beaumont, ed., *Divided Visual Field Studies of Cerebral Organisation* (London: Academic Press, 1982).
- Annett, M. A. *Handedness and Brain Asymmetry: The Right Shift Theory* (New York: Psychology Press, 2002).
- Annett, M. A., & Ockwell, A. Birth Order, Birth Stress and Handedness, *Cortex*, 16(1980), 181–188.
- Bakan, P. Left Handedness and Birth Order Revisited, *Neuropsychologia*, 15 (1977), 837–840.
- Beaumont, J. G. Handedness and Hemisphere Function, in S. J. Dimond & J. G. Beaumont, eds., *Hemisphere Function in the Human Brain* (London: Elek Science, 1974).
- Beaumont, J. G., & McCarthy, R. A. Dichotic Ear Asymmetry and Writing Posture, *Neuropsychologia*, 19 (1981), 469–472.
- Birkett, P. Measures of Laterality and Theories of Hemispheric Processes, *Neuropsychologia*, 15 (1977), 693–696.
- Bradshaw, J. L., & Gates, A. Visual Field Differences in Verbal Tasks: Effects of Task Familiarity and Sex of Subject, *Brain and Language*, 5 (1978), 166–187.
- Bradshaw, J., Nettleton, N. C., & Taylor, M. J. Right Hemisphere Language and Cognitive Deficit in Sinistrals?, *Neuropsychologia*, 19 (1981), 113–132.

- Bryden, M. P. Response Bias and Hemispheric Differences in Dot Localization, *Perception and Psychophysics*, 19 (1976), 23–28.
- Bryden, M. P. Measuring Handedness with Questionnaires, *Neuropsychologia*, 15 (1977), 617–624.
- Bryden, M. P. Strategy Effects in the Assessment of Hemispheric Asymmetry, in G. Underwood, ed., *Strategies of Information Processing* (London: Academic Press, 1978).
- Bryden, M. P., Roy, E. A., McManus, I. C., & Bulman-Fleming, M. B. On the Genetics and Measurement of Human Handedness, *Laterality*, 2 (1997), 317–336.
- Buffery, A. W. H., & Gray, J. A. Sex Differences in the Development of Perceptual and Linguistic Skills, in C. Ounsted & D. C. Taylor, eds., *Gender Differences: Their Ontogeny and Significance* (London: Churchill-Livingstone, 1972).
- Carter, R. L., Hohenegger, M., & Satz, P. Handedness and Aphasia: An Inferential Method for Determining the Mode of Cerebral Speech Specialization, *Neuropsychologia*, 18 (1980), 569–574.
- Clements, A. M., Rimrodt, S. L., Abel, J. R., Blankner, J. G., Mostofsky, S. H., Pekar, J. J., et al. Sex Differences in Cerebral Laterality of Language and Visuospatial Processing, *Brain and Language*, 98 (2006), 150–158.
- Cohen, G., & Freeman, R. Individual Differences in Reading Strategies in Relation to Handedness and Cerebral Asymmetry, in J. Requin, ed., *Attention and Performance, VII* (Hillsdale, NJ: Erlbaum, 1978).
- Coren, S. (Ed.). *Left-Handedness: Behavioural Implications and Anomalies* (Oxford, UK: North-Holland, 1990).
- Davidoff, J. Hemispheric Differences in Dot Detection, *Cortex*, 13 (1977), 434–444.
- Ecuyer-Dab, I., Tremblay, T., Joannette, Y., & Passini, R. Real-Life Spatial Skills, Handedness, and Family History of Handedness, *Brain and Cognition*, 57 (2005), 219–221.
- Fairweather, H. Sex Differences: Little Reason for Females to Play Midfield, in J. G. Beaumont, ed., *Divided Visual Field Studies of Cerebral Organisation* (London: Academic Press, 1982).
- Faurie, C., Schiefenhövel, W., Le-Bomin, S., Billiard, S., & Raymond, M. Variation in the Frequency of Left-Handedness in Traditional Societies, *Current Anthropology*, 46 (2005), 142–147.
- Frith, U., & Vargha-Khadem, F. Are There Sex Differences in the Brain Basis of Literacy Related Skills? Evidence from Reading and Spelling Impairments after Early Unilateral Brain Damage, *Neuropsychologia*, 39 (2001), 1485–1488.
- Geffen, G., & Traub, E., Preferred Hand and Familial Sinistrality in Dichotic Monitoring, *Neuropsychologia*, 17 (1979), 527–531.
- Geffen, G., & Traub, E. The Effects of Duration of Stimulation, Preferred Hand and Familial Sinistrality in Dichotic Monitoring, *Cortex*, 16 (1980), 83–94.
- Grouios, G., Tsorbatzoudis, H., Alexandris, K., & Barkoukis, V. Do Left-Handed Competitors Have an Innate Superiority in Sports?, *Perceptual and Motor Skills*, 90 (2000), 1273–1282.

- Hannay, H. J., & Boyer, C. Sex Differences in Hemispheric Asymmetry Revisited, *Perceptual and Motor Skills*, 47 (1978), 317–321.
- Hardyck, C. A Model of Individual Differences in Hemispheric Functioning, in H. Whitaker & H. A. Whitaker, eds., *Studies in Neurolinguistics, Volume 3* (London: Academic Press, 1977).
- Hardyck, C., & Petrinovich, L. F. Left-Handedness, *Psychological Bulletin*, 84 (1977), 385–404.
- Hardyck, C., Petrinovich, L. F., & Goldman, R. D. Left-Handedness and Cognitive Deficit, *Cortex*, 12 (1976), 266–279.
- Heim, A. W., & Watts, K. P. Handedness and Cognitive Bias, *Quarterly Journal of Experimental Psychology*, 28 (1976), 355–360.
- Herron, J. (Ed.). *Neuropsychology of Left-Handedness* (New York: Academic Press, 1980).
- Hicks, R. A., & Beveridge, R. Handedness and Intelligence, *Cortex*, 14 (1978), 304–307.
- Hicks, R. A., Elliott, D., Garbesi, L., & Martin, S. Multiple Birth Risk Factors and the Distribution of Handedness, *Cortex*, 15 (1979), 135–137.
- Hines, D., Fennell, E. B., Bowers, D., & Satz, P. Left Handers Show Greater Test-Retest Variability in Auditory and Visual Asymmetry, *Brain and Language*, 10 (1980), 208–211.
- Hines, D., & Satz, P. Cross-Modal Asymmetries in Perception Related to Asymmetry in Cerebral Function, *Neuropsychologia*, 12 (1974), 239–247.
- Hiscock, I., Inch, R., Hawryluk, J., Lyon, P. J., & Perachio, N. Is There a Sex Difference in Human Laterality? III. An Exhaustive Study of Tactile Laterality Studies from Six Neuropsychology Journals, *Journal of Clinical and Experimental Neuropsychology*, 21 (1999), 17–28.
- Hiscock, M., Inch, R., Jacek, C., Hiscock-Kalil, C., & Kalil, K. M. Is There a Sex Difference in Human Laterality? I. An Exhaustive Study of Auditory Laterality Studies from Six Neuropsychology Journals, *Journal of Clinical and Experimental Neuropsychology*, 16 (1994), 423–435.
- Hiscock, M., Israelian, M., Inch, R., Jacek, C., & Hiscock-Kalil, C. Is There a Sex Difference in Human Laterality? II. An Exhaustive Study of Visual Laterality Studies from Six Neuropsychology Journals, *Journal of Clinical and Experimental Neuropsychology*, 17 (1995), 590–610.
- Hiscock, M., Perachio, N., & Inch, R. Is There a Sex Difference in Human Laterality? IV. An Exhaustive Study of Dual-Task Interference Studies from Six Neuropsychology Journals, *Journal of Clinical and Experimental Neuropsychology*, 23 (2001), 137–148.
- Hutt, C. *Males and Females* (London: Penguin, 1972).
- Isaacs, K. L., Barr, W. B., Nelson, P. K., & Devinsky, O. Degree of Handedness and Cerebral Dominance, *Neurology*, 66 (2006), 1855–1858.
- Kail, R., & Siegel, A. Sex and Hemispheric Differences in the Recall of Verbal and Spatial Information, *Cortex*, 14 (1978), 557–563.
- Lalumière, M. L., Blanchard, R., & Zucker, K. J. Sexual Orientation and Handedness in Men and Women: A Meta-Analysis, *Psychological Bulletin*, 126 (2000), 575–592.

- Leiber, L., & Axelrod, S. Not All Sinistrality is Pathological, *Cortex*, 17 (1981), 259–271.
- Levy, J. Possible Basis for the Evolution of Lateral Specialization of the Human Brain, *Nature*, 224 (1969), 614–615.
- Levy, J. Psychological Implications of Biological Asymmetry, in S. J. Dimond & J. G. Beaumont, eds., *Hemisphere Function in the Human Brain* (London: Elek Science, 1974).
- Levy, J. Cerebral Asymmetry and the Psychology of Man, in M. C. Wittrock, ed., *The Brain and Psychology* (New York: Academic Press, 1980).
- Levy, J., & Gur, R. C. Individual Differences in Psychoneurological Organization, in J. Herron, ed., *Neuropsychology of Left-Handedness* (New York: Academic Press, 1980).
- Levy, J., & Nagylaki, T. A Model for the Genetics of Handedness, *Genetics*, 72 (1972), 117–128.
- Levy, J., & Reid, M. Variations in Cerebral Organization as a Function of Handedness, Hand Posture in Writing, and Sex, *Journal of Experimental Psychology: General*, 107 (1978), 119–144.
- Maccoby, E. E., & Jacklin, C. N. *The Psychology of Sex Differences* (Stanford, CA: Stanford University Press, 1974).
- McFarland, K., & Anderson, J. Factor Stability of the Edinburgh Handedness Inventory as a Function of Test-Retest Performance, Age and Sex, *British Journal of Psychology*, 71 (1980), 135–142.
- McGlone, J. Sex Differences in Functional Brain Asymmetry, *Cortex*, 14 (1978), 122–128.
- McGlone, J. Sex Differences in Human Brain Asymmetry, *Behavioral and Brain Sciences*, 3 (1980), 215–263.
- McKeever, W. F. A New Family Handedness Sample with Findings Consistent with X-Linked Transmission, *British Journal of Psychology*, 91 (2000), 21–39.
- McKeever, W. F., & Hoff, A. L. Evidence of a Possible Isolation of Left Hemisphere Visual and Motor Areas in Sinistrals Employing an Inverted Handwriting Posture, *Neuropsychologia*, 17 (1979), 445–455.
- McKeever, W. F., & Van Deventer, A. Visual and Auditory Language Processing Asymmetries: Influences of Handedness, Familial Sinistrality and Sex, *Cortex*, 13 (1977), 225–241.
- McKeever, W. F., & Van Deventer, A. Inverted Handwriting Position, Language Laterality and the Levy-Nagylaki Genetic Model of Handedness and Cerebral Organisation, *Neuropsychologia*, 18 (1980), 99–102.
- McManus, I. C., & Bryden, M. P. The Neurobiology of Handedness, Language, and Cerebral Dominance: A Model for the Molecular Genetics of Behavior, in M. H. Johnson, ed., *Brain Development and Cognition: A Reader* (Malden, MA: Blackwell, 1993).
- Metzger, R. L., & Antes, J. R. Sex and Coding Strategy Effects on Reaction Time to Hemispheric Probes, *Memory and Cognition*, 4 (1976), 167–171.
- Miller, E. Handedness and the Pattern of Human Ability, *British Journal of Psychology*, 62 (1971), 111–112.

- Newcombe, F., & Ratcliff, G. Handedness, Speech Lateralisation and Ability, *Neuropsychologia*, 11 (1973), 399–407.
- Porac, C., & Coren, S. The Dominant Eye, *Psychological Bulletin*, 83 (1976), 880–897.
- Rasmussen, T., & Milner, B. Clinical and Surgical Studies of the Cerebral Speech Areas in Man, in K. J. Zülch, O. Creutzfeldt, & G. C. Galbraith, eds., *Cerebral Localization* (Berlin: Springer-Verlag, 1975).
- Raymond, M., & Pontier, D. Is there Geographical Variation in Human Handedness?, *Laterality*, 9 (2004), 35–51.
- Rizzolatti, G., & Buchtel, H. A. Hemispheric Superiority in Reaction Time to Faces: A Sex Difference, *Cortex*, 13 (1977), 300–305.
- Roberts, J., & Engle, A. Family Background, Early Development, and Intelligence of Children 6–11 Years (Rockville, MD: National Center for Health Statistics, 1974; DHEW Publication No. HRA 75-1624).
- Sappington, J., & Topolski, R. Maths Performance as a Function of Sex, Laterality, and Age of Pubertal Onset, *Laterality*, 10 (2005), 369–379.
- Sasanuma, S., & Kobayashi, Y. Tachistoscopic Recognition of Line Orientation, *Neuropsychologia*, 16 (1978), 239–242.
- Schachter, S. C., & Ransil, B. J. Handedness Distributions in Nine Professional Groups, *Perceptual and Motor Skills*, 82 (1996), 51–63.
- Searleman, A. Subject Variables and Cerebral Organization for Language, *Cortex*, 16 (1980), 239–254.
- Searleman, A., Tweedy, J., & Springer, S. P. Interrelationships among Subject Variables Believed to Predict Cerebral Organisation, *Brain and Language*, 1 (1979), 267–276.
- Semmes, J. Hemispheric Specialization: A Possible Clue to Mechanism, *Neuropsychologia*, 6 (1968), 11–26.
- Szaflarski, J. P., Binder, J. R., Possing, E. T., McKiernan, K. A., Ward, B. D., & Hammeke, T. A. Language Lateralization in Left-Handed and Ambidextrous People: fMRI Data, *Neurology*, 59 (2002), 238–244.
- Umiltà, C., Brizzolara, D., Tabossi, P., & Fairweather, H. Factors Affecting Face Recognition in the Cerebral Hemispheres: Familiarity and Naming, in J. Requin, ed., *Attention and Performance, VII* (Hillsdale, NJ: Erlbaum, 1978).
- Voyer, D. On the Magnitude of Laterality Effects and Sex Differences in Functional Laterality, *Laterality*, 1 (1996), 51–83.
- Warrington, E. K., & Pratt, R. T. C. Language Laterality in Left-handers Assessed by Unilateral ECT, *Neuropsychologia*, 11 (1973), 423–428.
- Warrington, E. K., & Pratt, R. T. C. The Significance of Laterality Effects, *Journal of Neurology, Neurosurgery and Psychiatry*, 44 (1981), 193–196.
- Zaidel, E., Aboitiz, F., Clarke, J., Kaiser, D., & Matteson, R. Sex Differences in Interhemispheric Relations for Language, in F. L. Kitterle, ed., *Hemispheric Communication: Mechanisms and Models* (Hillsdale, NJ: Erlbaum, 1995).

PART IV

APPLICATIONS

Neuropsychological Practice

The neuropsychologist who applies a knowledge of the functions of the brain to clinical practice is likely to find that a major, although not the only, role is assessment of patients. The assessment may be to assist in the diagnosis and localization of any cerebral pathology, but it is more likely to be undertaken to establish a patient's current functional status, described in psychological terms, to act as a basis for the design of remedial or rehabilitative therapy, and to monitor the progress of treatment and recovery.

Neuropsychologists are now centrally involved in the design and execution of therapy programs, contributing their specialized knowledge of the structure of human abilities, of the psychological aspects of training, and of specific behavioral methods to the efforts of the remedial team. This role for neuropsychologists within rehabilitation may seem obvious, but until about 30 years ago neuropsychology was principally concerned with diagnosis, rather than treatment. It was the introduction of scanning technologies, initially the computerized tomography (CT) scans (see Chapter 13) that liberated neuropsychology from its then primary role of assisting diagnosis in neurology and neurosurgery. Nevertheless, although considerably more clinical time is being devoted to rehabilitation and management, the major concern of most clinical neuropsychologists continues to be the assessment of cognitive abilities. In any clinic, neuropsychologists will typically be expected to provide an opinion on one of three questions: (1) whether the patient is suffering from any cerebral pathology and, if so, its nature, and whether focal or diffuse; (2) if focal cerebral pathology is present, which behavioral functions have been affected; and (3) how the patient can best be helped to return toward normal functioning.

The way in which answers might be sought to these three questions is sketched out below, but a general point should first be made. In trying to introduce as clearly as possible the relation between lesions and deficits, and the procedures of assessment and treatment, there is a danger that neuropsychological practice may seem to be simply a matter of following a well-charted routine. Nothing could be further from the truth. Neuropsychological practice involves sensitive and emphatic insight into the patient's problems (and not just the cognitive problems), and a creative and questioning intellectual approach to the dysfunction, no less than in any other area of clinical psychology. As a naive graduate student I expected neurological patients to be much easier to deal with than psychiatric patients—after all they were only medically ill, like people with broken legs or liver complaints; they would, I thought, be cooperative, well motivated, and grateful for my help. My view of all types of patient has since matured. Many neurological patients are indeed a pleasure to meet, but, largely as a result of their neurological state, some may be poorly motivated, distractible, and irritable. They often tire easily and suffer from headaches, causing bad temper, and consultation requires patience and empathy. The most important personal quality in any neuropsychologist is patience; an assessment may have to be spread, in brief sessions, over many days, and rehabilitation might take months of daily regular work. This makes clinical practice no less challenging and rewarding, but it is as well to remember the difficult conditions under which much assessment and therapy is carried out when reading the following sections.

TESTING FOR BRAIN DAMAGE

This is one of the classic demands directed to clinical psychologists or specialized neuropsychologists. It is, however, the demand that is least easy to satisfy. When psychologists are asked to give opinions as to whether a particular patient shows any form of brain damage or not, they are likely to refuse to answer so general a question, or at least to feel little confidence in their ability to answer it.

The issue may seem a little outdated, and in part it is since the introduction of modern scanning, but the question still arises. A patient has suffered a mild or moderate head injury in a road traffic accident some years previously but continues to complain of cognitive and behavioral problems. The scans reveal no abnormality. Does the patient have enduring neuropsychological consequences of the head injury, despite the fact that we might expect them to have cleared up within the first year or so after the accident? Or are these problems resulting from a psychological

reaction to the accident and its aftermath, and not directly attributable to the head injury at all? This is a common issue in the neuropsychological clinic, and it is important to find the correct answer. If a direct result of brain injury, at over 2 years from the accident, the problems are likely to endure for the remainder of the patient's life. If the problem is a secondary psychological reaction, then there is a good prospect that if this is treated, most commonly by cognitive behavior therapy (CBT), the patient may recover.

The difficulty in identifying the presence of damage to the brain arises with the assumption that there is some validity in the notion of a generalized phenomenon that can be called "brain damage." It should by now be clear that there are a variety of pathological processes that can result in cerebral lesions: trauma from blows that may or may not pierce the skull (*open* or *closed* head injuries); tumors; cerebral diseases; poisoning by toxic substances; strokes; brain hemorrhages; and the side effects of disorders of other bodily systems. These pathological processes may result in a diffuse pattern of lesions throughout the brain, or in more or less localized or focal lesions. The lesions may be in continuous progressive development, slow or rapid; or they may be static, the result of a single traumatic event. They may be chronic, of long standing, or acute and recently acquired. Lesions, of course, affect different hemispheres and different lobes of the brain, and the evidence presented in Part II showed how these variables could be related to different resulting deficits. Also take into account the patient's age, the premorbid level of achievements and abilities, and the current level of motivation, and it is easy to see what a dramatic range of behavioral disturbances, in both variety and severity, may follow damage to the brain.

Much time and effort were devoted during the decade following World War II to trying to establish some single factor that would characterize "brain damage"; it was hoped that by measuring such a factor it would be possible readily to determine the presence or absence of brain damage. The result of this research was, in essence, that no single factor could be identified. The only single aspect of psychological function that has any claim to be generally affected by any cerebral pathology is psychomotor speed. This is the speed with which simple tasks, usually requiring some active motor participation in response, can be carried out. It involves both speed of thinking and speed of motor execution. This might be useful were it not that this factor is equally affected by most functional psychiatric states, ruling out its application to the detection of brain damage.

The problem of *screening* for the presence of brain damage is nonetheless a practical clinical issue, and a number of single tests are employed to test for the presence of brain damage. Heaton, Baade, and

Johnson (1978) have usefully reviewed studies of the performance of these tests in making what is often the most difficult discrimination: between organic cerebral dysfunction and psychiatric states. They found that if chronic and process schizophrenics (those with long-lasting and severe psychotic illnesses) were included, then the tests performed very poorly. If, however, these patients were excluded, then the median success rate for determining brain damage was about 75%. The tests, in descending rank of success, included the Bender Gestalt Test (success enhanced by use of the Background Interference Procedure), the Benton Visual Retention Test, the Trail Making Task, and the Memory for Designs Test. The ability of these tests to discriminate between brain-damaged and psychiatric patients, excluding the chronic and process schizophrenics, was about equivalent to their ability to distinguish between brain-damaged and normal patients. It is a matter of opinion whether you regard these tests as successful and justified for use in clinical practice, but it is obvious that discrimination rates in the region of 75% leave very considerable room for improvement.

An additional factor must also be taken into account in assessing the utility of these and other assessment tests: the effect of base rates. There is an interesting statistical phenomenon (most lucidly explained in Vernon, 1964) that results in the ability of a test result to identify a certain characteristic being dependent on the base rate of that characteristic, the base rate being the proportion of individuals in the tested population who possess the characteristic. Discrimination will be best when the characteristic occurs in 50% of the population, and will decline dramatically as the base rate falls. In research studies that, as part of their design, employ equal-sized groups of those with and without brain damage, the success of a test in picking out those with brain damage will be considerably greater than when trying to screen for brain damage in a population where such damage might be expected to occur in, perhaps, only 20% of cases. Care should be taken that the base rates of the research studies match those of the target clinical populations.

It is also the case that when older people are to be tested, and determination of *cerebral dementia* in older adults is a difficult and yet clinically important problem, the tests that are often employed may perform even less well. The Newcastle-upon-Tyne study (Savage, Britton, Bolton, & Hall, 1973) found that indices derived from the Wechsler Adult Intelligence Scale (WAIS) tended to identify only a third to a half of the patients with organic conditions, at the same time misclassifying up to a third of normal subjects or psychiatric patients as organic. The Allen Index, which takes the difference between the summed scaled scores for the Information and Comprehension subtests and for the Digit Span and Digit Symbol subtests, correctly identified 52% of the patients with

organic problems but went on to declare 48% of the psychiatric patients and 62% of the normal subjects as brain damaged! Two more specialized tests, a paired associate learning test and the Modified New Word Learning Test, performed rather better, but not at a satisfactory level. It is sometimes pointed out that in screening, false positives are less serious than false negatives because they will result in further investigation, following which pathology will be ruled out, rather than resulting in pathology being missed. This is to some extent true, but it depends somewhat on the context. In the case of the aged, where organic and functional illnesses are most difficult to distinguish, it might well mean that a patient failed to receive treatment for a remediable functional psychiatric illness on the assumption that the problem was a progressive untreatable cerebral dementia.

Battery tests, which we shall shortly discuss, have also been used to detect generalized brain damage. They have had rather better success than the single tests, the Halstead-Reitan Neuropsychological Test Battery showing a general level of discrimination of about 80% (Boll, 1981), and the Luria-Nebraska Neuropsychological Battery a level of about 86%, for the presence of brain damage. This is to some extent because they sample a broad range of psychological functions, and therefore have a greater opportunity to pick up whatever signs of dysfunction are present. The improved success comes at the expense of very lengthy testing, which makes these procedures impractical as a general screening device for the presence of brain damage.

Before moving on to discuss the examination of specific functions, some of the difficulties that underlie the assessment of behavioral deficits should be pointed out. Two logical approaches can be taken to testing: direct and indirect. In *direct testing*, the patient's performance is measured premorbidly and then again following the onset of the lesion. This is the ideal state of affairs, but it is rarely possible. Occasionally, particularly in the case of servicemen, there will be recent formal assessments of intelligence and cognitive abilities that form an accurate baseline against which to measure subsequent decline. However, when this information is not available, the only direct approach is by repeated testing, perhaps weekly or monthly. Leaving aside the problems of teasing out the effects of familiarity with the test instrument, repeated testing may enable the development of the functional state to be charted, but it will not give any information by which to assess the extent of decline from premorbid abilities.

As the direct approach is often not feasible, an *indirect approach* must be adopted. One version of this is to find some index by which the patient's premorbid level of functioning can be inferred. At one time it was thought that vocabulary ability was not affected by cerebral dam-

age. This idea was based upon a further notion that vocabulary ability remains unaffected when other abilities decline during normal aging (which is, incidentally, not true when stated this simply). It was argued that if brain dysfunction was like normal aging, then vocabulary would remain stable and could be used as a pointer to premorbid intelligence. It is not surprising that this method has not been found to be valid. Vocabulary is, of course, affected by some cerebral lesions, and the way in which it is affected depends on the previous levels of ability and education; even hospitalization, in itself, is known to produce a decline in vocabulary ability. This approach has therefore fallen out of favor. However, one more recent development, the New Adult Reading Test (NART; Nelson & O'Connell, 1978), which is a simple test of the patient's ability to read a list of words, has been shown to predict with reasonable accuracy the premorbid intelligence level of adults with dementia, and is also widely used to infer premorbid ability following all kinds of trauma. The list employed is composed of irregular words—those that cannot be read by the standard grapheme to phoneme conversion rules—and so the pronunciation has had to be learned for these words, also sometimes called “exception” words. An example would be *salmon*; the rules predict that this should be pronounced “sal-mon” but, since it is an irregular word, this is not the correct way to pronounce it. You just have to know how to say it. The NART is now in a second edition and has been joined by the Wechsler Test of Adult Reading (WTAR), which is constructed on exactly the same principle.

The only other approach that remains to the neuropsychologist, and another indirect one, is the use of inferences from the patient's history. It is possible to piece together from the patient's educational attainments, occupational choice and success, and pastimes and interests, a fairly accurate picture of the level and pattern of the patient's cognitive abilities. It will be easier in some cases than others, particularly if examinations have recently been passed, or particular occupations successfully taken up. The prudent clinician will generally check an estimate derived from one of the tests of adult word reading against this kind of estimate; it is not uncommon to see individuals who simply were more interested in sports, or motorcycles, than in their academic subjects, and these individuals may attain relatively poorly on the formal test while having achieved more successfully in their subsequent occupational life in an activity for which they are motivated, often based on practical rather than academic skills. However, these data are all that the clinician may have to work with; this is yet another challenge to the neuropsychologist's professional skill in creating an accurate and valid assessment from the information that is available or may be collected, both by direct observation and by indirect inference.

THE ASSESSMENT OF SPECIFIC FUNCTIONS

It was noted in Chapter 1 that there are three distinct traditions in the approach to neuropsychological assessment of specific functions. These traditions are behavioral neurology; the use of neuropsychological batteries; and the individual-centered normative approach. They can loosely be said to typify the historical traditions of clinical neuropsychology in the Soviet Union, the United States, and Britain respectively.

Behavioral Neurology

This approach derives from the influential work of the Russian neuropsychologist A. R. Luria and is individual centered and clinical in nature. The goal of neuropsychological assessment is not a quantitative measurement of patients' difficulties, but a qualitative analysis and description of their problems. Rather than employing psychometric procedures to identify abnormal performance by statistical means, with reference to a normal population, the emphasis is on behaviors that any normal individual of the age, background, and general ability of the patient should be able to perform. When such behaviors cannot be generated, then a deficit has been demonstrated. Particular attention is paid to the qualitative aspects of *how* a task is performed, instead of merely to the absolute level of performance that is observed.

The neuropsychological examination has four essential aspects. First, the psychologist begins from his or her knowledge of the different types of dysfunction that follow cerebral lesions, that is, from a *model* of the organization of the brain. Second, in order to locate the areas of dysfunction to be investigated in depth, the initial stages of examination explore in a preliminary fashion the optic, auditory, and kinesthetic processes together with motor behavior. Third, in analyzing the behavioral deficits observed, a distinction is maintained between deficits that follow from a primary failure in the system under investigation and those that are a secondary result of some more general fundamental failure. Last, the examination must include tests of complex integrated activity, such as speech, reading, writing, comprehension, and problem solving. The aim is a careful qualitative analysis of the entire range of patients' activities, of the difficulties that they experience, and the mistakes they make.

The investigation therefore begins with a general evaluation of the basic "individual analyzers" (optic, auditory, and so on) of Luria's model, considered in terms of the levels of direct sensory reaction, mnemonic organization, and complex mediated operations (Luria's terms). The tests available are short and are selected for their appropriateness to the patient. There is no rigid pattern of administration or scoring. The

investigation then moves into a second phase designed to investigate in detail the problems of the individual patient, a period of individualized qualitative exploration.

While maintaining a high regard for the insightful and persuasive nature of much of Luria's model, and an admiration for Luria's clinical skills, most Western psychologists have found this approach difficult to apply. However, considerable assistance to those attempting to employ the approach was given by Christensen's publication of a systematized version of the tests used in Luria's clinic (Christensen, 1974). While covering only the initial stages of the investigation, it provides a source of materials and procedures for use in assessment. The text is divided into main sections on topics including "Acoustico-motor Organization," "Higher Cutaneous and Kinesthetic Functions," "Impressive Speech," and so on. Each of these is divided into subsections for which a number of simple test procedures are supplied. The Investigation of Acoustico-motor Organization is broken down into the investigation of perception and reproduction of pitch relationships, and the perception and reproduction of rhythmic structures. The first of these is further subdivided into the perception of pitch and the reproduction of pitch relationships and musical melodies, the second into perception and evaluation of acoustic signals and motor performances of rhythmic groups. For each of about three or four dysfunctions that might be observed under each of these subdivisions, a lesion site is suggested. For example, in motor performance of rhythmic groups, using the four little tests of rhythmic reproduction suggested, if the patient does not fully apprehend the incorrectness of his or her performance, a right temporal lesion is indicated, but a lesion of the frontotemporal division of the cortex is indicated if the patient exhibits marked incoordination in all tests. This version does not reflect fully the philosophy behind Luria's work but it conveys something of the character and flexibility of the approach. For a good description of the application of Luria's approach, the paper by Luria and Majovski (1977) is a useful introduction to the fuller account in Luria (1980).

The advantages of Luria's method are first that it is based upon an explicit theoretical foundation, his model of cerebral organization, although this model (as noted on p. 59, for example) has not always been supported by empirical evidence. Second, it emphasizes the qualitative aspects of performance and is flexible in approaching the diagnosis of functional deficits. Particularly in view of our imperfect knowledge of cerebral organization and function, this latter aspect might be expected to result in greater accuracy and finer resolution in the final description of a patient's difficulties.

The disadvantage is that the system depends almost entirely upon the clinical acumen and skill of the neuropsychologist. In the hands of

Luria, the results were impressive, as the published case reports demonstrate. However, the approach demands a comprehensive grasp of the details of Luria's complex theoretical model. There are no rigorous investigations of the validity of the procedures in routine application, and the result, in the West, has been some suspicion of the "clinical-analytical" approach, and a reluctance to apply it in regular practice.

Neuropsychological Batteries

The strict psychometric approach, in complete contrast with behavioral neurology, has been more popular in the United States than elsewhere. While it would be false to give the impression that this is the only approach adopted there, it is the only region where the employment of neuropsychological batteries has been taken up with any enthusiasm and, if anything, the trend is toward increasing popularity.

Historically, there have been a number of batteries that have been widely used in clinical practice. However, two are currently of major importance: the Halstead-Reitan and the Luria-Nebraska.

The Halstead-Reitan battery has been gradually developed by Reitan and co-investigators over a number of years from tests originally selected, on a largely pragmatic basis, by Halstead in the 1940s. The exact tests that make up the battery vary a little from clinic to clinic, but the tests seek to cover as comprehensive a range of behavioral functions as is possible. The Halstead Category Test (see Figure 3.1) is a concept attainment task involving learning, memory, mental efficiency, and adaptability. The Tactual Performance Test is a formboard test with repeated presentations in which improvement in speed can be observed, culminating in a blindfold trial in which the patient must remember the location of the holes that take each shape. The Rhythm Test is drawn from the Seashore Tests of Musical Ability (see p. 76), and the Speech Sounds Perception Test involves multiple-choice identification of auditorily presented nonsense words. The Finger Oscillation Test examines simple motor speed. Most versions of the battery include all the above tests.

Also employed are the "allied procedures," comprising the Trail Making Test, in which numbered or lettered circles are to be joined in sequence, or by alternating letters and numbers, by a pencil line; the Strength of Grip Test; and the Sensory-Perceptual Examination, a test of tactile, visual, and auditory modalities. To these are added three tests of tactile perception and a modified form of the Halstead-Wepman Aphasia Screening Test. The full WAIS is often also given.

It will be appreciated that this battery takes a long time to administer. Even with uninterrupted testing a period of 6 to 8 hours is required, and with a distractible or easily fatigued patient testing may have to be

undertaken over many days. Nevertheless, the battery has been widely used, and a great deal is known about its reliability, validity, and psychometric structure. The battery, in common with most procedures, has some difficulty in identifying chronic schizophrenics as psychiatric patients, but otherwise the results are quite impressive. Reitan, working from the test protocols alone, without any additional information about the patient, is able to identify the presence of brain damage, distinguish diffuse from focal lesions, and lateralize and localize focal lesions, with an accuracy of around 80%. This may to some extent reflect Reitan's clinical skills, but even relatively untrained personnel can show remarkable diagnostic success using the battery. There has been extensive investigation of the performance of the battery when applied to criterion groups of brain-damaged and psychiatric patients and normal subjects in a long series of studies for validation and cross-validation (in which diagnostic criteria established in one research sample are tested out upon an independent sample).

One particularly interesting development has been the creation of a computer-supported automated diagnostic process that operates by a number of classificatory keys from the set of 41 scores yielded by the full battery (Russell, Neuringer, & Goldstein, 1970). This program had an accuracy rate of 88% for detecting brain damage, 80% for lateralizing left and right hemisphere lesions, and 62% for identifying diffuse lesions. This compares well with traditional methods, and the computer agreed with clinical neurologists and neuropsychologists as often as they agreed with each other.

In favor of this battery, then, is the extensive evaluation of its validity that has been published. It covers a broad range of functions and has the ability to identify complex test patterns and relationships across a number of tests, which may typify certain specific conditions. Nevertheless, the test carries a heavy penalty in the time taken in administration, and it does not cover all possible neurological deficits. Its heavy reliance on motor performance also rules out its use with certain patients. It has, however, proved a useful instrument, and may form the basis of further improved versions.

The second, and much more recently developed, battery is that generally known as the Luria-Nebraska Neuropsychological Battery, more formally referred to as the Standardized Version of Luria's Neuropsychological Techniques. This is just what it is, an attempt to take Luria's procedures and assemble a battery of normatively based tests from them.

The battery is formed of 269 items that cover the main components of the Luria Investigation as described, for example, by Christensen (see p. 324). These components comprise motor function; rhythmic and pitch skills; tactile functions; visual (spatial) functions; receptive speech;

expressive speech; writing; reading; arithmetic; memory; and intellectual processes. Each item is scored according to formal protocols. Summary Pathognomic, Left Hemisphere, and Right Hemisphere scores are calculated, and a profile across the individual scales may be drawn.

There is much less research available on the Luria–Nebraska battery than the Halstead–Reitan battery. Nevertheless, reports to date are extremely encouraging and suggest that it is at least as powerful in identifying both the presence and the type of brain damage. A recently published cross-validation study (Golden, Osmon, Moses, & Berg, 1981) found that the presence of brain damage was correctly diagnosed in 86% of cases, the lateralization was correct in 78%, localization in 92%, and the quadrant of the brain in which the lesion was situated was correctly identified in 84% of cases.

This battery does have the advantage that it takes considerably less time to administer than the Halstead–Reitan, at around 2½ hours, and it relates more directly to the patient’s problems on a clinical level. However, it requires that the user be aware of Luria’s formal model, and the greatest clinical value will be derived from it only when qualitative as well as quantitative aspects of performance are taken into account. The authors of the test (principally Charles Golden) specifically state that the test depends upon both the standardized and qualitative interpretive systems that the battery can provide. Nevertheless, this battery has been taken up with some enthusiasm, particularly in North America. (Several books that clinical case examples of the use of both these batteries are listed at the end of the chapter.)

The Individual-Centered Normative Approach

This approach, typical of clinical practice in Britain, stands between the two approaches already described. It relies to some extent on a formal psychometric approach, but it emphasizes the need to tailor the assessment to the nature of a particular patient’s difficulties. British neuropsychologists have generally considered batteries to be inefficient, wasteful of time and resources, and unlikely to provide a full and accurate description of the dysfunction under investigation. The aim of formulating such a description, which goes beyond a simple diagnostic classification to an understanding of the behavioral deficits in psychological terms, has always been kept firmly in view.

The strategy of assessment has therefore been, as in the Russian approach, to conduct a broad general survey of the patient’s functions, identifying areas of difficulty and pursuing an analysis of these particular areas. Each investigation thus becomes an individual examination of the possibly unique state that the patient exhibits. Unlike the Russian

approach, however, the particular tests employed are rooted in the empirical tradition. Performance is carefully analyzed and scored, and reference made, whenever possible, to established norms against which to judge the patient's performance. Where individual experiments are set up to test some hypothesis about the patient's cognitive state, attention is paid to the scientific design of the procedures, and there will be a formal statistical evaluation of the results.

The procedure of neuropsychological consultation, after collection of informal data about the patient's complaint, and a preliminary interview with the patient, might often begin with the WAIS (Kaufman & Lichtenberger, 1999; Tulsky et al., 2003). Although never designed with such an application in mind, the quality of this test and the range of functions that it examines make it the most popular instrument for detailed general evaluation of intellectual functions. The degree to which a neuropsychologist may be willing to infer deficits from the pattern of results on the WAIS varies widely, and few are likely to consider it a sufficient assessment, although a great deal can be inferred by a skilled clinician from this test alone.

Having identified the general areas of difficulty, and undertaken tests of primary sensory functions if appropriate, the neuropsychologist will have begun to formulate hypotheses about the nature of the deficit, and perhaps the lesion site. The next stage is to employ specific individual testing procedures to evaluate these hypotheses. The tests are of the type described in Chapters 3 to 6 when discussing the effects of specific lesions, and include the assessment of language function, which was discussed on pp 150–151. Because these tests have been shown to be affected by lesions of certain areas, then as a corollary the tests can be used to locate the sites of unidentified lesions. There is an enormous number of such tests available. Lezak (Lezak, Howieson, Loring, & Hannay, 2004) in her valuable and comprehensive guide to neuropsychological assessment lists about 300, but most clinicians will routinely use perhaps a couple of dozen, calling on less popular tests when an unusual or baffling case is encountered.

For example, if a frontal lesion is suspected, there are three areas of function that are likely to be examined. Concept attainment and abstraction may be tested with the Wisconsin Card Sorting Test (Figure 3.3), or the Halstead Category Test (Figure 3.1). Planning and the integrated execution of complex actions may be investigated with the Porteus mazes (Figure 3.5), the Trail Making Test; or the tests contained within the Behavioural Assessment of the Dysexecutive Syndrome (BADS). Finally, the Verbal Fluency Test might be used to check on "frontal" aspects of verbal function. There will, however, be wide variation in the practice of individual neuropsychologists (Rabbitt, 1997).

Because these tests are insufficient to provide a comprehensive coverage of behavioral functions, the gaps will be filled by the development of special experiments to investigate individual hypotheses about the deficits under examination. If a patient's poor motor performance is thought to be due to poor kinesthetic feedback, then artificial feedback could be introduced under experimental conditions to see if performance improves. If the problem is thought to be due to a deficit of verbal memory, then additional cues could be used to determine the level of the patient's performance. As another example, a patient's difficulty in performing mental arithmetic could be examined by presenting problems in different forms, verbally encoded or formally abstract, in written or mental form, in order to tease out whether the difficulty is one of abstracting the problem, of employing an appropriate strategy for solution, or of undertaking the mechanics of computation. The possibilities are limited only by the ingenuity of the neuropsychologist and the sophistication of his or her model of cognitive processes.

It must be remembered that the investigation aims not only to classify the lesion, but also to understand the patient's difficulty in psychological terms. This form of cognitive analysis (Shallice, 1988) attempts to specify the dysfunction in terms of the rate and accuracy of information processing, and the operation of the normal repertoire of cognitive strategies. The component processes are identified and measures of the efficiency of each employed. This psychological analysis can in turn be related to areas of localized cerebral function, but it can also form a basis for the management, treatment, and rehabilitation of the patient. This would be lacking from a simple diagnosis of the lesion and its site. An excellent demonstration of how the cognitive psychologist's knowledge of reading processes can help in assessing patients, and of how data from the clinic can be fed back to develop our understanding of normal function, has been provided by Patterson (1981).

This approach relies heavily on the skills and insight of the individual clinician. There is the risk that areas of function will be neglected, or that complex patterns of functional interaction will be missed. Nevertheless it seems the most intelligent approach to neuropsychological assessment, if practiced by a capable and sensitive clinician.

ASSESSMENT IN PRACTICE

All this may seem rather bewildering; there are various approaches and a myriad of tests that might be employed. While the choice of approach and specific procedures is generally dictated by the clinical question of what is to be discovered about the patient's state, and for what purpose,

it may assist in clarifying matters if I describe my own practice in conducting examinations for medicolegal purposes. Here the mission is fairly broad, to perform a psychological analysis of any deficit from which the individual is suffering, normally as a consequence of a road traffic accident or an occupational accident, and to analyze the impact of any such deficit in everyday life, now and in the future. I conduct these examinations about twice each week, and the reasonable period for which it is possible to assess an individual is limited to about 3 hours. I should very clearly emphasize that this is just an example and that while many of my colleagues perform slightly different examinations, I do not claim that mine is superior or that there are not other equally valid methods for examining clients. Nevertheless, this basic assessment has evolved over many years, and some considerable thought has gone into it, partly because every so often I have to stand up in court as an expert witness and defend it!

I should also make clear that while this is the basic form of the assessment it is regularly varied to take account of specific issues and circumstances. The client may be so severely affected cognitively, or by visual or motor handicaps, that these tests are inappropriate. The client's first language may not be English, which creates specific problems for assessment. It is rare that this set of tests is administered as described without some adaptation; but I do have a notional standard form of the assessment that illustrates the principles adopted in conducting it.

Before seeing a client it will have been usual to review the client's full medical history, and to have read accounts of the traumatic incident and seen medical records relating to hospitalization following the accident. There may also be school records and occupational records, and expert opinions already prepared by other neuropsychologists and experts in related fields such as neurology, neurosurgery, orthopedics, psychiatry and rehabilitation medicine. So, one is reasonably well informed about the history before the client arrives. Even so, after trying to put the client at ease, it is important to spend at least a further half hour obtaining the client's own view of his or her problems and clarifying the history. Actually observing clients while they describe their difficulties and their experience provides important data, as does observation of their performance on the formal tests. That is why in Britain, unlike in North America, it is considered important that the neuropsychologist actually administer all the tests, rather than have a technician conduct the testing and provide the test scores. While the formal psychometric data are the core of the assessment, interpretation and analysis are also informed by the qualitative clinical observations made of the client.

The formal assessment generally begins with one of the adult word reading tests, and I happen to prefer the WTAR, despite certain limita-

tions. The results of the WTAR will be combined with information about the client's educational and occupational history to infer an estimate of his or her likely general cognitive ability before the accident occurred. This will then be compared (formally and statistically) with the assessment of the current level of general cognitive ability obtained from the third edition of the Wechsler Adult Intelligence Scale (WAIS-III). This battery provides not only overall IQ indices for the major functional areas, but also factor-based index scores that may indicate a significant slowing in mental speed, or a deficit in working memory reflecting problems with attention and concentration, for example, even in the presence of intact "core" verbal and visuospatial abilities. Highly specific deficits may be apparent on particular subtests.

After this battery, which may take up to an hour in a slightly modified form, the client usually needs a break. A cup of tea or coffee helps to dissipate fatigue, and I often enjoy the opportunity simply to chat with clients in an informal way. It is also surprising and relevant to note how once clients are engaged in conversation on a topic that is of interest to them, their mood may lighten and their psychological function improve.

Next comes a battery of memory tests and my preference is for the Wechsler Memory Scale, third edition (WMS-III), partly for its inherent psychometric quality, and partly because formal statistical comparisons can be made with the results of the WTAR and the WAIS-III to determine whether current memory function is impaired with respect to estimated premorbid ability, or with respect to the current level of general cognitive ability. However, certain other batteries are in widespread use, and I commonly use the Camden Memory Tests or the Doors and People Test as additional or alternative tests of recognition memory. A scale of verbal learning may also be specifically employed, or the Rivermead Behavioural Memory Test if the standard tests prove too demanding.

This survey of general cognitive abilities and a more detailed exploration of memory provide a broad assessment of cognitive functions but with one important omission: tests of frontal executive functions. It was noted in Chapter 3 how severe and handicapping problems of executive function can occur in the absence of any abnormality apparent on other tests. Some specific tests of this area of function are therefore next included. My favorites are the Cognitive Estimates Test; a test of verbal fluency; the Trail Making Test; and the Brixton Test (which may be considered a briefer and more palatable alternative to the Wisconsin Card Sorting Test). Elements of the BADS might also be administered. Specific tests of other areas of function such as word naming or visual object and space perception might also be included here.

My 3 hours are starting to run out by now, but time remains to conduct a brief survey of the client's psychological state through the Beck

Anxiety and Depression Scales and, in cases where the interview has suggested that it might be important, a scale to assess symptoms of post-traumatic stress disorder. I have always considered it appropriate and courteous to provide some information to clients about the outcome of the assessment and potential treatment and rehabilitation options, and to answer any questions the client may have. The client leaves, and it just remains to score the tests, think hard about the implications of the findings, and prepare a lengthy and detailed report!

REHABILITATION

It is difficult to overemphasize the dramatic change that has occurred in neuropsychology with respect to rehabilitation. Only 25 years ago when preparing the first edition of this book, I wrote: "I wish that it were possible to devote a full chapter to treatment and rehabilitation, but unfortunately this would not be justified. While there are encouraging developments, neuropsychologists have not traditionally been heavily involved in therapy for brain-damaged patients." How things have changed, and for the better.

Clinical neuropsychologists now work in a variety of environments, and it is probably a minority who work in an acute setting in direct contact with neurologists and neurosurgeons, commonly in Britain in a regional neurosciences unit. A majority of specialist clinical neuropsychologists now work in a rehabilitation context, either within a brain injury rehabilitation unit that is likely to be, at least in part, residential or, increasingly, attached to a community rehabilitation team providing services in the client's own home. Many of the managers or clinical leaders of these units and teams are neuropsychologists. Nevertheless, rehabilitation teams are strongly multidisciplinary and depend upon active collaboration among specialists in neurorehabilitation aspects of neurology, neuropsychiatry, nursing, occupational therapy, physiotherapy, rehabilitation medicine, social work, and speech and language therapy.

One of the difficulties in developing neurological rehabilitation has been to formulate a model of *how function is reinstated*, when that does occur. As there is no significant regrowth of damaged tissue in the central nervous system (although neuroscience may yet provide a solution to this critical problem), what process results in lost functions being regained? There are at least four ways in which this might happen. First, recovery may result from the reinstatement of function at its original site, in tissue that has not been irreversibly damaged but only temporarily upset by processes associated with the acute effects of the lesion. Second, recovery may result from the adoption of the function of the

damaged area of cortex by an area that did not originally serve it. Some have considered that this process of neural relocation might be fundamentally programmed into the system. Third, there may simply be adaptation to the deficit. As most high level performance can be achieved by the mobilization of a variety of component processes, it may be possible to learn to perform the old skills by new processes that avoid the site of dysfunction in the system. Finally, some have thought that recovery reflects the original tissue regaining control of functions that have been temporarily taken away by other sites during the acute phase of the illness (Andrewes, 2001; see also Chapter 10). Without an accepted model of how recovery is achieved, it is difficult to plan a rational program of therapy that will be both effective and efficient.

However, in practice, there are two approaches to reestablishing function: relearning an ability through training and practice or adapting to the deficit by learning alternative methods or strategies to achieve the desired function. The latter can be thought of as similar to the behavior of traffic when a road in a city is closed. Vehicles simply find a diversion around the obstacle (the lesion in the road network) and are nevertheless able to reach their goal (achieve functional performance).

The progress of recovery, unaided or with the assistance of rehabilitation, can last over a very long period, and improvement continuing over at least 6 years has been recorded in some studies. Nevertheless, it is generally accepted that the most important period of recovery, if it is going to occur, is during the first year following injury, and especially during the first 6 months, with a gradual plateau developing during the second year, at the end of which natural recovery is essentially complete. There have, however, been few formal studies of the course of recovery. The exceptions have been long-term studies of the war injured over periods of up to 30 years (Newcombe, 1969; Teuber, 1975), which show certain deficits persisting throughout that period, and recent studies of the psychosocial effects of closed head injury (Ponsford, 1995; Richardson, 2000). These studies have shown great variation in the social functioning regained by the patients, related in part to the severity of the injury, but also to the patient's premorbid personality and intelligence.

However, there are a number of forms of active therapeutic intervention by neuropsychologists. Aphasia therapy, shared with speech and language therapists, has already been discussed (see p. 151) and forms an important aspect of this work. In more general terms, training has been the dominant strategy employed. This has sometimes been within the context of a belief that practice of a skill might actually play some part in the direct physiological reinstatement of that function, and it is sometimes known as "brain function therapy" (Buffery, 1977; Powell, 1981). More often, the approach has been simply pragmatic, in that it is

accepted that the patient cannot perform a certain activity, and psychological knowledge of learning is applied to teach the patient to perform it.

The kind of contribution that the psychologist can bring to the design of *training programs* is in performing task analyses and constructing appropriate training routines (Golden, 1981). This is where the cognitive analysis carried out during assessment can be of great value. The components of the dysfunctional skill must be recognized if the training is to be accurately directed at the dysfunctional processes. Once the components have been identified, they can be practiced within tasks in which the level of difficulty can be varied, and the errors committed by the patient controlled. Error-free learning has been shown to be particularly effective in neurorehabilitation (Wilson, Baddeley, Evans, & Shiel, 1994) and the principle is now widely employed. Emphasis should, in any event, be placed upon careful monitoring of the patient's progress, and on providing immediate, direct, and clear feedback about actual performance. Biofeedback (for example, of neuromuscular activity) and other forms of concurrent enhanced monitoring of performance have sometimes been used to aid patients in maintaining an accurate internal representation of their performance and its effects.

Where direct learning approaches are inappropriate or ineffective, it may be productive to introduce new *cognitive strategies* to the patient. For example, the patient with a verbal memory deficit may be taught to use explicit visual-imagery associations and nonverbal mnemonics to improve memory performance. Alternatively, patients may be encouraged to deepen their encoding of stimuli by thinking directly about associations with the material, constructing appropriate cues, and practicing regular rehearsal, so that words are less likely to be forgotten and may be retrieved when recall is attempted. If there is currently a dominant approach to neuropsychological rehabilitation, it is *cognitive rehabilitation*, in which attempts to intervene in the operation of cognitive processes are seen as facilitating the reacquisition of function (Riddoch & Humphreys, 1994; Stuss, Winocur, & Robertson, 1999).

Specific *behavioral methods* have also been explored, using operant, or occasionally classical, conditioning techniques, although they are currently less widely employed than cognitive approaches. Existing behavior may be shaped or new behavior established by the use of explicit reinforcement strategies, the patient being rewarded for the appropriate performance of some target behavior. Inappropriate behavior can similarly be reduced by the withdrawal of rewards, or by punishment regimes, although punishment is now regarded as singularly ineffective and in most cases ethically unacceptable.

An example of an elegant and purely behavioral approach is provided by Wood (1987). A patient regularly sat with head bowed, despite having the muscular ability and motor control to maintain a more normal posture. In this position he was understimulated by events occurring around him and unlikely to engage in social interaction. The patient was provided with a personal music player, and his favorite music, but the player was also equipped with a mercury tilt-switch so that the music only played when the patient lifted his head, stopping when his head dropped. This simple intervention resulted in a clear increase in the time the patient held his head in a more appropriate position and, after a period, not only when the player was on his head.

Recent developments in behavioral approaches, which may be particularly important when the patient is so impaired cognitively that benefit cannot be obtained from cognitive approaches, have centered around *positive programming*. Rather than being concerned with undesirable behaviors and the need to direct rehabilitation at reducing them, the emphasis is on increasing the frequency of desirable behaviors. It is generally possible to be more successful in increasing desirable behaviors than in removing undesirable ones, and an increase in positive adaptive behaviors, especially if they are incompatible with those that are undesired, simply results in their replacing the problem behaviors (Rothwell, LaVigna, & Willis, 1999).

Reality orientation therapy should also be mentioned as it is so widely employed with confused patients, particularly confused older people (Miller, 1977). This form of milieu therapy has been successfully used with elderly patients with dementia and is designed to provide artificially the basic information that the patient requires in order to conduct sensible interaction and generate appropriate behavior. A simple example is to greet Mrs. Brown, wandering about the ward at 2 a.m., with "Hello Mrs. Brown, you are in Barchester Hospital, and it's two o'clock in the morning. You ought to be in bed, asleep. Can I help you, dear?" rather than simply "What's the matter, dear?", which does not assist the patient to maintain appropriate orientation for time and place, and behave accordingly. The rationale for this form of therapy is clear.

In general, there has been a fruitful and increasing emphasis on holistic approaches to rehabilitation (Ben-Yishay, 1996; Prigatano, 1999; Sohlberg & Mateer, 2001). Here, the idea is not to concentrate upon specific handicaps, but to consider the whole individual in his or her personal, social, and possibly occupational, context. Rehabilitation may not be an attempt to restore the individuals to their previous condition, as this may be inappropriate and unrealistic for a variety of reasons, but to work toward the best adaptation that can be achieved for

patients in their new personal and social context. There is an interaction between the condition of the patient and the environment in which he or she lives, and rehabilitation will be about adapting the environment appropriately, as much as it is about trying the “fix” the patient’s problems.

CONCLUSION

The efforts of the neuropsychologist are of course only one element in the operation of a clinical team. In an acute context, besides the neuropsychologist, and the neurologist or neurosurgeon who heads a team of medical staff, there are radiographers, electroencephalographers, pathologists and biomedical laboratory staff, medical physicists, speech and occupational therapists, medical social workers, and of course the nursing staff. A neuropsychiatrist may well also bring special skills into play. The diagnostic information and the recommendations for management and treatment that the neuropsychologist makes will therefore be evaluated in the light of findings and opinions contributed by other members of the team. The level of accuracy of neuropsychological assessment may seem disappointingly low, and there is undoubtedly considerable room for improvement in the validity of assessment procedures, but taken in the context of other medical and physical investigations, it can contribute crucial information to the understanding of the patient’s condition.

A variety of general approaches and specific procedures have been used in determining the presence of brain damage and in assessing its nature and location. Many of these methods show a moderate degree of success, and are of undoubted clinical value, but at present none seems outstanding in accuracy or efficiency. However, with a shift in emphasis to the psychological analysis and functional description of a patient’s neuropsychological problems, neuropsychological assessment has found an important new role in the understanding of handicap, the planning of rehabilitation and management, and the future adaptation of those with injury or disease. In this role neuropsychological assessment has proved more successful.

In a dramatic change over the past 25 years, the majority of clinical neuropsychologists now work in a context in which rehabilitation is the primary goal. In a team, not uncommonly led by a neuropsychologist, the neurorehabilitation disciplines together provide intensive rehabilitation to assist those with neurological injury or disease to minimize their handicaps, and to optimize their adaptation to the physical and psychological problems of everyday living in a context of good psychological health.

FURTHER READING

Further reading in relation to the general topic of clinical applications is to be found at the end of Chapter 1, but more specialized texts that focus on assessment include:

- Christensen, A.-L. *Luria's Neuropsychological Investigation: Text* (Copenhagen, Denmark: Munksgaard, 1974).
- Golden, C. J., Osmon, D. C., Moses, J. A., Jr., & Berg, R. A. *Interpretation of the Halstead-Reitan Neuropsychological Test Battery* (New York: Grune & Stratton, 1981). This includes copious case material.
- Groth-Marnat, G. *Neuropsychological Assessment in Clinical Practice: A Guide to Test Interpretation and Integration* (New York: Wiley, 2000).
- Hobben, N., & Milberg, W. *Essentials of Neuropsychological Assessment* (New York: Wiley, 2002).
- Kaufman, A. S., & Lichtenberger, E. O. *Essentials of WAIS-III Assessment* (New York: Wiley, 1999).
- Lezak, M. D., Howieson, D. B., Loring, D. W., & Hannay, H. J. *Neuropsychological Assessment* (New York: Oxford University Press, 2004). An authoritative guide to general procedures and specific tests.
- Lichtenberger, E. O., Kaufman, A. S., & Lai, Z. C. *Essentials of WMS-III Assessment* (New York: Wiley, 2001).
- Spreen, O., & Strauss, E. *A Compendium of Neuropsychological Tests: Administration, Norms, and Commentary* (Third edition, New York: Oxford University Press, 2006).

See Chapter 7 for references to aphasia therapy, but on recovery and rehabilitation the most useful texts are:

- Ponsford, J. *Traumatic Brain Injury: Rehabilitation for Everyday Adaptive Living* (Hove, UK: Psychology Press, 1995).
- Riddoch, M. J., & Humphreys, G. W. (Eds.). *Cognitive Neuropsychology and Cognitive Rehabilitation* (Hove, UK: Erlbaum, 1994).
- Sohlberg, M. M., & Mateer, C. A. *Cognitive Rehabilitation: An Integrative Neuropsychological Approach* (New York: Guilford Press, 2001).

REFERENCES

- Andrewes, D. G. *Neuropsychology: From Theory to Practice* (Hove, UK: Psychology Press, 2001).
- Ben-Yishay, Y. Reactions on the Evolution of the Therapeutic Milieu Concept, *Neuropsychological Rehabilitation*, 6 (1996), 327–343.
- Boll, T. J. The Halstead-Reitan Neuropsychology Battery, in S. B. Filskov & T. J. Boll, eds., *Handbook of Clinical Neuropsychology* (New York: Wiley, 1981).
- Buffery, A. W. H. Clinical Neuropsychology: Review and Preview, in S. Rachman, ed., *Contributions to Medical Psychology* (Oxford: Pergamon, 1977).

- Christensen, A.-L. *Luria's Neuropsychological Investigation: Text* (Copenhagen, Denmark: Munksgaard, 1974).
- Golden, C. J. *Diagnosis and Rehabilitation in Clinical Neuropsychology* (Second edition, Springfield, IL: Charles C. Thomas, 1981).
- Golden, C. J., Osmon, D. C., Moses, J. A., Jr., & Berg, R. A. *Interpretation of the Halstead-Reitan Neuropsychological Test Battery* (New York: Grune & Stratton, 1981).
- Heaton, R. K., Baade, L. E., & Johnson, K. L. Neuropsychological Test Results Associated with Psychiatric Disorder in Adults, *Psychological Bulletin*, 85 (1978), 141–162.
- Kaufman, A. S., & Lichtenberger, E. O. *Essentials of WAIS-III Assessment* (New York: Wiley, 1999).
- Lezak, M. D., Howieson, D. B., Loring, D. W., & Hannay, H. J. *Neuropsychological Assessment* (New York: Oxford University Press, 2004).
- Luria, A. R. *Higher Cortical Functions in Man* (Second edition, New York: Basic Books, 1980).
- Luria, A. R., & Majovski, L. V. Basic Approaches Used in American and Soviet Clinical Neuropsychology, *American Psychologist*, 32 (1977) 959–968.
- Miller, E. The Management of Dementia: A Review of Some Possibilities, *British Journal of Social and Clinical Psychology*, 16 (1977), 77–83.
- Nelson, H. E., & O'Connell, A. Dementia: The Estimation of Premorbid Intelligence Levels Using the New Adult Reading Test, *Cortex*, 14 (1978), 234–244.
- Newcombe, F. *Missile Wounds of the Brain* (Oxford, UK: Oxford University Press, 1969).
- Patterson, K. E. Neuropsychological Approaches to the Study of Reading, *British Journal of Psychology*, 72 (1981), 151–174.
- Ponsford, J. *Traumatic Brain Injury: Rehabilitation for Everyday Adaptive Living* (Hove, UK: Psychology Press, 1995).
- Powell, G. E. *Brain Function Therapy* (London: Gower, 1981).
- Prigatano, G. P. Motivation and Awareness in Cognitive Neurorehabilitation, in D. T. Stuss, G. Winocur, & I. H. Robertson, eds., *Cognitive Neurorehabilitation* (New York: Cambridge University Press, 1999).
- Rabbitt, P. (Ed.). *Methodology of Frontal and Executive Function* (Hove, UK: Psychology Press, 1997).
- Richardson, J. T. E. *Clinical and Neuropsychological Aspects of Closed Head Injury* (Second edition, Hove, UK: Psychology Press, 2000).
- Riddoch, M. J., & Humphreys, G. W. (Eds.). *Cognitive Neuropsychology and Cognitive Rehabilitation* (Hove, UK: Erlbaum, 1994).
- Rothwell, N. A., LaVigna, G. W., & Willis, T. J. A Non-Aversive Rehabilitation Approach for People with Severe Behavioural Problems Resulting from Brain Injury, *Brain Injury*, 13 (1999), 521–533.
- Russell, E. W., Neuringer, C., & Goldstein, G. *Assessment of Brain Damage: A Neuropsychological Key Approach* (New York: Wiley, 1970).
- Savage, R. D., Britton, P. G., Bolton, N., & Hall, E. H. *Intellectual Functioning in the Aged* (London: Methuen, 1973).

- Shallice, T. *From Neuropsychology to Mental Structure* (Cambridge, UK: Cambridge University Press, 1988).
- Sohlberg, M. M., & Mateer, C. A. *Cognitive Rehabilitation: An Integrative Neuropsychological Approach* (New York: Guilford Press, 2001).
- Stuss, D. T., Winocur, G., & Robertson, I. H. (Eds.). *Cognitive Rehabilitation* (New York: Cambridge University Press, 1999).
- Teuber, H.-L. Recovery of Function after Brain Injury in Man, in *Outcome of Severe Damage to the Central Nervous System*, Ciba Symposium 34, new series (Amsterdam: Elsevier, 1975).
- Tulsky, D. S., Saklofske, D. H., Chelune, C. J., Heaton, R. K., Ivnik, R. J., Bornstein, A., et al. (Eds.). *Clinical Interpretation of the WAIS-III and WMS-III: Practical Resources for the Mental Health Professional* (San Diego, CA: Academic Press, 2003).
- Vernon, P. E. *Personality Assessment* (London: Methuen, 1964).
- Wilson, B. A., Baddeley, A. D., Evans, J. J., & Shiel, A. Errorless Learning in the Rehabilitation of Memory Impaired People, *Neuropsychological Rehabilitation*, 4 (1994), 307–326.
- Wood, R. L. *Brain Injury Rehabilitation: A Neurobehavioural Approach* (London: Croom Helm, 1987).

Neuropsychiatry and Neuropsychology

Early in my career, I submitted an academic paper to a highly respected psychiatric journal on the subject of hemisphere laterality and schizophrenia. The anonymous, but presumably eminent, reviewer rejected the paper not on the merit of its contents but because it was considered that to look for abnormal brain organization in schizophrenics was an outdated and futile exercise, of no interest to current psychiatry.

In *A Pelican at Blandings*, published shortly before in 1969, P. G. Wodehouse has Galahad Threepwood say: “That was the night you were so disturbed because she hummed and giggled, giving you the impression that something had gone wrong with the two hemispheres of her brain and the broad band of transversely running fibres known as the corpus callosum and that she was, in your crisp phrase, potty.”

Wodehouse seems to have been the more prophetic of the two in view of the dramatic increase in interest in neuropsychological variables in psychiatry over the past 30 years. Neurologists and psychiatrists have always been interested in mental states that result from cerebral pathology, the area known as neuropsychiatry or organic psychiatry, but this interest has now been extended to disorders that would not traditionally fall within this field, such as schizophrenia and affective psychosis. Neuropsychiatry has now grown into an independent medical specialty, standing astride psychiatry and clinical neuropsychology.

NEUROPSYCHIATRY

It has been clear for a very long time that abnormal mental states can follow from damage to the brain. Some of the changes associated with focal lesions that were discussed in Part II are obviously of a psychiatric nature, and there are in addition changes that follow from diffuse brain diseases, and from generalized trauma associated with blows to the head.

It should, however, be remembered that not all neurological conditions involving the brain produce psychiatric symptoms. In a large study of more than 3,500 war veterans with brain injuries, about a third of whom had penetrating wounds, lasting cognitive impairment was present in only 2%, character changes in 18%, psychosis in 8%, and severe neurosis in 11% (Hillbom, 1960). The incidence of mental changes following the development of tumors is rather higher, in the range of approximately 50% to 80% among patients in various studies, but on the other hand the incidence of cognitive and psychiatric sequelae after closed head injuries producing concussion and temporary amnesia is remarkably low. Such cases do nevertheless occur.

The variety of psychiatric conditions associated with organic states is a special area of study in its own right, particular patterns of psychiatric impairment being associated with, for example, the various cerebral diseases (see David, Fleminger, Kopelman, Lovestone, & Mellers, 2008). However, there are a number of general symptoms that are often seen to accompany organic nervous disease. Patients frequently show some disturbance of consciousness, varying from mild inattention to disorientation, delirium, unconsciousness, and coma. Sleep may also be disturbed, either in quantity, in pattern, or in the electrical rhythms recorded through the night. Impairment of memory is also common, and patients tire easily and tend to perseverate in their thoughts or their activity. They are often irritable and exhibit frequent changes in emotion, and their behavior may also be relatively disinhibited.

Some of these symptoms have been found to be associated in the *postconcussional syndrome* (sometimes called posttraumatic neurosis), which is characterized by severe headache, giddiness and blackouts, fatigue and difficulty in concentrating, loss of confidence, depression, anxiety, irritability, and intolerance of noise. Patients may also complain of insomnia and show a reasonable but particular concern for, and awareness of, their head.

Epilepsy might also be expected to be associated with psychiatric disorders. However, although epilepsy is a common aftereffect of cerebral trauma or disease, epileptics in general show relatively low rates of psychiatric disorder. Studies of the incidence of mental abnormality in epileptics commonly find that only about 5% are ever diagnosed as suf-

fering from a serious psychiatric condition. This compares with a rate of about 1% for the general population. There does, however, seem to be a much clearer relationship between temporal lobe epilepsy and psychiatric disorder. This form is found disproportionately among epileptics admitted to mental health facilities.

The abnormal behavior can take the form of psychiatric symptoms following seizures, or less commonly of a chronic psychotic state rather similar to paranoid schizophrenia. A particular form of personality, typified by stubbornness, concreteness, lack of spontaneity, egocentricity, and unctuousness has also been described as typical of epileptics. However, these descriptions were based upon institutionalized epileptics, not those living in the general community (as almost all are), and it seems probable that any personality changes that do occur can be regarded as secondary reactions to being handicapped by epilepsy, rather than as primary effects mediated by changes in cerebral systems.

Cerebral *dementia* is, of course, invariably accompanied by psychiatric changes. Dementia, seen most commonly in older people but also in certain presenile degenerative diseases, involves the generalized and progressive loss of cortical tissue from the brain. Most dementias are, at present, irreversible. As the cortex is lost, mental functions decline progressively. There is a global deterioration of memory, thinking, motor performance, emotional responsiveness, and social behavior. As the illness develops, speech may be affected, behavior becomes increasingly unreasonable and often disruptive, until finally the control of basic and vital processes becomes disorganized. It is a distressing and pathetic progression familiar to many with aging relatives.

Finally, of course, it should be remembered that there are a range of specific psychiatric effects that can follow damage to the brain, and these will depend upon the site of the lesion, its speed of development, the nature of the pathology, and particular characteristics of the individual patient.

There are also certain rather bizarre neuropsychiatric conditions that may follow head injury. These conditions are generally described as rare, and they are certainly uncommon, but their apparent rarity is, in my experience, partly attributable to the reluctance of individuals with head injuries to report these problems; they are afraid of being regarded as “mad” and it often takes time to gain the trust of the patient before the symptoms are described. As these syndromes are striking, and have recently been of particular interest to neuropsychologists, four typical conditions will briefly be described: Capgras, Cotard, and Frégoli syndromes, and obsessive–compulsive disorder. A fuller account, with case studies, can be found in Halligan and Marshall (1996).

In *Capgras syndrome* the patient believes that imposters have replaced family members and friends. These imposters behave exactly

like the people who they have replaced, and are essentially indistinguishable from them. While this is a rare feature of a rare condition, there are cases in which the patient has threatened, harmed, or even killed the person they believe to be an imposter, as a way of trying to unmask the perceived deception. While not all cases of Capgras syndrome are linked to neurological disease, up to half the cases are, and in particular abnormalities of the right hemisphere have been reported in these patients. Ellis and Young (1990) have provided a cognitive interpretation of this syndrome, and a more recent review can be found in Ellis and Lewis (2001). The syndrome can be surprisingly disabling. A teacher whom I saw after a head injury sustained in a road traffic accident experienced these delusions not only with respect to members of her family but also her colleagues at school, and was quite unable to sustain a return to work because of the distress which it caused to her.

Cotard syndrome appears rather similar to Capgras syndrome and, in my experience, the two can occur together. Cotard syndrome is often described as a syndrome of nihilism. Patients describe the feeling that they have died, and may also have the delusion that their bodies have been replaced by corpses. A complaint may be made of having lost possessions, status, or strength, or that certain internal organs are missing. Right hemisphere lesions are also associated with Cotard syndrome and, as with Capgras syndrome, there may be impairments of face processing. Some patients with Cotard syndrome may be clinically depressed, and a distinction is sometimes drawn between two forms of the disorder: one form linked to severe depressive illness, and one form based upon a delusional state.

Another form of delusional misperception occurs in *Frégoli syndrome*. Commonly, there is a paranoid element to this condition and patients identify an individual whom they believe to be persecuting them. This paranoid delusion is, however, extended to the idea that this person can disguise him- or herself to appear like other people with whom the patient comes into contact, usually for a sinister purpose. Despite the lack of any similarity between the (believed) persecutor and the person believed to be the persecutor-in-disguise, the patient believes them to be the same person. This syndrome can occur in association with degenerative conditions and also, transiently, in toxic states including cannabis use. A cognitive interpretation for Frégoli syndrome is also offered in Ellis and Young (1990).

Obsessive-compulsive disorder (OCD) is less rare following brain injury, and obsessional features that fall short of a full psychiatric diagnosis are not at all uncommon, especially after frontal brain injury. Often, the patient has a previous tendency to mild obsessiveness that is exacerbated by the head injury. Excessive orderliness, a concern with

cleanliness, a fixed routine, and excessive checking behavior are the common features. However, severe compulsions can sometimes occur, and I have seen patients who cannot sleep because they feel compelled to continually check that the door is locked or the gas stove turned off. Very rarely, the OCD may take a form that includes violent compulsive thoughts. Such thoughts are of too grotesque, macabre, and disturbing a nature to be described here, but one of mildest images described to me by a patient was that as we sat and talked he could not stop himself from imagining that he was removing all the skin from my arm with a cheese grater. These thoughts that intrude into everyday consciousness occur in individuals without previous violent tendencies or interests and are typically so distressing that they avoid all social interaction, either staying alone within their home, or moving to a remote and isolated location where they are unlikely to meet other people. For a neuropsychological account, see Tallis, Pratt, and Jamani (1999); for an overview of these and other similar disorders, see O'Carroll (2003). The neurological basis of delusions, more generally, is discussed by Gilleen and David (2005), and the role of the frontal lobes in Salloway, Malloy, and Duffy (2001).

This, very briefly, is the subject matter of organic psychiatry, and is composed of *organic* states or reaction types. In traditional psychiatry these have always been distinguished from *functional* states. Although the distinction is not entirely clear-cut, functional states are those where no clear physical pathology can be established. Whether this is because they do not follow from physical pathology, or because we are ignorant of the physical basis of each condition, is a matter for debate, but the distinction has nevertheless been traditionally maintained. Functional disorders have generally been accepted to include the major psychotic states of schizophrenia and bipolar affective illness. However, there has been a need to reexamine this distinction, particularly in the light of new evidence suggesting that abnormal neuropsychological organization might be shown to be typical of certain of these disorders.

A word should be said here about the psychiatric terms referred to in this chapter. A distinction is usually made between neuroses and psychoses. In the *neuroses*, which are less severe and usually require no more than outpatient treatment, the patient maintains contact with reality and has some insight into his or her problems. The symptoms are likely to be anxiety, depression, or avoidance behavior, but without hallucinations or extreme deviations in thought, mood, or action. *Psychoses* are more serious and generally require admission to a hospital. The symptoms may be primarily in mood (manic-depressive or affective psychosis, mania being intense excitement and overactivity, depression a state of mute, expressionless, and perhaps agitated withdrawal) or in changes in thought or action (hallucinations, delusions, bizarre behavior) in states usually termed schizophrenic. These psychotic patients gen-

erally have little contact with reality and limited awareness of their own condition. (If you feel uncertain about the distinctions being made here, it would be worth reading the section on abnormal psychology in an introductory psychology text.)

FUNCTIONAL STATES AND LATERALITY

Some general points should be made before considering in detail the studies of lateral cerebral organization in psychiatric patients. The first is that a rather simple model of psychiatric disorder is often assumed, in which mental illness is seen as exactly like a physical disease. It has long been recognized that there are problems with this view, particularly in accounting for the effects that psychological and social variables have upon abnormal behavior. Secondly, although considerable advances have been made in recent years, there is still considerable unreliability in the use of psychiatric diagnostic labels. Some terms, particularly *schizophrenia*, are rather loosely used and encompass a variety of rather different disorders. Thirdly, scientific rigor is lacking in many of the studies. Clinical studies are difficult to conduct, but in comparison with the experimental studies discussed in Part III, research with psychiatric patients has been methodologically of poor quality. Little care has been taken over the specification of patient groups, over the measurement of cerebral asymmetries, and over the interpretation of data collected. A variety of experimental findings have been squeezed uncomfortably into a mold formed by the researchers' expectations. The findings, and the strong claims made for them, must be treated with some caution, although the research questions remain of considerable interest and importance.

Studies of lateral cerebral organization in psychiatric patients seem to have been undertaken in a number of laboratories at about the same time, but one important influence was the work of Flor-Henry. Early on he observed that different psychiatric symptoms appeared to be associated with left- and right-side temporal lobe epilepsy. A great variety of forms of evidence are now quoted in support of laterality differences in functional psychiatric states, but many of the early observations were also of this kind, concerned with lateralized organic states or lateralized symptoms and signs.

Lateralized Symptoms

Among lateralized signs in psychiatric patients are conversion symptoms, in which disturbance is expressed by some overt physical manifestation, such as hysterical paralysis or loss of sensation. Such "conver-

sions” have been reported to be found more commonly on the left side of the body than on the right, especially in females (Galín, Diamond, & Braff, 1977), although in another study, of 759 cases with conversion symptoms, only 29 showed lateralization, and among these, 12 were left- and 17 right-sided (Bishop, Mobley, & Farr, 1978). Other studies have found at best only marginal significance for a left-side preponderance of psychogenic symptoms. Fleminger, McClure, and Dalton (1980) used an interesting suggestion test to show that suggestion would operate more powerfully upon left-sided responses. They did indeed find such an effect, not only for the psychiatric patients they studied, however, but also for the psychiatric nurses they used as controls.

Pain has also been reported to be more frequent or intense on the left side of the body, especially when it can be considered psychogenic in origin. The inference is that the increased pain is associated with a more “emotional” right hemisphere. However, some of the most careful studies have failed to find any asymmetry, in particular a study of a large sample of patients attending a pain clinic, using data that were not collected with any intention of later analysis for lateral asymmetry (Hall, 1981).

An alternative approach has been to examine the performance of psychiatric patients on standard neuropsychological test batteries to see what kind of organic dysfunction such performance might suggest. Flor-Henry, Fromm-Auch, Tapper, and Schopflocher (1981), used a Reitan-type battery with hysterical and schizophrenic patients. The results were complex, but it was argued that they supported a model of bilateral frontal pathology being associated with hysteria, but with greater importance being placed upon a dominant (left) hemisphere dysfunction. Hysteria, it was argued, can be viewed as a special form of schizophrenia in this context. Similar results, of schizophrenia being linked with dominant hemisphere dysfunction, particularly of the temporal and temporal-parietal regions, were also found by Taylor and co-investigators using an aphasia test and a neuropsychological test battery (Taylor, Greenspan, & Abrams, 1979; Taylor, Redfield, & Abrams, 1981). Not only were Kronfol, Hamsher, Digre, and Waziri (1978) able to show, using standard tests, a right hemisphere abnormality in depressed patients before treatment, but they were also able to show that unilateral ECT (see p. [old 258]), administered to either side of the head, improved “right hemisphere” performance on the tests.

In this context, a study by Hommes and Panhuysen (1971) using the Wada technique with depressed patients has been much quoted. They found that there was a negative relationship between the depth of depression and the degree of left hemisphere speech dominance. They also found that the injection of sodium amytal resulted in improved

mood, whether the injection was to the left or right, but especially when it was on the left. They concluded that normal mood regulation depends on the integrity of left hemisphere functional dominance, although their results have also been taken by others to implicate a right hemisphere dysfunction in depressed mood.

While research into lateralized symptoms in neuropsychiatric disorders has declined in recent years, a more recent review can be found in Green, Sergi, and Kern (2003).

Physiological Processes

The study of ECT has already been mentioned above and in the context of laterality research, and the findings can be turned around to provide evidence for lateralized brain dysfunction in depressive conditions.

A second physiological approach has been to look at electrophysiological parameters. It has long been known that psychiatric patients may show mild abnormalities in their EEG. Abrams and Taylor (1980), for instance, found 44 of 159 psychiatric patients with functional psychoses to have clinical EEG abnormalities, and these were more commonly observed in the left temporal region, and in those patients who had “formal thought disorder.”

Some studies have taken a more systematic approach to the study of ongoing EEG, and Flor-Henry, Yeudall, Koles, and Howarth (1979) found atypical brain function in the left frontal region in obsessive-compulsive patients. Similar results, of greater left hemisphere variability of alpha in paranoid schizophrenia and greater right hemisphere variability in depression, have also been reported (Rochford, Weinapple, & Goldstein, 1981). Coherence studies (see p. 268) have yielded rather complex results, but have if anything tended to suggest a deficit in interhemispheric integration, rather than a lateralized dysfunction in one of the hemispheres (Shaw, Brooks, Colter, & O'Connor, 1979; Weller & Montagu, 1979). The theme of functional disconnection as demonstrated by EEG coherence has been extended by Higashima and colleagues (Higashima et al., 2007), who specifically identified a disconnection between the frontal and temporal lobes of the left hemisphere as being responsible for acute psychotic symptoms in schizophrenia.

As might be expected, evoked potentials (see p. 269) have been studied as well as on-going EEG. Some studies have supported a left hemisphere dysfunction in schizophrenia (Roemer, Shagass, Straumanis, & Amadeo, 1978; Shagass, Roemer, Straumanis, & Amadeo, 1980), although this last study also found evidence of a similar left hemisphere dysfunction for depression, rather than the right-sided dysfunction that was expected. The hypothesis of a failure in interhemispheric communi-

cation was supported by the study of Jones and Miller (1981) examining somatosensory evoked potentials, which found no difference in the latency of components following ipsilateral or contralateral stimulation. This is an area of research from which it is often difficult to draw conclusions given the complexity of the findings, but a useful review may be found in Gruzelier, Galderisi, and Strik (2002).

The modern imaging techniques (see Chapter 13) have also begun to make a contribution to an understanding of neuropsychiatric conditions. A PET study of medicated schizophrenics identified an abnormal laterality of the supplementary motor area as responsible for auditory verbal hallucinations (“hearing voices”), interpreted as a failure to correctly attribute speech generated by the brain to the self (Stephane et al., 2006). More generally, left hemisphere abnormalities in brain function in schizophrenia, determined by a variety of imaging methods, have been described by Gur and Chin (1999), and links with neurotransmitter systems proposed by Doty (2003).

MRI studies have also established that there is a greater asymmetry of the Sylvian fissure in schizophrenia characterized by hallucinations and thought disorder (Shapleske, Rossell, Simmons, David, & Woodruff, 2001). Brambilla and colleagues (Brambilla et al., 2007), also using MRI, found an abnormally reduced and inverse hemisphere cerebral blood volume in a large group of patients with schizophrenia, and they argued that abnormal distribution of the blood within the brain may not only be associated with schizophrenia, but account as well for the brain atrophy and cognitive deficits also seen in this group.

Finally, it seems relevant to note two drug studies. Carr (1978) has reported evidence that piracetam, which is known to facilitate inter-hemispheric neural transmission, had the effect of improving certain aspects of schizophrenic performance, including scores on certain memory and learning tasks. Chlorpromazine, which is one of the major tranquilizers used in the control of schizophrenic symptoms, has also been shown to decrease lateral asymmetry in auditory temporal discrimination (Hammond & Gruzelier, 1978).

None of this evidence seems of great significance taken alone, but it all contributes to a general picture of disturbed cerebral organization that might characterize some psychiatric patients.

Experimental Tasks

Beaumont and Dimond (1973) were the first to use the divided visual field technique with psychiatric patients. They examined within- and between-hemisphere matching using a variety of stimuli and found evidence for some degree of lateralized dysfunction, but also for a more sig-

nificant deficit in interhemispheric communication. Some later researchers have found a similar effect (Pic'l, Magaro, & Wade, 1979), but others have found clearer evidence for a specific lateralized deficit (Gur, 1978; Tucker, Antes, Stenslie, & Earnhardt, 1978). Colbourn (1982) has thoroughly reviewed the divided visual field studies with psychiatric patients and concludes that it is possible to interpret the findings in terms of a left hemisphere dysfunction being associated with schizophrenia, although the findings for other conditions are much less clear. However, he goes on to point out that other interpretations of the evidence should also be examined.

Dichotic listening has been employed rather more often than the visual techniques. Here again the evidence has been rather confused. However, it again falls roughly into two areas. There are those who argue for a lateralized deficit from the ear differences that have been obtained. Bruder and Yozawitz (1979) found evidence for a right hemisphere abnormality in patients with affective disorders, but no evidence of an abnormality in schizophrenia. With a similar group of patients, an abnormal pattern of lateralization, which returned toward normal with ECT treatment, was also found by Moscovitch, Strauss, and Olds (1981). Schizophrenics were found to have an enhanced right ear advantage in the study of Nachshon (1980), and Tucker et al. (1978) also found a right ear attentional bias in patients with anxiety syndrome. Findings from dichotic listening were also considered alongside other sources of data by Mitchell and Crow (2005). Their thinking focused on the role of language functions in the right hemisphere for successful social communication, and the idea that left and right hemisphere functions were inadequately segregated in psychosis. Their hypothesis is that language functions supported by the right hemisphere are essential to accurately interpreting the intent of an interaction, and that a deficit in these functions in schizophrenics may be the basis of their problems with social interaction.

Other researchers have interpreted their findings differently. Lishman, Toone, Colbourn, McKeekan, and Mance (1978), studying recently recovered psychotic patients, found evidence of abnormal auditory laterality but concluded that this was more typical of an interhemispheric integration deficit than of a left hemisphere dysfunction. Against the general trend, Wexler and Heninger (1979) found increasing laterality to be associated with a decreasing probability of psychotic thought and behavior, so that they formulated their view of the schizophrenic deficit in terms of a failure of interhemispheric inhibition. Lastly, Walker, Hoppes, and Emory (1981) have reanalyzed the data of Nachshon's study, which they argue rather support a hypothesis of faulty interhemispheric transfer.

Any conclusion with respect to auditory laterality has been considerably complicated by the finding of Gruzelier and Hammond (1979) of asymmetries in hearing thresholds, with diurnal variation, in schizophrenic patients.

There are also studies of lateral performance asymmetry in the tactile modality, but they all point toward a deficit in intermanual transfer and integration, rather than specific lateralized deficits (Carr, 1980; Dimond, Scammell, Pryce, Huws, & Gray, 1980).

A relatively high level of agreement has also been shown by studies of lateral eye movements. These have, in general, found a tendency for an increase in rightward conjugate lateral eye movements (or a reduction in left movements) in schizophrenia (Gur, 1978; Schweitzer, 1979; Tomer, Mintz, Levi, & Myslobodsky, 1979; Tucker et al., 1978). The interpretation placed upon these findings has generally been that this demonstrates the presence of some abnormal process originating from the dominant left hemisphere.

It should also be noted that there have been reports of abnormal distributions of hand preference within psychiatric groups. The overall results have been, however, somewhat unclear. Some investigators (Gur, 1977; Lishman & McMeekan, 1976) have reported an increase in left handedness among schizophrenic patients. Alternately, some have found an excess of right handers among these patients (Taylor, Dalton, & Fleming, 1980). There are undoubtedly difficulties in assessing the handedness of psychiatric patients by questionnaire measures, and this may contribute to some of the variability in the findings.

By now you may feel thoroughly confused by the variety of studies and the diversity of the findings. A whole range of different methods of investigation has been used with all kinds of different patients, and the result has been a very complex set of research data. This rather selective survey of the studies, with rather more references than usual, has been presented in order to illustrate just how difficult it can be to make sense of some particular field of investigation. However, we must try and make what sense we can out of the research, and it seems useful to do this in the light of the two major theoretical hypotheses that have been adopted.

THEORETICAL MODELS

Three types of model have been proposed to link lateral asymmetry with functional psychiatric disorder. The first was proposed by Galin (1974, 1977), and is the only one to be based upon *psychodynamic* concepts. Galin derived his ideas from split-brain research and argued that psychi-

atric states might be likened to the activity of the disconnected right hemisphere. Specifically, the right hemisphere could be associated with the Freudian “primary process” and by its relative disconnection from the conscious, speaking left hemisphere could be the source of unconscious processes and repression. This is an intriguing theory, but partly because a psychodynamic approach is not fashionable in contemporary psychiatry, certainly among those psychiatrists likely to be interested in biological correlates of mental illness, relatively little attention has been paid to Galin’s ideas.

The other two theoretical models will probably already be apparent from the sketch of the research evidence given above. These are of *specific lateralized cerebral dysfunction* and of *impaired interhemispheric integration*. The concept of a specific dysfunction has been more actively promoted, especially by Flor-Henry (Gruzelier & Flor-Henry, 1979; Flor-Henry, 1978, 1979). Flor-Henry based his theories on extremely diverse evidence, of the kind that has been cited already, and while the theory is fairly complex, it boils down to the idea that functional disturbances can be linked with dysfunction of the frontal and temporal regions of the brain. If the abnormality is on the dominant side, then the illness will be of a schizophrenic nature. If the abnormality is on the nondominant side, then the disorder will be an affective one with symptoms of depression more likely than symptoms of mania. Flor-Henry, supported by others, argues quite directly that the research evidence unequivocally supports his theoretical model.

The alternative model, which stems from the work of Dimond (Beaumont & Dimond, 1973; Dimond, 1980; Dimond et al., 1980), proposes that certain functional states, principally schizophrenia, can be viewed as a disconnection syndrome. The psychiatric symptoms are associated with a failure of interhemispheric transfer across the corpus callosum. In this it reflects Galin’s model, but is formulated in terms of information-handling processes rather than psychodynamic mechanisms. It has not been claimed that such a model can explain all the evidence that is sometimes cited for neuropsychological effects in psychiatric illness, but it is proposed as a more satisfactory explanation of many of the findings obtained by using well-controlled experimental tasks with schizophrenic patients. As we have seen, the results of a number of studies provide clear support for such a model.

A variant on the disconnection model is the *valence* model proposed by Davidson and colleagues (Davidson & Irwin, 1999). Their idea is that the anterior parts of the brain, together with the amygdala, on the left side support positive emotions and approach behavior, while the same structures on the right support negative emotions and withdrawal behavior. Damage to one or the other frontal lobe will disturb the bal-

ance between the two systems through a loss of inhibition by one hemisphere over the other. The evidence for this hypothesis has been reviewed by Andrewes (2001), and is derived not only from experimental tasks but also from stroke patients. Some of the evidence is particularly persuasive; the study of patients administered unilateral sodium amytal (the Wada technique; see p. 86) found depressed mood to be associated with left sided suppression, while elated mood was found after right sided suppression. However, drawing conclusions from studies of patients with strokes is far from straightforward, and the issue is complicated by the possibility of either denial or alexithymia, the faulty perception of one's own emotions.

Before assessing the relative validity of these models, one or two methodological and analytical points should be noted. In this book it has often been necessary to stand back and consider methodological issues before assessing the value of any conclusions that might be drawn, and it often seems that the methodological difficulties overshadow the significance of the outcome of the research. This is simply part of the nature of neuropsychology at present, and whether it stems from the immaturity of this field of research or the complexity of the processes under investigation is difficult to say.

There are a whole host of procedural problems that I do not intend to spell out at length. These include the difficulties of undertaking laterality research and of obtaining laterality measurements, which have been discussed in preceding chapters. Added to these are the problems of undertaking clinical studies, and particular problems associated with psychiatric research. It is difficult to standardize the diagnostic procedures employed to classify groups of psychiatric patients; in addition many have been institutionalized for varying periods, and almost all are being treated with a variety of powerful drugs acting upon the central nervous system. These problems have been extensively discussed in a number of reviews (Colbourn, 1982; Marin & Tucker, 1981; Merrin, 1981).

One problem that cannot go undiscussed, however, is the difficulty of interpreting abnormal lateral asymmetries. This is particularly relevant when considering a model of specific lateralized dysfunction. The problem is that once the concepts of interhemispheric inhibition or overactivation are introduced into the discussion of research findings, then almost any model of lateralized dysfunction can be supported by post hoc interpretation of the results. Let me take two studies as examples. This is not to spotlight these studies as examples of bad science—in fact both seem well conducted and yield valuable data—but they are useful to illustrate the problems that are frequently encountered in interpretation of the findings.

Gruzelier and Hammond (1980) examined schizophrenic performance in a dichotic listening task. Among other findings they found that in the serial position effect with digit pairs of equal loudness, there was a tendency for the left ear to lack primacy in report. They discussed this as evidence in support of the expected *left* hemisphere processing impairment. Their argument was, roughly, that as the reporting processes rely upon verbal encoding, which can be considered a left hemisphere activity, then even though the effect was at the left ear, it demonstrated a left hemisphere impairment. They may be correct, and I have taken their result out of the context of their other findings, but it still seems that the result could as easily be taken as direct evidence of an alternative right hemisphere deficit.

In a second study, Gur (1979) asked subjects with schizophrenia to judge pairs of altered pictures that were presented simultaneously or successively. Control subjects did better overall, and were equally fast in the simultaneous and successive conditions. The patients with schizophrenia were faster in the successive than the simultaneous condition. Gur argues that this pattern of performance is typical of subjects with right brain damage, which must therefore be evidence for *left* hemisphere overactivation in schizophrenia. Even assuming that the parallel with brain damage allows a valid inference to be drawn, the conclusion might obviously just as well be that people with schizophrenia have a defective right hemisphere.

So what conclusions may be drawn about laterality and functional psychiatric states? Of the scholarly reviews, one concludes that despite the methodological difficulties, there is consistent support for a model of left hemisphere overactivation or dysfunction in schizophrenia (Newlin, Carpenter, & Golden, 1981). Three other reviews, however, are driven to the view that there is no consistent support for any of the models proposed, and each makes certain recommendations that are designed to improve the quality of research in this field (Marin & Tucker, 1981; Merrin, 1981; Wexler, 1980).

My own view continues to be that this last position is the only possible conclusion at the present. Many of the theoretical discussions have been far too selective in the evidence that they have considered, and most have indulged quite shamelessly in post hoc interpretation that has been in line only with the hypotheses being promoted. The basic research evidence is still too sketchy to allow any clear conclusions to be drawn, and it is certainly much too early to proclaim that functional psychiatric states can be explained in terms of (a particular model of) abnormal neuropsychological processes. There are a variety of other psychological models of psychotic behavior, for instance in terms of an attentional disturbance associated with heightened arousal, and many of

these models are at least as powerful in explaining the data as are the neuropsychological theories. Whether neuropsychological models will come to reflect models cast in other conceptual terms, and derived from different levels of behavioral analysis, remains to be seen, but the thought undoubtedly provides at least one avenue for future research. What is now established without reasonable doubt is that there is a neuropsychological dimension to many psychiatric disorders, even if the precise mechanisms that underlie the dysfunction have yet to be clarified.

CONCLUSION

Neuropsychology has been able to make an increasingly valuable contribution to the study of the mental sequelae of organic pathological processes in the area known as neuropsychiatry or organic psychiatry. The psychiatric effects of gross damage to the brain have long been recognized, and neuropsychology is able to clarify and illuminate the processes involved by developing its own models of the processes that underlie brain–behavior relationships.

Almost no clear conclusions have emerged from the upsurge of interest in the neuropsychological concomitants of functional psychiatric states. There has been a refreshing reevaluation of the processes that might contribute to schizophrenic and affective psychotic disorders. Many exciting theoretical models have been formulated, but despite the growing research effort, it is still too early to say whether a particular neuropsychological dysfunction can be identified as accompanying a given functional state. This area is nevertheless one of the most exciting and challenging of any in contemporary neuropsychology.

The problems and difficulties that arise when we attempt practical applications of neuropsychology illustrate not only the fundamental deficiencies of our knowledge but also the importance of understanding how physical systems generate the highest levels of thinking, feeling, and consciousness. We have made considerable progress in unraveling the nature of brain–behavior processes, and yet there is much still to be discovered, not only about the details of functional relationships, but about the fundamental principles that govern the operation of those functions. To discover the answers involves an engagement with essential philosophical issues, a rigorous and creative approach to experimental design, and a questioning and critical appraisal in the construction of theories. The answers—and they will be found—will be of fundamental importance for our understanding, not only of disordered behavior, but also of everyday human action, and the essence of the humanity of humankind.

FURTHER READING

On the topic of neuropsychiatry, a readable general introduction and an excellent reference source are, respectively:

Cummings, J. L., & Trimble, M. R. *Concise Guide to Neuropsychiatry and Behavioural Neurology* (Second edition, Washington, DC: American Psychiatric Publishing, 2002).

David, A. S., Fleminger, S., Kopelman, M., Lovestone, S., & Mellers, J. *Lishman's Organic Psychiatry: A Textbook of Neuropsychiatry* (Oxford, UK: Blackwell, 2008).

A more neuropsychologically oriented discussion, with case examples, is:

Halligan, P. W., & Marshall, J. C. (Eds.). *Method in Madness: Case Studies in Cognitive Neuropsychiatry* (Hove, UK: Psychology Press, 1996).

Further reading on laterality and functional disorders is to be found in:

Gruzelier, J. H., & Flor-Henry, P. *Hemisphere Asymmetries of Function in Psychopathology* (Amsterdam: Elsevier/North Holland, 1979).

Hugdahl, K., & Davidson, R. J. (Eds.). *The Asymmetrical Brain* (Cambridge, MA: MIT Press, 2003).

Marin, R. S., & Tucker, G. J. Psychopathology and Hemispheric Dysfunction: A Review, *Journal of Nervous and Mental Disease*, 169 (1981), 546–557.

Merrin, E. L. Schizophrenia and Brain Asymmetry: An Evaluation of Evidence for Dominant Lobe Dysfunction, *Journal of Nervous and Mental Disease*, 169 (1981), 405–416.

REFERENCES

Abrams, R., & Taylor, M. A. Psychopathology and the EEG, *Biological Psychiatry*, 15 (1980), 871–878.

Andrewes, D. G. *Neuropsychology: From Theory to Practice* (Hove, UK: Psychology Press, 2001).

Beaumont, J. G., & Dimond, S. J. Brain Disconnection and Schizophrenia, *British Journal of Psychiatry*, 123 (1973), 661–662.

Bishop, E. R., Mobley, M. C., & Farr, W. F. Lateralization of Conversion Symptoms, *Comprehensive Psychiatry*, 19 (1978), 393–396.

Brambilla, P., Cerini, R., Fabene, P. F., Andreone, N., Rambaldelli, G., Farace, P., et al. Assessment of Cerebral Blood Volume in Schizophrenia: A Magnetic Resonance Imaging Study, *Journal of Psychiatric Research*, 41 (2007), 502–510.

Bruder, G. E., & Yozawitz, A. Central Auditory Processing and Lateralization in Psychiatric Patients, in J. Gruzelier & P. Flor-Henry, eds., *Hemisphere Asymmetries of Function in Psychopathology* (Amsterdam: Elsevier/North Holland, 1979).

- Carr, S. A. The Effects of Piracetam (UCB 6225) on Interhemispheric Transfer and Memory in Chronic Schizophrenics, *Bulletin of the British Psychological Society*, 31 (1978), 64–65.
- Carr, S. A. Interhemispheric Transfer of Stereognostic Information in Chronic Schizophrenics, *British Journal of Psychiatry*, 136 (1980), 53–58.
- Colbourn, C. J. Divided Visual Field Studies of Psychiatric Patients, in J. G. Beaumont, ed., *Divided Visual Field Studies of Cerebral Organisation* (London: Academic Press, 1982).
- David, A. S., Fleminger, S., Kopelman, M., Lovestone, S., & Mellers, J. *Lishman's Organic Psychiatry: A Textbook of Neuropsychiatry* (Oxford, UK: Blackwell, 2008).
- Davidson, R. J., & Irwin, W. The Functional Neuroanatomy of Emotion and Affective Style, *Trends in Cognitive Science*, 3 (1999), 11–21.
- Dimond, S. J. *Neuropsychology* (London: Butterworth, 1980).
- Dimond, S. J., Scammell, R., Pryce, I. J., Huws, D., & Gray, C. Some Failures of Intermanual and Cross-Lateral Transfer in Chronic Schizophrenia, *Journal of Abnormal Psychology*, 89 (1980), 505–509.
- Doty, R. W. Interhemispheric Abnormalities in Schizophrenia and Their Possible Etiology, in E. Zaidel & M. Iacoboni, eds., *The Parallel Brain: The Cognitive Science of the Corpus Callosum* (Cambridge, MA: MIT Press, 2003).
- Ellis, H. D., & Lewis, M. B. Capgras Delusion: A Window on Face Recognition, *Trends in Cognitive Science*, 5 (2001), 149–156.
- Ellis, H. D., & Young, A. W. Accounting for Delusional Misidentifications, *British Journal of Psychiatry*, 157 (1990), 239–248.
- Fleminger, J. J., McClure, G. M., & Dalton, R. Lateral Response to Suggestion in Relation to Handedness and the Side of Psychogenic Symptoms, *British Journal of Psychiatry*, 136 (1980), 562–566.
- Flor-Henry, P. Gender, Hemispheric Specialisation and Psychopathology, *Social Science and Medicine*, 12 (1978), 155–162.
- Flor-Henry, P. On Certain Aspects of the Localisation of the Cerebral Systems Regulating and Determining Emotion, *Biological Psychiatry*, 14 (1979), 677–698.
- Flor-Henry, P., Fromm-Auch, D., Tapper, M., & Schopflocher, D. A Neuropsychological Study of the Stable Syndrome of Hysteria, *Biological Psychiatry*, 16 (1981), 601–626.
- Flor-Henry, P., Yeudall, L. T., Koles, Z. J., & Howarth, B. G. Neuropsychological and Power Spectral EEC Investigations of the Obsessive–Compulsive Syndrome, *Biological Psychiatry*, 14 (1979), 119–130.
- Galín, D. Implications for Psychiatry of Left and Right Cerebral Specialization, *Archives of General Psychiatry*, 31(1974), 572–583.
- Galín, D. Lateral Specialization and Psychiatric Issues, *Annals of the New York Academy of Sciences*, 299 (1977), 397–411.
- Galín, D., Diamond, R., & Braff, D. Lateralization of Conversion Symptoms: More Frequent on the Left, *American Journal of Psychiatry*, 134 (1977), 578–580.
- Gilleen, J., & David, A. S. The Cognitive Neuropsychiatry of Delusions: From

- Psychopathology to Neuropsychology and Back Again, *Psychological Medicine*, 35 (2005), 5–12.
- Green, M. F., Sergi, M. J., & Kern, R. S. The Laterality of Schizophrenia, in K. Hugdahl & R. J. Davidson, eds., *The Asymmetrical Brain* (Cambridge, MA: MIT Press, 2003).
- Gruzelier, J., & Flor-Henry, P. *Hemisphere Asymmetries of Function in Psychopathology* (Amsterdam: Elsevier/North Holland, 1979).
- Gruzelier, J. H., Galderisi, S., & Strik, W. Neurophysiological Research in Psychiatry, in I. J. J. Lopez, W. Gaebel, M. Maj, & N. Sartorius, eds., *Psychiatry as a Neuroscience* (New York: Wiley, 2002).
- Gruzelier, J. H., & Hammond, N. V. Gains, Losses and Lateral Differences in the Hearing of Schizophrenic Patients, *British Journal of Psychology*, 70 (1979), 319–330.
- Gruzelier, J. H., & Hammond, N. V. Lateralized Deficits and Drug Influences on the Dichotic Listening of Schizophrenic Patients, *Biological Psychiatry*, 15 (1980), 759–779.
- Gur, R. E. Motoric Laterality Imbalance in Schizophrenia: A Possible Concomitant of Left Hemispheric Dysfunction, *Archives of General Psychiatry*, 34 (1977), 33–37.
- Gur, R. E. Left Hemisphere Dysfunction and Left Hemisphere Overactivation in Schizophrenia, *Journal of Abnormal Psychology*, 87 (1978), 226–238.
- Gur, R. E. Cognitive Concomitants of Hemispheric Dysfunction in Schizophrenia, *Archives of General Psychiatry*, 36 (1979), 269–277.
- Gur, R. E., & Chin, S. Laterality in Functional Brain Imaging Studies of Schizophrenia, *Schizophrenia Bulletin*, 25 (1999), 141–156.
- Hall, W. D. Does Pain Occur More on the Left?, *Bulletin of the British Psychological Society*, 34 (1981), 23.
- Halligan, P. W., & Marshall, J. C. (Eds.). *Method in Madness: Case Studies in Cognitive Neuropsychiatry* (Hove, UK: Psychology Press, 1996).
- Hammond, N. V., & Gruzelier, J. H. Laterality, Attention and Rate Effects in the Auditory Temporal Discrimination of Chronic Schizophrenics: The Effect of Treatment with Chlorpromazine, *Quarterly Journal of Experimental Psychology*, 30 (1978), 91–103.
- Higashima, M., Takeda, T., Kikuchi, M., Nagasawa, T., Nirao, N., Oka, T., et al. State-Dependent Changes in Intra-hemispheric EEG Coherence for Patients with Acute Exacerbation of Schizophrenia, *Psychiatry Research*, 149 (2007), 41–47.
- Hillbom, E. After-effects of Brain Injuries, *Acta Psychiatrica et Neurologica Scandinavica*, suppl. 60 (1960), 36–47.
- Hommes, O. R., & Panhuysen, L. H. H. M. Depression and Cerebral Dominance, *Psychiatria Neurologia Neurochirurgia*, 74 (1971), 259–270.
- Jones, G. H., & Miller, J. J. Functional Tests of the Corpus Callosum in Schizophrenia, *British Journal of Psychiatry*, 139 (1981), 553–557.
- Kronfol, Z., Hamsher, K. D., Digre, K., & Waziri, R. Depression and Hemispheric Functions: Changes Associated with Unilateral ECT, *British Journal of Psychiatry*, 132 (1978), 560–567.

- Lishman, W. A., & McMeekan, E. R. L. Hand Preference Patterns in Psychiatric Patients, *British Journal of Psychiatry*, 129 (1976), 158–166.
- Lishman, W. A., Toone, B. K., Colbourn, C. J., McMeekan, E. R. L., & Mance, R. M. Dichotic Listening in Psychotic Patients, *British Journal of Psychiatry*, 132 (1978), 333–341.
- Marin, R. S., & Tucker, G. J. Psychopathology and Hemispheric Dysfunction: A Review, *Journal of Nervous and Mental Disease*, 169 (1981), 546–557.
- Merrin, E. L. Schizophrenia and Brain Asymmetry: An Evaluation of Evidence for Dominant Lobe Dysfunction, *Journal of Nervous and Mental Disease*, 169 (1981), 405–416.
- Mitchell, R. L., & Crow, T. J. Right Hemisphere Language Functions and Schizophrenia: The Forgotten Hemisphere?, *Brain*, 128 (2005), 963–978.
- Moscovitch, M., Strauss, E., & Olds, J. Handedness and Dichotic Listening Performance in Patients with Unipolar Endogenous Depression Who Received ECT, *American Journal of Psychiatry*, 138 (1981), 988–990.
- Nachshon, L. Hemispheric Dysfunctioning in Schizophrenia, *Journal of Nervous and Mental Disease*, 168 (1980), 241–242.
- Newlin, D. B., Carpenter, B., & Golden, C. J. Hemispheric Asymmetries in Schizophrenia, *Biological Psychiatry*, 16 (1981), 561–582.
- O'Carroll, R. The Clinical Presentation of Neuropsychiatric Disorders, in P. W. Halligan, U. Kischka, & J. C. Marshall, eds., *Handbook of Clinical Neuropsychology* (Oxford, UK: Oxford University Press, 2003).
- Pic'1, A. K., Magaro, P. A., & Wade, E. A. Hemispheric Functioning in Paranoid and Nonparanoid Schizophrenia, *Biological Psychiatry*, 14 (1979), 891–903.
- Rochford, J. M., Weinapple, M., & Goldstein, L. The Quantitative Hemispheric EEG in Adolescent Psychiatric Patients with Depression or Paranoid Symptomatology, *Biological Psychiatry*, 16 (1981), 47–54.
- Roemer, R. A., Shagass, C., Straumanis, J. J., & Amadeo, M. Pattern Evoked Potential Measurements Suggesting Lateralized Hemisphere Dysfunction in Chronic Schizophrenics, *Biological Psychiatry*, 13 (1978), 185–202.
- Salloway, S. P., Malloy, P. F., & Duffy, J. D. (Eds.). *The Frontal Lobes and Neuropsychiatric Illness* (Washington DC, American Psychiatric Publishing, 2001).
- Schweitzer, L. Differences in Cerebral Lateralisation among Schizophrenics and Depressed Patients, *Biological Psychiatry*, 14 (1979), 721–733.
- Shagass, C., Roemer, R. A., Straumanis, J. J., & Amadeo, M. Evoked Potential Evidence of Lateralised Hemispheric Dysfunction in Depressive Psychosis, *Electroencephalography and Clinical Neurophysiology*, 49 (1980), 26P.
- Shapleske, J., Rossell, S. L., Simmons, A., David, A. S., & Woodruff, P. W. R. Are Auditory Hallucinations the Consequence of Abnormal Cerebral Lateralization? A Morphometric MRI Study of the Sylvian Fissure and Planum Temporale, *Biological Psychiatry*, 49 (2001), 685–693. [and see *Biological Psychiatry* 50 (2001), 394 for erratum]
- Shaw, J. C., Brooks, S., Colter, N., & O'Connor, K. P. A Comparison of Schizophrenic and Neurotic Patients Using EEG Power and Coherence Spectra, in

- J. Gruzelier & P. Flor-Henry, eds., *Hemisphere Asymmetries of Function in Psychopathology* (Amsterdam: Elsevier/North Holland, 1979).
- Stephane, M., Hagen, M. C., Lee, J. T., Uecker, J., Pardo, P. J., Kuskowski, M. A., & Pardo, J. V. About the Mechanisms of Auditory Verbal Hallucinations: A Positron Emission Tomographic Study, *Journal of Psychiatry & Neuroscience*, 31 (2006), 396–405.
- Tallis, F., Pratt, P., & Jamani, N. Obsessive Compulsive Disorder, Checking and Nonverbal Memory: A Neuropsychological Investigation, *Behaviour Research and Therapy*, 37 (1999), 161–166.
- Taylor, M. A., Greenspan, B., & Abrams, R. Lateralized Neuropsychological Dysfunction in Affective Disorder and Schizophrenia, *American Journal of Psychiatry*, 136 (1979), 1031–1034.
- Taylor, M. A., Redfield, J., & Abrams, R. Neuropsychological Dysfunction in Schizophrenia and Affective Disease, *Biological Psychiatry*, 16 (19H1), 467–478.
- Taylor, P. J., Dalton, R., & Fleming, J. J. Handedness in Schizophrenia, *British Journal of Psychiatry*, 136 (1980), 375–383.
- Tomer, R., Mintz, M., Levi, A., & Myslobodsky, M. S. Reactive Gaze Laterality in Schizophrenic Patients, *Biological Psychology*, 9 (1979), 115–127.
- Tucker, D. M., Antes, J. R., Stenslie, C. E., & Earnhardt, T. M. Anxiety and Lateral Cerebral Function, *Journal of Abnormal Psychology*, 87 (1978), 380–383.
- Walker, E., Hoppes, E., & Emory, E. A Reinterpretation of Findings on Hemispheric Dysfunction in Schizophrenia, *Journal of Nervous and Mental Disease*, 169 (1981), 378–380.
- Weller, M., & Montagu, J. D. Electroencephalographic Coherence in Schizophrenia: A Preliminary Study, in J. Gruzelier & P. Flor-Henry, eds., *Hemisphere Asymmetries of Function in Psychopathology* (Amsterdam: Elsevier/North Holland, 1979).
- Wexler, B. E. Cerebral Laterality and Psychiatry: A Review of the Literature, *American Journal of Psychiatry*, 137 (1980), 279–291.
- Wexler, B. E., & Heninger, G. R. Alterations in Cerebral Laterality During Acute Psychotic Illness, *Archives of General Psychiatry*, 36 (1979), 278–288.
- Wodehouse, P. G. *A Pelican at Blandings* (London: Herbert Jenkins, 1969).

Author Index

Page numbers followed by an *f* indicate figures.

- Aaron, W., 287
Abel, J. R., 311
Aboitiz, F., 295, 314
Abrams, R., 346, 347, 355, 359
Aerts, J., 290
Afshar, A., 129, 133
Agid, Y., 163, 179
Ahmed, A., 70
Albert, M. L., 126, 132, 152, 158
Alderman, N., 64, 71
Alexander, G. E., 163, 178
Alexandris, K., 308, 311
Amadeo, M., 347, 358
Amochaev, A., 276, 287
Anderson, C., 177–178
Anderson, J., 298, 313
Andreone, N., 355
Andresen, D. R., 227, 240
Andrewes, D. G., 21, 333, 337, 352, 355
Andrews, K., 171, 178
Annett, M. A., 298, 300, 301, 303, 304, 308, 310
Antes, J. R., 232, 243, 295, 313, 359
Antunes, J. L., 129, 132
Arambel, S. R., 241
Ardila, A., 156, 212, 217
Ardouin, C., 193–194
Arnett, P. A., 162, 178
Asenjo, A., 186*f*, 193
Ashwal, S., 178
Atkinson, R. C., 232, 243
Axelrod, S., 302, 313
Aziz, T. Z., 184, 194

Baade, L. E., 319, 338
Babiloni, C., 249, 259
Baciu, M., 230, 242

Baddeley, A. D., 92, 334, 339
Bakan, P., 301, 310
Baker, E., 152, 156
Bannister, R., 116
Barberi-Berger, S., 162, 178
Barker, P. B., 287
Barkoukis, V., 308, 311
Barr, W. B., 302, 312
Barrett, G., 279, 287
Bartlett, J., 190, 193
Barton, M. I., 101, 102, 111, 116, 117
Bates, G., 177
Batir, A., 193–194
Battersby, W. S., 124, 133
Bauby, J.-D., 173, 178
Beaton, A. A., 146, 156
Beaumont, J. G., 20, 107, 117, 177, 207, 211, 216, 217, 221, 223, 229, 233, 235, 238, 240, 242, 247, 257, 259, 267, 274, 275, 277, 278, 286, 287, 288, 290, 291, 306, 309, 310, 348–349, 351, 355
Bebout, L. J., 231, 244
Beeman, M., 207, 217
Beeson, P. M., 150, 156
Behar, C., 194
Behrmann, M., 146, 153, 156
Bell, G. A., 212, 217
Bell, M. A., 275, 288
Belmore, S. M., 253, 259
Bender, M. B., 124, 133
Benson, F. D., 66, 71, 111, 116, 127, 137, 142, 152, 156, 157, 216
Benson, P. J., 132, 133
Benton, A. L., 67, 70, 110, 114, 116
Ben-Yishay, Y., 335, 337
Berenbaum, S. A., 256, 259

- Berg, R. A., 327, 337
 Berkelbach van der Sprenkel, J. W., 189, 195
 Berlin, C. I., 247, 249, 255, 259
 Berlucchi, G., 225, 238, 241
 Bernardin, L., 162, 179
 Berndt, R. S., 153
 Berry, T., 152, 156
 Bertelson, P., 253, 261
 Besner, D., 231, 241
 Beveridge, R., 307, 312
 Billiard, S., 297, 311
 Binder, J. R., 314
 Bion, P. J., 221, 229, 244
 Birkett, P., 298, 310
 Bishop, E. R., 346, 355
 Bisiach, E., 105, 116, 126, 132
 Bjorklund, A., 184, 195
 Black, F. W., 49, 70, 89
 Black, S. E., 94
 Blanchard, R., 296, 312
 Blanchet, S., 237, 241
 Blanken, G., 137, 158
 Blankner, J. G., 311
 Bloom, F. E., 41
 Bodis-Wollner, I., 177
 Bogen, G. M., 140, 156
 Bogen, J. E., 140, 156, 199–200, 207, 210,
 217, 246, 260
 Boiler, F., 93, 126, 133
 Boll, T. J., 337
 Boller, F., 164, 178, 321
 Bolton, N., 320, 338
 Boly, M., 179
 Bor, D., 70
 Bornstein, A., 339
 Boronat, C. B., 282, 288
 Bourne, V. J., 223, 228, 240, 241
 Bowers, D., 256, 260, 305, 312
 Bowers, J. K., 260
 Boyer, C., 294, 312
 Braaten, A. J., 184, 194
 Bradshaw, J. L., 216, 230, 231, 237, 241, 244,
 251, 252, 253, 259, 260, 294, 307, 310
 Brady, S. A., 254, 260
 Braff, D., 346, 356
 Brambilla, P., 355
 Bramham, J., 193
 Brancucci, A., 249, 259
 Brassington, J. C., 177
 Bridges, P., 190, 193
 Brindley, G. S., 129, 133
 Brinkman, C., 225, 241
 Britton, P. G., 320, 338
 Brizzolara, D., 238, 241, 294, 314
 Broca, P., 9
 Brody, B. A., 101, 117
 Brooks, S., 347, 358–359
 Brouwers, E. Y.M., 221, 242
 Brown, J. W., 138, 156
 Brown, R. G., 164, 178
 Brown, W. S., 278, 288
 Bruder, G. E., 349, 355
 Bryden, M. P., 230, 234, 241, 254, 255, 259,
 294, 295, 297, 298, 311, 313
 Bub, D., 250, 259
 Buchsbaum, M., 279, 288
 Buchtel, H. A., 294, 314
 Buffery, A. W.H., 293, 311, 333, 337
 Bulman-Fleming, M. B., 248, 260, 297, 311
 Bunge, M., 21
 Bures, J., 221, 242
 Burgess, N., 93
 Burgess, P. W., 64, 71
 Burton, L. A., 228, 241
 Butters, N., 85, 93, 101, 102, 113, 116, 117,
 166, 178
 Butterworth, B., 109, 116, 117
 Buxbaum, L. J., 288
 Caccappolo-van Vliet, E., 149, 156
 Cahn, A., 229, 243
 Caine, E. D., 178
 Callaway, E., 273, 276, 286, 288, 289
 Campbell, R., 21, 92
 Capitani, E., 126, 132
 Cappa, S. F., 281, 287, 290
 Caramazza, A., 149, 157
 Cardebat, D., 237, 243
 Carlson, N. R., 41, 192
 Carpenter, B., 353, 358
 Carr, S. A., 348, 350, 356
 Carter, R. L., 135, 156, 302, 311
 Caspary, P., 179
 Caudrey, D., 255, 260
 Cerini, R., 355
 Cermak, L. C., 84, 85, 166
 Cermak, L.S., 93, 178
 Chabardes, S., 193–194
 Chabris, C. F., 229, 241
 Chandra, P. S., 194
 Chang, J. W., 189, 194
 Chapman, L. F., 48
 Chase, T. N., 163, 178–179
 Chelune, C. J., 339
 Cherbuin, N., 225, 241
 Chiarello, C., 207, 217, 232, 241
 Childs, N., 178
 Chin, S., 357
 Christensen, A.-L., 57, 70, 324, 326, 337, 338
 Clare, A., 185, 192, 193
 Clark, R. E., 94
 Clarke, J., 295, 314
 Clarkson, D., 278, 291
 Clayton-Davies, J., 181, 194
 Clements, A. M., 296, 311
 Cloning, I., 125, 132
 Cloning, K., 125, 132
 Code, C., 21, 94, 207, 217
 Cohen, G., 226, 233, 234, 237, 241, 242, 307,
 311
 Colbourn, C. J., 224, 242, 255, 259, 349, 352,
 356, 358

362 Author Index

- Coleman, M. R., 171, 178, 179
 Coles, G. H., 287
 Colley, M., 238, 241
 Colter, N., 347, 358–359
 Coltheart, M., 146, 148, 150, 156
 Conway, M. A., 92
 Cook, S. D., 177
 Cools, R., 193–194
 Cooper, R., 286
 Coren, S., 297, 298, 302, 303, 310, 311, 314
 Corkin, S., 80, 81, 93, 94, 97, 117, 189, 193
 Cormack, A., 280
 Coslett, H. B., 288
 Cousin, E., 230, 242
 Cowey, A., 128, 132
 Cox, P. J., 233, 242–243
 Craig, J. E., 251, 259
 Cranford, R., 178
 Crea, F., 225, 241
 Critchley, M., 77, 93, 114, 116, 117, 156, 259–260
 Crossley, D., 193
 Crossman, A. R., 41
 Crow, T. J., 251, 261, 349, 358
 Crutcher, M. D., 163, 178
 Cullen, J. K., Jr., 247, 259
 Cummings, J. L., 355
 Curry, S. H., 279, 290
 Cutting, J. E., 250, 259

 Dafoe, C. G., 239, 242
 Dalglish, T., 70, 189, 193
 Dalton, R., 346, 350, 356, 359
 Damasio, A. R., 66–67, 69, 70, 126, 132, 251, 259–260
 Damasio, H., 126, 132, 251, 259–260
 Darwin, C. J., 249, 254, 260
 David, A. S., 341, 348, 355, 356, 356–357, 358
 Davidoff, J., 227, 229, 242, 311
 Davidson, R. J., 49, 216, 274, 288, 351, 355, 356
 Davis, A. E., 278, 288
 Davis, M. H., 178, 179
 Davis, R., 231, 235, 241, 242
 Davison, L. A., 71
 Dax, M., 140
 Day, J., 229, 230, 242
 De Ajuriaguerr, J., 112*f*
 De Pesce, M., 210, 218
 De Renzi, E., 116
 De Valois, K. K., 132
 Dean, C. E., 189, 193
 Decker, H., 275, 288
 Dedon, M. F., 289
 Degueldre, C., 290
 Delargy, M., 178
 Delgado, J. M. R., 34
 DeLong, M. R., 163, 178
 DeLuca, J., 162, 178, 287
 Demarest, R. J., 41
 Demeyer, W., 114, 117

 Démonet, J. F., 237, 243
 Denise, P., 241
 Desgranges, B., 241
 Desmedt, J. E., 273, 286, 288
 Deutsch, G., 216
 Devinsky, O., 302, 312
 Diamond, M. C., 41
 Diamond, R., 346, 356
 Digre, K., 346, 357
 Dimond, S. J., 210, 214, 217, 221, 226, 240, 242, 348–349, 350, 351, 355, 356
 DiStefano, M., 225, 241
 Dobelle, W. H., 129, 132
 Doble, J. E., 177–178
 Dolce, G., 177, 275, 288
 Donchin, E., 267, 274, 275, 277, 279, 286–287, 288, 289, 290
 Donnett, J. G., 93
 Dory, R. W., 356
 Doubleday, C., 278, 291
 Doyle, J. C., 276, 289
 Doyon, B., 237, 243
 Drago, D., 279, 288
 Drewe, E. A., 59, 70
 Drislane, F. W., 125, 133
 Dubois, B., 163, 179
 Duffy, J. D., 358
 Duncan, J. D., 70

 Earnhardt, T. M., 359
 Ebadi, M., 177
 Eccles, J. C., 211, 217
 Ecuyer-Dab, I., 298, 311
 Efron, R., 246, 260
 Ehrlichman, H., 258, 260, 274, 288
 Eichenbaum, H., 89, 93
 Elias, L. J., 248, 260
 Elliot, K., 179
 Elliott, D., 302, 312
 Elliott, K., 175
 Ellis, A. W., 146, 156, 221, 244
 Ellis, H. D., 343, 356
 Ellis, R. R., 278, 289
 Elson, L. M., 41
 Emory, E. A., 349, 359
 Emslie, H., 64, 71
 England, M. A., 41
 Engle, A., 307, 314
 Eustache, F., 233, 241, 244
 Evans, A. C., 60, 70
 Evans, J. J., 64, 71, 334, 339
 Evans, M. E., 64, 71
 Ewing, C. T., 254, 260

 Fabene, P. F., 355
 Fairweather, H., 294, 297, 311, 314
 Farace, P., 355
 Farah, M. J., 132
 Farr, W. F., 346, 355
 Farrelly, J., 253, 259
 Farrington, L. J., 221, 242

- Faure, S., 233, 241, 244
 Faurie, C., 297, 311
 Fedio, P., 181, 193
 Feinstein, A., 177
 Feirtag, M., 32
 Fennell, E. B., 256, 260, 305, 312
 Fleminger, J. J., 341, 346, 350, 356, 359
 Fleminger, S., 355, 356
 Flight, J., 255, 262
 Flor-Henry, P., 346, 347, 351, 355, 356, 357
 Follett, K., 184, 195
 Folstein, S. E., 177
 Fountas, K., 185, 194
 Fox, N. A., 275, 288
 Foxe, J. J., 279, 291
 Frackowiak, R. S.J., 93, 283, 289
 Frank, E., 93
 Franklin, S., 148, 157
 Freedman, M., 177
 Freeman, C., 185, 186*f*, 193
 Freeman, R., 307, 311
 Freeman, W., 193
 Freides, D., 254, 260
 Freud, S., 125
 Friedman, A., 239, 242, 277
 Friedman, D., 288
 Frisé, L., 132
 Frith, C. D., 93, 283, 289, 294
 Frith, U., 311
 Fromm-Auch, D., 346, 356
 Frost, D., 127, 133
 Funkiewiez, A., 184, 193–194
 Funnell, E., 153
 Furst, C., 211, 217
 Fuster, J. M., 69, 70
- Gage, P., 61–62, 62*f*
 Galaburda, A. M., 280, 289
 Galderisi, S., 348, 357
 Galen, 8
 Galin, D., 278, 289, 346, 350–351, 356
 Gall, F. J., 9
 Gamble, M., 260
 Gandevia, S. C., 209, 217
 Gao, F., 89, 94
 Garbesi, L., 302, 312
 Gardner, H., 152, 156
 Gargiulo, M., 194
 Garoutte, B., 41
 Garraux, G., 290
 Garvin, E. A., 228*f*
 Gaskill, S. J., 239, 242
 Gates, A., 251, 252, 260, 294
 Gates, E. A., 230, 231, 241
 Gazzaniga, M. S., 200–201, 205, 206, 209,
 210, 212, 216, 217, 218, 220, 246, 261–262
 Geffen, G., 255, 260, 303, 311
 Geschwind, N., 39, 110, 117, 127, 132, 137,
 156, 157, 280, 289
 Gevins, A. S., 276, 289
 Ghent, L., 59, 71, 97, 103, 117
- Giacino, J. T., 171, 178
 Gillard, J. H., 287
 Gillean, J., 356
 Gill-Thwaites, H., 175, 178
 Girvin, J. P., 129, 132
 Globus, G. G., 212, 218
 Godfrey, J. J., 250, 260
 Golden, C. J., 327, 334, 337, 338, 353, 358
 Goldman, R. D., 307, 312
 Goldman-Rakic, P. S., 61, 70
 Goldstein, G., 326, 338
 Goldstein, K., 9, 49–50
 Goldstein, L., 358
 Gollin, E. S., 83*f*
 Gomez, C., 163, 178–179
 Goodale, M., 132
 Goodglass, H., 137, 156
 Gordon, H. W., 206, 218, 251, 252, 260
 Gotlib, I. H., 275, 289
 Granholm, E., 166, 178
 Gray, C., 350, 356
 Gray, J. A., 293, 311
 Green, M. E., 347, 357
 Greenberg, M. S., 192
 Greenspan, B., 359
 Grimsell, D., 231, 241
 Groth-Marnat, G., 337
 Grouios, G., 308, 311
 Gruzelier, J., 348, 350, 351, 353
 Gruzelier, J. H., 355, 357
 Gupta, S., 194
 Gur, R. C., 313, 350, 353
 Gur, R. E., 350, 357
 Gutchess, A. H., 282, 289
- Hagen, M. C., 359
 Haig, A. J., 177–178
 Haines, S. J., 192
 Halderman, L. K., 241
 Hall, E. H., 320, 338, 346
 Hall, W. D., 357
 Halliday, A. M., 279, 287
 Halliday, E., 279, 287
 Halligan, P. W., 21, 105, 107, 116, 117, 157,
 342, 355, 357
 Halstead, W., 48
 Hamilton, S. E., 229, 241
 Hamm, J. P., 252, 261
 Hammeke, T. A., 314
 Hammond, N. V., 348, 350, 353, 357
 Hamsher, K. D., 346, 357
 Hanley, J. R., 146, 157
 Hannay, H. J., 294, 312, 328, 337, 338
 Hannula, D. E., 89, 93
 Hardyck, C., 231, 242, 303, 304, 306, 307,
 310, 312
 Harper, P., 177
 Harris, P. G., 278, 289
 Harshman, R. A., 252, 256, 259, 261
 Hawryluk, J., 295, 312
 Haynes, W. O., 275, 276, 289, 290

364 Author Index

- Head, H., 9
 Heaton, R. K., 319, 338, 339
 Hebb, D. O., 48
 Hécaen, H., 112*f*
 Heeley, D. W., 133
 Heim, A. W., 307, 312
 Heimberger, R. F., 114, 117
 Held, R., 127, 133
 Hellige, J. B., 216, 226, 233, 242–243
 Helm, N., 152, 158
 Heninger, G. R., 349, 359
 Henry, R. G., 253, 260
 Henson, R. A., 77, 93, 259–260
 Herron, J., 275, 290, 297, 310, 312
 Herzog, H., 70
 Hicks, R. A., 302, 307, 312
 Hicks, R. E., 233, 243
 Higashima, M., 347, 357
 Higginson, C. H., 162, 178
 Hillary, F. G., 287
 Hillbom, E., 341, 357
 Hillis, A., 149, 153, 157
 Hillyard, S. A., 209, 210, 217, 218, 279, 287, 289
 Hines, D., 238, 243, 305, 312
 Hippocrates, 8
 Hiscock, I., 295, 312
 Hiscock, M., 254, 260, 295, 296, 312
 Hiscock-Kalil, C., 295, 312
 Hobben, N., 337
 Hoff, A. L., 309, 313
 Hoff, H., 125, 132
 Hohenegger, M., 135, 156, 302, 311
 Hole, G. J., 228, 241
 Holland, A. L., 152, 157
 Hommes, O. R., 357
 Homskaya, E. D., 59, 70
 Hoppes, E., 349, 359
 Horn, S., 179
 Houeto, J. L., 194
 Hounsfeld, G., 280
 House, A. S., 250, 262
 Howard, D., 148, 157
 Howarth, B. G., 347, 356
 Howell, P., 254, 260
 Howes, D., 137, 157
 Howieson, D. B., 328, 337, 338
 Hubel, D. H., 120, 132
 Huber, W., 152, 158
 Hugdahl, K., 216, 259, 355
 Humphrey, N., 128, 132–133
 Humphreys, G. W., 132, 334, 337, 338
 Hur, K., 184, 195
 Hutt, C., 292, 312
 Hutton, J. T., 138, 157
 Huws, D., 350, 356

 Iaccino, J. F., 216
 Inch, R., 254, 260, 295, 312
 Ippolito, D., 184, 195
 Irwin, W., 351, 356

 Isaacs, K. L., 302, 312
 Israelian, M., 295, 312
 Ivnik, R. J., 339

 Jacek, C., 295, 312
 Jacklin, C. N., 292, 313
 Jackson, J. H., 10, 69
 Jain, S., 194
 Jamani, N., 344, 359
 James, M., 117
 Jay, M., 125, 133
 Jeeves, M. A., 200, 218, 248, 261
 Jenkins, J., 152, 158
 Jennett, B., 177, 178
 Jennings, J. R., 287
 Jimenez-Pabon, E., 152, 158
 Joannette, Y., 21, 94, 298, 311
 Johansen, B. H., 285, 289
 Johanssen-Horrbach, H., 137, 158
 John, E. R., 267, 291
 Johnson, K. L., 319, 338
 Johnson, R. C., 251, 260
 Johnson, S. K., 162, 178
 Johnsrude, I. S., 178
 Johnston, R. S., 133
 Johnstone, J., 275, 290
 Jones, G. H., 348, 357
 Jones, L., 177
 Jones-Gotman, M., 88, 93
 Jonides, J., 232, 243
 Jordan, J. R., 133

 Kacinik, N. A., 241
 Kaehkönen, S., 284, 289
 Kail, R., 294, 312
 Kaiser, D., 295, 314
 Kalil, K. M., 295, 312
 Kallman, H. J., 253, 254, 261
 Kaplan, E., 137, 156, 166, 179
 Katz, D. I., 178
 Katz, R., 177–178
 Kaufman, A. S., 328, 337, 338
 Kawai, K., 185, 194
 Kay, J., 146, 157
 Kelly, D., 190, 193
 Kemper, T. L., 280, 289
 Kenealy, P. M., 20, 177
 Kennard, C., 128, 132, 133
 Kern, R. S., 347, 357
 Kertesz, A., 137, 138, 140, 142, 143, 150, 152, 157, 274, 286, 289
 Kikiuchi, M., 357
 Kim, C. H., 189, 194
 Kim, J. W., 189, 194
 Kimberg, D. Y., 288
 Kimura, D., 109, 117, 247, 251, 261
 Kinsbourne, M., 233, 238, 239, 240, 243, 254, 258, 260, 261, 277, 289
 Kirik, D., 184, 195
 Kischka, U., 21, 157
 Klatzky, R. L., 232, 243

- Knight, R. T., 71
 Knowles, J., 177
 Kobayashi, Y., 294, 314
 Kolb, B., 20, 55, 56*f*, 70
 Koles, Z. J., 347, 356
 Kolodny, J., 70
 Koo, M. S., 189, 194
 Kopelman, M., 92, 341, 355, 356
 Koslow, S. H., 273, 286, 288
 Krack, P., 193–194
 Krashen, S., 252, 261
 Kronfol, Z., 346, 357
 Kuskowski, M. A., 359
 Kutas, M., 267, 273, 279, 286–287, 288, 289, 291

 Ladavas, D., 210, 218
 Ladavas, E., 217
 Lai, Z. C., 337
 Lalumière, M. L., 296, 312
 Lambert, A., 252, 261
 Landsdell, H., 194
 Langdon, R., 146, 156
 Lange, H. W., 179
 Lansdell, H., 181, 194
 Lashley, Karl, 9
 Lassone, M., 200, 218
 Laureys, S., 290
 LaVigna, G. W., 338
 Lazarson, A., 41
 Le Bomin, 297, 311
 Leach, L., 166, 179
 Le-Bomin, S., 311
 Lechevalier, B., 241
 Lecours, A. R., 21, 94
 Lederman, S. J., 231, 244
 Ledlow, A., 243, 277, 289
 LeDoux, J. E., 200, 205, 209, 210, 212, 216, 218
 Lee, J. T., 359
 Leehey, S. C., 229, 243
 Leh, S. E., 285, 289
 Leiber, L., 243, 302, 313
 LeMay, M., 280, 289
 Leng, N. R.C., 85, 94
 Leo, G. J., 162, 179
 Lerner, B. H., 188, 194
 Lesk, D., 140, 157
 LeVay, S., 93
 Levi, A., 350, 359
 Levy, J., 204, 207, 208*f*, 210, 218, 228, 241, 298, 303, 304, 306, 307, 308, 313
 Lewis, M. B., 343, 356
 Lezak, M. D., 328, 337, 338
 Liberman, A. M., 249, 261
 Lichtenberger, E. O., 328, 337, 338
 Lichtheim, L., 137
 Liepmann, H., 110
 Lim, V. K., 252, 261
 Lindell, A. K., 239, 243
 Linford-Rees, W., 187, 194

 Lishman, W. A., 349, 350, 358
 Littlewood, C., 171, 178
 Litvac, L., 233, 242–243
 Litvan, I., 163, 178–179
 Loring, D. W., 328, 337, 338
 Lovestone, S., 341, 355, 356
 Low, D. W., 275, 290
 Luck, S. J., 210, 218, 286
 Luria, A. R., 10, 39, 58*f*, 59, 69, 70, 106*f*, 124*f*, 138, 157, 323–325, 327, 338
 Luzzatti, C., 105, 116
 Lyon, P. J., 295, 312
 Lyons, F. M., 261

 Maccoby, E. E., 292, 313
 Macdonald, D., 187, 194
 Mack, J. L., 126, 133
 Magaro, P. A., 349, 358
 Maguire, E. A., 89, 93, 283, 289
 Mahoney, R. A., 275, 290
 Majovski, L. V., 324, 338
 Malcolm, S., 248, 261
 Malhi, G., 193
 Malloy, P. F., 358
 Maltete, D., 194
 Mance, R. M., 349, 358
 Mangun, G. R., 210, 218
 Marin, R. S., 352, 353, 355, 358
 Markowitsch, H. J., 93
 Marsden, C. D., 164, 178
 Marsh, G. R., 274, 279, 287, 290
 Marsh, J. T., 278, 288
 Marsh, N. V., 177
 Marshall, J. C., 21, 107, 117, 148, 150, 152, 156, 157, 342, 355, 357
 Marsolek, C. J., 227, 240
 Martin, G. N., 21
 Martin, S., 302, 312
 Martinez, C., 248, 261
 Martone, M., 166, 178
 Marzi, C. A., 238, 241
 Masterson, R. B., 120, 133
 Mateer, C. A., 140, 335, 337, 339
 Matteson, R., 295, 314
 Mawdesley, C., 183, 194
 Mayes, A. R., 92, 93, 278, 288, 290
 McBride, K., 137, 158
 McCabe, P., 140, 157
 McCallum, W. S., 279, 290
 McCarthy, G., 267, 275, 277, 286–287, 288, 290, 309
 McCarthy, M., 107, 117
 McCarthy, R. A., 231, 243, 310
 McClure, G. M., 346, 356
 McCreery, D. B., 52, 71
 McFarland, K., 298, 313
 McFie, J., 114, 117
 McGlone, J., 293, 294, 295, 296, 310, 313
 McKee, J., 126, 132
 McKeegan, E. R.L., 349, 350, 358
 McKeever, W. F., 294, 298, 309, 313

366 Author Index

- McKenna, J., 256, 262
 McKiernan, K. A., 314
 McLellan, L., 179
 McManus, I. C., 248, 260, 297, 298, 311, 313
 McMeekan, E. R.L., 358
 Mellers, J., 341, 355, 356
 Menon, D. K., 178
 Merrin, E. L., 352, 353, 355, 358
 Metzger, R. L., 232, 243, 295, 313
 Michie, P. T., 279, 290
 Migo, E., 93
 Milberg, W., 337
 Miller, E., 307, 313, 335, 338
 Miller, J. J., 348, 357
 Milner, A. D., 132, 133
 Milner, B., 55, 56f, 59, 60, 62, 70, 76, 80, 86,
 88, 93, 97, 117, 123, 125, 133, 135, 157,
 185, 194, 302, 314
 Mintz, M., 350, 359
 Miozzo, M., 149, 156
 Mishkin, M., 60, 71
 Mitchell, R. L., 251, 261, 349, 358
 Mobley, M. C., 346, 355
 Mohr, E., 163, 178–179
 Moniz, E., 185
 Montagu, J. D., 347, 359
 Montaldi, D., 93
 Moore, W. H., Jr., 275, 290
 Morais, J., 253, 254, 261
 Mori, S., 284, 290
 Moscovitch, M., 89, 94, 237, 243, 349, 358
 Moses, J. A., Jr., 327, 337
 Mostofsky, S. H., 311
 Mueller, H. M., 275, 291
 Muggia, S., 164, 178
 Munday, R., 171, 175, 178
 Murdoch, B., 157
 Murphy, L., 171, 178
 Mushahwar, V. K., 52, 71
 Myslobodsky, M. S., 350, 359

 Näätänen, R., 279, 290
 Nachshon, L., 349, 358
 Nagasawa, T., 357
 Nagel, T., 212, 218
 Nagylaki, T. A., 298, 313
 Nauta, W. J.H., 32
 Neary, D., 41
 Nelson, C. A., 41
 Nelson, H. E., 322, 338
 Nelson, P. K., 302, 312
 Netter, F. H., 41
 Nettleton, N. C., 307, 310
 Neuringer, C., 326, 338
 Neville, H. J., 273, 291
 Newcombe, F., 307, 314, 333, 338
 Newlin, D. B., 353, 358
 Newton, M. P., 187, 194–195
 Nichelli, P., 126, 132
 Nicholls, M. E., 239, 243
 Nieuwenhuys, R., 41

 Nirao, N., 357
 Noback, C. R., 41
 Noordmans, H. J., 189, 195
 Noth, J., 179
 Nutt, J. T., 163, 179

 Obeso, J. A., 163, 179
 O'Carroll, R., 344, 358
 Ockwell, A., 302, 310
 O'Connell, A., 338
 O'Connor, K. P.A., 358–359
 O'Connor, V., 84, 93, 347
 Ogden, J. A., 81, 94
 Ogilvie, A. D., 193
 Ojemann, G. A., 140, 157, 202, 219
 Oka, T., 357
 O'Keefe, J., 93
 Olanow, C. W., 163, 179
 Olds, J., 349, 358
 Ornstein, R., 275, 290
 Orstein, R., 290
 Osmon, D. C., 327, 337, 338
 Ossleton, J. W., 286
 Ostrosky-Solis, F., 212, 217
 Owen, A. M., 60, 70, 171, 179

 Panhuysen, L. H.H. M., 357
 Papanicolaou, A. C., 287
 Papçun, G., 252, 261
 Pardo, J. V., 359
 Pardo, P. J., 359
 Park, D. C., 282, 289
 Park, I. H., 189, 194
 Parkin, A. J., 82, 85, 92, 94
 Parrot, M., 237, 243
 Parsons, T. D., 184, 194
 Passini, R., 298, 311
 Patterson, K. E., 146, 148, 150, 153, 156, 157,
 237, 244, 329, 338
 Paul, K. J., 248, 261
 Peigneux, P., 283, 290
 Pekar, J. J., 311
 Penfield, W., 94
 Perachio, N., 295, 312
 Perani, D., 281, 287, 290
 Pereira, E. A., 184, 194
 Perelman, E., 212, 218
 Perret, L., 55, 71
 Perrett, D. I., 133
 Perry, C., 146, 156
 Persaud, R., 193
 Petersen, S. E., 61, 71
 Petrides, M., 60, 70
 Petrinovich, L. F., 303, 307, 310, 312
 Peyrin, C., 230, 242
 Pfeiffer, R. F., 177
 Pickard, J. D., 178
 Pic'l, A.K., 349, 358
 Piercy, H., 112f
 Pillon, B., 163, 179
 Pincus, J. H., 184, 194

- Ploska, A., 163, 179
 Poeck, K., 152, 158
 Poffenberger, A. T., 224
 Poldroll, K., 179
 Polson, M. C., 239, 242
 Ponsford, J., 333, 337, 338
 Pontier, D., 297, 314
 Poppel, E., 127, 133
 Porac, C., 298, 314
 Posner, M. I., 61, 71
 Possing, E. T., 314
 Post, F., 194
 Powell, G. E., 175, 179, 333, 338
 Pratt, P., 302, 303, 344, 359
 Pratt, R. T.C., 314
 Preilowski, B., 213, 218
 Preissl, H., 290
 Presbrey, T. W., 261
 Pressman, J. D., 192
 Pribram, K. H., 59, 70
 Prigatano, G. P., 335, 338
 Pringle, H., 107, 117
 Prochazka, A., 52, 71
 Provins, K. A., 248, 261
 Pryce, I. J., 350, 356
 Prito, A., 285, 289
 Puccetti, R., 212, 218

 Querné, L., 233, 244
 Quest, D. O., 129, 132

 Rabbitt, P., 328, 338
 Rambaldelli, G., 355
 Randolph, J. R., 162, 178
 Ranganath, C., 89, 93, 275, 289
 Ransil, B. J., 308, 314
 Rao, S. M., 162, 179
 Rapcsak, S. Z., 150, 156
 Rapin, I., 277, 288
 Rasmussen, T., 76*f*, 97, 117, 135, 157, 184, 194, 302, 314
 Rastle, K., 146, 156
 Ratcliff, G., 307, 314
 Raymond, M., 297, 311, 314
 Rebert, C. S., 275, 290
 Reches, A., 126, 132
 Redfield, J., 346, 359
 Reeves, A., 201, 219
 Reid, M., 308, 313
 Reitan, R. M., 49, 71, 114, 117
 Remington, R., 252, 261, 278, 291
 Repp, B. H., 247, 261
 Richards, B., 89, 94
 Richardson, J. T.E., 333, 338
 Riddoch, M. J., 334, 337, 338
 Rimrod, S. L., 311
 Risse, G. L., 200, 209, 218
 Ritter, W., 277, 288
 Rizzolatti, G., 238, 241, 294, 314
 Roberts, J., 307, 314
 Roberts, T. S., 129, 132

 Robertson, I. H., 105, 116, 117, 334, 339
 Robin, A., 187, 194
 Robinson, C. S., 241
 Rochford, J. M., 358
 Rodd, J. M., 178
 Rodgers, J. E., 193
 Rodgers, M. A., 256, 262
 Roemer, R. A., 347, 358
 Rogers, M. J.C., 20
 Rogers, S. A., 184, 194
 Rolls, E. T., 66, 69, 71
 Romani, G. L., 249, 259
 Rose, D., 212, 218
 Rosenbaum, R. S., 89, 94
 Rosenfeld, J. P., 275, 289
 Rosini, P. M., 249, 259
 Ross, L. E., 232–233, 244
 Rossell, S. L., 348, 358
 Rossi, E. M., 284, 289
 Rothwell, N. A., 338
 Roy, E. A., 297, 311
 Rugg, M. D., 275, 277, 279, 287, 288, 290–291
 Ruggiero, D. A., 41
 Rushton, D. N., 129, 133
 Russell, A., 256, 262
 Russell, E. W., 326, 338
 Rylander, G., 48
 Ryu, S. I., 129, 133

 Sadler, R. M., 185, 194
 Saffran, E. M., 288
 Sait, P. E., 249, 262
 Saklofske, D. H., 339
 Salamy, A., 276, 287
 Salloway, S. P., 358
 Santhanam, G., 129, 133
 Sanyal, S. K., 185, 194
 Sappington, J., 296, 314
 Saron, C. D., 274, 279, 288, 291
 Sasanuma, S., 294, 314
 Satz, P., 135, 136, 156, 158, 256, 260, 302, 305, 311, 312
 Savage, C. W., 212, 218–219
 Savage, R. D., 320, 338
 Sazbon, L., 177
 Scammell, R., 350, 356
 Schachter, S. C., 308, 314
 Schacter, D. L., 92
 Schafner, R. E., 276, 289
 Schiebel, A. B., 41
 Schiefenhövel, W., 297, 311
 Schmit, V., 235, 242
 Schopflocher, D., 346, 356
 Schroeder, C. E., 279, 291
 Schuell, H., 152, 158
 Schupbach, M., 184, 194
 Schurr, P., 187, 194
 Schweitzer, L., 350, 358
 Scoville, W. B., 185, 186*f*
 Searleman, A., 256, 261, 301, 305, 314

368 Author Index

- Segalowitz, S. J., 231, 244
 Seitz, R. J., 70
 Semmes, J., 59, 71, 98, 102–103, 117, 305, 314
 Sensory-Perceptual Examination, 325
 Sergeant, J. A., 209, 217, 219
 Sergi, M. J., 347, 357
 Severn, J. M., 9
 Shagass, C., 358
 Shallice, T., 21, 64, 65, 65*f*, 71, 113, 117, 329, 339
 Shalman, D. C., 79, 94
 Shapleske, J., 348, 358
 Shaw, J. C., 268, 286, 291, 347, 358–359
 Shears, C., 241
 Shenoy, K. V., 129, 133
 Shiel, A., 179, 334, 339
 Shimazu, H., 185, 194
 Shoulson, I., 178
 Shucard, D. W., 278, 291
 Shucard, J. L., 278, 291
 Sidtis, J., 206, 219, 246, 261–262
 Siegel, A., 294, 312
 Silberstein, R. B., 278, 289
 Silverberg, R., 126, 132
 Sim, T. C., 248, 261
 Simmons, A., 348, 358
 Simson, R., 277, 288
 Singh, V. P., 194
 Smith, E., 178
 Smith, J. C., 278, 288
 Smith, J. R., 185, 194
 Smylie, C. S., 212, 218
 Snyder, M., 274, 288
 Sohlberg, M. M., 335, 337, 339
 Sparks, R., 152, 158
 Sparr, S. A., 125, 133
 Spellacy, F., 248, 261
 Sperry, R. W., 199–200, 204, 207, 210, 211, 212–213, 217, 218, 219, 220
 Spinnler, H., 126, 132, 133
 Spreen, O., 337
 Springer, S. P., 200, 206, 209, 216, 218, 219, 246, 249, 250, 261–262, 301, 314
 Spurzheim, J. C., 9
 Squire, L. R., 92, 94
 Stachowiak, F.-J., 152, 158
 Stark, C. E.L., 94
 Stenslie, C. E., 359
 Stephane, M., 359
 Stern, M., 149, 184, 195
 Stern, Y., 156
 Stierman, I., 255, 260
 Straumanis, J. J., 347, 358
 Strauss, E., 337, 349, 358
 Strik, W., 348, 357
 Strominger, N. L., 41
 Studdert-Kennedy, M., 254, 262
 Stuss, D. T., 66, 69, 71, 334, 339
 Sugano, H., 185, 194
 Suh, H. S., 189, 194
 Sunaga, S., 185, 194
 Swanson, J. M., 243, 277, 289
 Sweet, W. H., 184, 194
 Swencionis, C., 275, 290
 Swick, D., 273, 291
 Szaflarski, J. P., 302, 314
 Tabossi, P., 294, 314
 Takeda, T., 357
 Tallis, F., 344, 359
 Tang, K., 288
 Tanguay, P. E., 278, 291
 Tapper, M., 346, 356
 Tassinari, G., 225, 241
 Taub, J. M., 278, 291
 Taylor, A. M., 100, 117
 Taylor, L., 71
 Taylor, M. A., 346, 347, 355, 359
 Taylor, M. J., 253, 259, 307, 310, 350
 Taylor, M. L., 152, 158
 Taylor, N., 274, 288
 Taylor, P. J., 359
 Teasdale, J. D., 193
 Teng, E. L., 255, 262
 Terbeck, D., 252, 261
 Teuber, H.-L., 10, 49, 59, 60, 69, 71, 80, 93, 97, 103, 117, 123, 124, 133, 333, 339
 Thatcher, R. W., 267, 291
 Thompson, R., 107, 117
 Threepwood, G., 340
 Thwaites, H., 175, 179
 Tobias, E., 278, 287
 Tomer, R., 350, 359
 Toone, B. K., 349, 358
 Tooth, G. C., 187, 194–195
 Topolski, R., 296, 314
 Torjussen, T., 128, 133
 Traub, E., 255, 260, 303, 311
 Tremblay, T., 298, 311
 Trevarthen, C., 204, 207, 208*f*, 210, 213, 218, 219
 Trimble, M. R., 355
 Tripathi, M., 194
 Troster, A. I., 184, 194
 Tsorbatzoudis, H., 308, 311
 Tucker, D. M., 275, 291, 350, 359
 Tucker, G. J., 184, 194, 349, 352, 353, 355, 358
 Tueting, P., 273, 286, 288
 Tulskey, D. S., 328, 339
 Tweedy, J., 301, 314
 Tzeng, O. J.L., 231, 242
 Uecker, J., 359
 Umilta, C., 238, 241, 294, 314
 Unverzagt, F., 162, 179
 Valenstein, E. S., 185, 191, 193, 195
 Van Buren, J. M., 181, 193
 Van der Linden, M., 290
 Van Deventer, A., 294, 309, 313
 Van Huijzen, C., 41

- van Rijen, P. C., 189, 195
 Van Wagenen, W. P., 199
 van Zijl, P., 284, 290
 VanderGriff, A., 185, 194
 Vanderplas, J. M., 228f
 Vargha-Khadem, F., 294, 311
 Vaughan, H. G., Jr., 279, 291
 Venna, N., 125, 133
 Vernon, P. E., 320, 339
 Vetter, R. R., 261
 Vignolo, L. A., 77, 94
 Vilkki, J., 181, 195
 Vogel, P., 199–200, 207
 Volpe, B. T., 212, 218
 Voogd, J., 41
 Voyer, D., 255, 256, 262, 295, 314
- Wada, J. A., 278, 288
 Wade, E. A., 349, 358
 Wakely, J., 41
 Waldeier, H., 275, 288
 Waldman, A. D., 287
 Walker, E., 349, 359
 Wallesch, C.-W., 21, 94
 Wallesch, C. W., 137
 Wallesch, C.-W., 158
 Walters, B. C., 192
 Wang, W. S.-Y., 231, 242
 Ward, B. D., 314
 Ward, T. B., 232, 244
 Warrington, E. K., 83, 94, 100, 111, 113, 117, 302, 303, 314
 Watson, M., 179
 Watson, P. J., 182, 195
 Watts, J. W., 185, 186f, 193
 Watts, K. P., 307, 312
 Waziri, R., 346, 357
 Wearing, D., 94
 Weaver, F., 184, 195
 Webster, R., 226, 243
 Weigl, E., 152, 158
 Weinapple, M., 358
 Weinberger, A., 258, 260
 Weinstein, S., 59, 71, 97, 102–103, 117
 Weisenberg, T., 137, 158
 Weiskrantz, L. A., 83, 94, 128, 133
 Weiss, M. S., 250, 262
 Weiss, S., 275, 291
 Weller, M., 347, 359
 Welter, M. L., 194
 Weniger, D., 152, 158
 Wepman, J. M., 152, 158
 Wernicke, C., 9, 137
 Wexler, A., 177
 Wexler, B. E., 349, 353, 359
- Whishaw, I. Q., 20, 55, 70
 Whitaker, H. A., 140, 202, 219, 250, 259
 White, M. J., 227, 244
 Wickens, A., 192
 Wiener, M. S., 274, 288
 Wiesel, T. N., 120, 132
 Willems, P. W., 189, 195
 Williams, J., 163, 178–179
 Willis, T. J., 338
 Wilson, A., 261
 Wilson, B. A., 64, 71, 92, 94, 175, 179, 334, 339
 Wilson, D. H., 200–201, 206, 209, 212, 218, 219, 246, 248, 261–262
 Wilson, S. L., 179
 Winkler, C., 184, 195
 Winocur, G., 334, 339
 Witelson, S. F., 258, 262
 Wodehouse, P. G., 340, 359
 Woerdeman, P. A., 189, 195
 Wolfe, J., 166, 178
 Wolff, H. G., 48
 Wood, C. C., 278, 291
 Wood, R. L., 335, 339
 Woodruff, P. W.R., 348, 358
 Woods, D. L., 279, 287, 289
 Woods, S. P., 184, 194
 Wray, A., 207, 219
 Wyke, M., 251, 262
- Yamada, T., 185, 194
 Yamashita, H., 284, 289
 Yarbush, A. L., 71
 Yeudall, L. T., 347, 356
 Yiend, J., 193
 Yingling, C. D., 276, 289
 Young, A. W., 146, 156, 221, 223, 229, 244, 343, 356
 Yozawitz, A., 349, 355
 Yu, B. M., 129, 133
 Yund, E. W., 246, 260
- Zaidel, D., 212–213, 219
 Zaidel, E., 207, 210, 212–213, 216, 219, 244, 295, 314
 Zakzanis, K. K., 162, 166, 179
 Zangwill, O. L., 10, 114, 117, 140, 158
 Zatorre, R. J., 252, 262
 Zeitlin, G. M., 276, 289
 Zeki, S., 132, 133
 Zeman, A., 212, 219
 Ziegler, J., 146, 156
 Zihl, J., 128, 132, 133
 Zucker, K. J., 296, 312
 Zurif, E. B., 152, 156, 249, 262

Subject Index

Page numbers followed by an *f* or a *t* indicate figures or tables.

- Ability. *see* Intelligence
- Absences, epilepsy and, 89–90
- Abstract thought, 49–50
- Abundance, electroencephalogram and, 268
- Acalculia, 108, 114
- Acoustic agnosia. *see* Auditory agnosia
- Acoustico-motor organization, 324
- Acquired dyslexia, 146, 148
- Activation, 233
- Activity, medial structures and, 66
- Acuteness, lesions and, 47*t*
- Adaptability, prefrontal cortex and, 68*t*
- Addiction, 191
- AER. *see* Auditory evoked response
- Afferent pathways, 181
- Afterbrain, 28*f*
- Age considerations
 - lesions and, 46, 47*t*
 - rehabilitation of aphasics and, 151
- Aggressive behavior
 - orbital cortex and, 63
 - subcortex and, 185
 - temporal lobe and, 84
- Agnosia
 - acoustic, 77
 - associative visual, 126
 - color, 125
 - defined, 77
 - finger, 99, 114
 - somatosensory, 98
 - visual, 78, 125
 - visual spatial, 126
- Agnosic alexia, 127
- Agraphia, 114, 127, 137, 146–150, 154–155*t*, 209
- Akinesia, 184
- Alexia, 127, 137, 146–150, 154–155*t*
- Alexithymia, 66, 352
- Allesthesia, 108
- Alzheimer's disease, 159
- Ambidexterity, 300, 302, 308
- Amblyoptic defect, 121
- Amnesic aphasia, 137, 144, 154–155*t*
- Amnesic syndrome, 80–85, 92*t*
- Amplitude, evoked response and, 272
- Amygdala, 32*f*, 34, 34*f*, 80, 82, 84, 188*f*, 189, 351–352
- Analytic processing, 306
- Anesthesia, 97, 189
- Angular gyrus, 114, 139*f*, 140
- Anhedonia, 63, 66
- Anomic aphasia, 137, 141*f*, 144, 154–155*t*
- Anosognosia, 98
- Anoxia, handedness and, 302
- ANS. *see* Autonomic nervous system
- Anterior, defined, 23*f*, 24
- Anterior aphasia, 137
- Anterior commissure, 36, 37*f*, 201*f*
- Anterior mesial cortex, 140
- Anterior parietal cortex, 96*f*, 115*f*
- Anterior temporal lobectomy, 86
- Anterior thalamus. *see* Thalamus
- Anterograde amnesia, 80, 83
- Anticipation, frontal lobe and, 66
- Antidepressant medications, 183
- Anxiety, 189, 190, 344, 349
- Aphasias
 - assessment of, 150–151
 - classifications of, 154–155*t*
 - defined, 134
 - forms of, 141–150
 - rehabilitation of, 151–153
 - varieties of, 136–139

- Apraxia, 109–112, 283
 Aqueduct of Sylvius, 25*f*
 Arabia, 8
 ARAS. *see* Ascending reticular activating system
 Arcuate fasciculus, 139*f*, 140
 Arithmetical computations, 56–57, 108–109, 115*f*, 296, 306, 308, 326–327. *see also* Acalculia
 Articulatory control, 98
 Ascending reticular activating system, 30, 180
 Asomatognosia, 98–99
 Assessment
 aphasia and, 150–151
 cognitive function and, 50–51
 frontal lobe syndrome and, 64
 intelligence and, 48
 practice and, 329–332
 of specific functions, 323–329
 testing for brain damage and, 318–322
 Association cortex. *see* Tertiary cortex
 Association value, 227
 Associative visual agnosia, 126
 Astereognosis, 98
 Asymbolia, 98, 99
 Ataxia, 161
 Attention
 dichotic listening and, 252–255
 evoked potentials and, 279
 prefrontal cortex and, 61, 68*t*
 temporal lobe and, 78–80, 92*t*
 Attentional models of divided visual field studies, 235*f*, 238–239
 Attenuation, electroencephalogram and, 268
 Audition
 auditory nerves and, 30
 commissurotomy and, 205–206
 comprehension and, 142
 dichotic listening and. *see* Dichotic listening
 epilepsy and, 90
 nerves and, 30
 short-term memory and, 113, 115*f*
 temporal lobe and, 72, 74–77, 74*f*, 92*t*
 Auditory agnosia, 77
 Auditory evoked response, 269, 271*f*
 Automatic behavior, epilepsy and, 89–90
 Autonomic nervous system, 22, 30, 31, 33
 Autotopagnosia, 99
 Avoidance, 344
 Avoidance learning, 35
 Awareness. *see also* Consciousness
 brain injury and, 174*t*
 completion and, 122
 diencephalon and, 31
 states of low awareness and, 170
 Balance, cerebellum and, 31
 Basal ganglia, 28*f*, 32, 34, 52, 162, 181, 183
 Behavioral approach to rehabilitation, 334–335
 Behavioral neurology, 5, 323–325
 Between-object neglect, 105–107
 Biasing system, 66–67
 Bilateralization, 300, 305
 Bimanual skills, 213
 Binocular vision, 226–227
 Biological intelligence, 48
 Bipolar recording, electroencephalogram and, 266
 Birth order, handedness and, 302
 Blackouts, epilepsy and, 89–90
 Blind spots, 122
 Blindsight, 127–128, 285
 Blood pressure, 30
 Body loops, 66
 Body sense, 98–101, 115*f*
 Boston classification, 137–138
 Bradykinesia, 162
 Brain
 lateral view of, *Plate I*
 midsagittal view of, *Plate II*
 Brain imaging. *see* Imaging
 Brain injury, 167–168, 168*f*, 318–322. *see also* *specific injuries*
 Brain stem, 28*f*, 29–31, 29*f*
 Branches of neuropsychology, 4–5
 Breathing, 181
 Brightness, 226
 Brighton Phrenological Institute, 9
 British neuropsychology, 12–13, 13*t*
 Broca's aphasia, 137, 141–142, 154–155*t*
 Broca's area, 51, 51*f*, 61, 68*t*, 137–138, 139–140, 139*f*
 Calcarine fissure, 119*f*, 120
 Callosal agenesis, 200
 Cannabis use, 343
 Capgras syndrome, 342–343
 Caudal, defined, 23, 23*f*
 Caudate, 32*f*, 34
 Cause, lesions and, 47*t*
 Central aphasia, 137, 143–144, 154–155*t*
 Central fissure, 36, 37*f*
 Central nervous system
 brain stem and, 29–31
 cerebellum and, 31
 cerebral cortex and, 35–40
 diencephalon and, 31–35
 divisions of the brain and, 27–29
 environment of, 24–25
 overview of, 22
 pertinent terminology regarding, 23–24, 23*f*
 spinal cord and, 26–27
 Cerebellum, 28*f*, 29, 29*f*, 31
 Cerebral blood flow. *see* Regional cerebral blood flow
 Cerebral commissurotomy. *see* Commissurotomy
 Cerebral cortex, 28*f*, 32–33, 35–40, 37*f*, 39*f*, 139, 280
 Cerebral dementia. *see* Dementia
 Cerebral dominance, 299–301
 Cerebral ischemia, 120
 Cerebral metabolism, 174*t*

372 Subject Index

- Cerebral organization, gender and, 292–297
 Cerebrospinal fluid, defined, 24–25
 Cerebrovascular accidents, 24
 Cheyne-Stokes breathing, 181
 Chlorpromazine, 348
 Chorea, defined, 33
 Chronic progressive multiple sclerosis, 160
 Cingulate gyrus, 34, 34*f*, 61, 188*f*, 189
 Clinical neuropsychology, 4, 10, 11–14, 13*t*
 CNS. *see* Central nervous system
 Cognitive neuropsychology, 17–18
 Cognitive strategies, 230, 334
 Coherence analysis, 268
 Coherence spectrum, 268–269, 347
 Color agnosia, 125
 Color anomia, 125–126
 Color perception, 226
 Coma, 168–169, 174*t*
 Commissures, 36, 37*f*, 201*f*
 Commissurotomy
 consciousness and, 210–211, 211–215
 dichotic listening and, 246
 effects of, 202–211
 overview of, 199–200
 patients and, 200–202
 Common reference recording,
 electroencephalogram and, 267
 Comparative neuropsychology, 5–6
 Completion, 122
 Conduction aphasia, 137, 143–144, 154–155*t*
 Conflicting activity, commissurotomy and, 214
 Consciousness
 commissurotomy and, 210–211, 211–215
 Somatic Marker Hypothesis and, 66–67
 Constructional apraxia, 110–111, 115*f*
 Contralateral limb activation, 107
 Contralateral postcentral gyrus, 96–97
 Contralateral rule, 299–300
 Contre-coup injuries, 122
 Conventional representations, 100*f*
 Coordinate processing, 237
 Corollary discharge, 60
 Coronal plane, defined, 23*f*, 24
 Corpus callosum, 34*f*, 36, 37*f*, 127, 188*f*, 199, 200, 201*f*, 209, 225
 Cortical blindness, 128
 Cortical functioning, integration of, 130–131
 Cotard syndrome, 342, 343
 CP. *see* Chronic progressive multiple sclerosis
 Cranial nerves, brain stem and, 30
 Cross-modal integration, 92*t*
 CSF. *see* Cerebrospinal fluid
 Culture, handedness and, 297
 CVA. *see* Cerebrovascular accidents

 Damage, brain. *see* Brain injury
 Decerebrate animals, 29–30
 Declarative memory, 88
 Deep dyslexia, 146, 148–149
 Deep reflexes, 26
 Degenerative diseases. *see* Diseases

 Déjà vu, 90
 Delusions, 344
 Dementia, 163–164, 320–321, 342
 Demyelination, 160
 Denial, 122
 Depersonalization, 90
 Depression, 59, 62, 189, 190, 303, 343, 344, 352
 Depth perception, 226–227
 Dermatomes, 26
 Design fluency, premotor cortex and, 53, 68*t*
 Detection, 61
 Developmental dysgraphia, 146
 Developmental dyslexia, 146
 Dichotic listening
 attention and, 252–255
 gender and, 295
 handedness and, 305
 index of lateralization and, 255–256
 lateral ear asymmetries and, 247–252
 lateralization and, 136
 overview of, 245
 psychiatric patients and, 349
 technique of, 245–247
 temporal lobe and, 74
 Diencephalon, 28*f*, 31–35, 32*f*
 Direct pathways, 224–225, 225*f*
 Direct testing, 321
 Direct-lexical reading, 147–148, 147*f*
 Discrimination, 61, 320
 Diseases, 159–160, 319, 341. *see also specific diseases*
 Disequilibrium, 184
 Divided visual field studies. *see also* Vision
 anatomy of visual pathways and, 222*f*
 deficits and, 123, 202
 evidence regarding, 224–233
 gender and, 294, 295
 overview of, 220
 psychiatric patients and, 348–349
 technique of, 221–223
 theories of, 234–239
 Dominance. *see* Cerebral dominance;
 Handedness
 Dopamine, 163
 Dorsal, defined, 23, 23*f*
 Dorsolateral prefrontal cortex, 61, 66
 Drastic restructuring, 249
 Drawing, parietal lobe and, 115*f*
 Dressing apraxia, 110, 115
 Drive, orbital cortex and, 66
 Drug use, 343. *see also* Medications
 Dual minds. *see* Commissurotomy
 Dualism, 7
 Dura mater, defined, 24
 Dynamic-structural model of divided visual field studies, 235*f*, 239
 Dysarthria, 98
 Dysesthesia, 97
 Dysgraphia, 146. *see also* Agraphia
 Dyslexia. *see also* Alexia

- Dysphasia, 181, 255
 Dystonia, defined, 33
- Ear advantage, 247
 Ear asymmetries, 247–252
 Echolalic aphasia, 145
 Efferent pathways, 181
 Egypt, 8
 Eipo, 297
 Electrode placement, electroencephalogram and, 266*f*
 Electrophysiology
 electroencephalogram and, 264–269, 274–276. *see also* Electroencephalogram (in Tests Index)
 evoked potentials and, 269–274, 276–279. *see also* Evoked potentials (in Tests Index)
 overview of, 263–264
 technique of, 279–280
 Emergent materialism, 7
 Emergent psychoneural monism, 7
 Emotion
 alexithymia and, 66
 diencephalon and, 31
 epilepsy and, 91
 Emotional tone, 248
 End brain, 28*f*
 Endogenous components, 273
 Enhancement, electroencephalogram and, 268
 Environment, somatic, 66
 Epilepsy, 80, 89, 182–183, 184–185, 187, 264, 341–342, 345
 Equilibrium, auditory nerves and, 30
 Equipotential theory, 9
 Ethical considerations
 brain injury and, 175–176
 research and, 191
 Exception words, reading and, 147–148
 Exhibitionism, 63
 Exogenous components, 273
 Experiential perception, 92*t*
 Experimental neuropsychology, 5, 10–11, 14–17, 257–258, 294, 303
 Expressive aphasia, 137
 Expressive speech, 61, 68*t*
 Extent, lesions and, 47*t*
 Extracerebral reference, electroencephalogram and, 267
 Extralemniscal anatomical pathway, 96
 Extrapyramidal movement, 162
 Eye movement, 57, 58*f*, 68*t*, 258, 350
 Eye opening, Glasgow Coma Scale and, 169*t*
- Facial control, 30, 53, 55, 56*f*
 Facial recognition, 78, 92*t*, 181, 227–229
 Falx, 36
 Fight or flight response, 33
 Finger agnosia, 99, 114
 Fingerprints, handedness and, 298
 Fixation, 221
 Flexion, defined, 27
- Fluent aphasia, 137
 Focal epilepsy, 184–185. *see also* Epilepsy
 Footedness, 298
 Forebrain, 28*f*
 Form, 113
 Form perception, 227
 Fornix, 32*f*, 34, 34*f*, 201*f*
 Free vision, 207, 258
 Frégoli syndrome, 342, 343
 Frequency, stimuli and, 249–250
 Frontal eye fields, 57, 61
 Frontal granular cortex. *see* Prefrontal cortex
 Frontal gyrus, 283
 Frontal lobe
 aphasia and, 145
 Broca's area and, 61
 cerebral cortex and, 36, 37*f*
 four divisions of, 51*f*
 intelligence and, 48–51
 lateralization of, 67–68
 memory and, 88–89
 methodological considerations and, 45–48, 47*t*
 modern theories of functionality of, 64–67
 orbital cortex and, 61–64
 prefrontal cortex and, 54–61
 specific functions of, 51, 68*t*
 Frontal lobe syndrome, 63–64
 Frustrative nonreward, 35
 Functional states, 344, 345–350
 Fusiform gyrus, 72, 73*f*
- Gait, 53, 181–182
 Gegenhalten, 52–53
 Gender
 cerebral organization and, 292–297
 handedness and, 256, 297–309
 lateralization and, 346
 Generalized epilepsy, 89
 Genetic factors, handedness and, 301, 306, 308
 Geniculostriate system, 120–121
 Geography, handedness and, 297
 Gerstmann Syndrome, 113–114
 Global aphasia, 137, 154–155*t*
 Global processing, 306
 Globus pallidus, 32*f*, 34, 84
 Glossopharyngeal nerves, 30
 Grand mal epilepsy, 89
 Grapheme to phoneme conversion, 147–149, 147*f*
 Gray matter, defined, 35
 Greece, 8
 Gyri
 defined, 36
 temporal, 72
- Hair whorl, handedness and, 298
 Half fields, 202
 Hallucinations, 90, 344, 348
 Handedness, 16, 134–136, 135*t*, 203*f*, 256, 274–276, 297–309
 HD. *see* Huntington's disease

374 Subject Index

- Hearing. *see* Audition
Hemianopia, 108
Hemifield advantage, 224
Hemi-inattention, 104–105, 108
Hemisphere lateralization. *see* Lateralization
Hemispheric encoding/retrieval asymmetry model, 237
Hemorrhage, brain damage and, 319
HERA. *see* Hemispheric encoding/retrieval asymmetry model
Herpes simplex encephalitis, 83–84
Heschl's gyrus, 73, 73*f*, 75, 92*t*, 139*f*, 140
Higher Cutaneous and Kinesthetic Functions, 324
Hippocampus, 32*f*, 34, 34*f*, 73, 80, 82, 84, 85, 188*f*, 200, 283
Historical perspective, 8–11, 13*t*
Holistic approach to rehabilitation, 335–336
Homosexual orientation, handedness and, 296
Horizontal plane, defined, 23*f*, 24
Hormones, handedness and, 306
Huntington's Chorea. *see* Huntington's disease
Huntington's disease, 164
Hydrocephalus, 182
Hypersexuality, 91
Hypophonia, 163
Hypothalamus, 28*f*, 29*f*, 32–33, 32*f*, 181, 188*f*, 189
Ideational apraxia, 109–110
Identity, 113
Ideomotor apraxia, 109
IHTT. *see* Interhemispheric transfer time
Illusions, epilepsy and, 90
Imaging, 280–285. *see also* Computerized tomography scan (in Tests Index); Functional magnetic resonance imaging (in Tests Index); Magnetic resonance imaging (in Tests Index); Magnetoencephalography (in Tests Index); Positron emission tomography (in Tests Index); Tractography (in Tests Index)
Impaired Consciousness Research Group, 171
Impaired interhemispheric integration, 351
Impulsivity, orbital cortex and, 63
Index of lateralization, 255–256
Indirect pathways, 224–225, 225*f*
Indirect testing, 321–322
Individual-centered normative approach, 327–329
Inferior, defined, 24
Inferior frontal gyrus, 283
Inferior temporal gyrus, 72, 73*f*, 120
Information-processing model of divided visual field studies, 237
Inhibition, sexual. *see* Sexual behavior
Injury. *see* Brain injury; Trauma
Insula, 72
Intelligence
 assessment and, 326–327
 cerebral cortex and, 35
 clinical neuropsychology and, 4
 frontal lobe and, 48–51
 handedness and, 299, 307–308
 Huntington's disease and, 165–166
 subcortex and, 181
Intensity, stimuli and, 249–250
Intentional movement, 115*f*, 168
Interactionist theory, 10
Interbrain, 28*f*
Interference, 233
Interhemispheric transfer time, 225, 236
Internal capsule, 28*f*
Intersensory association, 113
Intracarotid sodium amyltal, 86
Ipsilateral postcentral gyrus, 96
Isolation syndrome, 137, 145–146, 154–155*t*
Klüver-Bucy syndrome, 91
Korsakoff's disease, 82, 83
Language. *see also* Speech
 anatomical structures and, 139–141, 139*f*
 assessment of aphasia and, 150–151
 classification of aphasias and, 154–155*t*
 commissurotomy and, 206
 forms of aphasia and, 141–150
 gender and, 292–297
 handedness and, 306
 Huntington's disease and, 166
 lateralization of, 134–136
 parietal lobe and, 114, 115*f*
 rehabilitation of aphasics and, 151–153
 right hemisphere and, 212
 temporal lobe and, 92*t*
 varieties of aphasias and, 136–139
Latency jitter, 272, 272*f*
Lateral, defined, 23, 23*f*
Lateral asymmetry, 224, 226, 236, 251. *see also* Dichotic listening; Divided visual field studies; Lateralization
Lateral ear asymmetries, 247–252
Lateral eye movement, 258
Lateral fissure, 36, 37*f*
Lateral geniculate, 222*f*
Lateral globus pallidus, 84
Lateral performance asymmetries, 258
Lateral ventricles, 24–25, 25*f*
Lateral view of brain, *Plate 1*
Lateralization. *see also* Commissurotomy; Dichotic listening; Divided visual field studies
 audition and, 75, 77
 depression and, 303
 electroencephalogram and, 274–276
 frontal lobe and, 67–68
 functional states and, 345–350
 gender and, 294–296
 handedness and, 203*f*, 297, 300
 index of, 255–256
 language and, 134–136
 lesions and, 47*t*

- memory and, 88
- models of, 235*f*
- overview of, 18–19
- parietal lobe and, 103
- somaesthetic presentation and, 205
- surgery and, 185
- theories of, 350–354
- Left handedness. *see* Handedness
- Left hemisphere tasks, 67, 249–250
- Left-right discrimination, 103, 114, 115*f*
- Lemniscal anatomical pathway, 96
- Letter-by-letter reading, 146, 149
- Leukotomy, 185, 185–188, 186*f*
- Lexical decision, 231
- Lexical-semantic reading, 147–148, 147*f*
- Lightness, 237
- Limbic leukotomy, 189
- Limbic system, 28*f*, 34–35, 34*f*, 73
- Linguistic parameters, 230
- LIS. *see* Locked-in syndrome
- Lobectomy, 185
- Lobotomy, 185
- Localization, relative, 38
- Localizationist theory, 9
- Location, 113, 237
- Locked-in syndrome, 27, 168, 172–173, 174*t*
- Locomotion, cerebellum and, 31
- Logic of stimulus presentation, 15
- Longitudinal fissure, 36
- Long-term memory, 92*t*
- Lou Gehrig's disease, 159
- Macropsia, 125
- Macular region, 121
- Mammillary bodies, 34, 34*f*, 82, 84
- Marche a petit pas*, 53, 163
- Masking, 226
- Massa intermedia, 37*f*, 201*f*
- Masturbation, 63
- Materialism, 7
- Mathematical computations. *see* Arithmetical computations
- MCS. *see* Minimally conscious state
- Mechanical skills, 292–297
- Medial, defined, 23, 23*f*
- Medial geniculate body, 74*f*
- Medial temporal cortex, 84, 88–89
- Mediation, verbal, 59
- Medications, 162, 183, 184–185, 187–188, 348, 352
- Medieval thought, 8
- Medulla oblongata, 28*f*, 29, 29*f*
- Memory
 - auditory agnosia and, 77
 - divided visual field studies and, 232
 - electroconvulsive therapy and, 302
 - gender and, 296–297
 - limbic system and, 35
 - parietal lobe and, 113
 - prefrontal cortex and, 60, 68*t*
 - subcortex and, 181
 - temporal lobe and, 80–88, 92*t*
 - tonal, 76
- Meninges, defined, 24
- Meningitis, 24
- Mesencephalon, 28*f*
- Mesial, defined, 72
- Metacognition experiments, 207
- Metamorphopsias, 125
- Metencephalon, 28*f*
- Micropsia, 125
- Midbrain, 28*f*, 29–30, 29*f*
- Middle temporal gyrus, 72, 73*f*, 120, 139*f*
- Midsagittal view of brain, *Plate II*
- Minimally conscious state, 168, 171–172, 174*t*
- Monism, 7
- Monitoring, frontal lobe and, 66
- Monopolar recording, electroencephalogram and, 266–267
- Morse code, 252
- Motion perception, 226–227
- Motivation
 - cortical systems and, 131
 - diencephalon and, 31
 - orbital cortex and, 66
- Motor aphasia, 137, 141–142, 154–155*t*
- Motor cortex, 39*f*, 51, 51*f*, 52–53, 68*t*, 110
- Motor function
 - apraxia and, 102–112
 - assessment and, 326–327
 - brain injury and, 174*t*
 - Glasgow Coma Scale and, 169*t*
 - tertiary level of, 54
- Motor strip of the cortex. *see* Motor cortex
- MS. *see* Multiple sclerosis
- Multiple sclerosis, 159, 160–162
- Muscle tension, 279
- Muscular activity, cerebellum and, 31
- Musical ability, 76–77, 92*t*, 252, 278
- Myelencephalon, 28*f*
- Narrow brain, 28*f*
- Neocortex. *see* Cerebral cortex
- Neuroendocrine disorders, 182–183
- Neurological deficits model, 167*f*
- Neuropsychiatry, 340, 341–345
- Neuropsychology
 - branches of, 4–5
 - clinical, 4, 10, 11–14, 13*t*
 - cognitive, 17–18
 - comparative, 5–6
 - conceptual issues surrounding, 6–8
 - experimental, 5, 10–11, 14–17
 - historical background of, 8–11, 13*t*
 - overview of, 3–4
 - practice of. *see* Practice
- Neurorehabilitation. *see* Rehabilitation
- Neuroses, 344
- Neurosis, 190
- Neurosurgery, 182–185
- Newcastle-upon-Tyne study, 320
- Nonfluent aphasia, 137, 141–142, 154–155*t*

376 Subject Index

- Nonverbal stimuli, 225–229
- Nonvoluntary movement, 162
- North American neuropsychology, 11–12, 13*t*
- Nostril size, handedness and, 298
- Obsessive-compulsive disorder, 189, 190, 342, 343–344
- Occipital lobe
 - anatomy of visual pathways and, 222*f*
 - basic visual functions and, 121–123
 - blindsight and, 127–128
 - cerebral cortex and, 36
 - divisions of, 118–121, 119*f*
 - overview of, 118
 - specific functions of, 130*t*
 - speech and, 77
 - visual perceptual functions and, 123–127
 - visual prostheses and, 128–129
- Occipital pole, 120
- Occupations, handedness and, 308
- OCD. *see* Obsessive-compulsive disorder
- Ocular dominance, 298
- Olfaction
 - epilepsy and, 90
 - nerves and, 30
- Optic chiasm, 201*f*, 222*f*
- Optic nerves, 222*f*
- Optic neuritis, 161
- Optic radiation, 78
- Optic tract, 222*f*
- Orbital cortex, 51, 51*f*, 61–64, 68*t*
- Orbitofrontal cortex. *see* Orbital cortex
- Organic states, 344
- Organizational skills, 305–306
- Orientation, 61
- Out-of-body experiences, 90
- Pain, 96, 99, 182
- Paired associate learning, 88
- Paleocortex, 73
- Palinopsia, 125
- Parahippocampal gyrus, 72–73, 73*f*, 89
- Paralysis agitans. *see* Parkinson's disease
- Paranoid schizophrenia. *see* Schizophrenia
- Paraphasias, 141
- Paraplegia, 161
- Parastriate cortex, 118, 119*f*
- Parietal lobe
 - apraxia and, 109–112
 - cerebral cortex and, 36, 37*f*
 - divisions of, 96*f*
 - Gerstmann Syndrome and, 113–114
 - intersensory association and, 113
 - language and, 114
 - overview of, 95
 - short-term memory and, 113
 - somatosensory perception and, 96–98
 - spatial neglect and, 104–108
 - spatial orientation and, 101–104
 - specific functions of, 115*f*
 - speech and, 77
 - symbolic syntheses and, 108–109
 - tactile perception/body sense and, 98–101
- Parkinsonism, 33, 162, 181
- Parkinson's disease, 162–164, 183–184
- Pathological left-handedness, 299
- Patterned breathing, 181
- PD. *see* Parkinson's disease
- Pelopsia, 125
- Perceptual judgment, prefrontal cortex and, 59–60, 68*t*
- Performance asymmetries, 16
- Peripheral nervous system, 22, 26
- Peristriate cortex, 118, 119*f*
- Peri-Sylvian association cortex, 140, 145
- Permanent vegetative state, 170
- Perseveration, 55
- Persistent vegetative state, 170
- Personality, 4, 61–62, 68*t*, 84, 89–91, 92*t*
- Pharmacological treatment. *see* Medications
- Phase spectrum, 268–269
- Phobias, 190
- Phoneme conversion, 147–149, 147*f*
- Phonemic dyslexia, 148
- Phonetic discrimination, premotor cortex and, 53
- Phonological dyslexia, 146, 149
- Phosphenes, 121
- Photisms, 121
- Phrenology, 9
- Pia mater, defined, 24
- Pitch, assessment and, 326–327
- Planning, sequences of behavior and, 55, 68*t*
- Plasticity, 38
- PNS. *see* Peripheral nervous system
- Polyopsia, 125
- Pons, 28*f*, 29, 29*f*
- Porropsia, 125
- Postcentral gyrus. *see* Sensory strip of the cortex
- Postconcussional syndrome, 341
- Posterior, defined, 23*f*, 24
- Posterior aphasia, 137
- Posterior commissure, 36, 37*f*, 201*f*
- Posterior hypothalamus. *see* Hypothalamus
- Posterior parietal cortex, 61, 96*f*, 114, 115*f*
- Posttraumatic neurosis. *see* Postconcussional syndrome
- Postural mechanisms, cerebellum and, 31
- Posture, writing, 308–309
- Power, electroencephalogram and, 268
- Practice
 - assessment and, 329–332
 - assessment of specific functions and, 323–329
 - overview of, 317–318
 - rehabilitation and, 332–336
 - testing for brain damage and, 318–322
- Practice, divided visual field studies and, 232–233
- Prefrontal cortex, 51, 51*f*, 54–61, 56*f*, 68*t*
- Prefrontal leukotomy. *see* Leukotomy
- Premotor cortex, 51, 51*f*, 52–53, 68*t*

- Prenatal development, 296, 306
 Preplanning, frontal lobe and, 66
 Primary auditory cortex, 74
 Primary cortex, 39–40
 Primary process, 351
 Primary somatosensory cortex, 96
 Primary visual cortex, 120–121, 123, 130*t*
 Priming, 238
 Problem-solving skills, 55–56, 68*t*, 161–162
 Processing determinants, 236–237
 Programming, sequences of behavior and, 55, 68*t*
 Prosencephalon, 28*f*
 Prosopagnosia, 78
 Prostheses, 52
 Pseudo-depression, 59, 62
 Psychodynamic concepts, 350–351
 Psychoses, 344–345
 Psychosurgery, 182, 185–191, 188*f*
 Psychotropic drugs. *see* Medications
 Pure word blindness, 127
 Putamen, 32*f*, 34, 84
 PVS. *see* Permanent vegetative state

 Quasi-spatial syntheses. *see* Symbolic syntheses

 Rage reaction, 33
 rCBF. *see* Regional cerebral blood flow
 Reading. *see also* Alexia
 assessment and, 326–327
 cognitive processes of, 147*f*
 handedness and, 307–308
 occipital lobe and, 130*t*
 parietal lobe and, 114, 115*f*
 Recall, memory and, 85
 Receptive aphasia, 137
 Reflexes
 brain stem and, 30
 defined, 26
 righting, 33
 subcortex and, 180–181
 Regional cerebral blood flow, 16–17, 281, 282–283
 Regional equipotentiality, 10
 Regulation, verbal, 59
 Rehabilitation
 aphasia and, 151–153
 brain injury and, 173–175
 practice and, 332–336
 Reinforcement. *see* Rewards
 Reinstatement of function. *see* Rehabilitation
 Relapsing-remitting multiple sclerosis, 160
 Relative localization, 38
 Relative specialization, 67, 236
 Religion, handedness and, 297
 Renaissance, 8
 Respiratory function, brain injury and, 174*t*
 Response, prefrontal cortex and, 68*t*
 Response strategy, 254
 Reticular formation, 28*f*, 30
 Retrieval deficit, 166

 Rewards, 35, 66
 Rhinencephalon, 28*f*
 Rhythm, assessment and, 326–327
 Right handedness. *see* Handedness
 Right hemisphere tasks, 67, 249–252
 Right shift model, 300–301
 Righting reflexes, 33
 Right-left confusion. *see* Left-right discrimination
 Rigidity, 162–163
 Rolandic fissure, 36, 37*f*, 95
 Rostral, defined, 23, 23*f*
 Route following, 115*f*
 RR. *see* Relapsing-remitting multiple sclerosis
 Russian neuropsychology, 12, 13*t*

 Sagittal plane, defined, 23*f*, 24
 SAS. *see* Supervisory Attentional System
 Schema, defined, 65
 Schizophrenia, 90, 185, 187, 189, 191, 320, 342, 344, 345, 347, 349, 351, 353
 Scotomas, 121
 Screening, 319–320
 Secondary auditory cortex, 75
 Secondary cortex, 39–40
 Secondary somatosensory cortex, 97
 Secondary visual cortex, 120–121, 123, 125
 Seizures. *see* Epilepsy
 Selection of goals, frontal lobe and, 66
 Semantic associations, 113, 130*t*
 Semantic paraphasia, 144
 Sensation. *see* Somatosensory perception
 Sensory aphasia, 137, 142–143, 154–155*t*
 Sensory input. *see also specific senses*
 cranial nerves and, 30
 experimental neuropsychology and, 15
 reading and, 147–148, 147*f*
 temporal lobe and, 79
 Sensory strip of the cortex, 39*f*, 95, 97
 Sensory-motor functions, clinical neuropsychology and, 4
 Septal region, 34, 188*f*
 Septum, 34*f*
 Sequencing, dorsolateral prefrontal cortex and, 66
 SER. *see* Somatosensory evoked response
 Sexual behavior, 35, 63, 90–91, 92*t*, 181, 296
 Short-term memory, 113. *see also* Memory
 Side effects, brain damage and, 319
 Sighting dominance, 298
 Silent reading, 148
 Simple perceptual variables, 226
 Simultanagnosia, 126
 Simultaneous extinction, 108
 Sinistrality, 299. *see also* Handedness
 Site, lesions and, 47*t*
 Sleep-wake cycle, 169, 174*t*
 Smell, olfactory nerves and, 30
 Social behavior, 61, 63, 68*t*, 89–90
 Sodium amytal, 86, 302, 346
 Somaesthetic presentation, 205

378 Subject Index

- Somatic complaints, 190
- Somatic environment, 66
- Somatic Marker Hypothesis, 66–67
- Somatoparaphrenia, 98–99
- Somatosensory agnosia, 98
- Somatosensory evoked response, 269
- Somatosensory perception, 96–98, 115*f*, 271*f*
- Spatial long-term memory, 92*t*
- Spatial neglect, 104–108
- Spatial orientation, 101–104, 115*f*, 292–297, 306, 308, 326–327
- Special reflexes, 26
- Specialization, relative, 67
- Specific lateralized cerebral dysfunction, 351
- Speech. *see also* Language
 - assessment and, 326–327
 - cortical systems and, 131
 - expressive, 61, 68*t*
 - handedness and, 135*t*, 302, 306
 - as left hemisphere task, 249
 - reading and, 147*f*
 - short-term memory and, 113
 - temporal lobe and, 92*t*
- Spelling, premotor cortex and, 53, 68*t*
- Spinal cord, 26–27, 28*f*
- Spinal injuries, prostheses and, 52
- Splenium, 120
- Split-brain operation. *see* Commissurotomy
- Sports, handedness and, 308
- Stability, lesions and, 47*t*
- Stages of processing, 237
- States of low awareness, 170
- Stereotactic subcaudate tractotomy, 188
- Stimuli
 - brain injury and, 174*t*
 - dichotic listening and, 248
 - flashed, 127–128
 - logic of presentation of, 15
 - nonverbal, 225–229
 - somatosensory patterns and, 66
 - temporal lobe and, 75, 79, 92*t*
 - verbal, 229–233
- Stimulus enumeration, 226–227
- Stress, handedness and, 299, 302, 306
- Striate cortex, 118, 119*f*, 120, 121, 222*f*
- Stroke, 319, 352
- Stroop phenomenon, 55
- Structural models of divided visual field studies, 234–237, 235*f*
- Subcaudate tractotomy, 189
- Subcortex, 180–182
- Subcortical forebrain. *see* Diencephalon
- Subcortical visual system. *see* Secondary visual cortex
- Subtraction technique, 281
- Suicidality, 162, 166, 189
- Sulci, 36
- Superficial reflexes, 26
- Superior, defined, 24
- Superior colliculus, 222*f*
- Superior temporal gyrus, 72, 73*f*, 77, 139*f*
- Supervisory Attentional System, 65–66, 65*f*
- Supramarginal gyrus, 139*f*, 140
- Surface dyslexia, 146, 149–150
- Surgery
 - commissurotomy and. *see* Commissurotomy
 - neurosurgery and, 182–185
 - psychosurgery and, 182, 185–191, 188*f*
 - resulting amnesic syndrome and, 80
 - techniques used during, 75, 76*f*
- Sylvian fissure, 36, 37*f*, 97, 140, 348
- Symbolic syntheses, 108–109
- Syntactic closure, 277
- Tactile asymmetries, 295, 296
- Tactile perception, 98–101, 115*f*, 257–258, 326–327
- Taste, nerves and, 30
- Tectopulvinar system, 120–121
- Telegraphic speech, 141
- Telencephalon, 28*f*. *see also* Cerebral cortex
- Teleopsia, 125
- Temperature, somatosensory perception and, 96
- Temporal lobe
 - attention and, 78–80
 - audition and, 74–77
 - cerebral cortex and, 35, 36, 37*f*
 - medial structures and, 84, 88–89
 - memory and, 80–88, 84*f*
 - personality and, 89–91
 - specific functions of, 92*t*
 - speech and, 77
 - vision and, 78
 - visual perceptual functions and, 123
- Temporal lobectomy, 85–88
- Temporal pole, 73*f*
- Temporo-parieto-occipital junction, 126
- Tension, 190
- Tertiary auditory cortex, 77
- Tertiary cortex, 39–40
- Tertiary level of motor control, 54, 68*t*
- Tertiary visual cortex, 78, 92*t*
- Testicles, handedness and, 298
- Testing. *see* Assessment; Tests Index
- Thalamus, 28*f*, 29*f*, 32–33, 32*f*, 34*f*, 52, 61, 82, 84, 172, 188*f*
- Thought disorder, 348
- Timbre, 76
- Time, stimuli and, 249–250
- Tonal memory, 76
- Topographical perception, 115
- Touch, somatosensory perception and, 96
- Toxicity, brain damage and, 319
- Training programs, 334
- Tranquilizers, 348
- Transcortical motor aphasia, 137, 140, 145, 154–155*t*
- Transcortical sensory aphasia, 137, 145–146, 154–155*t*
- Transverse plane, defined, 23*f*, 24

- Trauma
 brain damage and, 319
 clinical neuropsychology and, 4
 handedness and, 299
 prefrontal cortex and, 61
Tremor, 163
Two-point threshold, 96
- Unconventional representations, 100*f*
Uncus, 73*f*, 80
Unilateral neglect, 104–105, 106*f*, 107, 115
- Vagus nerves, 30
Valence model, 351–352
Vegetative state, 168, 169–171, 174*t*
Ventral, defined, 23, 23*f*
Ventricular system, 24–25, 25*f*
Ventriloquism effect, 253–254
Ventrolateral frontal cortex, 61
VER. *see* Visual evoked response
Verbal ability, Glasgow Coma Scale and, 169*t*
Verbal fluency, 53, 59, 68*t*, 292–297. *see also*
 Language
Verbal long-term memory, 92*t*
Verbal mediation, 59
Verbal regulation, 59, 68*t*
Verbal stimuli, 229–233
Vigilance paradigm, 226
Violence, 63, 185
Visceral brain. *see* Limbic system
Vision
 anatomy of visual pathways and, 222*f*
 commissurotomy and, 204–205
 divided visual field studies and. *see* Divided
 visual field studies
 epilepsy and, 90
 free, 207, 258
 nerves and, 30
 occipital lobe and, 120, 121–123
 optic nerves and, 30
 temporal lobe and, 72, 78, 92*t*
Visual agnosia, 78, 125
Visual evoked response, 269, 271*f*
Visual field advantage, 224
Visual field asymmetry, 224
Visual field deficits, 123, 202. *see also* Divided
 visual field studies
Visual object agnosia, 99
Visual perception, 123–127, 130*t*, 326–327
Visual prostheses, 128–129
Visual reflexes, 30
Visual spatial agnosia, 126
Visuospatial agnosia, 101
Visuospatial properties, 113, 164, 166
Vocabulary, assessment and, 321–322
Voluntary eye movement, 57, 58*f*, 68*t*
VS. *see* Vegetative state
- Weight, of brain, 3
Wernicke-Korsakoff syndrome, 82
Wernicke's aphasia, 137, 142–143, 154–155*t*
Wernicke's area, 77, 137–138, 139*f*, 140
Wernicke's encephalopathy, 82
Whiplash effect, 122
Whole word reading route, 147–148, 147*f*
Within-object neglect, 105–107
Witzelsucht, 62
Working memory, 60–61
World War I, 9, 10
World War II, 10, 124, 319
Writing. *see also* Agraphia
 assessment and, 326–327
 commissurotomy and, 206–207
 handedness and, 306, 308–309
 reading and, 147*f*
 temporal lobe and, 92*t*

Index of Tests and Procedures

Page numbers followed by an *f* or *t* indicate figures or tables.

- AEPs. *see* Evoked potentials
AER. *see* Auditory evoked response
Air encephalography, 25
Allen Index, 320
Aphasia therapy, 333
Aubert task, 59–60
Auditory ambiguity task, 278
Auditory evoked response, 269, 271*f*
Averaged evoked potentials, 269. *see also*
 Evoked potentials
- Background Interference Procedure, 320
BADS. *see* Behavioral Assessment of the
 Dysexecutive Syndrome
BDAE. *see* Boston Diagnostic Aphasia
 Examination
Beck Anxiety and Depression Scales, 332–333
Behavioral Assessment of the Dysexecutive
 Syndrome, 64, 328, 331
Bender Gestalt Test, 320
Benton Visual Retention Test, 102, 102*f*, 110–
 111, 320
Block design task. *see* Kohs block design task
Boston Diagnostic Aphasia Examination, 150
Brain function therapy, 333
Brixton Test, 331
Brodmann's cytoarchitectonic maps, 36
- Camden Memory Tests, 331
CAT scan. *see* Computerized tomography scan
Category Test, 49
CAVE. *see* Cognitive Assessment by Visual
 Election
CBT. *see* Cognitive behavior therapy
CET. *see* Cognitive Estimates Test
Chimeric figures, 204–205, 204*f*, 208*f*
Cognitive Assessment by Visual Election, 172
Cognitive behavior therapy, 319
Cognitive Estimates Test, 64
Commissurotomy operation, 11
Compensation Therapy, 152
Computerized tomography scan, 263, 317, *Plate*
 III
Corsi block tapping task, 85, 86*f*
CT scan. *see* Computerized tomography scan
- Deblocking Therapy, 152
Design Copying, 67
Dichhaptic technique, 258
Dichotic Monitoring Test, 255
Diffusion-tensor imaging, 284
Digit Span, 320
Digit Symbol, 320
Doors and People Test, 331
DTI. *see* Diffusion-tensor imaging
Dual task experiments, 295, 296
- ECT. *see* Electroconvulsive therapy
EEG. *see* Electroencephalogram
Electroconvulsive therapy, 302–303, 346–347
Electroencephalogram, 17, 174*t*, 264–269, 265*f*,
 266*f*, 274–276, 347. *see also* Evoked
 potentials
Electromyography, 161
Embedded figures, 126, 127*f*
Encephalography, 25
EPs. *see* Evoked potentials
ERPs. *see* Evoked potentials
Event-related potentials, 269. *see also* Evoked
 potentials
Evoked potentials, 269–274, 270*f*, 271*f*, 272*f*,
 276–279, 284

- Fiber tractography, 284
 Finger Oscillation Test, 325
 Finger pulse volume, 279
 fMRI. *see* Functional magnetic resonance imaging
 Frenchay Aphasia Screening Test, 151
 FT. *see* Fiber tractography
 Functional magnetic resonance imaging, 171, 263, 281–282, 296, 302, *Plate V*
- Garvin figures, 227, 228*f*
 GCS. *see* Glasgow Coma Scale
 Glasgow Coma Scale, 169, 169*t*, 174*t*
 Gollin Figures, 83*f*, 100
 Gorham Proverbs Test, 67
- Halstead Category Test, 49*f*, 325, 328
 Halstead-Reitan Neuropsychological Test Battery, 12, 321, 325–327
 Halstead-Wepman Aphasia Screening Test, 151, 325
- Imaging, 280–285. *see also* Computerized tomography scan; Functional magnetic resonance imaging; Magnetic resonance imaging; Magnetoencephalography; Positron Emission Technology; Tractography
 Impressive Speech, 324
 In-between test, 99
- Kohs block design task, 55–56, 57*f*, 67, 111, 274
- Locomotor Map Following Task, 102–103, 103*f*
 Logical Memory subtest, 87
 Luria-Nebraska Neuropsychological Battery, 12, 321, 325–327
- Magnetic resonance imaging, 82, 161, 281, 283, *Plate IV*
 Magnetoencephalography, 283–284, *Plate VI*
 McGill Picture Anomalies Test, 79, 79*f*
 Meaningful Sounds Identification Test, 77
 MEG. *see* Magnetoencephalography
 Melodic Intonation Therapy, 152
 Memory for Designs Test, 320
 Minnesota Test for Differential Diagnosis of Aphasia, 150
 Mirror Drawing Task, 81*f*, 82–83
 Modified New World Learning Test, 321
 Money Road Map Test, 103, 104*f*
 Mooney Closure Faces Test, 100, 100*f*
 MRI. *see* Magnetic resonance imaging
- NART. *see* New Adult Reading Test
 Nerve conduction studies. *see* Electromyography
 New Adult Reading Test, 322
 Novelty tasks, 88
- Ongoing EEG. *see* Electroencephalogram
 Oxfordshire Villages Survey, 307
- PALPA. *see* Psycholinguistic Assessment of Language Processing in Aphasia
 PET. *see* Positron Emission Technology
 Phonological task, 296
 PICA. *see* Porch Index of Communicative Abilities
 Pool Reflections Test, 101, 101*f*
 Porch Index of Communicative Abilities, 150–151
 Porteus mazes, 56*f*
 Positive programming, 335
 Positron Emission Technology, 174*t*, 282–283, 348
 Psycholinguistic Assessment of Language Processing in Aphasia, 151
- Reality orientation therapy, 335
 Reitan Battery, 102, 346
 Reporter's Test, 151
 Rey-Osterrieth Figure, 87–88, 87*f*
 Rhythm Test, 325
 Rivermead Behavioural Memory Test, 331
 Road Map Test. *see* Money Road Map Test
- Schuell Short Examination for Aphasia, 150
 Seashore Tests of Musical Ability, 76, 325
 Seguin-Goddard Formboard, 98, 102
 Semmes Maps, 102–103, 103*f*
 Sensory Modality Assessment and Rehabilitation Technique, 175
 SER. *see* Somatosensory evoked response
 Skin conductance, 279
 SMART. *see* Sensory Modality Assessment and Rehabilitation Technique
 Somatosensory evoked response, 269, 271*f*
 Spatial Span, 84, 85
 Split-brain operation, 11
 SSEA. *see* Schuell Short Examination for Aphasia
 Standardized Version of Luria's Neuropsychological Techniques, 326
 Stick Construction Test, 102, 111
 Strength of Grip Test, 325
- Tactual Performance Test, 102, 325
 Task irrelevant probes, 278
 Three-Dimensional Construction Praxis, 111, 111*f*
 Time Orientation, 67
 Token Test, 151
 Torque Test, 258
 Tractography, 284–285
 Trail Making Task, 320, 328, 331
 two-point finger test, 99
- Unconventional Views of Objects Test, 100, 100*f*
- Vanderplas figures, 227, 228*f*
 Ventriculography, 25
 VER. *see* Visual evoked response

382 Index of Tests and Procedures

- Verbal Fluency Test, 67, 328
- Verbal Learning, 67
- Visual ambiguity task, 278
- Visual Communication Therapy, 152
- Visual evoked response, 269, 271*f*
- Visual Object and Space Perception Battery, 101
- Visuospatial task, 296
- VOSP. *see* Visual Object and Space Perception Battery
- Wada Test, 16, 86, 87, 88, 135*t*, 185, 255, 302, 303, 346
- WAIS. *see* Wechsler Adult Intelligence Scale
- Waveform averaging, 270*f*
- Wechsler Adult Intelligence Scale, 294, 307, 320, 325, 328, 331
- Wechsler Memory Scale, 85, 87, 331
- Wechsler Test of Adult Reading, 322, 330–331
- Weigl Sorting Test, 115*f*
- Weinstein Maps, 102–103, 103*f*
- Wessex Head Injury Matrix, 175
- Western Aphasia Battery, 138, 151
- WHIM. *see* Wessex Head Injury Matrix
- Wisconsin Card Scoring Test, 54–55, 54*f*, 164, 328
- WTAR. *see* Wechsler Test of Adult Reading
- X-ray photography, 25, 280. *see also* Computerized tomography scan