Fractured Minds: A Case-Study Approach to Clinical Neuropsychology, Second Edition

Jenni A. Ogden

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In memory of my mother,
who taught me that the mind and
spirit can rise above even the
most disabling illnesses,
and for my first two granddaughters,
Sophie and Belize,
born during the revision
of this book and with
their whole lives ahead of them.

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PREFACE

The second edition of *Fractured Minds* follows the same format as the previous edition, with two introductory chapters followed by 17 chapters that each focus on a different neurological disorder. In each of these chapters a theoretical section precedes an illustrative case study. All the chapters from the first edition have been revised to include pertinent new research findings, new treatments, and further discussion of many issues important to the discipline and profession of clinical neuropsychology. The original cases have, with one exception, been retained, and for some cases additional recent case material has been added. Thus the reader can discover what has happened to Rangi, following many years free of epilepsy, and Mark, the (now middle-aged) man with visual object agnosia and prosopagnosia. A new case has replaced the original case in the subarachnoid hemorrhage chapter, allowing the inclusion of an illustrative neuropsychological assessment report that includes the latest Wechsler scales.

Three entirely new chapters on multiple sclerosis, Parkinson's disease, and Huntington's disease have been added. These not only make the range of neurological disorders covered much broader but also provide a vehicle for describing and exploring motor disorders, neurosurgical treatment to alleviate motor symptoms, genetically inherited disorders, genetic counseling, gene therapy, and some of the many difficult ethical issues that go hand-in-hand with new technology and knowledge.

Throughout, my aim has been to reveal the people who stand behind each disorder: patients, families, clinicians, support groups, and researchers. I hope that their courageous and sad stories will enhance the reader's understanding of neurological disease processes and modern treatments, as well as add depth and color to my descriptions of how clinical neuropsychologists go about their business of unraveling the mysteries of brain–behavior relationships.

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Preface to the First Edition

I wrote this book for two reasons. My first reason was to relate the stories of some of the neurological patients who, over the years, have graciously admitted me into their lives, albeit in most cases for a brief time. The primary reason for my acquaintance with these people was either to assess their cognitive and psychological functioning and assist them with rehabilitation or to involve them in research projects with the goal of increasing our understanding of higher-level brain functioning.

My personal gain from contact with neurological patients is immeasurable. I have not only been privileged with intriguing glimpses into the human mind, but these people have also humbled me with their courage, humor, generosity of spirit, and determination to triumph over their illnesses, and for some, their uncomplaining acceptance of irretrievable losses and the certainty of an untimely death. As a PhD student new to neurosurgery and neurology ward rounds, I sometimes had to turn away from the bed so that the patient (and doctors) would not notice the tears in my eyes. Over the years I have conquered this "unprofessional" behavior and replaced it with a more subtle lump in my throat in response to the tragic stories, often masked by the brisk medical protocol that is commonplace in these wards. By sharing my experiences of how these "ordinary" folk cope with the extraordinary stress of a brain disorder, I hope to pass on something of their lives to the readers of this book.

My second reason for writing this book was to satisfy a need for an introductory text in clinical neuropsychology that would capture the attention of students and other health professionals interested in gaining a broad understanding of clinical neuropsychology, but without being required to learn "how to do it." The experiences of people with damaged brains and disordered minds seem to be intrinsically interesting to most people, perhaps because we can all relate in some small way to forgetting important information, not being able to say a word although we know we know it, or becoming clumsy and inefficient when we are overtired or intoxicated. Neurological disorders of one sort or another are common, and few people reach midlife without being touched by a family member or close friend with a head injury, dementia, stroke, or other neurological problem.

My own university teaching has convinced me that relating stories about real people as a pathway into the mysteries of neurological disorders and how they affect the mind generally works better than describing complex research studies and force-feeding facts about neuroanatomy and neuropathology. For example, an excursion into the world of patient H.M. not only results in an "emotional" understanding of what it might be like to have no memory but also introduces the reader or listener to the medial temporal lobes, epilepsy, and the many ways memory can be categorized.

Important theories and research studies pertinent to H.M.'s amnesia and other memory impairments can be included to give a general overview of the area and to encourage the interested reader to pursue them in more detail. Some of the neuropsychological tests commonly used to assess memory and other cognitive impairments can be described in general terms, and ethical, cultural, and other issues that are part and parcel of the clinical neuropsychologist's practice can be presented and discussed when they arise in the "natural" course of the case presentation.

This book thus represents a series of readings in introductory clinical neuropsychology. I hope that by the end of the book, the reader will have a broad view of clinical neuropsychology that will be sufficient in itself for many readers and that will encourage and prepare others for more advanced study. Each chapter includes a moderate number of references so that students can use the book as a springboard for further serious study. Chapters can be read (or used as a basis for a lecture, laboratory, or tutorial) in any order. However, for those new to this field, reading Chapters 1 and 2 first will provide a basis for understanding the following 14 chapters, each of which is centered around a particular neuropsychological disorder as experienced by one or two patients. Chapter 1 provides an overview of many different aspects of neuropsychology, including basic neuroanatomy, important assumptions and concepts understood by neuropsychologists, and a demystification of the "jargon" used in this field. Chapter 2 takes the reader through the steps of a neuropsychological assessment and briefly describes the more common tests referred to in the case studies.

Each case study is preceded by a section covering the main theoretical and neuropathological aspects of the disorder that often includes a sample of the relevant research in the area. Some chapters focus on the clinical assessment, treatment, and rehabilitation of common disorders, such as head injury, epilepsy, and dementia; others describe in straightforward language the research that is conducted to understand less common but fascinating disorders, such as the inability to recognize faces and objects by sight.

Although this book was written primarily for college and university students, it may also be of interest to health professionals who work with neurological patients. For example, practicing as well as student clinical psychologists; physical, speech, and occupational therapists; nurses; and junior doctors who work with neurological patients need to understand the kinds of cognitive impairments and other difficulties experienced by these people without needing to know in detail how to assess and rehabilitate the problems themselves. Increasingly, a multidisciplinary approach is taken in the assessment, treatment, and rehabilitation of neurological patients. For such an approach to work effectively, it is important that each professional understands, at least in a general way, the concepts and assessment measures used by other professionals. In addition, many of the ethical and professional issues discussed in the book are common to all health professionals. Sometimes patients (and their families) ask for books they can read to help them better understand their disorders. I hope one or more of the chapters in this book will assist in fulfilling that need for some people.

This is not a test manual or a text to prepare the reader to practice as a clinical

neuropsychologist, but its breadth and detail may be sufficient to convey the richness and fascination of working with this population. Individual cases are of real people whose names and other personal details have been changed to protect their identity. In no specific case has the patient or client's gender been changed, as this is often an important factor in that patient's assessment and rehabilitation. When writing in general terms about patients, clients, and health professionals, however, to avoid the clumsy and impersonal use of *she/he* and *her/his*, I have used one or the other pronoun in a fairly random manner. Of course, generally speaking, in all these instances I could have used the alternative pronoun just as easily.

I must accept responsibility for the ways in which I have expressed the different aspects of neuropsychology in this book, and I am sure many readers will disagree with me on a number of issues. Indeed, given the ever-increasing body of neuropsychological knowledge that pours out of scientific journals every month, many of the ideas expressed in this book may well be outdated by the time it appears on the shelf. That aside, I could not have written this book without the massed wisdom and help of numerous people over the years.

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ACKNOWLEDGMENTS

I am indebted to all the patients and their families who willingly gave up their time and cooperated with my tests and interviews, often when they themselves were going through one of the most significant crises of their lives. Although I am especially grateful to the 18 people and their families whose particular stories I have told here, I also thank the hundreds of patients who have, over the years, taught me much of what I know. I especially thank my own academic teachers and mentors, Michael Corballis and Suzanne Corkin, and Dorothy Gronwall, whose death in 2001 was a sad occasion, but whose legacy lives on. My mentors have supported me as well as taught me, and have even put up with my argumentative nature with good grace. Every clinical neuropsychologist needs a neurosurgeon or neurologist alongside, and Edward Mee has fulfilled that role for me. Not only has he taught me much about neuroanatomy, neuropathology, neurology, and even neurosurgery, but he has also maintained my belief that all health professionals, even busy surgeons, can take the time to care about the whole person, and not just the disorder that person endures.

I thank the staffs of the University of Auckland Department of Psychology, the Department of Neurology and Neurosurgery of Auckland Hospital, and the Clinical Research Center at Massachusetts Institute of Technology, all of whom have assisted my clinical, academic, and research endeavors in numerous ways over the years. The New Zealand Neurological Foundation, Inc., and the Health Research Council of New Zealand have supported many of my research projects, some of which have found their way into this book. Many of my colleagues and students have in various ways influenced and guided my neuropsychological thinking and clinical practice, encouraged me to write this book, and read drafts of chapters. In particular, I acknowledge Joe Bogen, Edith Kaplan, Muriel Lezak, Garry McFarlane-Nathan, Anne Aimola Davies (nee Maguire), Erana Cooper, Jennifer Hume, Laurie Miller, Gill Rhodes, Fred Seymour, Jon Simcock, Lynette Tippett, Guy Von Sturmer, and Kevin Walsh. I thank Meryl Hawkins, who drew the brains in Chapters 1 and 5, and my editor at Oxford University Press, Jeffrey House, who took a punt on a new author who wanted to write a novel disguised as a textbook, and who then asked for a second edition. I thank Fiona Stevens for all her guidance in the production of the second edition.

Finally, I thank my husband, John, and my children, Caroline, Jonathan, Josie, and Joachim, who have been listening to my stories about H.M., Michael, and others over the dinner table for more years than they care to remember. They may not feel obliged to take the second edition of the book to bed, but perhaps they will read it to my grandchildren to put them to sleep!

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CONTENTS

Fractured Minds

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1

Introduction to Clinical Neuropsychology

A Definition of Clinical Neuropsychology and Its Aims

This book is concerned with the lives of real people whose behavior, emotions, or thinking abilities have become disordered, disrupted, or unusual as a result of some type of brain disorder or damage. The study of human behaviors, emotions, and thoughts and how they relate to the brain, particularly the damaged brain, is the subject matter of clinical neuropsychology.

Clinical neuropsychology has both applied and academic aims. Applied aims include learning more about neurological disorders and diseases so that we can more accurately and usefully diagnose, treat, and rehabilitate people who suffer such disorders and, along with other disciplines, ultimately find ways to prevent their occurrence. The primary academic aim is to learn more about how the undamaged or "normal" human brain and mind work by carrying out experiments, usually in the form of cognitive tests, on brain-damaged people.

This introductory chapter describes the similarities and differences between clinical neuropsychology and other related disciplines. It then touches on functional neuroanatomy, important neuropsychological terms and concepts, the interaction of clinical practice and research, the roles of a clinical neuropsychologist, and cross-cultural issues in neuropsychology. Each of these topics demands a chapter or book to itself, and a few paragraphs on each will act only as a reminder of knowledge you already have or provide just enough material to help you understand most of the information in the case studies. To provide a general sense of the basic tools the neuropsychologist uses to understand what is going on in the minds of brain-damaged patients or clients, Chapter 2 describes the different aspects of the neuropsychological assessment. Chapters 3 to 19 each present one or two case studies chosen to illustrate particular neuropsychological disorders, such as aphasia, visual agnosia, and dementia. A number of other issues important to the clinical neuropsychologist are raised throughout the case studies. At the end of this introductory chapter is a list of topics keyed to the chapters that provide further information about them.

Relationship of Clinical Neuropsychology to Other Disciplines

A number of disciplines are closely related to clinical neuropsychology and overlap with it (Fig. 1-1). The main ones can best be conceptualized as a continuum with the brain at one end (neurology) and the mind at the other (cognitive psychology). *Neurology* is the study of the medical aspects of central nervous system disorders and treatments. Compared with neuropsychologists, neurologists tend to be more concerned with clinical symptoms and signs as indications of underlying neuropathology in the brain, spinal cord, and peripheral nervous system and less concerned with the details of the higher behaviors and cognitions mediated by the brain and how the detailed study of their breakdown can inform us about normal higher cognitive processes.

At the other, more academic, end of the spectrum lies *cognitive psychology*, a popular subdiscipline of academic psychology. Its aim is to understand the workings of the human mind by analyzing the higher cognitive functions and their components. Participants in cognitive psychology experiments are unimpaired people (usually undergraduate university students) rather than brain-damaged patients, and cognitive psychologists have developed many important experimental paradigms that allow measurement of minute differences in cognitive performance under controlled conditions. For example, the time required to perform different tasks or a single task under different conditions might be measured in milliseconds, and from these results inferences can be made about the cognitive processes underlying the behaviors.

Cognitive neuropsychology is a relatively recent label for a type of research that many neuropsychologists have been conducting for years. It is, as the name suggests, a hybrid of cognitive psychology and clinical neuropsychology. It concentrates on the detailed analysis of higher cognitive functions, often using similar paradigms to those used in cognitive psychology, but it studies brain-damaged patients rather than "normals" (McCarthy and Warrington 1990). In their hypotheses and analyses of deficits and their implications for the normal functioning of the brain, cognitive neuropsy-

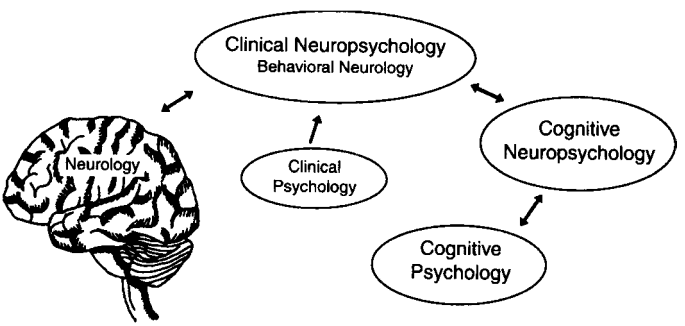

Figure 1-1 The discipline of clinical neuropsychology in relation to neurology and psychology.

chologists, although certainly not ignoring the brain entirely, tend to be less interested than clinical neuropsychologists in where the damage is and how it might be related to the impairment. Similarly, they are not interested in brain pathology, disease, and treatment on their own per se, but only as a means to the end of understanding the workings of the normal mind.

Thus, *clinical neuropsychology* positions itself between neurology and cognitive neuropsychology. It has a neurological interest in brain pathology and the resulting symptoms and a psychological interest in the analysis of higher cognitive functions, both to understand the workings of the normal mind and to develop better rehabilitation methods for patients. In practice, all the disciplines in Figure 1-1 overlap considerably, and many practitioners and researchers straddle two or more of these. Some neurologists specialize in clinical neuropsychology, and they are often known as *behavioral neurologists*. Clinical neuropsychologists who have an affiliation with a university psychology department as well as a hospital often carry out research that would best fit into the cognitive neuropsychology category. This is well illustrated by some of the case studies in this book that are more closely aligned with cognitive neuropsychology than clinical neuropsychology (see Chapters 3, 6, 8, and 19).

Other important areas that contribute to clinical neuropsychology include animal psychology and neuroscience, neuropharmacology, and human neurophysiology. This latter discipline measures the electrical brain waves of patients using electroencephalographs (EEG) and evoked potentials. In recent years rapidly developing neuroimaging technology has changed the face of neuroscience, and clinical neuropsychology has been one of the greatest beneficiaries. Computed tomography (CT) (see Chapters 6, 7, and 19 for examples) and magnetic resonance imaging (MRI) (see Chapter 8 and 14 for examples) permit us to visualize the anatomic structures and damage in the living brain, while cerebral blood flow techniques, positron emission tomography (PET), and functional MRI allow us to visualize the changing metabolism of the working brain. The relevance of these latter techniques lies in their potential to confirm and extend our hypotheses about brain–behavior relations. That is, when a non-brain-damaged person is speaking, does Broca's area (hypothesized to mediate speech) "light up" on a PET scan? Alternatively, when a patient has a large lesion in Broca's area (as confirmed on a CT brain scan) but still manages to speak, what area of the brain "lights up" when a PET scan is carried out on this patient?

Finally, but importantly, a practicing clinical neuropsychologist should first be an accomplished clinical psychologist, as will become evident in many of the case studies that follow. Even clinical neuropsychologists who restrict themselves to assessment and do not take an active part in rehabilitation and therapy require some clinical skills to enable them to build the rapport necessary to achieve a valid and useful assessment and to discuss in a sensitive manner the often distressing information about a patient's performance. In addition, patients often express strong emotions about their illness and their wider situation during their assessment, especially during the initial interview, and the clinical neuropsychologist should be able to respond professionally and sensitively. People with stable, long-term lesions who have volunteered as research

subjects are also entitled to sensitive treatment that does not exploit or disempower them.

Functional Neuroanatomy

The human brain is the most complex system in the animal kingdom, and it is well beyond the scope of this book to cover neuroanatomy in any detail. This section provides a brief, simplistic overview of the cortical areas and other neuroanatomical structures that are most closely related to the disorders of higher cortical functioning covered in this book. This section should serve as a reminder for readers who have studied neuroanatomy and provide some background for those who have not. For readers who wish to learn more about this important area, the neuropsychology texts by Lezak (1995) and Walsh (1994) have excellent, easy-to-read sections on neuroatomy for neuropsychologists; more detailed descriptions of neuroanatomy can be found in Mesulam (1985).

Gross Structure of the Brain

The brain has three major divisions: the cerebral hemispheres, the cerebellum, and the brain stem. Neuropsychology is most concerned with the cerebral hemispheres. Figure 1-2 shows lateral (from the side) and medial (split down the middle from front to back) views of the human brain. The *brain stem*, an upward extension of the spinal cord, consists of four parts: the medulla oblongata, pons, midbrain, and diencephalon. It is the life-support part of the brain as it controls respiration, cardiovascular function, and gastrointestinal function. It also contains the nuclei for the cranial nerves connected with the special senses, but it is not directly concerned with higher cognitive function. The *cerebellar hemispheres* are paired structures at the base of the cerebral hemispheres and are concerned mainly with motor coordination, muscle tone, and balance.

The *cerebral hemispheres* are paired structures above the midbrain and pons. They are covered by a highly convoluted layer of nerve cells called the *cerebral cortex*, or *grey matter*. The "hills" of the cortex are called *gyri* (singular, gyrus) and the "valleys" are called *sulci* (singular, sulcus). The axons or fiber tracts that connect the nerve cells to the rest of the brain form a layer directly below the cortex called the *white matter*. Deep within the hemispheres are further paired structures of grey matter called the *basal ganglia*. Chapter 15, which describes Parkinson's disease, provides more detail about the basal ganglia and their connections. The two hemispheres are separated by the *longitudinal fissure*, a deep groove that runs from the anterior frontal lobes to the posterior occipital lobes. The other main fissures are the *central (or rolandic) fissure* or sulcus, which separates the frontal from the parietal lobe, and the *lateral (or sylvian) fissure* or sulcus, which separates the temporal lobe from the frontal and parietal lobes. A tough band of interhemispheric fibers called the *corpus callosum* forms the major

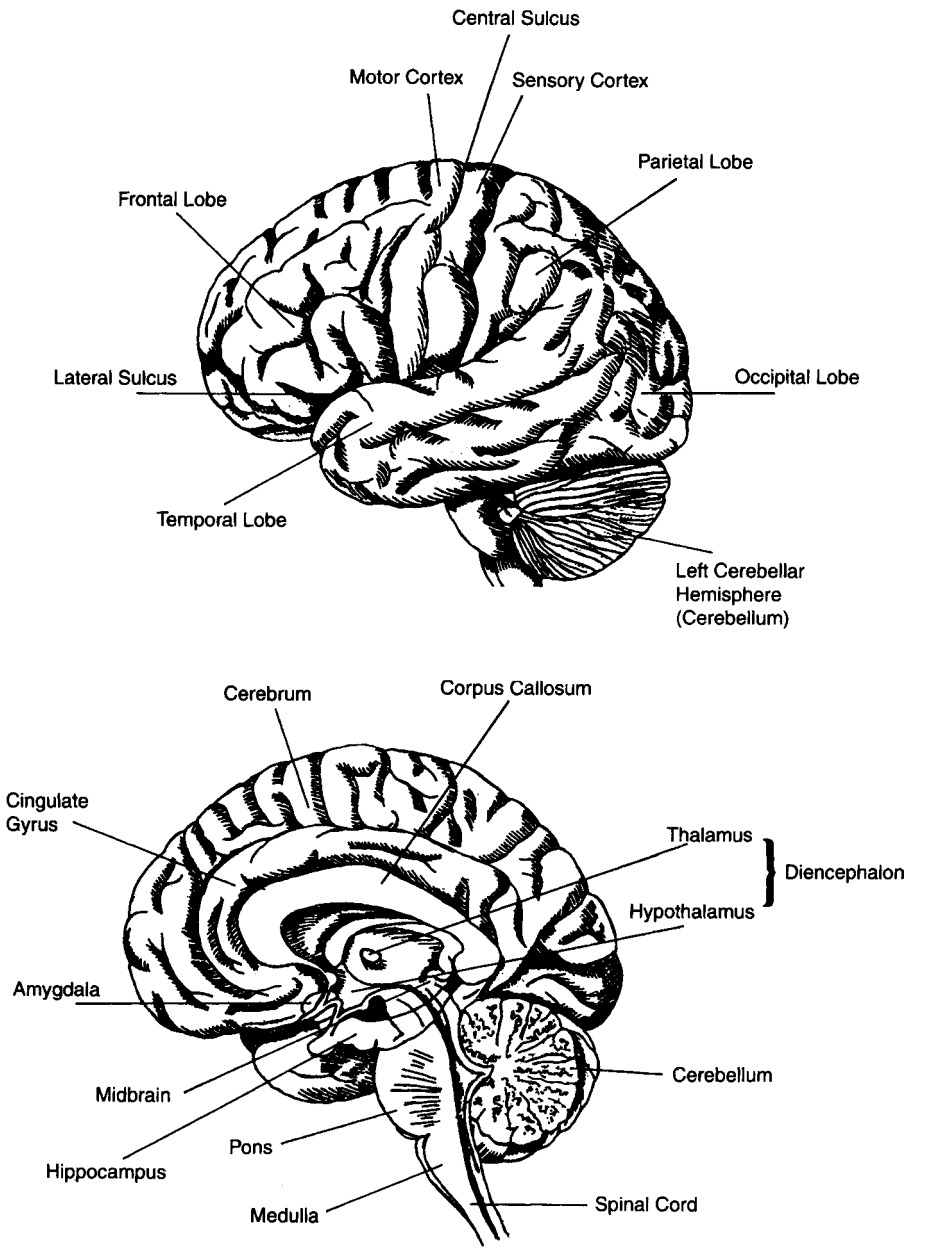

Figure 1-2 The upper figure is a lateral view of the left hemisphere; the lower figure a medial view of the right hemisphere of the human brain.

functional connection between the two hemispheres. Within each hemisphere, fiber tracts connect different parts of the hemisphere.

A system called the ascending *reticular formation* (RF) controls the overall arousal level of the cortex. The RF is a diffuse system of multisynaptic neuron chains traveling up through the brain stem. All the major sensory pathways send impulses via collateral axons to the RF, which relays them to a group of nuclei in the *thalamus*, paired grey matter structures deep in the brain on either side of the midline at the upper end of the brain stem. The thalamus serves as a relay center for motor pathways, many sensory pathways, and the RF. On reaching the thalamus, the impulses are relayed to the cerebral cortex, where they influence the level of mental alertness or sleep.

Within the brain lies the *limbic system*, which includes the hippocampus and amygdala, which lie medially to the temporal lobes; the cingulate gyrus, which lies along the medial surface of the frontal and parietal lobes; and some deep, midline structures in the brain, including the mamillary bodies. The limbic system is involved in emotion, motivation, and memory.

The brain has three coverings, called the *meninges*. The outermost thick, tough, covering is called the *dura mater* ("tough mother"), which adheres to the inner surface of the skull. The delicate, filamentous middle membrane, called the *arachnoid mater* ("spider mother"), is attached by cobweb-like strands of tissue to the fine *pia mater* ("little mother"), which adheres closely to the cortex. The *subarachnoid space* lies between the arachnoid mater and the pia mater and is filled with *cerebrospinal fluid* (*CSF*). Blood vessels also lie within the subarachnoid space and dip down in the sulci to supply deeper parts of the brain.

An inflammation of the meninges is called *meningitis*; one symptom of meningitis is a stiff neck, caused by the muscles of the neck contracting strongly (called guarding) to prevent bending of the neck and the subsequent painful stretching of the inflamed meninges.

The *ventricles* are lakes of CSF located deep within the hemispheres. The lateral ventricles, large paired structures in the center of each hemisphere, connect in the middle to form the third ventricle and, below that, the fourth ventricle. The CSF is continually formed by the choroid plexus within the ventricles and circulates through the ventricles and around the outside of the brain and spinal cord within the subarachnoid space. Excess CSF drains into the venous system from the subarachnoid space. If one of the small apertures between the ventricles becomes blocked, the CSF cannot flow out and the ventricles increase in size, causing increased intracranial pressure. This condition, known as *hydrocephalus*, can be corrected by a neurosurgeon placing a valve, or shunt, into the blocked ventricle to allow the CSF to flow through a tube into a body cavity.

The *cerebrovascular system* is too complex to describe in detail here, but in simple terms it involves two pairs of cerebral arteries: the *internal carotid arteries*, which supply the anterior parts of the brain, and the *vertebral arteries*, which supply the posterior parts of the brain. The two internal carotid arteries enter the skull and ascend on either side of the optic chiasm, where each artery branches to form the *anterior*

cerebral arteries (ACA) and *middle cerebral arteries* (MCA), one set in each hemisphere. The ACAs sweep forward to supply the medial and lower (inferior) surfaces of the frontal lobes, the medial surfaces of the parietal lobes, and the corpus callosum. The MCAs travel laterally within the lateral fissure and branch to supply much of the lateral surfaces of the frontal, temporal, and parietal lobes as well as parts of the inferior surfaces of the frontal lobes and medial surfaces of the temporal lobe. The MCA also branches to form the *striate arteries*, which supply the deeply situated *internal capsule*, the main passageway for the fiber tracts between the motor cortex and the spine (the *corticospinal tract* or *pyramidal tract*). The tiny diameter of the striate arteries makes them vulnerable to blockage, resulting in damage to the corticospinal tract and subsequent paralysis of the opposite side of the body. The MCA supplies 75% or more of the blood supply to the cerebral hemispheres.

The paired vertebral arteries enter the skull at the point where the spinal cord becomes continuous with the brain stem and join to form the single *basilar artery* on the undersurface of the brainstem; the basilar artery then divides to form paired *posterior cerebral arteries*, which supply the occipital lobes and parts of the medial and inferior surfaces of the temporal lobes, including the hippocampus. The internal carotid and vertebral arterial systems are linked at the base of the brain by a single *anterior communicating artery* and two *posterior communicating arteries*, forming a ring of vessels lying in the subarachnoid space, called the *Circle of Willis*. If one of the main arteries becomes blocked, the blood can pass around the circle to reach the deprived area. The Circle of Willis is a frequent site of weakenings on the artery wall, called *aneurysms*. If an aneurysm bursts, it expels blood around the brain in the subarachnoid space, causing a *subarachnoid hemorrhage* (see Chapter 12). A blockage in a vessel away from the Circle of Willis can result in the blood and oxygen supply being cut off to the part of the brain that vessel supplies, resulting in an area of brain death, called a *stroke* (see Chapter 5).

The venous system involves *superficial veins*, which drain the lateral and lower (inferior) surfaces of the hemispheres, and *deep veins*, which drain the internal area of the brain. The cerebral veins empty into channels within the dura mater called *venous sinuses*, which in turn empty into the large internal jugular vein.

Cerebral Cortex

Cortical zones

The cortex of each hemisphere can be divided in various ways, two of which are particularly useful for neuropsychologists. By dividing the cerebral hemispheres into primary, secondary, and tertiary cortical zones, as illustrated in Figure 1-3, the anatomical-functional relationships of the cortex can be conceptualized (Luria 1973; Mesulam 1985). The parietal, temporal, and occipital lobes lying behind the central sulcus constitute the *posterior cortex* and are involved mainly in a person's awareness of what is happening in the world. Each of these lobes can be divided into three zones.

The *primary zones* are primary projection areas in which incoming sensory in-

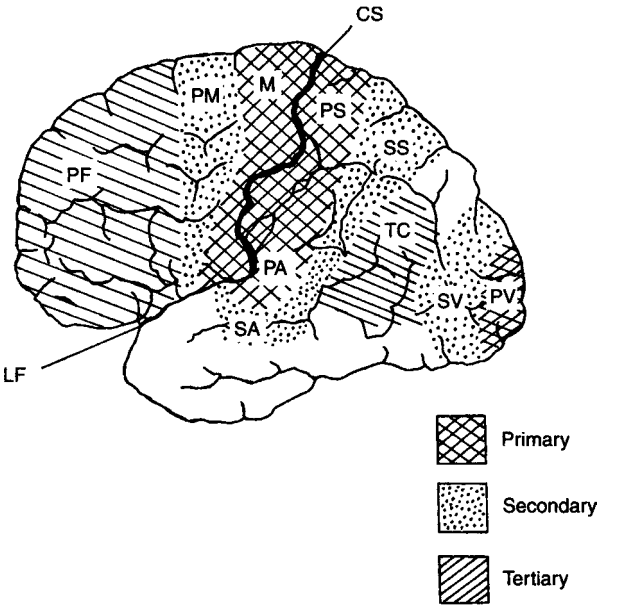

Figure 1-3 A diagram of a lateral view of the left hemisphere of the human brain divided into primary, secondary, and tertiary cortical zones. CS, central (rolandic) sulcus; LF, lateral (sylvian) fissure; M, motor strip; PM, premotor cortex; PF, prefrontal cortex; PS, primary sensory cortex; SS, secondary sensory cortex; PA, primary auditory cortex; SA, secondary auditory cortex; PV, primary visual cortex; SV, secondary visual cortex; TC, tertiary (multimodal) cortex.

formation is projected to sense-modality-specific neurons. Each side of the body is mapped topographically onto the primary sensory strip of the opposite (contralateral) hemisphere. Thus, a touch on the index finger of the right hand is projected to specific neurons in the primary sensory cortex of the left *parietal lobe* (the postcentral gyrus lying directly behind the central sulcus). The position of the finger would be projected to other specific neurons in the primary zone. The topographic pattern of neurons within the primary sensory strip of the parietal lobe can be conceptualized as a person hanging upside down with his foot hanging over the longitudinal fissure into the medial side of the hemisphere, with the trunk and hand represented on the lateral surface of the hemisphere and the face represented at the lower edge of the lateral surface at the edge of the lateral or sylvian fissure. The primary zone of the temporal lobes is concerned with sounds, and different frequencies are represented in different parts of the primary zone. Similarly, the primary zone of the occipital lobes represents specific parts of the visual field. Damage to specific areas of the primary cortex results in highly specific deficits of sensation in the topographically related body part or sense organ.

The *secondary zones* (also called the *association cortex*) lie adjacent to the primary zones. The neurons in these zones, unlike those in the primary zones, do not have a direct topographic relationship with sensory information relayed from a particular body part or sense organ. Instead, they receive the modality-specific information from their primary cortex and integrate it into meaningful wholes. Thus, the secondary cortex is concerned with perception and meaning within a single-sense modality. Damage to parts of the secondary cortex can therefore result in an inability to perceive or comprehend what one is touching or hearing or seeing, depending on whether the damage is in the parietal, temporal, or occipital secondary zones.

The *tertiary zones* lie at the inner borders of each lobe so that the parietal, temporal, and occipital tertiary zones overlap. At this level, modality specificity disappears, and integration of information across sense modalities occurs. Damage to the tertiary zones can lead to complex higher cognitive disorders that involve transmodal integration (e.g., writing to dictation). The tertiary zones also have links with the limbic system, which is involved in emotion and memory; therefore, disorders resulting from damage to the tertiary cortex may also involve abnormal emotional components.

The *frontal lobes* lie anterior to the central sulcus and are concerned mainly with acting on knowledge relayed to the posterior part of the cerebral cortex from the outside world. The frontal lobes can also be divided into three zones. The *primary zone*, or *motor strip*, is on the precentral gyrus, immediately anterior to the central sulcus, and parallels the sensory strip in that each side of the body is mapped topographically (like a person hanging upside down) onto the primary motor strip of the opposite (contralateral) hemisphere. The *secondary zone* (association cortex), also called the *premotor cortex*, mediates the organization of motor patterns, such as riding a bicycle.

The *tertiary zone*, also called the *prefrontal cortex*, is a large area situated at the anterior pole of the brain; it includes both the lateral cortex and the *basomedial* (or orbitomedial) cortex, which lies between the two hemispheres and extends to the underside of the frontal lobes above the eyes. The *tertiary cortex* is involved in executive functions, including planning, organization, and abstract thinking. Because they also have rich connections with the limbic system, the prefrontal lobes are intimately involved with mood, motivation, and emotion, and damage to them can result in many and varied impairments involving the interactions of motivational and emotional states and executive functions.

Cortical lobes The division of the cortex of each hemisphere into four lobes is the most often used concept in clinical neuropsychology. Although the lobes are often viewed as separate areas and are frequently linked to specific functions, they are in fact divisions of convenience rather than true anatomic divisions. Nevertheless, these divisions serve a useful purpose in discussions of brain–behavior relations. The four cortical lobes of the left hemisphere are labeled in Figure 1-2, and the right hemisphere is divided up in the same way. The large frontal lobes form the anterior part of the

brain, and the parietal, temporal, and occipital lobes make up the portion posterior to the central sulcus.

All three posterior lobes (in each hemisphere) are involved in the awareness, perception, and integration of information from the outside world, although their connections with the limbic system ensure that the way the world is experienced is influenced by the individual's mood, motivation, and past experiences. Generally, the *parietal lobe* is involved in functions involving tactile sensations, position sense, and spatial relations. The *left* parietal lobe has a bias toward sequential and logical spatial abilities, such as perceiving the details within a spatial pattern, whereas the *right* parietal lobe is more involved with the holistic appreciation of spatial information. The left parietal lobe also appears to mediate the ability to calculate, which involves both logical and spatial concepts. The right parietal lobe is especially good at conceptualizing complex spatial relations, and people with right parietal lesions often have extreme difficulty copying complex patterns or working out how to put jigsaw puzzles together.

The *temporal lobes* are concerned primarily with auditory and olfactory abilities, but they are involved in integrating visual perceptions with other sensory information. They also mediate some memory functions, especially those involved in new learning. Their intimate connections with the hippocampus, a part of the limbic system, allow the integration of emotion and motivation with the sensory information relayed from the outside world to the posterior lobes of the hemispheres. The *left* temporal lobe is concerned more with verbal and sequential functions; it includes the language comprehension area and is involved in new verbal learning and memory. The *right* temporal lobe tends to be more concerned with nonverbal functions, such as the interpretation of emotional voice tone and emotional facial expression and the appreciation of music and nonlanguage sounds. It also appears to play a part in nonverbal learning and memory, although this role is not as clear as the left temporal lobe's role in verbal memory.

The *occipital lobes* are the visual lobes, and they mediate sight, visual perception, and visual knowledge. A patient with a large lesion of the right occipital lobe may have a complete left-visual-field defect (loss of vision) in the visual fields of both eyes (called a homonymous hemianopia), and a patient with bilateral lesions of the primary visual cortex at the very pole of the occipital lobes will be unable to see although his eyes function normally. This condition is termed *cortical blindness*. Visual-field defects can also occur if the visual pathways are damaged at other points. A lesion in the right temporal lobe that damages the optic radiation as it travels from the optic chiasma to the occipital cortex will result in a visual-field defect in the upper left quadrant of both eyes. A lesion of the right parietal lobe that damages the optic tract will result in a visual-field defect in the lower left quadrant of both eyes. Visual-field defects are straightforward sensory defects resulting from damage to the primary projection cortex and the optic fibers traveling to them.

Lesions of the occipital secondary or association cortex can result in a number of strange disorders, particularly when the lesions are bilateral. For example, the patient

described in Chapter 8 has bilateral medial occipital lobe lesions; although he can see and describe the form of objects, he is unable to recognize what it is he is seeing. This condition is called *visual agnosia*. Again, there is some functional division between the occipital lobes of the left and right hemispheres, with the *left* occipital lobe being more concerned with visual language functions such as reading, and the *right* occipital lobe being more concerned with visually judging the orientation of lines or objects in space.

The *frontal lobes*, which are anterior to the central sulcus, are concerned with motor functions and executive functions such as forming abstract concepts and planning and executing actions based on the information received from the posterior cortex. Motor functions are mediated by the primary and premotor frontal cortex, and the left frontal lobe includes the speech area *Broca's area*. The executive functions are mediated by the prefrontal lobes and are integrated with emotional and motivational states via part of the limbic system (the *cingulate cortex*), which forms the medial parts of the frontal lobes.

The functional verbal–nonverbal division between the left and right prefrontal lobes is less marked than in the posterior lobes, but it nevertheless can be demonstrated with some neuropsychological tasks. For example, patients with left frontal lesions are frequently less able to produce words beginning with a specific letter under time pressure than those with right frontal lesions; conversely, patients with right frontal lesions are sometimes less able to create different designs than patients with left frontal damage.

Other deficits resulting from frontal-lobe damage include recent memory deficits (*frontal amnesia*), wherein the patient is unable to use memory strategies (e.g., logically structuring the material he wishes to memorize) and as a result has difficulty learning and recalling new information. The other main area of disturbance found after frontal-lobe lesions is related to the close anatomic and functional connections between the limbic structures and the frontal lobes and is often described as a *personality change* (see Chapter 9). This can be the onset of an apathetic, aspontaneous, or even mute state or an increase in aggression or the display of inappropriate behaviors.

Functional Systems

Simple motor and sensory functions, and even some more complex perceptual functions, are mediated by a particular group of neurons; therefore, damage to these neurons results in an unambiguous deficit. For example, damage to the area of motor cortex that mediates hand movements results in a paralysis of the hand on the opposite side of the body. Many of our higher cognitive functions, such as reading or memory, are, however, the result of quite complex *functional systems*, composed of a number of different brain areas working together to produce a behavior. The concept of a functional system was proposed by Luria (1973), who further proposed that in terms of double dissociation, damage to area *A* will result in the impairment of a factor or subcomponent *a*, and all functional systems that include this factor will suffer. Like-

wise, when area *B* is damaged, all functional systems that include subcomponent *b* will suffer.

As an example, damage to the right parietal lobe may impair the ability to conceptualize spatial relationships. In turn, this impairment may disrupt many functional systems and result in a wide range of behavioral deficits. The patient may no longer be able to do jigsaw puzzles, may become easily lost in an unfamiliar environment, may have difficulty learning and remembering new tasks that have a spatial component, and may no longer be able to perform calculations on paper or mentally that involve carrying figures from one column to another (a spatial task).

The concept of functional systems suggests one possible way of overcoming an impairment. If the patient can find a new way to reach the same endpoint while avoiding the necessity to include the impaired subcomponent, then recovery of function is possible. For example, he may be able to overcome his calculation difficulty by using a calculator that does not require a spatial ability but that simply requires pressing the right numbers and mathematical symbols in the correct order. In some cases of spontaneous recovery of function, the impaired functional system may restructure itself, perhaps by bypassing the damaged neurons. Nearby undamaged neurons can sprout new dendrites that "fill the gap" left by the dead or damaged neurons and connect with the dendritic trees of undamaged neurons in other cortical areas. The new cortical area could either "learn" the cognitive subcomponent that was previously mediated by the now-damaged neurons or could supply a different cognitive subcomponent that allows the functional system to remain viable, albeit using a slightly different process. This restructuring would, of course, take place outside the awareness of the patient, although he may unknowingly assist the process by continuing to practice the impaired behavior.

Disconnection Syndrome

A number of disorders are thought to result from an anatomic disconnection between two cortical areas (Geschwind 1965). One example is provided by a type of apraxia (ideomotor apraxia; see Chapter 6), wherein the patient is unable to perform skilled movements to verbal command but can perform them spontaneously. Several mechanisms have been put forward to explain this condition, but one disconnection explanation is that it is caused by damage to the fiber connection (the *arcuate fasciculus*) between the language comprehension area in the posterior left temporal lobe and the motor association cortex in the left frontal lobe.

The disconnection can be within one cerebral hemisphere as in the above example, or it can be between hemispheres, as in the case where the corpus callosum connecting the hemispheres is damaged. For example, damage to the anterior section of the corpus callosum can result in a disconnection between the verbal comprehension area in the left hemisphere and the motor strip in the right hemisphere. As a consequence, the patient may not be able to comb his hair on verbal command with his left hand

(innervated by the right motor strip) but can do so with his right hand, as the left motor strip is still connected to the left verbal comprehension area.

Experiments with split-brain subjects who have had the corpus callosum cut as a treatment for epilepsy have produced many examples of a "pure" disconnection syndrome (see Chapter 18). For example, an object flashed in the right visual field (and therefore projected to the left hemisphere) can be described or named by the split brain subject because the speech faculty is also in the left hemisphere. An object flashed in the left visual field (and therefore projected to the right hemisphere) cannot be described in speech or writing, but the isolated right hemisphere, via the left hand, can respond nonverbally to the object it sees by pointing to a matching stimulus or to the name on a list (Sperry et al. 1969). The finding that the right hemisphere can point to a name on a list demonstrates that it does have the ability to comprehend simple language, although it cannot express itself in words.

Neuropsychological Terminology

In the company of most medical and scientific disciplines, neurology and neuropsychology are well endowed with their own jargon. Although jargon should be avoided wherever possible, to understand the vast neurological and neuropsychological literature it is necessary to have a grasp of the most common of these terms. For example, *deficit, dysfunction, symptom, impairment*, and *disorder* are used synonymously and can refer to any motor, sensory, perceptual, behavioral, psychological, emotional, or cognitive abnormality. A *syndrome* refers to a group of symptoms that characteristically occur together after brain damage (see Chapter 6). In many cases, jargon terms can provide shorthand descriptions for complex disorders. Fortunately, a few simple rules can simplify their interpretation for the beginner; indeed, it can even be fun trying to work out what deficits a patient should have by breaking down the diagnostic label into its component parts.

Any label containing *phasia* refers to a speech disorder; *graphia* refers to writing, and *lexia* to reading. *Praxia* means to work or perform purposeful actions, and *gnosia* means to know. If the base word is prefixed by an *a*, strictly speaking it means that that function is completely absent (e.g., *agnosia* means not to know); prefixed by *dys*, it means partial impairment (e.g., *dyslexia* means to have a marked reading difficulty). However, these conventions are often not adhered to, and a patient labeled as having expressive aphasia may not be totally mute but more accurately may be dysphasic.

Sometimes the main label is preceded by a common English word that signifies the specific type of disorder. Therefore, *visual agnosia* means not to know what one is seeing, and *tactile agnosia* means not to know what one is touching. A patient with *dressing apraxia* has difficulty with the actions related to dressing and may try to put his left leg into the right sleeve and his right leg into the left leg of the garment, thus getting into an impossible tangle. In addition, many terms can be partially, if not fully, understood if the base of the word is known. For example, *prosopagnosia* denotes an

inability to recognize or know faces, and *anosognosia* is to deny knowledge, as in the case of a patient who denies she has paralyzed limbs. Some patients who suffer from cortical blindness deny that they are blind; this disorder is termed *visual anosognosia*. Many of these terms appear in the following case studies and are defined when they first arise.

Other terms commonly used to describe brain–behavior relations include *unilateral* and *bilateral*, which refer to damage in one hemisphere or both hemispheres, respectively, and *contralesional* and *contralateral*, which refer to impairments (or body parts) and lesions that are opposite each other. For example, a paralyzed right arm is caused by a lesion in the contralateral (left) motor strip; alternatively, a lesion in the arm section of the left motor strip causes paralysis of the contralesional (right) arm. In another example, a patient may ignore or neglect stimuli in the contralesional hemispace (the side of space opposite the brain lesion).

Assumptions that Underlie Clinical Neuropsychology

The study of brain-damaged patients to understand the workings of the normal brain and mind relies on two important assumptions. The first is that the brain of the patient was normal before the brain damage, an assumption that is often challenged when patients with long-term neurological conditions are used as experimental subjects in an attempt to understand normal brain functioning. For example, patients with long-standing epilepsy who later in life undergo neurosurgery in an attempt to cure their epilepsy often participate in experiments to discover what impairments result from removing the temporal lobe (see Chapters 3 and 4). The argument that their brains may have been organized differently from normal as the result of their epilepsy can to a large extent be dismissed as we assess more patients without epilepsy who demonstrate the same sort of impairments after traumatic temporal lobe injury.

Experiments with patients who have undergone a surgical splitting of the *cerebral commissures* (the large band of fibers connecting the two cerebral hemispheres), again in an attempt to control severe, long-term epileptic seizures, face similar criticisms (see Chapter 18). Fortunately, the results of experiments on normal people, in which stimuli are briefly flashed to one hemisphere or the other, generally support the findings of the split-brain studies, suggesting that their brains do not differ greatly from normal.

The second assumption underlying both cognitive and clinical neuropsychology experiments is that we can generalize about brain–behavior relations from one "normal" human to another. The main criticism of this assumption is the evidence that not all patients with a lesion to a specific area of the brain suffer the same impairments. For example, most but not all adult patients who sustain damage to the inferior, posterior left frontal gyrus (Broca's area) suffer impairment of language. Nevertheless, as the result of numerous studies of the impairments of brain-damaged people, it is generally accepted that it is valid to make broad generalizations about brain–behavior

relationships from one human to another. The most obvious of these generalizations is that the left cerebral hemisphere is dominant for speech in most people.

Making generalizations in the case of children is more difficult, as the brain develops functions at different rates as the child grows older. Thus, the practice of child clinical neuropsychology and experiments with brain-damaged children, although having much in common with adult neuropsychology, require different tests with age-appropriate normative data and different clinical methods and skills. A number of good books have been written about this subject area for readers with a particular interest in the neuropsychological problems of children (Anderson et al. 2001; Sattler 1992; Spreen, Risser, and Edgell 1995).

Focal Lesions and Diffuse Brain Damage

A *focal lesion*, as its name suggests, is damage restricted to a circumscribed area of the brain. *Lesion* is a general term used to describe any type of focal brain damage. *Infarct* or *infarction* refers to any area of dead brain. The most common cause of a focal lesion is a stroke, caused by a blockage or spasm of a cerebral artery and a loss of blood and oxygen to the part of the brain that artery supplies (see Chapter 5). Focal lesions can also be caused by a circumscribed area of bleeding that forms a blood clot within the brain substance (an intracerebral *hematoma*; see Chapter 5).

In an open head injury the skull is fractured, and an object may penetrate the skull and underlying brain (e.g., bone fragments, bullets, or metal from an automobile accident), damaging the brain tissue through which it passes. The neurosurgeon usually cleans the wound, removing the object and any damaged tissue and debris, and leaving a clean focal lesion that does not affect the rest of the brain. Studies of the impairments demonstrated by patients with lesions from penetrating objects or focal strokes have contributed a great deal to our understanding of the functional organization of the brain (e.g., Luria 1970; Newcombe 1969). Neurosurgical operations to remove or resect tumors or to remove parts of the brain that cause epilepsy (as in the case of temporal lobectomies) also result in focal lesions (see Chapters 3 and 4).

Some viruses attack specific areas of the brain, causing focal damage, often bilaterally. For example, the herpes simplex encephalitis virus usually targets the medial temporal lobes and sometimes attacks the inferior temporal and frontal areas as well, resulting in severe memory deficits and, in some cases, executive deficits (Utley, Ogden, Gibb, McGrath, and Anderson 1997). Brain tumors and brain abscesses are also focal lesions in the sense that they cause neural death by destroying neurons directly or via pressure effects. However, malignant tumors, while appearing to have a circumscribed boundary on a CT brain scan, may be widespread with no clear division between diseased and healthy brain. It is therefore important to be cautious when proposing associations between a tumor lesion in a specific area of the brain and the impairments that the patient demonstrates (see Chapters 6 and 7).

Diffuse brain damage refers to damage that affects many areas of the brain, as in

Alzheimer's disease and other dementias (see Chapter 17). The damage can often be visualized on a CT or MRI scan or at postmortem as *atrophy*, which is shriveled or shrunken cortex and white matter, signifying neuronal death, and usually affecting large areas of the cortex. Brain atrophy decreases the brain mass and allows the fluid-filled ventricles in the middle of the brain and the subarachnoid space around the brain to expand. Other disease processes such as meningitis and encephalitis can also result in widespread brain damage that is sometimes transient and sometimes permanent.

Progressive diseases other than AD, such as Huntington's disease (HD), Parkinson's disease (PD), and multiple sclerosis (MS), cause increasing impairment of motor and cognitive functions as time goes on. HD (see Chapter 16) is a progressive degenerative genetic disorder, and 50% of the children of a parent with HD will develop the disease (Mendelian autosomal dominant inheritance) and inevitably will become demented. The caudate nucleus, putamen, and globus pallidus become severely atrophied, but as the disease advances, brain atrophy is widespread and thus diffuse.

PD (see Chapter 15) is a disease of unknown cause that usually becomes apparent in the sixth decade and involves neurotransmitter deficits (dopamine) as a result of loss of dopamine-producing neurons in the substantia nigra of the basal ganglia. Although the majority of PD patients suffer from severe motor disorders and a number of mild to moderate cognitive impairments, only a small percentage become demented. As the dopamine deficits stemming from the atrophied basal ganglia result in widespread deficits in other parts of the brain, especially the prefrontal cortex, on functional and neuropsychological assessment, especially in advanced cases, PD patients may display a picture that is typical of diffuse damage.

MS (see Chapter 14) is an autoimmune, inflammatory disease that often first becomes apparent in adults in their 30s. As it results in the episodic occurrence of many plaques of demyelination of white matter pathways in the CNS that can later recover or remain as plaques, it causes a range of fluctuating focal motor and cognitive deficits. In many cases, as the disease progresses, the numerous focal lesions that are present at any one time can result in a functional picture that is more typical of diffuse damage. This damage is rarely sufficient to result in dementia, but it often causes memory impairments, poor sustained attention, and slowed information processing.

Diffuse damage can also occur after closed head injury, when the head hits a moving object and the brain accelerates inside the skull. This can cause stretching and tearing of neural axons, as well as numerous small areas of bleeding and infarction where the brain scrapes against the skull (see Chapters 10 and 11). A type of stroke called a *subarachnoid hemorrhage*, caused by the rupture of a cerebral artery and the expelling of blood into the subarachnoid space around the brain, can also result in diffuse cortical damage (see Chapter 12), as can severe and chronic cases of neurotoxicity caused by long-term exposure to organic solvents (see Chapter 13).

Many brain pathologies cause swelling in the vicinity of the damage; this swelling is termed *edema*. Edema, particularly common in association with malignant tumors, has the effect of increasing the area of dysfunctional brain. As the edema resolves (often when the patient is treated with steroids), the area of dysfunctional brain de-

creases, and often impairments subside dramatically (see Chapters 6 and 7). Massive brain swelling, most common after severe head injury, can compress the brain stem, resulting in the patient's death.

Cerebral Dominance, Lateralization of Function, and Specialization

The idea that in humans the left hemisphere has a strong relationship with language functions was first suggested by Broca (1861), who discovered that speech was impaired following damage to the posterior portion of the third convolution of the left frontal lobe (often called *Broca's area*). This discovery was followed closely by Wernicke's (1874) observation that a lesion in the left superior temporal gyrus (often called *Wernicke's area*) resulted in difficulties with comprehending language. Since that time, numerous studies on brain-lesioned patients have proved beyond doubt that in at least 92% of right-handers and 69% of left-handers (Milner 1975), the left hemisphere is specialized not only for the verbal functions of speech, language comprehension, reading, writing, and verbal memory but also for a number of other functions as well: functions that involve sequential, logical thinking, such as the ability to conceptualize mathematical relationships, and other functions, such as the ability to tell left from right or to carry out skilled acts on verbal command (Strub and Geschwind 1983).

For many years, the left hemisphere was viewed as the dominant hemisphere, and the right hemisphere as the minor or nondominant hemisphere, implying that the right hemisphere did not have its own, equally important areas of specialization. More recently, it has been established that the right hemisphere is "better" at some tasks than the left; in particular, tasks involving stimuli that cannot be readily verbalized. Included are nonverbal memory functions (Milner 1968b), interpretation of nonverbal emotional expression (Gainotti 1984), and visuospatial functions generally (De Renzi 1982). Some researchers also believe that the right hemisphere is dominant for some forms of attention in light of evidence that it has the ability to attend to both sides of space, whereas the left hemisphere is confined to attending to the right side of space (Heilman 1982).

Information from studies on commissurotomized (split-brain subjects) carried out by Sperry et al. (1969) and their many followers have generally confirmed the verbal/sequential specialization of the left hemisphere and the nonverbal/visuospatial specialization of the right hemisphere, although it is now clear that these distinctions are far from absolute (see Chapter 18). The left hemisphere is capable of quite complex spatial tasks, although many such tasks may be performed better by the right hemisphere; the right hemisphere has some ability to comprehend simple language and is involved in extralinguistic aspects of language, such as voice tone (Searleman 1977). In general, however, the specialization of the left hemisphere for language functions is more pronounced than the specialization of the right hemisphere for visuospatial functions, which have a greater degree of bilateral (both hemispheres) representation.

The term *lateralization of function* is simply another way to express hemispheric specialization in the sense that language is lateralized to the left hemisphere. The term

specialization is also used to describe the mediation of particular functions by specific cortical areas within a hemisphere. For example, the occipital cortex is specialized for visual perception.

The old concept of absolute left cerebral dominance is hard to put to rest. It has even been extended in the popular press to include an absolute specialization of the right hemisphere for "creative thinking," the ability to draw, and "female" traits of sensitivity and gentleness. The now-popular right hemisphere is also often publicized as the seat of the subconscious, controlled and subdued by the left hemisphere with its overpowering "male" traits of competitiveness, logical reasoning powers, and poor ability to show emotion. Although this makes a good story and sells parapsychology books and even cars ("Buy a car for your right creative and passionate hemisphere") and may be fostered by the political correctness of emphasizing "female" attributes and criticizing "male" attributes, there is no solid evidence for these extreme claims. As an example of the absurdity of such views, the idea that creativity per se is the specialized realm of the right hemisphere appears quite incongruous when one considers the great literary writings of humankind. Why these should be considered less creative than great art or architecture is decidedly unclear. There is even evidence to suggest that musical appreciation, intuitively a nonverbal function, is mediated predominantly by the right hemisphere in nonmusicians but by the left hemisphere in musicians. It has been suggested that for musicians, music is conceptualized as a sequential, logical "language," thus explaining its mediation by the left hemisphere (Sergent, Zuck, Terriah, and MacDonald 1992).

The current scientific view, backed by considerable evidence, is that whereas each hemisphere has certain specialist abilities (often also represented to a lesser degree in the other hemisphere), in the unimpaired brain the two hemispheres work as a team, and neither should be considered dominant. When the term "cerebral dominance" is used, it should be justified by the addition of the specialist functions to which it refers; for example, the (left) hemisphere is dominant for language.

Functional Plasticity

The concept of *plasticity of function* is, in one sense, the reverse of specialization. It refers to the ability of some areas of cortex to take on functions not normally attributed to them. The clearest examples of this occur in the case of hemispherectomy, where an entire hemisphere is removed or stripped of its cortex because it is so badly damaged or diseased that it no longer functions normally. These damaged hemispheres often cause uncontrollable seizures and disturbed behaviors and inhibit the normal functioning of the healthy hemisphere (see Chapter 19).

When brain damage or hemispherectomy occurs in childhood, the other intact hemisphere is often able to take over many of the functions of the damaged hemisphere. Damasio, Lima, and Damasio (1975) studied a child whose right hemisphere was damaged at the age of 5 years, resulting 7 years later in uncontrollable seizures and disturbed behavior. After a right hemispherectomy, her seizures stopped, her be-

havior improved, and assessments demonstrated that her left hemisphere could perform many of the visuospatial tasks usually mediated primarily by the right hemisphere, in addition to language functions. Similarly, when left hemispherectomies are performed after damage in childhood, the right hemisphere can take over sophisticated language functions (Ogden 1996), although it appears that some visuospatial abilities are compromised (Ogden 1988a, 1989; see also Chapter 19). Doubt continues over whether plasticity, at least for language, is possible only following childhood damage (perhaps up to the age of 12 to 15 years) or whether it can occur at any age if enough time has elapsed to allow new areas of brain to take over the functions. This issue has not been resolved because of the lack of patients who survive for long recovery periods after massive damage or disease to one hemisphere in adulthood (St. James-Roberts 1981).

Plasticity also refers to the concept that there can be a window of opportunity in development during which a particular function becomes established. Put in these terms, the hemispherectomy studies suggest that language becomes established up to the age of 12 years or so, primarily in the left hemisphere, but in the right hemisphere if the left is damaged. If the child is isolated completely from language during those first 12 years, they will never be able to establish normal language thereafter—in either hemisphere. Other functions may also develop preferentially only within a particular age range. One study that assessed visual imagery in blind people provided some evidence for plasticity for this function (although not for hemispheric specialization), as people who were blind from birth or went blind before about 12 years of age reported little or no visual imagery (including visual dreams) in adulthood, whereas people who went blind after the age of 12 years still commonly utilized visual imagery (and reported visual dreams) in adulthood after many years of total blindness (Ogden and Barker 2001).

Double Dissociation of Function

An individual patient with brain damage in a particular area may show impairment on one test and not on another. Although this discrepancy may indicate that the impaired ability is mediated by the lesioned area of cortex, and the unimpaired ability is not (a single dissociation), an alternative explanation is that the test used to assess the impaired function was simply more difficult than the test used to assess the unimpaired function. That is, if the tests were of equal difficulty, both would be impaired by damage in this area and possibly by damage in other areas as well. This ambiguity can be overcome by a method called the *double dissociation of function* (Teuber 1955). Simply put, it states that to confirm the independence of functions, symptom *a* must appear in association with lesions in area *A* but not with those in area *B*, and symptom *b* must appear with lesions in area *B* but not with those in area *A*.

This principle is particularly useful in discriminating functions that appear very similar. For example, if a patient with damage in a particular area of his brain is impaired with respect to a specific cognitive ability (e.g., he cannot memorize verbal

information) but is not impaired in other components of the same general ability (e.g., he can memorize nonverbal, visuospatial patterns), and a second patient demonstrates the reverse pattern (e.g., he can memorize verbal material but not nonverbal material), this proves that the two types of memory are dissociable and do not simply vary with respect to difficulty level. In addition, the areas of the brain damaged in each case must be associated in some way with the impaired function. Indeed, verbal memory difficulties frequently occur after damage to the medial part of the left temporal lobe (called the *hippocampus*), and there is some evidence that nonverbal memory is affected by right hippocampal damage in some patients (see Chapters 3 and 4).

It should be noted, however, that failure to find double dissociations does not necessarily mean that specific associations do not exist between the impaired function and the area of damaged brain: Performance on a particular task can be influenced by a number of factors, such as the multiple discrete functions involved in a test or the level of test difficulty (Walsh 1985, p 26). In addition, if two neural structures that each mediate separate, discrete functions are anatomically close together, the chances of finding two patients with lesions (and therefore the associated impairments) confined to only one of these neural structures becomes very small. An example is the difficulty of deciding whether a *syndrome* (a cluster of symptoms that appear together following brain damage to a particular area of the brain) is the result of one underlying cognitive deficit causing many symptoms or results from many separate deficits, each mediated by different neural structures that lie very close together (see Gerstmann's syndrome, Chapter 6).

Neuropsychological Research and Clinical Practice

One of the most stimulating aspects of clinical neuropsychology is the continual interplay between research and clinical practice. In part, this interplay is a function of the relatively recent emergence of clinical neuropsychology as a discipline separate from neurology and psychology, but it is also related to the intrinsic nature of clinical neuropsychology. For example, neuropsychology researchers who are not interested in the clinical assessment and rehabilitation of patients with neurological conditions nevertheless require excellent clinical skills and knowledge if they use neurological patients as their research subjects. A patient who is excessively anxious will not produce valid results on neuropsychological experiments, and first must be helped to relax. The researcher who is unaware of the more subtle psychological effects of depression, fatigue, and head injury on higher cognitive functions may unknowingly produce research that is seriously flawed. It is important for researchers to keep abreast of the clinical literature to ensure that they are aware of new clinical findings about neurological disorders that may have implications for their own research.

Research skills are just as important for the practicing clinical neuropsychologist as clinical skills are for the researcher. First, it is important to obtain up-to-date normative data on many of the tests clinicians use, and these data are often gathered by

clinicians working as researchers. Second, clinicians need a good grasp of appropriate research methods to design and evaluate specific tests to examine complex higher cognitive functions in different patients because the importance of individual differences increases with the complexity of the cognitive function being assessed. Third, the neuropsychological experimental literature can provide clinicians with new tests and paradigms that they can adapt for their own clinical purposes.

Roles of a Clinical Neuropsychologist

An individual clinical neuropsychologist not only can fulfill diverse roles as a clinician in an acute or outpatient neurosurgical–neurological service, in a rehabilitation service, or in private practice but also can become involved in research. This research can involve either intensive study of interesting single cases (see Chapters 3, 6, 8, and 19) or large group studies of neurological patients (e.g., Ogden 1985a; Ogden, Mee, and Henning 1993a; Ogden, Utley, and Mee 1997; Utley et al. 1997). In the setting of an acute or outpatient service, the clinical neuropsychological investigation may make an important contribution to the diagnosis of a disorder. For example, it might be important to establish whether a patient is depressed or in the early stages of dementia. Patients who are possible candidates for a temporal lobectomy as a cure for temporal lobe epilepsy must be carefully assessed by a neuropsychologist to ensure that the temporal lobe that will be left intact after neurosurgery is undamaged, and therefore capable of mediating memory (see Chapter 4). Patients in trials of new drug regimens can benefit from cognitive testing before and after the drug regimen is established (See Chapter 15). Assessments over time that monitor the changing cognitive status of patients with progressive diseases (see Chapters 14, 15 and 17) or following treatments such as neurosurgery (see Chapters 4 and 12) and radiotherapy can provide information to guide the patient's rehabilitation and, in addition, can provide data on long-term prognosis for future patients with similar conditions. The methodology required for carrying out longitudinal studies assessing interventions (e.g., drug treatments, neurosurgery, psychotherapy) for the symptoms of neurological disorders is fraught with difficulties and challenges, as many factors such as spontaneous recovery independent of the intervention, and practice effects that occur if the same neuropsychological test is given to the patient on multiple occasions, must be taken into account. In addition, even when assessing patients with the same neurological disorder, the patient group is likely to be heterogeneous on numerous variables such as age, educational level, and ethnicity (Towgood, Ogden, and Mee 2004).

The neuropsychologist is often a key staff member in rehabilitation programs, and in a head injury or stroke rehabilitation service, the neuropsychologist will usually work within a multidisciplinary or interdisciplinary team. In the multidisciplinary team, a number of different disciplines (e.g., occupational and physical therapists, speech pathologists and clinical psychologists and neuropsychologists) independently assess the needs of the patient and accordingly plan a rehabilitation program within

their discipline. Although regular team meetings are usually held to discuss the patient's progress and ensure all team members are aware of what other team members are trying to accomplish, it sometimes happens that many different goals are being worked toward simultaneously, which can cause confusion and overload for the patient.

In the interdisciplinary team, the patient is often assessed in a more holistic way, often by a key therapist on the team, who will then request more specialized assessments as necessary. The key therapist, in collaboration with other team members, then establishes and prioritizes goals for rehabilitation toward which each therapist then works, but within their own discipline. This ensures that the patient's progress toward the stated goals is reinforced by all the therapists and avoids the situation in which one therapy discipline might unknowingly interfere with the program or goals of another therapist. In either team situation, in addition to assessing the client and planning and supervising the cognitive and psychological aspects of the client's program, the neuropsychologist may also act as rehabilitation coordinator or key therapist with responsibility for overseeing the total rehabilitation program of particular clients.

In many countries the demand for neuropsychological expertise in medicolegal cases is growing. A neuropsychological report on an accident victim with a possible head injury may be required to support claims for compensation. In legal suits involving neurologically impaired clients, before a court hearing, the neuropsychologist may be asked to prepare a written submission based on the neuropsychological assessment of the client and to act as an expert witness at the hearing. For example, a head-injured client may bring a charge against a person whose actions caused the head injury or against a person he believes has taken advantage of his reduced cognitive abilities to exploit or abuse him. In the reverse situation, a client with neurological damage may be accused of a deed his lawyer argues he should not be held responsible for because of his cognitive disabilities. Whatever the reason for the court case, the neuropsychologist who acts as an expert witness must be ready to put forward a reasoned and tight argument based on her professional observations and opinion that is supported by the established body of knowledge in the relevant area.

Understanding Neuropsychology through Case Studies

The detailed examination of a selection of interesting case studies can provide not only a relatively user-friendly, jargon-free description of a range of neurological disorders, both common and rare, but can also serve to highlight the clinical and human aspects of the patients who suffer these disorders. Telling the stories of individual victims of brain damage allows their patterns of cognitive functioning and rehabilitation to be described within an ecological context. Throughout the case studies, issues that commonly arise in the practice and research of neuropsychology are introduced. Such issues include patient-centered concerns (e.g., cultural and psychosocial aspects, family involvement, patient rights), and clinician-centered concerns (e.g., personal

involvement and burnout, interaction with other health professionals, conflicts between research and clinical objectives, ethical issues).

The purpose of broadening the case studies in this way is to stimulate thinking and discussion about these issues. Another aim is to highlight the importance of conceptualizing problems and assessing and rehabilitating patients, their families, and their caregivers as an integrated "system" with all the humanistic and ethical aspects that entails, rather than as a disorder in isolation. By the end of the book, in addition to coming to know individuals and their families who have suffered and coped with brain damage, the reader should have gathered an overview of the theory and practice of neuropsychology. The reader should also have some idea about the types of rehabilitation programs and levels of recovery that are possible with different types of brain damage.

To understand the case descriptions, a sophisticated knowledge of neuroanatomy and specific neuropsychological tests is not necessary. Brief descriptions of the main tests mentioned can be found in Chapter 2. When it is helpful to do so within the context of a particular case study, the purpose of the neuropsychological tests used and their results are described in general terms.

I suggest that Chapters 1 and 2 be read before the individual case studies, but each case study chapter stands on its own and can be read in isolation from the others and in any order. Different topics, concepts, and issues are distributed through the case studies, depending on where they best fit within the context of the case. A list of some of the main topics and the chapters that include substantial discussion of them follows.

Neuropsychological Impairments

Agnosias (autotopagnosia, visual object agnosia, prosopagnosia): Chapters 6 and 8.

Amnesia and memory impairments: Most chapters refer to memory impairments as they are so common and important in a wide range of neurological disorders; however, Chapters 3, 4, 9, 10, and 17 are the most informative.

Aphasia and language disorders: Chapter 5. Chapters 18 and 19 provide discussions of language abilities in split-brain and hemispherectomized people.

Apraxia: Chapter 6.

Dementia: Chapters 16 and 17.

Diffuse brain-damage impairments: Chapters 10, 11, 12, 13, 14, 16, and 17.

Disconnection syndromes: Chapters 5, 6, and 18.

Focal brain damage impairments: Chapters 3, 4, 5, 6, 7, 8, 9, and 12.

Frontal-lobe (executive) syndrome: Chapters 9, 10, 15, 16, and 17.

Gerstmann's syndrome: Chapter 6.

Postconcussion syndrome: Chapter 11.

Hemineglect (unilateral inattention): Chapters 7 and 12.