

A Coursebook on
**Aphasia and Other
Neurogenic
Language Disorders**

FIFTH EDITION

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Contents

Preface to the Fifth Edition ix
About the Author xi

PART I: BRAIN AND LANGUAGE

- CHAPTER 1 Overview of Neuroanatomy and Neurophysiology 3**
The Human Nervous System 4
Neurons and Nerves 4
The Peripheral Nervous System (PNS) 10
The Central Nervous System (CNS) 12
The Protective Layers of the Brain 30
Cerebral Blood Supply 31
References 38
- CHAPTER 2 Neurodiagnostic Methods and Neuropathology 41**
Neurodiagnostic Methods 42
Pathologies of Neurologically Based Communication Disorders 52
References 65

PART II: APHASIA

- CHAPTER 3 Aphasia: Prevalence, Definition, and Classification 69**
Prevalence of Aphasia and Associated Diseases 71
Definitions of Aphasia 74
Limitations of the Classic Aphasia Classification 75
Alternative Aphasia Classifications 78
General Evaluation of Aphasia Classification 85
Overview of Aphasia Symptoms 87
Nonfluent Aphasias 92
Fluent Aphasias 103
References 116
- CHAPTER 4 Assessment of Aphasia 121**
Overview of Assessment 122
Screening for Aphasia 123
Diagnostic Assessment of Aphasia 124
Assessment of Functional Communication and Quality of Life 144
Assessment of Bilingual and Ethnoculturally Varied Individuals 148
Diagnostic Assessment Summary 150
Differential Diagnosis 152
References 153
- CHAPTER 5 Treatment of Aphasia 165**
Behavioral Treatment of Aphasia Is Effective 166
Generalization and Maintenance Are Still Problems 169

Most Treatment Programs Are Partial	170
Treatment of Auditory Comprehension Problems	171
Treatment of Verbal Expression	173
Social Approaches to Aphasia Rehabilitation	183
Treatment of Reading and Writing Problems	185
Group Treatment for People With Aphasia	187
Virtual Reality Therapy	189
Telerehabilitation for Aphasia	190
Augmentative and Alternative Communication	191
Medical Treatment of Persons With Aphasia	195
References	198

PART III: RIGHT HEMISPHERE DISORDER

CHAPTER 6	Right Hemisphere Disorder	207
	Hemispheric Asymmetry	208
	Right Hemisphere Functions	209
	Neuropathology of Right Hemisphere Disorder	212
	Overview of Right Hemisphere Disorder	212
	References	229
CHAPTER 7	Assessment and Treatment of Right Hemisphere Disorder	231
	Initial Screening	233
	Standardized and Nonstandardized Assessment Tools	235
	Treatment of Persons With Right Hemisphere Disorder	237
	Treatment Targets and Strategies	238
	Social Communication Skills	239
	Visual Neglect	242
	Impaired Attention	245
	Deficit Awareness	249
	Abstract Language	250
	Other Communication Disorders	252
	References	253

PART IV: TRAUMATIC BRAIN INJURY

CHAPTER 8	Traumatic Brain Injury: Causes and Consequences	259
	Incidence and Prevalence of TBI	260
	Common Causes of TBI	262
	Types of Brain Injuries	264
	Primary Effects of TBI	271
	Secondary Effects of TBI	272
	Variables Related to Recovery	274
	Neurobehavioral Effects of TBI	275
	Overview of Communication Disorders in TBI	276
	Impaired Memory, Thinking, Reasoning, and Planning Skills	280
	References	282

CHAPTER 9	Assessment and Management of Traumatic Brain Injury	287
	Overview of Assessment	288
	Initial Screening	290
	Diagnostic Assessment	291
	Assessment of Consciousness and Responsiveness	292
	Assessment of Memory	293
	Assessment of Reasoning and Planning Skills	295
	Assessment of Communication Disorders	296
	Overview of TBI Treatment Research	301
	Specific Treatment Targets and Strategies	303
	Teaching Compensatory Strategies	308
	Skill Maintenance Program	312
	Group Therapy	313
	Promoting Community Reentry	316
	Cognitive Rehabilitation	319
	References	321

PART V: THE DEMENTIAS

CHAPTER 10	The Dementias: Prevalence, Causes, and Types	329
	Decreasing Incidence and Increasing Prevalence of Dementia	330
	Prevalence of Dementia in Varied Populations	331
	What Is Dementia?	332
	Reversible and Rapidly Progressive Dementias	334
	Dementia of the Alzheimer's Type	336
	Vascular Dementia	348
	Dementia With Lewy Bodies	350
	Frontotemporal Dementia	351
	Parkinson's Disease	357
	Huntington's Disease	360
	HIV-Associated Neurocognitive Disorder	362
	References	363
CHAPTER 11	Assessment and Management of Persons With Dementia	369
	Chapter Outline	369
	Learning Objectives	369
	Assessment of Dementia	370
	Clinical Management of Dementia	377
	Intervention for Communication Deficits	381
	The Caregiver Program	390
	References	401

Glossary 407

Index 421

Preface to the Fifth Edition

The first edition of this book on aphasia and other neurogenic communication disorders was one of the first to be developed as a coursebook—a new format for teaching and learning. Instructors and students alike have liked this format because it makes both teaching and learning a bit easier and more efficient than the traditional lecture method and classroom note-taking.

The coursebook format was originally designed as an effective instructional package that reduced the amount of note-taking needed. The coursebook also reduced the variability in the accuracy and completeness of notes students take. The use of this type of book promotes class discussion as the students are not as busy taking notes in the class as they otherwise would be.

My students who have used this book for the first time in a course I taught on aphasia and related communication disorders gave me much positive feedback. Students have found the coursebook a valuable means of integrating textbook information with class notes they take. They have a single source of information that is easier to study than a text on the (literally) one hand and the class notebook on the other. They have expressed a preference for this type of book for all of their courses. I would like to thank them for their comments and suggestions. I also welcome feedback from instructors who use this coursebook.

This fifth edition is not only a coursebook but also a stand-alone textbook on courses in aphasia and other neurologically based communication disorders, whether offered at the undergraduate or graduate level. This new textbook retains the coursebook format so the instructors can make lecture notes and students can write down instructor's notes on the right half of each page of the text.

The text offers a comprehensive description and critical review of basic and applied information on aphasia, right hemisphere disorder, traumatic brain injury, and dementia—the four major language and communication disorders associated with neurological pathologies. The relationship between the brain and language, major features of aphasia and other disorders, their assessment, and treatment have been described in streamlined and clinician-friendly lan-

guage. Critical review of theories, assessment, and treatment research helps speech-language pathologists distinguish what is valid from the questionable in the professional and scientific literature. All assessment and treatment chapters give an outline of comprehensive and practical procedures, integrating current practices the clinicians might readily use.

New to the Fifth Edition

The new edition is a thorough revision of both the structure and content of the book. Several new topics and areas of research in understanding and treating the disorders are reviewed and critically evaluated:

- Part I of the book has been restructured under the heading “Brain and Language” to describe the neuroanatomical bases of language and language impairments associated with neuropathological variables. Research on brain and language continues to use newer technologies producing a dynamic field of theories and applications. The chapter on neurodiagnostics has been revised and expanded to include a variety of surgical, radiological, and imaging procedures that help understand the relation between the brain structure and function and their involvement in language production and comprehension.
- Part II has been reorganized into three chapters on aphasia. Chapter 3 offers a comprehensive review of aphasia prevalence, definition, and classification. Newer perspectives on intraoperative cortical brain mapping and alternative classification of aphasia based on recent research on the dual-stream hypotheses related to brain and language have been reviewed with critical evaluation to help the clinicians. Chapters 4 and 5 on assessment and treatment of aphasia offer a more comprehensive review of established and newer procedures. The chapter on treatment is expanded to include telerehab, drug treatment, brain stimulation, and technologically based interventions. All

major language intervention techniques are reviewed with outlined recommendations for clinicians.

- Part III on the right hemisphere disorder consists of two chapters that have been revised to reflect current terminology, research, and clinical practice issues. Sections on neglect, deficit awareness, social communication, and abstract language intervention have been updated.
- Part IV offers the most recent research on traumatic brain injury rehabilitation. Research on teaching compensatory strategies, group therapy, and community reentry has been updated with clinical recommendations.
- Part V consists of two chapters on dementia with new information on changing incidence and prevalence patterns of dementia, infectious and rapidly progressive dementias, frontotemporal dementias, primary progressive aphasias, and HIV-associated neurocognitive

disorders. An expanded clinical management and caregiver programs are included.

I am gratified that many instructors and students have found this innovative tool of teaching and learning useful and I am thankful for the many positive comments I have received from instructors across the country.

I am very pleased that this new edition is being published by Plural Publishing, the leading publishing house in communication disorders and related medical specialties. I would like to thank the editorial department headed by an able editor, Valerie Johns. Her kind and continuous help is greatly appreciated. I would also like to thank Lori Asbury, Production Manager and Jessica Bristow, Production Editor for their excellent help throughout the development and production process. Angie Singh, the president of Plural, has been a friend and supporter for decades. I thank her and all her efficient staff at Plural Publishing.

1

OVERVIEW OF NEUROANATOMY AND NEUROPHYSIOLOGY

Chapter Outline

- The Human Nervous System
- Neurons and Nerves
- The Peripheral Nervous System (PNS)
- The Central Nervous System (CNS)
- The Protective Layers of the Brain
- Cerebral Blood Supply
- References

Learning Objectives

After reading this chapter, the reader will:

- Define the terms of anatomic orientation
- Describe the central and the peripheral nervous system
- Describe and distinguish the various types of neural cells
- Distinguish cranial and spinal nerves both structurally and functionally
- Describe the surface structure of the cerebral cortex
- Describe the cerebral locations of language, motor, and auditory areas
- Describe the relevant subcortical structures of the brain
- Distinguish the different protective layers of the brain
- Describe the arteries that supply the particular regions of the brain

Communication disorders described in this book follow recent brain trauma or diseases that develop over time. These disorders include aphasia, dementia, traumatic brain injury, and right hemisphere disorder. Therefore, to appreciate the physical basis of these communication disorders, a basic understanding of the central nervous system is essential. Also essential is a familiarity with the medical neurodiagnostic methods. Speech-language pathologists (SLPs) need this knowledge to effectively assess and treat communication disorders associated with brain diseases or trauma. The knowledge will also promote more effective interprofessional collaboration with other professionals, including the primary care physicians and neurologists, who are also involved in the care of persons with communication disorders associated with brain diseases or trauma.

This chapter offers only a simplified overview of neuroanatomy and neurophysiology. It is expected that students will have had detailed coursework in anatomy, physiology, and neurology of speech and language. Students are referred to other sources for details (Andreatta, 2020; Bear et al., 2020; Bhatnagar, 2017; Blake & Hoepner, 2023; Haines & Mihailoff, 2017; Seikel et al., 2020; Vanderah & Gould, 2020).

The Human Nervous System

Anatomically, the nervous system is divided into the central nervous system (CNS) and the peripheral nervous system (PNS). The CNS includes the cerebrum, diencephalon, midbrain, cerebellum, pons, medulla, and the spinal cord.

The PNS is a network of cranial and spinal nerves that connects the brain with the peripheral organs. An overview of these structures follows. Figure 1–1 shows the main division of the nervous system.

Neurons (nerve cells) and nerves (neural fibers) are the basic building blocks of the nervous system. We begin with these two structures.

Neurons and Nerves

Neurons send and receive chemically mediated electrical signals, often referred to as *neural messages*

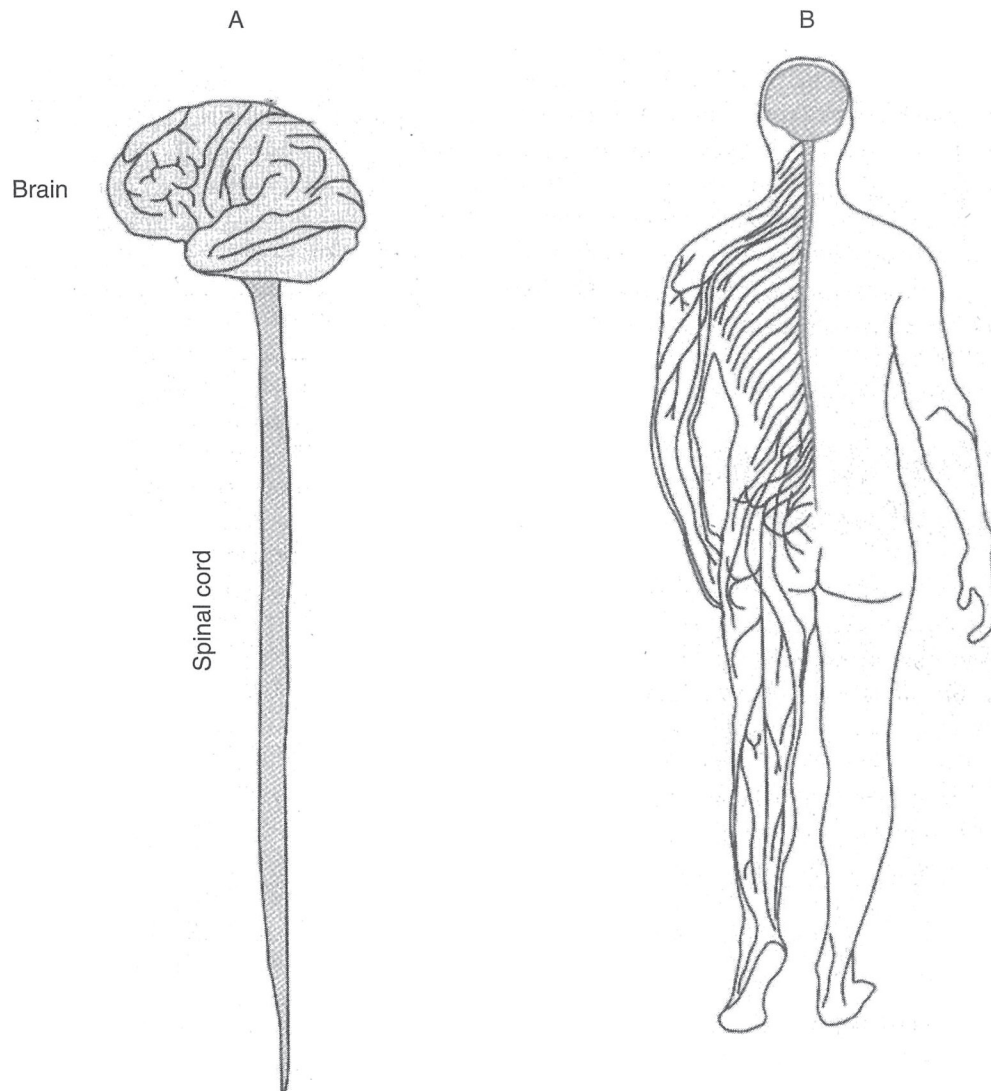


Figure 1-1. The two main divisions of the nervous system: **A.** The central nervous system. **B.** The peripheral nervous system, which includes the cranial and spinal nerves.

or *information*. Neurons receive such signals from other neurons through their dendrites and transmit the same through their axons to other neurons.

The size and shape of neurons vary a great deal as there are a variety of neurons. Figure 1–2 shows a typical neuron and some common variations. Neurons have three parts: soma (cell body), dendrites, and axon. Collectively, dendrites and axons are called neurites.

The **cell body** (*soma*; *somata*, plural) contains the *nucleus* and *cytoplasm*. The cytoplasm is enclosed with neuronal membrane, which regulates the flow

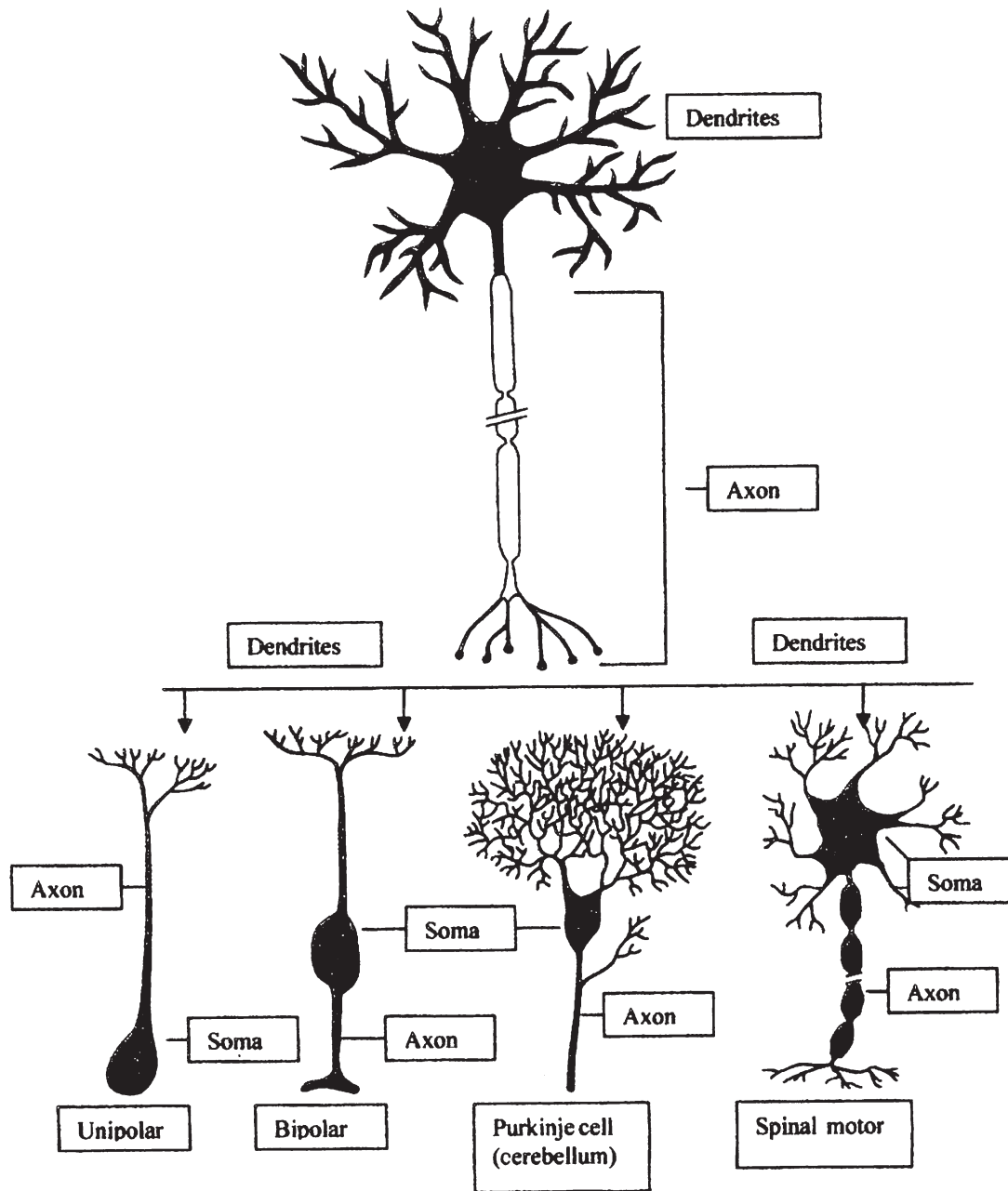


Figure 1-2. Varieties of neurons.

of chemicals (and chemically transmitted signals) to and from the cell.

Cytoplasm includes everything the soma contains except for the nucleus. The term *protoplasm* includes both the nucleus and cytoplasm. The chromosomes, which contain the genetic material called deoxyribonucleic acid (DNA), are contained within the nucleus.

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Dendrites and axons are two kinds of nerve fibers. **Dendrites** (*tree* in Greek) are short, unmyelinated fibers that extend from the cell body. Each neuron may contain several dendrites. The cell is *unipolar* when only one dendrite extends from the cell body, it is *bipolar* when two dendrites extend from it, and it is *multipolar* when several dendrites extend from it.

Dendrites receive information from axons of other cells. Hence, they are classified as *afferent* (receptive). Dendrites transmit information thus received to the cell body.

While cells may have multiple dendrites, they only have a single axon (*axle* in Greek). An **axon** is the nerve fiber that is longer than dendrites and originates from soma at a cone-shaped region called the *axon hillock*. Looking like a string of sausages, the tubular axon is thinner at its origin and becomes thicker as it becomes longer. Axons are not structurally intact; there are structural gaps in them (Vanderah & Gould, 2020).

Axons may be myelinated or unmyelinated, although the thicker, longer axons are *myelinated* (wrapped in a thin layer of white, protective, and fatty material called **myelin**). The myelin sheath helps increase the speed of conduction of neural impulses across the axon. Some demyelinating diseases degrade or destroy this protective sheath, resulting in slow or disrupted conduction of neural messages, causing motor, visual, sensory, or cognitive dysfunctions. A prominent demyelinating disease is *multiple sclerosis*.

An axon finally branches into many small filaments called **telodendria**. The terminal points of an axon are capped with small structures called **terminal buttons**. The terminal buttons are the functional contact points between neurons. Axons are functionally *efferent* in that they transmit signals away from the cell body to other neurons or muscles.

To communicate with each other, an axon of one neuron makes contact with another neuron through multiple methods. An axon may make a contact (*synapse*) with (a) a dendrite of another neuron (*axo-dendritic synapse*), (b) the soma of another neuron (*axosomatic synapse*), (c) another axon of a neuron (*axoaxonic synapse*), or (d) an *effector cell* such as a

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skeletal muscle. The point at which two neurons come in contact with each other is called a **synapse**, a neural junction. The several smaller branches of an axon at its end form synaptic terminals that come in contact with a dendrite, the soma, or an axon of another neuron or motor muscle. The contact, however, is not physically continuous as the neural junction has a small space or a gap, called the **synaptic cleft**. The synaptic cleft is a structural gap, not a chemical gap. The cleft is filled with a specialized form of protein.

Once injured or destroyed by a disease, axons of the central nervous system do not regenerate to recover their function. Dead neural cells leave a clump of debris. Consequently, in cases of severe brain injury or tissue loss due to disease, the cognitive functions may not be recovered fully.

The **glial cells** (also known as glia, meaning glue) are nonneural cells of the nervous system. Also called neuroglia, these nonneural cells provide a structural framework for the neural cells. Glia outnumber neural cells by at least 5:1 and occupy half the volume of the brain. Glial cells do not receive or send messages. Instead, they help maintain neural metabolic activity and regulate concentrations of neurotransmitters.

Normal transmission of signals within the neural network is essential for both typical communication and cognitive functions. Various neuropathologies disrupt the efficient and speedy neural transmission of messages, giving rise to several communication problems and general behavioral deficits.

Because of the synaptic cleft and other structural gaps in the human nervous system, neural transmission of information is unlike a pure physical transmission over uninterrupted lines of fibers. Neural transmission across the synaptic gaps is an electrochemical process of information exchange.

Neurotransmitters

Chemical compounds known as **neurotransmitters** contained within the axon terminal buttons establish contact between two cells by diffusing themselves across the synaptic space. This diffused neurotransmitter becomes bound to receptors in the postsynaptic membrane (neuron receiving the

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