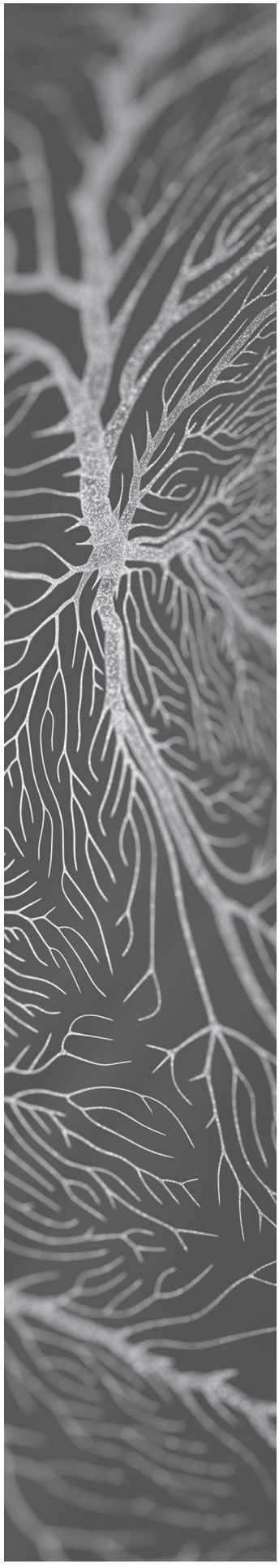

A Coursebook on

Aphasia and Other Neurogenic Language Disorders

SIXTH EDITION

M. N. Hegde, PhD

Plural Publishing Inc.

PLURAL PUBLISHING
INC.

9177 Aero Drive, Suite B
San Diego, CA 92123

Copyright © 2028 by Plural Publishing, Inc.

Typeset in 11/13 Adobe Garamond by Flanagan's Publishing Services, Inc.
Printed in the United States of America by Integrated Books International

All rights reserved, including that of translation. No part of this publication may be reproduced, stored in a retrieval system, used to train Artificial Intelligence platforms, or transmitted in any form or by any means—electronic, mechanical, recording, or otherwise, including photocopying, web distribution, or information storage and retrieval systems—without the prior written consent of the publisher.

For permission to use material from this text, contact:
Email: permissions@pluralpublishing.com
Web: www.pluralpublishing.com/authors/request-permissions

Every attempt has been made to contact the copyright holders for material originally published in another source. If any have been inadvertently overlooked, the publisher will gladly make the necessary arrangements at the first opportunity.

NOTICE TO THE READER
The views, opinions, and interpretations expressed in this publication are solely those of the author(s) and do not necessarily reflect the views, policies, or positions of the publisher. References to specific products, services, or organizations, or inclusion of third-party content, do not imply endorsement by the publisher.

Every effort has been made to ensure the accuracy and conformity of the information in this book with current medical and health service practices. However, the authors, editors, and publisher are not responsible for errors or omissions or for outcomes resulting from the application of this content. No expressed or implied warranties are made regarding the completeness or accuracy of the material. Some diagnostic protocols or medications discussed may not be FDA-approved for the conditions or dosages indicated. Use of this information remains the responsibility of the practitioner, who is expected to stay informed of evolving standards and practices.

A coursebook on aphasia and other neurogenic language disorders
Library of Congress Cataloging in Publication Control Number: 2026045322
ISBN-13: 978-1-63550-713-3
ISBN-10: 1-63550-713-8

Contents

Preface *ix*
About the Author *xi*

PART I: BRAIN AND LANGUAGE

CHAPTER 1 **Essential Neuroanatomy, Neurophysiology, and Neurodiagnostics 3**
Learning Objectives 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 31
Cerebral Blood Supply 33
Neurodiagnostic Methods 40

CHAPTER 2 **Neurological Models of Speech-Language Production 53**
Learning Objectives 53
The Classic Model 54
The DIVA Model 57
The Dual-Stream Model 58
Memory, Unification, Control Model 61
Computational Semantic Models 63
SCALED: A Social Communication Model 65
General Evaluation of Neurolinguistic Models 68
Clinical Implications of Neurolinguistic Models 69

PART II: APHASIA

CHAPTER 3 **Aphasia: Prevalence, Definition, Etiology, and Classification 73**
Learning Objectives 73
Prevalence of Aphasia and Associated Diseases in Varied Populations 75
Aphasia in Bilingual Persons 77
Definitions of Aphasia 79
Etiology of Aphasia 81
Major Classification of Aphasia 91
Overview of Aphasia Symptoms 93
Nonfluent Aphasias 99
Fluent Aphasias 111
Summary of Features Across Aphasia Types 124
Limitations of Aphasia Classification 125
General Validity of the Classic Classification of Aphasia 126

CHAPTER 4 **Assessment of Aphasia 129**
Learning Objectives 129
Objectives of Aphasia Assessment 130

Overview of Assessment Procedures 131
Strategies of Aphasia Assessment 133
Screening for Aphasia 133
Diagnostic Assessment of Aphasia 134
Assess Quality of Life 167
Assess Bilingual and Ethnoculturally Varied Individuals 169
Consider Telepractice in Aphasia Assessment 174
Consider AI-Assisted Aphasia Assessment 175
Write a Diagnostic Assessment Summary 179
Make a Differential Diagnosis 181
Appendix 4–1 183
Appendix 4–2 184
Appendix 4–3 185
Appendix 4–4 186
Appendix 4–5 187
Appendix 4–6 188

CHAPTER 5 Treatment of Aphasia 189
Learning Objectives 189
Strengths of Aphasia Treatment Research 190
Limitations of Treatment Research, Evidence, and Reviews 191
Most Treatment Programs Are Partial 194
Comprehensive Treatment Programs 195
Intensive Treatment Programs 196
Treatment for Specific Aphasic Deficits 201
Social Approaches to Aphasia Rehabilitation 216
Group Treatment for People With Aphasia 218
Augmentative and Alternative Communication 219
Virtual Reality Therapy 225
Telerehabilitation for Aphasia 226
AI-Assisted Treatment of Aphasia 227
Communication Partner Training in Aphasia 236
Executive Function Training in Aphasia 240
Medical Treatment of Persons With Aphasia 241

PART III: RIGHT HEMISPHERE DISORDER

CHAPTER 6 Right Hemisphere Disorder 247
Learning Objectives 247
Hemispheric Asymmetry 248
Right Hemisphere Functions 249
Neuropathology of Right Hemisphere Disorder 253
Overview of Right Hemisphere Disorder 254
Perceptual and Attentional Deficits 255
Affective Disorders 262
Communication Disorders 263
Other Deficits 274
Appendix 6–1 275

CHAPTER 7 Assessment and Treatment of Right Hemisphere Disorder 277
Learning Objectives 277
Case History and Interview 279
Initial Screening 280
Standardized Screening Tests 281
Standardized and Nonstandardized Assessment Tools 282
Treatment of Right Hemisphere Disorder: The Current Status 285
Treatment Targets and Strategies 286
Overview of Treatment 287
Social Communication 288
Left Neglect 297
Impaired Attention 301
Impaired Inference 305
Reduced Awareness of Deficits 308
Abstract Language 311
Other Communication Disorders 313

PART IV: TRAUMATIC BRAIN INJURY

CHAPTER 8 Traumatic Brain Injury: Causes and Consequences 319
Learning Objectives 319
Incidence and Prevalence of TBI 320
Common Causes of TBI 323
Classification of Brain Injuries 325
Primary Effects of TBI 332
Secondary Effects of TBI 333
Variables Related to Recovery 335
Neurobehavioral Effects of TBI 335
Communication Disorders in TBI 338
Impaired Cognitive Functions 344

CHAPTER 9 Communication Assessment and Management in Traumatic Brain Injury 349
Learning Objectives 349
Overview of Assessment 350
Initial Screening 352
Diagnostic Assessment 353
Assessment of Consciousness and Responsiveness 353
Assessment of Functional Communication Disorders 355
Assessment of Social Communication Skills 357
Assessment of Language Comprehension 357
Assessment of Dysarthria Associated With TBI 358
Assessment of Impaired Cognitive Functions 359
Assessment of Quality of Life 367
Overview of TBI Treatment Research 368
Developing a Comprehensive Communication Intervention Plan 370
Augmentative and Alternative Communication 380
Skill Maintenance Program 385
Group Therapy 387

Promoting Community Reentry 392
Research Evidence on Cognitive Rehabilitation 394

PART V: THE DEMENTIAS

CHAPTER 10 The Dementias: Prevalence, Causes, and Types 401
Learning Objectives 401
Decreasing Incidence and Increasing Prevalence of Dementia 402
Prevalence of Dementia in Varied Populations 404
What Is Dementia? 405
Reversible Dementia 406
Rapidly Progressive Dementia 407
Dementia of the Alzheimer's Type 408
Vascular Dementia 423
Lewy Body Dementia 426
Frontotemporal Dementia 428
Parkinson's Disease 437
Huntington's Disease 440
HIV-Associated Neurocognitive Disorder 443

CHAPTER 11 Assessment and Management of Persons With Dementia 445
Learning Objectives 445
Assessment of Dementia 446
Overview of Clinical Management of Dementia 457
Measuring the Effects of Interventions for Dementia 460
The Question of Cognitive Training 461
Medical Treatments 463
The Three Elements of Communication Intervention 465
Direct Communication Intervention for Persons With Dementia 466
Augmentative and Alternative Communication 475
The Caregiver Program 482
Telepractice 495
Posttreatment Quality of Life 497

Glossary 501
References 513
Index 539

Preface

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 textbooks 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 the 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.

Over the multiple editions, the coursebook too has evolved. Although it still keeps its original coursebook format, efforts to cover as much information as needed for a course have transformed this new sixth edition into a stand-alone textbook as well. It is now appropriate as a hybrid coursebook–textbook for courses in aphasia and other neurologically based communication disorders offered at the undergraduate or graduate level. Instructors can continue to write their lecture notes right on the pages of the book, next to the text. Students can continue to write down instructor's notes on the right half of each page of the text.

The new edition is also a response to instructors who have offered their thoughtful comments on the previous edition. Their suggestions have been incorporated throughout the book. Some instructors had suggested a need for better coverage of neurolinguistic theoretical models that try to explain the brain–language relations. In response, this new edition contains a new chapter on models of language. Major language models are described and critically evaluated for their clinical significance. To make room for this, the neurodiagnostic methods, which were in a separate chapter, have been integrated into the first chapter on neurophysiological bases of communication disorders.

Cognitive communication disorders is a popular term to refer to the disorders associated with brain dysfunction. Although the author is not convinced that an addition of the term *cognitive* to *communication disorders* offers theoretical or clinical advantages, descriptive and critical reviews of *cognitive communication* and *cognitive interventions* are included in the relevant chapters.

The text retains its comprehensive descriptions and critical reviews 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. All chapters have been updated and expanded to include new research information published since the fifth edition.

Wherever appropriate, new information on technologically oriented assessment or treatment has been included. AI-assisted assessments and treatments are included and evaluated for their current status and relevance to clinical practice. Where substantial information on teleassessment and teletreatment is available, it has been included.

Most of the chapters have been reorganized for better understanding of information. More headings and subheadings have been created for easier access to specific information. Critical review of theories, assessment, and treatment research helps speech-language pathologists distinguish 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.

The PluralPlus companion website includes new assessment protocols that clinicians may access,

download, modify, and use in their clinical practicum or practice. The website also includes color illustrations of the brain and other structures. To control the cost of the text to the students, illustrations are in black and white in the book. Most of those illustrations, however, are in color on the companion website.

I am gratified that many instructors and students have found this innovative tool of teaching and learning useful. 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, the leading publishing house in communication disorders and related medical specialties. I would like to thank Plural Publishing's editorial department headed by an able editor, Valerie Johns. Her kind and continuous help is greatly appreciated. I would also like to thank Jessica Bristow, production editor; Sean Adams, production manager; and Ari Brin, the project editor, for their excellent work on this new edition. Angie Singh, the president of Plural Publishing, has been a friend and supporter for decades. I thank her and all her efficient staff at Plural Publishing.

New to the Sixth Edition

A Coursebook on Aphasia and Other Neurogenic Language Disorders, Sixth Edition is a textbook for a course or courses on aphasia, right hemisphere disorder, traumatic brain injury, and dementia. It is organized in a unique and interactive coursebook format I have developed in teaching my courses. The format divides the pages into two columns. The first column to the left of the page contains the printed text and the second column to the right has lined spaces for the student to take lecture notes.

The coursebook offers the same level of integrated and critical information on all the neurogenic communication disorders. Both the theoretical and practical information on the disorders, their assessment, and treatment are described in a user-friendly style that is technical as well as easy to understand.

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:

- The first chapter on neuroanatomy and physiology includes an overview of neurodiagnostic methods that help determine various sites of the brain may be damaged, resulting in communication disorders.
- A new chapter (Chapter 2) has been written on the neurolinguistic models or theories that are proposed to explain how the brain processes speech and language functions.
- AI-assisted assessment and treatment information and their critical evaluations have been added to relevant chapters.
- Teleassessment and teletreatment, to the extent that research supports, have been discussed in relevant chapters.
- Communication partner training, social communication and participation, and community reentry of persons with traumatic brain injury have all been strengthened with new research.
- Quality of life assessment is included in relevant chapters.

About the Author

M. N. (Giri) Hegde, PhD, professor emeritus of Communication Sciences and Disorders at California State University–Fresno, holds a master's degree in experimental psychology from the University of Mysore, India; a post-master's diploma in medical (clinical) psychology from Bangalore University, India; and a doctoral degree in speech-language pathology from Southern Illinois University Carbondale.

Dr. Hegde is a specialist in fluency disorders, language disorders, research methods, and treatment procedures in communicative disorders. He has made numerous presentations to national and international audiences on various basic and applied topics in communicative disorders and experimental and applied behavior analysis. With his deep and wide scholarship, Dr. Hegde has authored several highly regarded and widely used scientific and professional books, including *Clinical Research in Communicative Disorders* (with P. Salvatore), *Introduction to Communicative Disorders*, *A Coursebook on Aphasia and Other Neurogenic Language Disorders*, *Hegde's Pocket-Guide to Communication Disorders*, *Hegde's Pocket-Guide to Treatment in Speech-Language Pathology*, *Hegde's PocketGuide to Assessment in Speech-Language Pathology*, *Assessment of Communication Disorders in Children* (with F. Pomaville), *Assessment of Communication Disorders in Adults* (with D. Freed), *Clinical Methods and Practicum in Speech-Language Pathology*, and several others. He has served on the editorial boards of scientific and professional journals and continues to serve as an editorial consultant to the *Journal of Fluency Disorders*.

Dr. Hegde is a recipient of various honors, including the Outstanding Professor Award from California State University–Fresno, CSU-Fresno Provost's Recognition for Outstanding Scholarship and Publication, the Distinguished Alumnus Award from the Southern Illinois University Department of Communication Sciences and Disorders, and Outstanding Professional Achievement Award from District 5 of the California Speech-Language-Hearing Association. Dr. Hegde is a fellow of the American Speech-Language-Hearing Association.

1 Essential Neuroanatomy, Neurophysiology, and Neurodiagnostics

Learning Objectives

After reading this chapter, the reader will:

- Define the terms of anatomic orientation
- Describe the central and the peripheral nervous system
- Give an overview of neural cells and their types
- Distinguish the structures and functions of cranial and spinal nerves
- Describe the surface structure of the cerebral cortex
- Describe the cerebral locations of language, motor, and auditory functions
- Describe the relevant subcortical structures of the brain
- Distinguish the different protective layers of the brain
- Describe the structure of cerebral blood supply
- Summarize the essential neurodiagnostic methods

Communication disorders described in this book include aphasia, right hemisphere disorder, traumatic brain injury, and dementia. These conditions are all associated internal or external factors that affect brain function. Therefore, an understanding of the neurological bases of these disorders is essential to (a) assess and treat neurologically based communication disorders and (b) effectively interact with the medical professionals involved in the care of persons with brain diseases or trauma.

This chapter offers only a simplified overview of neuroanatomy, neurophysiology, and neurodiagnostics. 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, 2023; Bhatnagar et al., 2024; Blake & Hoepner, 2027; Seikel et al., 2027; Tellis & Hunter, 2025).

The Human Nervous System

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 CNS (brain) and the peripheral system of nerves that run throughout the body.

Neurons and Nerves

Neurons (nerve cells) and nerves (neural fibers) are the basic building blocks of the nervous system. **Neurons** send and receive chemically mediated electrical signals (*neural messages*). Neurons receive 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.

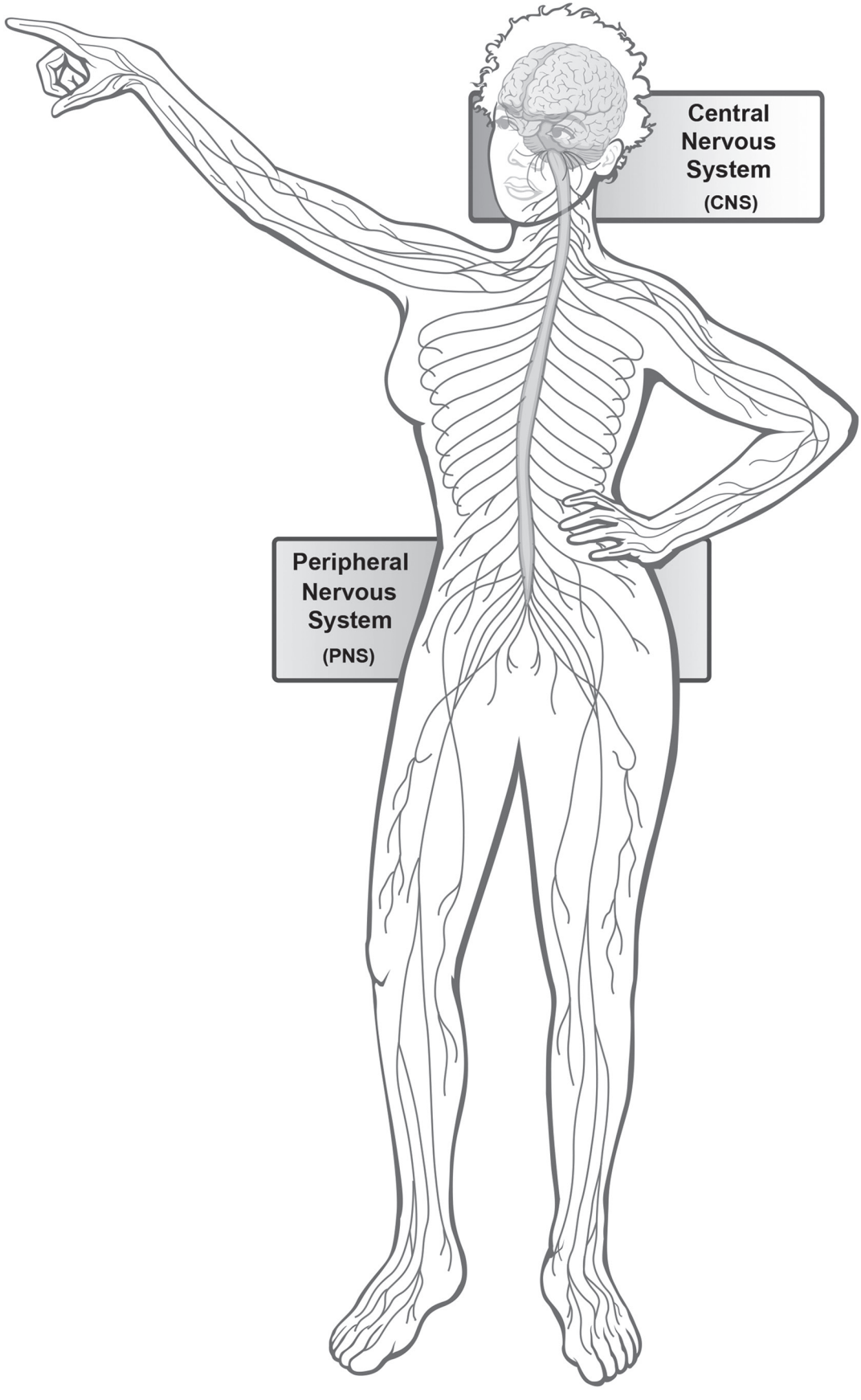

Figure 1–1. The central and the peripheral nervous system. *Source:* From *Neuroscience Fundamentals for Communication Sciences and Disorders, Second Edition* by Andreatta, R. D. Copyright © 2023 Plural Publishing, Inc.

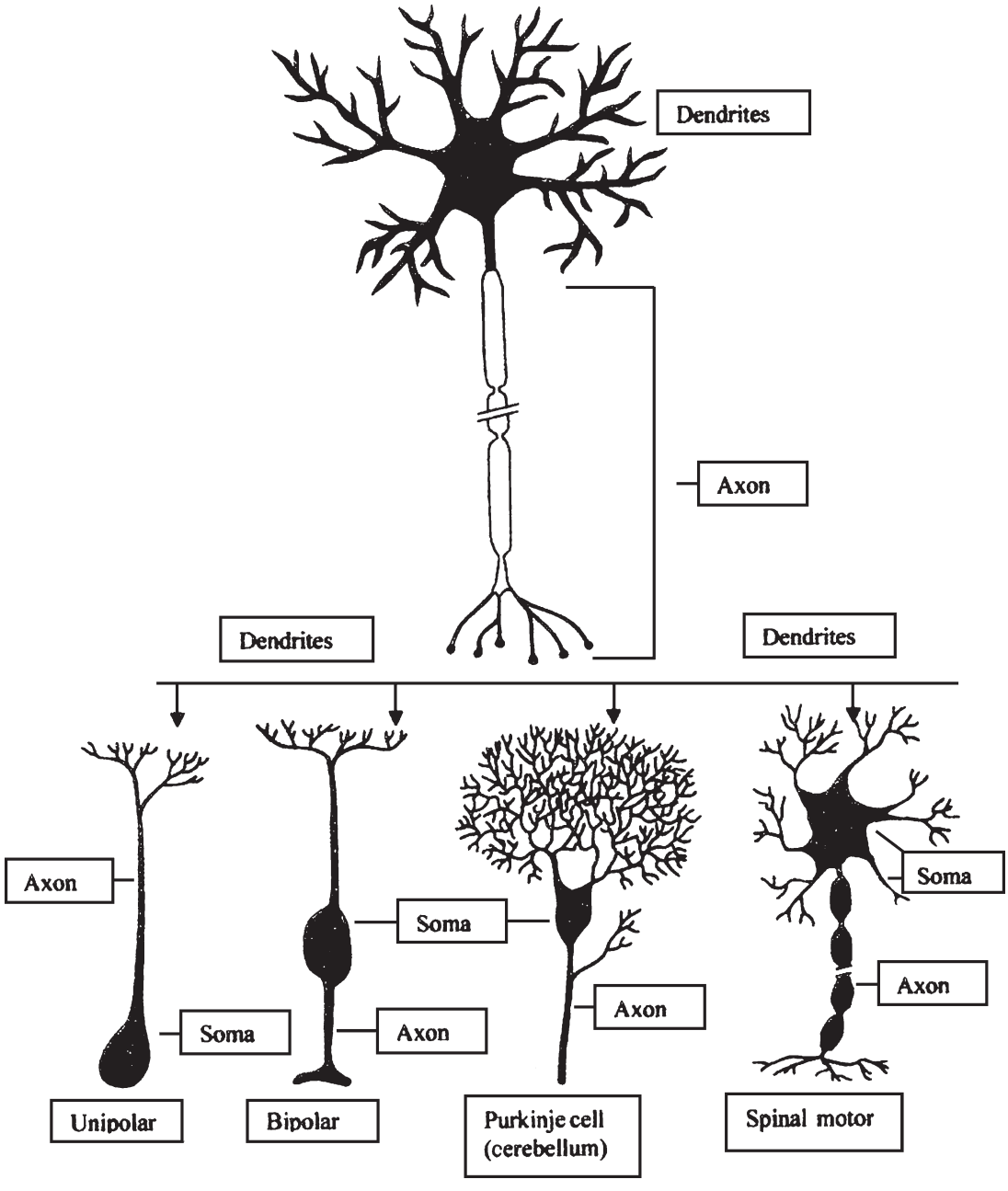

Figure 1–2. Varieties of neurons.

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 **nucleus** contains the chromosomes, within which are the genetic material called deoxyribonucleic acid (DNA). The **nucleus** houses DNA. The **cytoplasm** includes everything the soma contains except for the nucleus. Enclosed

with neuronal membrane, the cytoplasm regulates the flow of chemicals and chemically transmitted signals to and from the cell.

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.

Although 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.

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 (e.g., *multiple sclerosis*) degrade or destroy this protective sheath, resulting in slow or disrupted conduction of neural messages, causing motor, visual, sensory, or cognitive dysfunctions.

An axon finally branches into many small filaments called **telodendria**. The terminal points of an axon are capped with small structures called **terminal buttons**, which 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 (*synaps*e) with (a) a dendrite of another neuron (*axodendritic 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

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.

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.

The **glial** or **neuroglia** (glia means *glue*) are nonneural cells of the nervous system that provide a structural framework and support 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. Injured or destroyed axons of the CNS 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 neural disease, the communication and cognitive functions may not be recovered fully.

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 impulse). The diffused neurotransmitter may either excite or inhibit the next neuron in a chain.

Among the several varieties of neurotransmitter chemical compounds, amino acids and amines are

critically important. **Amino acids** include gamma-aminobutyric acid (GABA), glutamate, and glycine. **Amines** include acetylcholine (Ach), dopamine, epinephrine, histamine, norepinephrine, and serotonin. Each neuron specializes in releasing a certain kind of neurotransmitter. For instance, most CNS synapses release amino acids (glutamate, GABA, and glycine) to accomplish fast transmission of messages. At neuromuscular junctions (and throughout the PNS), Ach mediates fast message transmission; its action in the CNS is somewhat slower. Ach primarily mediates voluntary movement. All varieties of neurotransmitters are involved in slower forms of message transmission.

Neurological diseases and trauma affect neurotransmission, causing several neurological and psychiatric disorders. Dopamine deficiency due to the death of dopamine-producing cells in the brainstem are related to the tremor and poor control of movements in Parkinson's disease. Alzheimer's disease may be associated with deficiencies in Ach in the hippocampus and frontal cortex. On the other hand, excessive production of dopamine in the forebrain may be involved in schizophrenia.

GABA, an amino acid and a derivative of glutamate, is the main neurotransmitter in the brain. Although glutamate is excitatory, GABA is inhibitory. Death or degradation of neurons that produce GABA in basal ganglia (especially in the caudate and putamen) causes the involuntary movements of Huntington's chorea.

Afferent and Efferent Nerves

Bundles of axons, dendrites, or both, specializing in certain functions, are called **nerves**. Two major kinds of nerves of interest to us are motor nerves and sensory nerves.

Motor nerves, or **motor neurons**, cause muscle contractions (movement) or glandular secretions. Because the motor nerves transmit impulses away from the CNS, they also are called **efferent nerves**. Efferent nerves terminate at an effector, which leads to glandular secretion or muscle movement. Motor or efferent neurons have their origins in the brain and terminate in a gland or muscle. In essence, the