Acquired Language Disorders

A CASE-BASED APPROACH

Second Edition

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Contents

Accompanying PowerPoint slides and CD include lecture material for:

Review of Neuroanatomy
The Target Model
Assessment in Acquired Language Disorders (ALD)
Nonfluent Aphasia
Fluent Aphasia
Subcortical Aphasia
Primary Progressive Aphasia (PPA)

Acquired Dyslexia and Dysgraphia
Right Hemisphere Disorder (RHD)
Traumatic Brain Injury (TB1) and Post Concussive Syndrome (PCS)
Dementia
Encephalopathy

Preface

Introduction

New and experienced clinicians may find it challenging when attempting to integrate theoretical knowledge and research into clinical practice. As professors teaching the graduate courses in acquired language disorders and clinical practicum, we realize that it isn't until the clinician actually encounters a person with a specific disorder that academic knowledge and practice coincide. Given our years of experience as both teachers and practitioners, it is our intention to bridge the gap between theory and practice by providing the reader with a case-based approach to understanding acquired language disorders (ALD). To further our goal in making ALD come to life for the reader, we have developed a model that depicts the individual's language and cognition following a cerebrovascular accident or other neurologic event. We refer to this as the Acquired Language Disorders Target Model, and each of the 14 cases that we discuss has a corresponding diagram within the chapter.

In our experience as professors at the graduate level, the student benefits from the graphic features of the ALD Target Model because it facilitates a concrete understanding of the linguistic and cognitive characteristics of each case. This model combined with features of the various disorders, case analyses, and treatment considerations connects theoretical knowledge with practical application. In our opinion, this case-based approach matches the needs of speech-language pathologists practicing in health care today.

How the Book Is Organized

Each chapter includes a fictional person based upon an actual case that was treated in a health care setting, private practice, or home health environment to exemplify a specific acquired language disorder. These case scenarios were developed based on actual patients who the authors or their colleagues have evaluated and treated. This brings to life each communication impairment for the learner, who can better conceptualize the specific characteristics of the disorder in the context of a real person. For purposes of anonymity and confidentiality, the patients' names and identifying information have been changed.

As practicing speech-language pathologists, we believe it is essential to understand not only the basic pathophysiology of a disease process associated with an acquired language disorder, but also the functional effects it may have on a person's life. The fundamentals presented here allow the reader to participate in discussions with other professionals and family members. The student or practitioner can then use this information to build a foundation for assessment and therapeutic approaches, which are found in Appendix D.

Special Features

The 14 cases in this book provide a comprehensive overview of the assessment process, major aphasic syndromes, right hemisphere disorder, traumatic brain injury, dementia, encephalopathy, and other etiologies affecting the ability to communicate. The final chapter provides detailed information about past, present, and future considerations in treating individuals with ALD. We discuss therapeutic approaches currently in use and include new trends in treatment.

Each chapter is based on a case study and includes eight sections:

- **Characteristics** of the disorder including neurologic correlates.
- **Case Scenario** providing a brief overview of the case history.

- **Diagnostic Profile** including language expression, speech production, auditory comprehension, reading, written expression, cognition, and behavioral symptoms of the case.
- The **ALD Target Model** presents a visual representation that captures the type and degree of language impairment as well as any areas of cognition that may be affected.
- **Functional Analysis** consists of a narrative that succinctly summarizes the case and helps the clinician understand the impact of the disability on daily life.
- **Critical Thinking/Learning Activity** poses questions designed to help the student or clinician develop problem-solving and practical skills necessary to maximize the patient's progress.
- **Treatment Considerations** provide areas to consider for rehabilitation based on the patient's strengths and weaknesses, individualized to his/her psychosocial context. General therapeutic objectives are also provided.
- **Therapeutic Options** include possible treatment considerations pertinent to the case, most of which are further described in Appendix D.

The Acquired Language Disorders Target Model

We developed the Acquired Language Disorders Target Model™ (ALD Target Model™) from an *embedded language framework*. This model is shown in Figure 1 and reflects the influence that cognition plays in normal communication and, by extension, in the rehabilitation of people with acquired language disorders. The physical appearance of the model depicts a schematic relationship between language and cognition as well as the relationship among functional language modalities.

There are five primary domains of the ALD Target Model: Language, Attention, Memory, Executive Functions, and Visual Spatial skills. The Language domain includes four areas: expression, comprehension, reading, and writing. Expression (E) and comprehension (C) involve the verbal modality; whereas reading (R) and writing (W) involve the visual modality. Although as speech-language pathologists we are clinically oriented to the Language domain, we must not neglect the other four cognitive areas of functioning because they are integral to functional communication.

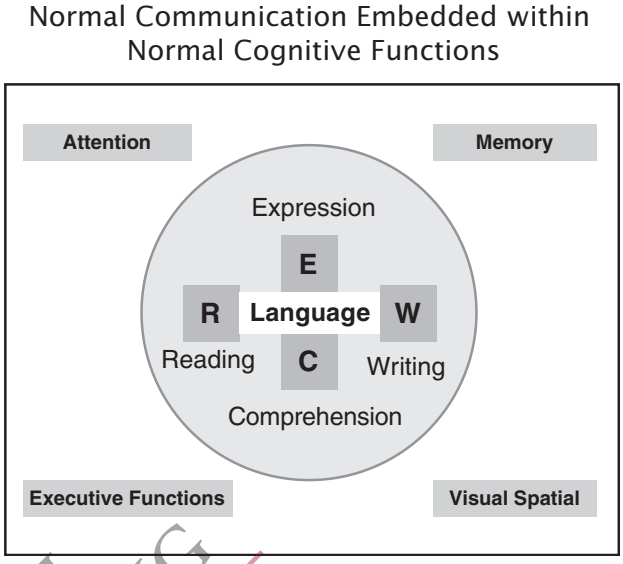

Figure 1. The Acquired Language Disorders Target Model.

For an individual who has normal communicative functions, the lettered squares (E, C, R, and W) remain attached to the rectangle containing the word *Language*. For an individual with an acquired language disorder, the lettered squares move further away from the Language rectangle to reflect greater impairment. For each type of acquired language disorder, the pattern is different. For example, in a person with an expressive nonfluent aphasia (Broca's), the E square and the W square are placed outside the circular border to indicate a severe degree of impairment. Depending on the acquired language disorder, any or all of these language modalities may be impaired at varying degrees. This ranges from normal, to mild-moderate, to moderate-severe, to severe-profound. Impairment level is depicted in Figure 2.

The ALD Target Model reflects our agreement with Helm-Estabrooks and Albert (2004): assessing the cognitive domains of attention, memory, visuo-

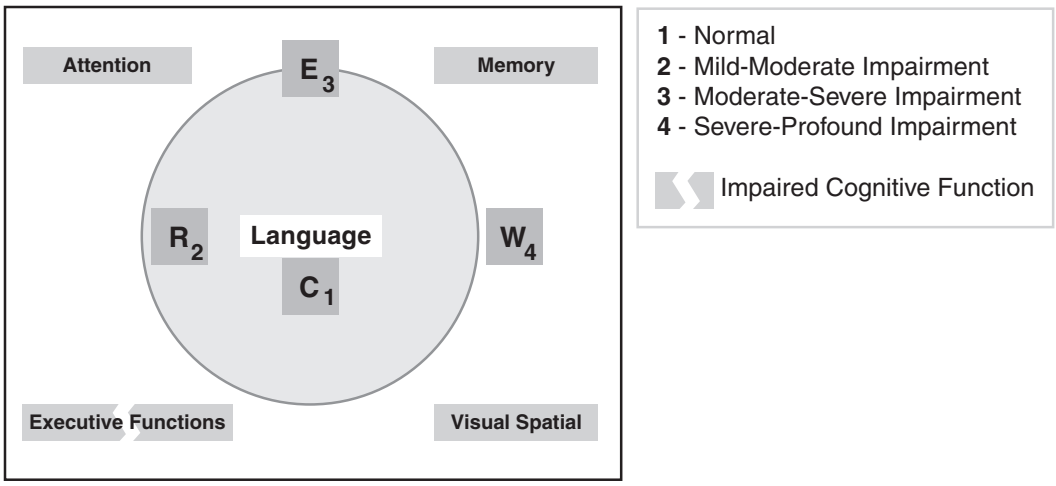

Figure 2. The key to understanding the Acquired Language Disorders Target Model.

spatial skills, and executive functions in aphasia should be a component of the standard evaluation for treatment planning. The reader will notice a fractured line through any of the four cognitive domains that are impaired in each specific case presented. For example, in an individual with a severely impaired memory, the box labeled *Memory* has a fractured line going through the word.

How to Use This Book

For the Student and the Practitioner

- A graphic image of the ALD Target Model representing each disorder enhances the student's or practitioner's understanding of cognitive-linguistic changes pertinent to that specific case.
- The value of the case-based approach to ALD is that it facilitates comparisons among types of patients. This optimizes more accurate decision-making for planning treatment. As a learning tool, this approach helps the student or practioner attach clinical information to a case that is represented with a physical image
- The ALD Target Model combined with the Functional Analysis can be very useful for clinical practice in a health care setting. This permits the student or practitioner to integrate the neurologic, cognitive, linguistic, and functional aspects of each patient to formulate a holistic picture for treatment.
- PowerPoint slides supporting lectures are provided for the topical chapters in the book.
- Critical thinking questions are provided for each case to facilitate clinical decision-making skills, and Appendix G provides a case-based exam to "test your knowledge."
- Many current treatment approaches (23) are provided to assist the practitioner in planning a program for each patient.
- Each case has a one-page diagnostic profile that describes each patient's language expression, speech, auditory comprehension, reading, written expression, cognition, and behavioral symptoms.

For the Instructor

- An overview of basic neuroanatomy for acquired language disorders is provided.
- This book offers a detailed summary of many formal and informal assessments

and treatment programs for those with ALD.

- There are 14 case-based acquired language disorders, each with assessment and treatment considerations, to facilitate class discussion and clinical problem solving.
- There is a section on past, present, and future considerations with a historical overview of efficacy and evidence in treating those with ALD.
- PowerPoint slides correspond to the text and offer important lecture material, diagrams, illustrations, and online links for teaching.
- Charts, tables, and figures including the ALD Target Model help categorize and concretize the various acquired language disorders.
- Functional treatment can be easily planned using the Functional Communication Connections Worksheet with samples provided.
- A Test Your Knowledge examination is also provided using seven case-based examples.

Reference

Helm-Estabrooks, N., & Albert, M. L. (2004). *Manual of aphasia and aphasia therapy* (2nd ed.). Austin, TX: Pro-Ed.

Chapter 1

AN OVERVIEW OF NEUROANATOMY AND NEUROPHYSIOLOGY RELATED TO ACQUIRED LANGUAGE DISORDERS (ALD)

The Neuron

The brain has more than 100 billion neurons, or nerve cells. These structures comprise the building blocks of the nervous system and are its functional work horses. Each neuron is composed of a body, referred to as the soma; filamental extensions called dendrites; and longer fibers called axons. Each neuron has one axonal fiber that can measure from micrometers to meters in length. The axon functions as a conductor of electrical impulses. Dendrites receive stimuli or input from other neurons, and axons send stimuli to other neurons, glands, or muscles (Webb & Adler, 2008). These neurons communicate with each other electrochemically via neurotransmitters (a discussion of neurotransmitters appears in this chapter; Figure 1–1).

The nervous system has sensory neurons (receptors) and motor neurons (effectors). Sensory neurons are sensitive to light, sound, touch, temperature, smell, and chemical input, and transmit sensory information from the environment via the nervous system. Motor neurons receive excitation from other cells and send impulses to the muscles instructing them to contract and to the endocrine glands to regulate hormonal secretions. Input from sensory neurons can be transmitted to motor neurons, for example, a sensory neuron may detect a dangerous stimulus and respond by alerting interneurons in the spinal cord to notify the motor neurons to remove that body part in danger. At the endpoint or terminal of the nerve cell, neurotransmitters are released into the synaptic space between the cells. Neurotransmitters are biochemical compounds that help neurons communicate, acting as messengers between them (Figures 1–2 and 1–3).

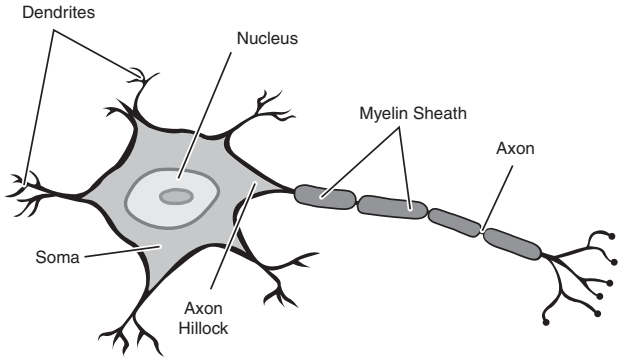

Figure 1–1. Neuron.

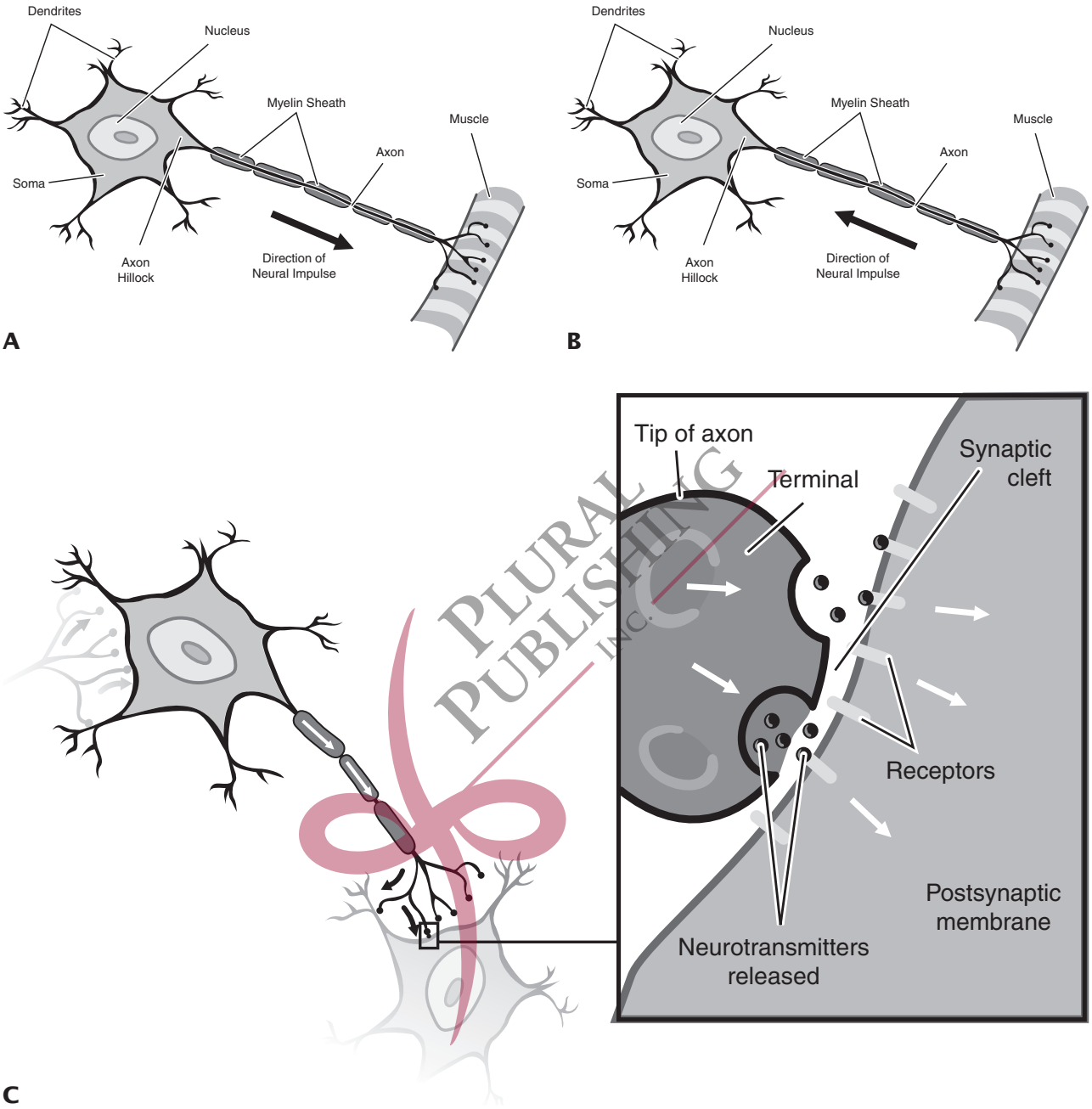

Figure 1–2. A. Motor neuron. **B.** Sensory neuron. **C.** Events at synapse.

Neurotransmitters

Neurotransmitters are chemicals that assist in the regulation of the brain's ability to control metabolic activity, speech and language, motivation, personality, mood states, and cognition including attention and memory (Bhatnagar, 2002). Each neuron releases neurotransmitters at the synapse, which is where the bulb of the axon makes contact with the dendrites. The neurotransmitter passes across the synaptic cleft and bonds with the receptor site on the postsynaptic

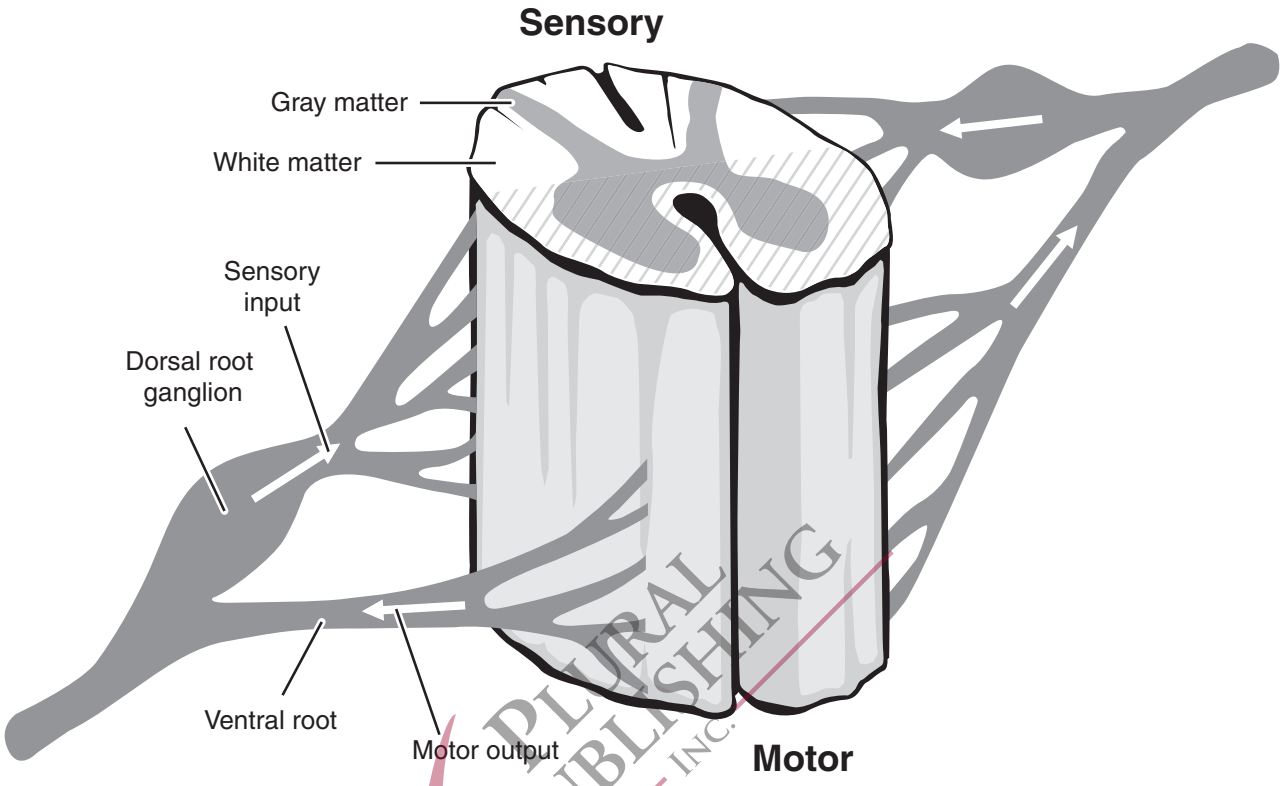

Figure 1–3. Spinal cord cross-section.

membrane. This results in a change in the electrical current across the cell membrane and the nerve fibers. The change in the electrical valence of the cell is referred to as the action potential. An excess or depletion of neurotransmitters can have significant effects on functioning. For example, excess dopamine interacting with other factors has been linked to schizophrenia, and a depletion of dopamine concentration contributes to Parkinson's disease.

There are two main types of neurotransmitters: the small molecules and the large molecules, also known as neuropeptides. The small molecule transmitters include acetylcholine, serotonin, dopamine, norepinephrine, glutamate, histamine, and gamma aminobutyric acid (GABA). In this group, GABA is primarily inhibitory whereas glutamate is excitatory. Yet in many cases, neurotransmitters can be either excitatory or inhibitory depending on the receptor site. Dopamine can act in this way. The large molecule neuropeptides include vasopressin, somatostatin, neurotensin, enkephalin, and endorphins. These neuroactive substances are hormone-mediated and affect the body's metabolic functioning. A pituitary peptide such as endorphin is opioid-like and functions in pain management. Neuroactive peptides may be specific to particular organs and have multiple roles in the body. Both groups of neurotransmitters are crucial to a person's feelings of pleasure, pain, stress, cravings, the promotion of sleep and rest, and emotional attachment, as well as basic metabolic functioning (Schwartz, 1991; Webb & Adler, 2008; Table 1–1).

The Brain: A Brief Review of Structure and Function

The central nervous system consists of the brain and spinal cord. Each segment of the spinal cord has both sensory and motor nerves that innervate our skin, organs, and muscles. During brain development in

Table 1–1. Selected Neurotransmitters

Neurotransmitter	Distribution	Proposed Impact
Acetylcholine	It is the primary neurotransmitter of the peripheral nervous system (PNS) and important to the central nervous system (CNS) as well. It is concentrated in the basal forebrain, striatum, and reticular formation. It is also concentrated within regions of the brainstem involved with cognition and memory.	Involved in voluntary movement of skeletal muscles and viscera including spinal and cranial nerves. Drugs that affect cholinergic activity within the body impact heart rate, bladder function, digestion, and may cause dry mouth. This neurotransmitter is also important to sleep-wake cycles. Decreased cholinergic projections on muscle cells are found in myasthenia gravis. Decreased projections in the hippocampus and orbitofrontal cortex are related to Alzheimer's disease.
Dopamine	Concentrated in neuronal groups in the basal ganglia. Dopaminergic projections originate in the substantia nigra and have terminals in the cortex, amygdala, and nucleus accumbens.	Decreased dopamine in the brain is linked to Parkinson's disease. An increase of dopamine in the forebrain is linked to schizophrenia. Dopamine is involved in cognition and motivation and is related to wanting pleasure associated with love and addiction.
Norepinephrine	Norepinephrine neurons are found in the pons and medulla. Most are in the reticular formation and locus ceruleus.	Important to maintaining attention and focus. It increases excitation in the brain and is involved in wakefulness and arousal. It is also associated with the sympathetic nervous system and feelings of panic, fight, or flight.
Serotonin	Synthesized from the amino acid tryptophan and found in blood platelets and the gastrointestinal tract. Terminals are localized in nerve pathways from the nuclei at the center of the reticular formation.	Controls mood, regulates sleep, involved in perception of pain, body temperature, blood pressure, and hormonal functioning. Low levels are associated with depression. It is also involved in memory and emotion.
GABA	A major neurotransmitter with cells found in the cerebral cortex, cerebellum, and hippocampus. GABA projections are inhibitory from the striatum to the globus pallidus and substantia nigra to the thalamus.	Loss of GABA in the striatum is linked to a degenerative disease that causes involuntary abnormal movements (Huntington's chorea). It is associated with the inhibition of motor neurons.

childhood, neurons create new connections with other neurons. At birth, the brain weighs about 350 grams (12 ounces) and is about 1,000 grams (2.2 pounds) at 1 year old. As an adult, the brain weighs approximately 1,200 to 1,400 grams (2.6 to 3.1 pounds) and does not have the ability to create new connections with other neurons, as most neurons cannot be replaced. This section discusses the brain's covering, the ventricles, and the following major structures of the central nervous system: the cerebral cortex, brainstem, subcortical structures, cerebellum, and the neural pathways.

The Coverings of the Brain, Ventricles, and Cerebrospinal Fluid

There are three layers of tissues, the meninges, that protect the brain. They include the dura mater, arachnoid membrane, and pia mater. Between the

arachnoid membrane and pia mater is the subarachnoid space. This space contains blood vessels and cerebrospinal fluid (CSF).

The CSF protects the brain. It is a clear and colorless fluid that circulates throughout the brain and the spinal cord cushioning and protecting them from injury. There are four ventricles within the brain: two lateral ventricles, the third ventricle, and the fourth ventricle. Each ventricle contains the choroid plexus, which is the structure that produces the CSF. The CSF flows from one ventricle to the next and finally into the subarachnoid space. It is reabsorbed back into the blood. The lateral ventricles are connected to the third ventricle, and the third ventricle is connected to the fourth. Blockage in any of the spaces can cause CSF to back up, leading to a number of serious medical conditions including hydrocephalus, which increases pressure on the brain (http://www.sickkids.ca/childphysiology/cpwp/brain/csf.htm; Figures 1–4 and 1–5).

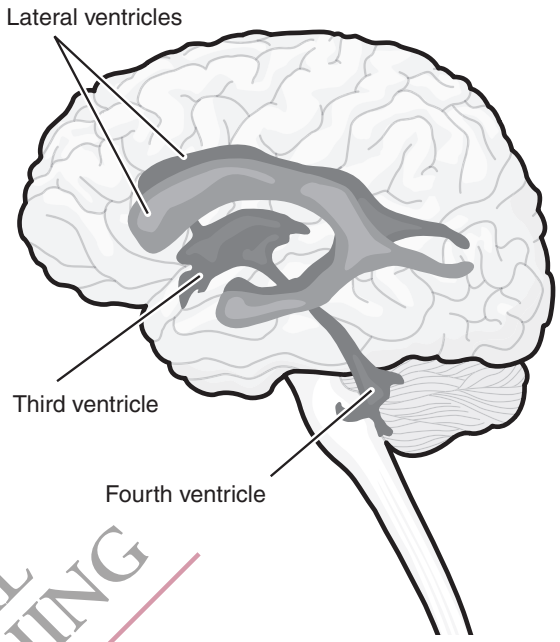

Figure 1–4. Ventricles.

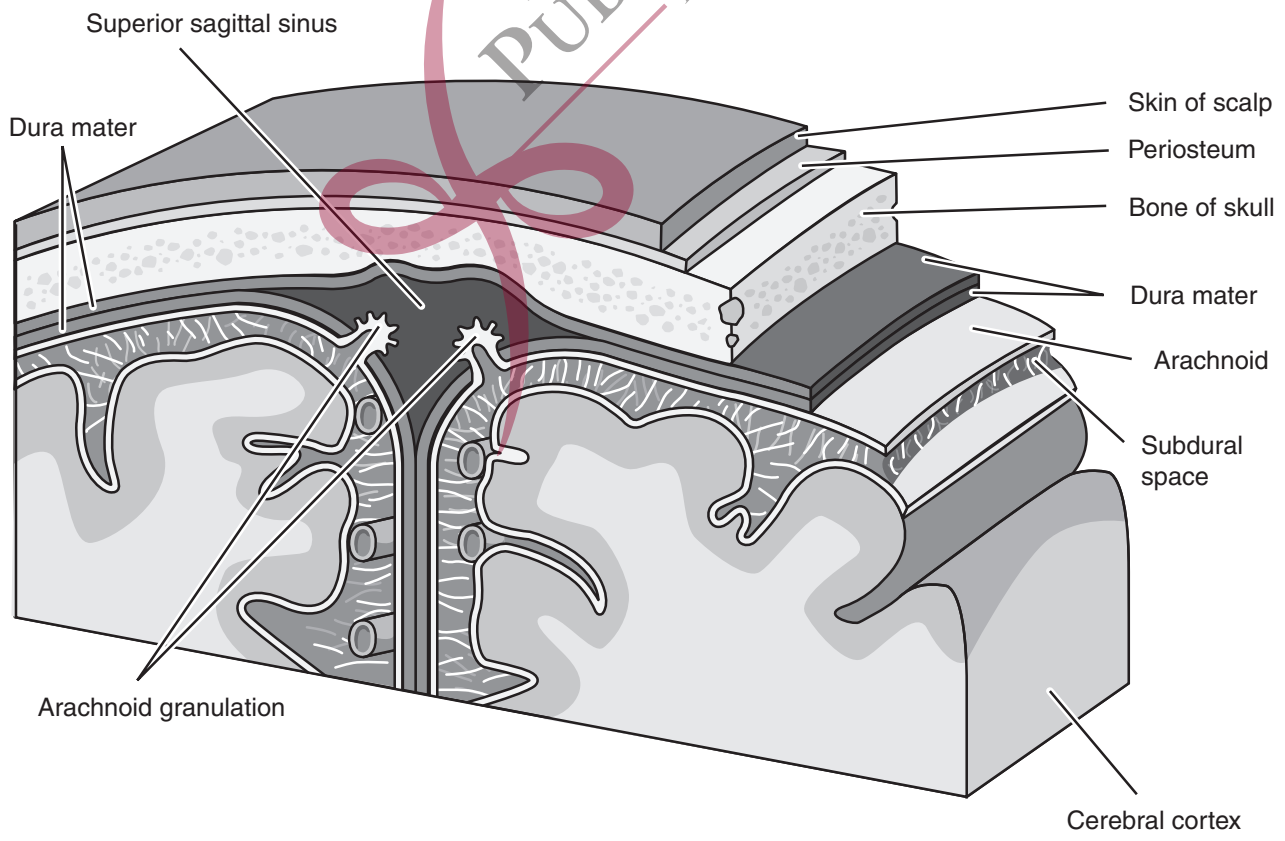

Figure 1–5. Meninges.

Appendix G

TEST YOUR KNOWLEDGE

- This examination focuses on *critical thinking,* given detailed clinical information.
- There are 50 questions on this examination, based on seven case scenarios. Read the case scenarios very carefully. This is a skill that you must master in this discipline.
- Choose the one best response and circle your response on the answer sheet below and darken the appropriate corresponding responses.

Case #1

This patient is a 60-year-old WM found down by his son in the living room of a house that they share. The patient was not breathing and had no pulse. The patient's son started CPR after calling 911, and he continued with CPR until EMS arrived, 10 minutes later. EMS found the patient to be unconscious, not breathing, and pulseless. They implemented the cardiac arrest protocol. A respiration and a pulse were restored. The son told EMS that the patient must have been down "for at least 10 minutes" before he called 911. The patient was transferred to the EMS vehicle and taken to the local hospital.

The patient was stabilized in the emergency department and transferred to the cardiac care unit. Two weeks post-event, the critical care attending consulted speech-language pathology to assess the patient's cognitive-linguistic status in preparation for eventual transfer to the next level of care. The chart review indicated that the patient had variable levels of alertness; mumbles "occasionally"; is totally dependent on nursing for his activities of daily living. Nursing reports that he "just lays there most of the day." His family is present in the afternoon hours, and they are asking for "rehab."

1. Based on the history above, it is most likely that this patient is presenting with:
   a. Lewy body dementia
   b. PPA
   c. Anoxic encephalopathy
   d. RHD

2. As the family is asking for "rehab," is it reasonable to assume that the DRS would be the appropriate rating scale for this patient as part of your assessment?
   a. Yes, because the DRS is a scale related to functioning post-hospitalization and therefore describes the patient from a rehab potential perspective.
   b. Yes, because the DRS assesses brain injury.
   c. Yes, because the DRS is the best and most efficacious assessment instrument for all anoxic patients.
   d. No, I would still use the Rancho rating system for this patient.

3. This family is asking for inpatient rehab services at a free-standing rehabilitation hospital 2 weeks post event. This may pose a counseling and education problem for you. How would you handle this?
   a. I would refer to social work and step aside.
   b. I would notify the resident and tell her that this family is irate and unrealistic and demand a social work consult from her.