

**The Right Hemisphere  
and Disorders of  
Cognition and Communication**

*Theory and Clinical Practice*



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*Theory and Clinical Practice*

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# Preface

This book covers decades of work by researchers in a variety of fields who all have been interested in what happens in the right side of the brain. It is designed for advanced graduate students and practicing clinicians interested in neurogenic disorders of cognition and communication. The perspective is from the field of speech-language pathology, but the knowledge should be useful for a broad range of professionals interested in cognition and communication.

The first chapter provides an introduction to right hemisphere brain damage (RHD) and some of the reasons why patients and clients with RHD often do not receive the same recognition or treatment as survivors of left hemisphere strokes. The second chapter provides a review of some fundamentals of clinical practice, including the World Health Organization's structure for viewing health and disability, cultural awareness, evidence-based practice, and practice-based research. While it may seem odd to have two introductory chapters, they serve very different purposes: one to introduce the population, and the second to set the stage for working with that population. It is important to approach assessment and treatment with consideration of clients' personal and environmental contexts, their cultural background, and plans to assess treatment effectiveness all firmly in the front of your mind. Thus, the review of these areas appears before the chapters on the disorders.

The remaining chapters all begin with an overview of the construct and how that construct is processed in the intact right hemisphere. This is followed by how the construct is affected by RHD and what we currently know about assessment and treatment. Given the current state of the art and science in the area of RHD, the assessment and treatment sections are relatively scarce in terms of concrete evidence-based practice. With this current reality, it is crucial to have a solid understanding of cognitive and communication processes and the theories of how they are affected by RHD to guide clinical decision making. The treatment sections build upon what we do know, and contain many suggestions based on evidence from the traumatic brain injury (TBI) literature and theoretically based expert opinion. For areas in which the research and the theories are solid enough to support treatment approaches, I provide specific suggestions for approaching treatment (e.g., language comprehension). For other areas (e.g., anosognosia), explicit approaches that go beyond the existing expert opinion or evidence from TBI are not provided because the theoretical support is not strong enough for me to feel comfortable doing so.

There are many possible ways in which the chapters could be organized, because the areas of cognition and communication overlap and interact. Indeed, communication *is* a cognitive process. They are divided here because in the field of

speech-language pathology we tend to think of language and communication as separate from other cognitive processes. In this book, aspects of communication are presented first, followed by the other cognitive areas. The pragmatics chapter provides a model of social communication that sets the stage for all of the processes

discussed in the book, thus it is the first “content area” following the introductory chapters. This is followed by language comprehension and prosody. The remaining chapters cover cognition: attention and neglect, executive function and anosognosia, and finally memory.



# Foreword

About 23 years before I had the pleasure of reading the prepublication chapters of Margaret (Peggy) Blake's wonderful, informative new book, I had asked her to do the same thing for me. At that time I was a young(er) professor and was thoroughly delighted to have convinced Peggy to come work with me as a PhD student. Fast-forward several years, past her assiduous work on several of my grants, a number of our joint publications, and multiple research projects of her own, and Peggy had become the only PhD graduate I know whose dissertation committee required not even one change in her thesis document. The clarity of thought and style connoted by this fact continue to be evident in the current volume.

Peggy quickly developed into, and has remained, an influential sister-in-arms in the pursuit and evaluation of knowledge about the nature, assessment, and management of cognitive/language disorders in adults with damage to the right side of the brain. There weren't many investigators interested in the topic 23 years ago and there still aren't—a fact that makes me even prouder of Peggy's continued, substantive leadership through her research and publications, educational offerings, and professional service roles. It has been a joy to collaborate with her and to learn from her over the years, having watched her grow into the expert who, among other considerable contributions to the field, wrote the clear, engaging, and authoritative volume you have in your hands.

The entire book is terrific, but I particularly loved Chapter 1, in which Peggy astutely comments on how and why patients with right hemisphere disorders (RHD) often get "lost in the system." She elaborates on the discrepancy in detecting and intervening with the problems of right versus left brain stroke patients, beginning with the earliest medical contacts and proceeding through various clinical assessment and management processes. The rest of the chapter provides additional important introductory material about the population of adults with RHD. Peggy really connects with readers through fun thought experiments about vital right hemisphere contributions to communication. This chapter also emphasizes essential issues such as patient/symptom heterogeneity, thinking beyond the standard clinical stereotypes, and common research problems.

Chapter 2 helps to lay a strong foundation for clinical work with the people who have RHD. It is an extremely useful guide to viewing the existing evidence with an appropriately critical eye, and to helping readers understand how they can be involved in expanding this evidence. The chapter focuses in part on the nature of evidence and different sources of evidence, along with challenges to clinical assessment and evidence-based practice. It also offers some solutions to these challenges. For example, Peggy calls the lack of data and investigation "a golden opportunity" to apply a practice-based evidence

model with the RHD population, by gathering evidence in typical clinical situations to influence management practices.

The remaining chapters each tackle cognitive/language areas that are often affected by RHD: pragmatics and social communication (including discourse production), discourse comprehension, prosody, attention, neglect, executive functions, awareness, and memory. In each chapter, Peggy begins by introducing relevant theories and models and reviewing evidence on normal right hemisphere functioning. Each chapter ends with coverage of RHD symptoms, assessment, and treatment considerations. The coverage is typically comprehensive and always clear and understandable. Periodic sidebars help to clarify difficult concepts, making the material even more engaging. Tables and figures provide useful summaries or illustrations, including, for example, the extremely helpful table that depicts manipulations that affect performance in the chapter on Neglect, and the excellent examples of the “contextualization process” for novel idioms in the chapter on Language Comprehension.

These chapters admirably bring together vast, complex, and often contradic-

tory bodies of literature. In addition, they offer the best clinical solutions currently available, including borrowing from the evidence about other populations with similar disorders and theoretically based possibilities. Equally important, Peggy’s approach provides clinicians with reminders and tools that will help them find the best solutions next week, next year, and many years from now.

This is a really opportune moment for Peggy’s book. The literature on normal right hemisphere function has boomed—much of it after investigators who were interested in left brain functioning saw the right hemisphere activation in their investigations of the brain bases of normal language and cognitive processes. Theory and evidence about right hemisphere disorders and their clinical management have continued to grow since the last book of this sort. And the literatures on evidence-based practice and practice-based evidence have blossomed. Bringing these literatures together in a comprehensible and enlightening way does a real service for readers of all kinds. I enjoyed reading every word and can’t wait to see what Peggy does next.

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# Acknowledgments

This book would not have been possible without the many very smart, dedicated, amazing people who influenced my educational journey through the “other side” of the brain. My first introduction to right hemisphere brain damage (RHD) came in my graduate program at Arizona State University when Dr. Leonard LaPointe suggested that I focus on RHD for my Master’s thesis. I am forever indebted to him for that suggestion, as it was the launching point for my career researching, teaching, writing, and wondering about the right hemisphere. My path was solidified when I spent several years working with Connie Tompkins at the University of Pittsburgh. She began as my mentor, and became my colleague and friend. The year I spent at the Mayo Clinic working with Joe Duffy and Edy Strand strengthened not only my clinical skills, but also my appreciation for other parts of the brain, like the basal ganglia and the left hemi-

sphere. It also allowed me the opportunity to collaborate with Penny Myers and add a more direct clinical component to my views on RHD. Thanks also to the many colleagues who have been supportive of my work throughout the years.

While working on this book I received valuable assistance with collecting, sorting, and reviewing information from several students at the University of Houston: Natalie Ewing, Dionne Dias, Aaron Rodriguez, Kelly Tobey, and especially Jessica Connors, who spent many hours poring over psychometric properties of cognitive and communication assessments. Thanks also to Jerry Hoepner and Rik Lemoncello who lent their expertise to the chapters on executive function and practice-based evidence. Finally, thanks to Kalie Koscielak at Plural, who was so helpful at every stage of this process, and to the reviewers who took the time to read the book draft and make smart, thoughtful suggestions for changes.

## **RH Function**

Lateralization of function and lateralized asymmetries are not unique to humans; they have been reported rather extensively in both vertebrates and invertebrates (Corballis, 2014). Most primates and marine mammals show LH dominance for action dynamics, and all primates studied thus far show RH dominance for emotion. Some species (e.g., frogs, mice) also show LH dominance for vocalization.

The RH has long been thought to have greater interconnectivity than the LH. Early work supported this idea based on the greater amount of white matter in the RH compared with the LH (Gur et al., 1980). More recent studies employing a variety of imaging techniques (magnetoencephalography, near infrared spectrometry) have provided additional evidence of differential white matter organization. The RH appears to have greater functional interconnectivity than the LH (Gootjes, Bouma, Van Strien, Scheltens, & Stam, 2006; Iturria-Medina et al., 2011; Li et al., 2014; Medvedev, 2014). The organizational patterns suggest that the RH is better at general information processing such as integration processes, in contrast to the LH, which is more efficient at specialized processing such as language and motor action (Iturria-Medina et al., 2011; Li et al., 2014).

It has been suggested that cognitive changes associated with aging reflect differential changes in the hemispheres. An early theory suggested that aging affected the RH more than the LH, resulting in RHD-like symptoms in older adults (Goldstein & Shelly, 1981). Neuroimaging

evidence to support this theory is inconsistent (Rajah & D'Esposito, 2005); some studies do report asymmetrical changes related to aging (e.g., Dolcos, Rice, & Cabeza, 2002; Goldstein & Shelly, 1981; Miller, Myers, Prinzi, & Mittenberg, 2009), but others indicate that age-related changes in the size of structures are roughly equivalent across the hemispheres (Raz et al., 2005; Salat et al., 2004). While differential changes have been observed in regions within the prefrontal cortex, with greater changes in anterior and dorsal regions of the RH compared with the LH, these prefrontal changes occur in tandem with bilateral (and symmetrical) changes in the ventral regions (Rajah & D'Esposito, 2005). The RH may influence aging in other ways. Robertson (2014) suggests that the RH-biased networks for arousal, sustained attention, awareness, and response to novelty may underlie the construct of cognitive reserve, in which individuals with higher education, higher IQ, and more complex job responsibilities appear to have some "protection" against cognitive deficits related to brain injury and neurodegenerative disease.

## **History of Understanding RH Functions**

Historically, discovery of localization of neurological function began with studying patients. This is true for RH functions. Much of our understanding of what the RH does comes from early studies of patients with focal lesions and an exploration of their deficits. While there were occasional descriptions of deficits attrib-

uted to RHD beginning in the late 1800s<sup>1</sup> (see review by Heilman, Bowers, Valenstein, & Watson, 1986), it was not until the mid-1900s that specific functions of the right hemisphere were explored in earnest (see reviews in Blake, 2016; Heilman et al., 1986; and Searleman, 1977).

Case studies and experiments involving visuoperceptual deficits, visuospatial agnosia, and unilateral neglect began appearing in the 1940s (McFie, Piercy, & Zangwill, 1950). While the early reports suggested that these disorders could not be unequivocally linked to RHD, it was not long before the RH was considered “dominant” for visuoperception. Language and communication were addressed in the 1960s, with the suggestion that RHD could affect abstract and complex language processing (Critchley, 1991; Eisenson, 1962).

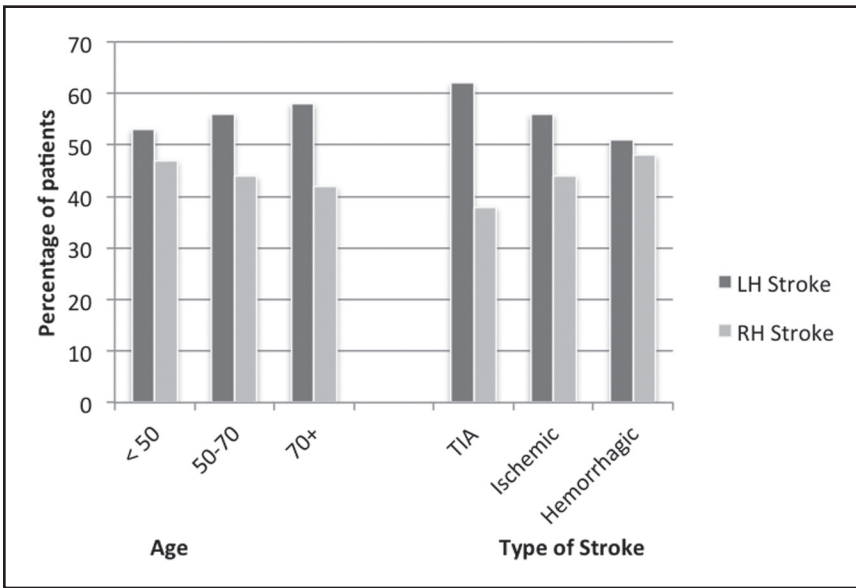
In the 1970s there was a dramatic increase in the number of studies of emotion, prosody, visuoperception, and unilateral neglect (Blake, 2016; Ross, 1984) that led to the current understanding that the RH is dominant for these functions. During that same time frame, descriptive studies of language and communication supported ideas proposed by Critchley (1991) and Eisenson (1962) that RHD resulted in changes to “extra-linguistic” or complex language, including interpreting connotative meanings, story morals or gist, comprehending sarcasm and humor, and other forms of nonliteral language (Blake, 2016; Perecman, 1983; Wapner, Hamby, & Gardner, 1981). Development of theories to explain the language and communication deficits occurred in the

1990s, with Myers’ (1990) inference failure hypothesis, Tompkins and colleagues’ suppression deficit hypothesis (Tompkins, Lehman, & Baumgaertner, 1999; Tompkins, Baumgaertner, Lehman, & Fassbinder, 2000; Tompkins, Lehman-Blake, Baumgaertner, & Fassbinder, 2001), and Beeman’s (1998) coarse coding hypothesis. These are discussed in more detail in Chapter 4, along with the first treatments for language deficits associated with RHD that were published in the 2000s. The clinical history of RHD thus is relatively new, beginning nearly a century after the dedicated interest in the LH.

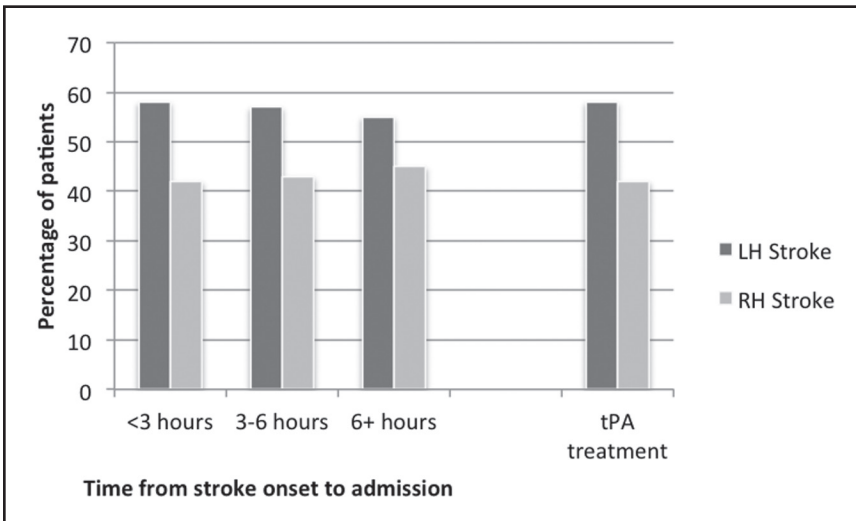
### **Identification and Treatment of Right Hemisphere Stroke**

RH and LH strokes occur at approximately the same frequency (RH 45%; LH 55%) (Foersch et al., 2005; Hedna et al., 2013). However, there are stark differences in the recognition and treatment of RH and LH strokes (Figures 1–1 and 1–2). To fully grasp the issues, it is important to understand stroke treatment. Currently, the most effective medical treatment for ischemic stroke is tissue plasminogen activator (tPA), a “clot-busting” drug. When administered within 4 hours after the onset of a stroke, it can dissolve the clot, restore blood flow, and substantially reduce the amount of tissue damage and resulting disability, thus significantly improving a patient’s prognosis. Beyond

<sup>1</sup>Hughlings Jackson described visuoperceptual deficits in 1876; Babinski described anosognosia and changes to affect in 1914 (Langer & Levine, 2014).



**Figure 1-1.** Differences in age of onset and stroke type based on side of lesion (based on Foersch et al., 2005).



**Figure 1-2.** Timing of admission and pharmacological treatment based on side of lesion (based on Foersch et al., 2005). *Note.* tPA, the gold standard clot-busting treatment for ischemic strokes.

the 6-hour point, tPA has little effect. Thus, early identification of stroke is critical to receiving the best care.

Adults with LH stroke are more likely to get to an emergency department within the critical time frame, are more likely to

get tPA, and tend to spend shorter amounts of time in acute care settings. In contrast, adults with RH stroke may not get to the hospital in time to receive the best medical treatment and typically have poorer outcomes (Fink et al., 2002; Foersch et al., 2005; Hedna et al., 2013; Wee & Hopman, 2005). This is particularly evident for transient ischemic attacks (TIAs): of TIAs diagnosed in the hospital, 63% are in the LH, and only 38% in the RH. The reason for this is not likely to be physiological, as RH strokes occur nearly as often as LH. Rather, it may be a difference in the rate of recognition of the mild signs or symptoms of LH versus RH TIAs (Foersch et al., 2005).

Characteristics associated with early arrival to a hospital are provided in Table 1–1. The physical and somatosensory signs (hemiparesis or hemisensory deficits) should occur equally as often from LH and RH strokes. However, in most cases aphasia probably is more obvious than the cognitive-communication deficits associated with RHD and is more likely to be recognized as a problem by patients or family members. Fink (2005) suggests that the presence of anosognosia, or reduced awareness of deficits, could be a major barrier to the recognition of RH stroke symptoms. Anosodiaphoria, or reduced concern for deficits, may also play a role. A person who appears to downplay any changes may be able to convince a spouse or family member not to seek medical attention for him/her. While these suppositions make logical sense, it is unknown how common anosognosia and anosodiaphoria are in initial presentation of stroke and if they are actual contributors to recognition of stroke signs and symptoms.

**Table 1–1.** Characteristics Influencing Timing of Arrival to an Emergency Department

<i>Early Arrival</i>
Good social network
Severe stroke
Hemorrhagic stroke (versus ischemic or TIA)
Signs/symptoms include:
sudden confusion
speech/language problems
hemiparesis
loss of consciousness
<i>Late Arrival</i>
Live alone
Mild stroke
Right hemisphere stroke
Female*

*Note.* \*Females take 46% longer to get to an emergency department and wait 49% longer for treatment at hospitals than males.

*Sources:* Foersch et al., 2005; Jorgenson et al., 1999; Maze & Bakas, 2004; Turan et al., 2005.

The bias in diagnosis and treatment of stroke persists once an individual arrives at a hospital. Two of the most commonly used stroke scales are the National Institutes of Health Stroke Scale (NIHSS; Brott et al., 1989) and the Scandinavian Stroke Scale (SSS; Scandinavian Stroke Study Group, 1985). Both assess motor, sensory, and language functions and are used to assess severity of stroke. However, both are notably biased toward LH signs. Of the 42 points on the NIHSS, seven are related to language function to identify aphasia. Only two points are related to unilateral neglect, and those are based on

observation of performance on a picture description task, not on a specific assessment of unilateral neglect. The SSS has 10 of 58 points related to language, but none for any deficit related to RHD. In a study examining the relationship between NIHSS scores and amount of tissue damage, for mild strokes (scores 0–5) individuals with RHD had twice as much tissue loss (8.8 cc) as those with left hemisphere brain damage (LHD) (3.9 cc), with comparable NIHSS scores (Fink et al., 2002). Additionally the NIHSS is relatively insensitive to cognitive deficits. In one recent study, approximately 40% of patients with an NIHSS score of 0 (extremely mild stroke) had at least one cognitive deficit (Kauranen et al., 2014).

Physicians and researchers from Johns Hopkins (Agis et al., 2010; Gottesman et al., 2010) have suggested several additions to the NIHSS to increase its sensitivity to RHD. One is to evaluate content units (CUs) produced in response to the Cookie Theft picture description task. A variety of measures of CUs (CU/minute, ratio of CU from left and right sides of the picture, number of interpretive CUs [see Chapter 4]) were related to tissue loss in various areas of the RH (Agis et al., 2010). The addition of visual extinction and line bisection tasks (see Chapter 7) also increased the sensitivity of the NIHSS to RH lesion size (Gottesman et al., 2010).

In addition to the stroke scales, neurologists and physicians have other ways to evaluate specific stroke-related deficits. While aphasia often is readily apparent after an LH stroke, it can be relatively objectively screened using a set of easy-to-administer tasks. The same is not true for cognitive-communication deficits associ-

ated with RHD. It is likely that aphasia, which occurs in about 50% of adults with LH strokes, is one of the primary concerns of neurologists. However, cognitive-communication deficits occur with about the same frequency—in about 50% of adults with RH strokes (Blake, Duffy, Myers, & Tompkins, 2002; Côté, Payer, Giroux, & Joannette, 2007)—but may not be considered at all.

Unilateral neglect is arguably the best-known deficit related to RHD. Indeed, the presence of neglect increases a patient's chance of receiving tPA by approximately 40% (Di Legge, Fang, Saposnik, & Hachinski, 2005). It is commonly assessed by asking a patient to draw simple pictures such as a butterfly or an analog clock. Such representational drawing tasks are not very sensitive and may identify only a small percentage of individuals with unilateral neglect (Appelros, Nydevik, Karlsson, Thorwalls, & Seiger, 2003). Additionally, according to a recent study of acute stroke, unilateral neglect occurs in only about 25% of patients, and the presence of neglect alone identifies only 63% of RH strokes (Dara, Bang, Gottesman, & Hillis, 2014). Thus, even a sensitive measure of neglect will not fix the imbalance in the recognition of LH and RH stroke.

Beyond the initial diagnosis and medical treatment, the absence of clear patterns of deficits and a standard label for “right hemisphere cognitive-communication disorders” creates problems in both research and clinical practice. In research studies, often there are no a priori criteria to identify and exclude the potential participants who have no cognitive or communication disorder. This adds to the heterogeneity of participant samples, reducing the power



of the experiments and the strength of the conclusions that can be drawn. Additionally, there are no standard clinical procedures for determining the presence of a cognitive-communication disorder. This is complicated by the limited options for valid, reliable, and sensitive assessment tools (see discussion in Chapter 2).

The disparities continue after a patient is sent home, in regard to available resources. General resources for stroke survivors obviously would be the same for RHD and LHD. However, an individual with aphasia has numerous resources for advocacy groups, support groups, and sources of education (Aphasia Access, National Aphasia Association, Aphasia Now, etc.). A patient with “cognitive-communication deficits” or some other vague diagnostic label will have a much harder time finding resources or education sources specific to his/her deficits.

### **Impact of Deficits Associated With RHD**

A variety of studies have been conducted to identify predictors of stroke outcome. While there are many discrepancies across studies, some general patterns are apparent in relation to deficits associated with RHD (Table 1–2). The length of stay in a medical setting, either in acute care settings or acute and subacute settings combined, has been related to severity of stroke, the presence of unilateral neglect,

and the presence of cognitive deficits (Appleros, 2007; Gillen, Tennen, & McKee, 2005; Jorgenson et al., 1999; Kong, Chua, & Tow, 1998; Pedersen et al., 1996). Functional status at discharge has been linked to stroke severity, age, unilateral neglect and anosognosia, depression, and presence of cognitive deficits (Meijer et al., 2005; Paolucci et al., 1996; Pedersen et al., 1996; Vossel, Weiss, Eschenbeck, & Fink, 2012; Wee & Hopman, 2005). The likelihood of being discharged to a dependent-living environment is related to older age, anosognosia for illness, unilateral neglect, and presence of cognitive deficits (Jehkonen et al., 2001; Kammersgaard et al., 2004; Paolucci et al., 1996; Wee & Hopman, 2005).

The presence of cognitive deficits impacts a variety of outcomes. However, what constitutes a “cognitive deficit” is not clear. While in speech-language pathology aphasia is generally considered in its own category and deficits such as attention, memory, and executive function are put into a “cognitive” category,<sup>2</sup> many other disciplines do not make this distinction. Thus, the outcomes linked to cognitive disorders described above are linked to problems in attention, memory, executive functions, and/or aphasia. A second issue is how cognition is measured. Often, general screenings such as the Mini Mental State Exam (Folstein, Folstein, & McHugh, 1975) are used as indicators of cognitive deficits. Such tools are designed only to screen for such deficits and are not sensitive measures of cognition (see discussion in Chapter 2).

<sup>2</sup>Language is a cognitive function, and thus aphasia is a cognitive disorder. However, there is a long-standing tradition in speech-language pathology to think of language separate from the “other cognitive disorders.”

**Table 1–2.** Predictors of Stroke Outcomes

	<i>Stroke Severity</i>	<i>Age</i>	<i>Depression or Anxiety</i>	<i>Cognitive Deficits</i>	<i>Unilateral Neglect</i>	<i>Anosognosia</i>	<i>Visuoperceptual Deficits</i>
Length of stay (acute/subacute settings)	x			x	x		
Functional status upon discharge	x	x	x	x	x	x	
Discharge to dependent-living setting		x		x	x	x	
Long-term recovery	x	x		x			
Participation level outcomes	x	x		x	x		x
Quality of life	x	x	x				
Mortality	x	x		x			x
Independence in activities of daily living					x	x	x

# 9

## Awareness

Awareness of one's own abilities, deficits, strengths, and weaknesses can impact performance in daily life, vocational success, and participation in rehabilitation. Awareness can be impaired following brain injury, particularly when the RH is damaged. This chapter will cover definitions, types, and models of awareness, how the RH is involved, and how to assess and treat deficits of awareness.

### **Anosognosia**

The word "anosognosia" comes from Greek and means "without knowledge of disease." In clinical practice it is used to refer to the reduced awareness of either acquired deficits or the consequences of those deficits. The term was initially coined in 1914 by Babinski in reference to reduced awareness of hemiplegia (Babinski, 1914; translated by Langer & Levine, 2014). However, descriptions of reduced awareness first appeared over 30 years earlier when von Monakow described such a

deficit in relation to symptoms of Korsakoff's syndrome. Anton and Pick (known today as namesakes of types of cortical blindness and frontotemporal dementia) also described aspects of reduced awareness in the late 1800s (Prigatano, 2010a).

As with many disorders, there are inconsistencies in terminology (Table 9–1). "Anosognosia" is commonly used for reduced awareness of specific impairments, most often hemiparesis and unilateral neglect. Anosognosia is the label used in research with stroke survivors, while "Impaired Self-Awareness" (ISA) is preferred in the literature on TBI. ISA is defined more broadly; in addition to referring to reduced awareness of a specific deficit, it encompasses the functional implications of that deficit, the patient's expectations for recovery, differential awareness for different domains, and adherence to treatment (Orfei, Caltagirone, & Spalletta, 2009). Some even use ISA synonymously with metacognition (Schmidt, Lannin, Fleming, & Ownsworth, 2011), as both refer to one's understanding of one's own strengths and limitations and

**Table 9–1.** Terminology Related to Anosognosia

<i>Terms</i>	<i>Description</i>
Anosognosia	Reduced awareness of acquired deficits; typically used in relation to reduced awareness of specific impairments
Denial of deficit	Connotes that there is some awareness that allows for psychological refusal to acknowledge the deficit (conscious or unconscious)
Impaired self-awareness	Commonly used in TBI literature to refer to reduced awareness; can be used for specific impairments or general awareness; often includes insights about consequences and motivation to participate in therapy
Lack of insight	Connotes higher-level cognitive deficit implicating higher-level reasoning; reduced understanding/awareness of the consequences of an impairment
<i>Types of Unawareness</i>	
Explicit awareness	Ability to verbally report the presence of a deficit
Implicit awareness	Changes in behavior related to the presence of a deficit (e.g., to avoid failure related to a deficit)
<i>Related Disorders</i>	
Alexithymia	Reduced use of emotion-related words
Anosodiaphoria	Reduced emotional reaction to, or concern for, deficits
Asomatognosia	A form of disturbed sense of ownership in which a patient believes his impaired limb is missing or does not belong to him
Misoplegia	Hatred of one's limbs or body part(s)
Personification	Refer to and treat a limb as if it were its own being (e.g., naming one's arm)
Somatoparaphrenia	A form of disturbed sense of ownership in which a patient feels her impaired limb belongs to someone else

how those will impact performance on daily activities. Prigatano and Morrone-Strupinsky (2010) use the label “anosognosia” to refer to a complete unawareness of a specific impairment, and use “ISA” to refer to partial unawareness that may be a stage of recovery from anosognosia.

“Lack of insight” can be described as a component of ISA. It connotes a cognitive deficit implicating higher-level reasoning. This may occur in some patients who have a reduced awareness of the *consequences* of a deficit, even if they do have awareness of the deficit itself. A patient who

can describe his hemiparesis, but in the next sentence talk about how he plans to resume his weekend bike rides with his son once he is discharged from the hospital could be described as having reduced insight.

The phrase “denial of deficit” often is used synonymously with anosognosia in clinical practice (Prigatano & Klonoff, 1998). However, denial is very different from reduced awareness. In order to deny that something exists, you must be aware of it and consciously reject it. Using the phrase “denial of deficit” may cause families to erroneously believe that the patient is being difficult or refusing to admit a problem, when in reality the patient is not aware of the existence of the deficit at a conscious level.

Another commonly used but not quite accurate label is “unawareness.” While some researchers and clinicians use “unawareness” synonymously with anosognosia, the former suggests a complete loss of awareness and does not convey the nuances of the disorder, in which a patient may be aware of hemiparesis but not of cognitive deficits; have different levels of awareness of upper and lower extremity weakness (Berti, Ladavas, & Della Corte, 1996); demonstrate awareness that appears to increase or decrease depending on the questions asked; or may not verbally report hemiparesis but never try to stand up unassisted (Mograbi & Morris, 2013; Nurmi & Jehkonen, 2014; Orfei et al., 2007). For these and other reasons, Prigatano (2013) cautions that anosognosia should not be considered a unitary disorder.

Nurmi and Jehkonen (2014) highlight some of the difficulties and inconsisten-

cies in research on anosognosia. First, as described above, there are inconsistencies in definitions and terminology. Second is the distinction between explicit and implicit awareness (Fotopoulou, Pernigo, Maeda, Rudd, & Kopelman, 2010; Mograbi & Morris, 2013; Moro, Pernigo, Zapparoli, Cordioli, & Aglioti, 2011). Explicit unawareness is measured by verbal responses to questions (e.g., is there anything wrong with your arm?). Implicit unawareness, in contrast, is observed in patients’ behaviors. A patient who does not verbally acknowledge her hemiplegia but who never attempts to get out of bed without assistance might have implicit but not explicit awareness of her deficit. Another example of implicit awareness comes from studies that employ bimanual tasks (e.g., Cocchini, Beschin, Fotopoulou, & Della Sala, 2010; Moro et al., 2011). Some individuals with explicit anosognosia will use strategies to complete bimanual tasks that suggest implicit awareness of upper limb paralysis. For example, when asked to lift a two-handled tray, they will lift with one hand in the middle of the tray instead of attempting to lift from the two ends. In studies of Alzheimer’s disease, some patients may have emotional reactions to failure despite not being able to explicitly acknowledge the poor performance (Mograbi & Morris, 2013). Not all individuals with anosognosia have preserved implicit awareness. Evidence from priming studies (Fotopoulou et al., 2010; Nardone, Ward, Fotopoulou, & Turnbull, 2007) suggests that some patients with anosognosia show reduced activation of relevant disability-related words (e.g., weakness, walk) compared with individuals with hemiparesis but intact awareness,

indicating that explicit and implicit awareness can be affected differentially.

Vocat and Vuilleumier (2010) suggest that the dissociation between implicit and explicit awareness could be due to two separate monitoring systems. One is a subcortical system that provides implicit, automatic monitoring of “affective relevance of a mismatch between a goal and the outcome” (p. 267). The other is a cortical system residing in frontal and parietal lobes which provides “conscious error detection based on the quality of feedback and on access to attentional and executive networks” (p. 267). Damage to the former would cause a deficit of implicit awareness, and the latter would result in problems with explicit awareness.

### **Models of Anosognosia**

There have been a variety of theories of anosognosia over the years. Some of the earliest were motivational or psychodynamic theories in which anosognosia was described as a form of psychological denial that was used as a defense mechanism (Weinstein & Kahn, 1955). While the terminology “denial of deficit” lingers, strong versions of these theories have been discarded in light of disconfirming evidence. Anosognosia has been identified in acute stages of stroke recovery, before patients have had a chance to experience their deficits (e.g., attempting to walk to the bathroom with a hemiparetic leg) or the broader consequences of them (e.g., not being able to drive with hemiparesis). Without the experience of the loss, there is no need for a psychological defense against it. Other evidence against this theory is the fact that some patients

can be aware of some deficits (e.g., hemiparesis) but not others (e.g., unilateral neglect) (Berti et al., 1996; Bisiach, Vallar, Perani, Papagno, & Berti, 1986). Turnbull, Fotopoulou, and Solms (2014) argue that such evidence does not spell the death knell for the idea that anosognosia may have an emotional component related to a defense mechanism. They argue that emotional deficits associated with RHD result in the person viewing the world as he would like it to be, as opposed to the reality. Thus, for some clients, emotion and motivation may play a strong role in anosognosia.

Geschwind (1965) suggested a disconnection model, in which verbal reports of awareness were disrupted by a disconnection between the RH sensory and proprioceptive processing areas and the LH language areas. If this were true, then there should be dissociations between verbal and nonverbal assessments of awareness. These dissociations have not consistently been found.

More recent theories use anatomical models and include different levels of awareness. Higher-level, conscious awareness is thought to be controlled primarily by the prefrontal regions. Low-level, modality-specific awareness is localized posteriorly, in the temporal and parietal lobes. Damage to either region could result in reduced awareness.

McGlynn and Schacter’s (1989) Conscious Awareness System (CAS) resides primarily in the prefrontal regions and works in concert with judgment, insight, and self-reflection processes. Damage to the CAS may result in a global unawareness of self. The input from modality-specific systems (visual, somatosensory) is intact, but the signals are not processed

correctly by the damaged CAS, thus resulting in incorrect interpretation and self-monitoring of the sensory input. In this model, cognitive and affective states may be part of the presentation of anosognosia.

In contrast to the CAS model are the modality- or domain-specific accounts of anosognosia (Bisiach, 1990). Damage to the temporal and parietal sensory processing areas may result in disruptions to connections or signals sent to the frontal lobes for processing by the CAS. For example, an RH parietal lesion may result in unilateral visual neglect. If information about the incomplete visual representation is not sent to the frontal lobes, or if erroneous information is sent (e.g., the visual representation is complete), then the central processor will not detect a problem, resulting in reduced awareness of the unattended visual field.

The theories with the most empirical support purport that anosognosia for hemiparesis is caused by a disruption in the motor control system. The motor system is thought to control intention to move, the movement itself, and a comparison between the intended movement and the actual movement based on sensory feedback. Disruption to either the intention or the comparator system has been implicated. According to the feed-forward model (Heilman, 1991; Wolpert, 1995), there is a loss of intention to move. In the intact system, the intended movement would be compared with the actual movement, and discrepancies would be noted. However, if there is a loss of intention, then there would be no discrepancy with an absence of actual movement. In the feedback model (Berti & Pia, 2006; Spinazzola, Pia, Folegatti, Marchetti, & Berti, 2008; Wolpert, 1995), the disruption occurs

in the comparison process. The intended (desired) and predicted results match, but the “comparator” does not correctly identify a mismatch between these two states and the actual movement. If no mismatch is identified, then there is no awareness that the movement was incorrect or did not occur as planned. This model can explain the phenomenon of illusory movement, in which patients report that they felt a movement occur, even in the face of contradictory visual and sensory feedback (Feinberg, Roane, & Ali, 2000; Fotopoulou et al., 2010). Jenkinson, Edlestyn, Drakeford, and Ellis (2009) reported that adults with anosognosia for hemiparesis are impaired in determining whether they had seen or imagined pictures or had performed, observed, or imagined actions. They tend to recall having seen pictures or performed actions that had only been imagined, indicating a deficit in reality monitoring.

## Related Disorders

There are several disorders that are related to, or commonly co-occur with, anosognosia (see Table 9–1). In some cases, the disorders are erroneously considered to be parts of the same problem. First is unilateral neglect. Some researchers appear to equate unilateral neglect and anosognosia: “right hemispheric stroke is usually associated with neglect, which reduces awareness of neurological deficits” (Foersch et al., 2005, p. 392). While patients with unilateral neglect often have anosognosia for neglect, the deficits are distinct disorders that can be dissociated (Appelros, Karlsson, & Hennerdal, 2007; Berti et al., 1996; Bisiach et al., 1986; Vocat et al., 2010).

Additionally, some individuals with anosognosia for unilateral neglect can be aware of other deficits, such as hemiplegia.

Second is anosodiaphoria, which is a reduced concern for deficits, or reduced emotional expression related to those deficits (Babinski, 1914/Langer & Levine, 2014). This too has been dissociated from anosognosia. Some patients may be aware of their deficits and be able to identify and describe their hemiparesis but show no apparent concern over the loss of motor control. It is not clear whether there is reduction in emotional experience or if the problem is in the expression of emotion. Related to the latter is alexithymia, a reduced use of emotional words (Heilman & Harciarek, 2010; Jorge, 2010). Again, these two deficits may co-occur, but just because a person is not using many emotional words does not mean that he or she is not experiencing emotional responses.

Third, some individuals with anosognosia develop delusional beliefs about their hemiparetic limbs (Bottini et al., 2010; Giacino & Cicerone, 1998). Several of these fall under the category “disturbed sense of

ownership” in which patients do not feel that a paretic limb really belongs to them (Karnath & Baier, 2010). One form of this is asomatognosia, in which they believe their limb is missing. In another form, somatoparaphrenia, patients attribute the impaired limb to someone else.

Other phenomena include misoplegia, in which patients develop a hatred for the impaired limb, and personification, in which patients develop a name and personality for the impaired limb. For example, a patient who names her hemiplegic arm “Connie” and gives reports about how Connie is doing on a particular day would be showing signs of personification. These delusional beliefs are productive deficits, in which there is an exacerbation or production of additional function, while anosognosia itself is a defective disorder in which there is reduction of function (Bottini et al., 2010).

Finally, confabulation can be observed in some individuals with anosognosia. The source of confabulations is not well studied, but they are thought to be an unconscious response to behaviors that

### **SIDEBAR**

My first experience with anosognosia was with a patient who had been diagnosed with a right hemisphere tumor. His initial symptoms included getting lost in the hardware store in which he worked and bumping into the wall when walking down a hallway in his house. During a preoperative assessment in which he was asked about the latter problem, he explained: “My wife hangs too many pictures on the wall and I don’t like them. So when I’m walking down the hall, I hit them so they fall off the wall.” He confabulated an explanation for the symptoms because he was not consciously aware of the unilateral neglect caused by the RH tumor.