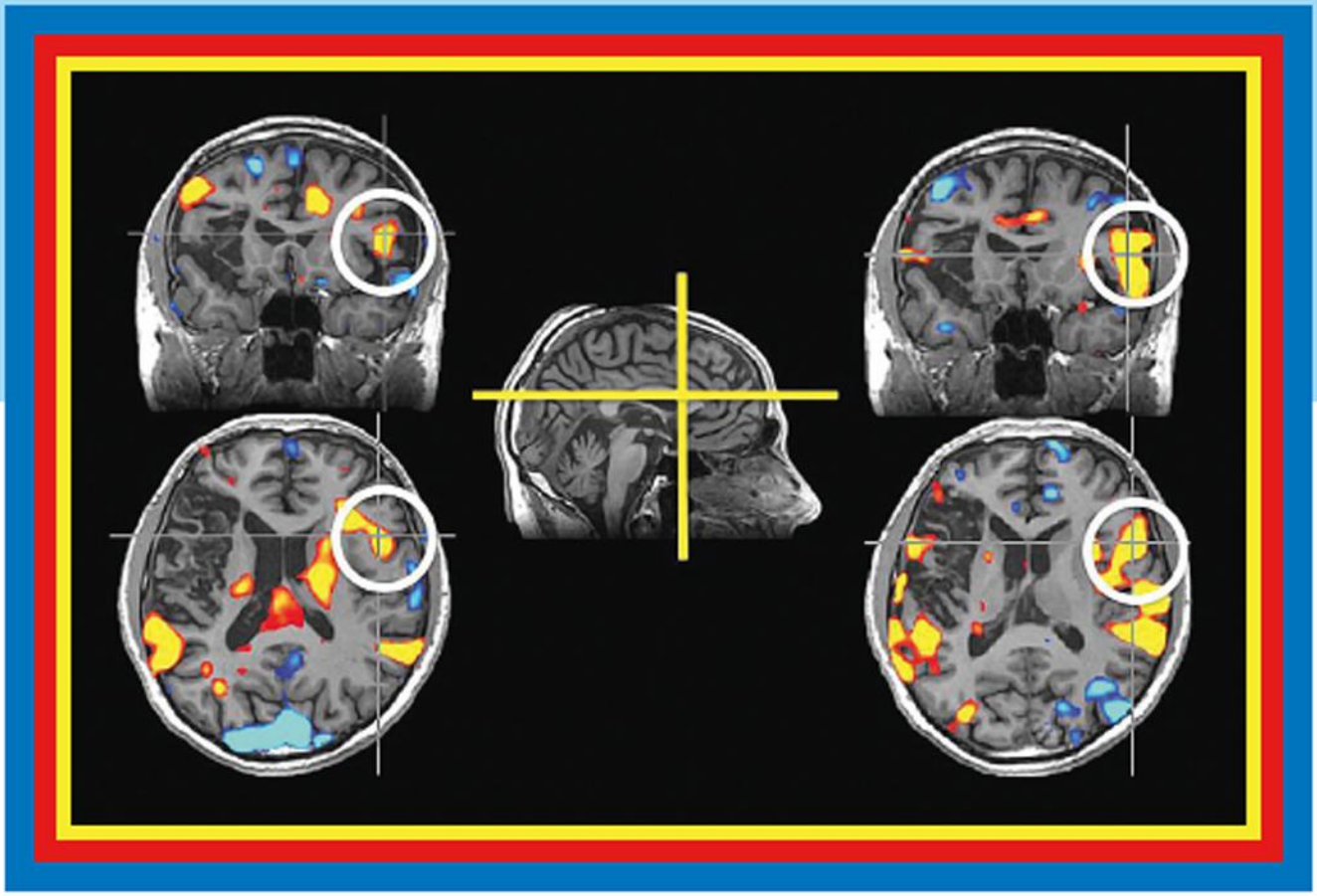
Aphasia and Related Neurogenic Language Disorders

Fifth Edition

Leonard L. LaPointe
Julie A. G. Stierwalt



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Aphasia and Related Neurogenic Language Disorders

Fifth Edition

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To Pierre Paul, for your inspiration and labor. So glad we share a birthday, and I'd share cake if I could.

Leonard L. LaPointe

To Kyle, Jordan, and Harrison. Your constant love and support means the world to me.

Julie A.G. Stierwalt

Contents

Preface to the Fifth Edition

Amid the patisseries and bistros of the Latin Quarter with its students and tourists sipping espresso and enjoying the salted caramel flavored dessert of the week reside Leborgne and Lelong, whose brains still float in the formaldehyde of history in the Musée Dupuytren*. These French brains symbolize the largesse of the Master French brain. [Paul] Broca's most profound contributions were to be that of the final intellectual frontier, the human brain. His work has provided foundation and principled theoretic rationale for modern and surely forthcoming advances in what we know about our brain, our* cerveau*, and our most human trait, the ability to communicate. Our brain is the origin of our being and our identity. It is you. It is me. It is us.*[1]

What's past is prologue. Prefaces in previous editions of this book have invoked images that arise from Navajo prayers and ancient Chinese poems. The writers of these special images create evocative figurative language and wispy metaphors that are perfect examples of the full use and interplay of human language and emotion. Language and emotion are many times inseparable. Paul Broca knew that and he, too, has molded thought and words into not only remarkably apt imagery, but also milestones of neuroscience. His extraordinary contributions on the use and loss of words are rife with life and full of moments, revelations, homecoming, remembrances, exile, admonitions, belonging, grief, and coping. All of these are part of enjoying and using human language, despairing at its loss, and marveling at its recovery. The metaphor of wreck and raft is still appropriate.

Remarkably, this is the fifth edition of this collection of ideas and scholarship on brain damage and communication loss. It is archived in libraries and in classrooms throughout the world. Again, we have assembled a group of experts on brain-based disorders of communication who have been there: in the clinics, hospitals, research labs, and classrooms. Previous editions were Presidents ago, and the original Thieme compilation was crafted more than a generation hence. Time flies when you are rescuing language. Now, all of a sudden it is 2018. A new generation of aphasiologists is slipping into the fore. The science and the art are changing. When the first edition of this book was born, we knew nothing of flat screens, Twitter, ATMs, the Internet, and DVDs; and blackberry was a fruit. We knew nothing of iTouch, iPod, iPhone, or iPad. We had tape-based answering machines and navigation done mostly with the stars and paper maps, and not by satellites that plotted and tracked global positioning. Our garages and attics were full of junk until we discovered eBay and now our garages and attics are refilled with transferred junk. Hand sanitizers are ubiquitously available. We bought heads of lettuce and not salads in a bag. We watched YouTube emerge and coax the universe to broadcast itself on everything from how to play blues ukulele to how to prepare Moroccan couscous.

Significant changes have arrived in health care and bioscience as well. The Genome Project was a dream just a blink ago, and brain fitness and neural plasticity were considered by many to be fanciful. Now, some are touting research and breakthroughs on experience-based neuroplasticity as a breakthrough and illustrating ways in which the brain changes with intervention.[2] The human brain should no longer be considered immutable. Changes in neuroarchitecture and neuroconnectivity that are directly associated with behavioral treatments are being reported and carefully studied every day, and the idea that carefully selected and programmed therapeutic experiences in the proper dosages can actually change the brain is one of the most exciting developments in brain and rehabilitative science in many a moon—or maybe ever. That is precisely why this new edition on this expanding topic of neurorehabilitation of aphasia and related disorders is necessary and topical.

We have radically overhauled this book for the fifth edition. Gone are the chapters on selected types of aphasia. We are grateful to the contributors of the previous editions, and we now welcome a new generation of experts on a wider array of topics. With this edition, we retain the humanistic nature and the fabric of care that was characteristic of previous editions. We have added a chapter on neuroanatomical basics that is richly illustrated from Thieme's deep vault of exquisite illustrations. We have added chapters on telepractice, digital and electronic advances, funding and reimbursement for speech and language services, and the interactions of communication and cognition. We also have expanded a penetrating look at practice in neurogenic communication disorders in the acute care hospital setting. In fact, this chapter comes to us from one of the premier hospital settings in the United States, the Mayo Clinic. We also have up-to-date information on neural imaging and brain-based communication disorders. In response to reviews from users of previous editions, we have added chapters as well on disorders of comprehension, on syntax and linguistic-based disorders, and on communication pragmatics and discourse. We have incorporated the social model of aphasia intervention with a detailed account of life participation and group approaches to treatment. Notably, we have recruited chapters on assistive technology and on treatment effectiveness and evidence-based practice. Our contributions on specific syndromes include attention to primary progressive

aphasia, right hemisphere damage, dementias, and traumatic brain injury, with an expanded focus on polytrauma and blast injuries. We have updated as well our well appreciated chapter on resources for both families and clinicians. The book is sprinkled with pedagogical tools such as sidebars, glossaries, discussion questions, test questions, case examples, and pearls of wisdom. This fifth edition is quite different from previous editions. Additionally, we have added as co-editor Dr Julie A.G. Stierwalt of the Mayo Clinic Rochester, who has contributed deftly and mightily to this new edition. I believe we have been responsive to the reviews of user clinicians, user students, and user academics and now have a book that covers the waterfront of aphasia and related neurogenic communication disorders. I hope you find it both instructive and useful. The Navajo prayers, Chinese poets, and the landmark neuroscientist Paul Broca all have found ways to express the frustration and hope that are characteristic of lost language and lives turned upside down. Thank you for your daily dedication to helping people climb onto the raft and reach shore.

Leonard L. LaPointe, Co-Editor

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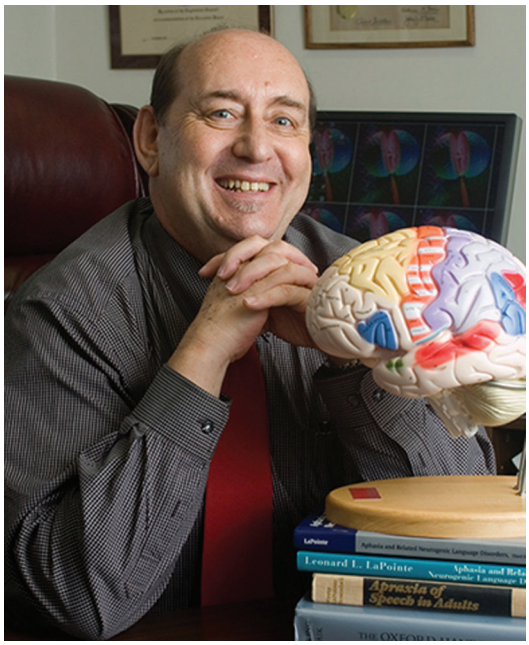

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Julie A.G. Stierwalt, PhD, received her bachelor's and master's degrees from the University of Northern Iowa, and her doctoral degree from the University of Iowa. She is currently a Senior Associate Consultant in the Division of Speech Pathology, Department of Neurology at Mayo Clinic, where she serves patients with a wide range of speech, language, cognitive, and swallowing impairments. Dr. Stierwalt is a Fellow of ASHA with numerous journal publications, chapters, and a coauthored textbook.

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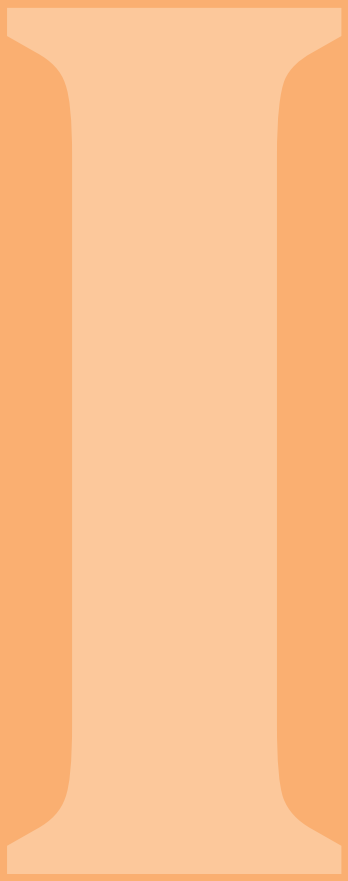
Section I

Foundations and Practicalities



1 Brain Basics

Leonard L. LaPointe

Introduction

The brain is a modest yet spectacular thing. In this 3.5 pounds of gelatinous squish tissue rests everything we are or have ever been. The brain is the seat and soul of our fears, our joys, our achievements, our relationships, our creativity, our happiness, our sadness, our identity, our memory, and our history. The brain allows us to acquire language, to interact with one another, to cajole and console, to cheer, to thank, to joke, to sing off-key, to talk to the animals, and to aspire. The miracle of language makes us human, more so than the opposable thumb and the ability to conceptualize death and subsequently fear it. Pessimists among us would say that the brain is inextricably linked to abilities to lie, steal, cheat, go to war, and ruin our nest. But, for all human foibles, there is ample evidence that language and intraspecies communication is responsible for the sum of human achievements, altruism, and decency. Debates on what makes us human have boiled for generations but always atop the list of human attributes is language and sophisticated communication.[1] The brain has been rhapsodized in poem and song and called everything from an enchanted loom that weaves a never-ending stream of dissolving patterns to a computer on steroids.[2]

Listen to Diane Ackerman[3] talk about the brain:

> *Shaped a little like a loaf of French country bread, our brain is a crowded chemistry lab, bustling with nonstop neural conversations. Imagine the brain, that shiny mound of being, that mouse-gray parliament of cells, that dream factory, that petit tyrant inside a ball of bone, that huddle of neurons calling all the plays, that little everywhere, that fickle pleasure dome, that wrinkled wardrobe of selves stuffed into the skull like too many clothes into a gym bag.*[3]

What a poetic and rapturous characterization of what used to be regarded as something that showed no more capacity for thought than a cake of suet or a bowl of curds. How times have changed. The brain in all its wonder forms the infrastructure for all that will be discussed in this new edition of our book. This chapter presents an abbreviated précis of some brain basics. The discussion is not meant to be exhaustive or complete, but rather presents some common ground for understanding the subsequent chapters in this book.

The brain and the human nervous system are vital to the perception and production of processes that take place inside (enteroception) or outside (exteroception) the body. Not the least of these processes is internal and external communication. The brain keeps us in touch with our environment. It allows all of our sensory perceptions (taste a hot fudge sundae; hear the chirping of a chickadee; view the sunset at Cedar Key; touch the soft fur on the head of Annie, the golden retriever) as well as everything we do to move or communicate within the environment (swim to the raft at Sawyer Lake; hit a drop shot in tennis; do a full twisting dismount from the pommel horse; operate the lever of the recliner chair; fuss with the television's remote control; or pick up the baby and smell she needs a diaper change). In our specialty areas of human exchanges of information and feelings, the brain is responsible for the vast spectrum of human communication, such as writing a poem; saying you are sorry; texting the important message that you are in the produce section of the market, near the broccoli, and will be home soon; or telling your daughter you love her.

This topic is important to learn because it forms the foundation for subsequent courses in human communication and its disorders. It will aid greatly in solving clinical problems and is the anchor of knowledge that fastens itself to many clinical problems in both children and adults. Children have tiny nervous systems; this not only is relevant to the adult disorders and the role these brain basics play in both diagnosis and treatment of the myriad disorders of human communication but also will help clinicians and researchers understand the basis of many of these maladies. The current state of neuroscience is advancing rapidly. We know a lot more today about basic genetic and microscopic aspects of our nervous systems and the technology of applications to diagnosis, treatment, and malleability of brain

structures, and its enigmatic nest of connections is becoming less of a mystery and more understood.

Basics of Neuroscience

Given the diversity of human communication's perceptual and production tasks, the human body has evolved an intricately complex nervous system that can be subdivided and discussed in various ways, as can be found in the study of Schuenke et al[4] as well as in the relatively new neuroanatomy book designed for communication science and disorders by LaPointe.[5] The principles of classification vary; a common one is to divide the nervous system morphologically into a peripheral nervous system (PNS) and a central nervous system (CNS). The brain and spinal cord comprise the CNS, and they are seamlessly interconnected in a functional unit (**Fig. 1.1**). The PNS is formed by the nerves that emerge from the brain and spinal cord (cranial and spinal nerves) and are distributed into the periphery of our vast collection of muscles, tissues, and organs. The brain and spinal cord consist of neurons or brain cells that have been characterized as gray matter or white conducting part of a neuron, known as the axon, covered by a fat-like myelin sheath that looks white upon gross examination of a specimen and, hence, the term *white matter*.[4] The cell bodies of neurons, on the other hand, are not covered with myelin and appear gray to the naked eye, and hence the term *gray matter*. To help protect the brain from external injury, which is not entirely possible in such instances as slamming into a bridge abutment or drunkenly diving into the shallow end of the swimming pool from a balcony, the CNS is encased in bony structures (vertebrae and cranial bones). Between the bones and the CNS are coverings (meninges) of the brain and spinal cord. The meninges are composed of three layers: pia mater ("pious mother"), arachnoid ("spiderlike"), and dura mater ("tough mother"). It is always a pleasure to demonstrate these coverings in my neuroanatomy class by soliciting the cooperation of usually three males from the class whose heads are then covered with plastic wrap as they assume the postures of piety (pia mater), Spider-Man (arachnoid), and a tough mother (dura mater).

CNS Divisions

The CNS can be further studied and divided into the telencephalon (new brain), diencephalon (between brain), and the brainstem. For our purposes in this book, the telencephalon, which is composed of the cerebrum, including the later evolved cerebral cortex, is of most interest to us. The lobes of the mirrorlike left and right hemispheres of the cerebral cortex are traditionally divided morphologically and functionally into the frontal lobes, temporal lobes, parietal lobes, occipital lobes, and the hidden insular lobes. Although the external surfaces of the left and right cortical hemispheres look alike, like sisters, they can be very different. We will discover this as the stories in this book unfold. Furthermore, as a method of labeling morphologic and functional areas of the cerebral cortex, a traditional numeric labeling system developed by Brodmann is used in many anatomic and physiologic depictions.

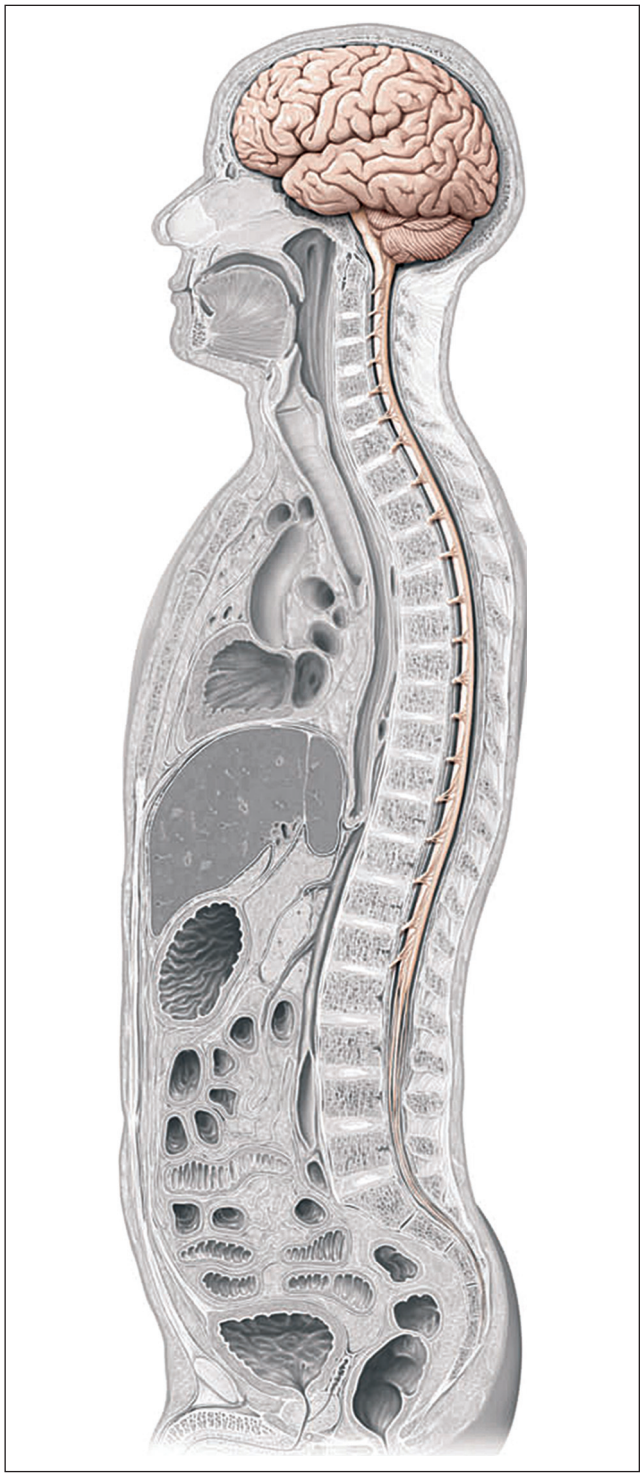


Fig. 1.1 Central nervous system, in situ, left lateral view. (Reproduced with permission from Schuenke et al. Illustration by Markus Voll.)[4]

Figs. 1.1, **1.2**, and **1.3** depict the morphologic and functional basics of the CNS. In **Fig. 1.3**, a left lateral view, the primary sensory and motor areas are shown in red and the areas of the association cortex are shown in shades of green. Projection tracts begin or end in the primary sensory or motor areas. More than 80% of the cortical surface area is association cortex, which

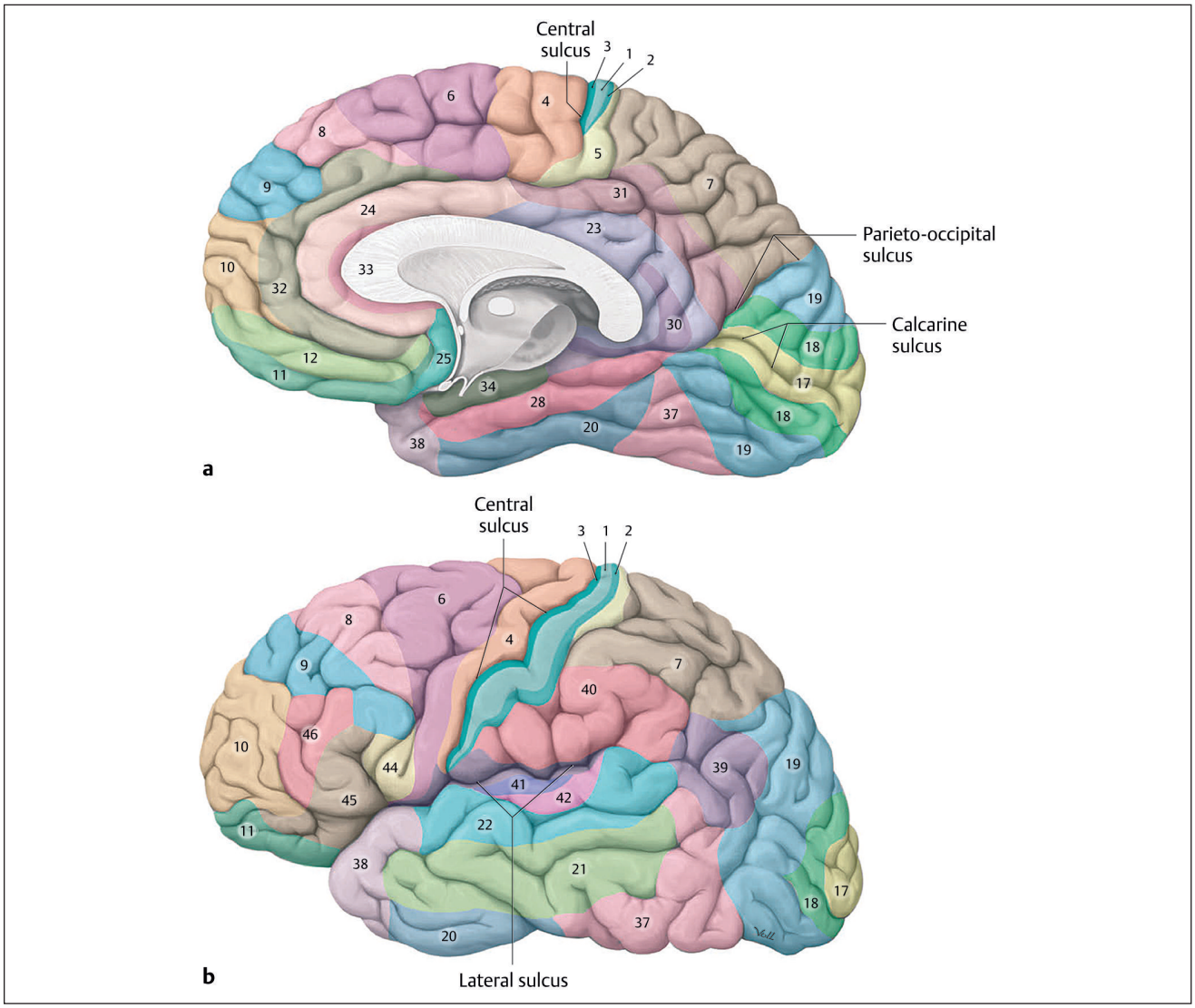


Fig. 1.2 Brodmann's areas in the neocortex. (**a**) Midsagittal section of the right cerebral hemisphere, viewed from the left side. (**b**) Lateral view of the left cerebral hemisphere. (Reproduced with permission from Schuenke et al. Illustration by Markus Voll.)[4]

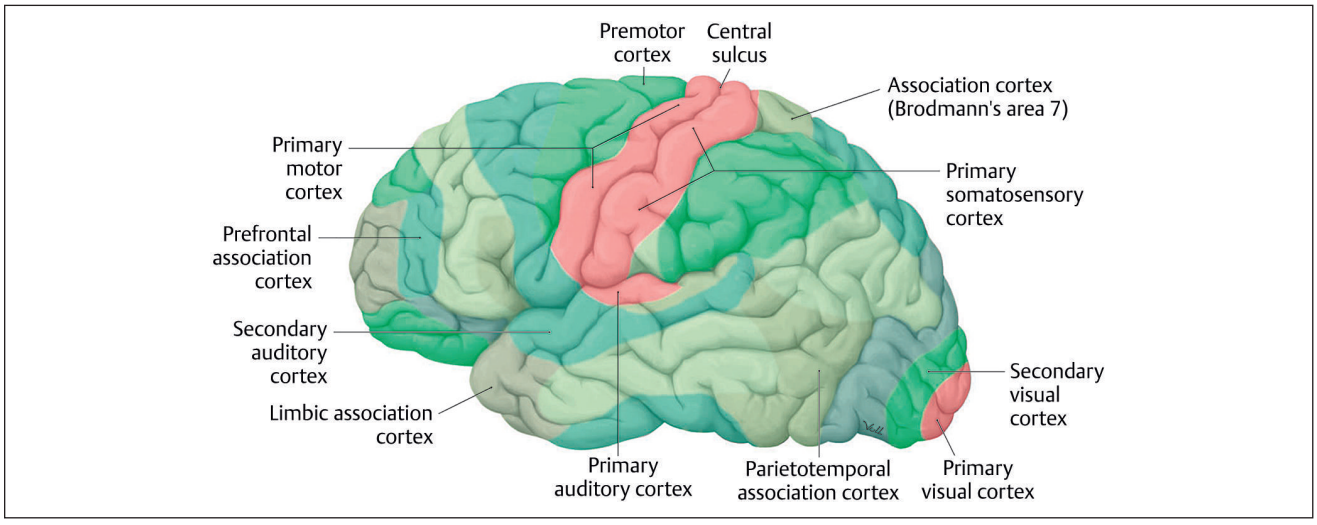


Fig. 1.3 Functional organization of the neocortex, left lateral view. (Reproduced with permission from Schuenke et al. Illustration by Markus Voll.)[4]

is richly interconnected with the primary sensory and motor areas (Box 1.1).

Box 1.1 Pearl

Leonardo da Vinci is arguably the single most pungently creative and historically dominant person in human history or herstory. Now over 600 years down the road, a posthumous study of his neuroanatomy is unravelling the secrets of how his astounding brain was able to function at such an extraordinary level. In an incredible book, *Leonardo's Brain: Understanding Da Vinci's Creative Genius*,[6] surgeon, inventor, and author Leonard Shlain created a startling exploration into da Vinci's brain. Shlain, who died after publishing the book, ironically of brain cancer, reveals how the "undereducated, left-handed, nearly ambidextrous, vegetarian, pacifist, gay, singularly creative Renaissance male" was able to contribute to both science and art at levels surpassing nearly any other human. Based on neuroimaging, Shlain postulates that di Vinci's unusual white matter, brain wiring, and rich neural connectivity contributed to his rare gifts of intellect and creativity.

When neurons are activated, they consume glucose and oxygen to remain healthy and viable, and that is why disruption of the blood supply to the brain is a primary type of neuropathology that can create many of the problems discussed in this book. The primary neurovascular systems that deliver nutrients to the neurons or brain cells are the vertebral and carotid arterial systems (Fig. 1.4). The middle cerebral arterial division of the carotid system is particularly associated with damage to the language areas of the brain. Fig. 1.4 shows the primary arterial systems that nourish the brain.

The brain contains several language areas whose damage is associated with the clinical signs and symptoms outlined in subsequent chapters. Although the box-and-arrow oversimplified diagrams of yesterday are recognized in today's understanding of parallel distributed neural processing and the rich

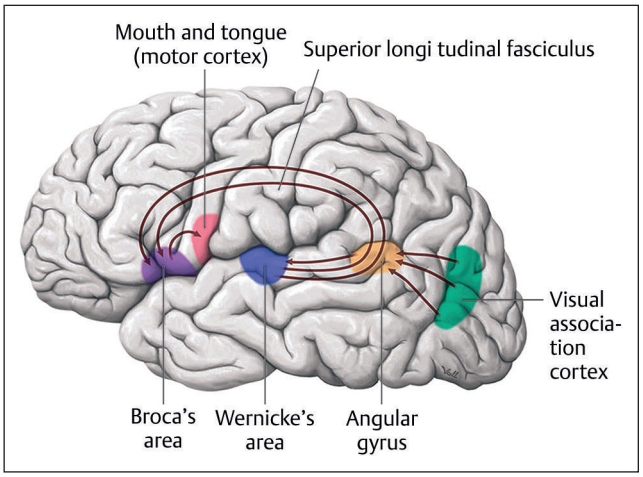


Fig. 1.5 Language areas in the normally dominant hemisphere, lateral view. (Reproduced with permission from Schuenke et al. Illustration by Markus Voll.)[4]

interconnectivity that is involved in complex language and cognitive functions, there still are recognized areas that play important roles in cognitive-communicative activities. The Wernicke area (Brodmann's area 22) in the dominant cerebral hemisphere is highly associated with language comprehension and understanding, and Broca's area (Brodmann's areas 44 and 45) is associated with language production (Fig. 1.5). The areas around the parietal, temporal, and occipital lobe borders (supramarginal and angular gyri) are a third primary language area that has been associated particularly with reading and writing. Fig. 1.5 shows primary speech and language systems of the brain.

The dorsolateral prefrontal cortex and the ventromedial prefrontal cortex have emerged as important areas related to the complex cognitive functions, including all of the behavioral characteristics included in the nebulous term *executive function* (Fig. 1.6). Fig.1.6 shows areas of the brain related to complex cognitive functions.

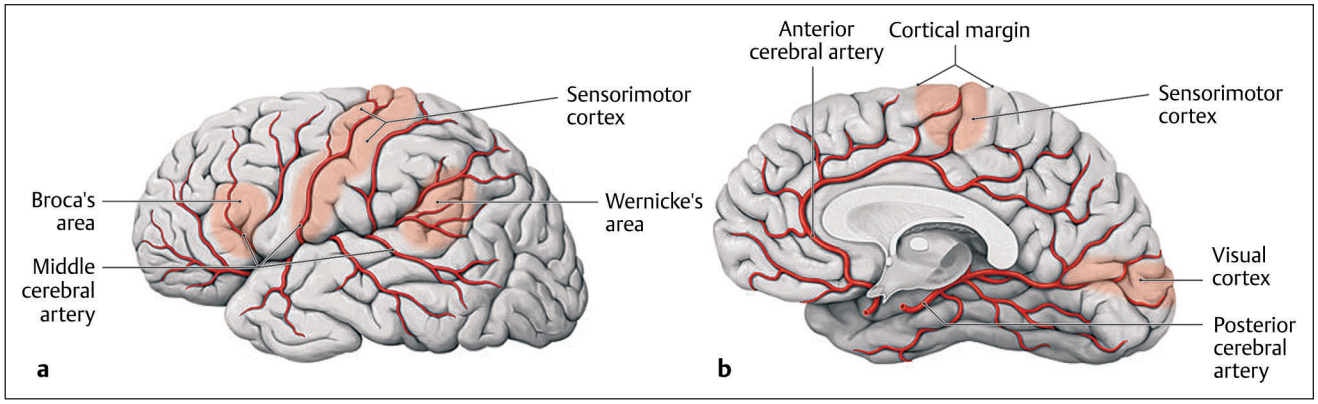


Fig. 1.4 Arteries of the cerebrum and functional centers on the surface of the cerebrum. (**a,b**) Lateral and medial view of the left cerebral hemisphere. Regions supplied by the branches of the middle cerebral artery are shaded orange. The motor and sensory speech centers (Broca's and Wernicke's areas) are supplied by branches of the middle cerebral artery. Therefore, aphasia would suggest an occlusion of the middle cerebral artery. (Reproduced with permission from Schuenke et al. Illustration by Markus Voll.)[4]

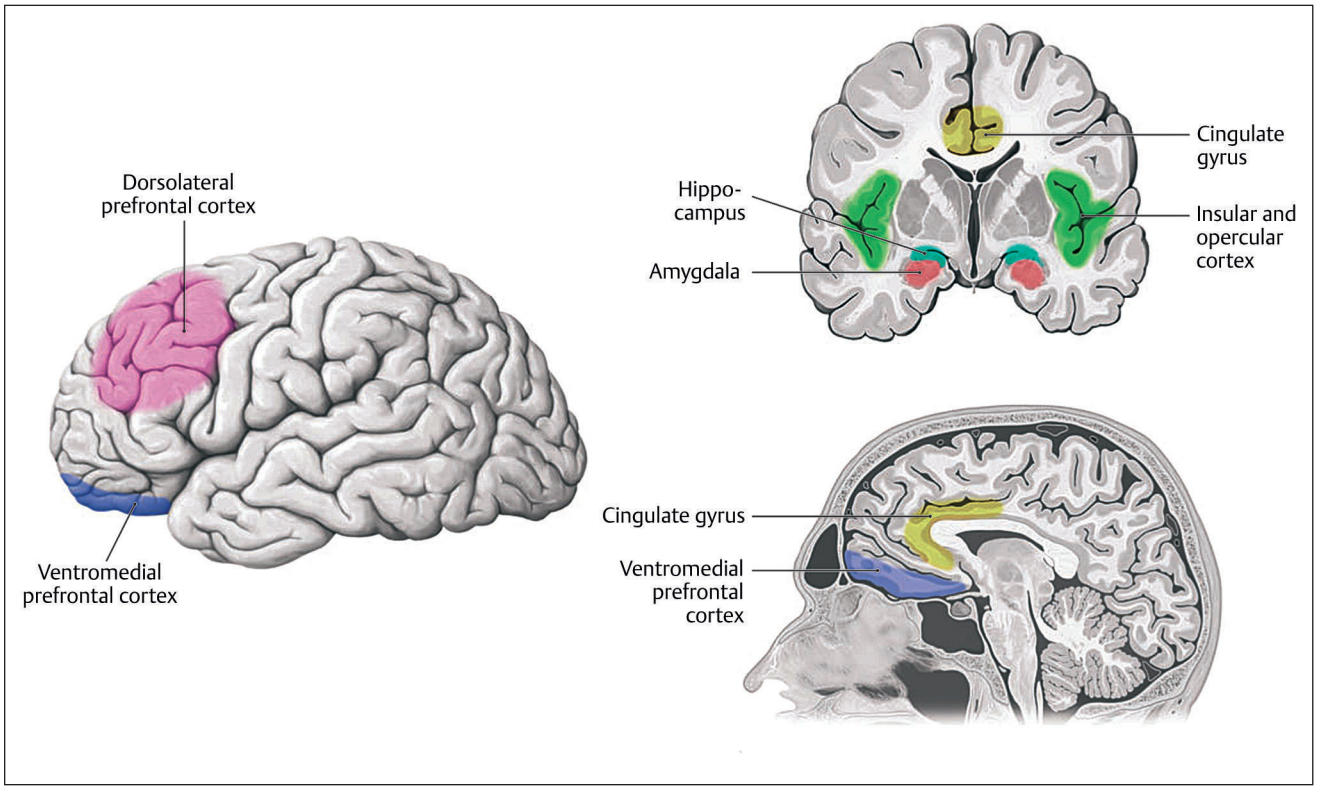


Fig. 1.6 Lateral view of the left hemisphere. (Reproduced with permission from Schuenke et al. Illustration by Markus Voll.)[4]

Summary

This chapter presents an overview of the human nervous system with focus on aspects of the CNS and PNS that are vitally related to human communication and cognitive processes. The chapter is not meant to be a detailed consideration of the nervous system, as particulars can be found in other sources such as LaPointe's *Atlas of Neuroanatomy for Communication Science and Disorders*.[5] It should, however, serve as a basis for a better understanding of the neurogenic disorders presented in subsequent chapters of this book.

Chapter Review

- What makes us human? What separates us from the gibbons and lizards? Make a list of your ideas.
- Research what human behaviors might be included in the term executive function.
- What are the primary divisions of the human nervous system?
- If you had a lesion (damage) to the left middle cerebral artery, what functions would you likely lose?

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2 Humanistic Basics: Accommodation, Adjustment, and *Aristos*

Leonard L. LaPointe

I heard them talking at my bedside. They didn't think I could understand. They were talking about putting me in a nursing home. I couldn't enter the conversation. Powerless.

I thought they had cut my tongue out. I had gone in for surgery on my wisdom teeth; they put me under general anesthesia; and I developed a stroke during the surgical procedure. When I awoke and struggled to find words, I thought for sure I had lost my tongue. But it was far worse than that, as I was to find out. My tongue was fine but I couldn't come up with the right words or put them in the right order. And then when I tried to write or read, I had the same trouble. And it sounded like everyone was speaking a language I didn't know. Talk about terrifying!

Betty[1]

And I'm lost … I am lost, completely lost, have to get to … somewhere, Omaha I think. The radio is out, or rather for some reason picks up only Bucharest.

Emily Stilson[2]

A lot of the accident victims I've met will need help the rest of their lives. And most of them ran out of insurance long ago … It's been rough. Sometimes I feel like Cha'kwaina, the kachina called One Who Cries. I'll keep trying though. I can still create beauty from silver. I won't lose my spirit. We have a rich tradition of persistence. Maybe by the Hawk Moon I'll be doing better.

Bennett[1]

I cannot read a road map … I was good at maps and things … now I couldn't even find my way home … Also, I can't play chess worth a tinker's damn anymore. I can't seem to figure out the moves or the strategies … I guess chess is out. I used to play like a Russian. Now I play like a cushion … Can you help me with any of this?

Russell[1]

I'm more happy and content than I ever thought I could be. Life with Carl is different, but just as fulfilling. He now makes the same bed that I used to have to help him into not so long ago … I never tried to be strong before … I didn't have to.

Eileen Wilson[3]

So ironic that rehab and chronic conditions are not well covered in America. In Sweden or Australia we'd be taken care of. Here, we're at the mercy of a broken healthcare system. How much is a word worth, anyway? How much is the power of speech worth?

The fat cat HMO CEOs need to have their regular meetings at the St. Regis and in the Bahamas so they can discuss how to save money by cutting physical therapy and speech therapy. I wish I were in Sweden.

Nobody will believe this, but in some ways I may be better off after the stroke than before. My life now has meaning. I guess that's what Viktor Frankl meant in his book.

Name withheld by request

Introduction

These are the voices of survivors and those close to them. In the following chapters, we will learn about a daunting array of communication and cognitive disorders that arise from a shattered or compromised nervous system: aphasia, in all its variants; right hemisphere syndrome; traumatic brain injury; and the language of dementia. These are described by the experts in our text to help us understand the nature and characteristics of these disorders, learn about their pathophysiology, and discover some of the clinical hurdles of their assessment and treatment. This chapter is an attempt to humanize these disorders and conditions. They happen to people and it is good to keep that in mind constantly. Although it is comforting to learn that some neuropathologies seem to be declining in incidence (such as the annual incidence of stroke in industrialized nations), it is equally sobering to learn that *survival* has increased comparably, with a net result of the existence of more disabled stroke survivors and their families who require support and assistance. Not all who suffer these pathologies of the nervous system retain chronic residuals of the damage. Some recover completely (perhaps as many as 30% of stroke survivors, for example), with no trace of impairment and resume their lives in much the same fashion as before.

In 2010, the carotid artery in Sean Maloney's neck froze, paralyzing much of his right side and damaging a portion of his brain used to produce speech. Brain scans taken by Dr. Amit Etkin of the Stanford University School of Medicine reveal vacant holes on the left side where the stroke hit. Months later new scans showed new activity on the right side of the brain as Maloney relearned how to speak (**Fig. 2.1**).

On the car ride home from the hospital after suffering a stroke in February 2010, Intel executive Sean Maloney insisted that his wife take him to his boat. With little use of his right arm, he could only row in circles, but he was nonetheless determined to prove wrong his doctor's prognosis that he would never row his scull again. The same drive that got him back into his boat enabled him to relearn how to speak and ultimately return to work to become chairman of Intel China, leading the company's largest market.

For the majority, however, recovery is less than complete. This chapter is a reminder that these devastating disorders happen to people: to our parents, to our children; to our immigrant grandfather; to our neighbor, the railroad brakeman who tended his tomatoes; to our seventh grade teacher who taught us *The Courtship of Miles Standish*; and to us.

Viktor Frankl, whose words were remembered by a survivor with aphasia, is as evocative today as when he formulated his message on life's meaning based on his experiences in a concentration camp. In fact, for some, aphasia and imposed silence are not unlike a concentration camp in many respects. Frankl's book *Man's Search for Meaning* has inspired millions.[4] According to a survey conducted by the Library of Congress, *Man's Search for Meaning* has a secure place on a list of the 10 most influential books. At the time of the author's death in 1997, the book had sold 10 million copies in 24 languages. Frankl's multilayered message is that meaning in life is found in every moment of living; life must never cease to have meaning, even in suffering and death, and even with aphasia. If we can detect or discover meaning, and if we can infuse our rehabilitation with it, we can live life fully despite handicaps, roadblocks, or tainted

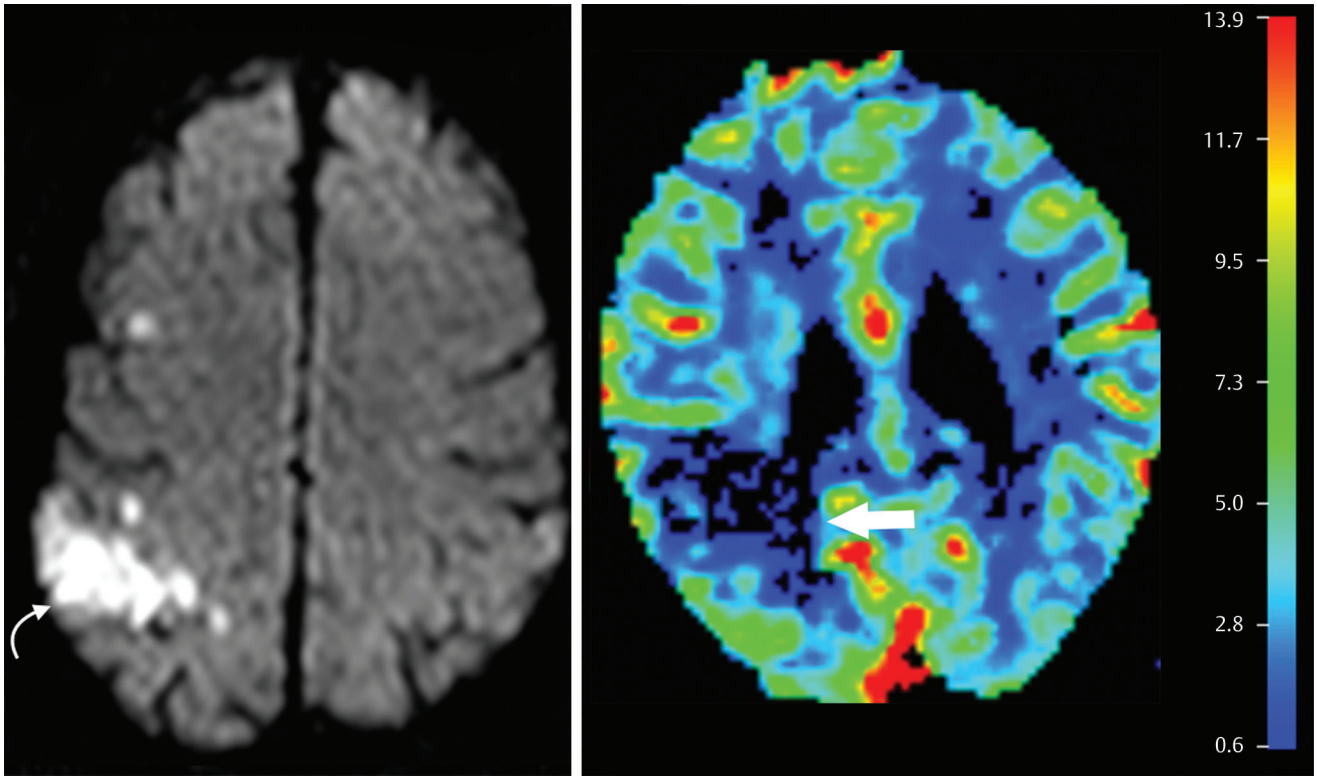

Fig. 2.1 Brain scan of stroke. (Reproduced with permission from Leite C, Castillo M. Diffusion Weighted and Diffusion Tensor Imaging. New York, NY: Thieme Publishers; 2016.)

Fig. 2.2 *The Wreck* (from painting by Aivazovsky).

circumstances. Frankl's book is important reading, even in these contemporary times of Twitter, Facebook, Kindle, and electronic omni-connectivity. Our therapy and the road to recovery should strive to infuse significance into the process. Many times, as Frankl so powerfully pointed out, direction and fulfillment is based on discovery of meaning. One path to meaning in life is by recognizing others, getting outside ourselves for a bit, and doing good works. This, as Frankl might say, could be a remarkable and healing theme for some of our activities and tasks of rehabilitation. When we are no longer able to change a situation, we are challenged to change ourselves. This is the crux of *Aristos*, as we will see, and the golden nugget from Frankl that we can use during our daily grappling with healing against pig-headed odds.

The Wreck

Most of the individuals who have acquired one of the disorders described in this volume will have a life forever refashioned. The challenge then becomes not only how to lessen the effects of the disability, but also how to achieve accommodation and a degree of adjustment to a transformed existence. How does one accept a radically altered life? How does a family adjust to a burden that may be 10-fold on Friday from what it was last Monday? Is it possible to accept and deal with chronicity and a prodigiously changed existence? These questions flirt with the darkly philosophical. Can relative happiness be attained or regained? Is life worth living? Is there precedent for dealing with the illness experience and coping with chronicity? Hundreds of dead philosophers have grappled with these questions. Those of us who believe in the tenets of the rehabilitative process must embrace and champion the affirmative. Although the magnitude of the struggle is certain, it is possible to make gains and to achieve balance. Internal factors as well as resources in the family, community, and society influence the struggle to cope. They also play a major role in the outcome. These elements shape the theme of this chapter. Coping with chronicity is a topic largely missing from many sources. We will explore some of the psychosocial factors that accompany these disorders as well as the attitudes and strategies that allow people who have been dealt a bad break to accommodate, to adjust, and perhaps to accept drastically altered circumstances while retaining a worthwhile quality of life. Frankl's concept of finding meaning may be an understudy in this great drama, and it may be time for the understudy to emerge into a more prominent role (**Fig. 2.2**).

As the aging Ella Mae Cheeks Johnson implied in **Box 2.1**, everyone has difficulties, but it is not necessary to amplify.

Box 2.1 Pearl

"I told Ella Mae Cheeks Johnson, then age 105 that she was the only person over 80 who I'd ever met who never referred to her physical infirmities or health problems. To which she replied, "I have my difficulties; I do not rejoice in them."

—Patricia Mulcahy, *It Is Well with My Soul: The Extraordinary Life of a 106-Year-Old Woman*

Impairment, Disability, Handicap, Activities, Participation

The social context of neurogenic communication disorders should not be divorced from the clinical aspects, but frequently it is. Others are adamant that the social context, especially the family, must be an integral part of the condition. Some experts go so far as to define the disorders within the context of how they affect families. Wahrborg defines aphasia as a disruption of

family life.[5] Earlier, McKenzie Buck, a speech-language pathologist who suffered a stroke, recovered sufficiently to write an insightful treatise on aphasia, in which he viewed the aphasia as a "family disease."[6] Others, such as Sarno,[7] Code and Muller,[8] and Pandolfo,[9] have written insightfully about psychosocial concomitants that accompany aphasia. Sarno has advocated that it is an oversimplification to view aphasia simply as a "neurological problem," a "linguistic problem," or a "language pathology." To do so is to lose much of the broader perspective of the impact of the condition.

The World Health Organization (WHO) has generated a classification schema that has drawn important distinctions among such terms as *impairment* (the pathology itself), *disability* (the consequences or impact on everyday personal, social, and vocational life), and *handicap* (the value that the individual, family, or community places on the disability, and the degree to which the individual is disadvantaged). Disability and handicap can vary greatly across relatively similar levels of impairment. Furthermore, the levels of disability and handicap can vary a great deal across people, depending on differences in the resources, values, and attitudes of the individual and family. These variants necessitate prudent identification and assessment if suitable plans of intervention are to be formulated. The WHO has refined its definition of health. In contemporary understanding, health is not only the absence of infirmity and disease but also a state of physical, mental, and social well-being. Newer conceptualizations of well-being also incorporate concepts of an individual's activities and participation. The WHO has proposed international classifications of functioning (ICF), and current updates can be accessed at the WHO Update Platform: https://extranet.who.int/icfrevision/nr/loginICF.aspx.[10]

The fabric of the perceived disability and handicap suffered by individuals and families with neurogenic disorders is intertwined with a web of psychosocial elements. With the exception of some slowly progressive dementias, the other disorders covered in this book make their appearance suddenly. Life is explosively transformed. It will never be the same. Thrust upon a person and family are mutations in life role and employment, financial stability, living environment, sexuality, leisure lifestyle, social integration, independence, and control. All of this may be wrapped in modified mood or personality, the perception of stigma, emotional turmoil, and alteration of the very core of identity and self-esteem. Ewing and Pfalzgraf have humanized and personalized some of these psychosocial barriers, as well as the struggle and courage involved in the rehabilitative process, by tracing the journey of six men and women who survived stroke.[3] Ed, Betty, Ted, Paula, Tom, and Carl weave important lessons of their emotional reactions, changed relationships, resumption of activities, new responsibilities, and coping strategies. Their stories can be fitted alongside those of Tan, Betty Penner, Jimmy Parker, Ken Kimber, Mr. Babcock, Uncle Jimmy, Dr. Anderson, Martha, Mr. Kopit, Emily the Wingwalker, Tom Reyhons, Kelly Hocker, and the thousands of others who trod the path. All had to endure ponderous life adjustments and psychosocial hurdles. We learn from each of them.

In the meantime, we are in the context of a time of great transition and technologic revolution in rehabilitation. These are the days of neuroplasticity, dendritic sprouting, facilitative connectivity, and other 21st-century approaches to rehabilitation that have only been dreamed of in the past. Evidence for a long-term alteration in brain function associated with a therapy-induced recovery in neurologic populations increasingly appears in the literature. For instance, constraint-induced movement therapy (CIT) can significantly change cortical excitability measured by transcranial magnetic stimulation in both affected and unaffected hemispheres.[11] What was regarded as fringe science 35 years ago is currently at the cutting edge of rehabilitative neuroscience. Neuroscientists now know that with the right training the brain can reshape itself to work around dead and damaged areas, often with clear-cut benefits. As Norman Doidge explains in *The Brain that Changes Itself*, the doors are opening to personalized and neurophysiologically based recovery from brain injury.[12] In the early days of neuroscience, we had a preoccupation, some would say obsession, with the question of "*Lesion, lesion, where is the lesion?*" A great deal of effort, even among rehabilitative neuroscientists, has been directed toward identifying the functions of anatomically distinct regions of the brain. From these experiments emerged a body of evidence that supported what Doidge calls the *localizationist paradigm*: neuronal function mapped to specific brain locations—the boxes-and-arrows view of brain structure and function. Models of parallel-distributed neural processing have shown that the oversimplified, boxes-and-arrows, Radio Shack model of complex neural functioning is fine for some functions and behaviors, but remarkably reductionistic for others. Doidge has strong objections to the permanence of the localizationist paradigm for higher and not-so-higher cortical functions, arguing instead that the brain is highly plastic—so much so that regions that normally carry out one function can learn through concentrated experience to carry out another. The implications of this remarkable degree of brain cell plasticity are wide ranging, and Doidge does an admirable job in surveying the landscape through the use of both case studies and clever experimental results.

So, the future looks somewhat brighter, and technology and advances in neuroscience will continue to provide rays of hope, but there is still a long way to go and in the end, even if we can implant and grow stem cells or somehow stimulate the development of new neuroarchitecture, the whole of the human being will still be the essential domain of rehabilitation.

The Illness Experience

What is it like to be ill? What is it like to suffer life-altering disability and handicap? Our ability to imagine or empathize with those who are chronically disabled is limited. A traditional approach to understanding the illness experience has emanated from the education and focus of health care professionals on the *disease*. Little emphasis has been placed on the perspective of the individual with the disease. As Susan Sontag offered, as long ago as 1988, most of us are destined to live in two worlds, the kingdom of the well and the kingdom of the sick. Illness is the night side of life, a more arduous citizenship. Everyone who is born holds dual citizenship, in the kingdom of the well and in the kingdom of the sick. Sontag's words in her influential book *Illness as Metaphor* echo true; and finally some ethnographic and qualitative research is illuminating these two kingdoms.[13] As Sontag suggests, we all prefer to use only the good passport; sooner or later each of us is forced, at least for a spell, to identify

ourselves as people of that other place. Only a stellar author, a master of words like Sontag, could so capture the meaning of the illness experience. Perhaps, this work by Sontag set the stage for qualitative researchers to try to reach inside the illness experience and allow us to better understand it. Trends in some of the health care literature appear to be developing along paths that try to understand the illness experience and try to develop research directions that incorporate qualitative as well as quantitative research strategies.

Morse and Johnson[14] and, more recently, Larsen[15] have written insightfully about the illness experience. They have proposed some models for understanding and studying it and have reported some important qualitative research across several different types of chronic illnesses or conditions. They illuminate the role of such strategies as those used in ethnographic research, with careful attention to in-depth interview methods, participant observation, and what they refer to as "symbolic interactionism," or the experiential aspects of human behaviors. Morse and Johnson explain "grounded theory" as the essence of conducting qualitative research on the illness experience and explain how health care researchers can transcribe, analyze, and interpret informant interviews to reveal such themes as coping strategies, social support, and participation in treatment decisions.[14] They catalogue a rich array of descriptions of the illness experience and make no apologies for their intensity or poignancy. The strain of trying to endure, the shock of institutionalization, and the suffering and uncertainty of the illness experience were clearly evident in the reflections of those who were interviewed:

> *When you're thrown into a pool, and you can't swim, and you don't have a life preserver, that's the way I felt for a long time. In other words, struggling. It was hard to get up in the morning knowing that you were going to have another one of those days that was unpredictable. The pain of watching somebody that you love going through pain, going through agony; that is the hardest part. That is the hardest part of the whole thing to deal with that. That was my hardest part—it wasn't the caring or the cleaning up or the helping her to the washroom or getting the pills.*[14]

An update on the Illness Constellation Model is provided by Davis, who uses the WHO ICF as a framework for her book *Rehabilitation: The Use of Theories and Models in Practice*.[16] The book explains a variety of models of rehabilitation and explains differences in theories of rehabilitation across professions. The Morse and Johnson Illness Constellation Model remains viable and usable even some 25 years after its introduction and has spawned increasing interest on the illness experience and coping with chronicity.

Stages

Arising from these encounters is a broader understanding of the illness experience. Morse and Johnson propose a more comprehensive view of illness, which they call the Illness Constellation Model, and it is contrasted with traditional views.[14] In the Illness Constellation Model, the experience is viewed not only from the perspective of the physical signs and symptoms, but also from the perspective of the adaptation and coping behaviors exhibited by the ill person within the social context of the effects of the events on family and friends.

Table 2.1 presents an adaptation of Morse and Johnson's conceptualization of the stages that the individual and significant others presumably traverse through the evolution of an illness.[14] Morse and Johnson have contributed mightily to the germination of this new perspective in understanding the illness experience.

These stages arose from the common themes expressed by individuals, families, and friends who were interviewed in depth throughout the course of their illnesses or conditions. Surely, the insights gained from this type of qualitative research can aid us in our task of knowing what to expect and how emotions and behaviors evolve during the course of having to deal with neurogenic conditions. Although each stage may not be represented with the same duration or in precisely the same way across the different neurogenic conditions we encounter (e.g., in stroke, the onset is sudden and the stage of uncertainty may be compressed into a matter of seconds or minutes), the fundamental aspects of these stages may have considerable relevance to the conditions with which we deal. No doubt considerable individual variation occurs across individuals and families as well, but an understanding of the major themes that may be represented during the course of illness can be useful to us in predicting and dealing with reactions of those we see.

Stage 1 of the illness experience for many conditions starts with the individual's beginning to suspect that something is wrong. Family and friends may be drawn into this process of suspicion, and doubts begin to creep into the thinking of everyone that perhaps something is amiss. If the signs or symptoms are

Table 2.1 Stages of the illness experience

Stage	Self	Others
Stage 1		
Uncertainty	Suspecting Reading the body Being overwhelmed	Suspecting Monitoring Being overwhelmed
Stage 2		
Disruption	Relinquishing control Distracting oneself	Accepting responsibility Being vigilant
Stage 3		
Regaining self	Making sense Preserving self Renegotiating roles Setting goals Seeking reassurance	Committing to the struggle Buffering Renegotiating roles Monitoring activities Supporting
Stage 4		
Regaining wellness	Taking charge Attaining mastery Seeking closure	Relinquishing control Making it through Seeking closure

insidious, questions begin to crop up about each subtle change in the body that may be perceived. Family and friends begin to monitor behaviors and tune in with more attention focused on any type of action or sign that may signify deviance from the norm. The ill person listens to his or her body with concern and tries to identify triggers that mark any change in signs or symptoms. Finally, the course of the condition may proceed to a point where the ill person and others become overwhelmed. This is the point where in many conditions "cure shopping" and multiple opinions are sought to add support or validation to the realization that something truly is seriously wrong.

In Stage 2, sick people realize that they have no choice. Control must be relinquished, and choices become a medical prerogative. This is the stage where an individual is transformed into a "case" or a "patient." The loss of control and a resolution to turn all decision-making over to professionals are felt by the individual and the family. The sick person feels that he or she is merely an object who is undergoing a process of having things done to him or her:

> *I can't do anything about it now. I just have to wait for these tests. They're doing some tests. I just get wheeled here and wheeled there. I just have to trust them, I guess. I've had all these doctors and people talking to me. Some are nice. Some could use a few lessons in talking to people who don't know a lot about medical things. It's in their hands now, I guess.*[14]

In Stage 2, the family and friends feel compelled to accept responsibility. They get on with the tasks of notifying other relatives and friends and in fetching basic items for the sick person even though they may be consumed with anxiety and fear. Helping turns into a coping strategy at this point. Responsibilities are clarified and assumed:

> *I've got to be strong. I don't want the kids to think we're all falling apart. Now is my chance to do something. He did so much for me for 30 years. I wouldn't want to be anywhere else right now. He needs me. There's an old song that goes "He holds the lantern while his mother chops the wood." Wait a minute. I don't know if that relates to this. What the hell is wrong with me? Well, anyway I'm here, and I've got to be strong and I'll help with this.*[14]

Stage 3 is the struggle to regain self. The process may not become actualized or complete during this phase, but now people begin to try to make sense of everything. They mull and ponder and ask questions about the details of how they behaved during the acute phase. They go over the events in their minds and examine in excruciating detail the events leading up to the illness to try to discover the "real" cause. They ask, "Why me?" and finally, after ruminating, they begin to accept the fact that their lives may be inexorably altered. This is where uncertainties of identity, self-image, and self-esteem begin to be formulated. This is where ill people struggle to preserve a sense of self. Responsibilities and the identity associated with vocation, profession, or work are stripped from most ill people at this point. They feel that their accomplishments and the cocoon of things that they have done or are interested in are unrecognized or ignored:

> *I was a composer and conductor, for heaven's sake. I've conducted in most of the great music venues of the world. They don't know any of that. They treat me like an eccentric old man. They use all these medical terms. I'd like to get them on my turf. I bet they don't know Berlioz from their bum. This whole experience is "Fantastique." Hector would understand. He was a little crazy, too. They sure don't know who I am. Maybe that's because I'm not anymore.*[14]

Arthur Kopit, the playwright who crafted many successful and critically acclaimed plays and scripts, is the author of *Wings*, a powerful play about aphasia.[2] Once, when this writer and Dr. Jay Rosenbek were lecturing at a conference in Dallas where Kopit was the headline speaker, we had the opportunity to spend a long and intriguing night together, discussing and exploring the effect of aphasia on identity. Kopit's insights were astonishing. He had done much of his research in preparation for writing *Wings* at the Rusk Rehabilitation Institute. The play was motivated by his tormented experience with his father's severe aphasia. Kopit observed that his father's identity was seriously torqued or twisted by the experience of aphasia. He related that his father would ask him, in convoluted aphasic fashion:

> *Arthur, when am I going to be me again? This is not what I had in mind for "me." After some more rehab will I be me again?*

These are trenchant observations about the effect of aphasia on identity and self and about the struggle people engage in at this stage of the illness experience. The role of identity metamorphosis in neurologic conditions and chronic illness has been a relatively neglected area of attention and clinical research effort. Shadden has written poignantly about aphasia as identity theft, and she writes from both an academic and family constellation perspective and motivation.[17] Our colleagues in Australia also have enlightened what we know about psychosocial aspects of aphasia, including the impact of aphasia on self and identity.[18]

Márquez recognizes in **Box 2.2** that life is full of many unpredictable changes and a new self is sometimes required.

Box 2.2 Pearl

"He allowed himself to be swayed by his conviction that human beings are not born once and for all on the day their mothers give birth to them, but that life obliges them over and over again to give birth to themselves."

—Gabriel García Márquez, *Love in the Time of Cholera*

In Stage 3, the family and friends now solidify their commitment to the struggle. They realize that they must get organized so that they can be useful. At this stage, the family also begins to assume a buffering role and engage in activities that protect the sick person. They hover and attempt to shield the person from concern and worry and the mundane responsibilities of business at home. Sometimes the buffering becomes excessive, and this leads to exacerbation of the ill person's sense of loss of identity and responsibility.

This is also a stage in which ill persons and their families begin to *renegotiate roles* as recovery progresses. Ill people now start to regain some control over home and work by giving instructions, issuing orders, and making decisions even while still hospitalized. Beginning to exert some assertiveness

over their outside lives aids them in beginning to feel like who they were rather than like anonymous "patients." Morse and Johnson point out that this is the stage where the ill person attempts to feel useful once again, and even though the roles may have drastically shifted and they have become dependent, it is an important time for the family members to realize that they can now relinquish decision-making roles and other tasks that can once again be assumed by the ill person.[14] Morse and Johnson use the metaphor of dance and comment that in this stage the sick person and the well person must learn to lead, follow, change direction, and accept and relinquish a variety of tasks. This is also the important stage where the ill person begins to *set goals*. Planning is an important marker for all sorts of recovery processes and certainly no different in the stages of dealing with neurogenic conditions. Accomplishment of small goals is an important component of this phase, although a risk is that some individuals feel that an all too frequently recurring situation is that a person with aphasia is given a goal, struggles to achieve the goal, and finally achieves it, only to have the outcome suggest another goal that the person feels helpless to achieve. Professionals and family can spend more time and exposure on the successfully achieved objectives at this stage, even though this may slow progression to the next objective. A more crystallized appreciation of progress along with enhanced motivation to continue can be the result of this approach. The family usually engages in *monitoring activities* at this time as well. They express concern that the ill person "not overdo it," and they chaperone, hover, and remain close, ready to intervene whenever they perceive that they are needed.

In this stage of regaining self, the ill person needs much reassurance and experiences much trial-and-error behavior on many activities of daily living (ADLs). Lifting heavy objects, making a telephone call, dealing with the checkbook, and resuming sexual activity may be approached with tentativeness:

> *I'm afraid to do too much. Should I or shouldn't I do it? I feel I need to ask someone if I can do these things. Can I do this myself again? Well, let's give it a try. If I'm going to get back driving, I guess I got to start somewhere. The longest journey begins with the smallest step. Is that right?*[14]

The family members have an important role at this stage, and Morse and Johnson note that they usually recognize the uncertainty of the ill person and engage in supporting behavior. They make lifestyle changes, try to second guess needs, read or try to learn about the illness, try not to notice or comment on devastating effects of the condition, and try to remain positive (even though their real feelings may be thinly veiled). This is the stage where family members shower praise and encouragement and try to generate a sense of hope. Professionals can foster the development of this phase in families by their own recognition and support of positive family trends.

In Stage 4, regaining wellness, the ill person enters into the rehabilitative phase of a more fully regained sense of self. This stage may or may not necessarily coincide with hospital discharge. Now is the time that rehabilitation continues and, depending on the severity of the residual impairment and disability, the ill person begins to regain more control over his or her own life, dependency may be changed, and former relationships may be assumed. Of course, there may be very real limits to how much premorbid normalcy is achieved. Roles and responsibilities may be permanently altered. The ill person wants to *take charge* and may feel that he or she must demonstrate his or her regained health, altered roles, or regained competencies. Proving him- or herself to others becomes a frequent process during this stage. Families and friends may *relinquish control* at this stage. Although some degree of hovering and monitoring may be maintained at this point, usually it is covert, as ill persons increase their efforts to demonstrate control over their lives. At this stage, ill people learn to trust regained or new abilities and further define and perhaps even accept their limitations. Confidence grows. Days are marked by promotions to higher levels of premorbid functioning and *attaining mastery*. More sorties into the community are attempted, and gradually the realization that life can go on and in fact be worth living begins to be believed. Comparisons are made either with others or with perceived levels of previous functioning to assure that objectives have been achieved or that progress has been made.

This stage can progress for a long time, and unfortunately is incompletely achieved by many, particularly those who fixate on a goal of nothing less than return to "normal" or premorbid functioning. Families may be guarded at this stage and focus on making it through with the concern that there may be setbacks or recurrences. They express the feeling that they do not want to become too enthusiastic about progress "in case something happens." Acceptance of altered lifestyle and making the best of a bad situation is as difficult for some family members at this stage as it is for the ill person.

The ultimate place to be in this final stage, and a major task of ill persons and their families, is that of seeking closure. Getting on with life is a resolution expressed by those who achieve this closure. Putting the nightmare behind them, continuing to work on rehabilitation or prevention, accepting restrictions, changes, and limitations, and planning for the future are healthy markers that signal closure. Some even generate tasks of writing about their experiences, finding ways to help others, or becoming active with support groups or organizations that deal with their condition or illness:

> *I'm real active with the aphasia newsletter. My writing is slow and not as easy as it once was, but I feel I'm helping others by getting this word out. I also enjoy talking to your class. Teaching those young people what it is like to have aphasia is an important lesson, I think. They always have such good questions. But they need to have the view from under the skin of one. Plus the brownies are always good.*[19]

Adjustments are made. Searches for meaning are launched. New duties, hobbies, activities are nurtured:

> *This painting is all new to me, even though I have to do it with only one hand. Let me show you some of my work. This is a watercolor that we are going to use for this year's Christmas cards. I know it's not perfect. But I get a lot of pleasure from it now. You like this one of the Model A. I had an old Model A once. Did you ever hear of a rumble seat? Boy, a lot could get done in the rumble seat. I wonder if that's why they named it that. Whatever. My painting means a lot to me now. I probably never would have done this if I hadn't had a stroke.*[19]

Sometimes the search for meaning and adjustment is fostered by renewed altruism, less egocentricity, and learning how to become more other-oriented:

I never did much volunteering before. Now I realize that we're not alone on this planet, and there are a lot of people who need help out there. It's nice to just help others. I've been real active in the Special Olympics the last two years. I go there as a volunteer and help out with timing some of the races and stuff. I tell you these kids do a real nice job. It's all relative, I guess. They're not the fastest in the world, but boy that accomplishment of finishing an event is just as meaningful as the big-time Olympics. I guess there's nothing quite like having one of those kids jump into my arms when they finish an event. I'm glad I can help with this. It makes me realize that there's more to life than just sitting at home and stewing over my own problems.[19]

Simmons-Mackie has referred to and conducted aspects of ethnographic research methods that can prove fruitful in aphasia. Damico et al[20] and Elman[21] have advocated methodologic alternatives and presented examples of ethnographic qualitative research as it can be applied to aphasia.

Certainly, multimethod research is appropriate to questions about coping, accommodation, and adjustment to these conditions. Although quantitative research has an honored part of our tradition, a few things can get lost in sterile numbers. Emotions, beliefs, and strategies of wellness need to be explored with equal vigor.

Being a Patient

One of the most pervasive consumer complaints about health care delivery is the perception that "patients" are dehumanized, institutionalized, robotized, and stripped of identity and individualism when control is wrenched from them as they are cloaked in the cape of "patient."[22] It is so different on the other side of the fence. The depersonalization of the process is captured remarkably by the late and dearly missed gifted writer-neurologist Oliver Sacks as he relates his own feelings of foreboding and alienation upon becoming a patient:

One's own clothes are replaced by an anonymous white nightgown; one's wrist is clasped by an identification bracelet with a number. One becomes subject to institutional rules and regulations. One is no longer a free agent; one no longer has rights; one is no longer in the world-at-large. It is strictly analogous to becoming a prisoner, and humiliatingly reminiscent of one's first day at school. One is no longer a person—one is now an inmate. One understands that this is protective, but it is quite dreadful too. And I was seized, overwhelmed, by this dread, this elemental sense and dread of degradation, throughout the dragged-out formalities of admission, until—suddenly, wonderfully—humanity broke in, in the first lovely moment I was addressed as myself, and not merely as an "admission" or "thing."[23]

Health care professionals must guard against the snare of routineness in our procedures and daily interactions despite the fact that we may have administered the same test or treatment paradigm dozens of times. LaPointe in "On Being a Patient" emphasizes affinity with what may be an individual's first experience with a very foreign ordeal.[22] Procedures need to be explained. Rationales need to be revealed and clarified. Questions need to be answered. Fears need to be calmed. Dignity and the uniqueness of the individual need to be preserved. We are reminded of this compellingly when we become patients ourselves and suddenly are thrust into a very different role in the health care environment. I have been aware of this myself on several occasions. "So you're the cysto," announced a nurse prior to wheeling me for a medical procedure. I wanted to respond, "I'm a bit more than the 'cysto.' I'm a person who is scared at the moment and worried about my future and a bit more complex than a shorthand name for a medical procedure." The illness experience can be endured better with the assistance of empathic, compassionate professionals who are in tune with the most efficient ways to nurture adjustment and accommodation.

Mood Alterations and Depression

Of course he's depressed. He just had a stroke. It's perfectly natural to go through a period of mood alteration after a major medical crisis, so we can expect some depression. Be patient. He'll snap out of it.

This is not an unfamiliar reaction relayed by families of people with neurologic conditions when they report their perceptions of depression to health care professionals. For years, mood alteration and depression have been conceived as natural accompaniments to most major medical illnesses. The unfortunate consequence of this interpretation is that treatable conditions may then be unattended. The familiar parallel that we have heard so many times is "He will outgrow it" when discussing a child's developmental aberration that may need intervention. Some mood alterations do run a natural course and become self-limiting, but many do not. Increasingly, altered emotional state, mood disorders, and overt clinical depression are being recognized as all too frequent components of certain neurogenic conditions. In stroke, for example, prospective studies of depression have revealed that as many as 46% of persons with acute stroke can be expected to suffer either major or minor depressive symptoms.[24] Kouwenhoven et al have expanded and updated what we know about poststroke depression and contemporary ideas on treatment.[25] They concluded depressive symptoms are common in the acute phase after stroke and associated with persistency of depression and mortality after 12 months. A gold standard for the measurement of depressive symptoms in relation to stroke is still missing especially in the presence of aphasia but careful observation from both clinicians and caregivers can tip us off to its existence. Depression in stroke should not go untreated, as it has for years.

Contemporary approaches to depression that accompany stroke and a variety of other neurologic conditions are more prone to evaluate and treat the disorder instead of waiting for it to fade away. Particularly in aphasia, where people are at a genuine disadvantage in expressing their mood alteration because of communication barriers, it is becoming acknowledged that undue suffering can result from allowing people to wallow in their depression with no attempt at alleviation. The work of Robinson and colleagues at Johns Hopkins University and elsewhere has shed new light on depression and stroke, and perhaps the greatest contribution of this enlightenment has been

the realization that depression can be treated successfully instead of just allowing it to run its course.[24] The neurologic, physiologic, diagnostic, and treatment implications of poststroke depression have been reviewed meticulously by Swindell and Hammons.[26] Robinson and colleagues continue their programmatic research on stroke and depression.[24]

Recent work on depression and stroke from Sweden has confirmed once again that depression is a major concomitant of the disorder.[27] In this study, the frequency of depression was 34% in persons with stroke and 13% in a sample controls. The risk of depression in a large sample of people with stroke was increased in both men and women and in all age groups but not related to the predominant side of hemispheric damage.

Depression following stroke can be categorized as either major or minor. Either variety can disrupt life and well-being, although at differing levels of severity. A major depressive episode is characterized by prominent, persistent, dysphoric mood that may include loss of interest or pleasure in nearly all activities or pastimes, including participation in rehabilitation. Appetite and sleep are frequently disturbed, and the depression can be accompanied by psychomotor agitation or retardation, decreased energy, or expressed or implied feelings of worthlessness, self-reproach, guilt, restlessness, social withdrawal, or irritability. Minor depression or *dysthymic* mood alteration usually has some but not all of these features. These characteristics and the framework presented by Swindell and Hammons can provide a useful structure for appreciation and appraisal of depressive episodes that accompany stroke.[26]

Several theories have been advanced to explain the mechanisms underlying depression. Included in these are a neuropharmacologic model, neuroendocrine models, and neuroanatomic models. Robinson and colleagues have highlighted two critical variables affecting poststroke depression, namely location of lesion and time poststroke.[24] Persons who are at greatest risk of becoming depressed are those in the first 2 years after the onset of the stroke and those who have cerebral damage in the anterior portion of the left hemisphere. Although the correlation of site of lesion with the appearance of depression after stroke is somewhat controversial, many studies propose a convincing case for interruption of certain neurochemical distribution pathways related to mood stability by the same left hemisphere lesions that create the language disruption of aphasia. Certainly, other factors affect depression and mood alteration, including premorbid social adjustment, the individual's support system, and the presence or absence of cognitive impairment. Without treatment, the duration of poststroke depression typically lasts 7 to 8 months, but the disorder has been known to reappear more than 10 years after the onset of the stroke.[24] Spencer et al have studied and synthesized the psychosocial outcomes of stroke in their report of a longitudinal study of depression risk.[28] They stress the importance of addressing the depressive symptoms in stroke survivors and their caregivers. They present a well-crafted rationale for considering poststroke depression: (1) depression is a significant barrier to rehabilitation, (2) any person suffering from mental torment unquestionably deserves treatment, (3) the consequences of unrecognized and untreated depression can result in increased use of health care services and longer hospital stays, and (4) depression has the potential to increase morbidity and mortality from suicide. Overarching all of these reasons is the realization that alleviation of depression can dramatically alter quality of life of individuals who suffer it as well as that of caregivers and families. Poststroke depression is a serious disorder that is often neglected. Clinicians can assume an important role on the health care team not only in the identification of mood disorders and in becoming patient advocates for attention to and treatment of depression, but also in the education of caregivers and family members about treatment options. Without adequate appreciation, diagnosis, and treatment of mood disorders that can accompany neurologic disorders, little hope exists for reducing the impact of the reverberations of these complications. The speech-language pathologist has a compelling role in this venture, particularly since they see persons with aphasia frequently and regularly.

Adaptation to Chronic Illness

Only in recent years has much attention been directed toward adaptation to chronic illness. The psychological implications of the illness experience can evolve into the need to deal with a chronic condition that may never go away or be significantly improved. Many neurologic conditions fall into this latter category. That is not to suggest that complete recovery does not occur in some instances, as mentioned earlier, but in far more cases there exist significant residuals that turn out to be a part of a very altered lifestyle. Stroke, for example, is the third leading cause not only of mortality in older persons, but also of chronic long-term disability.[29] In a recent investigation of stroke survivors with aphasia, the results are not comforting. Dalemans et al reported there was considerable variation in the social participation of people with aphasia after stroke.[30] Not surprisingly, they concluded aphasia negatively affects long-term social participation, together with other factors including functional ADL performance, age, and gender. Environmental factors and personal factors were not found to independently contribute to the level of social participation of stroke survivors with aphasia. None of this is surprising but it certainly supports the notions that we should be emphasizing social participation in our treatment paradigms. The figures vary for other causes of neurologic conditions, but for all, the evolution to chronicity is more typical than not. Coping with chronicity should be high on our priorities when we deal with stroke survivors and their families.

Chronicity

Chronicity implies long duration. Concepts associated with chronic medical conditions include "irreversible," "enduring," and sometimes "recurring." That being the rather bleak forecast for most with chronic neurologic conditions, it is surprising that more attention has not been paid in the health care community to strategies designed to help people and families deal with chronicity. Certainly, that is part of the enfolded responsibility in the counseling aspects of the job of the clinicians. As Martz and Livneh point out, adjustments to chronic illness must be made over and over, and these adjustments vary across disease processes, age, sources of social support, social mobility, family environment, and internal characteristics of the chronically ill individual.[31] All chronicity, however, may be marked by any

number of fears, real or imagined. These include, but are not limited to, the following:

- Fear of death.
- Fear of incapacitation.
- Fear of pain.
- Fear of abandonment.
- Fear of spreading the disease or condition to others.

Stages of Chronicity

Gregg et al have amplified these apprehensions and have written as well about the stages of emotional reactions associated with chronicity.[32] As with most stages, such as the well-publicized "stages of grief," recent research has found that certain caveats are necessary when interpreting them. Some people do not experience the stages at all; others experience only some of the stages; and still others experience the stages in a different order. However, certain identifiable emotional patterns or reactions are more likely to emerge at certain stages than at others. Parallels can also be seen in the general reactions to the illness experience described earlier. The stages of emotional reaction reported to be associated with chronic conditions include the following[32]:

- *Shock*: This has been described as a period of "psychic numbness," with muted responses to external stimuli.
- *Realization*: Gradual awareness of the reality of the situation and the seriousness of the consequences begin to seep into the individual. Now, strong feelings of anxiety or depression begin to grow based on the perception that death, permanent loss of function, or unpredictability may characterize the horizon.
- *Denial*: This is a defensive retreat from the situation. The ill person simply refuses to acknowledge or accept the existence of seriousness of the situation. It is important to remember that in certain neurologic conditions, as is well chronicled in our chapter on right hemisphere syndrome, the lesion that causes the condition can also affect those portions of the nervous system that are responsible for appropriate emotional reactions. Denial and misperception are an integral part of the sign and symptom complex in right hemisphere syndrome and may not be a reaction to chronicity. Other conditions, such as diffuse neurologic damage in traumatic brain injury or dementing diseases, also could result in pathologic emotional conditions and reactions. In many cases of chronic illness, however, denial can be a part of the adjustment to the realization of chronicity. Denial, as they say, is not just a river in Egypt.
- *Mourning*: Grieving the loss of full health is a common reaction. With realization and awareness of the meaning of changed functioning may come a period of mourning and depression. This is also a stage that may be characterized by anger, irritability, frustration, uncooperativeness, and all of the other behavioral accompaniments to depression listed earlier. Sleep loss, reduced energy level, listlessness, and little interest in worldly events or socialization also can mark this stage.
- *Adaptation*: This is the stage that has been referred to as the "acceptance" phase. It is a much more positive stage of the whole experience of chronicity and an end goal, but a stage that not necessarily everyone realizes. The difference between successful, positive rehabilitation and less than optimal rehabilitation can be determined largely by the degree to which the chronically ill persons and family penetrate this stage. It is marked by increased ability to work through other emotional reactions to the condition or illness, a realistic acceptance of limitations, and an ability to plan for the future and to look forward to the future. It would not be unusual to observe that individuals with chronic conditions may in fact retreat into earlier stages of emotional response with the appearance of complications or recurrence, but the stage is marked by an attitude of accommodation, participation in rehabilitation, and a genuine appreciation of the positive assets that remain in life.

Although it has been a goal since the dawn of humankind to attain happiness, and many spiritual, philosophical, and pharmacologic strategies have been proposed and practiced to achieve it, a reality of human existence is that reaching a constant state of nirvana, pleasure, joy, exhilaration, bliss, or contentedness is not probable. But on balance more pleasure than pain can be achieved, even in the face of chronic medical conditions, and health care professionals have a responsibility to help ill people and their families on the journey to this state of relative equilibrium. Radcliffe et al have delved into the coping of couples with chronic illness issues.[33] They reported illness narratives have mainly focused on individual patients' accounts, and particularly those of people experiencing the onset of chronic illness in midlife. However, as longevity increases and medical care improves, a growing number of older people are spending their later life with their partner and both experience multifarious experiences. Radcliffe et al examined the shared creation of meanings among older stroke survivors and their spouses and the implications for individual and couple identity.[33] Joint biographical narrative interviews were held with 13 stroke survivors aged 75 to 85 years and their spouses. The analysis examined both narrative content and narrative style. Three main types of couples were identified. The "united couple" described couples who pulled together and emphasized their accommodation of the stroke and normality as a couple, despite often considerable disability, and was strongly buttressed by collaborative interaction in interviews. Caring relationships were distinguished as "positive," and were characteristic of self-reliant couples who took satisfaction in how they managed. Finally, "frustrated" couples emphasized the difficulties of caring and hardships experienced and were characterized by a conflict in narrative style. These authors shed light on how stroke survivors and their caregivers manage chronic illness; some well, some not so well.

Research in Adaptation

Individuals

Although there are not a lot of studies in the literature to guide health care professionals along the rehabilitative path to nurturing, fostering, or achieving adaptation, there is increasing

attention to it these days. Much of what is being studied about adaptation to chronicity appears to be grounded in *systems research*. In fact, the Contingency Model of Long-Term Care, as espoused by Hymovich and Hagopian, includes a variety of systems as a major component of the model.[34] The systems encompassed in the individual person as well as the family, and its subsystems form the core of the model. The more external suprasystems of community and society play an important role also. Theories of adaptation to chronicity can be developed and traced through all of the systems that revolve around and within individual, family, community, culture, and society. As is pointed out by Hymovich and Hagopian, all systems are important because they interact with and are mutually influenced by each other.[34] Some of these systems have received far more attention than others in the research on adaptation.

A complex set of variables exists in each individual that can illuminate differences across persons in capacity to adapt. Age, physiologic responses, cognitive abilities, education level, and gender have all been associated with these differences. Perhaps, the most intriguing set of variables are those that have been rather nebulously labeled as *personality characteristics*. Among the personality characteristics thought to have something to do with the ability to adapt to chronic conditions are such factors as temperament, hardiness, introspection, dispositional optimism, and locus of control.

Temperament

Differences in behavioral styles have been long observed and relegated to the personality constellation of temperament. Negative temperament characteristics have been studied in children and adults and have been associated with the behaviors of high activity level, low adaptability, withdrawal from new stimuli, distractibility, unpleasant mood, lack of persistence, and irregular or unpredictable behavioral style. More positive features of temperament generally are just the opposite of the traits cited.

Hardiness

Resistance to stress is the primary feature that has been studied in determining individual differences in hardiness. This factor has been coined and studied by Kobasa in people who do not become ill when faced with serious stressors.[35] The concept refers to a set of attitudes toward challenge, commitment, and control. All of these subfactors can mediate one's reaction to stress. Hardy people view change as a challenge; are deeply involved and committed to various belief systems, institutions, or other areas of life; and strongly believe they can influence events. Maddi and numerous others have amplified the concept of hardiness in studies of personality attributes that contribute to adequate coping.[36] Contemporary researchers advocate *hardiness* as a synonym for emotional resilience, and believe that hardiness may have a direct effect on a chronically ill person's ability to cope or use social support.[37]

Introspection

The tendency for a person to turn inward, examine self-motivations, and explore personal feelings has been the personality inclination termed introspection. Although this trait has many qualities that would be evaluated as positive and is associated with such qualities as sensitivity, empathy, and creativity, some research suggests that introspective persons tend to be upset by stressful life events and have negative self-evaluations.[34]

Dispositional Optimism

This is the tendency to judge expectations optimistically rather than pessimistically. The cliché example of viewing the glass as half full rather than half empty characterizes dispositional optimism. Perhaps, the extreme of unrealistic dispositional optimism is represented in the cartoon of a person viewing a glass and exclaiming, "I like to think of this glass as one-eighth full." Optimism has been found to be positively correlated with problem-focused coping, the tendency to seek social support, and acceptance/resignation when situations are perceived as uncontrollable. Optimism also has been found to be negatively correlated with denial and distancing. Pessimistic behavior has been associated with a tendency to focus on stress and ventilate feelings, and with coping strategies that imply disengagement from goals.[34]

Nes and Segerstrom did a meta-analysis of 50 studies with a total of over 11,000 participants and found the relation between dispositional optimism and better adjustment to diverse stressors may be attributable to optimism's effects on coping strategies.[38] Dispositional optimism was found to be positively associated with approach coping strategies aiming to eliminate, reduce, or manage stressors or emotions, and negatively associated with avoidance coping strategies seeking to ignore, avoid, or withdraw from stressors or emotions. Obviously, dispositional optimism is more likely to produce advancement to the stage of accommodation and adjustment in rehabilitative struggles, but little empirical research appears to be addressed to chronic neurologic conditions and personality characteristics of survivors. This personality characteristic and its relationship to stroke and aphasia is a critical gap in our literature and research effort.

Locus of Control

This is a psychological construct that relates to the degree that a person believes his or her own actions influence events in the world. Some individuals believe that events are in the hands of fate, luck, chance, or powerful others. Culture and societal beliefs mold these values, and many cultures ascribe much more power to fate and forces beyond the individual's control than is typical in Western culture. Manochiopinig et al, for example, reveal genuine differences in the perception and adjustment to aphasia in the Thai culture.[39] I have noticed this cultural difference myself in the enriching experience of thrice teaching at a medical school in Bangkok over the past several years. One's perception of the locus of control over health matters may be related to compliance, and those who believe that they have no control over health courses or events are less likely to comply with recommendations or courses of rehabilitation. Recent research in Southeast Asian cultures has indicated that the gap between Western and Thai approaches to rehabilitation may be changing somewhat.[40]

Family Systems and Caregivers

Much attention has been directed of late to the concept of family systems theory in health and in illness. As mentioned earlier, many conceive of certain neurologic conditions, such as aphasia, as being a family disease.[5,6] According to family systems theory, illness in one member affects all family members. Communication patterns, roles, and relationships can be vastly altered within families in the presence of chronic illness. Concepts such as the amount of interdependence among family members have been studied relative to its relationship to adaptation to chronic illness.[34] The degree to which a family is *enmeshed* (minimal differentiation among members, all members overly involved with the ill person, and difficulty in determining who is in charge of family decisions) and the degree to which a family may be *disengaged* (rigid subsystem boundaries within the group, with members functioning largely autonomously rather than interdependently) have been found to be important predictors of the ease or difficulty of mobilizing families during rehabilitation. Also, families that can be characterized as having an open system (many social relationships and interactions with religious, school, health institutions) have greater access to social networks and support during attempts to adapt to chronic illness. Families that are more closed appear to be more vulnerable during periods of crisis. Table 2.2 lists some of the characteristics of healthy and vulnerable families.

Not nearly enough consideration has been given to the needs of caregivers of people with chronic illness. Although some research has been conducted on perceptions of families and caregivers, particularly regarding perceptions of aphasia, there is only a shred of ongoing research in this area. Some studies have been done on involving families in the treatment process, with suggestions as to how family roles can be changed to improve treatment.[41] The attitudes, beliefs, perceptions, and knowledge of the disorder may greatly influence the degree to which families can become involved, not to mention the degree of effectiveness of their participation. In aphasia, some research has pointed out the remarkable lack of understanding of aphasia by spouses. In a study of the perception and definition of aphasia by spouses, Plumridge et al found that nearly 24% of spouses thought that aphasia was related to "loss of the mind" or a "decrease in intelligence."[42] Nearly 20% thought that "language loss" was due to the "inability to produce voice," "inability to move the tongue," or the "inability to talk because of low intelligence." Although some of our family education efforts are paying off, it is obvious that we must continue to investigate the knowledge, misperceptions, fears, and attitudes of caregivers and other family members if we are to obtain optimal family participation in the rehabilitation process. Aphasia rehabilitation efforts in Japan, Australia, and the United Kingdom have advanced and advocated the role of the family and conversational partners in aphasia treatment. The family is a vital part of the quality of life of the person with aphasia, as documented by Worrall and Holland and many others.[43]

That spouses can have a positive effect on recovery and adaptation to neurologic conditions seems to be a truism, but not enough research has investigated specifics of the issue. Quality of life issues and the social model of aphasia are redirecting emphasis from an impairment-based aphasia to a new sociology of aphasia. This is emphasized by LaPointe and by Worrell and Holland.[19,43]

In a review of adaptation to chronic illness, Krupp defines adaptation as making the best of a bad bargain.[44] The person who deals successfully with chronic illness is the one who continues to live and function as fully as possible until death is appropriate and inevitable. The challenges of adaptation are suggested to include acceptance of the sick role and compromise. Krupp points out that denial or nonacceptance on the part of a spouse or family can constitute a tremendous adaptational complication. Sometimes, caregivers, physicians, and other health care professionals may fail to recognize the magnitude of the challenge of compromise and too easily criticize the patient for meeting it with difficulty or only partially. This phenomenon was apparent to the author several years ago, when dealing with the perceptions of some family members who commented on the plateauing of change in an uncle who had suffered an extensive left hemisphere lesion with subsequent profound aphasia of many years' duration. Even after careful explanation of the extent of the cerebral damage and rather limited prognosis for recovery of speech, one family member stated, "If he only tried harder and was a little more motivated, he would have regained his speech." Adaptation to chronic illness has received considerable attention since Krupp's pioneering treatise on it. More recently in 2014, Falvo has presented some modified ideas on this difficult adaptation process.[45] Adaptation and acceptance is the target for many with chronic aphasia, and additional research on this concept is welcomed.

An extensive report of family support in rehabilitation reviewed 30 published empirical studies on family support in rehabilitation, and although there were methodologic problems with some of the studies reported, the report concluded that family support is a crucial social support in adaptation and adjustment. Patterson and Garwick presented a cogent review of the impact of chronic illness on families.[46] They suggested that families need to achieve balanced functioning by developing and maintaining their resources as well as

Table 2.2 Characteristics of healthy and vulnerable families

Healthy families	Vulnerable families
Open system	Closed system
Permeable boundary	Tight boundary
Many social institution connections (religious, school, community)	Few social relationships
Well-defined structure	Weak social supports
Access to resources	Restricted access to resources
Receptive to change	Unreceptive to change
Good intrafamily communication	Poor communication

Source: Reproduced with permission from Gregg et al.[32]

their coping behaviors for meeting the continually changing demands that they face. In this report, successful adaptation to chronic illness is suggested to be best promoted by focusing on the family system as the unit of intervention. Another crucial point of emphasis is that families should be encouraged to balance illness needs with other family needs. This issue and the myriad other challenges of the impact of chronic illness on the family constellation are treated in considerable depth in a classic book on the topic, now in its ninth edition, by Larsen.[15]

The Raft

It is clear from the previous discussion that family systems and particularly caregivers are crucial ingredients in adaptation to chronic illness. In this fifth edition of our book, Hancock provides many resources and examines several strategies of intervention for caregivers and families of people struggling with chronicity. Her chapter is an updated and insightful version of resources that are available to survivors, caregivers, and family members.

The now defunct but still available journal of the American Speech-Language-Hearing Association (ASHA) presents an excellent forum on working with families by several contributors.[47] Blosser served as guest editor for this symposium that imparts in-depth discussion of such topics as changes in the American family, suggestions on how people cope, preassessment planning with families, ideas on making time count, reaching families through technology, and forming a therapeutic alliance with older adults. DePompei, one of the contributors, offers a list of suggestions from the literature on how people cope (with the underlying assumption that considerable individual and cultural variability may exist).[47] **Table 2.3** is an adaptation and expansion of this guide with the help of some of my colleagues and students.

Table 2.3 How do people cope?

Embrace a cause	Plant a garden and watch it grow
Develop a new interest, craft, and hobby	Talk to a theologian (priest, minister, rabbi, guru)
Count one's blessings or accentuate the positive	Read spiritual passages
List one's assets	Read inspirational stories
Proceed one day at a time	Meditate
Reduce negative thoughts	Pray
Cultivate and maintain humor	Spend time at peaceful places (woods, desert, rivers, seashore, and mountains)
List strengths of family members; realize you are not alone	Visit house of worship
Dress up	Lunch with a monk
Trivialize the trivial	Monkey with your lunch
Treasure little gains	Interact with old or new friends
Keep a journal	Do things with family
Notice elemental things (sunrise, trees, coffee flavors)	Join support groups
Plot improvements physically	Get involved in community activities
Continue normal routines	Help others
Listen to music, explore new music	Take a course
Play the accordion	Tell a joke
Dance	Watch a goofy movie
Exercise	Go shopping
Fish	Play with kids
Clean house, tidy up, and organize	Visit new places
Cry	Get a pet
Laugh	Read a good book
Rest	Read about the disorder or condition
Eat cleverly	Join local, state, or national organizations or foundations
Walk the dog	Talk to other families with similar problems
Take a hike	Attend a workshop
Soak in the Jacuzzi	Listen to an audiotape or view videotapes; ask questions
Get a massage	Surf the Internet
Play pool	Seek information

Note: Thanks to many of my students over the years who have contributed to this list.

George, Paul, and John were aware in their construction of evocative lyrics that life is bathed in the plea for coping as is evident in **Box 2.3**.

Box 2.3 Pearl

"Give me love
Give me love
Give me peace on earth
Give me light
Give me life
Keep me free from birth
Give me hope
Help me cope, with this heavy load
Trying to, touch and reach you with,
heart and soul"

—George Harrison

Aristos

Accommodation to and acceptance of misfortune in life and of conditions that may not be able to be changed much is a perilous voyage. John Fowles has penned many important works but none as insightful as *The Aristos*.[48] In it, he discusses the necessity of hazard for humankind. What we call suffering, disaster, misfortune, tragedy, or death is the price of living. How we react to these inevitabilities is the measure of how we evaluate and relish our lives. The concept of *Aristos* is taken from the ancient Greek, and it means "making the best of a given situation."

The notion of *Aristos* can aid people on this hazardous voyage. Fowles reminds us that for every wreck there is a raft.[48] Those who have endured neurogenic disorders and conditions and those who help them must never lose sight of the profundity in the rainbow of rehabilitation. If full restoration is not possible, the struggle to improve still is. The process of grappling to optimize and achieve what can be achieved within a given situation is nearly always worth the pain and effort. We have a lot to learn about how to maximize recovery as well as how to attain a state of accommodation and acceptance, but it is extremely important to keep in view the positive end of choosing to get involved with the hardships of others. The Navajo metaphor of dark mist curtains, toil, adversity, frustration, and disappointment can be balanced with equally sanguine images illuminated by LaPointe in earlier editions of this book as well as in his essay on the sociology of aphasia.[19]

Happily I recover.
My eyes regain their power. My head cools.
My limbs regain their strength. I hear again.
My voice restore for me.
In the white of the wings are the footsteps of morning.

For every wreck there *is* a raft. Recovery. Restoration. Relearning. Renaissance. Surmounting barriers. Adjustment. Acceptance. *Aristos*. All of these are good reasons to get up in the morning. They provide not only solace to the individuals who expend the effort to face these storms but also the balance and fulfillment so necessary for the professionals who choose a life of helping others climb the raft and look for shore (**Fig. 2.3**).

Fig. 2.3 *Aristos*[48]: making the best of a given situation ("Remembering Annie," public domain).

Summary

This chapter attempts to humanize the person with aphasia. A person with aphasia is not an "aphasic" or a "stroke survivor" or a disease or catastrophic medical condition. The person is your mother, your grandfather, your brother, your uncle, or you. This chapter reviews the illness experience, coping mechanisms, and the concept of *Aristos*: the ancient Greek word from the works of John Fowles that means "making the best of a given situation."

Chapter Review

- Make a list of the top five things you do to raise your spirits when you are depressed or blue.
- Visit a nursing home or extended care facility. Seek out a resident or client who rarely has a visitor. Chat with him or her for 30 minutes. Ask him or her for advice on how to live a good life.
- Simulate aphasia when ordering lunch at a café or deli. Misname the vegetables and ask for chocolate on them. Maintain a serious demeanor.
- Text a friend and use a jargon or nonsense word for every fifth word. Evaluate the reaction.

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3 Aphasia Theory, Models, and Classification

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Introduction

A theory is generally understood to be "a system of ideas or statements held as an explanation or account of a group of facts or phenomena."[1] While a range of criteria have been proposed regarding what constitutes a proper theory, any theory should allow generalization and should be testable. In essence, a theory should allow predictions beyond the data upon which it was based, and should be able to be falsified. There is no generally agreed upon, unified theory of aphasia, although a few have been proposed. That is, there is no single theory that has gained acceptance in the scientific communities that accounts for the wide range of behaviors that represent phenomena or for the array of persons to whom the term applies. The term *aphasia* applies to language-specific behaviors—not to the anatomy, physiology, cognitive mechanisms, or even communication impairments that may underlie the linguistic behaviors or are the result of the impaired linguistic behaviors.

Theories are closely linked to definitions, models, and classification systems. A good definition is a first approximation to a theory. Although sometimes used interchangeably in the aphasia literature, models differ in some respects from theories as they attempt to visualize or formalize a theory in a way that allows it to be tested; in essence, the model puts the theory to work.[2] For practical reasons, models therefore tend to simplify aspects of the theory and may not factor in particular aspects of the related theory as the emphasis may be placed on the utility of the model to provide a test in the real world. As with theories, the scope and complexity of models varies greatly, from a simple analogy (e.g., lexical access as a file search) to complex computational modeling of relearning.[3] The goals of models vary accordingly, from simulating a specific phenomenon to providing an explanation to possibly predicting behavior. Models can be useful without reference to theory. In aphasiology, there has been a gradual increase in the development of models and less emphasis on broader unifying theories, perhaps reflecting a clinical focus and assumption that such targeted modeling will provide a closer step toward treatment or a window into the difficult encapsulation of the complexities of aphasia within a single theory.

So, for what can models and theories of aphasia be used? The simplest answer is to explain or better understand the phenomena of aphasia. Ideally, such models and theories would enable prediction of the characteristics of an individual with aphasia, given data that constrain the theory. Theories and models of aphasia by necessity have grounding in conceptions of normal language function and also may seek to further this understanding by studying pathological language. Theories and models of aphasia also can incorporate neurobiological considerations, although this is not obligatory. Some claim that certain cognitive neuropsychological theories seek to provide information about assessment, diagnosis, prognosis, and treatment,[4] but this is a contentious issue (discussed later in the chapter), as there are few theories or models of aphasia that actually provide direction regarding the treatment of the language or communication impairment.

The Dominant Paradigm

Although there is no unified theory of aphasia, there is a scientific paradigm under which the majority of scientific community practices. That paradigm is best described as the "classical associative connectionist" paradigm and is best represented by the classification system most frequently in use today across the world (discussed in greater detail later). There are, however, many challenges to this paradigm. Before addressing these, we will briefly describe the neoclassical connectionist model of aphasia that underpins this paradigm. Following the pioneering work of Broca, the models proposed by Wernicke and by Lichtheim and the later reformulation of Geschwind generally assume the existence of language centers located in discrete regions of the brain. A fundamental tenet of this paradigm is that these centers, composed of association cortex,

hold representations required for particular language functions (e.g., speaking, listening, repeating, writing, reading) and that information flows between these centers via dedicated pathways.[5,6] In this formulation, a posterior language center (posterior superior temporal cortex now known as Wernicke's area) holds auditory language representations. A lesion of this center not only impairs auditory comprehension but also influences production. An anterior language center (third frontal convolution of the left hemisphere or Broca's area) was said to hold speech motor representations. Lesions of this center disrupt spoken word production but leave auditory comprehension intact. Disruption of the arcuate fasciculus connecting posterior and anterior language centers is assumed to cause auditory repetition deficits. These models and the rise of the "diagram makers" have been profoundly influential in both neurological aphasiology and clinical aphasiology and still hold sway in various ways today, despite considerable criticism on linguistic and anatomical grounds.[7,8,9]

One key limitation of this dominant paradigm is its attempt to link broad aphasic signs and symptoms to discrete anatomical structures. The classification of aphasia derived from this system (discussed later) vastly oversimplifies the complex array of signs in an individual and does not account for a large majority of patient symptom complexes. There is now clear evidence that language functions are not solely housed in these discrete language centers or that lesions in these areas are consistently associated with deficits in these functions (or vice versa) (for current neurobiological models of language, see the discussion later in the chapter). Critically, language is no longer understood in terms of these rudimentary functions but instead as several identifiable processing components leading to this end point (e.g., word production), with breakdowns occurring in one or more of the subcomponents that contribute to the overall function. The emergence of this approach underpinned the development of cognitive neuropsychological models of language that have emerged as a relatively new paradigm for understanding aphasia (discussed later). Besides offering the chance to specify language impairments with greater precision, and in doing so guide assessment and treatment efforts more practically, one key reason for the rise of these information processing models is that they rarely attempt to attach function to discrete neural structures, thereby avoiding the pitfalls of earlier "diagram makers."[10] Theoretical perspectives that represent substantive departures from the classical associative connectionist model include Brown's *microgenetic*[11] theory and the *unitary linguistic*[9,12] theory (see McNeil and Copland,[13] for a discussion of these theoretical perspectives) as well as the cognitive neuropsychological perspective and computational approaches to modeling aphasia presented in the following.

Cognitive Neuropsychological Models

Cognitive neuropsychological models assume that the study of individuals with brain injury can inform our understanding of normal language processes. According to this approach, the language faculty is understood to be represented and organized in modules that are comprised of several separable components that are domain specific in the sense that they are specialized to process only a certain type of input or representation.[14] These modules are assumed to operate in an encapsulated manner, such that information is processed within a module independent of information processed elsewhere in the system. Critically, it is assumed that brain damage can disrupt a module or processing component selectively, leaving other components to operate normally. The use of such models then allows the identification of the locus of the deficit through hypothesis-driven testing, which may then provide a starting point for therapy. Several such models have been proposed for single word processing[15,16] and sentence processing.[17,18] A typical model of single word processing (e.g., Lesser and Milroy, 1993) provides a means of understanding breakdowns in auditory and visual word processing and includes discrete modules representing stages of acoustic or visual word analysis, phonological and orthographic input lexicons, a semantic system, phonological and orthographic output lexicons, and assembly buffers leading to written or spoken forms. Input and output routes are specified between modules, and alternative routes are assumed to exist for orthographic and phonological conversion without accessing the lexicon. Such models provide a useful means of visualizing the locus of word processing deficits in an individual through hypothesis-driven testing, focusing in part on those tasks that purportedly allow some fractionation of the system.

These information processing models have been particularly influential in providing a framework for assessment and treatment that links clinical concerns with theoretical considerations, as well as providing the psychological underpinnings for the most common signs and symptoms in aphasia. These models are, however, not without their limitations. There is considerable debate regarding the independence or true modularity of the various processing components; and if they are not truly modular, exactly how different do processors interact and what happens within a processing module? The development of these models has been largely built on single case studies, meaning that an assumption is necessarily made that the functional architecture is identical in all brains.[19] Questions have also been raised about the overreliance on somewhat rare double dissociations to confirm fractionation of functions, and it has been argued that in a complex and adaptive neurological system responding to insult, language functions may still be carried out but in a way that does not have clear implications for normal function.[20] Accommodation, adaptation, and neural reorganization following brain injury pose challenges to some of the basic assumptions of some cognitive neuropsychological models. One method for disentangling the near-impenetrable complexity of "doing" language and of the consequences of impairments to it has been the development and use of computational connectionist modeling.

Computational Models

Criticisms regarding the lack of detail or specificity of the interactions between modules of cognitive neuropsychological models have been levied. A more precise understanding of the processing involved has been approached in computational

connectionist, interactive, or parallel distributed processing models that have been applied to aphasia. Computational connectionism holds that various aspects of language can be represented as patterns of activity over interconnected sets of simple neuronlike processing units that occur in a competitive and cooperative fashion.[21,22] It should be noted that the neuronal structure is an analogy and such computational models do not necessarily make claims regarding neurobiology, although there are certain advantages to applying a model with some characteristics of actual neural structures to the study of brain disorders. Patterns of activity within this architecture are enabled through differentially weighted connections between units that may be manually fixed (e.g., in certain interactive activation models) or may develop through supervised learning, commonly involving an error-correcting procedure or "back-propagation" whereby repeated comparisons between the output of a given system and the desired target are made and connection weights are altered to reduce errors.[18,19] Representations within computational models may be distributed (as described earlier) or localized, where the representation such as a word is linked to a specific processing unit. The degree of interaction also varies between models that propose several steps that are both top down and bottom up. That is, interactive activation models, such as the two-step model of lexical access, have been used to model aphasic errors[23,24] and highly interactive models have been used that involve settling upon a representation through a pattern of activity across connected processing units.[25]

Aspects of aphasia have been addressed in computational models where "lesions" are introduced by impairing units, weakening or destroying the connections between units, introducing noise, or accelerating the rate of decay. The value of computational models in aphasia has been tested primarily in terms of how well they accommodate aphasic data. For instance, Martin et al[26] demonstrated that increasing the decay rate in an adaptation of Dell's localist interactive spreading activation model replicated the errors observed in an individual with deep dysphasia (characterized by semantic errors in repetition) and, impressively, that changes in errors with recovery also could be simulated. More recently, two different adaptations of this model were compared in their ability to fit naming errors in a large case series of people with aphasia. It was demonstrated that "lesions" weakening semantic and/or phonological connections were more accurate in modeling variable aphasic naming errors.[21] Such computational models have also been employed to make predictions regarding aphasia treatment and recovery (Martin, Fink, Laine, & Ayala, 2004)[27,28,29] and have provided new insights into how treatment may be approached.[22] Limitations of computational models include the observation that forms of learning involved in certain models are not consistent with actual learning mechanisms and cannot account for certain patient data,[21,29] that the phenomena modeled are necessarily simplistic or narrow interpretations of aphasia, and that damaging these models to elicit language deficits sometimes occurs in ways that are biologically implausible. Some of these issues have been addressed by recent neurocomputational models such as that developed by Ueno et al,[30] which represents a computational model based on patient data that incorporates neuroanatomical data and constraints, based on current models of language.

Contemporary Neurobiological Models of Language

Significant advances in functional and structural neuroimaging over the past 30 years have provided the basis for neurobiological models of language directly relevant to, and in part based on, aphasia. The current dominant paradigm in this respect is the notion of dual (ventral/dorsal) streams supporting language. Such models are acknowledged as being grounded in Wernicke's original conceptualization of two pathways arising from the auditory cortex, and share properties with dual-stream models of auditory and visual processing. According to Hickok and Poeppel's model,[31] following spectrotemporal analysis of speech in the supratemporal plane, phonological processing occurs bilaterally (involving the middle to posterior portions of the superior temporal sulcus) and engages with a ventral stream involving posterior portions of the middle temporal gyrus (with a weak left-hemisphere bias) which acts as a lexical interface, mapping phonological input onto conceptual-semantic representations. The ventral stream also involves a left hemisphere–dominant combinatorial network including the anterior middle temporal gyrus and inferior temporal sulcus. The dorsal stream involves a left lateralized dominant sensorimotor interface (corresponding to an area of the sylvian fissure at the parietal-temporal boundary) linked to an anterior articulatory network involving the posterior inferior frontal gyrus, premotor cortex, and insula (also left dominant).

This model is a notable departure from the traditional conception of brain–language relationships in several respects, including the regions involved (including the anterior temporal lobe and middle temporal gyrus in addition to right hemisphere mechanisms), the language processes accounted for, and its bilateral nature.[32] Diffusion imaging evidence has allowed further refinement of the connections within these proposed streams, suggesting involvement of the arcuate and superior longitudinal fascicle in the dorsal stream and highlighting the role of the extreme capsule in connecting the middle temporal lobe and ventrolateral prefrontal cortex, consistent with the ventral stream.[33]

While various aspects of the model are well supported by converging imaging, lesion, and stimulation data, several of its structural components are still points of contention. It is important to note here that testing and refining such neurobiological models using fMRI (functional magnetic resonance imaging) data in healthy individuals alone is fraught with difficulties, given that such data do not solely reflect regions critical to a given language operation. Instead, advancing and testing such models is increasingly being achieved more fruitfully with large lesion–symptom mapping studies in aphasia. Importantly, the dual-stream model provides a potentially useful neurobiological framework for understanding impairments of phonology and lexical-semantics (production and comprehension) in aphasia. Fridriksson et al[34] recently tested the dual-stream model using voxel-based symptom mapping and principal component analysis (on an extensive range of language measures relevant to this model) in a large cohort of individuals with aphasia (n = 138). The findings were generally consistent with Hickok and Poeppel's model,[31] with two principal behavioral components characterized as reflecting form to articulation

(dorsal stream) and form to meaning (ventral stream) processing deficits with associated lesion locations generally in agreement with the original proposal. Mirman et al[35] also applied voxel-based lesion–symptom mapping and factor analysis on a large range of word production and comprehension measures in a large cohort of individuals with aphasia (n = 99). They found a distinction between both phonology versus semantics and comprehension versus production which supported several aspects of the described dual-stream model but did not find evidence for the involvement of the left inferior frontal cortex, planum temporale, or posterior superior temporal gyrus in the dorsal route supporting speech production.

The discovery of mirror neurons has inspired the development of other neurobiological models of language, based on the assumption that motor systems involved in speech production are also necessarily involved in speech perception and/or that language representations of actions (action semantics) are partially coded by motor systems involved in performing those actions.[36] While such proposals have garnered significant interest and there are clearly links between language and motor systems, there are also numerous fundamental challenges to these claims. These include findings of no activation in relevant motor regions that is specific or selective for action words,[37] failure of damage to motor speech areas to impair speech recognition including in those classified as having Broca's aphasia, and identification of patients with mixed transcortical aphasia who demonstrate intact motor speech systems (and the ability to repeat) with impaired speech comprehension suggesting a problem mapping speech onto lexical-semantic representations.[38] Some have argued that understanding or processing of abstract representations may be facilitated or influenced by sensory–motor associations, but such comprehension is not dependent on the motor system suggesting a middle ground in this debate (Mahon & Caramazza).[39,40] Many uncertainties remain regarding the precise nature of interactions between language and motor systems and the implications for aphasia.

Theories and Models of Aphasia Rehabilitation

Although a comprehensive model may provide a convincing explanation of how particular language processes are impaired in an individual, it provides no specific information regarding how to carry out treatment (e.g., what tasks, what stimuli or responses to target, treatment duration or intensity). Several critical ingredients for successful treatment are not generally integrated into current models relevant to aphasia. Knowledge of relearning, unassisted versus treatment-induced recovery, the neurophysiological modulation of language recovery, and a swathe of motivational and personal factors are often overlooked. There has been increased interest in applying principles of neuroplasticity observed in animals, such as saliency, high intensity, repetition, and learned misuse when developing a useful rehabilitation model for aphasia.[40] However, caution must be applied when translating these principles, as demonstrated recently by Dignam et al,[41] who found advantages for distributed compared to high-intensity aphasia therapy, consistent with distributed learning effects observed in healthy adults, suggesting that principles of healthy human learning must also be considered when developing an approach to aphasia rehabilitation. Applying models of language dysfunction to derive treatment approaches also may reach an impasse when attempting to do this from a purely functional approach treating the brain as a black box. If language treatment on this basis was always successful, one could argue that considerations of the neurobiological mechanisms underpinning treatment and recovery were not necessary, and in fact the clear majority of aphasia treatment proceeds in this way.

However, this argument is problematic given that manipulating the brain mechanisms underlying treatment success may further enhance the *degree* of improvement. For instance, neurobiological adjuncts to aphasia therapy (e.g., pharmacotherapy or transcranial direct current stimulation) may further boost treatment effects.[42,43] More importantly, treatments of aphasia are not invariably successful,[44] with treatment response being difficult to predict based on behavioral observations alone. For example, Van Hees et al[45] observed that patients with predominantly semantic versus phonological deficits did not respond preferentially to treatments targeting semantics versus phonology, respectively. This leaves open the possibility that when treatment fails or the effects are limited, it is one of these missing pieces of the puzzle, such as neural and neurochemical mechanisms associated with treatment success or relearning mechanisms, that needs to be integrated into any model of aphasia treatment for an individual. Basso[46] asserts that a theory of aphasia therapy should at a minimum include (1) a means for deriving hypotheses regarding the functional impairment and a model of the processes to be treated, (2) knowledge of what forms of language impairment are treatable, (3) hypotheses regarding the neural basis of recovery, (4) any other factors besides the brain damage that may affect recovery, (5) a theoretical conception of learning in individuals with brain damage, and (6) critically, exactly how to treat each functional impairment. Although some of these criteria may be addressed to some degree (e.g., 1 and 2), and there is emerging evidence regarding the forms of learning relevant to aphasia rehabilitation,[47,48] the state of current knowledge leaves clinical and theoretical aphasiologists poorly positioned to fulfill many of them in order to develop such a theory. In addition to providing some directions regarding treatment of aphasia, theories and models of aphasia have driven the development of various classification systems.

Classification Systems

Over 30 different classification systems for aphasia have been identified[11] and newer categories such as "Primary Progressive Aphasia"[44,49] and subcategories for existing ones such as "Logopenic Aphasia"[50,51,52] emerge intermittently. These and the other classification systems that have appeared in the literature over the past one and a half centuries are summarized in **Table 3.1**. A classification of aphasia is a taxonomy. The construction of a taxonomy involves the orderly classification of phenomena that occur naturally, into appropriate categories that are assigned suitable and correct names. Aphasic taxonomies are in part numerically derived with the number of shared phenotypic characteristics used to determine the category or class. Each characteristic is usually given equal weight, and the number of

instances of that relevant behavior adds to its scaling to develop a pattern of characteristics across a finite set of behaviors. A good example of this process is seen in the Boston Diagnostic Aphasia Classification System.[53] In this system, a set of eight parameters or behaviors (articulatory agility, phrase length, grammatical form, melodic line, paraphasias in running speech, word finding relative to fluency, sentence repetition, and auditory comprehension) are rated or quantified from observed performance on specific tasks. The magnitude of impairment on each of these tasks determines the rating on that category and the relative magnitude of impairment across behaviors or modalities determines the classification. The reason that these specific behaviors were chosen for the classification system, as opposed to any number of other possible behaviors (e.g., decreased phonological similarity effect, abnormal word length effect, impaired short-term memory span, increased number of sound substitutions, impaired spelling, reading or topic maintenance, reduced number of story propositions), in persons with aphasia is because it is these behaviors that are predicted by the model on which the classification system is based. That is, the Wernicke/Lichtheim model of language (a classical associative connectionist-based model), augmented by Geschwind's modifications, makes explicit predictions about differential impairments of these specific eight behaviors. There is little doubt that these behaviors can, on occasion (as low as 30% when using the Boston Diagnostic Aphasia Examination [BDAE] criteria[53]), be impaired differentially and that patterns do emerge that correspond to the predictions of the model. However, if specific components of language are not represented in discrete "centers" (e.g., semantic operations in Wernicke's areas) or if specific language tasks are not compiled via the specific pathways represented in the model (speech repetition requiring auditorily received speech sounds at Wernicke's area being transmitted via the arcuate fasciculus to Broca's area for speech production), then the classification system might be challenged on theoretical grounds alone. This model has been criticized on just such grounds.[8] However, as stated earlier, good models can be tested. Testing and, when warranted, modification of models are legitimate. Catani et al,[54] for example, have proposed a modification of the Wernicke/Lichtheim model with anatomical evidence that they claim can account for previously unaccounted patterns of behavior subtending the disconnection causing "conduction aphasia." Such model testing and revision is the essence of the scientific method, and when accommodations to the existing model can reasonably be argued, it is difficult to summarily dismiss the model on which the dominant classification system is based. There are, however, other criteria for the establishment of a legitimate classification system for aphasia.

Table 3.1 Summary of aphasia classification systems with reference to assumed anatomical correlates

Classification proponent	Anterior to rolandic fissure	Posterior to rolandic fissure	Subcortical	Other (assume diffuse language representation[a] or diffuse damage[b] or focal other[c])
Broca[55]	Aphemia	Verbal amnesia		
Bastian[56]	Aphasia	Amnesia; word blindness		
Wernicke[57]	Transcortical motor; cortical motor	Transcortical sensory; cortical sensory; conduction	Subcortical motor; subcortical sensory	Total[b]
Marie[58]				Aphasia[a]
Pick[59]	Expressive	Impressive		Amnestic[a]; total[b]
Head[60]	Verbal	Syntactic; nominal		Semantic[a]
Kleist[61]	Word muteness; anarthria	Word deafness; reception	Anarthria	Amnesic[a]
Nielsen[62]	Transcortical motor; Broca's; subcortical motor	Wernicke's; transcortical sensory	Subcortical motor	Amnestic[a]
Goldstein[63]	Transcortical motor; central motor; peripheral motor; peripheral sensory	Wernicke's sensory; transcortical sensory; central	Peripheral motor; peripheral sensory	Amnestic[a]; mixed echolalia[b]
Eisenson[64]	Predominantly expressive	Predominantly receptive		Amnestic[a]
Weisenberg and McBride[65]	Expressive	Receptive		Amnesic[a]; expressive/receptive[b]
Brain[66]	Broca's	Central		Nominal[a]; total or global[b]
Wepman and Jones[67]	Syntactic	Pragmatic jargon; pure word dumbness		Semantic[a]; pure word deafness[c]
Russell and Espir[68]	Motor	Central; alexia		
Gloning et al[69]	Motor	Sensory; conduction		Pure word deafness[c]
Bay[70]	Echolalia; cortical dysarthria	Sensory		Pure[a]

Table 3.1 (*continued*)

Classification proponent	Anterior to rolandic fissure	Posterior to rolandic fissure	Subcortical	Other (assume diffuse language representation[a] or diffuse damage[b] or focal other[c])
Luria[71, 72]	Dynamic; efferent motor	Sensory; acoustic-amnestic; afferent motor		Semantic[a]
Goodglass et al[73]	Nonfluent	Fluent		
Howes[74]	Type A	Type B		
Schuell et al[5]	Group 3 (minor group 1)	Group 4; group 5		Group 1 (simple aphasia)[a]; group 2[a]; group 5[b]
Geschwind[75,76]	Transcortical motor; Broca's	Wernicke's; conduction; anomia; transcortical sensor		
Hecaen and Dubois[77]	Expressive 1; expressive 2;	Sensory 1; sensory 2; expressive 3; sensory 3 (attentional disorder)		Amnesic[a]
Brown[78]	Transcortical motor; agrammatic anarthria	Semantic; phonemic		Anomic[a]
Lecours[79]	Broca's 1; Broca's 2	Wernicke's 1; amnesic (mild Wernicke's); Wernicke's 2; conduction		
Adams and Victor[80]	Isolation of speech area; Broca's; pure word muteness	Wernicke's; conduction' isolation of speech area		Anomic[a]; total[b]; pure word deafness[c]; pure word blindness[c]
Mohr et al[81]	Big Broca's; little Broca's			
Kertesz and Phipps[82]	Transcortical motor; isolation; Broca's	Wernicke's; efferent conduction; afferent conduction; transcortical sensory; isolation of speech area		Anomic[a,b]; global[b]
Hecaen and Albert[83]	Transcortical motor; pure motor	Sensory; alexia with agraphia; conduction; transcortical sensory; mixed transcortical		Amnesic[a]
Benson[84]	Transcortical motor; mixed transcortical; Broca's; aphemia	Wernicke's; alexia with agraphia; conduction; transcortical sensory; mixed transcortical	Aphasia of Marie's quadrilateral space; thalamic; striatal; white matter aphasia	Anomic[a]; global[b]; pure word deafness[c]; alexia without agraphia[c]
Mesulam[49]				Primary progressive aphasia[b]
Gorno-Tempini et al[52]				Logopenic progressive aphasia[b]

Source: Adapted from McNeil.[12]
Notes: See McNeil[12] for additional detailed anatomical localization of lesion assumed for each aphasia type.
[a]Assumed that a lesion in many areas of the left hemisphere would cause the unified syndrome.
[b]Assumed that a diffuse lesion caused the syndrome.
[c]Assumed that a focal lesion caused the primary characteristic within the context of more diffuse damage.

Mayr et al[85] suggest that taxonomy begins with the separation of "… the almost unlimited and confusing diversity of individuals in nature into easily recognizable groups, to work out the significant characters of these groups, and to find constant differences between similar ones." Uniqueness and consistency of group membership are important constraints placed on finding significant characteristics and constant differences. The observation of a novel behavior or even a pattern of behaviors within an individual, even if replicated in another individual, does not make a classification (see, e.g., the creation of the "logopenic aphasia" category). There are rules and forms of evidence that must be followed in order to establish a valid and reliable classification system. Few of the aphasia classification systems summarized in Table 3.1 have been subjected to the

rigors of scientific (psychometric) analysis and hence represent the products of clinical observation and intuition rather than the results of empirically derived characteristics of groups with constant differences between similar ones. The difficulties in constructing a valid classification system in aphasia parallel the difficulty of the diagnosis. As suggested by Akbarzadeh-T and Moshtagh-Khorasani,[86] "Aphasia diagnosis is a particularly challenging medical diagnostic task due to the linguistic uncertainty and vagueness, inconsistencies in the definition of aphasic syndromes, large number of measurements with imprecision, natural diversity and subjectivity in test objects as well as in opinions of experts who diagnose the disease." Nonetheless, both diagnosis and classification in aphasia are possible. With the strict adherence to appropriate psychometric principles of test design and construction, along with careful construction of falsifiable models of aphasia, the development of valid, reliable, and clinically useful classification systems is an appropriate goal and perhaps an achievable objective.

With the challenges imposed by theory and measurement, it is perhaps inevitable that there has been a need to make more finite and manageable the task of finding recurring behaviors that are consistent with the definition and theory of aphasia. Commonly co-occurring behaviors, typically observed clinically rather than derived theoretically, provide the impetus to define a category or class of aphasia. Either a theoretically or a clinically driven motivation for the construction of a classification system for aphasia is legitimate but a reconciliation of the two purposes is necessary for any system to achieve the clinical utility required by the clinician and avoid the scrutiny of theorist. In our judgment, the classification systems available to date have failed to achieve this level of success. Nonetheless, the most frequently used classification systems will be discussed. These include the *fluent/nonfluent* dichotomy and the *classical associative connectionist* (commonly referred to as the *Boston* Diagnostic Aphasia Classification System).

The classical associative connectionist classification system has been objectified as the *Boston* Diagnostic Aphasia Classification System and the Western Aphasia Battery (WAB)[87,88] derived system. These will be discussed together, as the categories are similar, although the criteria for admission to an aphasia "type" are somewhat different. These classification systems represent two derivational mechanisms; one obtained from clinical observation (*fluent/nonfluent*), and one predicted from a model of language performance (classical associative connectionist).

Boston Diagnostic Aphasia Classification

As discussed previously, the classical associative connectionist model is a product of the Wernicke/Lichtheim model, with small but important modifications by Geschwind's[75,76] neoclassical associationism. A brief review of Geschwind's disconnection framework and its contribution to the classification of aphasia is provided by Catani and ffytche.[89] According to Catani and ffytche, Geschwind revived the then dormant associative connectionist model of brain function and added two new components to the classical model. First, he revived and expanded a phylogenetic principle that established the role of association cortices that connected the phylogenetically more mature (with earlier myelinization) primary sensory, motor and interhemispheric areas (the latter two added by Geschwind). The second component added by Geschwind was the freeing from limbic mediation of information exchange between vision, audition, and somesthesis via association cortices. This cross-modal limbic system independence allowed the development of higher cortical functions (e.g., language) by development of association cortices in the parietal lobe, particularly the angular gyrus. Additionally, and perhaps most importantly, Geschwind included lesions of the association cortices as well as the white matter tracts that connected them as part of the disconnected tissue that formed the disconnection syndromes. For aphasia, that would then allow the inclusion of primary sensory and motor association area lesions in the categorization of aphasic syndromes. Although the "classic" aphasia disconnection of the arcuate fasciculus causing a repetition deficit in the presence of intact self-generated speech production (the hallmark of conduction aphasia) was well established before Geschwind's manifesto, the inclusion of association area lesions in the disconnection syndromes made way for a revival of the classical association connectionist model for classifying aphasia.

The differentiation of aphasia "types" according to the neo-associative connectionist classification system as interpreted by the BDAE involves deriving a rating of performance on eight parameters of speech and language production and the average performance on three auditory comprehension subtests (articulatory agility, phrase length, grammatical form, melodic line, paraphasias in running speech, word finding relative to fluency, sentence repetition, and auditory comprehension) rated on a 7-point equal-appearing interval scale or by percentile scores for the sentence repetition and auditory comprehension parameters. The syndromes derived from these behaviors and this test include Wernicke's aphasia, Broca's aphasia, conduction aphasia, anomic aphasia, transcortical motor aphasia, and global and mixed nonfluent aphasia. Importantly, transcortical sensory aphasia was not delineated in 2001 characterization of aphasic syndromes in the BDAE; however, several "pure aphasias" were discussed but without data or specific profiles: aphemia, pure alexia, optic aphasia, pure word deafness, pure agraphia, unilateral tactile aphasia, unilateral agraphia, unilateral apraxia, and hemioptic aphasia. It is important to note that earlier descriptions of the syndromes characterized differences across parameters as "impaired" or "intact"[90,91]; however, the allowance of ranges of impairments on each of the parameters has added a measure of credibility to the categories, as some aphasiologists have argued that these functions are rarely or never unimpaired in persons with aphasia.[12,92]

The tasks within the BDAE and the stimuli used to elicit the behaviors to be rated are based on experimental evidence, assembled over a long time in large measure by Goodglass and colleagues. It is important to note, however, that the motivation for the selection of these tasks, as opposed to the myriad tasks that could be chosen, is based on the neo-associative connectionist model of language impairments in aphasia, and in large measure, it is those behaviors that contribute to their classification. The success in this effort varies from 30 to 80% placement of an individual within one of the categories, depending on the individual assigning the category.[53] It is also apparent that the differentiation of syndromes from these selective behaviors can rest on a very minor difference in behaviors and profile. For example, differentiation of Wernicke's aphasia from conduction aphasia can be based only on the degree of auditory

comprehension impairment despite vastly different proposed mechanisms and patterns of language and communication impairments.

Although the classification system derived from the WAB is a direct descendent of the Boston Classification System, the aphasia categories are not isomorphic between the two. The WAB produces eight categories of aphasia (global, Broca's, isolation, Wernicke's, transcortical motor, transcortical sensory, conduction, and anomic). The aphasia types are derived from test scores on the parameters of fluency, auditory verbal comprehension, repetition, and naming/word finding.[87] Compared to the BDAE, the WAB classifies nearly 100% of the patients because it "forces" individuals into a category. Wertz et al[88] reported that there is only 27% agreement between the two assessments in classification. Importantly, the WAB provides greater directions for test administration and scoring, yielding the opportunity for great examiner and test–retest reliability. The WAB has also undergone considerably greater psychometric development than the BDAE and, unlike its predecessor, has established test–retest reliability. Nonetheless, there remain psychometric issues with the WAB,[93] and the underlying neuroanatomical assumptions of the classification and the language tasks used to elicit the behaviors from which the categories are derived are fundamentally the same.

Fluent/Nonfluent Classification

If we start with the premise that aphasia is, in some form, a disorder involving language, and not speech production at the basic sensory or motor levels, then the systems used to classify aphasia should in some way relate to language and not to sensory or motor processes involved in the externalization of language (i.e., speech). Poeck[94] attributed the origin of the terms "*fluency*" and "*nonfluency*" in aphasia to Wernicke[95] who described two distinct groups of aphasic patients' spontaneous speech production. Wernicke noted that the *fluent* group had poor comprehension, while the *nonfluent* had good comprehension, equating *sensory* and *motor* aphasia to *fluent* and *nonfluent* aphasia. The terms and the dichotomous classification remained dormant and essentially buried for almost a century. Benson[96] was instrumental in its revival. He considered the characteristics of speaking rate, prosody, pronunciation (articulation), phrase length, speech effort, pauses, press of speech, word choice, paraphasia (phonemic, semantic, neologistic), and verbal stereotypes (recurring utterances) as the cardinal features to consider in differentiating persons according to their fluency. Perhaps, the appeal of Benson's[96] study, and the eventual influence it has had on the widespread adoption of the classification system, was derived from the correlation of lesion location (based on isotope brain scans) with "fluency." He concluded that patients with lesions *anterior* to the fissure of Rolando had "*nonfluent*" signs and patients with lesions *posterior* to the fissure of Rolando had "*fluent*" signs.

Using factor analyses and the 10 categories of Benson,[96] Kerschensteiner et al[97] verified the presence of two distinct behavioral clusters corresponding in part to Benson's *fluent* and *nonfluent* categories with the hierarchically arranged features of phrase length, pauses, prosody, speaking rate, and effort. Linguistic processing impairments can reduce the number of words or morphemes that are produced before a pause or break in the phrase occurs. A language impairment could also increase the number of pauses inserted in a connected speech sample or decrease the overall rate of speech because of semantic, syntactic, lexical, or phonological impairments, impair the prosodic contour, and increase the perceived "effort" used to speak. Speech motor impairments also can create each of these features of speech production that are attributed to the language system, and in the absence of careful exclusion of motor speech disorders that frequently co-occur with aphasia (even in persons with lesions posterior to the Rolandic fissure), the classification can be applied to persons with aphasia but for the wrong reasons. For example, the features of phrase length, pauses, prosody, speaking rate, and effort are all associated with, and in some instances used to define, the presence of apraxia of speech.[98] Apraxia of speech, in the absence of co-occurring aphasia, has been found with lesions occurring *posterior* to the fissure of Rolando.[99] The probability of misdiagnosis or misattribution of signs in the application of the *nonfluent* category seems to be high. This does not appear to be the case for the use of the *fluent* category; however, its application frequently results from the absence of *nonfluent* signs rather than the assessment of the same parameters used to determine nonfluency. *Nonfluency*, even when carefully objectified with explicit and measurable criteria that have been employed to date, is difficult to unambiguously assign to any level of the speech production system. That is, deficits of the motor system are at least as likely (if not more likely) candidates for explaining the nonfluency characteristics of the spoken language of the aphasic individual as deficits of the language processing system. Short phrases, poor articulatory agility, and poor melodic line (characteristics used in the *Boston* classification system) are all potential products of an impaired motor system and not necessarily a linguistic system. Careful differential diagnosis *can* differentiate these very different disorders, but they are rarely done in the assignment of aphasia categories and their resultant assignment to mechanisms. There is scarce evidence that the differential diagnosis was done in the selection of the subjects on which these classifications were formed.

The criteria for the classification of *fluent* or *nonfluent* aphasia are frequently undefined by researchers and clinicians, and the criteria are not universally agreed upon. Nonetheless, it is sometimes possible to infer the author's intentions from patient descriptions. Some define "*nonfluent*" aphasia by the presence of agrammatic or paragrammatic speech. It is sometimes used as a substitute for the previously rejected "motor" aphasia category or as shorthand for Broca, transcortical motor, or global categories. Some have used it as a substitute for the presence of co-occurring motor speech impairments. Others define "*fluent*" aphasia by the absence of agrammatic or paragrammatic speech, as a substitute for the previously rejected "sensory" aphasia category, or as shorthand for any one of the Wernicke, transcortical sensory, anomic, or conduction categories or by the absence of a concomitant motor speech disorder. Kertesz[100] attempted to set specific criteria for the diagnosis of "*fluent*" and "*nonfluent*" aphasia as part of the WAB. He attempted to rate *fluency* from primarily language variables, attempting to minimize "… disturbances of prosody and dysarthria, which may not be aphasic features in all cases." He incorporated constructs such as meaningfulness (propositionality), number of words, agrammatism (telegraphic speech), phonemic paraphasias

(jargon), recurring stereotypic utterances, intonation (rhythmic pattern), and "mumbling" and low vocal intensity (the final three of which are consistent with motor speech impairments). Goodglass[91] suggests that the strictest definition of fluency was in terms of the number of words per uninterrupted group (usually less than four words per string, excluding stereotyped expressions) that the patient can "occasionally" produce. In this sense, nonfluency implies a preponderance of paraphasias and word-finding problems. He defined *fluency* by: preserved articulatory facility (agility), preserved prosody (rhythm, rate, and melodic line over the span of single words, phrases, or entire sentences—noting syllabic stress and monotone), preserved grammatical skills including the presence of markers of grammatical relationships, morphological markers of verb tense, person, and number, word order, subordinating constructions, bound morphemes, and omissions or substitutions of functor words.

As with any classification system, the *fluency/nonfluency* dichotomy needs to account for a reasonable array and pattern of phenomena such as lesion locations, psycholinguistic deficits, modality involvements, communicative deficits, and appropriate and differential treatments. In fact, the *fluency/nonfluency* categorization *does not* correspond well to the gross lesion localization of pre- or post-Rolandic fissure—perhaps the easiest of the criteria to meet.[101,102]

Poeck[94] posed and answered two questions about the *fluent/nonfluent* dichotomy in aphasia. He asked about the heuristic (scientific) and practical value of this classification compared to other widely used systems, and about the value the classification holds for research. His answers were essentially "… in the negative" and something approaching "none." He viewed the adoption of this classification system as a conceptual and methodological step backward. Furthermore, Poeck speculated that everybody assumes to know what *fluent* and *nonfluent* speech production is. However, the fact is that it is a highly unreliable diagnosis among professionals, and thus as a shorthand for communicating core characteristics of a patient, it not only has the limitations and invalidities of all classification systems, but also is insidiously dangerous because of its presumed simplicity.

There seems to be a legitimate and perhaps helpful use of the term "fluency" as applied to language competence/processing and motor behaviors. There may be a legitimate and useful application for the differentiation of normal speakers of a particular language. For example, many people are described as "fluent" or "hyperfluent" speakers. There is, however, little agreement among professionals on the characteristics that make them "fluent" speakers. The clinical and research use of the "*fluent*" and "*nonfluent*" aphasia classification is a conceptual step backward and should be discontinued until the construct can be objectified, theoretically justified, empirically validated and found reliable, broadly taught to all relevant disciplines, and found useful clinically for prognosis or some other purpose.

To date, there is insufficient converging evidence to accept one classification system and reject all others. It may be that different purposes for classifying persons with aphasia will yield different classification systems and that a single unified theoretically derived system will remain elusive. What is apparent is that the classification systems proposed and investigated to date leave the theorist and the clinician in search of an adequate taxonomy for any of the purposes for which one might want to put an additional label to the diagnosis of aphasia. Until considerably more evidence is assembled, the person with aphasia may be best served by careful description of their signs and symptoms that are relevant to the problem at hand for which a label would be substituted. That is, lesion location implied by the aphasia type is substantively unnecessary given the sophistication and availability of brain imaging technology. Treatments have not been validated that can be applied to a specific aphasia type. Classification assignment has not been shown to provide prognostic information about expected recovery from aphasia, either assisted (with treatment) or unassisted.

Summary

In sum, the dominant classical association connectionist model of language in aphasia has been challenged and found wanting in many respects by recent neurobiological models of language based on advances in our knowledge of brain structure and function and the consequences of brain lesions on specific language operations. Yet, while the linguistic and anatomical assumptions of the traditional model are clearly oversimplified, inaccurate, and inadequate, the original classification system and its variants based on this model remain steadfast. It is the opinion of these authors that all classification systems proposed to date fail to provide the theoretical justification or the clinical utility for any purposes to which they might serve, to justify their adoption and use. There, however, remains a need to communicate among professionals the salient aspects of the individuals' language and communication abilities and impairments. In spite of the logic and evidence that the classification systems proposed to date fail at every turn, history suggests that it is unlikely that professionals will abandon the notion of "aphasia with adjectives." With this "zombie science," there are, however, some cautions that require consideration when persons with aphasia are assigned a "type." As suggested, three and a half decades ago "if aphasic patients are classified, classification should be done with an awareness of the temptations to see what is not there, to miss what is there and to ignore individual differences."[12]

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4 Neuroimaging and Brain-Based Communication Disorders

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Introduction

Although neuroimaging techniques have been available for almost half a century, the last decade has been marked by an explosion in neuroimaging-related research. Vast improvements in neuroimaging techniques and methods now allow for more detailed assessment of brain structure as well as *in vivo* estimates of brain function in both normal and disordered populations. Following the advent of computed tomography (CT) imaging, it was possible to examine the relationship between brain damage and specific impairment in function. The field of aphasiology has benefitted from this improved technology, and several early studies examined the relationship between localized left hemisphere damage and aphasia types and severities.[1–3] More recent developments in nuclear health physics provided techniques to examine brain function associated with task-specific processing.[4] Unlike previous neuroimaging techniques, which only examined brain structure, techniques such as positron emission tomography (PET) and single-photon emission computed tomography (SPECT) allowed for functional neuroimaging. For example, using functional neuroimaging, it became possible to appreciate what brain areas are selectively recruited for speech and language processing.

Improvements in CT and PET methods not only have informed the relationship between brain structure, brain function, and behavior but also have improved diagnosis and treatment of various neurological events and disorders such as stroke and multiple sclerosis. Nevertheless, the discovery of nuclear magnetic resonance as a way to image the human body[5,6] brought about a surge in both structural and, later, functional neuroimaging.[7] In fact, the bulk of contemporary neuroimaging research relies on magnetic resonance imaging (MRI) as a way to assess both structure and function in the human brain. Today, MRI presents a powerful tool commonly used in the medical management of patients.

In addition to the aforementioned neuroimaging methods, several other techniques have emerged as a means to assess brain function. Electroencephalography (EEG) and magnetoencephalography (MEG) are two methods used to measure cortical brain waves associated with neural firing at the level of the scalp. These methods have good temporal resolution, allowing for real-time assessment of cortical activity during the execution of a specific cognitive task (for a review of the principles underlying EEG and MEG, see Buzsáki[8]). In addition to these methods, direct brain stimulation has also emerged as a part of the arsenal of tools available for the examination of brain function. Transcranial magnetic stimulation (TMS) allows for direct stimulation of the brain, whereby localized stimulation of the cortex allows for transient interruption of brain function in the targeted area. This enables direct examination of task impairment while a given cortical area is "paralyzed" for a very brief time period. Although TMS should not be viewed as a neuroimaging technique *per se*, it provides similar and sometimes complementary data to the more traditional neuroimaging methods such as CT and MRI.

Neuroimaging has become a staple in the study of aphasia and other related brain-based communication disorders. The following sections discuss specific neuroimaging techniques, followed by delineation of how each method has been used in the study of aphasia or a related disorder. We will focus first on structural neuroimaging techniques, followed by functional imaging techniques. It is important to note that this chapter should not be viewed as a thorough primer of the physics related to the prospective neuroimaging methods. Rather, the focus here will be primarily on clinical utility and application.

Structural Neuroimaging

CT in the Clinical Management of Stroke

CT scanning (also known as computerized axial tomography [CAT] scan) has been around since the early 1970s. It relies on X-ray imaging and allows for detailed visualization of the brain in three-dimensional space (**Fig. 4.1a**). Although CT of the brain includes significant radiation exposure, it is far cheaper